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Reichman's Emergency Medicine Procedures

Third Edition

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Preface

Emergency Medicine is extremely broad and the advances have been amazing in recent years. The field covers the neonate through the geriatric, surgical and medical, and encompasses all organ systems. Emergency Medicine is rapidly evolving. Procedural skills must supplement our cognitive skills. Achieving proficiency in procedural skills is essential for the daily practice of Emergency Medicine. We have produced a clear, complete, and easy to understand textbook of Emergency Medicine procedures. This new edition addresses the diverse topic of Emergency Medicine. This text will provide medical students, residents, advanced practice practitioners, and the seasoned Emergentologist with a single procedural reference on which to base clinical practices and technical skills.

The primary purpose of this text is to provide a detailed and step-by-step approach to procedures performed in the Emergency Department. It is expressly about procedures. It is not meant to be a comprehensive reference but an easy to use and clinically useful procedure book that should be in every Emergency Department. The contents and information are complete. It is organized and written for ease of access and usability. The detail is sufficient to allow the reader to gain a thorough understanding of each procedure. When available, alternative techniques or hints are presented. Each chapter provides the reader with clear and specific guidelines for performing the procedure. Although some may use this text as a library reference, its real place is in the Emergency Department where the procedures are performed. Despite its size, I hope that this book will find its way to the bedside to be used by medical students, residents, advanced practice providers, and practicing clinicians.

This book will satisfy the needs of a variety of backgrounds and training. While this text is primarily written for Emergentologists, many other practitioners will find this a valuable reference. This book is written for those who care for people with acute illness or injury. Medical students and residents will find this an authoritative work on procedural skills. Medical students, residents, nurse practitioners, physician's assistants, and practitioners with limited experiences will find all the information in each chapter to learn the complete procedure. Family Physicians, Internists, and Pediatricians will find this text useful to review procedures infrequently performed in the clinic, office, or urgent care center. Intensivists and Surgeons involved in the care of acutely ill patients will also find this book a wonderful resource. The experienced clinician can get a quick refresher on the procedure while enhancing their knowledge and skills. Physicians actively involved in education will find this text an easy to understand and well-illustrated source of didactic material.

The book is organized into sections with each representing an organ system, an area of the body, or a surgical specialty. Each chapter, with a few exceptions, is devoted to a procedure. This should allow quick access to complete information. The chapters have a similar format to allow information to be retrieved as quickly and as efficiently as possible. There are often several acceptable methods to perform a procedure. While alternative techniques are described in many chapters, we have not exhaustively included all alternative techniques. Key information, cautions, and important facts are highlighted throughout the text in bold type.

Each chapter, with a few exceptions, has a standard format. The relevant anatomy and pathophysiology is discussed followed by the indications and contraindications for the procedure. A list is provided of the necessary equipment. The patient preparation including consent, anesthesia, and analgesia is addressed. The procedure is then described in a step-by-step format. Cautions are placed where problems commonly occur. Alternative techniques and helpful hints for each procedure are presented. The aftercare and follow-up are discussed. Any potential complications are described including the methods to reduce and care for the complications. Finally, a summary contains a review of any critical or important information.

This book covers a wide variety of procedures that may be performed in a rural or urban Emergency Department. This includes procedures performed routinely or rarely; procedures that are often performed in the acute care, clinic, and office settings; procedures that are performed frequently in the daily practice of Emergency Medicine; and procedures that are seldom to rarely performed but critical to the practice of Emergency Medicine. Some procedures are uncommon, may not be known to the reader, and provide an opportunity to acquire new information that may be converted with proper practice and training into a useful skill. A few of the procedures are performed only by Surgeons and are included to promote understanding when the patient presents to the Emergency Department with a complication. This new edition has added chapters, algorithms, clinical pictures, cutting-edge technological advancements, radiographs, and tables based upon readers' comments, input, and suggestions.

We have drawn on a wide variety of authors. The majority of authors are residency-trained, board-certified, and practicing Emergentologists. We have the honor of having some contributors from outside the field of Emergency Medicine and who are experts in their own specialty. All authors do have biases because of differences in education, experience, and training. We have tried to base all recommendations on sound clinical and scientific data. However, we have not excluded personal experience or preferences when appropriate. In these cases, the authors also present alternative techniques.

This book has grown and changed with this third edition. I am happy and privileged to edit this third edition of the text. Continued input and suggestions from you, the reader, would be most appreciated. Let me know what additional procedures should be included or excluded in the future. Any errors, in the end, are mine. Please let me know of any mistakes or omissions, big or small, at eric.f.reichman@gmail.com.

Eric F. Reichman, PhD, MD

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Susan Gilbert is a wonderful medical illustrator and friend. Her input and assistance only added to the illustrations of the editions of this book. Working with her was easy, fun, and simple.

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SECTION 1

Introductory Chapters

1 Informed Consent

Eric Isaacs

This chapter is designed as a practical reference for the Emergency Physician (EP). It focuses on the unique challenges of informed consent in the Emergency Department (ED). It presents a practical guide for the informed consent process, reviews the exceptions, and offers suggestions on difficult scenarios of informed consent in the ED.

INFORMED CONSENT

The right of a patient to make decisions about their body, including the refusal of recommended procedures and treatment, is an important concept in medical practice with foundations in law and medical ethics. **Informed consent is the process of communication that demonstrates respect for a patient's right to make autonomous decisions about their health care. Informed consent is an ethical practice and a legal requirement for all procedures and treatments.**[1]

UNIQUE CHALLENGES OF INFORMED CONSENT IN THE ED

Each practice environment presents its own challenges to the process of obtaining informed consent. **Physicians frequently fail to fulfill all the requirements of obtaining informed consent.**[2-4] The ED presents significant challenges, which despite assumptions to the contrary, results in a greater need to spend time delivering information and engaging patients in their care decisions to the extent possible (**Table 1-1**). Time pressure and acuity are the most critical factors that influence the care paradigm in the ED. Care provided in the ED spans the full continuum of care as nonacute care is increasingly sought in the ED. Care in the ED addresses the full spectrum of society with patients from diverse health literacy, language origins, socioeconomic backgrounds, and recognized vulnerable populations (e.g., children, elderly, and prisoners). EPs need to be prepared to address the broad clinical needs of diverse patients under pressure without the traditional physician–patient relationship. Systemic constraints exacerbate this challenged professional context as patients have no choice in the treating physician or the treating facility. The location to transport the patient is often dictated by prehospital protocols. Tension may arise when a patient's wishes conflict with greater societal or institutional needs for efficiency and protocol compliance independent of the patient's preferences and needs. Examples include a trauma activation or a public health emergency. Increasing space constraints and crowding found in most EDs create a lack of privacy that can impede the free exchange of sensitive information. Procedural interventions in the ED are often concurrently diagnostic and therapeutic, further complicating informed decisions.

TABLE 1-1 Challenges for the EP to Spend Time Engaged in Conversation with a Patient

Lack of facility choice
Little privacy
No prior relationship
Pace of care challenges lay person decisions
Public health or system-imposed constraints
Time pressure

The torrent of complex medical information physicians provide patients is overwhelming in the most controlled settings. It is only made worse in the high-emotion and high-stress environment of the ED. EPs often make rapid decisions with limited information. Many of our colleagues in other specialties may not share this skill. The EP's expectations of patients must be equally, if not more, tolerant. The absence of an ongoing physician–patient relationship offers no basis upon which to build trust, elicit values, and draw preference knowledge. Lack of a prior relationship tests the ability to establish an immediate rapport with patients and renders patients' ability to express their values most important.

There may not be time to ponder the intricacies of medical ethics in the ED or to satisfy all the requirements of searching for the best surrogate decision maker when there is uncertainty about a patient's preferences or a potential refusal. Many EPs will default to doing as much as possible in these difficult situations.[5] There is often enough time to make a considered decision before acting in the most aggressive fashion. While some say that it is easier to withdraw care once the clinical picture becomes clearer, this aggressive course of action must be balanced with the knowledge that EP may be performing a painful or unwanted procedure on a patient who has previously made their wishes clear. Informed consent was often bypassed in the past under the presumption that a patient would want aggressive treatment. The scope of ED care and societal norms have shifted in recent years. **Informed consent for procedures in the ED needs to reflect the current standards of practice.**

LEGAL FOUNDATION FOR INFORMED CONSENT

Consent originates in the legal doctrine of battery (i.e., touching of the body without permission). The notion of protecting a patient from the bodily trespass of a procedural invasion was framed by Justice Cardozo in 1914: "[e]very human being of adult years and sound mind has a right to determine what shall be done with his own body; and a surgeon who performs an operation without his patient's consent commits an assault, for which he is liable in damages…."[6] By 1957, the notion of consent shifted from mere permission to an authorization following "the full disclosure of facts necessary to an informed consent."[7] Emerging at the same time as the bioethics movement's shift away from paternalistic medicine toward a patient's rights focus in medicine was *Cantebury v. Spence*.[8] **This case resulted in an appeals court establishing a physician's duty to disclose the risks and benefits of a procedure and its alternatives and introduced the reasonable patient standard. The reasonable patient standard is what a reasonable patient would need to know to make an informed choice, shifting away from the professional standard, what most physicians deemed necessary.** The standard for disclosure today varies by state.[9] As a result of the informed

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consent "duty," the legal and risk management function of informed consent (i.e., consent process that meets institutional and/or legal parameters for formal recognition, referred to as "effective consent") overshadows the ethically driven process of informed consent (i.e., consent as a communication process that demonstrates respect for a patient's autonomy, referred to as "autonomous authorization"). These two aspects serve distinct functions that are often conflated under "informed consent." Both are necessary for valid informed consent and are addressed separately throughout this chapter.[10]

The exception presuming permission to treat in an emergency has equally deep roots. Justice Cardozo's opinion continues, "[t]his is true except in cases of emergency where the patient is unconscious and where it is necessary to operate before consent can be obtained."[6] In *Canterbury v. Spence*, "the emergency exception" is included as a privilege from the duty to disclose when "the patient is unconscious or otherwise incapable of consenting, and harm from a failure to treat is imminent and outweighs any harm threatened by the proposed treatments." It also states that a "physician should, as current law dictates, attempt to secure a relative's consent if possible." **In the emergency context, one may presume permission: (1) to do what is necessary when (a) there is imminent harm from nontreatment and (b) when harm from nontreatment outweighs the harm from the proposed intervention; (2) where the patient is unconscious or unable to participate in care decisions; and (3) when the patient's preferences are not known and no surrogate is immediately available to provide authorization.**[11]

ETHICAL FOUNDATION FOR INFORMED CONSENT

In an era of patients' rights and shared decision-making, robust informed consent reflects a process of communication that secures that a patient "gives an informed consent to an intervention if (and perhaps only if) one is competent to act, receives a thorough disclosure, comprehends the disclosure, acts voluntarily, and consents to the intervention."[9]

It is not uncommon to encounter the challenge of a patient refusing a recommended procedure or intervention in a health care environment where there is an expectation for more active patient participation in health care decisions. Central to a strong patient–physician relationship is the desire to promote patient well-being and simultaneously respect patient autonomy. Conflict between EPs and patients may arise when views of what is in a patient's best interest differ between them. EPs with the greatest integrity come to work with the intention to act in the best interests of patients, and do so with a focus on the prevention and eradication of disease to preserve life and improve disability. **The patient may consent or refuse the recommendation after an EP has fully informed a capable patient about an intervention in an understandable way. An initial refusal of recommended treatment should begin a critical conversation that confirms all the elements of an informed refusal. The informed refusal process will respect patient autonomy by accepting a patient's view of well-being and may require honoring a refusal of the recommendation.**[12]

EP'S ROLE AND GOALS IN INFORMED CONSENT

The EP's role in the informed consent process is to provide patients the information needed to make their own decisions. Provide written sheets that cover all aspects of the procedure if available. It is important not to overwhelm a patient with too much information or complex clinical decisions. **Including patients in appropriate care decisions (e.g., the informed consent process for a procedural intervention) is an ethically important goal.**

TABLE 1-2 The Goals of EPs in the Informed Consent Process

Allow autonomous authorization (patient may consent or refuse)
Give information (more than we think we need to give)
Make information accessible
Offer guidance in weighing information
Support patients to make their own decision

EPs must pay attention to the informed consent process to accomplish the goal of respect for autonomy (Table 1-2). EPs need to provide more information to the patient than they think is needed. Research indicates that patients need more information than physicians think they need to feel "informed" in the decision-making process.[13] The need for a procedure seems obvious to the EP, and the balance of the considerations clearly tips in the favor of "do it." EPs must slow down to fully explain the rationale for their recommendation with patients and to offer patients information that allows their meaningful consideration of the recommendation so that they can reach their own decisions. A good guideline is to offer more time and information for procedures carrying greater risk.[14] **EPs must make the effort to work against the features of the ED (Table 1-1 and presumption of consent). Allow patients capable of engaging in their care decisions to express autonomous authorization.** This is achieved by giving patients sufficient information, in an understandable way, and by honoring their decisions.

COMPONENTS OF THE INFORMED CONSENT AND INFORMED REFUSAL PROCESS

Informed consent is the communication process that demonstrates and protects a patient's self-determination by providing a patient with decision-making capacity with sufficient, understandable information and allows the patient to make a voluntary, knowledgeable decision. There are five requirements that must be satisfied.[9] These include the patient having decision-making capacity, the EP providing sufficient information, the patient understanding the information, the patient giving consent in a voluntary fashion without coercion, and the patient communicating their decision (**Table 1-3**).

DECISION-MAKING CAPACITY

The terms "competence" and "decision-making capacity" are frequently used interchangeably, but their strict meanings are different. **Competence is a legal term with broader applications related to financial matters and the determination of personal choices. Decision-making capacity is a clinical term that speaks to the specific capacity to make a clinical decision.** Many people who are legally "incompetent" retain health care decision-making capacity. **If the patient does not have decision-making capacity, informed consent cannot be obtained and it must be obtained from a surrogate decision maker, or the patient may fall into an exception from informed consent.**

TABLE 1-3 Requirements of the Informed Consent Process[9]

1.	Does the patient have the decision-making capacity to make this decision?
2.	Has there been disclosure of relevant procedural information (including risks/benefits for intervention, alternatives, and nonintervention)?
3.	Has the information been presented in a way that is understandable to the patient?
4.	Has the information been presented in a way that allows the patient to make their own decision voluntarily while still being informed of the physician's recommendation?
5.	Has the patient communicated a decision?
6.	Does an exception apply?

DETERMINING DECISION-MAKING CAPACITY

The determination that a patient has decision-making capacity is at the core of informed consent. By default, EPs assume that a patient has capacity and confirm this through routine dialogue with the individual. Confirm six elements when there is a question about a patient's capacity to make an informed decision about procedures or treatment.[15] The patient must be able to: understand and process the options, weight the benefits and risks, apply a set of values and goals to the decision, arrive at a decision, communicate a choice, and demonstrate capacity to make the decision (**Table 1-4**).

Determination of capacity is a clinical decision based on the judgment of the EP regarding the patient's actual level of functioning and appreciation of the ramifications of the clinical situation.[16] The degree of capacity needed to understand risks and benefits of suturing a finger laceration differs from a cardiac catheterization. A patient may be able to understand one choice but not another. An Alzheimer patient who is pleasant, oriented to place, and oriented to year may be unable to appreciate the consequences of a decision. This patient may have capacity for some tasks but may lack the capacity to consent for a specific procedure (e.g., lumbar puncture).

The EP needs to assess the ability for the individual to weigh the risks considering their (i.e., the patient's) own values. An example would be the ramifications of a fracture reduction on the dominant hand. A construction worker or musician may decide different than an individual whose livelihood does not depend on perfect hand function.

A recognized element of decision-making capacity is whether the patient's decision is consistent over time. This is not necessarily applicable specifically to the ED. A possible heuristic is whether the decision is consistent with the person's narrative and values as expressed consistently over time in life choices. The decision-specific nature of capacity acknowledges that the level of capacity needed depends upon the complexity of the decision, with greater capacity needed for decisions with graver consequences. **The degree of capacity needed to consent does not necessary equal the degree of capacity needed to refuse a recommended intervention.**[12] Informed refusal will be discussed later in this chapter.

Decision-making capacity is a dynamic process and changes depending upon the patient's evolving condition and task in question. The ED patient may be able to participate to a greater or lesser extent depending on fluctuations in their condition and alterations of their sensorium from the administration of medications. Make efforts whenever possible to enhance the patient's decision-making capacity (e.g., reduce pain medication temporarily or visit patients at optimal times) to engage them to the fullest extent possible in their care.

Emancipated minor and adolescent laws vary from state to state.[9] **Emancipated minors are legally recognized as adults and responsible for their own finances and care.** They can provide fully informed consent. Know the local laws where minors who are not emancipated may give consent for sensitive conditions or procedures (e.g., those of a reproductive nature or substance abuse).

Informed consent may not be possible with some populations (e.g., young children and elderly with dementia). It is still possible to inform these patients of the procedure and to engage their assent. **Unlike consent, assent is not determinative.** It does offer the possibility of the individual participating in their care.[15]

TABLE 1-4 Elements to Determine if a Patient Has Decision-Making Capacity

1. The patient is able to understand and process the options presented.
2. The patient is able to weight the relative benefits, burdens, and risks of the options.
3. The patient is able to apply a set of values and goals to the decision.
4. The patient is able to arrive at a decision that is consistent over time.
5. The patient is able to communicate a choice.
6. The patient demonstrates capacity appropriate and sufficient to make this decision.

PATIENTS LACKING DECISION-MAKING CAPACITY

It is not possible to obtain informed consent when a patient lacks decision-making capacity. Necessary treatment may be provided to patients who lack decision-making capacity without obtaining the patient's informed consent. Make every effort to learn the patient's previously stated preferences for treatment (e.g., written advance directives or communication with a primary care provider). **Make efforts to obtain consent from a surrogate decision maker if prior preferences are not available.** A surrogate decision maker is a person entrusted with making health care decisions because they know the patient best and can bring the patient's values and goals into the clinical decision process. This role can be challenging for even the most capable decision makers. It is not uncommon for surrogates to have a role conflict between applying their own values and/or wishes and those of the patient.

EPs must pay attention to the language used when asking a surrogate decision maker for consent. Frame the discussion with phrases asking what the patient would want in the situation, such as "How would your father view this situation?" or "What would your father's preference be based on his values?" Avoid general phrases such as "What should we do?", "What do you want us to do?", or "What do you think he would want?" An EP can ask the surrogate "Why do you think he would choose that?" if the decision seems to stem from a role conflict. No prior conversation covers every clinical scenario perfectly, and the gravity of the decision can frequently be overwhelming for the surrogate.[12]

The choice of a surrogate decision maker may be obvious in some cases (e.g., the parent or legal guardian of a child). The choice can be more complex in other cases. Who may serve as a surrogate and their scope of authority varies by state. What if the appropriate surrogate is in question and there is no statutory guidance? A useful guide is that the surrogate's authority arises from a close relationship to the patient that affords accurate and informed communication of the patient's values. Refer challenges in resolving conflict between potential surrogates (e.g., siblings with different opinions regarding parental care) to an ethics committee or other institutional mechanisms to offer guidance unless emergent conditions make that impractical.

INFORMATION TRANSMITTAL

The EP must relate sufficient information about the procedure to the patient. This raises the questions of what information to present and how much to present. **Relevant information includes the risks and benefits of the procedure, any alternatives to the proposed course of action, and the consequences of nonaction.** The question remains how much information needs to be disclosed to patients, particularly considering the potential that legal action may be taken if an EP does not obtain informed consent properly.[17]

There are two standards that are commonly used, and these vary by state. The traditional "professional standard" requires the EP to provide information based on what the profession's standard of practice would deem necessary to disclose for a patient to be informed. The more common "reasonable person standard" requires the EP to include all the information that a reasonable patient would want to know to make a knowledgeable decision. Information that should be communicated includes: the patient's current medical condition and how will it progress if no treatment is given, the treatment

alternatives, the risks and benefits of each potential treatment and their probabilities, and the financial costs of each if those estimates exist. Finally, the EP should provide a personal recommendation as to the best alternative.[9]

UNDERSTANDABLE PRESENTATION OF INFORMATION

Information must be given in a way that is understandable. The patient must be able to adequately weigh the benefits, burdens, and risks of the treatment in the context of their own beliefs, goals, life, and values. The obvious differential in knowledge and understanding between patients and EPs may be exacerbated by language barriers, literacy, low educational levels, and numeracy.[18] Such barriers may be overcome by speaking at a level easy for the patient to comprehend, being sensitive to patients who may be unable to read, and being sensitive of patients who may not be highly educated. Understanding is bidirectional and necessitates that the EP confirms that the patient understands what they are told.[19] Communicating numbers (e.g., risk and probabilities) is the most complex task asked of the EP.[20] Frame numbers in multiple ways and present outcomes in positive and negative contexts to enhance informed consent.[20] For example, "three out of four children have no side effects, but one in four will have nightmares from this medication."

Language barriers are frequent in the ED and pose significant concern in obtaining and documenting informed consent.[21] Understanding languages is situational. It is imperative to know when to call an interpreter even though some EPs may have additional non-English language proficiency. Limited language skills allow the EP to extract some critical clinical information. Patients may need more information than the EP's skills allow. **Calling an interpreter may be essential for meeting a minimum standard of care.[22]**

VOLUNTARY NATURE OF THE DECISION

Forced treatment where any real choice is removed from the patient being involved in the decision-making process violates the doctrine of informed consent. Any form of coercion based on threats or intolerable consequences (e.g., the withholding of pain medication) would fall into this category. **EPs cannot manipulate patient decisions by withholding or distorting information that the EP believes may sway the patient toward a preferred course.** Persuasion is permissible. It is an obligation as trained professionals to synthesize the information and recommend a course of action. An appropriate recommendation includes laying out the risks, benefits, and reasoning behind the recommendation as well as explaining the reasoning for not selecting an alternate approach. EPs can utilize the resources of the patient's family or significant others to provide arguments in favor of a course of treatment. The EP must be careful to avoid overwhelming the patient, as the goal should be a shared solution by consensus and not forcing the patient to surrender to the wants of others.[9] Strategies to approach a patient's refusal are discussed in depth later in this chapter.

EFFECTIVE INFORMED CONSENT AND REFUSAL

There is a difference between the autonomous authorization informed consent (i.e., information and dialogue) and the effective informed consent (i.e., to meet legal and institutional requirements). **Document the discussion of the benefits, burdens, risks, and alternatives addressed in the discussion with the patient for the autonomous authorization to be recognized as effective and the entire informed consent to be valid.** Reference local institutional policies to confirm an effective informed consent or refusal.[10]

Some hospitals have patients sign "blanket" consent forms agreeing to all emergency tests and treatments upon their registration in the ED. Such consent forms provide no information regarding specific individual procedures.[23] These forms are not acceptable because they fail to respect patient autonomy. **Blanket consent forms cannot substitute for the usual informed consent process for procedures in the ED, where a dialogue with the patient is required.[24]**

EXCEPTIONS TO THE INFORMED CONSENT PROCESS

EMERGENCY EXEMPTION

Society's overriding assumption is that a person would want lifesaving treatment in an emergency. Consent to treatment is generally presumed under specific emergency circumstances where intervention is necessary to save life or limb, the harm of nontreatment is greater than the harm of the intervention, a patient is unable to participate in care decisions, and patient preferences are not known with no surrogate available. **This emergency exception is not absolute.** This is particularly true when there is clear evidence that the patient's wishes are contrary to the intervention being considered (e.g., prehospital advance directive or a wallet card stating no blood transfusions).

Some EPs believe that any patient in the ED qualifies for an emergency exception by being in the ED. This is not true. **Location by itself cannot be used to justify the emergency exception or to infer an "implied consent" for broad ED care. The emergency exception may be invoked only when the patient will be harmed by the delay necessary to obtain informed consent.[25]** The EP should ask themselves a few brief questions to determine if a patient meets the criteria for an emergency exception to informed consent (**Table 1-5**).

THERAPEUTIC PRIVILEGE

The therapeutic privilege is a disfavored concept but recognized exception. It excuses the EP from the duty to disclose in the limited circumstances where disclosure might create harm to the patient and interrupt the treatment process. This privilege is rarely invoked as it could almost negate the entire informed consent process. Therapeutic privilege may be applied when direct disclosure to a patient would create harm, generally recognized as occurring in some psychiatric conditions and for some cultural groups.[9]

WAIVER OF INFORMED CONSENT

The EP has a duty to disclose information. Patients may differ in how they approach their participation in care decisions. Some patients may prefer that another person (e.g., a close family member) receive health care information and make treatment decisions on their behalf (i.e., delegated autonomy). This may be due to personal preference or cultural variation. The delegation of the decision-making

TABLE 1-5 Questions to Justify an Emergency Exception

1. Will failure to treat quickly result in serious harm to the patient?
2. If their condition worsens, will the patient die or suffer serious harm before definitive care can be delivered?
3. Would most capable and reasonable people want treatment for this type of injury?
4. Is the patient unable to participate in care decisions?
5. Are the patient's preferences known or knowable in a timely way from a surrogate?
6. Is there any evidence that the patient would refuse this specific treatment?
7. Would failure to treat result in greater harm than the proposed intervention?

must be confirmed with the patient and not assumed based on cultural norms. The delegation reflects a patient's right to waive informed consent. **Honor the patient's choice to delegate that right to another person as it demonstrates an autonomous choice.**[15]

Some patients may interrupt the informed consent process after only partial information is disclosed and elect to follow the recommendation. If the EP confirms the patient's acceptance of the consequences of consent with only partial information, the EP may accept this as consent via waiver of the informed consent process.[25] The EP may accept a waiver of consent if the patient has capacity, understands that they are giving up an important right, and has made the request voluntarily. The EP who is uncomfortable with this responsibility may ask the patient to designate another person to assume this role.

IMPLIED CONSENT

Implied consent is a disfavored concept. It may be considered to "apply" in the very limited circumstances when an EP is undertaking a clinical activity with a well-known risk-benefit profile.[26] The most favored implied consent example is when a patient extends his arm for a blood draw. The volitional act of extending the arm is deemed as implied consent to the blood draw and its risks (e.g., pain and possible bruising). The assumption of "implied consent" poses a dangerous trap for the EP. **What an EP considers routine and well-known risks may differ greatly from what the patient knows.** This is particularly true in the ED where there is little trust and no knowledge of the patient's health literacy.

Emergency Medicine research shows at least 50% of patients wanted time spent on "detailed" information, including a review of the risks of only 1% chance of occurrence. For example, lumbar punctures are clinically safe and pose little risk. The patient perceives lumbar puncture as an invasive procedure that requires more information for informed consent.[13] **Implied consent is not sufficient when informed consent is required or possible.**[12]

UNREPRESENTED PATIENTS OR THE PATIENT ALONE

A patient who is unable to participate in care decisions and has no surrogate decision makers is known as the "unrepresented patient" or the "patient alone." These highly vulnerable patients have no social networks to assist the care team in navigating consent and care decisions.[27] Attention to clinical decision-making for this patient population is growing.[27] Statutory guidance on decision-making for this patient population varies by region. Review institutional policies to determine whether a policy exists for decision-making for the "unrepresented patient." Consultation with the ethics service is recommended in the absence of a policy, and make efforts to develop a consistent and transparent approach to care decisions for this vulnerable population.[15]

INFORMED REFUSAL

The EP often begins with the presumption that patients possess decision-making capacity to consent and refuse procedures. The EP may question a patient's capacity in clinical practice more readily when the patient disagrees with recommendations.

UNDERSTAND THE REASONS FOR THE REFUSAL

A refusal for a recommended intervention should be the beginning of an important conversation with the patient. A refusal of a recommendation when first proposed may seem a rebuff or potential time challenge. Approach a refusal with openness and curiosity. Help the patient not feel cornered into following the recommendation while confirming their informed refusal. A refusal is an opportunity to learn how to practice persuasive reasoning. A patient might have misheard numbers, or the proposed procedure may resemble a prior negative experience during the barrage of information disclosure. **Take time to listen to the patient's concerns and reasons for refusal.** This can help navigate the informed refusal process.

CONFIRM THE ADEQUACY OF INFORMATION WITH AN EMPHASIS ON UNDERSTANDABILITY

Reflect the patient's refusal reasons back to the patient so that they feel they have been heard. **It is important for the EP to acknowledge the patient's perspective, even if they disagree with the reasons.** This allows the patient to engage in listening as the EP provides additional information to support the recommendation. Normalizing an "irrational concern" allows the patient to feel "okay" and still follow the recommendation. For example, "I can understand that your sister's complication from procedural sedation several years ago would give you some concerns about this recommendation. I want to reassure you that today we take these additional steps…." Tailor the revised recommendation to address the concerns of the patient and focus on making sure that the information provided is simple, direct, and understandable.

ADDRESS BARRIERS TO UNDERSTANDING

Make significant efforts to enhance the patient's ability to understand the information when a refusal occurs. A professional interpreter must be utilized to compensate for any communication barriers to the patient's understanding in an informed refusal process. Revisit all the information from the initial discussion of information that occurred with an informal interpreter (e.g., family member or health care provider). Residual misinformation can prolong a patient's refusal. Start from the beginning of the clinical communication, even if it takes more time. This can often remedy the situation. Use language or pictures tailored to a patient's lower educational or functional level when necessary.[19] Address any anxiety and pain as quickly and as safely possible as they may contribute as a barrier to understanding.

CONFIRM CAPACITY TO REFUSE RECOMMENDATIONS

Is decision-making capacity a potential issue? **The EP must take steps to mitigate any factors leading to impaired decision-making so that the patient may participate in their care to the fullest extent possible.**

It was thought in the past that patients with certain diagnoses by default lacked decision-making capacity. Many clinicians now recognize that patients with severe mental illness, early dementia, and some organic brain syndromes are at risk for impaired decision-making but may possess decision-making capacity for selected procedures and treatments.[15] There are certain red-flag scenarios when an EP should scrutinize a patient's decision-making capacity with greater depth (**Table 1-6**). **Actions or decisions with greater consequences require a more intense evaluation of the patient's capacity.** A more careful evaluation of capacity is indicated when the patient's choice seems unreasonable or if the patient is unwilling to discuss their thought process. Chronic psychiatric and neurologic conditions remain a risk for, but should not be equated with, impaired decision-making. Cultural, educational, and language barriers impact the decision-making process. High levels of anxiety (e.g., untreated pain or the inevitable stress of the ED) are known to impair decision-making.[28]

TABLE 1-6 Red Flag Scenarios That Require Additional Assessments of the Patient's Decision-Making Capacity

Abrupt change in mental status
Anxiety or untreated pain
Chronic psychiatric or neurologic conditions
Cultural and language barriers
Extremes of age
Limited education
Patients readily consenting to invasive or risky treatment
Refusal of recommended treatment

Many providers outside the ED setting will utilize psychiatric consultations to assist with the evaluation of a patient's decision-making capacity. The utility of such a consultation is frequently limited by time and consultant availability. Consultations in the ED may prove useful when evaluating a thought or delusional disorder that may impede understanding.

EFFECTIVE DOCUMENTATION TO DOCUMENT THE INFORMED REFUSAL

Honoring a refusal of emergency treatment that would be beneficial or may result in decompensation or death is never easy. Use of the standard hospital "Against Medical Advice" form can create an adversarial relationship that an EP may find damaging to future patient interactions and the subsequent treatment plan. Anecdotal reports include cases where patients reconsidered their decision when presented with such a document. Document refusal of care for medicolegal protection and to confirm that clear communication with the patient had occurred.

The documented recommendations when a patient refuses treatment should include: the patient has refused the recommended procedure, test, or treatment; the patient's reasons for the refusal; and the consequences of the refusal were explained to the patient including the alternatives, if any, being offered or performed in lieu of the recommended procedure. Include statements that show the patient understood and continued to refuse the specific procedure or treatment and has the capacity to do so. Document that the patient's wishes are being honored against medical advice. **It would be preferable if the EP could have the patient read this documentation followed by the patient signing the medical record below this documentation in acknowledgement.**

Additional documentation is required when an EP recognizes a "red-flag" scenario for impaired decision-making (**Table 1-6**) or has other reasons for concern (**Table 1-7**). These are essential items that must be documented in these cases. Document the patient's medical condition and the procedure or treatment that is suggested, including the urgency and necessity. Document the patient's current decision-making abilities with a description of the impediments to capacity and the actions taken by the EP to maximize capacity. Include the availability of family or other surrogate decision makers and any relevant discussions.

Documentation will vary by institution and local laws. Being familiar with the appropriate measures to make an informed consent or refusal is effective is a critical part of the informed consent or informed refusal process in the ED.[29]

TABLE 1-7 Mnemonics for Documentation of Decision-Making Capacity Assessments

U *and* I GLAD
U–understanding of the procedure/discussion
I–impairing conditions
G–goals and values
L–logic used to decide
A–actual functioning
D–danger or risks of decision
CURVES*
C–choose or communicate (Can the patient make and communicate their choice?)
U–understand (Can the patient understand the risks, benefits, and alternatives?)
R–reason (Can the patient make a logical and rational choice?)
V–value (Is the choice consistent with patient values?)
E–emergency (Is there impending risk?)
S–surrogate (Is a surrogate available or is there any documentation guiding treatment?)

*The first four refer to the decision-making capacity. The last two refer to treatment without consent.

SHARED DECISION-MAKING

There has been much discussion about the concept of shared decision-making (SDM).[30-36] The concept was brought to the fore in the Institute of Medicine (IOM) report "Crossing the Quality Chasm."[30] It was in the context of improving quality and safety through patient-centered care, "care that is respectful of and responsive to individual patient preferences, needs, and values."[30] The report went on to specify "that patient values guide all clinical decisions."[30] There is a good deal of rhetoric surrounding patient-centered care in the literature, attempting to move clinicians away from the traditional role as the sole authoritarian and into the role of a partner in care. SDM is a way of actualizing these words, engaging patients in the essential role as a participant in their care, and breaking down communication barriers between providers and patients.

Expert consensus argues that SDM is different than informed consent.[31] Informed consent is used when there is one distinctly superior treatment choice. The informed consent process ensures the patient understands the risks and benefits from a particular procedure and consents to the treatment freely and without persuasion. SDM is a process entered when there is more than one reasonable course of treatment indicated for a particular clinical situation, each with its own set of outcomes and potential complications. The EP and the patient exchange information involving their expertise in the process of SDM. The EP shares the potential treatment options and describes the risks and benefits of each. The patient communicates their values and preferences regarding each treatment. This is not to say that all responsibility for decisions is placed on the patient. Each person contributes to the other's understanding of important aspects of the shared decision about how to move forward with treatment. **"SDM is best described as a conversation between the clinician and the patient in which they figure out together what to do to address this patient's situation."**[31]

The mechanical approach to implementing SDM seems to disturb the spirit of a personalized strategy for a particular patient. There are some fundamental components to include in a conversation. Clarify the patient's understanding of their condition. Identify the issue requiring treatment. Offer and describe options for treatment, emphasizing the advantages and disadvantages of each. Develop an understanding for the patient's values and how they may affect preferences for treatment. What matters most to the patient? Review the understanding of the patient's preferences and move toward a decision based on a combination of available treatment data and the patient's preferences.

The ED may not lend itself well to the original approach to SDM using interventions crafted by specialists, hospitalists, and primary care providers. These tools include risk calculators, decision aids, and conversation aids. Many of these were used outside the clinical encounter. Patient deliberation regarding options is a key task supported by SDM. Implementing SDM may not always be feasible in the ED with the pressures of acuity, flow, time, and variable volume.

TABLE 1-8 The Order for Surrogates for the Delegation of Decisions

Spouse
Adult child who has the consent of other children
Majority of adult children
Parent
A person authorized by the patient
Nearest living relative
Clergy member

SDM is already occurring in the ED as we work with patients on timing of cardiac disease risk stratification, choice of imaging modalities, wound care methods, and many other procedures and pathways.

SPECIAL CIRCUMSTANCES

Consent may be obtained over the telephone if the patient is unable to consent, the surrogate is not on premises, and the surrogate is only reachable by telephone. Have two persons on the phone with the surrogate during the consent process. Note the person's name and relationship on the consent. Have both persons on the phone sign the consent as witnesses. The general order of surrogacy is noted in **Table 1-8**.

Other issues with consent arise in the ED. A person in custody retains their right to consent except in emergencies and under court orders. Contact a minor's parent or guardian for consent if they are in custody except in an emergency, under a court order, or in a situation described previously. A minor placed in adoption or in the custody of the county or state requires contact with the welfare department for consent unless in emergency. A minor serving in the U.S. Armed Forces may give consent. Pregnant minors may consent to all care related to the pregnancy and newborn.

SUMMARY

The informed consent should be performed by the EP performing the procedure. Do not have the nurse obtain the consent. A written informed consent is preferred over a verbal consent. The written consent is a record of the verbal consent. Obtain verbal consent when the patient is unable to write. Have the verbal consent signed by two witnesses to the consent. The signed consent for treatment when the patient registers is not a substitute for a consent form for the procedure. Know the institution and state requirements for consent. Involve the ethics committee if time allows.

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2 Against Medical Advice

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INTRODUCTION

Patients electing to leave against medical advice (AMA) represent a growing population in the United States and provide unique challenges to the Emergency Physician.[1] **Discharge AMA is defined as the patient leaving before the Emergency Physician finishes the evaluation and establishes the disposition.**[2] It is estimated that 1% of Emergency Department visits result in a discharge AMA.[3,4] The prevalence rate of leaving AMA varies between Emergency Departments.[5] Discharge AMA patients often present again within a few days, resulting in increased costs associated with repeat testing and higher acuity therapeutic interventions due to worsening of their condition.[3] The AMA patient has an increased risk of repeated admission, increased admission length of stay, increased morbidity, and increased mortality.[2,6-17] This chapter provides an overview of the legal obligations to treat and elements associated with the refusal of care and discusses special populations that may be encountered.

Start with the assumption that the patient can make their own decisions, unless there is suspicion otherwise.[18-21] This practice is consistent with general principles of patient autonomy. **Any decisions made must be in the best interest of the patient.** Lack of decision-making capacity requires an assessment and documentation of how this was determined (**Table 2-1**). Lack of capacity requires an impairment of the patient's brain or mind significant enough to interfere with decision-making. The determination of decision-making capacity is specific to a relative point in time and does not apply to later decisions. The CURVES mnemonic was developed to be used in an acute setting such as the Emergency Department (**Table 2-1**).

The patient has their own reasons, good or not, for leaving AMA. Not every decision by a patient is considered reasonable by the Emergency Physician.[15,21] Patients often present voluntarily to the Emergency Department for evaluation and management. **Leaving AMA can be considered a withdrawal of the patient's consent signed when they initially presented for evaluation.**[22-24] **The patient has the right to participate in the medical decision-making process and may refuse any care offered.**[18-20] It may be impossible to change a patient's mind once they decide to leave AMA. Sometimes it is essential to treat the patient against their wishes (e.g., altered mental status, homicidal patients, life-threatening situations, public health risk with meningitis or tuberculosis, or suicidal patients). Document these exceptions clearly in the medical record.

Many Emergency Physicians struggle internally with the AMA patient. They want to allow the patient autonomy in the decision-making process while maintaining what they believe is in the patient's best interest (i.e., beneficence). A patient going against the Emergency Physician's recommendations may set up a poor physician–patient relationship for the encounter.

TABLE 2-1 The U and I GLAD and CURVES Mnemonics for Determining a Patient's Decision-Making Capacity

U *and* I GLAD
U–understanding of the procedure/discussion
I–impairing conditions
G–goals and values
L–logic used to decide
A–actual functioning
D–danger or risks of decision
CURVES*
Communicate: Is the patient able to choose and communicate this choice?
Understand: Does the patient understand the alternatives to treatment, benefits of treatment, and risks of leaving?
Reason: Can the patient make a rational choice?
Values: Is the patient's choice consistent with their values?
Emergency: Is there impending risk to the patient?
Surrogate: Are there patient surrogates available? Is there any documentation guiding treatment (e.g., advance directives)?

*The first four are for decision-making capacity. The last two are for treatment without consent.

TABLE 2-2 The Misconceptions Involved with Leaving AMA[4,5,22,25,26]

AMA means the patient leaves with nothing
Blood alcohol predicts decision-making capacity
Decision-making capacity is all-or-nothing
Decision-making capacity is consistent over time
Determining decision-making capacity compromises patient safety
Forms signed by the patient offers legal protection
Insurance will not pay if leaving AMA
Leaving AMA means you can't be sued
Legal competence equates to decision-making capacity
Minors cannot give consent
Only Psychiatrists can make an accurate assessment of decision-making capacity
Psychiatric diagnoses negate a patient's decision-making capacity
The AMA form must be signed

MISCONCEPTIONS OF LEAVING AMA

There are many misconceptions involving the patient who is leaving AMA (**Table 2-2**).[4,5,22,25,26] The main one is that a patient leaving AMA needs no further care. Offer pain medications for the patient's condition when appropriate. Do not withhold pain medications. The use of these drugs may make the patient more agreeable to completing the work-up and recommendations. This can be a new starting point and prevent the patient from leaving AMA. Offer several options for treatment if the patient refuses the primary and most ideal option. Many Emergency Physicians need to be educated regarding the misconceptions surrounding the patient leaving AMA.

RISK FACTORS FOR LEAVING AMA

Specific groups and complaints are associated with leaving AMA (**Table 2-3**).[10,16,17,27-34] Many complaints are associated with extensive and prolonged work-ups. Administrators and Emergency Physicians should anticipate the needs of this group and proactively intervene to improve upon completion of the planned work-up. Early identification of these patients may prevent discharges AMA and negative outcomes. Develop strategies to prevent patients leaving AMA. Be proactive to address anger, anxiety, and emotional distress among patients.

TABLE 2-3 The Risk Factors for Leaving AMA[10,16,17,27-34]

Abdominal pain	Lack of primary physician
Adolescents who register themselves	Lack of social support
Age 19–40 years	Lower socioeconomic status
Alcohol-related disorders	Lower triage categories
Black	Male
Chronic disease	Nonspecific chest pain
Headache	Prior AMA discharge
Hepatitis	Psychiatric disorders
Homelessness	Sickle cell anemia
Human immunodeficiency virus	Seen within last 72 hours in an Emergency Department
Lack of commercial insurance	
Lack of insurance	Substance abuse

TABLE 2-4 The Reasons Given for Adults and Children Leaving AMA[5,6,9,14,16,17,25,31,34-41]

Anxiety about other children at home	Job issues for other family members
Change their mind	Job issues for themselves
Chronic disease	Lack of confidence in health care system
Concern for pets	Lack of confidence in physician
Conflict with child caregivers at home	Living away from home
Delays in treatment	Long waits
Disagreements with physicians	Outside obligations
Dissatisfaction with care	Poor communication with physician
Elderly parents at home	Prolonged hospital stay
Faith in local healers	Refusal of referral
Faith in religious beliefs	Refusal of surgery
Faith in social customs	Second opinion
False perception of improvement	Spontaneous resolution of illness
Finances	Spontaneous resolution of pain
Frequent blood sampling	Transportation issues
Hunger	Travel issues
Improvement with treatment	Unknown (not noted)

Improve physician–patient communication. Nurses are often first to know the patient wishes to leave AMA. Train the nurses to proactively address concerns that may prevent a patient from leaving AMA. Consider involving case managers or social workers to ensure patient needs are met and improve communication.

REASONS FOR LEAVING AMA

Patients give many reasons for leaving AMA (**Table 2-4**).[5,6,9,14,16,17,25,31,34-41] The main reasons include communication issues, drug addiction, long wait times, inadequate pain control, outside obligations, physician personality, second opinions, and teaching hospital environments. Knowledge of the reasons for leaving AMA can improve the approach and management of these patients. Understanding the reasons for leaving AMA may allow Emergency Physicians and hospital administrators to address these issues and minimize adverse outcomes among this group. These patients are at risk for excessive morbidity, mortality, and increased associated costs.

Question the reason(s) the patient desires to leave AMA. Sometimes the resident or nurse can obtain this information as they typically have a closer relationship with the patient.[25] Consider involving family members and friends of the patient as allies to assist in convincing the patient to follow the recommendations. They may help the patient better understand the treatment and the consequences of the lack of treatment and reveal additional patient questions to be addressed. Apologize for any waits. **Do not become angry or frustrated when the patient wants to leave AMA.** This only upsets the patient and encourages them to leave even more. **Ensure the patient knows that you are on their side and have their best interest in mind.** Do not refuse to provide treatment if the patient wants to leave AMA. **Offer any treatment acceptable and appropriate for the patient's condition that they will accept.** Some care is better than no care.

DEFINING THE DUTY TO TREAT

Emergency Departments across the United States are bound by the Emergency Medical Treatment and Active Labor Act (EMTALA) requiring them to provide medical screening examinations and stabilization for all patients who present to the facility.[42] This obligation extends to Emergency Physicians who work at facilities that participate in one or more Centers for Medicare and Medicaid Services (CMS) programs. The timeline to which the obligation extends has been debated in the courts where allegations argue that EMTALA may even continue into the inpatient environment, as seen in the 2009 court case of *Moses v. Providence Hospital*.[43] One possible way the duty to treat may be terminated is via a patient's informed refusal of care.[44-46] Great care must be taken in completing the process of discharging a patient AMA in terms of fulfillment of EMTALA obligations. The patient often has a high risk of readmission and increased morbidity and mortality.[2,6-17,47,48]

ELEMENTS ASSOCIATED WITH REFUSAL OF CARE

Consent must be obtained prior to the treatment of a patient to avoid committing battery or the unwanted touching of a person (Chapter 1). Similarly, inform the patient completely before they make a final decision to refuse care.[26] The informed refusal of care is a process and requires more than having the patient simply sign the AMA form.

The patient may elect to refuse any or all treatment offered them during the hospital or Emergency Department encounter. **It is the responsibility of the Emergency Physician to evaluate the patient and ensure that all the elements listed below are met and then to clearly document the patient's informed decision-making process leading to refusal of care or discharge AMA.**

DECISION-MAKING CAPACITY

Decision-making capacity is sometimes simply referred to as capacity. It is determined by a physician and represents the patient's ability to make rational decisions.[21,49] Any physician, including Emergency Physicians, who cares for a patient can clinically determine if the patient has decision-making capacity.[25] Consulting a Psychiatrist or their delegated representative (e.g., Psychiatric Nurse Practitioner, Psychiatric Physician Assistant, or Psychiatric Social Worker) is not necessary in most cases. It may be necessary to contact a Psychiatrist or their representative on a case-by-case basis.[22] This is true when decision-making capacity cannot be determined or the patient is to be involuntarily committed (e.g., danger to others, danger to self, or incapable of self-care) to a facility. Decision-making capacity changes, is task-specific, is not all-or-nothing, and can be affected by many things (e.g., fatigue, medications, psychiatric disorders, and stress).

The term "decision-making capacity" is used by physicians. This is opposed to the legal term of competence as used by the courts.[25,50,51] These terms are often incorrectly used interchangeably by physicians. Only a court of law can decide competency and appoint a guardian to make important decisions for the patient.

The Emergency Physician must question the patient to determine if the patient has decision-making capacity (Table 2-1). The patient must have the ability to understand information related to their condition and treatment decisions. It is not possible to assess decision-making capacity unless the patient is fully informed. **The patient must have the ability to appreciate the significance of the information presented to them. The patient must explain the information presented rather than simply repeating it back. The patient must have the ability to weigh the treatment options and demonstrate reasoning. The patient must express their choice for treatment or refusal of treatment.** Failure of one part can result in lack of decision-making capacity. All this must occur in the patient not under the influence of alcohol or drugs or not with an altered mental status. The patient must not have a reason for involuntary commitment to a facility.

The Emergency Physician must first ensure the individual patient has the capacity to participate in their own decision-making process prior to engaging in a refusal of care discussion.[25] **Always ensure that**

capacity exists because the decision to refuse treatment may be viewed as unreasonable. The additional use of resources from psychiatry, if available, may be of benefit. Consider other conditions that affect a patient's ability to fully participate in their care (**Table 1-6**). Correct any reversible causes affecting the patient's decision-making capacity. A discussion must ensue regarding the disclosure of risk following a careful review of the patient's decision-making capacity.

Formal and structured assessment tools are often used to determine decision-making capacity.[52,53] These tools include the Aid to Capacity Evaluation (ACE), MacArthur Competence Assessment Tool (MacCAT), Montreal Cognitive Assessment (MoCA), and University of California San Diego Brief Assessment of Capacity to Consent (UBACC). These tools use standardized questions and scoring systems to objectively determine decision-making capacity. No specific test of decision-making capacity is better than another test. The tests take time to assess the patient and generate a score. Most of these tests are unfamiliar to the Emergency Physician who is untrained with their use.

Lack of decision-making capacity or refusal of treatment may allow the Emergency Physician to share information with friends and relatives. A person close to the patient can often convince the patient when the Emergency Physician is unsuccessful.[21] This option can be explored to assess the patient's best interest. The involvement of others shows that the Emergency Physician is advocating for the patient in solicitation of additional input to make the right decision. Another physician may intervene to provide care if a patient and Emergency Physician disagree. Consider another Emergency Physician taking over the care of the patient. Consider calling the Primary Physician if the patient has one. Offer to transfer the patient to another facility. Clearly document all offers and refusals.

DISCLOSURE OF RISK

The Emergency Physician must follow the principles established in the *Canterbury v. Spence* decision when disclosing risk.[54] This requires disclosure of the condition being treated, proposed treatment being offered, alternative treatment options, and risks associated with both treatment and refusal. **Take care to ensure the patient understands all available options.** Engaging family members, friends, or on-duty Emergency Department personnel in this discussion may prove beneficial.

INSURANCE PAYMENTS

A fallacy sometimes conveyed to patients is the idea that their insurance will not pay for the visit should they elect to leave AMA.[4,25,55] Many Residents and Attending Physicians believe insurance payments will be denied if the patient leaves AMA.[4] They often inform the patient of this to coerce the patient in remaining.[2,4] There are no documented instances of insurance companies denying the bill of a patient leaving AMA.[4] There are no policies of payment denial for leaving AMA. Insurance companies determine payment based on medical necessity. The Arkansas Supreme Court in *Loretta Long v. Arkansas Blue Cross and Blue Shield* ruled that services prior to discharge are payable because of benefits due to the patient before the AMA.[56]

Statements addressing the lack of insurance payment must be avoided verbally and on the AMA form.[2,15] This appears to be an "urban legend" passed down during residency training and often persists throughout a physician's career. There is often a breakdown in the physician–patient relationship when the patient is falsely warned of negative financial consequences if leaving AMA. The insurance payment should not be a concern when caring for the patient considering leaving AMA. **The Emergency Physician must respect the patient's autonomy when they do not agree.** Resident and Attending Physicians need to be formally educated on what to say, document, and do when the patient wants to leave AMA.

SPECIAL POPULATIONS

Obtaining a refusal of care or discharging AMA can be an anxiety-producing encounter while trying to provide care. This situation can become further complicated when a patient has consumed alcohol, is currently incarcerated, is a minor, or has an active psychiatric diagnosis. There are unique features to consider when dealing with these populations.

INTOXICATED PATIENTS

Patients who have consumed alcohol represent the most difficult of the special populations from whom to obtain informed consent or refusal.[29] The blood alcohol concentration can affect patients differently. The Emergency Physician often assumes that the acutely intoxicated patient lacks decision-making capacity. **The patient's decision-making capacity must first be established by the same standard as an individual who has not consumed alcohol before discharging an intoxicated patient AMA.**[57]

Each individual state may have specific laws regarding the ability to give consent while intoxicated. An intoxicated patient was considered unable to provide consent and a diagnostic procedure was completed against his request in *Miller v. Rhode Island*.[58] A New York court found the hospital and Emergency Physician could not detain an intoxicated person against their will in *Kowalski v. St. Francis Hospital*.[59]

Determining the degree of intoxication presents a challenge. Emergency Physicians have been previously shown to have poor ability in determining clinical sobriety. The patient often does not remember things that occurred while intoxicated when they become sober.[57] Serum and/or breath testing of alcohol does not directly correlate to a patient's degree of intoxication and is likely not helpful in determining capacity.[60,61] Documenting the patient's activities and ability to eat, walk, engage in conversation, and to rationally understand questions and discussions can be helpful as this suggests their ability to understand care options and treatment plans. Acutely intoxicated patients may have decision-making capacity regardless of their blood alcohol concentration.[15,57,62]

INCARCERATED PATIENTS

Patients who present in the custody of police or a correctional institution (e.g., jail or prison) represent another special population when considering the ability to refuse medical care. **Prisoners have the same rights to refuse or submit to medical care as the general population.** The standards described regarding capacity remain the same.[63] Unique to the incarcerated patient is their inability to determine where they are incarcerated. The correctional institution responsible for the patient may choose to supervise them in a jail ward or within a medical setting while respecting their right to refuse treatment if a patient who has capacity elects to refuse care.[63]

MINORS

Minors (e.g., those < 18 years old) represent a special population of patients who present to the Emergency Department and may elect to refuse care. State laws vary regarding types of treatment, age of consent, and conditions that apply to a minor who presents for emergent care. A minor making one decision may not have the capacity to make other decisions. Some states allow minors to

obtain contraception, treatment for sexually transmitted infections, and treatment for substance abuse without parental permission.

Minors must be emancipated and can be determined to have decision-making capacity if they meet the following qualifications. The qualifications for emancipation vary between states. They must have the ability to understand the diagnosis, treatment or lack of treatment, and that the choices have consequences. They must have the ability to understand the information presented to them. They must have the ability to make a decision based on the information they receive from the Emergency Physician. Minors must have the ability to understand the intervention, its benefits, and its risks. They must have the ability to understand any alternatives, along with the associated risks and benefits. The minor must make a choice between treatment and lack of treatment, or choose another person to make the decision on their behalf (e.g., usually a parent or spouse). The minor cannot be coerced or forced into a decision, and pressure should never be applied.

What about the minor who lacks decision-making capacity for any reason? Decisions are often made by parents or legal guardians.[5] Make an effort to involve the minor in order to gain their cooperation. Provide them with information in terms that they will understand based on their age.[35] Minors are vulnerable because they may not adequately understand the ramifications of a decision to leave AMA. Leaving AMA may not be in their best interest.

States work under the principle of *parens patriae*, or parent of the state. The state has an interest in the welfare of its citizens. This includes minors. The specifics regarding *parens patriae* vary among the states. *Parens patriae* is a mechanism for the state to override the rights of a parent and provide their substituted consent. ***Parens patriae* is not an option left to the Emergency Physician or hospital as a mechanism by which to override parental control.** Providing care in violation of parental consent may make an Emergency Physician and hospital liable for violating consent. **Do not proceed with care over parental objections without authorization from state authorities unless it is necessary to preserve life or limb.** Treatment in a true life-threatening situation can be considered prevention of child abuse, and the Emergency Physician may take emergent custody of the child. Get a second physician, if available, to agree and attest via signed documentation in a life-threatening situation to override the parents until the courts can render a decision. This may require separation of the minor from the parents with assistance from police or security.

PSYCHIATRIC PATIENTS

An active psychiatric diagnosis does not automatically mean the patient lacks decision-making capacity. An active psychiatric diagnosis may result in the lack of decision-making capacity. A psychiatric patient managed with appropriate medications can easily make decisions. Psychiatric patients may be in denial, dissatisfied with their treatment, fearful, mistrustful of the medical system, and/or paranoid. It may be necessary to contact a Psychiatrist or their delegated representative when managing psychiatric patients who refuse care.[22,52] This is true when decision-making capacity cannot be determined or in the setting where the patient is to be involuntary committed (e.g., danger to others, danger to self, or incapable of self-care) to a facility.

PATIENT-CENTERED APPROACH

The patient-centered approach uses shared decision-making in a collaborative effort between the Emergency Physician and patient (**Table 2-5**).[2,64,65] It takes into account scientific evidence along with patient goals, preferences, and values. Shared decision-making is based on the Emergency Physician's recommendations with the patient's right to accept or refuse the recommendation. Consensus and agreement are made between the Emergency Physician and the patient when determining the goals of care that affect the patient. A more agreeable plan is made when the Emergency Physician has clear information regarding the patient's motivation and values. This involves the exchange of information, deliberation, and decision-making. Good communication with the patient is essential to avoid dissatisfaction and frustration of the Emergency Physician and the patient.

TABLE 2-5 The Patient-Centered Approach for Leaving AMA[65]

Determine if the patient has decision-making capability
Is the patient alert and oriented?
Does the patient have mental impairment?
Does the patient have active mental illness?
Is the patient under the influence of alcohol or drugs?
Determine the patient's preferences and values
Don't stigmatize the patient
Don't berate the patient
Don't coerce the patient
Don't express frustration
Don't express anger
Don't mention insurance will not pay if they leave
Assure the patient the decision-making has nothing to do with their ability to pay
Assure the patient the decision-making is in their interest of well-being
Involve family members personally or by phone
What is the treatment plan if staying?
Discuss the benefits and risks if staying
Discuss how treatment will differ as an outpatient
Discuss the benefits and risks if leaving
Make and provide an outpatient treatment plan if patient leaves AMA
Provide follow-up
Provide prescriptions
Provide discharge instructions
Document everything in the medical record

The choice to designate the patient leaving AMA is made by the Emergency Physician when they do not agree with the patient decision. A patient-centered approach is used to support informed patient choices even if they conflict with physician recommendations. Be empathetic and nonjudgmental toward the patient. Engage the patient politely to determine their motivations behind their desire to leave AMA. Explore this motivation through discussion and avoid conflict that undermines the physician–patient relationship. Embrace and respect the informed decision made by a patient who has decision-making capacity.

DOCUMENTATION

Emergency Physicians and hospitals are not unequivocally protected from lawsuits and successful litigation resulting from bad outcomes simply because the patient signs the AMA form.[5,22,34,36,66,67] This is contrary to the belief of many physicians that the AMA form offers legal protection if the patient rejects their recommendations. Courts have found the AMA discharge terminates the physician–patient relationship and the physician's duty to treat.[45,46] Family members often believe more could have been done for an ill patient despite the irrationality of their thinking.[22] **The attending Emergency Physician, and not a resident or nurse, must interact with the patient contemplating leaving AMA and document the discussion.**

The Emergency Physician must document the situation and discussions to memorialize the encounter.[66] Clearly document the efforts offered to the patient to get them to stay. Emergency Physicians

do a poor job of documenting the encounters for patients leaving AMA.[32,36,68,69] The documentation involves extra time and disrupts the workflow of the Emergency Physician. The Emergency Physician may be sued years after the encounter. They may only have the encounter documentation to rely upon to refresh their memory.

Many institutions elect to use standardized forms to complete the discharge AMA process (**Figure 2-1**). Many Emergency Physicians use the hospital AMA form without a clear reason. It is used to document patient symptoms, to facilitate discussions with the patient, to improve documentation, and for the ease of completion.[70] The

73 **Prototype**
EMERGENCY PHYSICIAN RECORD
Competency for AMA Discharge
or Treatment without Consent

☐ All clinical information and issues reviewed / discussed with family, patient, other _____
☐ Relevant issues reviewed / discussed with patient / family
☐ No criteria for involuntary commitment

Cognition-
Oriented to person, place, time _____
Gives appropriate answers
Speaks coherently _____
No slurred speech
No signs of psychosis _____
No tangential thinking _____
No auditory hallucinations _____
No visual hallucinations
No delusional thinking _____
Abstract thought process intact _____
No suicidal ideations
No homicidal ideations _____
Gives rational explanation for refusal of care _____

Comprehension-
Aware of suspected diagnosis suggested by initiated screening exam:

Acknowledges understanding of reasons for recommendations regarding:
Medical treatment / intervention _____
Medical tests / procedure _____
Transfer to other medical facility _____
Admission to facility _____
Further observation / testing _____

The following risks of refusal of recommended care were disclosed to patient, and patient acknowledged risks:

RISKS	DISCLOSED	ACKNOWLEDGED
Death		
Neurologic		
Dysfunction		
Permanent mental impairment		
Loss of limb		
Loss of sexual function		
Loss of current lifestyle		
Worsened / chronic cond		
Other _____		

Suspected diagnosis(es) based upon initiated medical screening exam:

Outpatient treatment: _____

Follow-up plan: _____

	Patient Declined	Not Feasible
Offered transfer / other physician evaluation	☐	☐
	Patient Declined	**Not Available**
Offered to call patient's physician	☐	☐
Offered to speak with family / relative	☐	☐

CLINICAL IMPRESSION

Competent to make decisions regarding the medical care being offered?
__YES __NO

Discharge Instructions / Arrangements
☐ Discharge instructions were given to the patient / responsible party.
☐ Discharge instructions were NOT given to the patient / responsible party because:
☐ Patient / responsible party eloped
☐ Patient / responsible party refused
☐ Informed patient that they could return at any time if problems develop or if they change their decision regarding care.

Treating PA _____
Treating Physician _____

STATEMENT OF REFUSAL OF CARE:

(Obtain signature by patient / responsible person if possible)

I have read this paragraph. I understand that a doctor at this hospital wants to give me certain medical care. The doctor explained that care to me, and I understand what that care is. The doctor also explained to me what could happen to me if I leave here without having that care, and I understand what was said. I want to leave this hospital without receiving the recommended care.
I know that I am welcome to return to this hospital at any time to receive the recommended care or any other care that I may need at any time, regardless of my ability to pay for such care.

_____
Patient / Person Signing on Patient's Behalf

_____
Witness

☐ Patient / responsible person refused to sign this statement when requested to do so but indicated refusal of care in the following manner:

Other Comments: _____

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Circle positives, backslash negatives, check normals

FIGURE 2-1. A commercially available sample documentation for leaving AMA. (Courtesy of T-System Inc., Dallas, TX.)

form is often used to avoid further conversations with the patient. This "one size fits all" form is often just signed by the upset patient and witnessed by the staff. Signing the AMA form can appear to the patient as coercive or defensive and further exacerbate the poor physician–patient relationship.[70] The use of standardized forms has been shown to improve documentation of required elements. **Complete documentation and the patient's signature on the AMA form are not a substitute for the informed refusal discussion.**[34,71] The use of a hospital AMA form does not substitute for clear and specific documentation of the informed refusal documentation. Laws regarding liability are defined at the state level and vary based on jurisdiction.[34] Consider the AMA form as a document to make the patient aware of the benefits and risks associated with leaving AMA.[36] The Emergency Physician may elect to individualize and dictate the discussion with the patient (**Figure 2-2**). Document the exact words used when speaking to the patient.

Address the following elements when using a template form or directly documenting in the electronic medical record according to EMTALA guidelines: explain the clinical scenario, explain admission or treatment is medically advised, document that admission or treatment is refused by the patient, explain the potential consequences of self-discharge, and document that the patient takes responsibility for any adverse outcomes.[34,42] Include the date of the discussion, the time of the discussion, and those persons (e.g., family, friends, and/or hospital personnel) present. The patient should have decision-making capacity and not be under the influence of alcohol or drugs. The Emergency Physician and the patient should sign if electing to use a form. An alternative is to print out the medical record and have the patient sign it. Document the lack of the patient's signature if they refuse to sign, and have a witness to the refusal sign as well.

DISCHARGE

Provide the patient with a clear understanding of the discharge plan and alternative outpatient therapies.[15,68,72-74] Provide any prescriptions to the patient that may be required for an alternative treatment when leaving AMA. Provide prescriptions for pain control if appropriate for the patient's condition. Explain what to look for at home, medical reasons to return, and encourage the patient to return if they change their mind. Provide follow-up plans to the patient. Consider calling the follow-up physician to discuss the case, what was done, and why the patient left AMA to ensure appropriate care. Notify police and/or a social worker in cases of suspected child and elder abuse.

Patients electing to leave AMA can stimulate negative feelings among Emergency Physicians and staff. **Ensure that the patient feels welcome to return and resume care at any time.**[66,74,75] This includes persistence of symptoms, worsening of symptoms, or if the patient changes their mind. **Continue to be cordial and do not give the impression that it will be held against the patient if they choose to leave AMA.** Consider calling the AMA patient in 24 hours to ensure they are better, to inquire into their safety and well-being, and to see if they have any questions. Document this discussion.

Avoid a punitive encounter to increase the likelihood that patients will obtain the care needed.[15,74,75] The ability of the patient or their insurance carrier to pay is not an issue for the Emergency Physician to discuss with the patient. **The discharge and disclosure process must be free of coercion.** End the encounter on good terms with the patient. **Report all patients that leave AMA to risk management for review.**

The patient has decided to leave against medical advice because _____ _____. The patient has a normal mental status, is not under the influence of alcohol or drugs, and has adequate decision-making capacity regarding medical decisions. The patient appears to have insight, judgment, and reason. The patient refuses observation or admission and wishes to be discharged. The patient presents with _____ and I am concerned for _____. Staying for observation or admission we may be able to better treat you. The benefits and risks of leaving have been discussed and include _____ _____, worsening illness, chronic pain, disability, and death. The benefits of observation or admission have been explained including the availability of nurses and physicians, diagnostic testing, monitoring, and treatment. The patient understands and can state the risks of leaving and benefits of observation or admission. This was witnessed by me and _____. The patient was given the opportunity to ask questions about their medical condition, the risks of leaving, and the benefits of staying. The patient was treated with _____ _____. I offered to treat the patient with _____ if they stayed but the patient refused. I have spoken with Dr. _____ and the patient is to be followed up on _____ with Dr. _____. The patient was given prescriptions for _____ _____. The patient was given discharge instructions that included they may return at any time for care.

FIGURE 2-2. A hospital-made sample documentation for leaving AMA.

SUMMARY

Emergency Physicians face ethical, legal, and medical considerations as they encounter patients presenting for care who may ultimately elect to terminate their care plans in whole or in part. An effort should be made to recognize patients at risk for leaving AMA and attempts made to educate them as to the benefits and risks of leaving AMA. Maintain good communication with the patient. Ensure that the patient has no reason to be involuntarily hospitalized. Ensure the decision-making capacity for informed decision-making and clearly document these encounters. Fully inform the patient by reviewing the risks associated with failure to complete the work-up in terms of worsening morbidity and mortality. Encouraging the patient to return at any time for further evaluation and treatment is the best practice model for navigating potential pitfalls. The Emergency Physician should always fully explain the discharge process and follow-up plan and prescribe any appropriate medications despite the patient's choice to leave AMA.

The attending Emergency Physician is responsible for the discharge AMA. Residents and nurses can help with the process. Nurses can discharge the patient in the usual manner once the attending Emergency Physician fills out the documentation. Nurses can ensure the patient has all requirements (e.g., follow-up, instructions to return, prescriptions, questions answered, etc.) upon discharge.

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3 Family Presence

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INTRODUCTION

The topic of family presence has been discussed for over 30 years, and yet it remains controversial.[1-6] The proponents of the family being present during the resuscitation argue that it is the patient's and family members' basic human right to be present during the resuscitation. They maintain that barring or excluding families from witnessing critical events is paternalistic. Many people have already witnessed critical events in public, on television shows, on the news, on cable, and on reality shows. As a result, families are somewhat prepared and have expectations. Opponents of the family being present during resuscitation are concerned with litigation, the emotional fallout from the witnessed trauma, and disruptions during the resuscitation. The authors of this chapter strongly support the family being present during a resuscitation.

This chapter is designed to provide a reference for the Emergency Physician (EP) regarding the family being present during the resuscitation of patients in the Emergency Department (ED). It reviews the current literature on this topic and offers some suggestions.

BACKGROUND

The notion of the family being present during resuscitation dates back to the 1980s. Foote Hospital in Michigan started a program in response to families that wanted to be present during resuscitation.[7] Data from this program were presented in 1992 as formative research and have continued to be substantiated by more current publications.[8] The current literature shows the perspective of both the patient and the family.[9-22] Most of those surveyed, including the parents of children, feel it was their right to be present during the resuscitation or invasive procedure of a family member. Furthermore, when family members are present during the resuscitation, they are more likely to believe that everything that could have been done was done for their family member.[23] In addition, 67% of parents who were present during the resuscitation of deceased children felt that being present during the resuscitation helped them cope with the death of their child.[23]

Most patients with a critical illness would like to have their families present during resuscitation.[14,21] Concern that family members will suffer from posttraumatic stress disorder (PTSD) after witnessing their loved one resuscitated has not been substantiated.[24,25] A recent study of 65 family members of patients undergoing cardiopulmonary resuscitation (CPR) showed no difference in PTSD or depression in comparison to those who did not witness the resuscitation.[16] In a multicenter randomized study, PTSD-related symptoms were significantly lower in family members who witnessed the resuscitation than the control group.[17] Anxiety was also significantly lower in the intervention group that witnessed the resuscitation compared with those who did not witness the resuscitation. At 20 months of follow-up, there were no medicolegal claims of damages from the study participants.[17]

Professional organizations have positively endorsed the family being present during resuscitation.[26,27] The main points from the Emergency Nurses Association (ENA) position statement are listed in **Table 3-1**.[26] The American Heart Association (AHA) published in its 2010 guidelines for cardiopulmonary resuscitation and emergency cardiovascular care science that, "In the absence of data documenting harm and in light of data suggesting that it may be helpful, offering select family members the opportunity to be present during a resuscitation is reasonable and desirable assuming that the patient, if an adult, has not raised a prior objection."[27] The AHA identified this as a Class IIa recommendation with a level of evidence "C" for adults and a Class I recommendation with a level of evidence "B" for pediatric patients. In 2015, the European Resuscitation Council recommended that family be present during a resuscitation.[28]

Health care providers generally support the family being present during resuscitation and have identified barriers to its implementation.[29-33] Between 86% and 96% of nurses support the family being present during resuscitation compared to 50% to 79% of physicians.[29] The support decreases with lower levels of training.[5,30] The problems that health care providers perceive include

TABLE 3-1 The Emergency Nurses Association Position Statement on the Family Being Present During the Resuscitation

- There is some evidence that patients would prefer family members to be present during the resuscitation.
- There is strong evidence that family members wish to be offered the option to be present during invasive procedures and resuscitation of a family member.
- There is little to no evidence indicating that the practice of a family member being present is detrimental to the patient, the family, or the health care team.
- There is evidence that family members being present does not interfere with patient care during invasive procedures or resuscitation.
- There is evidence that health care professionals support the presence of a designated health care professional assigned to the family members present to provide explanations and offer comfort.
- There is some evidence that a policy regarding family member presence provides structure and support to health care professionals involved in this practice.
- Family member presence during invasive procedures or resuscitation should be offered as an option to the appropriate family members.
- Family member presence should be based on written institution policy developed in cooperation with the departments of social services, pastoral care, risk management, nursing, and medical staff (plus others as appropriate for the institution).
- Health care organizations should develop and disseminate educational resources to the public concerning the option of family presence during invasive procedures and resuscitation.

Source: Modified from reference 14.

anxiety, emotional stress, family litigation, limited space in the room, not enough staff to provide support to the family, prolonging a futile resuscitation, and staff distraction.[30-35] The health care provider should always show respect for the patient, whether or not the family is present.[36] These concerns and fears have not been supported in the literature.[2] In a study from the Children's Medical Center of Dallas, within the study period, no interruptions occurred, none of the family members were escorted out, and no disruptive behavior occurred in 100% of cases when family members were present during the resuscitation.[33] Other studies have shown that the resuscitation or invasive procedure was not affected by the presence of family members.[37-41]

HOSPITAL POLICIES

Several studies have focused on implementing hospital policies regarding the family being present during resuscitation.[42-47] The ENA and American Association of Critical-Care Nurses (AACN) have presented specific guidelines to be included in the policies. These guidelines include the following. Describe the benefits of family presence from the perspectives of the patient, the family members, and the health care providers. Establish criteria to assess family members so that patient care is not interrupted or delayed. Identify criteria to screen family members before offering the option of being present during the resuscitation. Family members who might be excluded are those who exhibit altered mental status, combativeness, emotional distress, or intoxication. Involve the family members in the decision process regarding declaration of death, invasive procedures, or aftercare. The policy should offer the option of the family being present during the resuscitation and support family members who choose not to be present. The policy must specifically address any research approved by the hospital and conducted in the ED because patient recruitment can be difficult with the family present.[48] A health care facilitator should be designated to consult with the health care team, to obtain consensus, to ensure proper timing, and to support family members (before, during, and after the resuscitation or invasive procedure).[49] The facilitator can be a chaplain, nurse, respiratory therapist, social worker, or other trained staff member. The option for family to be present during the resuscitation should not be offered if a facilitator is not available.[50,51]

A survey of over 1000 nurses showed that the policies are not consistently established and that only 5% of nurses worked where policies were established.[18] However, most health care providers who have experienced the family being present during the resuscitation would do it again.[52]

PEDIATRIC CONSIDERATIONS

In July of 2014, a technical report titled "Death of a Child in the Emergency Department" was published jointly in *Pediatrics* and *Annals of Emergency Medicine*.[53] The report reviewed the literature and pointed out that, in the studies of pediatric trauma resuscitations, all the milestones of care were performed timely regardless of family presence.[53,54] Most of the family members interviewed felt their presence was comforting to their children. However, a 2005 study found mixed results regarding child behavioral and emotional reflections about the family being present during the resuscitation.[55] In seven of the 17 studies, the family being present during the resuscitation resulted in a decreased level of distress. The other 10 studies demonstrated no significant difference between those who were present during the resuscitation versus those who were not. A common theme of these studies is that families believed everything was done for their loved one. In addition, numerous studies and position statements support the clinicians' ability to provide appropriate resuscitative care with the family present. O'Malley and colleagues point out this be done in the "setting of effective staff preparation, appropriate policy development and implementation, and, when staffing allows, providing designated personnel to attend to family members."[53]

Literature from the disciplines of pediatric emergency medicine, ethics, resuscitation, and nursing overall strongly supports the family being present during resuscitation. Multiple organizations, including the ENA, American Academy of Pediatrics (AAP), and American College of Emergency Physicians (ACEP), have published position statements recommending that all EDs that care for children have a policy regarding the family being present during the resuscitation of a loved one.[54]

SUMMARY

The controversy of the family being present during resuscitation has existed for some time. Review of the current literature supports family presence to be an accepted concept and practice. Most family members who have been present during a resuscitation have verbalized that they would do it again. Family members who observed the resuscitation experience reduced guilt and reduced time to accept the patient's death. They start the bereavement process earlier. The literature supports having an experienced staff member present whose only job is to support the family members during resuscitation. Data do not support the idea that family members are traumatized during resuscitation or interfere with any procedures. Medical communities should continue their efforts in establishing clear guidelines and protocols for the family being present during a resuscitation. Although most of this chapter covered family presence during resuscitation, the same principles apply to family presence during invasive procedures.

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4 Procedures on Recently Deceased

Bryan Darger and Eric Isaacs

INTRODUCTION

An issue relevant to procedural skills in Emergency Medicine and other specialties is allowing learners to practice invasive procedures on the recently deceased.[1,2] This often occurs immediately after the pronouncement of death and is a controversial practice. The use of the bodies of the dead for education has a long tradition in medicine. The use of the recently deceased is considered by some as a valuable resource because it improves the ability to save others in the future. Others believe consent is required from family members to preserve autonomy despite the uncomfortableness of asking for consent.

Teaching procedures have used various techniques (e.g., animals, cadavers, lectures, live patients, manikins, simulation, and videotapes). Each technique has advantages and disadvantages. These include cost, lack of reality, space, and time. The opportunity to practice lifesaving procedures is limited. This is especially true for realistic training.

Performing procedures on the recently deceased has been a topic of discussion over the past two decades and debated within the medical community. This chapter attempts to present a balanced overview of this topic and offer suggestions for best practices.

HISTORY AND CURRENT PRACTICE

Physicians and healers have been learning from the dead for millennia. The earliest-known description of circulatory anatomy is the Edwin Smith Surgical Papyrus from 1600 BC. Contemporary cadaveric dissection in the first year of undergraduate medical education training is a practice with a long precedent that has never been without controversy.

Various authorities (e.g., governments and religions) have restricted the practice of cadaver dissection for studying anatomy. A commonly cited belief is that the study or dissection of the dead without curative intent is tantamount to desecration of the corpse. There is a long intellectual and spiritual tradition of believing that the human body is sacred. This belief continues following death, and some reject dissection or manipulation as a form of desecration. This has been studied in the context of the autopsy. Some cultural or religious belief systems have requirements that the corpse of a decedent be buried whole and undisturbed. These beliefs may result in the reluctance of families to allow procedures or investigations on their loved ones after death.

An interesting footnote regarding the history of ambulance services is that they originally carried the dead to mortuaries in exchange for payment. The bodies of some patients were bought and sold to medical schools as teaching aids. Recently deceased bodies may offer the most lifelike opportunity for practicing new surgical procedures or learning anatomy.

Performing procedures on the deceased (e.g., central venous catheter placement or endotracheal intubation) is a long-standing tradition in medicine. Numerous authors have described the prevalence of this practice in the United States and abroad. This prevalence varies between specialties. More than 70% of neonatology training fellowships allowed trainees to practice endotracheal intubation and umbilical vein catheterization on recently deceased neonates.[3] This practice has decreased over the past several decades, perhaps due to advances in simulation technology. A recently published survey of Emergency Medicine residencies reported that 47% of program directors reported that this practice was ongoing at their institution, with or without official sanction.[4]

ETHICAL FRAMEWORKS AND OBJECTIONS

The general framework of medical ethics arises from four foundational principles (i.e., autonomy, justice, beneficence, and nonmaleficence). The question of whether this same framework can be readily applied to the topic of practicing procedures on deceased patients is a complex one because the "patient" is dead. The "patient" does not benefit from and is not harmed by the intervention. The "patient" does not have a formal moral standing. This may help the structured thinking about the topic.

The autonomy of a deceased person is questionable. Many do not make their preferences known before their death. The "patient" is not capable of voicing a preference or providing consent. This presents a problem for the requirement of autonomy unless these preferences were known beforehand. Collecting preferences after death is impossible outside of asking a proxy.

Beneficence and nonmaleficence are likewise difficult to consider with respect to the individual patient. The principle of beneficence may suggest that allowing a learner to practice a difficult and rare procedure on the newly deceased may allow future living patients greater chances at benefitting from an intervention and lessen the possibility of harm. A consequentialist ethical construct, a more utilitarian framework where the interests of an individual are subjugated to the interests of a larger population, might find that allowing a trainee to practice intubating someone who is dead meets the requirements of beneficence and nonmaleficence. This is due to the impact on future patients with a relative freedom from harm.

Consider the patients in whom this practice occurs to understand the principle of justice. The practice may not be equally distributed across different groups of patients or vulnerable populations (e.g., different financial classes, different sexual orientations, minorities, or those without families to speak on their behalf). It would fail this test of justice and be ethically wrong if one group were being used for procedural practice disproportionately.

PRACTICAL CONSIDERATIONS

Current practice attempts or has attempted to meet the above objections in different ways. Consent to perform procedures on the recently deceased is infrequently obtained. Policies regarding procedures on the recently deceased usually do not exist. Many lay people believe that practicing lifesaving procedures on the recently deceased is acceptable and that consent is required.[5,6]

INFORMED CONSENT

Multiple studies have examined the practicality of obtaining consent from families to practice resuscitation skills on the recently deceased. Residents and medical students learn in their training to address bereaved relatives and to have difficult conversations. The use of a loved one for procedural practice after they are deceased is a request that is intuitively uncomfortable. However, many families have given informed consent to use recently deceased babies for intubation if it helped to save other babies in the future.[3]

The reliance on familial consent sometimes does not follow the patient's wishes.[4] Presumed consent is problematic since the patient does not receive the benefit of the procedure. Health care personnel and medical trainees are often apprehensive and uncomfortable when procedures are performed on the recently deceased without consent. Obtaining consent respects the family, health care personnel, and medical trainees.

In Ohio, a person can be charged for practicing procedures on the recently deceased. The charge is abusing a corpse. This is based on the revised Code ORC 2927.01. The Code states the following: "(A) No person, except as authorized by law, shall treat a human corpse in a way that he knows would outrage reasonable family sensibilities. (B) No person, except as authorized by law, shall treat a corpse in a way that would outrage reasonable community sensibilities. (C) Whoever violates Division (A) of this section is guilty of abuse of a corpse, a misdemeanor of the second degree. Whoever violates Division (B) of this section is guilty of gross abuse of a corpse, a felony of the fourth degree."

SLOW CODES

An alternative form of teaching or allowing trainees to learn procedural skills is the continuance of resuscitative efforts with the purpose of allowing the practice of procedures (e.g., arterial line insertion, central venous line insertion, or pericardiocentesis), not because the resuscitation leader thinks it will lead to a successful resuscitation. This has been termed running a "slow code" by some. The American Nurses Association has issued a statement on so-called slow codes or submaximal resuscitation efforts.[7] They note that slow codes are not ethical and that partial codes often are not appropriate because they offer even less potential for survival than full codes.[7] With only a few exceptions, partial attempts to reverse a cardiac or pulmonary arrest are medically unsound because these interventions are often highly traumatic and consistently inefficacious.[8] A slow code often violates the principle of nonmalfeasance.

Some might question why this practice is not morally defensible when compared to practicing procedures on the recently deceased. Consider the potential harms, including cardiopulmonary resuscitation-induced consciousness and suffering, return of spontaneous circulation with poor neurologic outcome, and familial and medical professional distress. It quickly becomes clear why performing a slow code is ethically problematic.[9]

ADDITIONAL CONSIDERATIONS

Consider the relative invasiveness and lasting consequences on the body of various procedures when thinking about procedural practice in the deceased. Are some procedures that do not leave a mark on the outside of the body (e.g., endotracheal intubation attempt) or leave the body "intact" more acceptable to perform? Are more invasive procedures (e.g., surgical airways or a thoracotomy) that leave an external mark less acceptable? One study has suggested that family members are more comfortable with less invasive procedures than they are with more invasive procedures.[10]

Consider available alternatives for training. Clinicians practicing in an austere environment in developing countries have few alternatives for learning, so training on the recently deceased might be considered more permissible than if one practices in an environment with adequate hands-on opportunities with appropriate oversight and backup in living patients who can provide consent for a procedure, with ready access to preserved cadavers who gave antecedent consent to being used for learning, or with simulation facilities. Many manikins and animal models have been developed to address these issues but lack realism.

The legislature of individual states and the US Congress could develop a preauthorization form similar to organ donation. This form would have numerous advantages. The use of slow codes would be eliminated to teach procedures. Health care personnel would feel more comfortable. Hospitals would not have to make policies, and the administrators would not need to be involved. The need for family consent is avoided if the patient's wishes are known. The form would avoid using certain groups of patients more often than others.

NATIONAL GUIDELINES FROM THE AMERICAN MEDICAL ASSOCIATION

A series of articles and editorials in the lay press and the medical literature brought attention to the issue of performing procedures on the recently deceased. The Council on Ethical and Judicial Affairs of the American Medical Association (AMA) published a report on the topic in 2002.[11] A panel of experts was convened and published a document that focused on the question of the necessity of consent and offered the below recommendations regarding practice of procedures on the newly deceased.

Work to develop institutional policies that address the practice of performing procedures on the newly deceased for purposes of training. Include in any policies that the interests of all the parties involved are respected under established and clear ethical guidelines. Consider the rights of patients and their families, benefits to trainees and society, the potential harm to the ethical sensitivities of trainees, the risks to staff, the risks to the institution, and the risks to the profession associated with performing procedures on the newly deceased without consent. The lack of consent can damage the reputation of doctors and hospitals.

Address the following before trainees perform procedures on the newly deceased. The teaching of lifesaving skills should be the culmination of a structured training sequence, rather than relying on random opportunities. Training should be performed under close supervision. The environment and manner of performing procedures on the recently deceased should account for the wishes and values of all involved parties. Inquire whether the deceased individual had expressed preferences regarding handling their body or procedures performed after death. Request permission from the family in the absence of previously expressed preferences before performing procedures. Do not perform procedures for training purposes on the newly deceased patient when reasonable efforts to discover previously expressed preferences or someone with authority to grant permission for the procedure have failed.[9]

GUIDELINES FROM EMERGENCY MEDICINE PROFESSIONAL ORGANIZATIONS

The American College of Emergency Physicians (ACEP) Ethics Committee developed an information paper on the issues surrounding the practice of performing procedures on the newly dead but stopped short of developing an explicit statement on whether consent is required.[12] They recommended further research on the ethical ramifications, feasibility, public opinion, and consequences of asking for familial consent. Work has continued in these areas, although perhaps not at the pace requested.

An article published in 2004 offered a position from the Society for Academic Emergency Medicine.[13] It offered arguments both for and against this practice. It concluded by encouraging all Emergency Medicine training programs to develop a policy and make that policy available to the educators, institution, public, and trainees. It also recommended that families be asked for consent prior to practicing any procedures on the deceased.

SUMMARY

Procedural skills and proficiency are an incredibly important part of training in Emergency Medicine and many other medical specialties. Teaching these skills on living patients is the gold standard but has numerous potential downsides (e.g., the potential for unintended harm to patients as skills are attained). We are making rapid

TABLE 4-1 Some Recommendations for Using the Newly Deceased for Procedures

Conditions to be met:
- Consent is obtained before the procedure from a legal representative of the patient
- Documentation in the medical record of all other persons present
- Documentation in the medical record of any complications from the procedures
- Documentation in the medical record of consent and from whom
- Documentation in the medical record of the person performing the procedure
- Documentation in the medical record of a procedure note for any procedure performed
- Documentation in the medical record of the procedures performed
- Procedure performance is appropriate for the trainee
- Procedure performance is appropriate for the training program
- Procedure performance is supervised by faculty presence
- The cost will not be billed to the patient, the family, or the insurance company

Do not perform procedures if:
- Advance directives are against the procedures being practiced
- Consent is not obtainable due to lack of any legal representative
- Consent is not obtainable due to refusal of consent
- It is a medical examiner's case
- Member(s) of the health care team does not believe the procedure is appropriate
- The patient is a child unless the parents' consent
- Procedures are against cultural or religious beliefs
- Procedures interfere with an autopsy
- Procedures interfere with forensic evidence collection
- Procedures interfere with family visitation
- There is suspicion of patient abuse
- There is suspicion of patient neglect

progress as a profession in the use of simulation in training but have a long way to go before it can be used to its full potential. The practice of allowing trainees to perform invasive and noninvasive procedures on the recently deceased offers one way of gaining experience and technical skill without the possibility of patient harm.[9] The final recommendations are listed in **Table 4-1**.

Any personal or institutional approach to this practice must account for the ethical and pragmatic considerations. Balance the needs and interests of society and future patients with the legitimate interests of patients and their families. For the time being, it is likely that this practice of performing procedures on the newly deceased will continue. It should be done with care and thought. The consent of patients and their families will occur in ideal circumstances. Academic institutions and hospitals should talk about these issues before they arise and develop transparent policies that reflect a commitment to patient care above all else.

In the future, medical education and simulation will continue to progress. The use of simulation training mannequins and virtual reality technologies will become more prevalent, and the need for discussing this issue may be eliminated.

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5 Aseptic Technique

John S. Rose

INTRODUCTION

The proper use and understanding of aseptic technique are critical for the care of patients in the Emergency Department (ED). Aseptic technique dovetails with prescribed universal precautions and is central to the practice of Emergency Medicine. Knowledge of proper aseptic technique ensures that procedures performed in the ED provide maximal protection for the patient and the Emergency Physician while keeping the risk of contamination as low as possible.[1-19]

Wound infection and sepsis are the two major complications resulting from poor and improper aseptic technique. Other complications that may contribute to the patient's morbidity and mortality include increased length and cost of hospital stay, patient discomfort, scarring, and death. **Aseptic technique is warranted except in the direst circumstances.**

Numerous terms are used to describe the establishment and maintenance of a "sterile" environment. These include aseptic, disinfection, and sterile technique, to name a few. Many people often incorrectly interchange these terms. The proper definitions of the terms used to describe aseptic technique or associated with it can be found in **Table 5-1**.

ANATOMY AND PATHOPHYSIOLOGY

The skin and hair are colonized with various organisms. The stratum corneum layer of the epidermis is colonized with a polymicrobial flora. This includes molds, *Staphylococcus aureus, Staphylococcus epidermidis*, various *Streptococcus* species, viruses, and yeasts. Many of these organisms are nonpathogenic, even when placed in environments considered appropriate for infection. ***S. aureus* is the most common cause of wound infections.** It can result in an infection when introduced into deeper skin layers. Some species (e.g., *S. epidermidis*) are pathologic only when inoculated into deeper layers of the skin and soft tissue. A significant inoculation is required for most infections to create a critical level for microbial growth. **Aseptic technique decreases bacterial exposure and reduces the level of potentially pathologic organisms.**

TABLE 5-1 Definitions of Terms Used to Describe Aseptic Technique or Associated Processes

Term	Definition
Aseptic	Freedom from infection. Prevention of contact with microorganisms. Involves the use of sterile technique and skin disinfection.
Clean technique	The practice of using nonsterile equipment to perform procedures. This is considered as part of the universal body fluid precautions.
Disinfection	The cleaning of an area to make it free of pathogenic organisms and microbes.
Sterile field	The zone in which strict sterile technique is maintained. Generally consists of an area 3 to 10 times larger than the area of the primary procedure.
Sterile technique	The practice of utilizing sterile equipment and procedures to maintain an aseptic environment.
Super aseptic	Ultra-high state of an aseptic environment. Usually, this is achievable only in the operating room.

INDICATIONS

The role of aseptic technique in the ED is primarily for invasive procedures. Invasive procedures require varying degrees of aseptic technique. Placement of a small peripheral intravenous catheter may require no more than a brief wiping of the skin. A diagnostic peritoneal lavage requires Operating Room-level disinfection and strict sterile technique.

Routine and adequate provider disinfection involves careful hand washing, the use of clean and disinfected personal diagnostic equipment (e.g., stethoscopes), and wearing appropriately cleaned coats and clothing. This is critical in preventing iatrogenic infections in the ED. Aseptic technique in the ED can be referred to as clinical aseptic technique, since it is virtually impossible to achieve an Operating Room level of asepsis. **Clinical aseptic technique involves the combining of adequate disinfection with sterile techniques and protocols at the bedside.**

CONTRAINDICATIONS

There are very few contraindications to the maintenance of adequate clinical aseptic technique. One exception would be that extreme clinical circumstance in which time simply does not allow proper aseptic technique (e.g., an emergent thoracotomy). The Emergency Physician can still use sterile gloves and a quick application of an aseptic solution.

Always inquire about allergies and sensitivities to latex and antiseptic solutions. This information will affect the equipment that is chosen to properly prepare the patient.[8] Hospitals have a latex-free cart that contains equipment for use with latex-allergic patients. **Do not use povidone iodine solution in patients allergic to iodine.** Alternative agents include chlorhexidine and hexachlorophene preparations.

There are some relative contraindications to using some disinfectants. Do not use alcohol-based disinfectants near the eyes, inner ear, mucous membranes, or open wounds. Chlorhexidine can damage the corneal epithelium. Use only ophthalmic-approved iodine-based disinfectants. Chlorhexidine can cause deafness if it reaches the inner ear. Use aqueous-based disinfectants for the ears. Use chlorhexidine with caution around the mucous membranes. Check manufacturers' recommendations or use an aqueous iodine-based disinfectant.

EQUIPMENT

- Povidone iodine solution
- Chlorhexidine gluconate (i.e., chlorhexidine) or hexachlorophene-based solutions
- 70% isopropyl alcohol
- Sterile 4 × 4 gauze squares or applicator sticks
- Sterile gloves
- Face mask and eye protection
- Sterile drapes or towels
- Adequate lighting
- Sterile gowns
- Surgical hat
- Bedside procedure table

PATIENT PREPARATION

Inform the patient of what the procedure entails before performing any procedure in the ED. This should include an explanation of sterile technique and a request that the patient not touch the drapes or sterile equipment. Obtain any required informed consent (Chapter 1) before the patient is draped. The only exception to this is if an emergent and lifesaving procedure must be immediately performed.

Place the patient in the most comfortable position possible. Patient discomfort frequently results in movement and the potential loss of the sterile field. Use sedation and/or analgesia (Chapters 153 to 159) as necessary to facilitate proper patient positioning. The Emergency Physician must also be comfortably positioned if possible and have adequate lighting.

TECHNIQUES

Aseptic technique can be divided into skin disinfection and sterile technique. Skin disinfection removes any microorganisms found on the skin and decreases the potential contamination during the procedure. Sterile technique is performed for the same reason. There are different levels of aseptic technique, ranging from full aseptic technique (i.e., cap, mask, sterile drapes, sterile gloves, and sterile gown) to simple sterile gloves. The physician must use their judgment to determine which level is most appropriate to the task at hand.[8]

SKIN DISINFECTION

Disinfection involves the application and scrubbing of a disinfectant preparation onto the skin. Simple procedures (e.g., injections or venipunctures) may require little disinfection. Wipe the skin with gauze that has been impregnated with 70% isopropyl alcohol for simple procedures. The alcohol has an antibacterial effect. **The mere force of wiping the skin reduces bacterial counts.** No disinfection is used for simple venipunctures in some countries. More comprehensive skin preparation involves the use of a disinfectant agent (e.g., chlorhexidine solution or povidone iodine).

Chlorhexidine, povidone iodine, and 2% iodine tincture are the most commonly used skin antiseptic solutions. Povidone iodine solution is highly germicidal for gram-positive and gram-negative bacteria, viruses, fungi, protozoa, and yeasts.[7] It rapidly reduces bacterial counts on the skin surface and can last up to 3 hours.[7,11] Allow the iodine solution to dry and then wipe it from the skin with 70% alcohol prior to beginning the procedure. **The iodine solutions work by oxidation and cross-linking of sulfhydryl groups, killing bacteria as the solution dries.** Isopropyl alcohol can be applied

to the skin and scrubbed vigorously for 2 minutes to achieve disinfection, although this may cause skin irritation. Chlorhexidine or hexachlorophene preparations may be routinely used or used as substitutes in iodine-allergic or sensitive patients. These agents provide good bactericidal activity against gram-positive bacteria but somewhat less activity against gram-negative organisms.[8]

Chlorhexidine-based solutions are being used more commonly and are replacing the iodine-based solutions. Chlorhexidine provides much longer antimicrobial activity (e.g., up to 48 hours) and is gentler on the skin than iodine.[11-15] **Chlorhexidine destroys cell membranes of gram-positive and gram-negative bacteria while precipitating the intracellular contents.** Some preparations contain 70% isopropyl alcohol, further enhancing the antimicrobial activity.[12-14] The use of chlorhexidine solutions is superior to iodine solutions.[14,15,17,19]

Use a skin disinfectant for procedures other than simple venipuncture. Place the disinfectant solution onto a sterile sponge or sterile gauze if it is not supplied inside a single-use applicator. Historically, the application of disinfectant to the skin has been in a circular motion, beginning with the central area of the procedure and working out toward the periphery of the sterile field (**Figure 5-1**). There is no evidence to support this application method. It has been suggested that scrubbing in a back-and-forth motion may be preferable because it creates friction to dislodge microbes.[9,10] The back-and-forth motion drives the disinfectant solution into skin crevices and deeper layers, thus killing more bacteria and hopefully preventing infections.

Regardless of the disinfectant solution used, repeat the application process three or four times using a new sponge, gauze square, or applicator each time.[8] The technique of applying the disinfectant several times ensures that the central area where the procedure is to be performed is the most sterile area of the field. **The area of disinfection must be much larger than the primary area of the procedure, as the number of organisms increases toward the periphery of the prepped area.**

STERILE TECHNIQUE

General sterile technique is described followed by specific details for each step of the procedure. Strict sterile technique is virtually impossible in the ED. **Make every effort to maintain a sterile field to minimize infection.** Assemble all equipment necessary and place it on a small procedure stand. **Do not use the patient or their bed to set up supplies or equipment.** Patient movement and their irregular body surfaces can result in items becoming contaminated, breaking, or falling or iatrogenic needle sticks. Avoid having different components scattered around the procedure area. Open all sterile items, using proper sterile protocol, to have them available once the Emergency Physician has donned sterile gloves. Use anesthetic solution containers with removable caps. This allows the Emergency Physician to draw up anesthetic without having an assistant and minimizes the risk of occupational needle exposure. **Perform a thorough hand washing before the procedure.**

Use eye, face, and hair protection during the procedure. Apply these before donning sterile gloves and sterile gowns. Apply sterile gloves. Place sterile drapes or towels on the patient to form a field wide enough to allow for a comfortable work space. Drape the area near the patient closest to the bedside procedure table. This will minimize inadvertent contamination in moving from the table to the patient. Make a small flat sterile area near the procedure site to allow for placement of important items that must be immediately available. **Open all caps, position stopcocks, and prepare all devices prior to starting the procedure.** The likelihood of contamination increases if devices are not adequately prepared and require manipulation during the critical portion of a procedure. **Adhere to universal precautions guidelines.**

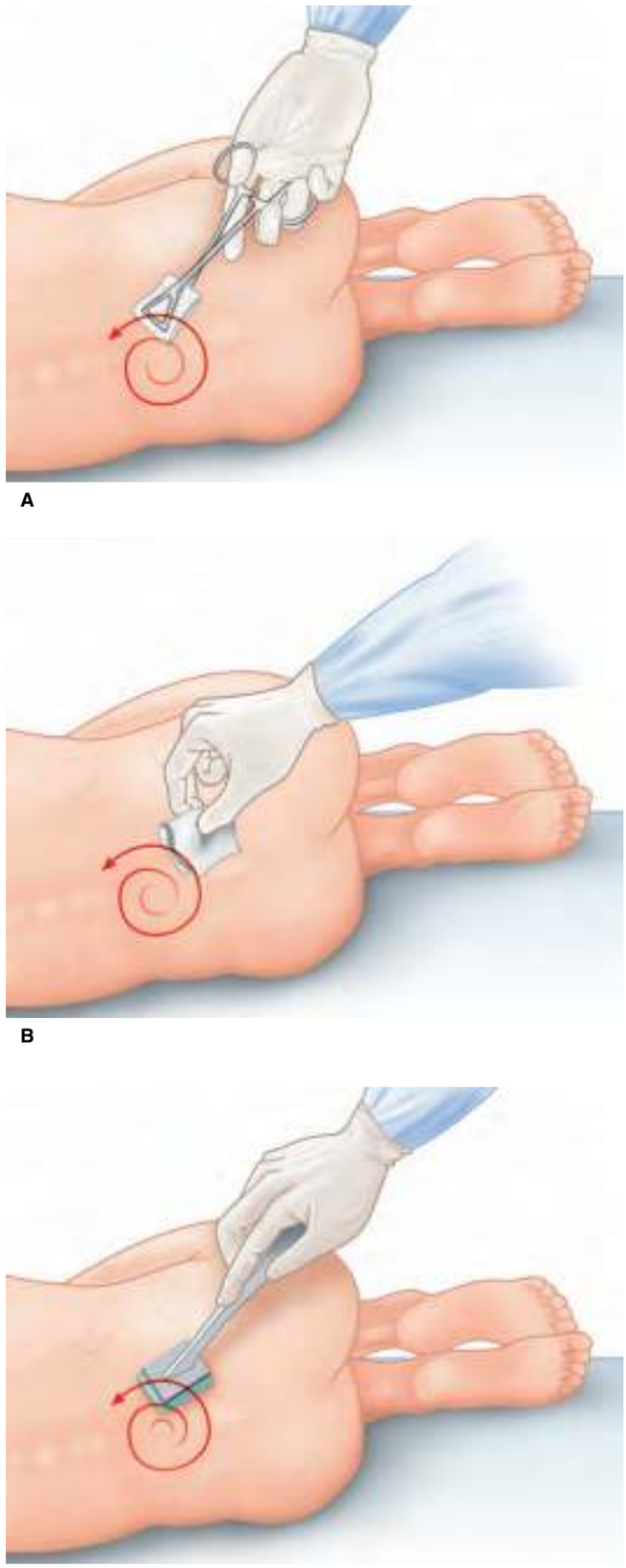

FIGURE 5-1. Preparation of the skin. Disinfectant solution is applied in a concentric circular pattern starting from the procedure site and working outward. Apply the disinfectant solution with sterile gauze held in a clamp (*A*), with sterile gauze held in a sterile gloved hand (*B*), or with a sponge on a stick (*C*).

OPENING A STERILE PACK

Always make sure that the outer wrapping is intact, the sterility expiration date has not passed, and the sterility indicator tape is the appropriate color before opening a sterile pack.[2] Wash your hands and then remove the outer wrap if applicable. Remove the sterility indicator tape (**Figure 5-2*A***). Place the sterile pack on a dry and level surface with the outermost flap facing away from you (**Figure 5-2*B***). Grasp the corners of the outermost flap (**Figure 5-2*B***). Hold your arms to the sides of the pack to avoid reaching over the sterile area. Lift the flap up and away (**Figure 5-2*B***). Open the side flaps by grasping the folded corner with a thumb and index finger and pulling the

A

B

C

D

E

FIGURE 5-2. Opening a sterile pack. ***A.*** Remove the sterility indicator tape. ***B.*** Grasp the edges of the outermost flap and open it away from you. ***C.*** Open the side flaps. ***D.*** Open the remaining flap toward you. ***E.*** The open pack.

flap to the side (**Figure 5-2*C***). Open the bottom flap (**Figure 5-2*D***). Grasp and open the bottom flap while stepping back to prevent contaminating the wrap on your clothing. **Make sure that your arms and clothes do not contaminate the contents of the pack when opening the flaps.** Repeat the procedure if the pack has an inner wrap.

PLACING STERILE SUPPLIES ON A STERILE FIELD

Sterile supplies are generally packaged in a hard peel-back pack (i.e., hard pack) or a soft peel-back pack (i.e., soft pack). The general principle of opening these is the same, although there are subtle differences. Hold the hard peel-back container in the nondominant hand with the flap facing the sterile field (**Figure 5-3*A***). Pull the flap toward you with the dominant hand so that the open end of the pack will be facing the field (**Figure 5-3*A***). Hold the container 15 to 20 cm above the sterile field. This ensures that if the contents fall, it will be onto the sterile field where they are wanted. Drop the contents of the sterile pack onto the sterile field taking care not to contaminate the field with the container (**Figure 5-3*B***).

Gloves and syringes are wrapped in soft packs. Grasp both sides of the unsealed edge of the soft pack and pull them apart slightly

A

B

FIGURE 5-3. Opening a hard peel-back container. ***A.*** Grasp the container with the flap facing the sterile field. Remove the flap. ***B.*** Drop the contents of the hard container onto the sterile field.

A

B

FIGURE 5-4. Opening a soft peel-back container. ***A.*** Grasp both sides of the unsealed edge and pull them apart. ***B.*** Face the pack toward the sterile field. Continue to open the edges until the contents fall onto the sterile field.

(**Figure 5-4*A***). Hold the open end facing the sterile field (i.e., away from you). Continue to open the soft pack. Fold the sides of the sterile packing back and over your hands to keep the contents sterile (**Figure 5-4*B***). Gently drop the contents of the soft pack onto the sterile field.

APPLICATION OF A MASK

Surgical masks serve a dual role in the performance of aseptic technique. Masks have been shown to decrease contamination of the sterile field that may result from aerosolized droplets from the mouth and nose. Masks protect the caregiver's mucous membranes from exposure and possible splashing during the procedure. Wear a mask with an eye shield during high-risk procedures.

Apply the mask before donning gloves and other sterile equipment. Secure the mask by placing the elastic straps around the ears, placing the elastic straps around the head, or tying the mask securely to the face with ties around the head and neck depending on the type and style of the mask. Pinch the metal nose clip securely to the bridge of the nose for a tighter fit and to minimize the gap between the mask and the nose.

HAND WASHING

An Emergency Physician must thoroughly wash their hands despite wearing sterile gloves for all sterile procedures. Do not overlook good hand washing technique. A full surgical scrub is neither necessary nor feasible in the ED.

Rinse your hands in warm water prior to applying antiseptic soap. Apply soap, lather your hands, and rub them together vigorously for approximately 10 seconds. Wash each wrist with the opposite hand. Interlace the fingers of both hands and slide them back and forth to clean the web spaces. Clean around the nails with the fingertips and nails of the opposite hand. Completely rinse each hand from the fingers downward. Repeat the procedure a second time if your hands were grossly contaminated. Dry your hands with a disposable towel. Turn off the faucet, using the towel with which you dried your hands. **Do not touch the faucet with clean hands to prevent them from being recontaminated.**

APPLICATION OF A CLEAN GOWN

A clean (i.e., nonsterile) gown is often used as an additional barrier to prevent contamination of the field and the Emergency Physician's clothing. Simply place your arms into the sleeves and pull on the gown with the opening toward the back. Secure the gown at the back of the neck and the lower back by tying the strings.

APPLICATION OF A STERILE GOWN

A sterile gown is worn for procedures requiring a stricter sterile technique (i.e., central venous access or diagnostic peritoneal lavage). Use the procedure previously described to open a gown's sterile soft pack. Grasp and pick up the gown just below the neckline, touching only the inner surface of the gown. Hold the gown up and let it unfold with the inside facing you. **Do not allow the gown to touch any nonsterile surfaces.** Insert your arms into the sleeves until the gown is in place. Have an assistant grasp the back of the gown, pull it completely on, and tie the strings securely.

APPLICATION OF STERILE GLOVES

Wash your hands thoroughly before putting on sterile gloves. Apply a clean gown at this point if it will be worn during the procedure. Open the outer wrap of the sterile gloves and remove the inner wrap (**Figure 5-5*A***). Place the inner wrap on a clean

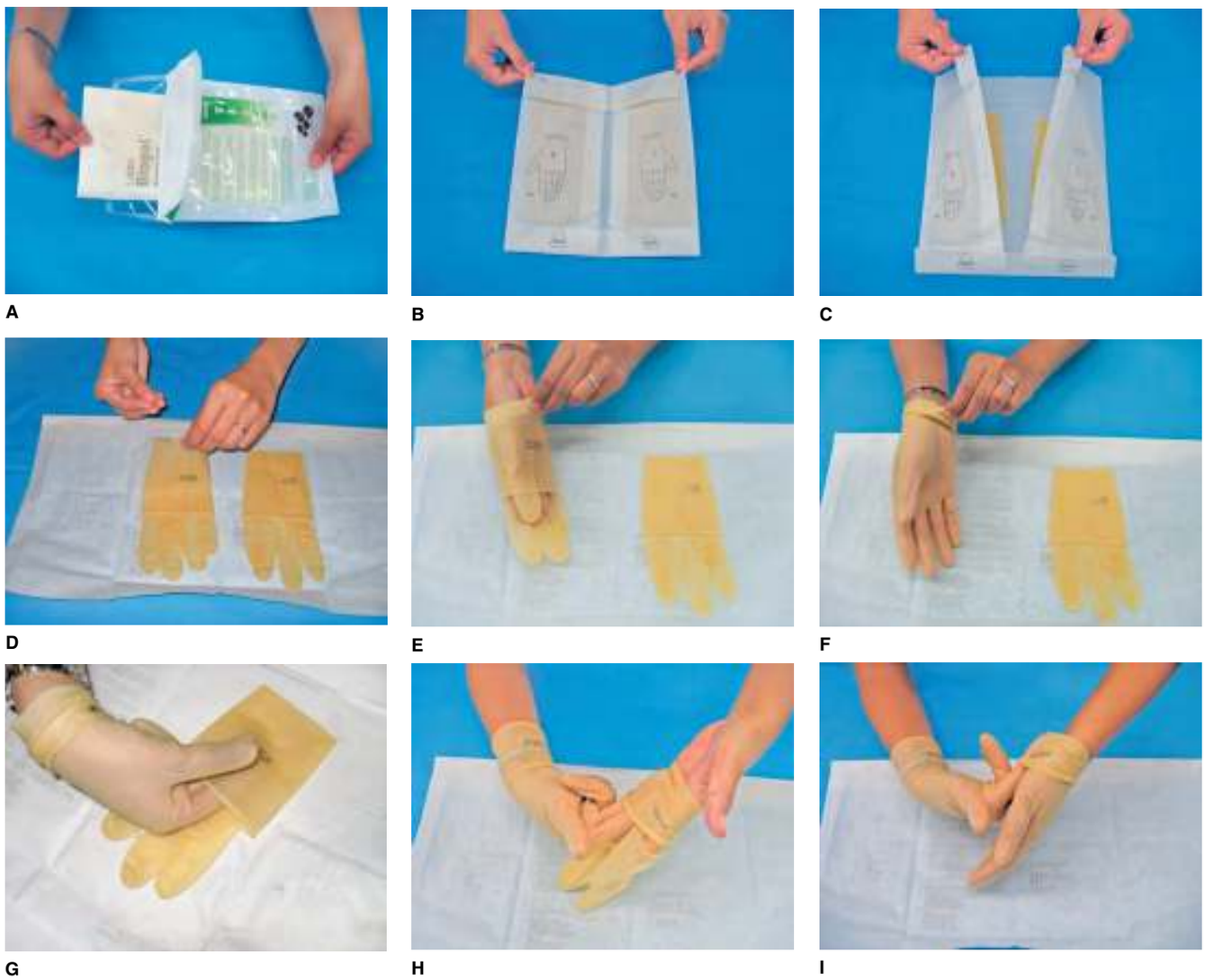

FIGURE 5-5. Application of sterile gloves. ***A.*** Open the outer wrap and remove the inner wrap. ***B.*** Unfold the inner wrap. ***C.*** Completely open the inner wrap. ***D.*** Grasp the cuff of a glove. ***E.*** Slip the glove onto the hand. ***F.*** Pull the glove onto the hand. ***G.*** Slip the gloved hand into the folded cuff of the second glove. ***H.*** Slip the glove onto the hand. ***I.*** Pull the glove onto the hand.

surface with the gloves' wrists facing toward you. Unfold the inner wrap touching only the outside edges (**Figure 5-5*B***). Open the inner wrap according to the procedure for opening a sterile pack (**Figure 5-5*C***). Apply a sterile gown if it will be worn during the procedure. Use the dominant hand to grab the opposite glove at the inner edge of its folded cuff (**Figure 5-5*D***). Slip the nondominant hand into the glove (**Figure 5-5*E***). **Be careful not to touch the outer surface of the glove (Figure 5-5*E*).** Pull the glove further onto the nondominant hand using the inner edge of the cuff (**Figure 5-5*F***). Place the fingers of the gloved nondominant hand into the folded cuff of the other glove (**Figure 5-5*G***). Slip the dominant hand into the glove (**Figure 5-5*H***). Pull this glove over the dominant hand using the cuff (**Figure 5-5*I***). Carefully unfold the cuff of each glove. **Use care not to touch the fingers and palms of the gloves to nonsterile skin.** Adjust each glove to ensure a snug fit over the fingers and hand.

REMOVAL OF PROTECTIVE CLOTHING

Remove protective clothing in a systematic manner to protect yourself and others from the contaminants on the gown and gloves (**Figure 5-6**).[20] Place all removed garments into the appropriate waste containers. The first step is to untie the gown (**Figure 5-6*A***). Have an assistant untie the neck strings of the gown or pull on both shoulders of the gown to break the neck strings. Untie the waist strings. Take off the gown by turning it inside out as it is removed (**Figure 5-6*B***). Roll up the gown with the contaminated surface facing inward and away from you (**Figure 5-6*C***). Dispose of the gown. Remove the gloves by turning them inside out. **Make sure that you do not touch the outside contaminated surface with ungloved hands.**[20] Use the dominant hand to grasp the cuff of the glove on the nondominant hand (**Figure 5-6*D***). Pull the glove inside out as it is removed and throw it away. Place the ungloved

FIGURE 5-6. Removal of protective clothing. ***A.*** Untie the gown. ***B.*** Remove the gown by turning it inside out. ***C.*** Roll up the gown with the contaminated side facing inward. ***D.*** Remove the glove from the nondominant hand. ***E.*** Remove the glove from the dominant hand. Remove the face mask and wash your hands.

fingers of the nondominant hand into the inside edge of the gloved dominant hand and remove the glove by pulling the glove inside out (**Figure 5-6*E***). Dispose of the glove. Remove the mask by untying its ties or removing the elastic straps from behind your ears. Dispose of the mask and wash your hands.

ULTRASOUND TRANSDUCER PREPARATION

Ultrasound is commonly used for many procedures. The application of sterile transducer covers allows for real-time and dynamic sonographic assessment during a procedure. Prepackaged transducer covers are available and are often included in procedure kits.

Open the prepackaged sterile transducer cover kit. Identify the sterile coupling gel, cover, and associated rubber bands to securely cover the transducer (**Figure 5-7*A***). Apply coupling gel to the transducer (**Figure 5-7*B***). Insert a gloved hand onto the front portion of the cover and push it inside out (**Figure 5-7*C***). Carefully grasp and hold the transducer through the cover (**Figure 5-7*D***). Apply the remaining cover over the transducer (**Figure 5-7*D***). Expel all the bubbles from the gel at the face of the transducer. Apply the supplied rubber bands to secure the cover in place (**Figure 5-7*E***).

DOUBLE GLOVING

Some Emergency Physicians wear two gloves in case the first one is cut. This offers some protection against the patient's blood and body fluids. The second glove sometimes protects against bloodborne pathogens. It may be hard to tell when the glove is cut if the skin is cut or intact until the glove is removed. Companies are now making double-dipped, dual-color gloves (**Figure 5-8**). These gloves are twice as thick as ordinary gloves. Cutting the outer white surface exposes the underneath color, making it easier to see glove perforations.

A

B

C

D

E

FIGURE 5-7. Applying the transducer cover. ***A.*** The contents of the sterile kit. (From left to right: transducer cover, rubber bands, and gel pack.) ***B.*** Apply the gel to the transducer. ***C.*** Insert a hand into the sterile cover. ***D.*** The transducer is grasped through the cover. ***E.*** The cover has been unrolled over the transducer. Rubber bands are applied to secure the sterile cover.

FIGURE 5-8. An example of a double-dipped dual-color glove.

COMPLICATIONS

Properly performed aseptic technique has very few complications. The primary risk is in patients with sensitivities or allergies to latex or the disinfectant preparations. Povidone iodine preparations are much less irritating than tincture of iodine. It is a good policy to clean all disinfectant off the patient at the end of the procedure to minimize any skin irritation. This is especially true of small children. Use alternate products for those patients with histories of allergies. **The main complication of improper aseptic technique is infection at the site of the procedure.[8] This only serves to underscore the need to perform aseptic technique properly.**

SUMMARY

Aseptic technique is an important component of all invasive procedures performed in the ED. Adequate skin disinfection and the proper use of sterile technique will greatly decrease the risk of iatrogenic infections. Aseptic technique allows a degree of protection for the Emergency Physician and the patient. Choose the level of asepsis required for the individual procedure.

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6 Basic Principles of Ultrasonography

Basem F. Khishfe

INTRODUCTION

The Emergency Physician (EP) has performed bedside ultrasound (US) for more than three decades. US is ubiquitous today in Emergency Medicine (EM). Each year, new EPs learn the skill, fresh evidence is brought to light supporting the practice, and novel indications are explored.[1] Technological advances have delivered smaller machines with improved image quality that are less expensive than ever before. US training is an important component of an EM residency program and a required core competency procedure of the Accreditation Council for Graduate Medical Education Residency Review Committee.[2] It is tested on board examinations and is endorsed by EM societies.[1,3] There are numerous opportunities for supplementary training in emergency US ranging from local courses to established fellowship training programs.

Safety considerations have also contributed to the acceptance of bedside US. Sonography is noninvasive, is safe in pregnancy, does not involve ionizing radiation, does not require nephrotoxic contrast agents, does not cause subcutaneous extravasation, and does not cause allergic reactions like other modes of imaging.[4] With increasing concern in the medical community over the long-term effects of ionizing radiation, US is recognized as an attractive alternative.[5] The U.S. Department of Health and Human Services Agency for Healthcare Research and Quality has highlighted US guidance for central line insertion as one of their top 10 recommended practices.[6]

The intent of this chapter is to provide an introduction to bedside US. The physician-sonographer should have a general understanding of the physics underlying the properties of an US wave.

"Knobology" is a colloquial term used to describe the study of the buttons, dials, switches, and knobs on the console of an US machine. **It is important that all users have a good sense of their machine's operational functions.** Typical US machines and transducers used in the ED will be described.

BRIEF HISTORY OF EMERGENCY ULTRASOUND

Bats and toothed whales have used sound for echolocation for millions of years. Dolphins produce a series of clicks that pass through the lipid-rich melon on their heads, an acoustical lens of sorts that focuses the sound waves into a beam. Returning echoes are processed to determine the location of objects for navigation and hunting.[7] Sonar (acronym for *so*und *na*vigation and *r*anging) is a maritime technique that uses sound waves for identifying oceanic objects. It was initially introduced in response to the sinking of the *Titanic* in 1912.[8] It was further developed and employed in World Wars I and II for submarine detection.[8]

In the early 1950s, a Radiologist named Douglas Howry and a team of other physicians introduced the first diagnostic US machine using a water-bath immersion tank. In the 1960s, direct contact (i.e., transducer to patient) scanners were developed, and the contemporary saga of US as a viable diagnostic modality commenced.[9] The technology was only found in specialized imaging laboratories. US technology improved rapidly. Real-time US was developed in the 1980s, allowing the viewing of images without delay between signal generation and the display monitor. US devices continued to improve, and technological advances have delivered smaller portable machines with improved image quality that are less expensive than ever before. The clinical applications growth paralleled those advancements. As early as the 1970s, Surgeons in Germany experimented with US to detect free fluid in the abdomen.[10] In the late 1980s, EPs began investigating the clinical use of US. The American College of Emergency Physicians (ACEP) offered its first course in emergency applications of US in 1990.

US has been examined over the past two decades and used for a wide spectrum of clinical conditions. A number of factors helped the development of emergency US, including the growing recognition of the utility of US information, the improved US technology, the accessibility to bedside US machines, the need for timely access to diagnostic imaging, and the endorsement of US by the EM societies. Several US manufacturers have developed machines targeted to EM, taking into consideration the specific indications and the less-than-forgiving work environment.

In the early years of emergency US, EPs often used large tank-like machines that were not conducive to a fast-paced work environment. Small portable machines that produce high-quality images are now available. Many US devices are the size of laptop computers that fit on maneuverable carts. Pocket-sized machines, not much larger than mobile phones, are being introduced and offer even greater portability. The notion of the "sono-stethoscope" promises devices as small, easy to use, and available as the stethoscope, but with the superior insight of sonography.

THE PARADIGM OF BEDSIDE ULTRASOUND

Bedside US offers a relatively new paradigm in clinical medicine. Traditional US involves a break in the patient–physician encounter. The multistep process involves the EP evaluating the patient and determining that an US is warranted, the Radiologic Technologist performing the study in an US suite, the Radiologist reviewing the images and generating a report, and the EP ultimately correlating that information back to the patient (**Figure 6-1**).

Bedside US creates a direct and immediate relationship between patient and EP (**Figure 6-1**). The technology is placed in the EP's hand for both image acquisition and interpretation. The EP can immediately synthesize US findings with clinical and laboratory data to paint a more complete diagnostic picture. A patient's condition can change on a moment's notice in the ED. US is a dynamic tool that can be used swiftly and serially throughout a patient's course.

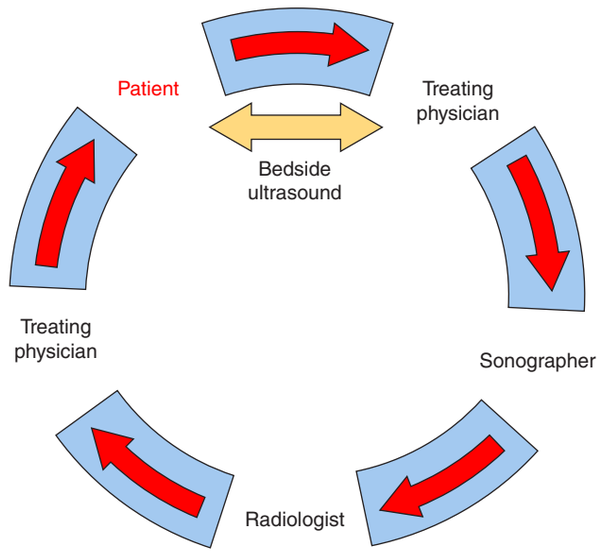


FIGURE 6-1. The paradigm of bedside US. The workflow of traditional US using the Radiology Department (blue circle with red arrows) is a multistep process that may take hours to days to complete. Bedside US (double yellow arrow) establishes an immediate and direct interaction between patient and physician. (Courtesy of Christopher Moore, MD, RDMS, RDCS.)

INDICATIONS

The primary indications of emergency US have traditionally included: cardiac US for the presence of pericardial fluid and for cardiac activity; abdominal US for the identification of free peritoneal fluid; aortic US for abdominal aneurysms; biliary US for the detection of gallstones and cholecystitis; renal US for hydronephrosis and nephrolithiasis; and pelvic US for the identification of an intrauterine pregnancy and the exclusion of an ectopic pregnancy (**Table 6-1**).[11-13] Basic emergency US has operated on the binary premise that emergencies are to be included or excluded by this diagnostic tool. The boundaries between primary and secondary indications have blurred, and the yes/no equation for the evaluation of emergencies has matured.

The list of indications for present-day emergency US is long and continually expanding (**Table 6-1**).[11-13] Another common indication is the evaluation of deep vein thrombosis. Lung US can be used to

TABLE 6-1 The Indications for Emergency Ultrasonography

Primary Indications	Secondary Indications	Procedural Indications
Abdominal	Abscess	Arthrocentesis
Aorta	Deep vein thrombosis	Foreign body localization
Biliary	Gastrointestinal	Lumbar puncture
Cardiac	Genital	Nerve blocks
First trimester pregnancy	Lung	Paracentesis
Renal	Musculoskeletal	Pericardiocentesis
	Ocular	Thoracentesis
	Shock	Transvenous pacer placement
	Other	Vascular access
		Other

diagnose a pneumothorax, pleural fluid, and interstitial lung diseases. US can be used for the evaluation of subcutaneous abscesses, peritonsillar abscesses, deep-space abscesses, and abdominal abscesses. Ocular US can be used to identify a retinal detachment, lens dislocation, vitreous hemorrhage, and foreign bodies and in optic nerve sheath assessment for increased intracranial pressure. Hypotensive patients or those in shock may be evaluated by US of the inferior vena cava and internal jugular veins to estimate central venous pressure. Gastrointestinal US is used for the identification of appendicitis, pyloric stenosis, and other conditions. Genitourinary US is used to look for testicular or ovarian torsion, intrauterine pregnancy, and ectopic pregnancy. Musculoskeletal US is used to diagnose joint effusions, tendinopathy, and fractures. Procedural US is used for guided assistance of vascular access, paracentesis, thoracentesis, pericardiocentesis, arthrocentesis, lumbar puncture, foreign body removal, nerve blocks, and other procedures.

ULTRASOUND PHYSICS

SOUND

Sound is a variation in pressure traveling through a medium, and it is described as a wave. We commonly understand the sound of our vocal communication to travel through air. Sound can also travel through fluid and solid structures. Pressure variations produced by sound waves mechanically displace or oscillate the particles of the medium. This oscillation produces cycles of higher and lower densities, or compression and rarefaction, respectively. Sound waves differ from water waves in that they are longitudinal, meaning the cycles of compression and rarefaction travel in the same direction as the wave. Imagine an analogous spring coil with a force exerted into its length. A wave, or region of compression, will pass down through the coil in a longitudinal or parallel fashion (**Figure 6-2**). It is important to have an understanding of the terms that describe sound waves as described in the following sections.

FREQUENCY AND PERIOD

Frequency is the number of cycles of pressure variation per 1 second. A single cycle begins at a baseline of absent sound, increases to a maximum value (i.e., compression), decreases to a minimum value (i.e., rarefaction), and returns to baseline (**Figure 6-2**). Frequency is measured in hertz with a unit of cycles per second and corresponds to the pitch or tone of a sound. The upper acoustical frequency of human hearing is approximately 20,000 hertz, beyond which is considered "US." Diagnostic US frequencies are generally in the range

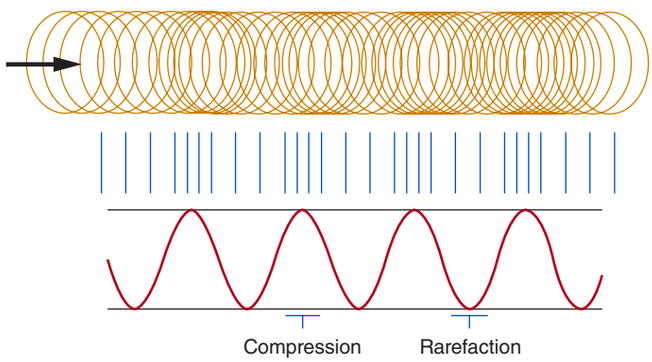


FIGURE 6-2. The longitudinal US wave. A force exerted in a parallel fashion along a coil will produce regions of compression and rarefaction. The particles of a sound wave vibrate in the direction the wave travels to create areas of high and low density and pressure. A sound wave is depicted as a classic waveform with peaks representing compression and valleys representing rarefaction.

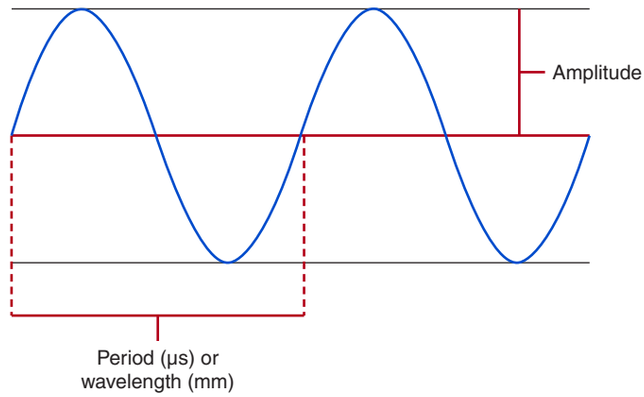


FIGURE 6-3. The characteristics of an US wave. Period is the duration in time of a single cycle of a wave. Wavelength is the distance in space. Amplitude measures the wave's variation (i.e., height) from baseline.

of 1 million to 10 million hertz or megahertz (MHz). US transducers operate on either the "low" or "high" end of this frequency spectrum, although for each transducer, frequency can be adjusted within a limited range. Frequency is a principal determinant of the resolution and penetration of an image.

Period is the time required for one cycle to occur. Thus, it is the inverse of frequency (**Figure 6-3**). In ultrasonography, period is typically measured in microseconds (μs).

WAVELENGTH AND PROPAGATION SPEED

While period measures the duration in time of a single cycle, wavelength measures the distance in space of a single cycle (**Figure 6-3**). Wavelength is related to the propagation speed and the frequency of a wave. It is represented by the following equation: wavelength = propagation speed ÷ frequency.

Propagation speed is the velocity at which sound travels through tissue. An average speed through soft tissues of 1540 m/s is generally assumed in basic US. Newer US machines incorporate the propagation speeds of different tissues to provide enhanced imaging.

AMPLITUDE, OUTPUT, AND BIOEFFECTS

Amplitude is a measure of the height or maximum variation of a wave from baseline (**Figure 6-3**). While frequency corresponds to the pitch or tone of a sound, amplitude is the "loudness" or volume of a sound. This correlates to the brightness of the image. Adjustments of the US machine's output affect the amplitude. Changing the gain produces a similar effect on received echoes. It is preferable to alter brightness on the back end with gain, rather than subject the patient to increased output. Diagnostic US has proven to be exceedingly safe. Concerns do exist over possible thermal and mechanical adverse bioeffects.[14] EPs should strive to perform studies in the shortest time frame, at the lowest output possible, and in-line with the safety acronym ALARA (*as l*ow as *r*easonably *a*chievable).

PULSED ULTRASOUND

The earliest US machines produced a continuous stream of US waves. Today's machines release pulses or packets of waves, that is, a few cycles of US at a time. The repetitive pulses are separated by gaps of no sound. Machines can generate pulses of varying duration, frequency, and fraction of time with respect to the soundless gaps. Pulsed US has been essential in the development of advanced imaging.

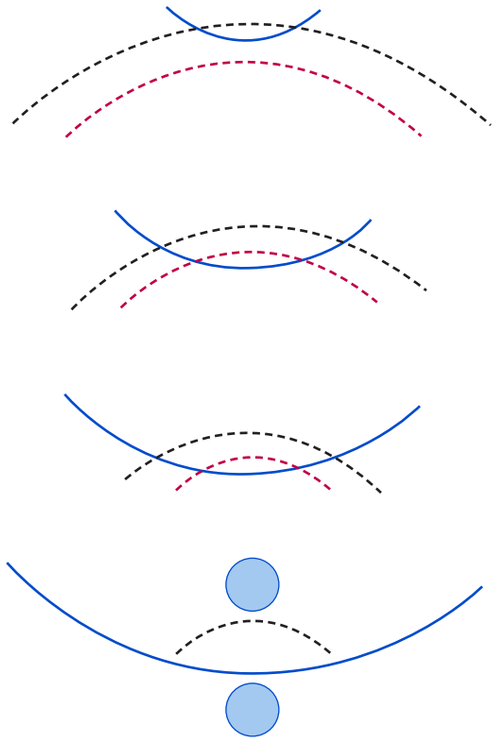


FIGURE 6-4. The axial resolution of structures. Blue curves represent the transmitted US beam. Red and black curves represent the returning echoes of the superficial and deep structures, respectively. Increasing the frequency shortens the wavelength and improves the axial resolution.

AXIAL RESOLUTION

Axial resolution refers to the ability of the US machine to distinguish two separate structures that lie on top of one another and in a parallel plane to the US beam (**Figure 6-4**). As viewed on the screen, this is one structure on top of another. The structures must produce two separate echoes for each to be recognized as distinct. The smaller the wavelength of the transmitted US beam, the closer in position can be the two tiny distinguishable structures. The size of the wavelength can be decreased by increasing the frequency of the US beam. Increasing the frequency delivers greater axial resolution. However, a sacrifice is seen in penetration because of increased attenuation at higher frequencies.

LATERAL RESOLUTION AND FOCUS

Lateral resolution refers to the machine's ability to distinguish two separate structures that lie side-by-side in a plane perpendicular to the US beam (**Figure 6-5**). As seen on the screen, this is one structure to the side of another. Lateral resolution is a function of the beam width. The narrower the beam, greater is the ability to produce separate echoes for two adjacent objects. Focusing narrows an US beam at specific depths. The focal zone is the narrowest part of the hourglass-shaped US beam. Focusing can be achieved automatically by the machine or manually by the operator. One focal zone or multiple focal zones can be set at specific depths to narrow the beam for optimal lateral resolution.

TEMPORAL RESOLUTION

Continuous US scanning is actually a collection of still frames displayed rapidly over time. US machines produce numerous frames per second as the scan beam is transmitted over and over again through the tissue. The number of frames per second is known as the frame rate. The greater the frame rate, the better is the temporal resolution and the smoother the moving image appears. High frame rates are particularly important for scanning moving structures such as the heart. Employing additional functions such as Doppler scanning may limit the machine's ability to produce high frame rates.

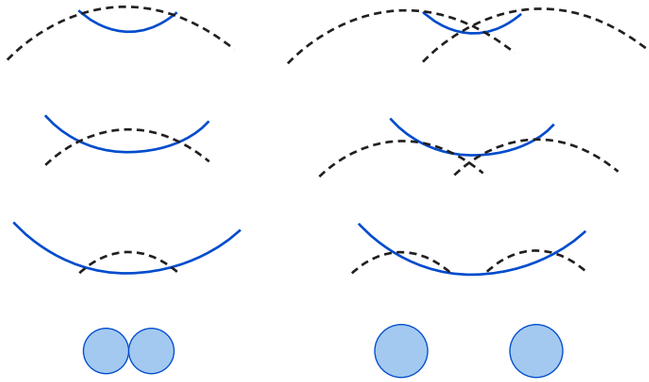


FIGURE 6-5. The lateral resolution of structures. Blue curves represent the transmitted US beam. Black curves represent the returning echoes. The structures must be able to produce separate echoes for the US machine to distinguish them. Focusing of the beam width improves lateral resolution.

ECHOES

Diagnostic US is predicated on sound waves not only transmitting through tissues but also reflecting back to the transducer (**Figure 6-6**). At an interface of two different tissues, the acoustic impedance of the tissues determines the proportion of transmission and reflection. Acoustic impedance is a measure of a tissue's resistance to sound penetration (i.e., density × propagation speed). Acoustic impedance is high in "hard" tissues such as bone, lower in visceral organs, and negligible in fluid. Reflection of an echo is a function of the difference between the acoustic impedance of two adjacent tissues. For instance, when a sound wave travels from soft tissue to bone, a significant proportion of the wave will reflect back to the transducer and a minimal amount will transmit through the bone for imaging of deeper structures.

When an US beam travels through parenchymal tissue without interfaces of homogenous acoustic impedance (e.g., liver), imaging is generated not by reflection but rather by scatter. Scattering occurs when a sound wave encounters particles smaller than its wavelength

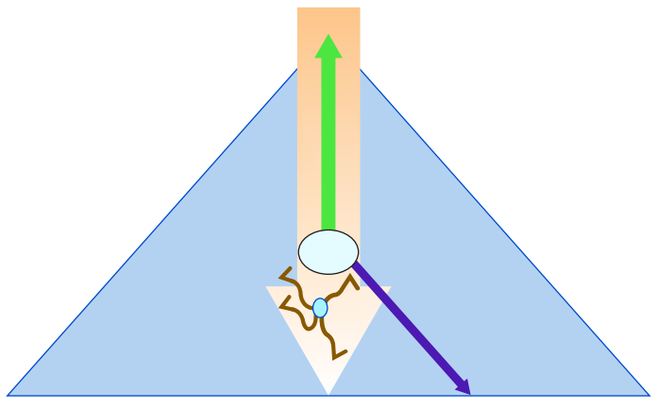


FIGURE 6-6. The echoes produced as the US beam penetrates tissue. The transmitted beam (orange) attenuates as it penetrates. A reflected echo (green) is produced at an interface. Refraction (purple) and scatter (brown) contribute to attenuation. Scatter produces the imaging within homogenous tissue.

or objects with rough and irregular surfaces. An analogous effect is seen when light is shone through fog. There is some transmission and reflection of the light, but it is the scattering of light that reveals the mass of fog.

In order for an US machine to generate an image, it must determine not only the intensity of the returning echo but also the location of the reflecting structure (i.e., reflector). With a given propagation speed (i.e., velocity of sound through tissue), the machine uses the travel time of the sound wave to calculate the distance the reflector sits from the transducer. The longer the travel time, the deeper is the reflector or structure.

ATTENUATION

As sound waves propagate through a medium, they start to lose amplitude and intensity, or attenuate (**Figure 6-6**). Attenuation is a result of absorption, reflection, refraction, and scattering of the US wave. Absorption is the conversion of sound to heat as the wave passes through tissue and accounts for the vast majority of attenuation. Absorption has very limited clinical effect for diagnostic US but is the basis of therapeutic US. Reflection is the "echoing" of the wave back to the transducer. Refraction and scattering are the redirection of waves upon encountering certain interfaces.

ULTRASOUND TRANSDUCERS (OR PROBES)

There are many types of transducers that vary in size, shape, construction, transmitted frequency, and function (**Table 6-2**). Transducers can be expensive and are at risk for damage in a busy ED. Transducers are often dropped and cords frequently run over by the wheels of the US machine. Creative solutions to prevent damage to dangling cords have been developed (**Figure 6-7**). The expense of replacing transducers should be factored into any warranty and maintenance considerations. Many US manufacturers offer multiport transducer connections that allow a number of transducers to be connected at once, each activated by the push of a button. Limiting the connecting and disconnecting of transducers decreases the chance of damaging fragile connector pins.

Transducers are the link between the US machine and the patient. The essential component of the transducer for generating an image is the piezoelectric element. The piezoelectric principle states that when an electrical voltage is exerted upon certain materials, a mechanical pressure or vibration will be produced. A transducer's piezoelectric elements, or crystals, when vibrated by an electrical voltage will generate a mechanical sound wave. The returning sound wave in turn vibrates the elements, creating an electrical voltage that carries image data back to the US machine.

FIGURE 6-7. The preservation technique for transducer cords. Segments of a coiled garden hose are sliced along their length and placed around the middle portion of a transducer cord. This prevents damage from the cart wheels running over dangling cords.

AUTOMATIC SCANNING

Transducers are constructed of numerous elements arranged along the width of the transducer. Unlike a flashlight that emits a single continuous beam of light, the US beam is a composite of pulsed firings of the elements. Automatic scanning is the electronic activation of the elements or arrays of crystals to generate a beam for a cross-sectional image. Two types of automatic scanning currently employed by transducers are sequenced array and phased array scanning.

SEQUENCED ARRAY SCANNING

Sequenced array scanning involves the sequential firing of groups of elements across the transducer assembly (**Figure 6-8**). One scan

TABLE 6-2 Common Types of US Transducers Used in the ED

Transducer type	Vascular	Abdominal	Endocavitary	Cardiac
Transducer shape				
Type of scanning	Linear sequenced	Curved sequenced	Microcurved sequenced	Phased array
Transducer frequency	High	Low	High	Low
Megahertz (MHz)	7–10	2–5	7–100	2–5

Photographs courtesy of Zonare Medical Systems.

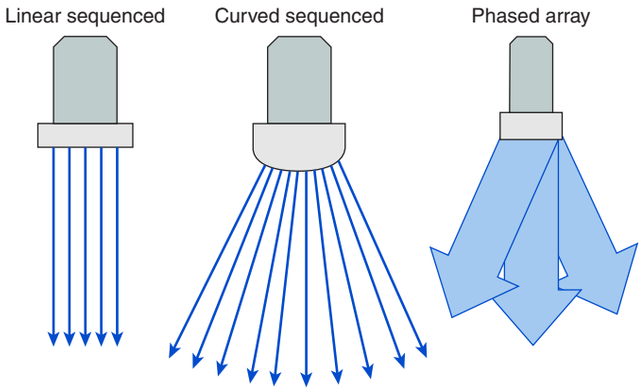


FIGURE 6-8. The basic types of transducers. Linear sequenced and curved sequenced array transducers transmit beams in a sequential fashion following the shape of the transducer head to produce rectangular and pie-shaped images, respectively. The beams of phased array transducers are steered by slight delays in the firing of the elements to produce a pie-shaped image.

line after another is generated along the width of the transducer. Linear transducers are flat-topped and produce a rectangular beam made up of parallel scan lines. Curved transducers transmit scan lines similarly, but following the curve of the transducer, and create a sector- or pie-shaped image. The elements are fired rapidly, and multiple frames of cross-sectional images are produced per second.

PHASED ARRAY SCANNING

Phased array transducers generally have a narrow flat-topped footprint. The tightly packed elements are electrically activated as a single unit, but with a slight time lag between each element, resulting in an angling of the pulse direction. The electronic activation and angling of each subsequent pulse are slightly changed. The resultant composite beam is a sector- or pie-shaped (**Figure 6-8**).

TRANSDUCER TYPES

The typical arsenal of transducers for emergency US includes a low-frequency curvilinear or phased array transducer, a high-frequency linear transducer, and an endocavitary transducer (**Table 6-2**). Numerous other types of transducers are manufactured. It is worthwhile to evaluate the options available before purchasing a transducer.

CONVEX TRANSDUCERS

Convex or curved transducers are low-frequency transducers that produce a sector-shaped image by sequenced array scanning. These are commonly used in EM. They are useful for most US examinations of the torso. The resolution of these transducers is inferior to that of linear transducers, but the greater penetration allows for imaging of relatively deep structures (e.g., aorta, gallbladder, liver, kidneys, heart). Microconvex transducers have a similar but smaller footprint with a tight curvature that allows for easier use between adjacent ribs.

PHASED ARRAY TRANSDUCERS

Phased array transducers are low-frequency transducers made for echocardiography and general imaging of the torso. These transducers have a small flat footprint ideal for maneuvering between the ribs and imaging the heart.

LINEAR TRANSDUCERS

Linear transducers are high-frequency transducers that are ideal for imaging superficial structures. They are generally wide, flat-topped, and produce a rectangular image by sequenced array scanning. These transducers are useful in the ED for imaging of blood vessels and guided line placement, skin abscesses, musculoskeletal pathology, pneumothoraces, ocular and testicular pathology, and a host of other superficial parts.

ENDOCAVITARY TRANSDUCERS

Endocavitary transducers are high-frequency transducers that have elements arranged in a tight curve at the end of a long handle. They are designed primarily for vaginal insertion and high-resolution imaging of the female reproductive organs. These transducers can be used for US-guided vascular access if no other high-frequency transducer is available. The small footprint and room afforded by the handle may even prove superior to a linear transducer in some instances. The transducer can be used in a patient's mouth for the evaluation of oral and pharyngeal pathology, such as a peritonsillar abscess.

FREQUENCY AND TISSUE HARMONIC IMAGING

Transducers may differ in inherent frequency. The frequency of each transducer can be adjusted within a narrow range of generally a few megahertz. Lowering the frequency may be beneficial if a greater depth is required to view pertinent structures. Conversely, increasing the frequency may improve visualization if a finer resolution is desired more than a deep penetration.

Tissue harmonic imaging is another means of enhancing US scanning. US echoes return from tissues in multiples of the transmitted frequency. The machine can filter out the transmitted frequency and focus on receiving the second harmonic frequency (i.e., twice the transmitted frequency). The returning harmonic beam is narrower than the beam of the fundamental frequency, and hence, lateral resolution is improved. Artifacts and distortions are also reduced with harmonic imaging.

COUPLING MEDIUM

Acoustic gel is the coupling medium used in diagnostic US. The gel obviates any air between the transducer and the patient to improve US beam transmission. Apply the gel across the footprint of the transducer or onto the patient's skin. A good rule of thumb is that too much gel is preferable to too little.

ULTRASOUND MACHINE INSTRUMENTATION

An US machine is made up of numerous components (**Figure 6-9**). The imaging system hardware consists of a beam former, signal processor, and image processor. The console allows the user to interface with and manage the imaging. The monitor screen provides for black and white as well as color viewing of the imaging. Transducers, single or multiple, have connecting ports onto the machine. Archiving tools, such as a printer or digital storage media, are available standard or as options. The following sections review the key functions that an EP should know when performing US examinations.

CONSOLE

The US console generally consists of a keyboard for data entry and numerous knobs, buttons, dials, and toggle switches for manipulating the images. Carefully read the user's manual or undergo a detailed operations briefing by the manufacturer's application

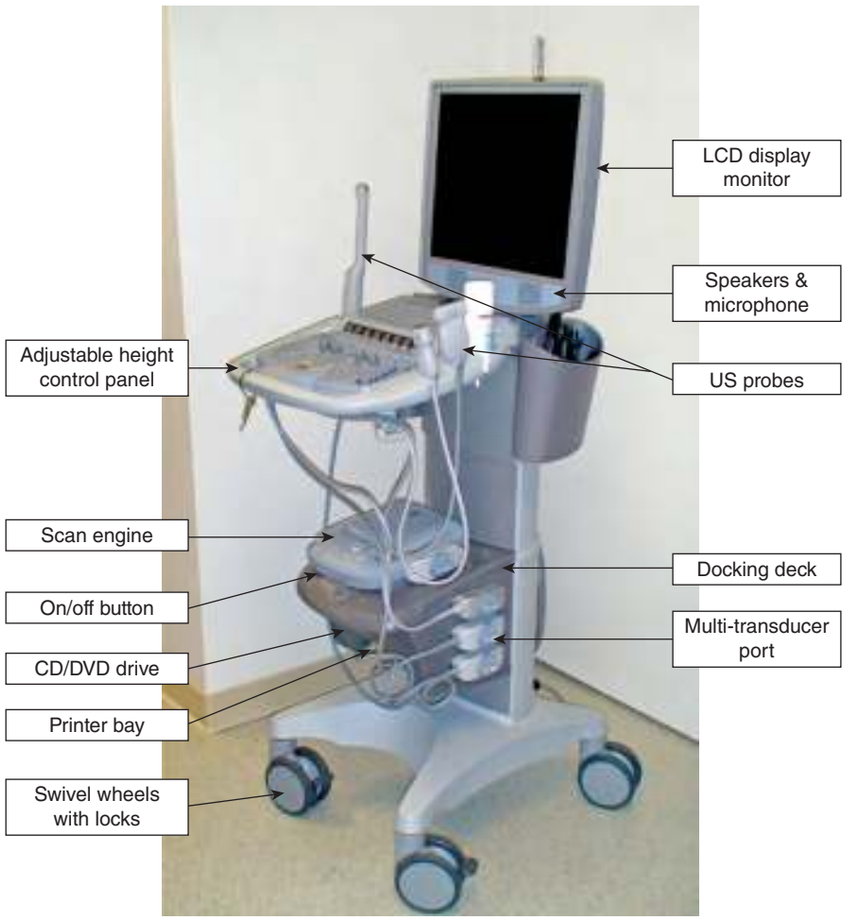


FIGURE 6-9. Components of a typical US machine.

specialist after purchasing a new machine. While there are many functions that are universal to US machines, they each have their proprietary functions.

There are numerous additional functions on the console. Magnify images with the "zoom" function. The "measure" button activates calipers that are moved with the trackpad or rollerball. This allows for precise measurements to the millimeter. Adjust the dynamic range to produce effects similar to modifying the contrast of a photograph. Other functions include gray-scale mapping, edge, and persistence for image alteration. A dual screen function displays two side-by-side imaging fields. Function keys can be set for easy access to commonly performed tasks.

GAIN

Gain is the US analog to the volume control on a radio. It alters the returning echo to amplify its intensity. Gain is measured in decibels and relates to the brightness of the image. Machines generally have a knob that can be dialed to adjust the gain, increasing or decreasing the overall brightness of the image (**Figure 6-10**).

Time gain compensation (TGC) allows for adjustments of gain at specific depths. Most TGC controls are comprised of a collection of sliding knobs arranged in a column (**Figure 6-11**). The knobs at the top of the column correspond to the near field, and those at the bottom correspond to the far field. The intensity of the beam is attenuated or dampened as an US beam penetrates to greater depths. TGC allows compensation for attenuation. For general abdominal imaging, arrange the sliding TGC knobs with a slope of increasing gain at greater depths. Use TGC to increase or decrease brightness at discreet depths of the image. Decreasing the gain can improve visualization to compensate for the artifactual brightness seen deep to a fluid-filled structure.

DEPTH

The depth of the field of view can be adjusted by a turn of a dial or toggle of a switch. Depth is conventionally measured in centimeters. Hash marks along the side of the image denote units of distance. Depth adjustments alter the penetration of the imaging beam and allow the user to appropriately "magnify" the organ or region of interest (**Figure 6-12**). The organ of interest will appear too large and its deepest portion may be cut off the screen if the depth is set too shallow (i.e., overmagnification). The organ of interest will appear too small in the near field with wasted space in the deeper regions if the depth is set too deep (i.e., undermagnification).

PATIENT DATA ENTRY

All machines allow the entry of demographic information about the patient and the US examination. The patient data entry screen presents numerous fields that can be filled by typing on the keyboard. These fields commonly include patient name, medical record number, date of birth, and a comments section, to name a few. Functions often available on this screen are examination and transducer selection.

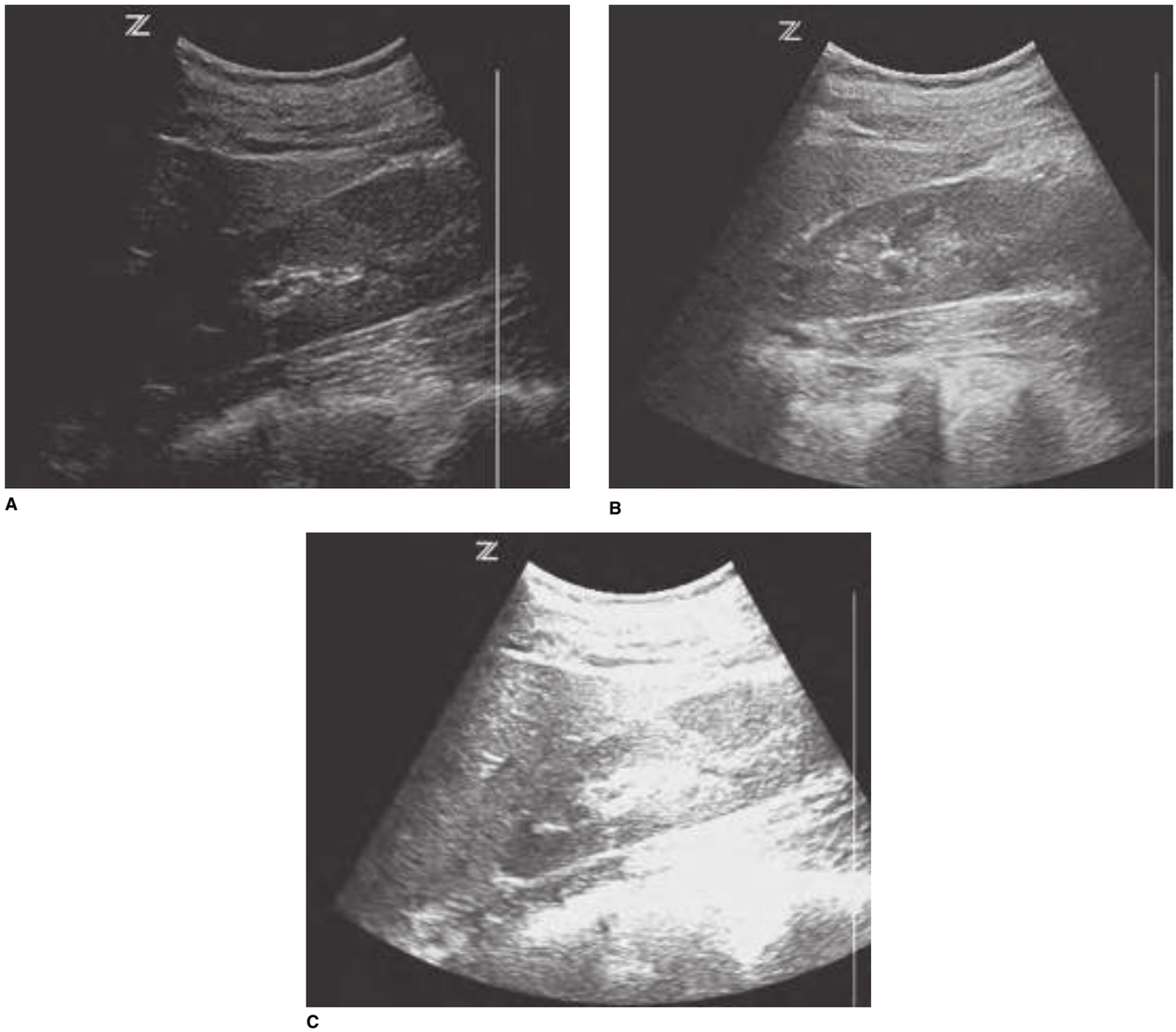


FIGURE 6-10. Gain adjustments brighten and darken the US image. These images of a kidney display a gain setting that is (*A*) too low, (*B*) appropriate, and (*C*) too high.

FREEZE AND CINE

Most machines allow both still images and video loops to be saved. Hitting the "freeze" button stops real-time continuous scanning. The most recent image is displayed on the screen with a few seconds of images (i.e., cine) available for review. Use the rollerball or trackpad to rewind through the cine memory of frames. This allows the viewing and subsequent saving of the most desired still image. A number of machines can allow the viewing of a loop of images as a video.

PRINCIPLES OF GENERAL ULTRASOUND IMAGING

DIMENSIONALITY

The US beam is flat, and the image displayed on the screen is a two-dimensional slice. Sound transmits through most tissues to allow for visualization of deep structures in the cross-sectional slice. Scanning the beam back and forth stacks multiple two-dimensional slices in a movie format and provides a sense of spatial orientation. Rotating the transducer 90° and scanning through the perpendicular plane helps to gain a three-dimensional comprehension of structures.

ORIENTATION

Orientation is entirely dependent on the how the transducer is placed on the patient. Each transducer has a marker that is a bump, ridge, or indentation. The marker correlates with an indicator on the screen to establish orientation. If the transducer is held with the marker aimed cephalad, the screen indicator is located on the left side of the screen (**Figure 6-13**). This results in the left side of the image representing the cephalad aspect and the right side the caudal aspect (**Figure 6-13**). The screen indicator is located on the left side for conventional radiology imaging. Most emergency US examinations require views with the transducer marker aimed to the patient's head and the patient's right. The image is displayed with the near field (i.e., closest to the transducer) at the top of the screen and the far field (i.e., farthest from the transducer) at the bottom of the screen.

FIGURE 6-11. Time gain compensation (TGC) controls on an US machine consist of a column of knobs that slide back and forth to adjust the gain at specific depths.

ECHOGENICITY

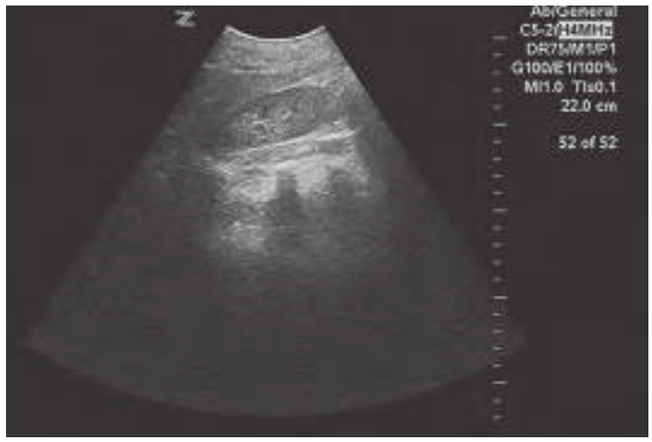
Fluid is anechoic. The US beam transmits through fluid without any reflected echoes. The echogenic silence is processed to generate black pixels on the screen. Highly reflective structures, such as the diaphragm and pericardium, are termed echogenic or hyperechoic. These structures will produce white imaging. Two structures of the same echo-texture are isoechoic. Less reflective tissue is hypoechoic and appears darker on the screen when comparing two tissues (**Figure 6-14**).

ARTIFACTS

Artifacts are images that do not accurately represent the anatomy in the reflected echo. Imaging artifacts are frequently encountered in ultrasonography. It is important to understand and expect certain artifacts to prevent misinterpretation of the images. Artifacts may also help to appreciate certain structures that would otherwise be less obvious.

SHADOWING

A highly reflective object allows very little of an US beam to transmit through it. Most of the beam is reflected back to the transducer, and the structure is represented as hyperechoic or white on the screen. The US machine interprets the lack of returning echoes deep to a structure that is hyperechoic as the lack of reflection, similar to that seen with fluid. This is seen as a black shadow on the screen (**Figure 6-15**). While shadowing from ribs may obscure deep anatomy, the shadows of gallstones aid in their identification.



A

B

C

FIGURE 6-12. Depth adjustments increase and decrease the penetration of the US beam. These images of a kidney display a depth setting that is: (*A*) too deep at 22 cm, (*B*) appropriate at 10 cm, and (*C*) too shallow at 6 cm.

REVERBERATION

Highly reflective interfaces may result in multiple reflections. Bouncing of the sound beam between the reflector and the transducer can create false echoes known as reverberation (**Figure 6-16**). It can be seen when the sound beam encounters two closely spaced

A

B

FIGURE 6-13. The orientation marker (white arrow) on the transducer corresponds with the indicator on the screen (red arrow).

interfaces, such as the two walls of a needle or the visceral and parietal pleura. The echo reflects back and forth between the interfaces, but with some transmission of the beam back to the transducer each time. These returning echoes generate artifactual reflections on the screen termed reverberation or comet tails (**Figure 6-17**).

MIRROR IMAGE

Mirror image artifacts are generated when an object is located in front of a very strong reflector. Structures seen on the near side of a bright reflector are displayed on the other side of the reflector as well. This is common with the diaphragm. Triangulation of the beam path delays the return time of the echo to mirror a second reflector (**Figure 6-18**). Mirror-image artifact can help to exclude a hemothorax by visualizing the liver or spleen cephalad to the diaphragm (**Figure 6-19**).

ENHANCEMENT

Attenuation does not occur when a sound beam passes through a fluid-filled structure. The beam's high intensity is maintained

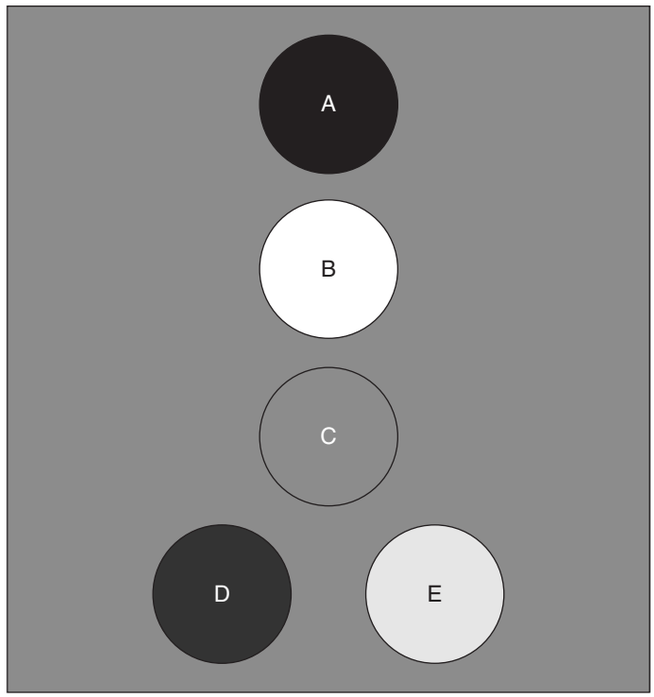


FIGURE 6-14. Echogenicity of structures are represented by their brightness on the screen. ***A.*** Anechoic fluid is black. ***B.*** Echogenic, or highly reflective, compared with adjacent tissues. ***C.*** Isoechoic, or same echo-texture, as adjacent tissue. ***D.*** Hypoechoic, or less echoic (i.e., darker) than adjacent tissues. ***E.*** Hyperechoic, or more echoic than adjacent tissues.

through the fluid. A strong echo is generated off the posterior wall of the fluid-filled structure. The artifact seen on the screen is a very hyperechoic region deep to the fluid-filled structure that may obscure the actual anatomy (**Figure 6-20**).

MISCELLANEOUS ARTIFACTS

A number of additional artifacts can interfere with imaging. Side lobes are weak beams emitted lateral to the central axis of the scanning beam that may produce false echoes. Section thickness artifact is a result of interfering information from the outside of the flat slice

FIGURE 6-15. Echogenic gallstones are present in the gallbladder. Shadowing is seen deep to the gallstones.

FIGURE 6-16. The two walls (white arrows) of a cylindrical bullet are seen superficially. Reverberation artifact produces an additional false echo (red arrow). Shadowing is seen deep to the bullet.

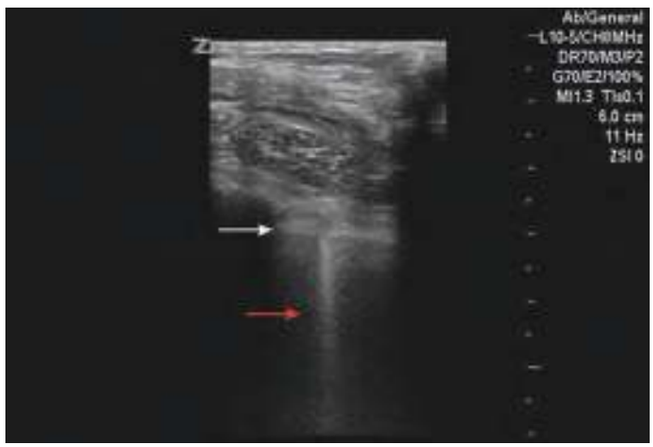


FIGURE 6-17. Each back and forth reflection between the visceral and parietal pleura (white arrow) results in an echo transmitted back to the transducer. Multiple echoes returning one after another produce echogenic artifacts, each deeper than the next. The artifact resembles a comet tail (red arrow).

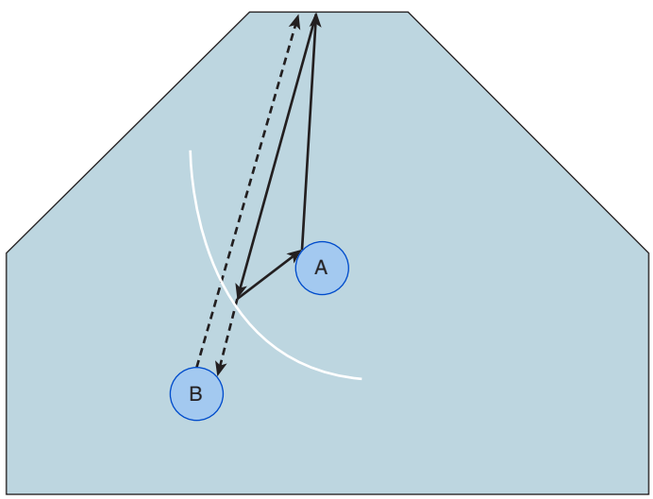


FIGURE 6-18. Bright reflectors like the diaphragm (white) can generate mirror-image artifacts. The initial beam (solid arrow) reflects off the diaphragm and then an anatomic structure (***A***) before returning to the transducer. The machine processes the delay as a signal from a deeper structure along the initial scan line (dashed arrow) and generates an artifact (***B***) on the screen.

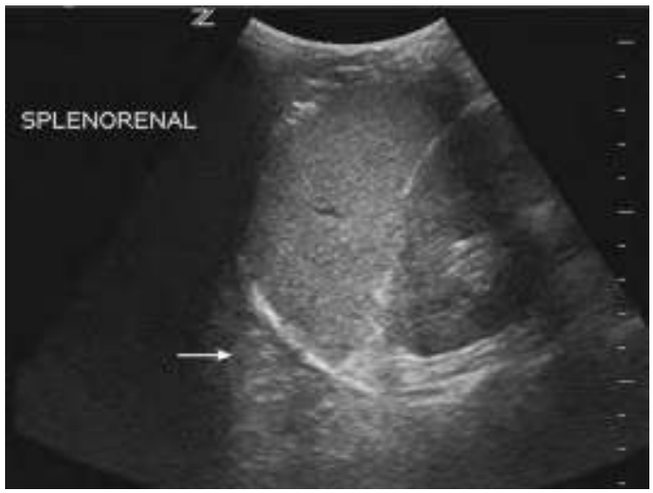


FIGURE 6-19. A mirror-image artifact (white arrow) of the spleen is seen cephalad to the diaphragm. This is distinct from fluid that appears black.

of the scanning beam. Refraction of the beam tangentially off an interface may produce a double image of a structure lateral to the original. Shadowing in the narrow angle of refraction may be seen deep to the interface.

ULTRASOUND MODES

BRIGHTNESS MODE

Brightness mode is commonly referred to as B-Mode. This is the basic scanning mode for US. It displays the standard two-dimensional gray-scale image.

MOTION MODES

Motion mode, or M-Mode, is used to assess moving structures. Activating M-Mode produces a vertical line on the image (**Figure 6-21**). The line can be moved left or right with the rollerball

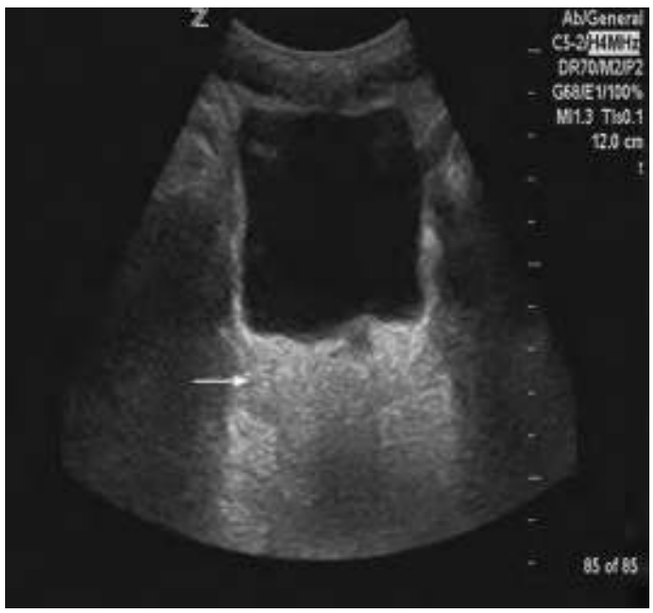


FIGURE 6-20. High-intensity echo returns from the posterior wall of the bladder and results in a hyperechoic region of enhancement artifact (white arrow).

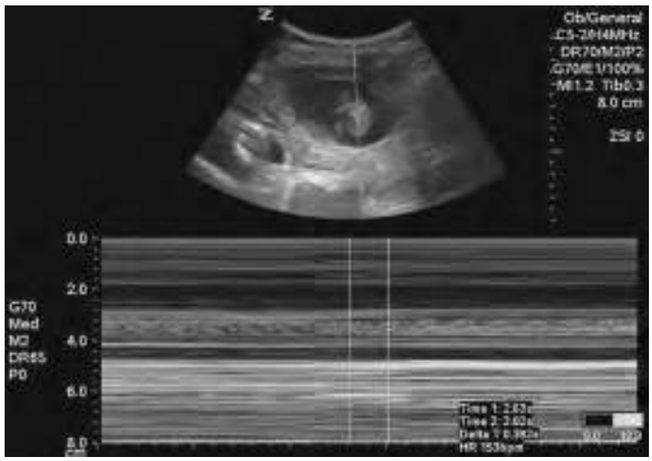


FIGURE 6-21. The M-Mode. The B-Mode image is displayed at the top of the screen. A green vertical line is seen over the B-Mode image. The area under the green line is displayed over time at the bottom of the screen. The fetal heartbeat is seen at a depth of 3.6 cm and measured at 153 beats per minute.

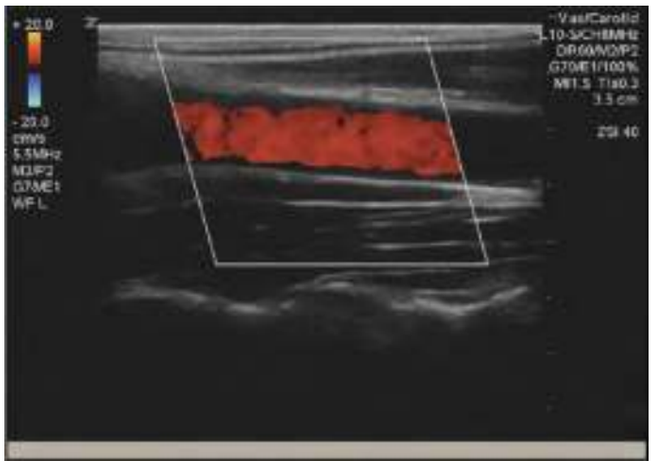


FIGURE 6-22. The color Doppler box, or region of interest (ROI), placed over the carotid artery reveals flow in the direction of the transducer (red).

or trackpad. The US beam penetrating the tissue along that single line is displayed in a continuous graphical manner on the bottom of the screen (**Figure 6-21**). The *x*-axis of the display is depth, and the *y*-axis is time. There will be no variation in the M-Mode display if the line is set upon immobile tissue. Movement along the line, such as a beating heart, allows measurements and rate determinations (**Figure 6-21**).

DOPPLER MODE

Doppler scanning allows the assessment of the presence, direction, speed, and character of blood flow and tissue movement. Doppler takes full advantage of the real-time dynamic nature of US. If an object is moving toward or away from the transducer, the frequency of the reflecting echo will be higher or lower, respectively, than the transmitted frequency. The Doppler shift is the difference between the transmitted and reflected frequencies. The US machine can calculate velocity using the Doppler shift. Turbulence, or the variance in velocity of multiple objects in flow, can be assessed. The complexities of Doppler physics and operations are beyond the scope of this chapter.

EPs can use Doppler in the assessment of deep venous thromboses, testicular or ovarian torsion, compromised vascular flow in the extremities, and inflamed or hypervascular tissues; to differentiate vessels from nonvascular structures for diagnostic and procedural purposes; and for advanced echocardiography.

COLOR DOPPLER MODE

A "C" button on the machine's console usually signifies color Doppler mode. Activating color Doppler produces a box, or region of interest (ROI), overlying the image. The ROI can be maneuvered over the desired area. The size of the box can be manipulated to be larger or smaller depending on the study being performed. Flow is displayed in a color schematic within the ROI (**Figure 6-22**). Flow toward the transducer is typically red. Flow directed away from the transducer is typically blue. Gradients of red and blue signify the speed of the flow.

PULSED WAVE DOPPLER MODE

Pulsed wave Doppler provides a quantitative assessment of flow velocities. Activating pulsed wave Doppler splits the screen, typically placing the image at the top of the screen and a dynamic graph of flow velocities at the bottom (**Figure 6-23**). A small "gate" can be positioned over a specific vascular area to measure flow. The size of the gate can be adjusted, allowing very narrow regions to be accurately assessed. The dynamic Doppler graph displays time on the *x*-axis and velocity on the *y*-axis. Movement toward the transducer is depicted as a positive velocity deflection. Movement away from the transducer is depicted as a negative deflection.

POWER DOPPLER MODE

Power Doppler is another method of displaying blood flow or tissue motion (**Figure 6-24**). It uses the same ROI box as color Doppler, but with only one shade of color. Power Doppler is very sensitive in identifying the presence of flow but does not reveal its direction. It is advantageous in identifying low flow states and subtle tissue motion and imaging small or deep vessels.

IMAGE ARCHIVING

There are numerous ways to archive US images. Still images can be sent to printers for hard copy archiving. Thermal printers are relatively small and can be attached to the US cart. The gray-scale

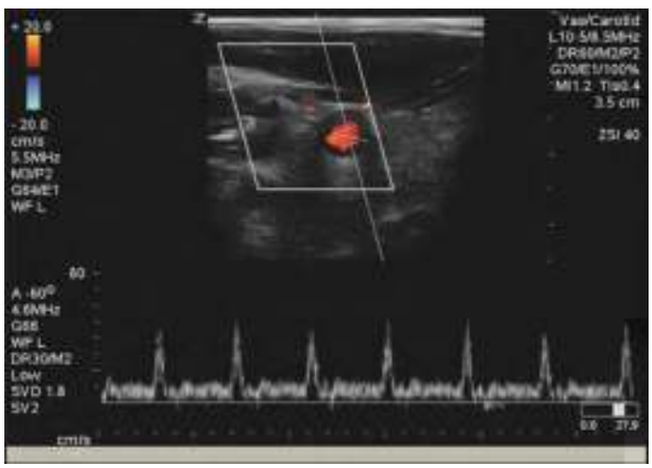
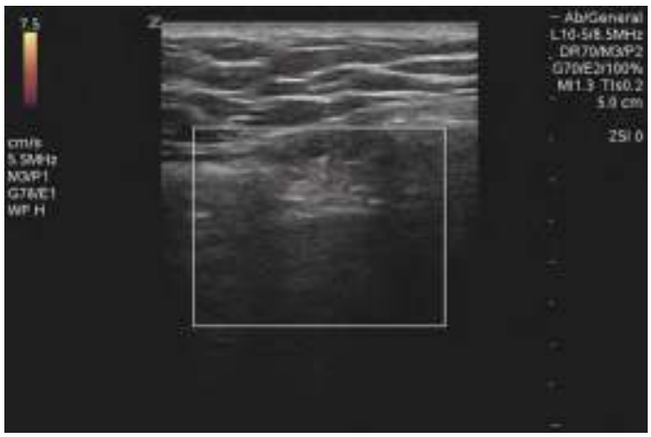


FIGURE 6-23. The pulsed wave Doppler mode. A Doppler gate is placed over the center of the carotid artery. Flow is displayed in active graphical format at the bottom of the screen. Positive deflections indicate systolic flow in the direction of the transducer.

A



B

FIGURE 6-24. The power Doppler mode. ***A.*** Normal lung slide at the pleural line. The tissue movement generates a power Doppler signal. ***B.*** No lung slide or tissue motion is seen in the setting of a pneumothorax.

printouts are fine reproductions of the image but are at risk for degradation over time.

Digital archiving allows users to save both still images and videos with a far reduced need for physical storage space. Examinations can be saved to hard drives built into the US machine or to peripheral devices. Machines today offer many options for peripheral storage including compact discs, digital video discs, external drives with universal serial bus connections, and magneto-optical disks.

Specific formats for digital archiving of medical imaging have been developed, in particular the digital information and communication in medicine (DICOM) format. Most US machines today have the ability to save images in the DICOM file format. Picture archiving and communication system (PACS) has been developed for viewing medical imaging in DICOM and other formats. These systems are employed by Radiology Departments for viewing US images, as well as computed tomography, magnetic resonance imaging, plain radiographs, and other imaging modalities. Advantages to saving DICOM images on PACS include improved organization, reliability, and the ability to transmit remotely.

ULTRASOUND-ASSISTED PROCEDURES

US-guided procedures are often performed in EM. A general discussion is provided in Chapter 7. More specifics on US-assisted procedures are included with each specific procedure. Sometimes, the EP does not have enough hands to hold the transducer and perform the procedure. Assistants are not always available to hold the transducer in position. A novel device is the Echosupport (Vygon SA, France).[15] It is a reusable, malleable, and sterilizable device (**Figure 6-25**). One end attaches to the patient's bed by a screw clamp. The other end securely holds the US transducer in position.

A

B

FIGURE 6-25. The Echosupport device. ***A.*** The device. (Courtesy of Vygon SA, France) ***B.*** Photo of the device being used on a patient. (Used with permission from reference 15.)

SUMMARY

US is fast becoming a commonly used modality in EM. All EPs should become familiar with US as it will soon become the standard of care for patient evaluation, patient management, and

procedural guidance. Take the time to investigate the various options available for machines, transducers, and accessories. A thorough understanding of the functioning and features of an US machine is essential before making clinical decisions. Training is readily available through US fellowships, continuing medical education courses, and US manufacturers.

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7 Ultrasound Assisted Procedures

Jehangir Meer and Brian Euerle

INTRODUCTION

Emergency Physicians commonly perform invasive procedures. These procedures have traditionally been taught using surface landmarks, with the assumption that anatomy is reliably similar from patient to patient. The increasing use of ultrasound (US) to assist in procedural guidance has demonstrated this is not the case. Using US to assist with procedures has numerous benefits (**Table 8-1**). It is safer for patients due to reducing complications. US improves patient comfort and satisfaction primarily because fewer attempts are required. The use of US usually decreases the duration of the procedure.

TABLE 7-1 Common ED Procedures that Use US Assistance or Guidance

Body region	Procedures
Abdomen and pelvis	Paracentesis
	Suprapubic bladder aspiration
	Suprapubic bladder catheterization
Airway	Endotracheal intubation
Bones and joints	Arthrocentesis
	Closed fracture reduction
	Fracture identification
	Joint dislocation
	Joint reduction
Chest	Pericardiocentesis
	Thoracentesis
	Cardiac pacing (transvenous and transthoracic)
Ear, nose, and throat	Peritonsillar abscess incision and drainage
Nervous system	Peripheral nerve blocks
	Lumbar puncture
Soft tissue and musculoskeletal	Abscess identification
	Abscess incision and drainage
	Foreign body identification and removal
Vascular	Arterial line placement
	Central venous access
	Peripheral venous access

The sonographer must make several decisions prior to beginning a procedure using US. Will the procedure be performed under real-time US guidance, or will US be used only to map the anatomy? Will one person (the sonographer) or two people (the sonographer and an assistant) be necessary? Which US transducer is the most appropriate for the procedure? Should the instrument or needle be imaged using the in-plane technique or the out-of-plane technique? Are needle guides necessary? What is the ideal location of the US machine in relation to the sonographer and patient? These general questions must be kept in mind when using US for invasive procedures and are discussed below.

This chapter reviews basic information regarding the use of US to assist or guide procedures in the Emergency Department (ED). US can assist in many commonly performed ED procedures (**Table 7-1**). The specific US technique for a procedure is described in the chapter for that procedure.

GUIDANCE VERSUS MAPPING

US can be used in one of two ways for procedural assistance: the dynamic technique or the static technique. The dynamic technique is also known as US guidance. **The sonographer uses US guidance in real time during the procedure to survey the anatomy, to confirm a diagnosis, and to visualize the needle or instrument as it enters tissue and reaches the target.** The static technique is also known as US mapping. **The sonographer uses US mapping prior to starting the procedure to map the local anatomy, to confirm a diagnosis, and to mark the site of needle entry. The US transducer is then put away and the procedure performed in the traditional fashion without real-time US.**

The decision between US guidance versus US mapping is influenced primarily by the degree of inherent danger of the procedure. Perform procedures that carry a higher risk or have greater technical difficulty (e.g., central venous access, pericardiocentesis, or foreign body removal) under US guidance. Perform lower risk procedures

(e.g., thoracentesis, paracentesis, or abscess incision and drainage) with US mapping. Another factor is sonographer experience. The experienced sonographer is more technically adept and comfortable doing procedures under real-time US guidance. Novice sonographers who do not have as much psychomotor training and experience may find it easier to use US mapping.

ONE-PERSON VERSUS TWO-PERSON TECHNIQUE

The decision to perform a procedure with one or two people is based on the sonographer's experience and the availability of an assistant. Sonographers with more experience often prefer to guide the US transducer and manipulate the instrument themselves. Their experience and expertise allows them to work with a higher efficiency without an assistant. Assistance during the procedure may not be an option during a busy ED shift, in an ED with single-physician coverage, or in an ED with limited personnel. The sonographer holds the US transducer with the nondominant hand and guides the needle or instrument with the dominant hand for one-person US guidance. This requires a degree of hand-and-eye coordination to maintain continuous alignment of the needle with the US transducer.

Novice sonographers may find it less daunting to use the two-person technique and work with an assistant. The assistant holds the US transducer while the sonographer guides the needle or instrument. The assistant's responsibility is to maintain alignment of the US transducer with the needle during the procedure. The sonographer must maintain good communication with the assistant throughout the procedure to maintain sight of both the needle tip and the target organ on the US monitor screen. This coordination is challenging when using a two-person technique. This is why most US-guided procedures are done by one person holding both the transducer and the needle.

TYPES OF TRANSDUCERS

The following general principle will be helpful in determining which US transducer is the most appropriate for a given procedure. **The higher the frequency of the US transducer, the better the resolution of the structures visualized, but the shallower the maximum depth of view.** In other words, use a high-frequency US transducer for superficial structures and a low-frequency US transducer for deep structures. Low-frequency US transducers include the curvilinear transducer and the phased-array transducer (**Table 6-2**). High-frequency US transducers include the linear transducer and the endocavitary transducer (**Table 6-2**). More than one transducer may be required for some procedures. Another factor in selecting the US transducer is its footprint or surface area. A larger footprint is recommended to get a larger scan image unless specific anatomic barriers (e.g., ribs) dictate a smaller footprint. The types of US transducers recommended for some specific procedures are listed in **Table 7-2**.

TABLE 7-2 Recommendations for US Transducer Type to Use for Specific Procedures

Transducer type	Procedure
Curvilinear	Endotracheal intubation
	Lumbar puncture
	Paracentesis
	Pericardiocentesis
	Suprapubic bladder aspiration
	Suprapubic bladder catheterization
	Transcutaneous cardiac pacing
	Transvenous cardiac pacing
Endocavitary	Peritonsillar abscess incision and drainage
Linear array	Abscess incision and drainage
	Endotracheal intubation
	Fracture identification
	Fracture reduction
	Lumbar puncture
	Peripheral nerve blocks
Phased array	Pericardiocentesis
	Thoracentesis
	Transvenous pacing

ORIENTATION OF THE NEEDLE AND THE TRANSDUCER

Two orientations or approaches are used for US guidance during procedures. The in-plane approach refers to placing the long axis of the needle in line with the long axis of the US beam (**Figure 7-1**). The out-of-plane approach refers to placing the long axis of the needle 90° to the long axis of the US beam (**Figure 7-2**). **It is paramount for the sonographer to keep sight of the needle regardless of which approach is used during the procedure.** It is helpful to have the US transducer indicator facing the same side as the marker on the top of the US monitor screen to maintain left-to-right alignment.

The in-plane approach allows the entire length of the needle to be visualized as it approaches the target (**Figure 7-3**). Depth perception is better with this approach. This is a more intuitive approach for some ultrasonographers. The main disadvantage of the in-plane approach is the poor lateral resolution. A needle located to the side of a structure may appear in the same plane as the structure when it is not. **This approach requires precise alignment of the US transducer with the needle. Otherwise, the sonographer can lose sight of the needle.** One procedure best performed with the in-plane approach is a peripheral nerve block.

The out-of-plane approach requires the sonographer to center the target of interest on the US monitor screen. The long axis of the

FIGURE 7-1. The in-plane approach of the needle to the US transducer.

FIGURE 7-2. The out-of-plane approach of the needle to the US transducer.

needle is positioned 90° to the long axis of the US beam, at the center of the long axis of the US transducer (**Figure 7-2**). The short axis view of the needle is visible on the US monitor screen and results in better lateral resolution (**Figure 7-4**). One disadvantage of this approach is the challenge to maintain sight of the needle tip. The sonographer must move the US transducer forward along with the needle tip as it is advanced. **The depth of the needle tip can be misjudged if one is not careful.** This can sometimes result in the inadvertent puncture of the anterior and/or posterior wall of the target structure or another adjacent structure.

FIGURE 7-3. In-plane US view of the needle (arrows).

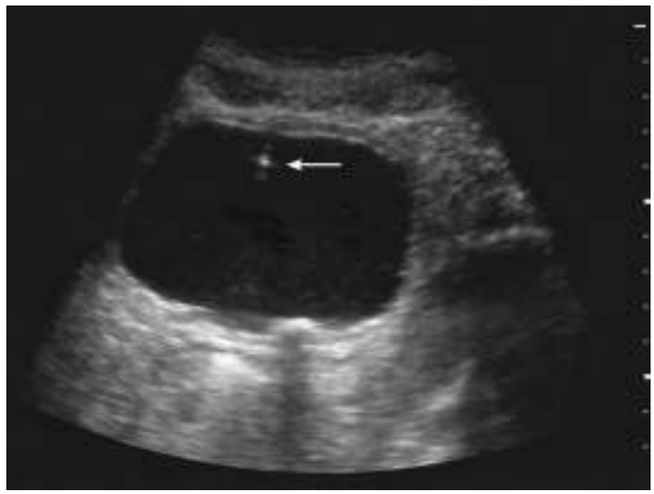

FIGURE 7-4. Out-of-plane US view of the needle (arrow).

MECHANICAL NEEDLE GUIDES

Needle guides are attachments to the US transducer that keep the needle in a predictable path during the procedure. They are often used by Radiologists for aspiration or biopsy of deep structures. The benefit of using a needle guide is that less hand-to-eye coordination is required to keep the needle aligned with the US transducer during the procedure. The disadvantage of a needle guide is that the path angle is fixed and cannot be changed in the middle of the procedure. A needle guide is not necessary for most of the procedures performed in the ED. It may be disadvantageous since it does not allow "on-the-fly" corrections of the needle path.

GENERAL TIPS FOR GUIDANCE

Always ensure there is a direct line of sight from the procedural field to the US machine (Figure 7-5). The sonographer should not need to turn their head to the side during the procedure to view the US monitor screen. This will greatly improve the sonographer's comfort during the procedure and increase the likelihood of success. Having to turn the head back and forth can result in movement of the needle, movement of the US transducer, an unsuccessful procedure, puncture of incorrect structures, and potential morbidity.

FIGURE 7-5. An example of a good setup with the US machine in relation to the sonographer and the patient.

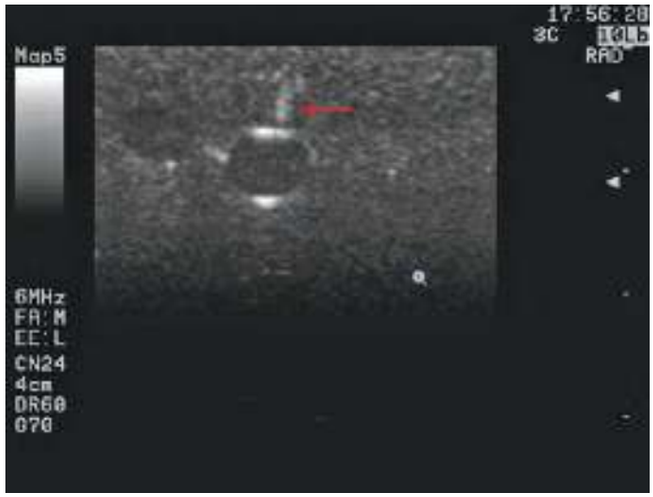

FIGURE 7-6. Ring-down artifact (arrow) showing the hyperechoic vertical line originating from the needle tip.

Hold the US transducer comfortably in the nondominant hand using a pencil-like grip and maintaining light contact with the patient. It is best to use short controlled movements to maintain visualization when manipulating a needle or other instrument under US guidance. Performing the procedure while seated can also help achieve the fine movements that are required.

Misalignment of the US transducer and the needle is the most common reason for the needle tip not being visualized during the procedure. **Stop advancing the needle if it is not visible on the US monitor screen.** Reposition the US transducer by dragging or fanning it back and forth over the area of the needle until the needle can be seen. Gently rocking or bouncing the needle within the soft tissue can sometimes assist in determining the location of its tip.

Indirect clues can help determine the location of the needle tip if it cannot be seen directly. These include the ring-down artifact (**Figure 7-6**) and the needle shadow artifact (**Figure 7-7**). The ring-down artifact is a bright hyperechoic streak caused by reverberation of the US beam from the highly reflective interface of the needle. The shadow artifact is a hypoechoic line below the needle caused by the needle blocking the US beam. Follow either artifact until the needle can be visualized on the US monitor screen. Continue to follow the needle until its tip is found.

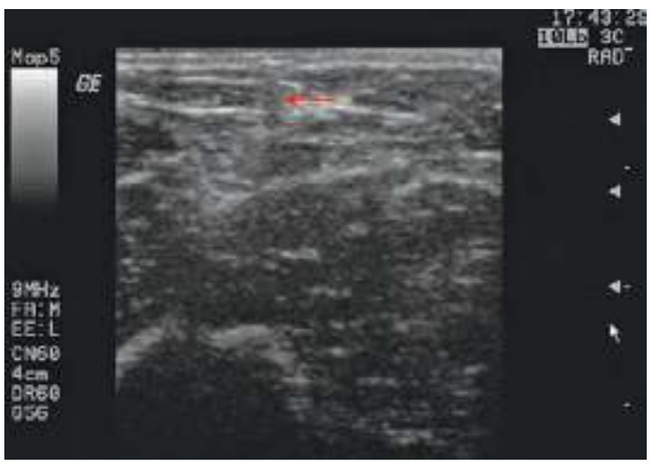

FIGURE 7-7. Needle shadow artifact (arrow) showing the hypoechoic vertical line originating from the needle tip.

Other factors can affect the ability to visualize the needle. Consider these when preparing to perform a procedure. Use the appropriate transducer and depth of field for the procedure. Smaller gauge needles produce smaller artifacts and are more difficult to visualize, especially in cross-section. It is easier to visualize needles perpendicular, or out-of-plane, to the US beam. Needles that are inserted at a shallow angle are typically easier to visualize than those at a steep angle. Consider using extra-reflective needles specific for ultrasonography. Their cost is hard to justify but may be acceptable for teaching. Normal needles are appropriately reflective, and it is difficult to justify these extra-reflective needles clinically. The beveled tip of the needle is easier to visualize than the shaft. The irregular surface of the bevel will reflect the US beam better than the shaft. Insert and advance the needle with a slight "to-and-fro" motion to make it easier to follow the needle path.

SUMMARY

The use of US assistance for procedures is an important skill set in the armamentarium of the Emergency Physician. It allows potentially dangerous procedures (e.g., incision and drainage of a peritonsillar abscess) to be performed in a safer manner due to visualization of the surrounding anatomy. It allows rapid confirmation of a diagnosis at the bedside and results in faster therapeutic interventions (e.g., pericardiocentesis). It usually results in greater patient satisfaction because of fewer attempts being required to successfully complete the procedure. ED ultrasonography can truly be a lifesaving modality when used by trained Emergency Physicians.

ACKNOWLEDGEMENTS

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8 Trauma Ultrasound: The FAST and EFAST Scans

Wes Zeger

INTRODUCTION

Evaluation of blunt trauma patients with ultrasound (US) has been described for over 30 years.[1-3] Its use in the United States in the early assessment of blunt abdominal trauma patients rapidly increased in the 1990s.[1] It is currently taught as an adjunct to the secondary survey in the Advanced Trauma Life Support (ATLS) course and in residencies.[1,4] US evaluation of the trauma patient decreases the costs in blunt trauma patients, resource utilization, and time to operative care.[5,6] There are many advantages to US (**Table 8-1**).[7] The focused assessment with sonography in trauma examination is known as the FAST examination and can be completed within 5 minutes.[1] It has replaced the need for a diagnostic peritoneal lavage (Chapter 84) in the initial assessment of all but a few trauma patients.[8,9] This chapter reviews the technique and interpretation of the FAST examination and the extended FAST, or EFAST, examination.

ANATOMY AND PATHOPHYSIOLOGY

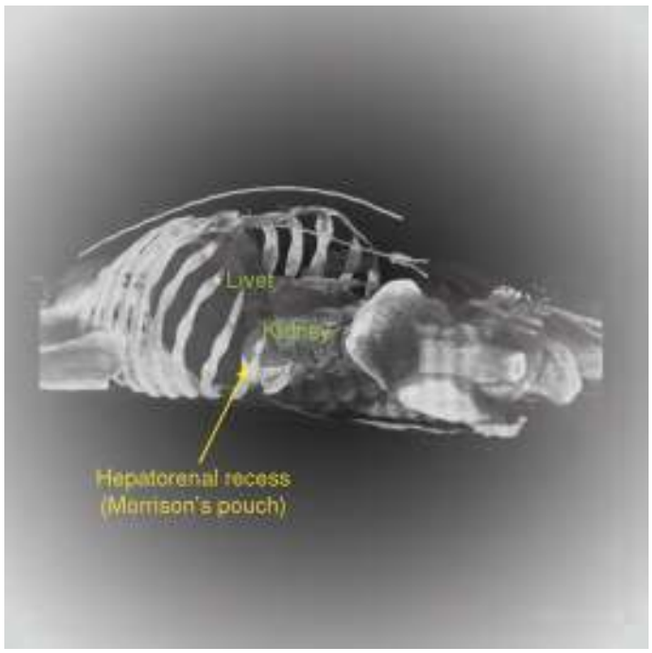
The FAST examination evaluates four anatomic areas or potential spaces for the presence or absence of fluid. These include the hepatorenal recess or Morrison's pouch, the splenorenal recess, the rectovesical or rectouterine space, and the pericardial space. **It assumes fluid present represents blood in the setting of trauma.** The presence of ascites, bowel fluid, and urine can appear similar using US. These four spaces represent the most dependent areas in the supine patient (**Figure 8-1**). The volume of fluid accumulation required for visualization by US ranges from 250 to 620 mL.[10,11] More experienced sonographers can visualize volumes closer to 250 mL.[10]

INDICATIONS

The FAST examination is performed after the primary survey. It can be performed in conjunction with ongoing resuscitative efforts. It is indicated when evaluating for the presence of intraperitoneal or pericardial blood in the setting of acute thoracoabdominal trauma. It is also useful in determining resource allocation in the setting of multipatient trauma scenarios.[1]

TABLE 8-1 Advantages of Using US

Completed quickly
Decreased cost
Decreased diagnosis time
Decreased resource utilization
Decreased time to operative care
Diagnose degree of fluid in abdomen
Diagnose degree of fluid in pericardium
Diagnose fluid in the abdomen
Diagnose degree of fluid in pericardium
Does not use intravenous contrast
High specificity for fluid
Integrated into primary and secondary survey
Lacks ionizing radiation
No need to move patient
Noninvasive
Performed at bedside
Reduces computed tomography scanning
Reduces diagnostic peritoneal lavage
Safe in children
Safe in pregnancy
Serial repeated exams



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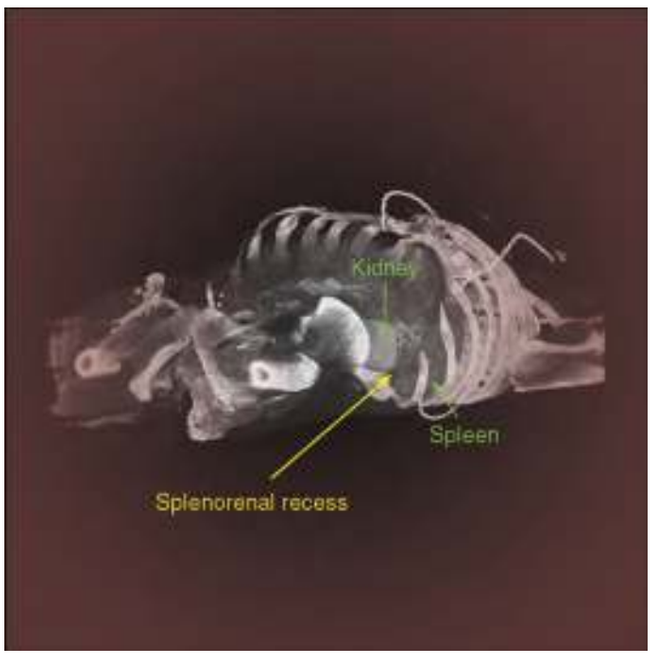

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FIGURE 8-1. The posterior reflection of the peritoneum is where blood initially layers in the supine patient. The hepatorenal (*A*) and splenorenal (*B*) recesses represent the posterior peritoneal reflections between the inferior pole of the kidney and the liver or spleen, respectively.

BLUNT THORACOABDOMINAL TRAUMA

The most studied use of the FAST examination has been in adult patients with blunt abdominal trauma. **The early diagnosis of hemoperitoneum and/or hemopericardium in the setting of**

blunt trauma is critical. The sensitivity of 78% to 90% and the specificity of 98% to 100% for the presence of hemoperitoneum varies depending on the comparison "gold standard."[9,12-15] It has been reported to approach 100% sensitivity and 100% specificity in hypotensive patients.[8,16] One study reported a much lower sensitivity of 42% and specificity of 98%.[17] The difference between these studies was how computed tomography (CT) was used as the gold standard.

Pediatric blunt trauma patients have been less well studied.[18-20] However, the FAST examination has shown a similar sensitivity and specificity.[21-24] Its sensitivity approached 90% but its specificity dropped when used to predict the need for operative intervention in pediatric trauma patients.[25] Current guidelines recommend evaluation of the pericardial space in adult and pediatric patients with blunt thoracoabdominal trauma.[26-28] This view of the pericardial space may be more cost effective in hypotensive patients.[29]

PENETRATING THORACOABDOMINAL TRAUMA

Decreased mortality using US in the assessment of trauma was first described in relation to penetrating chest trauma.[30] Its application in patients with penetrating abdominal trauma is less clear. The sensitivity ranges from 48% to 100%, but its specificity remains high at 98% to 100%.[15,31-34] Assessment of the pericardial space can be helpful in guiding operative intervention in the unstable patient.

CONTRAINDICATIONS

US is a noninvasive diagnostic modality. The FAST examination is contraindicated in the setting of trauma only if it would delay and negatively impact a clinically obvious need for emergent operative intervention. Do not perform and rely on the FAST examination if not properly trained or not supervised by those who have been adequately trained.[26]

EQUIPMENT

- US machine
- US gel
- US transducer, linear or phased array, 2.0 to 5.0 MHz
- US transducer cover or glove

A complete discussion of US equipment is beyond the scope of this chapter. Decisions regarding machines and transducers depend on the user, cost, and intended applications. The FAST examination can be performed with a good-quality, 2.0 to 5.0 MHz general abdominal transducer.[22] Transducers with smaller footprints allow for easier viewing between the ribs. Transducers of 5.0 to 7.0 MHz may better visualize children and small adults.

The US transducer is generally used directly against the patient's skin, with gel between them. Place the transducer in a cover or glove to prevent contamination of the transducer if the patient's skin is covered with blood, feces, urine, vomit, or other substances (Chapter 5). Place US gel in the transducer cover or glove before inserting the transducer. Squeeze out any air in the space between the tip of the transducer and the cover or glove.

PATIENT PREPARATION

Little to no preparation is required to perform the FAST examination. An informed consent is not needed. Tell the patient exactly what will and is happening to prevent them from moving. The US gel is often at room temperature and cold when it touches the skin. Wipe any blood, debris, dirt, and liquids from the patient's skin in the areas to be scanned. Place the US transducer in a transducer cover or glove if the patient's skin is contaminated with blood, dirt, urine, vomit, or other substances that may contaminate or damage the transducer.

TECHNIQUE

Performance of any US examination assumes the transducer marker orientation is generally cephalad or toward the right relative to the patient. The FAST examination traditionally demonstrates two-dimensional grayscale images of four views reflective of the anatomic potential spaces (i.e., the hepatorenal recess or Morrison's pouch, the splenorenal recess, the rectovesical or rectouterine space, and the pericardial space). View the rectovesical space, the area between the rectum and the bladder, in the male patient. View the rectouterine space, the area between the rectum and the uterus, in the female patient.

The overall examination time should be less than 5 minutes. It frequently can be completed in 1 to 2 minutes. More than one image of the area is required if complete views of the liver or spleen are not visualized with a single image. The order of image acquisition depends on the mechanism of injury. Morrison's pouch is generally imaged first in blunt trauma. This is the area where fluid will most likely first accumulate. This is followed by the splenorenal view, the rectovesical or rectouterine view, and finally the pericardial space. The pericardial space is often imaged first, followed by Morrison's pouch in penetrating trauma, then the remaining two views.

HEPATORENAL RECESS (MORRISON'S POUCH)

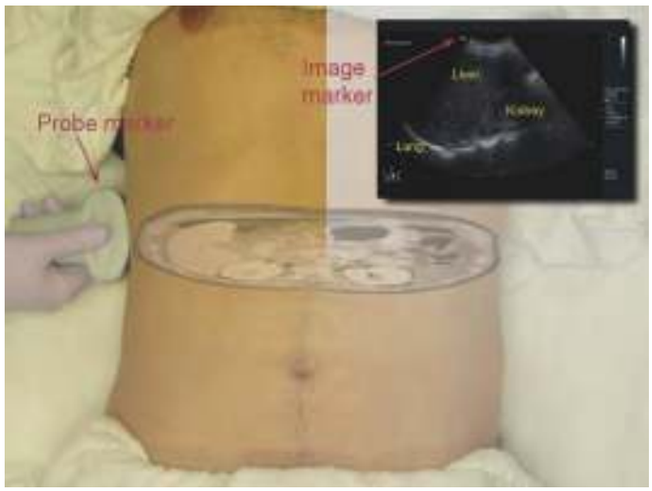
The hepatorenal recess is located between the inferior margin of the liver and inferior pole of the right kidney (**Figure 8-1*A***). Place the transducer on the midaxillary line at the level of the 8th to 11th ribs (**Figure 8-2*A***). Visualize the kidney and liver from the diaphragm to the inferior tip of the liver (**Figure 8-2*B***). **Visualize the space between the kidney and liver, including the inferior pole of the right kidney and the inferior liver tip, as fluid tends to accumulate near the tip of the liver first.**[35] A black echolucent stripe between the liver and the kidney represents blood and a positive fast exam (**Figure 8-3**). Rib shadows obscuring portions of the image may be prevented by a slight counterclockwise rotation of the transducer so it lies between the ribs.

SPLENORENAL RECESS

The splenorenal recess is located between the inferior margin of the spleen and inferior pole of the left kidney (**Figure 8-1*B***). Place the transducer on the posterior axillary line at the level of the 6th to 9th ribs (**Figure 8-4**). **The spleen and kidney should be visualized from the diaphragm to the inferior tip of the spleen.**[35,36] A black echolucent stripe between the spleen and the kidney represents blood and a positive FAST exam (**Figure 8-5**). Rib shadows obscuring portions of the image may be prevented by a slight clockwise rotation of the transducer so it lies between the ribs.

RECTOVESICAL OR RECTOUTERINE POUCH

The rectovesical or rectouterine space represents the most inferior-posterior reflection of the peritoneum. Place the transducer just superior to the pubic ramus (**Figure 8-6**). The transducer can be oriented in either the sagittal or transverse plane. View the rectovesical space (**Figures 8-6*A* and 6*B***) or the rectouterine space (**Figures 8-6*C* and 6*D***) in both the sagittal and transverse planes.



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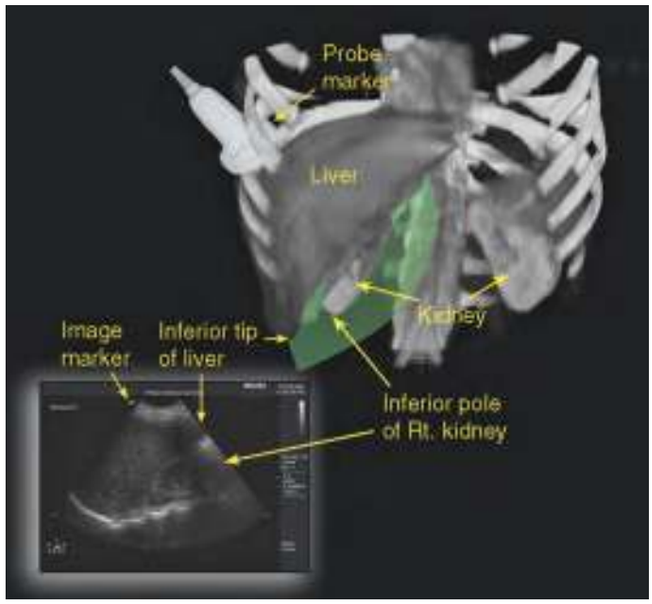


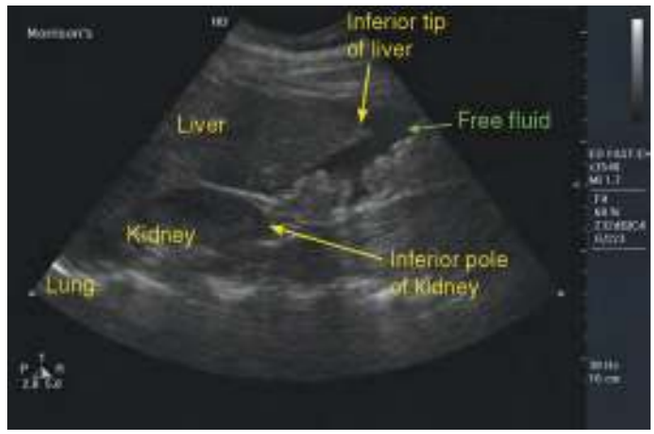
B

FIGURE 8-2. Imaging of the hepatorenal recess. ***A.*** US transducer placement with corresponding US screen image. ***B.*** A view of the US beam (green) as it passes through the liver and kidney with corresponding US image.

A black echolucent stripe represents blood and a positive FAST exam (**Figures 8-7 and 8-8**).

PERICARDIAL SPACE

Fluid accumulates initially in the inferior-posterior portion of the pericardial space in the supine trauma patient. The transducer and screen image orientation are different than traditional echocardiographic views. Place the transducer with the marker oriented toward the patient's right and the tip of the transducer aimed toward the patient's left shoulder (**Figure 8-9**). Use the liver as an acoustic window and increase the depth of the US beam to increase image quality. Fluid will initially accumulate near the top of the US image, representing the inferior portion of the pericardial space (**Figure 8-9 inset**). Visualize the inferior and superior areas of the pericardial space. A black echolucent stripe in the inferior or



A

B

FIGURE 8-3. A positive FAST exam of the hepatorenal recess. ***A.*** A black echolucent fluid stripe in Morrison's pouch. ***B.*** A normal scan for comparison.

superior areas of the pericardial space represents blood and a positive FAST exam (**Figure 8-10**).

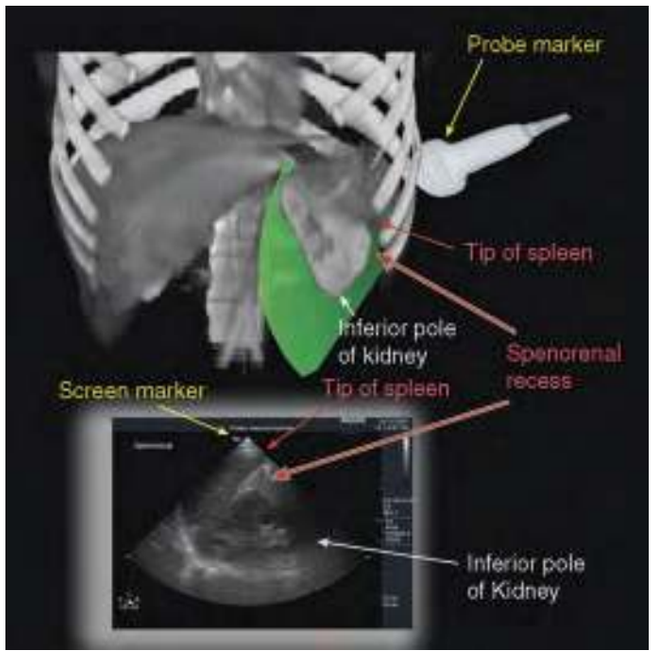
ALTERNATIVE TECHNIQUES

The incorporation of thoracic imaging coupled with the traditional FAST examination has been referred to as an extended FAST, or EFAST, examination. The additional imaging assesses for the presence of a hemothorax or a pneumothorax.[37-42] A hemothorax is best assessed by visualizing the inferior-posterior aspect of the plural cavity in the supine patient (**Figure 8-11**). As little as 20 mL of blood may be visualized.[33] This area is often visualized in routine views of Morrison's pouch but may require moving the US transducer superior one intercostal space. The lung edges are not normally visualized on US. The lung can be seen "floating" in fluid in the presence of a hemothorax (**Figure 8-11*B***). Another sign suggestive of a hemothorax is the "spine" sign (**Figure 8-11*B***). The portion of the thoracic spine above the diaphragm can be easily visualized with US. The thoracic spine is not normally well visualized on US superior to the diaphragm (**Figure 8-11*C***).

A pneumothorax has distinct findings using US. There is an absence of lung sliding. There are no comet-tail artifacts or B-lines. There are no lung pulsations with respiration. There may be one or two lung points, as described below.

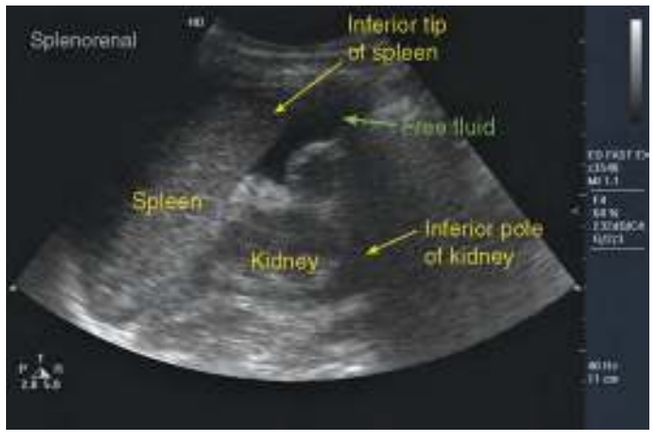
A pneumothorax is best assessed in the supine patient with a curvilinear, linear, or phased-array transducer. A high-frequency

A



B

FIGURE 8-4. Imaging of the splenorenal recess. ***A.*** US transducer placement with corresponding US screen image. ***B.*** View of the US beam (green) as it passes through the spleen and kidney.



A

B

FIGURE 8-5. A positive FAST exam of the splenorenal recess. ***A.*** A black echolucent fluid stripe. ***B.*** A normal scan for comparison.

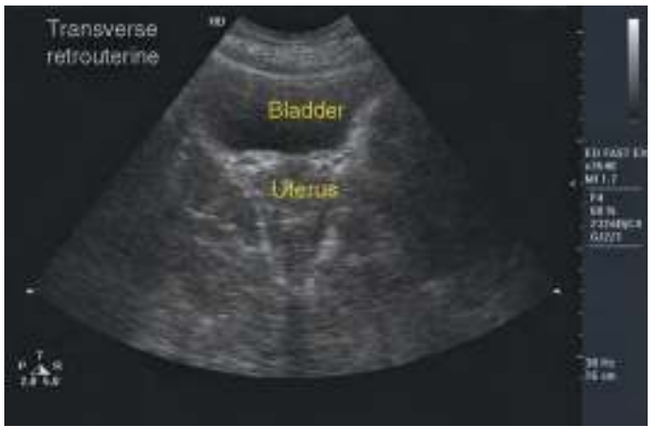
linear transducer used to image vascular structures is ideal but not required. Sensitivities and specificities range from 59% to 96% and 98% to 99%, respectively, depending on the gold standard.[43-45] Place the transducer on the anterior chest wall in the area bounded by the anterior axillary line, clavicle, nipple, and sternum. This position roughly correlates to the third or fourth interspace.

Start with the transducer on the midclavicular line between the nipple and the clavicle. Move the transducer within the area to obtain the best image possible. Position the transducer perpendicular to adjacent ribs to visualize the intercostal space (**Figure 8-12**). Visualize the interface between the parietal and visceral pleura as a hyperechoic white line. Visualize the normal sliding (i.e., to-and-fro movement) of the pleura on each other during respirations (**Figure 8-12*A***). The interface between the two pleura is known as the "sliding-lung" sign and is absent in a pneumothorax. Comet-tail artifacts are white hyperechoic reverberation artifacts visualized deep to the pleural line. Air present between the parietal and visceral pleura prevents visualization of lung sliding and comet tails in a pneumothorax.

Use M-mode to visualize the "sea-shore" sign (**Figures 8-12*A* and 8-12*B***). Visualize slightly wavy and parallel lines from the thoracic wall over an echogenic line (i.e., the pleural line) under which is a sandy pattern produced by the lung parenchyma. The parallel lines at the top of the screen represent the waves of the "sea." The sandy pattern at the bottom of the screen represents the "shore." Together, they form the "sea-shore" sign. The echogenic line between the "sea" and the "shore" represents the pleural lines. **The "sea-shore" sign indicates a normal chest wall–lung interface.** Air interposed between the chest wall and lung forms parallel horizontal echogenic lines and the loss of the "sea-shore" sign (**Figure 8-12*C***).

The lung point is pathognomonic for a pneumothorax.[46] The lung point is the transition between the sliding-lung sign and the lack of the sliding-lung sign (i.e., the edge of the pneumothorax). It is also seen when the air and lung can be visualized in the same US window. **The lung point is a dynamic sign**. It can only be visualized with the transducer fixed and observing the pleura during the entire respiratory cycle. This is 100% specific to confirm a pneumothorax. This may be difficult to visualize. The lung point can be mistaken for blebs, heart–diaphragm interface, heart–lung interface, lung fibrosis, lung scarring, pleural effusions, or severe pulmonary disease.

A



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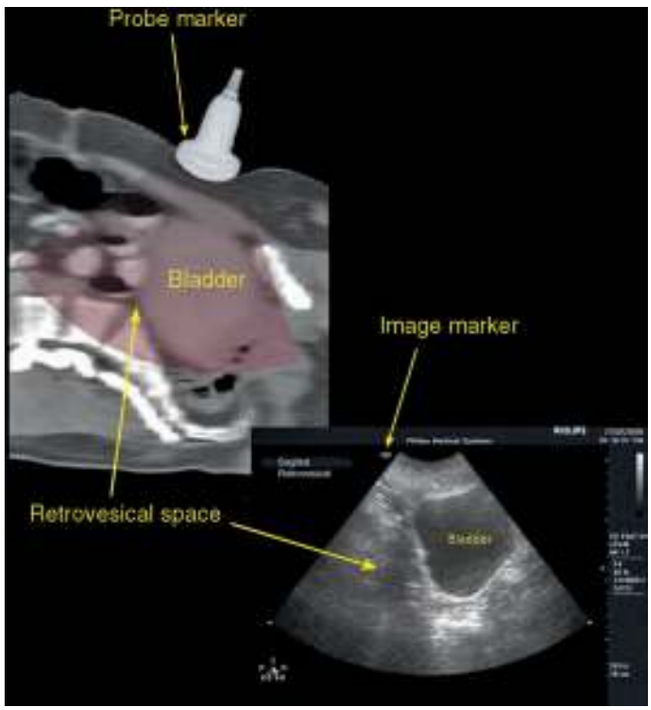

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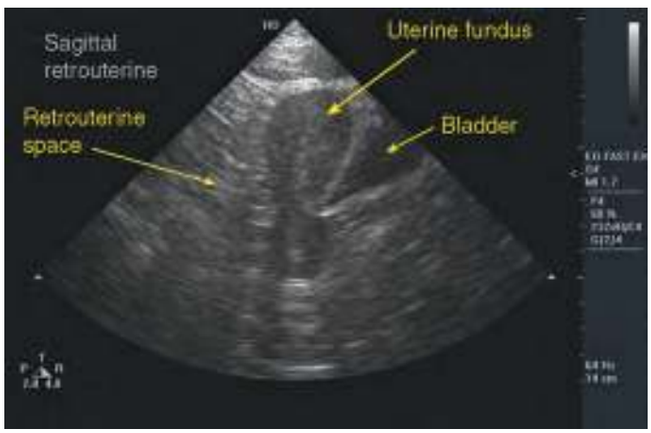

D

FIGURE 8-6. Imaging of the rectovesical and the rectouterine spaces. ***A.*** Transverse US transducer placement for the rectovesical view with corresponding US image. ***B.*** Sagittal view and transducer orientation of the rectovesical space displaying the US beam path (pink) and corresponding US image. ***C.*** Transverse view of the rectouterine space. ***D.*** Sagittal view of the rectouterine space.

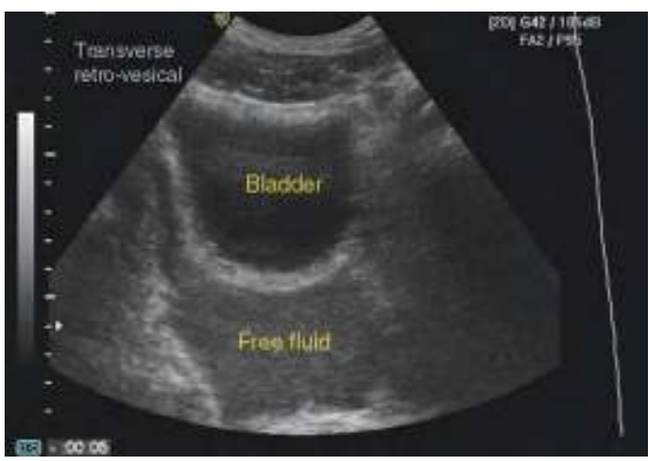

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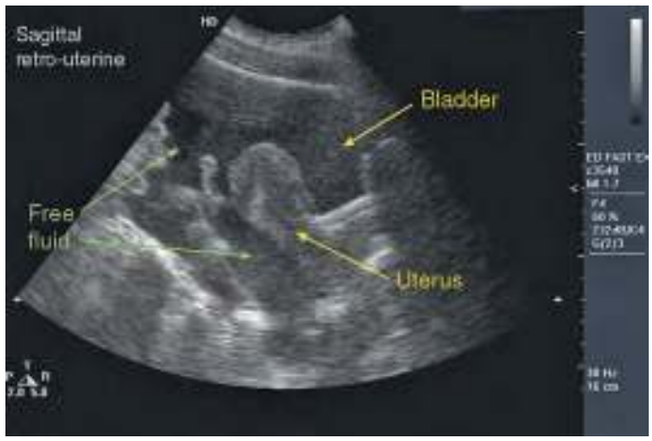
FIGURE 8-7. A positive FAST exam of the rectovesical space. ***A.*** Transverse view of a black echolucent fluid area. ***B.**** A normal scan for comparison.



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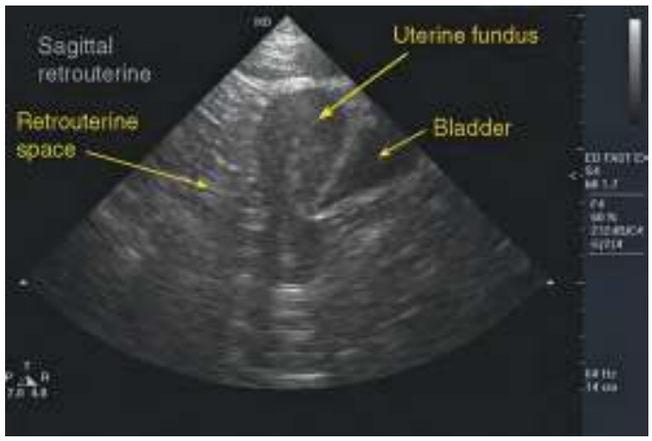


B

C

D

FIGURE 8-8. A positive FAST exam of the rectouterine space. ***A.*** Sagittal view of a black echolucent fluid area. ***B.*** A normal sagittal scan for comparison. ***C.*** Transverse view of a black echolucent fluid area. ***D.*** A normal transverse scan for comparison.

ASSESSMENT

The interpretation of each of the four views and of the overall FAST examination is classified as positive, negative, or indeterminate.[47] Positive examinations demonstrate black echolucent fluid accumulation in any one of the four views. Negative examinations and scans demonstrate no fluid with complete visualization of all structures. Indeterminate examinations are uncommon and occur when fat or bowel cannot be distinguished from peritoneal fluid or if there is incomplete visualization of any of the four views. Women and children may normally have a small amount of physiologic fluid in their pelvis.[48] This finding may lead to an indeterminate exam in the setting of trauma. Children are less likely to have free fluid in the pelvis despite an intraabdominal injury.[20,48,49]

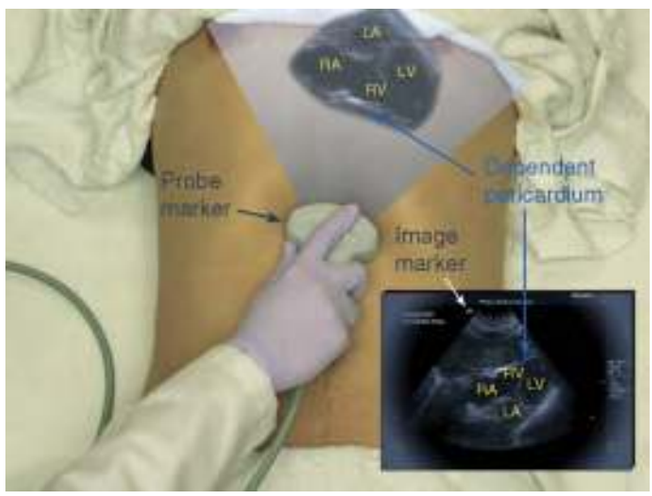


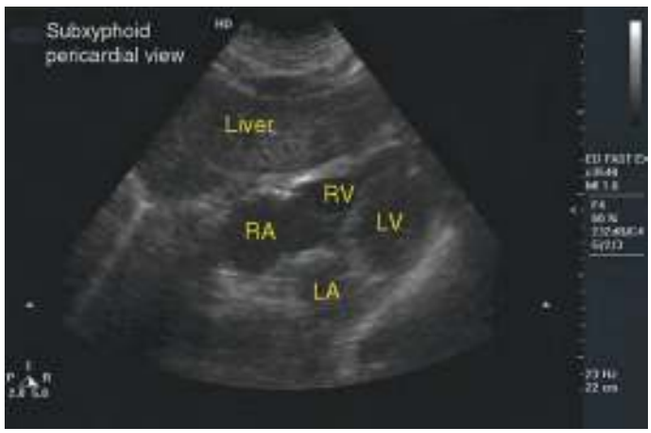
FIGURE 8-9. Imaging of the pericardial space. US transducer placement for the subxiphoid view with corresponding US image. (RV, right ventricle; RA, right atrium; LV, left ventricle; LA, left atrium.)

AFTERCARE

Save any US images that are used to make clinical decisions. This is for medical care of the patient and later quality assurance purposes. Label the images with the date, patient medical record number, patient name, and sonographer's name. Make any notes of importance on the images.

Multiple algorithms exist for the management of trauma patients.[1,4] These are differentiated into blunt trauma versus penetrating trauma and hemodynamically stable versus hemodynamically unstable. A positive FAST examination in the hemodynamically stable blunt trauma patient is typically followed with an abdominal CT scan.[50] A positive FAST examination in the hemodynamically unstable blunt trauma patient indicates the need for emergent operative intervention without further imaging. A positive FAST examination in adult

A



B

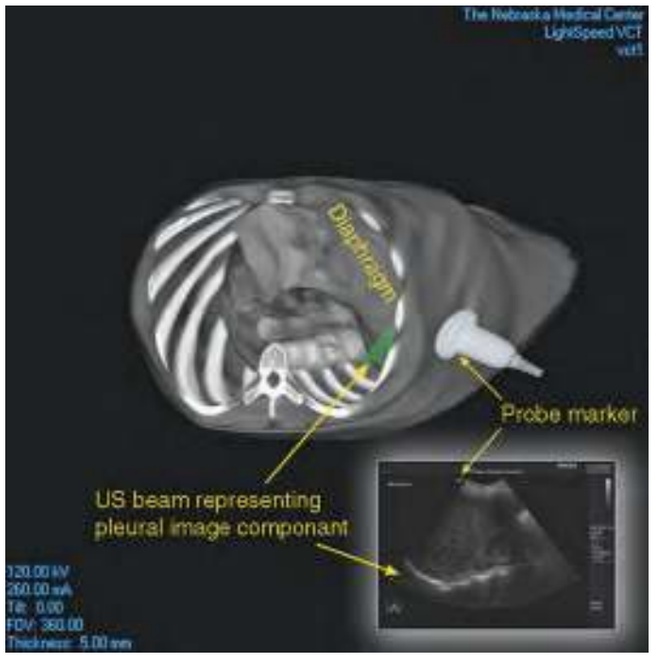
FIGURE 8-10. A positive FAST exam of the pericardial space. ***A.*** A large black echolucent area of fluid in the inferior-posterior aspect of the pericardial space and a small amount of fluid in the more superior area of the pericardial space. ***B.*** A normal scan for comparison.

patients with penetrating trauma has good correlation with need for a therapeutic laparotomy.[32] Management of these patients may vary between institutions. An unstable pediatric patient with a positive FAST examination may not necessarily proceed to laparotomy because the indications for operative management differ between adults and children.[4]

A negative FAST examination needs to be evaluated within the patient's clinical context. A negative FAST exam in the hemodynamically stable or unstable penetrating trauma patient is a reliable screen for the presence of intrapericardial blood. **It is not a reliable screening test for the presence of abdominal injury.**[18,51,52] A negative FAST examination in the hemodynamically stable blunt or penetrating trauma patient should be followed by serial clinical examinations and a repeat FAST examination or a CT scan.[31,50,53,54] Continue the work-up when the FAST examination is negative because of false-negative results are possible. A negative FAST exam in the presence of hemodynamic instability has a high sensitivity; consider an extraperitoneal source of bleeding for the instability. Indeterminate FAST examinations require a follow-up alternative diagnostic modality, usually a CT scan.

COMPLICATIONS

Performance of the FAST or EFAST examination has no associated complications. Inaccurate interpretation or inappropriate application of US findings in clinical decision making may complicate



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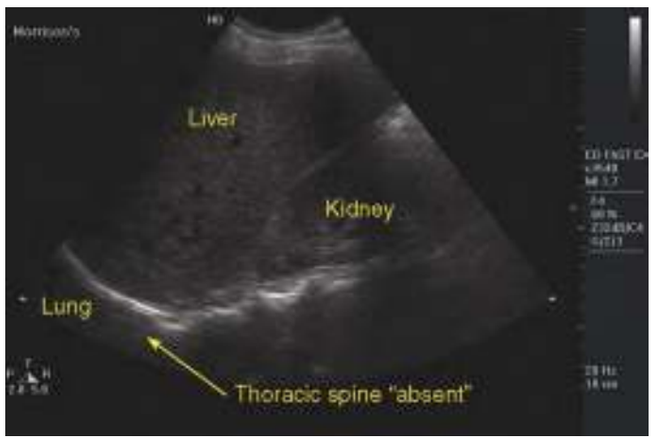
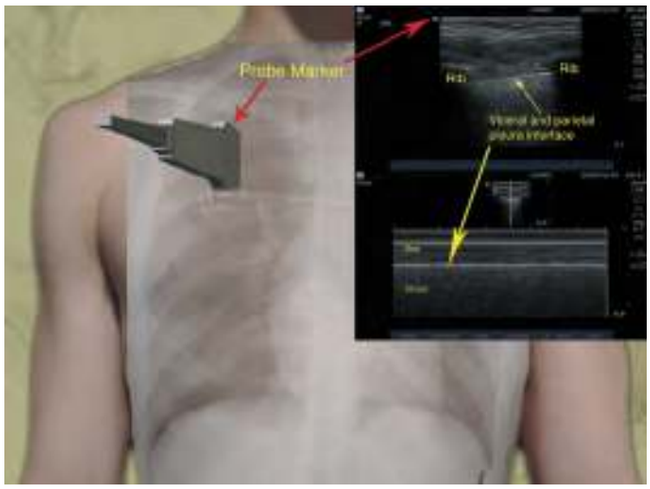


C

FIGURE 8-11. Imaging a hemothorax. ***A.*** The patient and US transducer position. The US beam path (green) generates the pleural component (inset). ***B.*** Blood in the pleural space provides an acoustic window to visualize the thoracic spine ("spine sign"). ***C.*** A normal scan for comparison.



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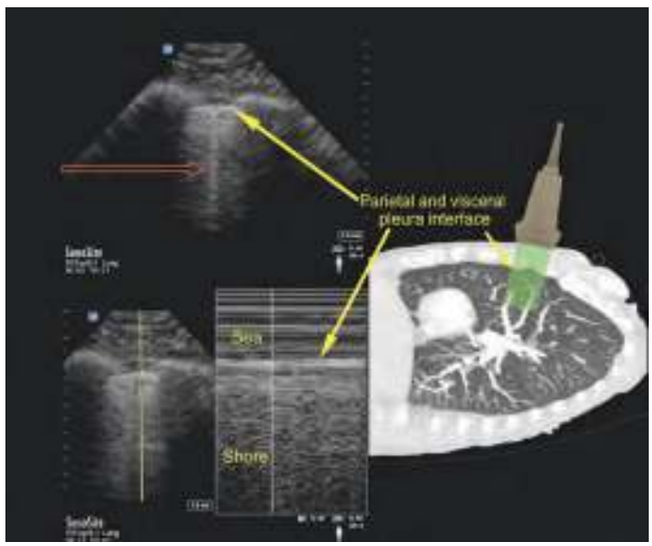

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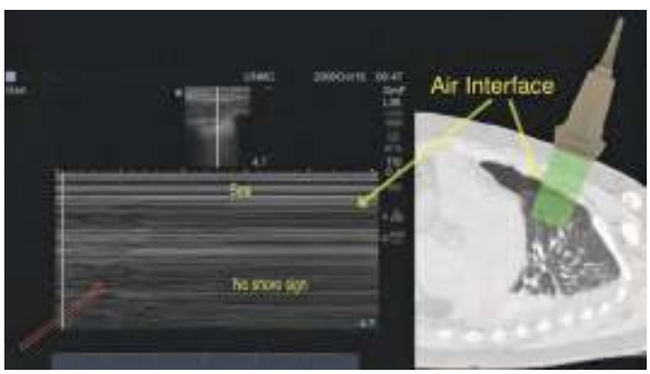

C

FIGURE 8-12. Imaging a pneumothorax. ***A.*** Transducer position is demonstrated with corresponding rib shadowing seen on both sides of the intercostal space. ***B.*** The echoic interface of the parietal and visceral pleura demonstrates the "sliding-lung" sign and the "sea-shore" sign (yellow arrow). It is visible when the lung and chest wall are in contact with each other. Note the bright echogenic stripe of the pleural interface between the chest wall ("sea") and the lung ("shore"). A "comet-tail" artifact may be seen with movement of the parietal pleura (red arrow). ***C.*** The "sea-shore" sign and the pleural interface are absent when a pneumothorax is present. The red arrow represents motion artifact.

TABLE 8-2 Common Errors

Not examining (i.e., fanning through) the depth of an organ
Not examining (i.e., fanning through) the length of an organ
Not performing serial examinations when the patient changes clinically
Not performing serial examinations with vital sign changes
Not realizing that clotted blood is mixed echogenicity
Not realizing the FAST examination cannot differentiate ascites from blood
Not realizing the FAST examination is insensitive for identification of small amounts of fluid
Not realizing the FAST examination is insensitive for retroperitoneal injuries
Not realizing the FAST examination is insensitive for solid organ injuries
Rib shadowing obstructing the view

its use. Its performance should not delay operative intervention if the patient has indications for the Operating Room (e.g., bowel evisceration).[54] Common US errors are noted in **Table 8-2**.

SUMMARY

The FAST and EFAST examinations are useful adjuncts in the management of blunt and penetrating trauma patients. They can be performed rapidly, are very specific, and can positively impact patient outcomes. Using US has many benefits (**Table 8-1**). Proper training is required to ensure good-quality images and appropriate interpretation. The false-negative rate makes the use of US more appropriate to rule in an injury than rule one out. Continue the work-up of a negative FAST or EFAST examination if the patient warrants it.

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SECTION 2

Respiratory Procedures

9 Essential Anatomy of the Airway

Divya C. Karjala, Serge G. Tyler, and Isam F. Nasr

INTRODUCTION

A thorough understanding of airway anatomy is essential for the performance of any airway procedure.[1-10] **Untoward events due to a procedure are usually the result of inexperience and/or an inadequate understanding of the regional anatomy.** The anatomy of the airway and airway procedures are no exception. An understanding of the anatomy of the airway will result in fewer attempts at intubation and improved success with fewer iatrogenic misadventures.

GENERAL ANATOMY

The upper airway comprises the nasal and oral cavities, the pharynx, and the larynx. The lower airway consists of the subglottic larynx, the trachea, and the bronchi.[8] Airway management typically involves the upper airway, which is the focus of this chapter. The anatomy of the pharynx, larynx, trachea, and principal bronchi is depicted in **Figure 9-1**.[11]

The nares serve as the functional beginning of the airway, namely warming and humidification of air.[4] The mucosa of the nasal passage is extremely vascular and fragile. It is susceptible to bleeding with minimal manipulation during instrumentation to establish an airway. The nasal blood supply originates from branches of the internal and external carotid arteries. It is wise to consider the use of a vasoconstricting agent, when appropriate, to help avoid epistaxis, which may obscure further attempts at securing the airway. Although patients tolerate nasal intubation better than oral intubation for a longer time, it is more important in an emergency to definitively secure the airway using a straightforward oral intubation. Choose the more patent side of the nasal cavity for instrumentation in patients with nasal septal deviation.[4]

The sensory innervation of the upper airway is provided by branches of several cranial nerves. The mucous membrane of the nose is innervated anteriorly by the anterior ethmoid nerve (i.e., ophthalmic division of trigeminal nerve) and posteriorly by the sphenopalatine nerve (i.e., maxillary division of trigeminal nerve). The tongue is innervated by the lingual nerve on its anterior two-thirds (i.e., a branch of the facial nerve) and posterior one-third by the glossopharyngeal nerve. The glossopharyngeal nerve also innervates the adjacent areas, the palatine tonsils, the undersurface of the soft palate, and the roof of the pharynx.[1]

The pharynx is a fibromuscular tube that extends from the base of the skull to the level of the cricoid cartilage. It connects the nasal and oral cavities with the larynx and esophagus, forming the oropharynx, nasopharynx, and hypopharynx. The velopharynx is the region of nasopharynx at the level of soft palate, a common site for upper airway obstruction in awake and anesthetized patients. An awake patient maintains the pharyngeal muscle tone to keep the airway patent. This tone is lost under general anesthesia, which promotes and causes upper airway obstruction. A chin lift and jaw thrust maneuver increases the tension on the pharyngeal muscles and counteracts the tendency of the pharyngeal airway to collapse. The posterior wall of the pharynx is made of the buccopharyngeal fascia, which separates the pharynx from the retropharyngeal space. Inadmissible advancement of a gastric or tracheal tubes can result in laceration of this fascia and a retropharyngeal dissection.[4]

The trachea measures 10 to 16.5 cm in an average adult.[4] The trachea is a tubular structure that begins at the level of the fifth or sixth cervical vertebra. It bifurcates at the level of the fifth thoracic vertebra into two primary bronchi. The posterior aspect of the trachea is flat and membranous, while its anterior and lateral aspect is lined by 16 to 20 horseshoe-shaped cartilaginous rings. The primary bronchi subsequently branch into three secondary bronchi on the right and two secondary bronchi on the left. The angle between the primary bronchus and the trachea on the left is more acute than on the right. This is due to the heart being located on the left side. This is clinically significant during aspiration and endobronchial intubations. **The more direct path on the right side due to the obtuse angle of the primary bronchi results in objects (e.g., food, fluid, foreign bodies) being more likely to enter the right lung.** The tracheal mucosa removes waste products by producing and moving mucus toward the pharynx via ciliary action. The trachea is richly innervated from the vagus nerve. This permits a vigorous cough reflex accompanied by hypertension and tachycardia if a foreign body is aspirated.

The inner diameter of the trachea varies between normal adult males and females, measuring about 15 to 20 mm.[4] The external diameter of a 7.5-mm internal diameter (ID) endotracheal tube is 11.0 mm. **Size must be taken into consideration in selecting an endotracheal tube.** These considerations usually preclude using endotracheal tubes much larger than 7.5 mm ID for normal adult females or larger than 8.0 or 8.5 mm ID for normal adult males.

ANATOMY OF THE LARYNX

The primary functions of the larynx are phonation and airway protection. The innervation of the larynx is relatively simple. The internal branch of the superior laryngeal nerve provides sensation above the vocal cords (i.e., vocal folds). The recurrent laryngeal nerve supplies sensation below the vocal cords. The recurrent laryngeal nerve provides the motor input to the intrinsic muscles of the larynx except to the cricothyroid muscle, which is supplied by the external branch of the superior laryngeal nerve. Bilateral injury to the recurrent laryngeal nerve will result in total airway closure due to unopposed stimulation of the vocal cord adductor (i.e., the cricothyroid muscle).[1]

There are three paired and three unpaired cartilages of the larynx.[2] The paired cartilages are the smaller arytenoid, corniculate, and cuneiform cartilages (**Figure 9-2**). The unpaired cartilages are the larger thyroid, cricoid, and epiglottic cartilages. The hyoid bone is not part of the larynx but has many ligamentous and

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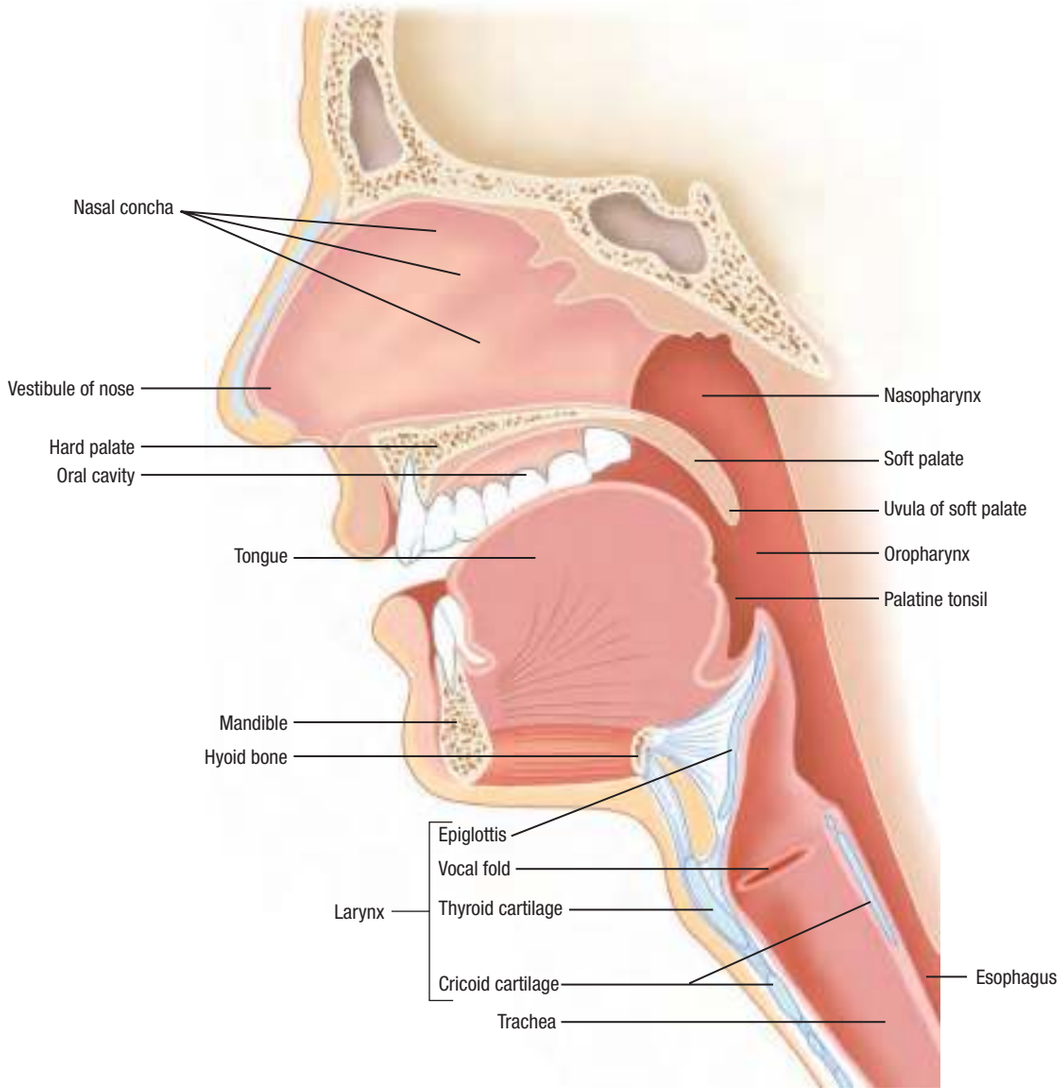

FIGURE 9-1. Anatomy of the upper airway as visualized in a midsagittal section through the head and neck.

muscular attachments to the larynx. The cricoid cartilage is signet ring-shaped, as opposed to the C-shaped cartilages of the trachea (**Figure 9-2**). Depression of the cricoid cartilage will put pressure on structures located posteriorly (i.e., the esophagus) because it forms a complete circle. **The application of posteriorly directed pressure on the cricoid cartilage during intubation is known as the Sellick maneuver (Figure 9-3). The Sellick maneuver will not prevent regurgitation from active vomiting. It has been shown to be effective in the prevention of passive regurgitation and subsequent aspiration.**[3] The Sellick maneuver can impair insertion of the laryngoscope, an airway introducer, and cause airway obstruction.

The cricoid cartilage is also an important landmark for locating the cricothyroid membrane. The cricothyroid membrane lies inferior to the thyroid cartilage and superior to the cricoid cartilage (**Figure 9-2**). The cricothyroid membrane is usually located at the level of the sixth cervical vertebra. **It is the anatomic location where emergency cricothyroidotomies and recurrent laryngeal nerve blocks are performed.**[12]

The three paired cartilages are located on the posterior aspect of the larynx (**Figure 9-2**). This position renders them vulnerable to injury during intubation.[2] It is less likely that these cartilages will become dislocated or otherwise injured by maintaining an anterior insertion of the laryngoscope blade, systematic visualization of the structures, and by not inserting it too deeply during intubation attempts. This is particularly true if a straight laryngoscope blade is used.

The hyoid bone is an important supporting structure of the upper airway. One of the attachments of the hyoid bone to the larynx is the hyoepiglottic ligament located at the base of the vallecula (**Figure 9-4**). This ligament is important because it is where the tip of the curved Macintosh laryngoscope blade is placed to move the epiglottis anteriorly and out of the path of vision during intubation. Another attachment of the hyoid bone to the larynx is the thyrohyoid membrane (**Figure 9-2**). It runs from the inferior border of the hyoid bone to the superior aspect of the thyroid cartilage. The internal branch of the superior laryngeal nerve passes through the thyrohyoid membrane just inferior to the lateral border of the hyoid bone (**Figure 9-2**). The internal branch of the superior laryngeal nerve is superficial at the thyrohyoid membrane and very easily anesthetized with an injection of local anesthetic solution.

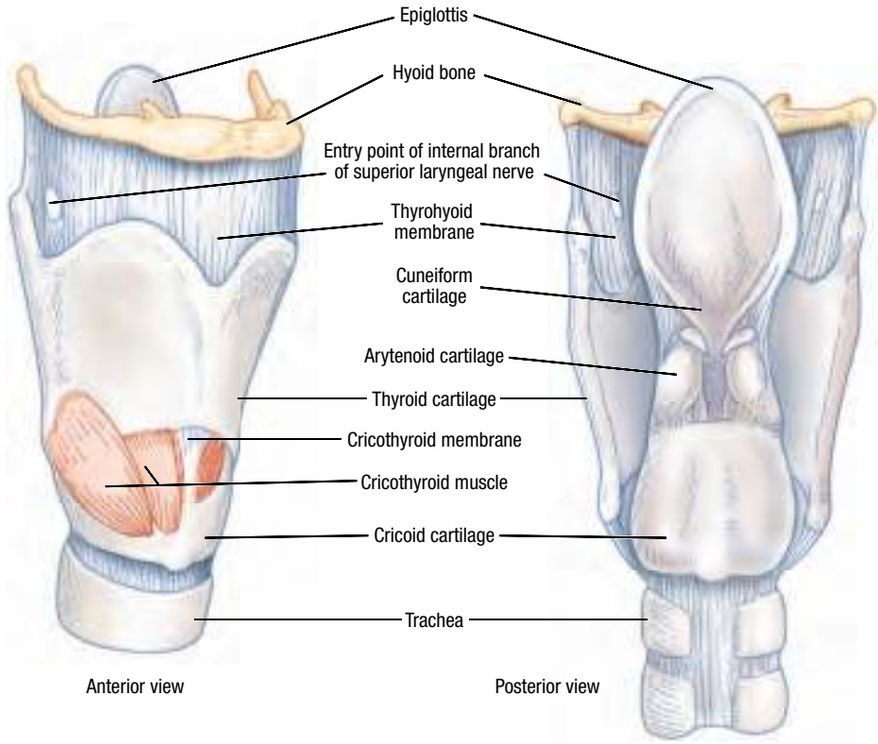

FIGURE 9-2. Right anterolateral and posterior views of the skeleton of the larynx. The thyroid cartilage shields the smaller cartilages of the larynx.

AIRWAY EVALUATION

The evaluation of the airway should always start with a thorough history. **An airway history should be conducted, when feasible, prior to the initiation of airway management in all patients.** It should include whether the patient has ever required intubation and if there was any difficulty. Additional history should focus on the patient's dentition, surgery of the airway, and surgery near the airway. There are many congenital syndromes (**Table 9-1**) and acquired conditions (**Table 9-2**) that can complicate airway management. Keep these in mind when performing the airway history and physical examination.

External evaluation of the airway is a critical step to a successful intubation and helpful in predicting a difficult intubation. External inspection should identify some obvious problems that may interfere with airway management. These include facial hair that prevents a good mask seal, cervical collars that restrict neck movement, face and/or neck trauma, severe micrognathia, or obesity.

The next steps in evaluating the airway may help to identify patients with potentially difficult airways. The distance between the thyroid cartilage ("Adam's apple") and the inside of the anterior aspect of the mandible is known as the thyromental distance. It should be at least 5 cm or about three large finger breadths in adults.[1] A lesser distance suggests the patient's vocal cords are

FIGURE 9-3. The Sellick maneuver. Posteriorly directed pressure is applied to the cricoid cartilage to occlude the esophagus and prevent regurgitation and subsequent aspiration of gastric contents.

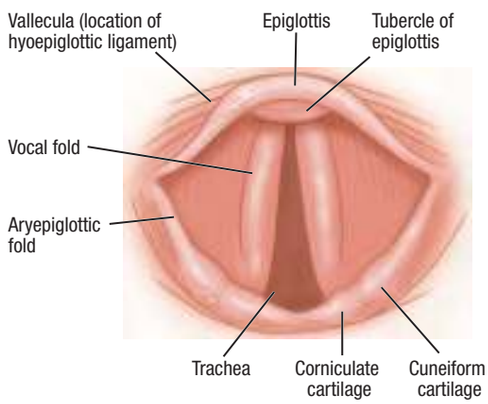

FIGURE 9-4. Laryngoscopic view of the larynx.

TABLE 9-1 Selected Congenital Syndromes Associated with Difficult Endotracheal Intubation

Syndrome	Description
Down	Cervical spine spondylolisthesis; large tongue, small mouth make laryngoscopy difficult; small subglottic diameter possible; frequent laryngospasm
Goldenhar (oculoauriculovertebral anomalies)	Mandibular hypoplasia and cervical spine abnormality make laryngoscopy difficult
Klippel-Feil	Neck rigidity because of cervical vertebral fusion
Pierre Robin	Small mouth, large tongue, mandibular anomaly; awake intubation essential in neonate
Treacher Collins (mandibulofacial dysostosis)	Laryngoscopy difficult
Turner	High likelihood of difficult intubation

Source: Modified from reference 1.

TABLE 9-2 Acquired Conditions Affecting the Airway and Associated with Difficult Endotracheal Intubation

Condition	Principal pathologic clinical features of the airway
Acromegaly	Macroglossia; prognathism
Acute burns	Edema of airway that worsens with time; secure airway early!
Angioedema	Obstructive swelling renders ventilation and intubation difficult
Arthritis (rheumatoid)	Temporomandibular joint ankylosis, cricoarytenoid arthritis, deviation of larynx, restricted mobility of cervical spine
Arthritis (ankylosing spondylitis)	Ankylosis of cervical spine; less commonly ankylosis of temporomandibular joints; lack of mobility of cervical spine
Benign tumors (cystic, hygroma, lipoma, adenoma)	Stenosis or distortion of airway
Diabetes mellitus	May have reduced mobility of atlantooccipital joint
Foreign body	Airway obstruction
Hypothyroidism	Large tongue and abnormal soft tissue (myxedema) make ventilation and intubation difficult
Infectious (croup and supraglottitis)	Laryngeal edema
Infectious abscess (intraoral and retropharyngeal)	Distortion and stenosis of airway and trismus
Ludwig's angina	Distortion and stenosis of airway and trismus
Malignant tumors (carcinoma of tongue, larynx, or thyroid)	Stenosis or distortion of airway; fixation of larynx or adjacent tissues secondary to infiltration or fibrosis from irradiation
Morbid obesity	Short, thick neck, and large tongue are likely to be present
Pregnancy	Edema of airway
Sarcoidosis	Airway obstruction (lymphoid tissue)
Scleroderma	Tight skin and temporomandibular joint involvement make mouth opening difficult
Temporomandibular joint syndrome	Severe impairment of mouth opening
Thyromegaly	Goiter may produce extrinsic airway compression or deviation
Trauma (head, face, or cervical spine injury)	Cerebrospinal rhinorrhea, edema of airway; hemorrhage; unstable fracture(s) of maxillae and mandible; intralaryngeal damage; dislocation of cervical vertebrae

Source: Modified from reference 4.

positioned more anteriorly than normal. **Distances less than 5 cm may indicate that visualization of the larynx during intubation may be difficult or impossible due to a lack of space in which to displace the tongue.**

The next step requires the patient to open their mouth maximally. The patient ideally will be in a seated or semisitting position. The distance between the maxillary and mandibular incisors in an average adult is 3 to 5 cm or approximately two large finger breadths.[5] Limited mouth opening may impair visualization of the airway as well as expose the teeth to damage during intubation.

Adults should be able to flex their cervical spine 35° and extend the cervical spine at the atlantooccipital joint 80° from a neutral position.[6] This range of neck movement allows for the alignment of the oral, pharyngeal, and laryngeal axes during orotracheal intubation (**Figure 9-5**). **This alignment of the axes provides the greatest chance for a successful intubation.** Evidence suggests slight head extension in infants and young children by placing a rolled towel behind their shoulders better aligns the vision of the glottic and laryngeal axes.[10] Observe the patient's neck for length and thickness. A short, excessively long, or thick neck may indicate difficulty in placing the patient in the sniffing position and aligning the airway axes.

The internal examination should evaluate the patient's dentition, palate, and tongue. Note any protuberant incisors (i.e., buck teeth), loose teeth, broken teeth, dental work, and dental devices. Prominent upper incisors may complicate the insertion of the laryngoscope blade, make laryngoscopy difficult, and predispose the patient to dental trauma. Assess the relation of the maxillary and mandibular incisors during normal jaw closure. Lack of an overbite forces the laryngoscope blade to enter the mouth in a more cephalad direction than normal. This can result in difficulty visualizing the airway. Observe the maxillary and mandibular incisors during voluntary protrusion of the mandible to determine the degree of temporomandibular joint mobility. Determine if the palate is normal, high and arched, or cleft. Determine if the tongue is elevated or larger or wider than normal in comparison to the oral cavity. Any abnormality can make the procedure of orotracheal intubation more difficult.

A common classification used by Anesthesiologists to grade the difficulty of laryngoscopy and intubation involves the identification of the size of the tongue in relation to the tonsillar pillars, the fauces, the soft palate, and the uvula.[7] **Instruct the patient to open their mouth and protrude their tongue maximally in the sitting position. The patient should not say "ahhh," as this distorts the anatomy and may falsely improve the airway classification.** The Mallampati classification, named after its author, has four grades or classes.[7] The anterior and posterior tonsillar pillars, the fauces, the soft palate, and the uvula can be fully visualized in class I (**Figure 9-6*A***). The fauces, the soft palate, and the uvula can be visualized in class II (**Figure 9-6*B***). The anterior and posterior tonsillar pillars are covered by the base of the tongue and not visible. Only the soft palate and the base of the uvula are visible in class III (**Figure 9-6*C***). None of the structures are visible in class IV (**Figure 9-6*D***). The predictive value of this classification is that during direct laryngoscopy, the entire glottis can be exposed in 100% of class I airways, 65% of class II airways, 30% of class III airways, and 0.1% of class IV airways.[7]

The airway evaluation is imperfect in predicting potential problems, and an airway strategy requires a combination of plans for each patient. Additional evaluation may be indicated in some patients to characterize the likelihood or nature of the anticipated airway difficulty. The findings of the airway history and physical examination may be useful in guiding the selection of specific diagnostic tests and consultation.

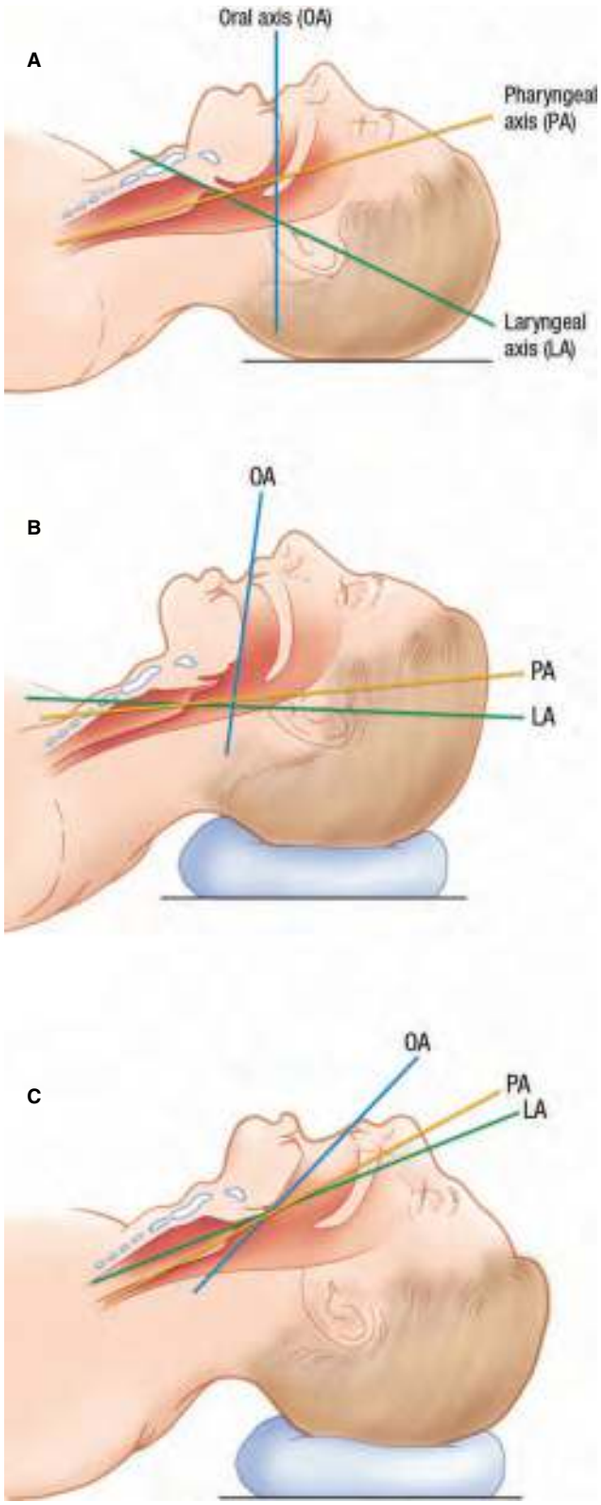

FIGURE 9-5. Head positioning for endotracheal intubation. ***A.*** The normal alignment of the oral, pharyngeal, and laryngeal axes. ***B.*** Elevation of the head about 10 cm with pads below the occiput, while the shoulders remain on the table, aligns the laryngeal and pharyngeal axes. ***C.*** Subsequent head extension, at the atlantooccipital joint, serves to create the shortest distance and most nearly straight line from the incisor teeth to the glottic opening.

FIGURE 9-6. The Mallampati classification. ***A.*** Class I. ***B.*** Class II. ***C.*** Class III. ***D.*** Class IV.

ANATOMIC DIFFERENCES BETWEEN THE ADULT AND THE YOUNG CHILD

There are numerous differences between the airway of an adult and that of a child.[11,13-15] The head-to-body ratio is larger in the child. This causes the neck to be flexed when the child is supine. Placing a rolled towel under the child's shoulders will correct the flexion. A child has a small mouth with a relatively large tongue as compared to an adult. This can make orotracheal intubation difficult. The presence of adenoidal tissue in the child makes nasotracheal intubation difficult and orotracheal intubation the preferred method.

The anatomic differences between the larynx of an adult and that of a young child are summarized in **Table 9-3** and **Figure 9-7**.[1] The vocal cords are more obliquely inclined in the child. The cricothyroid membrane is very small and narrow. **The most important difference is that the narrowest portion of the infant or young child's airway is below the level of the vocal cords at the level of the cricoid cartilage. The narrowest point of the adult airway is at the level of the vocal cords**. The cricoid cartilage is the only complete cartilaginous ring and the narrowest part of the trachea. An endotracheal tube may therefore pass through the vocal cords of a young child but might not advance past the cricoid cartilage due to normal anatomy. Forcing an endotracheal tube past the vocal cords in a young child may result in trauma to the airway and subsequent tracheal stenosis. Blind nasal intubation is not possible because the child's larynx is located more cephalad at the level of the third or fourth cervical vertebrae and angulated anterior to the glottis. The child's laryngeal inlet is narrow and more susceptible to obstruction. The U-shaped epiglottis and a more acute angle between the epiglottis and glottis cause the aryepiglottic folds to be more in the midline (**Figure 9-7*B***).

TABLE 9-3 Anatomic Differences Between the Child's and the Adult's Larynx

	Child larynx	Adult larynx
Size	Smaller	Larger
Shape	Lumen is funnel-shaped with the narrowest part below the vocal cords and within the cricoid ring	Narrowest part of lumen is at the vocal cords
Location	Higher, closer to the tongue base; vertical extent is opposite C3, C4, C5 vertebrae; more anterior	Vertical extent is lower, opposite C4, C5, C6 vertebrae
Epiglottis	Longer, narrower, and "U" shaped; the angle between glottis and epiglottis is more acute; increased chance of airway obstruction (see Figure 1-8)	Shorter and wider
Vocal cords	Angled in relation to the axis of trachea; shorter; more cartilaginous; more distensible; more likely to be injured	Perpendicular to the axis of trachea
Rigidity	The laryngeal cartilages are softer and more pliable	More rigid
Response to trauma	Mucous membrane is more loosely attached and swells more readily when traumatized or infected	Less vulnerable to trauma and infection

Differences also exist in the trachea. Children have a relatively shorter trachea. This makes both right main bronchial intubation and accidental extubation much easier. The narrower diameter of the trachea with smaller spaces between the cartilaginous rings makes a tracheostomy more difficult to perform. Consider using uncuffed endotracheal tubes in children less than 28 days of age to avoid injury and subsequent subglottic stenosis.[16] A correctly sized endotracheal tube should have a leak at 15 to 25 cmH_2O. Using the uncuffed endotracheal tube with a high leak pressure of more than 25 cmH_2O or a cuffed endotracheal tube can cause croup after extubation, an erosion of the mucosa, and/or subglottic stenosis. A cuffed endotracheal tube is recommended for anyone above the age of 28 days.[10,16-18]

The differences also continue into the chest. The infant thoracic cage is more compliant than that of the older child and adult. This can lead to more effective ventilation. The child's diaphragm is shorter and flatter than in the adult and has a decreased excursion during respiration. The child's relatively narrower airway results in an increased resistance to inspiratory and expiratory airflow. Children are more susceptible to airway edema and mucous plugs obstructing their narrow airway. Children have smaller and fewer alveoli. This results in less surface area for gas exchange compared to adults.

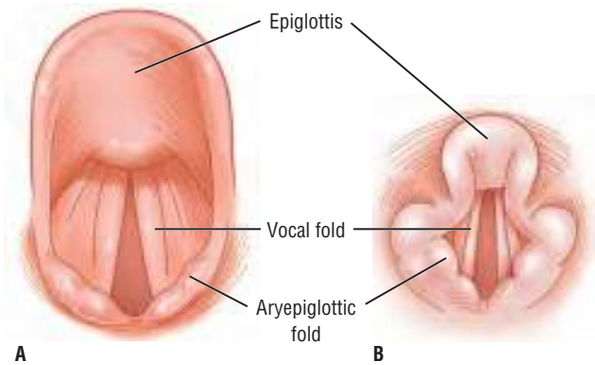

FIGURE 9-7. Differences between the adult's larynx (*A*) and the child's larynx (*B*).

ULTRASOUND OF THE AIRWAY

Ultrasound (US) is a newer development to evaluate the upper airway anatomy.[19] It is noninvasive, simple, portable, and provides no ionizing radiation. US facilitates the study of the upper airway structures (i.e., thyroid cartilage, cricoid cartilage, epiglottis, cricothyroid membrane, tracheal cartilage, and esophagus). US simplifies the assessment of airway anatomy for difficult intubation, detects upper airway pathology, helps determine endotracheal tube and laryngeal mask airway placement depth, and assesses airway size. US has proven benefit when performing emergency invasive airway procedures (e.g., cricothyroidotomy and tracheostomy). It can be used to predict postextubation stridor. An air column width ratio of 0.8 or less measured with US may predict the occurrence of postextubation stridor.[19]

SUMMARY

It is essential for Emergency Physicians to know the anatomy of the airway, especially the differences found in the young child. The airway assessment is an essential element in the preparation to intubate a patient. There may be limited information and time available to perform the full assessment in the Emergency Department. A quick airway assessment in every patient before orotracheal intubation will allow the Emergency Physician to effectively intubate.[14]

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10 Basic Airway Management

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INTRODUCTION

Airway management remains one of the most basic and important aspects of Emergency Medicine. The concepts and techniques described in this chapter can be applied in a variety of environments. Understanding the following concepts and having an opportunity to practice them will allow the provision of the most fundamental of all medical care, support of a patient's airway.

The primary purpose of airway management is to facilitate the transport of oxygen to the lungs. Without oxygen, the brain begins to die within minutes.[1] The secondary purpose of airway management is to protect the airway from aspiration or contamination with blood, fluids, or food. Airway management can be as simple as lifting a snoring patient's chin or as involved as awake, fiberoptic-guided endotracheal intubation.

The fundamental importance of airway management is reflected by the fact that much of Basic Life Support taught by the American Heart Association is concerned with this vital function.[2] **The mission of airway management is "to ensure a patent airway, provide supplemental oxygen, and institute positive-pressure ventilation when spontaneous breathing is inadequate or absent."[3] These three key aspects of airway management warrant repeating. Ensure a patent airway. Provide supplemental oxygen. Provide positive-pressure ventilation.**

Time is always critical when a patient needs airway support. The body's limited oxygen stores are rapidly exhausted once breathing stops. A healthy individual having maximally breathed 100% oxygen will begin to become hypoxic and have brain injury after 5 minutes of apnea. A sick patient breathing room air will become hypoxic almost immediately upon becoming apneic.[1]

Oxygenation and ventilation remain the essential goals of airway management. Inadequate ventilation may occur for a variety of reasons. Spontaneously breathing patients may develop an airway obstruction due to blood, food, secretions, or tissue obstruction from the loss of normal pharyngeal tone. The conscious patient with an airway obstruction will be in obvious distress and is more likely to have an obstruction due to a foreign body, laryngeal edema, laryngospasm, tissue swelling from an infection, or a tumor. The unconscious patient is at risk for aspiration of gastric contents despite spontaneous respirations. Secure the airway of an unconscious patient with intubation (Chapter 18) and mechanical ventilation (Chapter 36).

ANATOMY AND PATHOPHYSIOLOGY

The upper airway includes the nasal, oral, pharyngeal, and laryngeal anatomy and physiology. This highly complex system is responsible for conveying warmed and filtered air to the trachea and lungs while simultaneously allowing for passage of liquids and solids to the esophagus. Phonation is a secondary physiologic function of the larynx.[4] This highly sophisticated system allows us to drink liquid, eat food, breathe, and talk simultaneously. A profound system of reflexes is activated to protect the airway integrity if a small drop of liquid or a particle of food enters.[5]

The nasal cavity and the nasopharynx compose the area from the tip of the nose to the palate. The nasal cavity is bounded laterally by the bony framework of turbinates and medially by the nasal septum. This area is highly innervated by the ophthalmic and maxillary branches of the trigeminal nerve. The mucosa of the nasal cavity and the nasopharynx is highly vascular. This high degree of vascularity allows cool air from the environment to be warmed and humidified prior to entering the lungs. **It dictates that care be taken when nasal airways are placed.** It takes very little trauma to the nasal mucosa to cause significant epistaxis. The nasopharynx can be obstructed from congestion due to an upper respiratory infection, mucus, or polyps. The nasopharynx is oriented in an anteroposterior plane. **Insert the nasal airway or nasogastric tube perpendicular to the horizontal axis and not in a cephalad direction.**

The oropharynx extends from the palatoglossal fold down to the epiglottis. The primary structure contained within the oropharynx is the base of the tongue. The anterior two-thirds of the tongue is innervated by the lingual nerve, a branch of the facial nerve. The posterior one-third of the tongue, the tonsils, and the palate are innervated by the glossopharyngeal nerve.[6] Salivary glands located in the oropharynx can produce a significant volume of saliva and create potential problems for mask ventilation or intubation. Loose teeth can be inadvertently dislodged into the oropharynx and can become hazardous foreign bodies in the airway.

Edentulous patients present a unique set of problems for mask ventilation.[7] The lack of a maxillary alveolar ridge may allow the face mask to collapse into the airway. Redundant tissue due to lack of teeth tends to collapse into the airway. This makes mask ventilation extremely difficult without a nasal airway or oral airway. An appropriately sized and placed oral airway is the best way to overcome the problem of upper airway obstruction in the unconscious and edentulous adult patient.

The laryngopharynx extends from the epiglottis to the inferior border of the cricoid cartilage. The piriform recesses lie in the pharynx, on either side of the larynx, and the esophagus resides posteriorly. The larynx is positioned at the entrance to the trachea, acts as the sphincter of the pulmonary system, and is made up of nine cartilages to support this function.[6]

There are three paired and three unpaired cartilages. The unpaired cartilages are the cricoid, epiglottis, and thyroid cartilages. The paired cartilages are the arytenoids, corniculates, and cuneiform cartilages. **The cricoid cartilage is the only complete ring in the entire airway.** This presents a unique opportunity for the practitioner to help prevent gastric aspiration. **The cricoid cartilage can be firmly pressed posteriorly to pinch the esophagus against the cervical spine and prevent the passive regurgitation of gastric contents. This is known as the Sellick maneuver.[8] The cricoid cartilage is the narrowest point of the airway in the pediatric patient, while the glottic narrowing is the most narrow point in adults.** The cricoid and thyroid cartilages are the landmarks for identifying the cricothyroid membrane, which is essential in obtaining an emergent surgical airway.

The sensory and motor innervation of the larynx is derived from the vagus nerve.[6] The superior laryngeal nerve is a branch of the vagus nerve and gives rise to the internal branch. This provides the sensory innervation of the upper larynx. The superior laryngeal nerve gives rise to the external branch, which provides the motor innervation to the cricothyroid muscle (i.e., a vocal cord adductor). The recurrent laryngeal nerve provides the sensory innervation to the larynx below the vocal cords and motor innervation to all other laryngeal muscles.[6]

The trachea is approximately 15 cm long in an adult. It is composed of 17 or 18 C-shaped cartilaginous rings.[6] **The rings are essential to prevent the trachea from collapsing during the negative intrathoracic pressures generated on inspiration.**

INDICATIONS

The decision to institute airway support must often be made very quickly and frequently without the aid of laboratory results, pulmonary function tests, or radiographic studies. **The decision to institute emergent airway support is usually based on clinical judgment and the signs and symptoms of inadequate oxygenation and ventilation.** The signs of impending respiratory failure are agitation, cyanosis, dyspnea, tachypnea, and use of accessory muscles.[9] The patient will demonstrate extreme anxiety, audible wheezing or stridor, and aggressive attempts to clear the obstruction in the case of a partial airway obstruction. There may be no audible breath sounds if the obstruction is complete.

Perform a more formal evaluation of the indicators that warrant respiratory assistance if time exists (**Table 10-1**).[10] The ultimate signs indicating the necessity for airway assistance are hypoxia and hypercarbia. The most common etiologies resulting in the need for airway support are airway obstruction (e.g., food, foreign body, and vomit), cardiopulmonary arrest, drug overdoses, and toxic reactions. Impending ventilatory failure due to congestive heart failure, pneumonia, or severe asthma is a common indication for endotracheal intubation.

CONTRAINDICATIONS

There are no absolute contraindications for basic airway management. The contraindications for the various methods of endotracheal intubation are discussed in subsequent chapters.

EQUIPMENT

- Bag-valve-mask device
- Oxygen source
- Clear face masks, various sizes and shapes
- Oropharyngeal airways, various sizes
- Nasopharyngeal airways, various sizes
- Head strap
- Yankauer suction catheter
- Suction source
- Pulse oximeter
- Tongue blades or tongue depressors
- Water-soluble lubricant or anesthetic jelly
- Cardiac monitor
- Noninvasive blood pressure system

TABLE 10-1 Indicators That Warrant Respiratory Assistance

A-a gradient > 350 mmHg on 100% O_2
$PaCO_2$ > 55 mmHg
PaO_2 < 60 mmHg on 40% O_2
Respiratory rate > 35
SaO_2 < 90%
Vital capacity < 15 mL/kg

Data from reference 10.

TECHNIQUES

PATIENT POSITIONING

The first goal of airway management, regardless of a patient's ability to breathe spontaneously, is the establishment of a patent airway. This may be all that is required in a patient who has an upper airway foreign body or a patient who has suffered a loss of consciousness with loss of pharyngeal tone. **The importance of proper positioning cannot be overemphasized. The success of airway management is predicated on this very basic but often overlooked issue.** Placing the patient in the "sniffing" position, or lateral decubitus position, may correct many upper airway obstructions due to soft tissue impingement.[11] **The "sniffing" position is achieved by flexing the cervical spine approximately 15° and extending the atlantooccipital joint maximally (Figure 10-1).**[10] This is the position one subconsciously adopts in order to sniff and smell. **Omit head extension in the patient with cervical spine precautions. The sniffing position can be achieved with the chin-lift and/or jaw-thrust maneuvers.**

Obesity or large breasts on a patient often cannot be effectively managed in a supine position (Chapter 15). The normal sniffing position in an obese person is often not sufficient to relieve an airway obstruction (**Figure 10-2*A***). Place a ramp or shoulder roll under the patient's upper back to achieve the sniffing position (**Figure 10-2*B***).

JAW-THRUST MANEUVER

The jaw-thrust is one of the most basic maneuvers and an initial method of establishing a patent airway.[12] The tongue is attached to the mandible and may fall into the pharynx in the supine patient. **The goal of the jaw-thrust maneuver is to move the tongue away from the palate and posterior pharyngeal wall.** The jaw-thrust maneuver is a two-handed technique that can be used with the face mask and a second person to provide positive-pressure ventilation. Stand at the head of the patient and place fingers on the angles of the patient's mandible bilaterally and displace the mandible anteriorly (**Figure 10-3**). This maneuver elevates the tongue from the pharynx and allows air to flow unobstructed and posterior to the tongue.

FIGURE 10-1. The "sniffing" position for successful airway management.

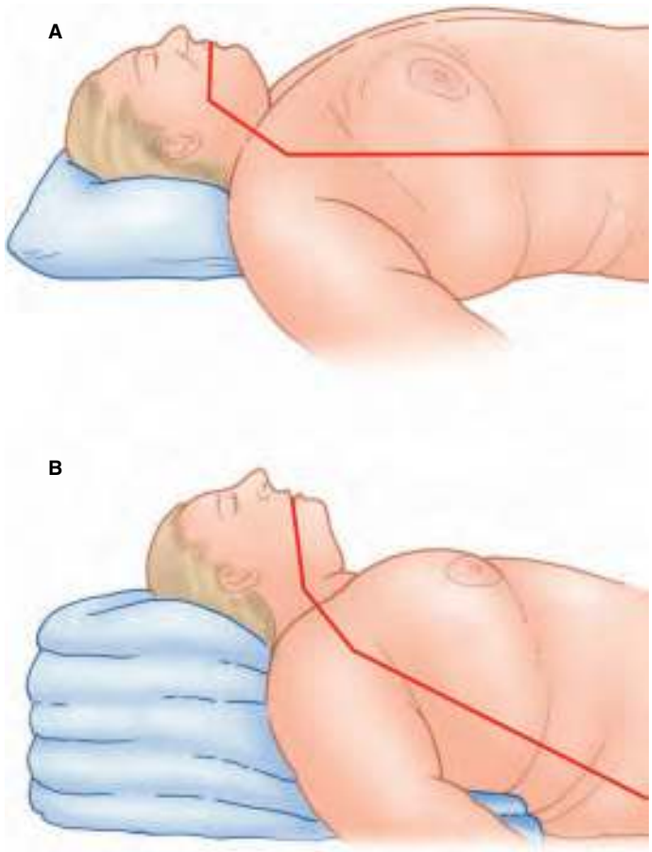


FIGURE 10-2. Airway management in the obese patient. ***A.*** The normal "sniffing" position is inadequate to open the airway. The red line represents the axis of the airway. ***B.*** A ramp placed under the head and shoulders will achieve the "sniffing" position.

CHIN-LIFT MANEUVER

The chin-lift is another basic maneuver and an initial method of establishing a patent airway.[12] Place the fingers on the inferior surface of the patient's mandible (**Figure 10-4**). **Do not place any of the fingers on the soft tissues of the submandibular space. This will elevate the tongue and cause further obstruction.** Lift the chin in an anterior and cephalic direction. The head may be tilted slightly posteriorly to aid in opening the airway.

FIGURE 10-3. The jaw-thrust maneuver.

FIGURE 10-4. The chin-lift maneuver.

NASOPHARYNGEAL AIRWAYS

The majority of airway obstruction occurs in the region of the pharynx.[13] Use various aids in addition to positioning to overcome obstruction and facilitate effective ventilation. The most commonly used devices are oropharyngeal (oral) and nasopharyngeal (nasal) airways. **Place a large enough airway to bridge the area of soft tissue impingement on the pharynx regardless of which device is chosen.**

Nasal airways (i.e., nasopharyngeal airways or nasal trumpets) are soft rubber, plastic, or silicone tubes that are inserted through the nostril and into the oropharynx just above the epiglottis. Nasal airways are available in numerous sizes (**Figure 10-5**). The proximal end has a flange that rests against the patient's nares and prevents the nasal airway from slipping backward into the nose and becoming a foreign body in the patient's airway. The larger the inner diameter, the longer is the tube. The nasal airway is more comfortable for the patient than an oral airway. Nasal airways carry the significant risk that their placement may result in epistaxis.[3,10] A size 30 or 32 French nasal airway is appropriate for most adults. It can be safely placed in the conscious, semiconscious, or unconscious patient. A nasal airway can be used when an oral airway cannot be placed (e.g., braces, oral trauma, seizures, trismus, etc.). **It is imperative to perform the jaw-thrust and/or chin-lift to prevent the tongue from obstructing the patient's airway when using a nasal airway.**

The Naso-Flo (Mercury Medical, Clearwater, FL) is a newer type of nasal airway (**Figure 10-5*D***). It is available in numerous sizes with and without oxygen ports. Oxygen connects to the elbow port on the proximal end. The oxygen port allows for higher levels of inspired oxygen. Some models are available with end-tidal carbon dioxide connectors attached to the proximal end.

Insertion of a nasal airway is a rapid procedure. Choose the proper size nasal airway by placing the flared end of the airway near the tip of the patient's nose. The distal end of the nasal airway should be at the external auditory canal. Liberally apply water-soluble lubricant or anesthetic jelly to the nasal airway. Apply a

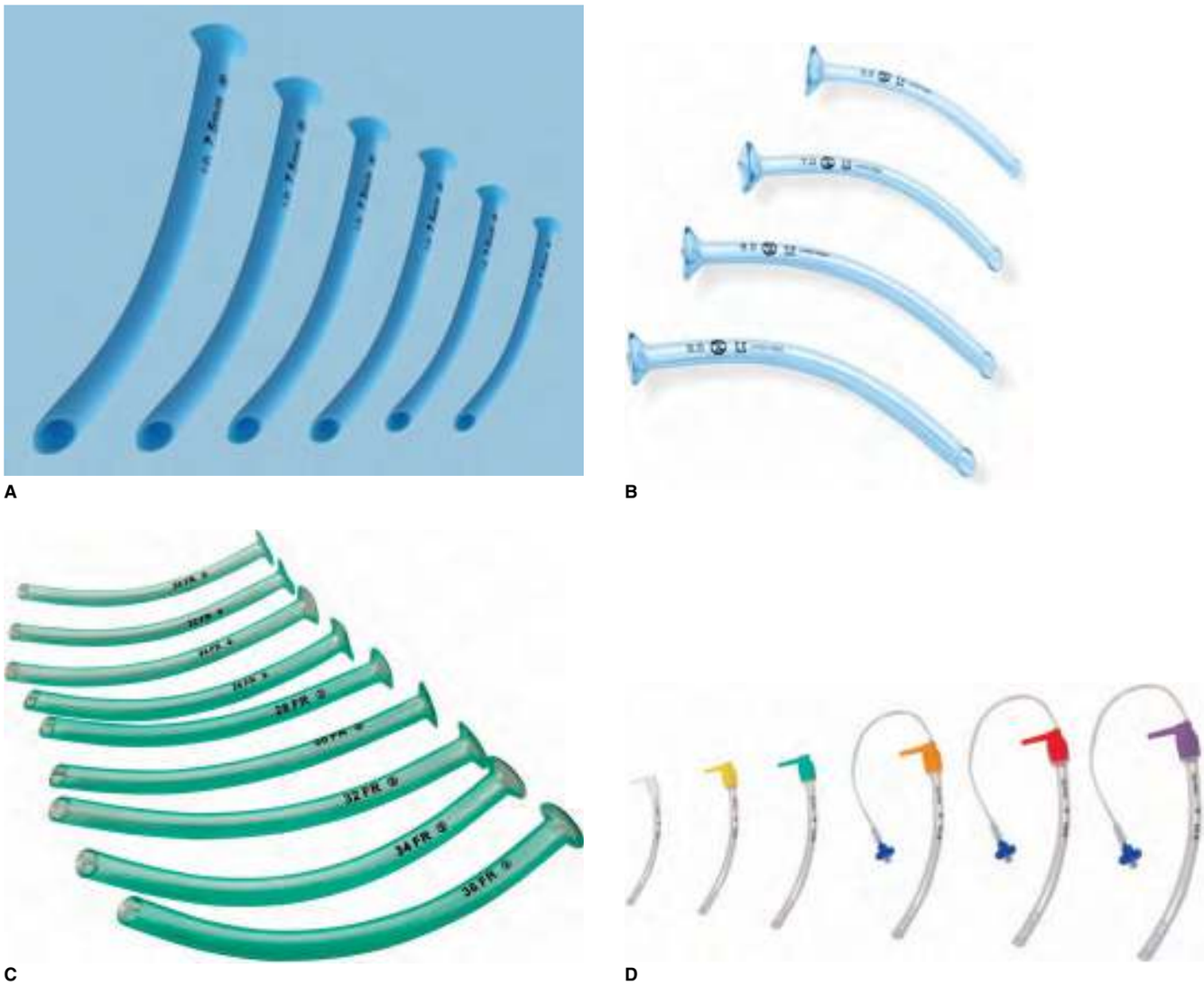


FIGURE 10-5. Examples of nasopharyngeal airways. ***A.*** Opaque blue. ***B.*** Clear. ***C.*** Opaque green. ***D.*** Naso-flow with and without an end-tidal carbon dioxide connection. (Courtesy of Mercury Medical, Clearwater, FL.)

vasoconstrictor to the patient's nasal mucosa if not contraindicated. Gently insert and advance the nasal airway with the beveled tip against the nasal septum (**Figure 10-6**). This will prevent epistaxis from the tip of the nasal airway getting caught on the inferior or middle turbinate. Insert and advance it along the floor of the nasal cavity adjacent to the septum. Continue to advance the nasal airway completely until the flared end is against the patient's nostril. Rotate the nasal airway 90° so it is concave upward. Slight rotation will often facilitate the passage of the nasal airway if resistance is encountered during insertion. Insert the nasal airway into the other nostril or use a smaller nasal airway if resistance is still encountered. Begin the administration of supplementary oxygen or positive-pressure ventilation with a bag-valve-mask device after insertion of the nasal airway.

Insertion of a nasal airway may be associated with complications. It may cause laryngospasm or vomiting if the device is too long. A nasal airway that is too long may be placed with its tip in the esophagus and result in gastric distention and subsequent aspiration. Nasal mucosal injury upon insertion can cause epistaxis and aspiration of blood.

FIGURE 10-6. Insertion of the nasopharyngeal airway.

OROPHARYNGEAL AIRWAYS

The oropharyngeal airway (i.e., OPA or oral airway) is a semicircular plastic device that holds the tongue up and away from the posterior pharyngeal wall (**Figure 10-7**). They are available in clear, color-coded, and a variety of other styles. Oral airways cause less trauma and are more easily placed than nasal airways. **Use oral airways only in unconscious patients. They may result in laryngospasm or vomiting if placed in a conscious or semiconscious patient.**[14]

Oral airways have many uses. The primary indication is to maintain a patent airway. It will prevent the patient from biting, lacerating, or occluding an endotracheal tube. It facilitates oropharyngeal suctioning by removing the tongue from the airway. It will protect the tongue from bites during seizure activity. Oral airways are sometimes used as bite blocks for inserting scopes orally.

The oral airway is often considered an accessory to the use of face mask ventilation after the face mask is difficult to ventilate the patient. This results in delays using an oral airway. Use an oral airway immediately in patients who are edentulous without their dentures, have a history of obstructive sleep apnea, have a nasal obstruction, are obese, or snore. **Use the oral airway in conjunction with the chin-lift or jaw-thrust.**

Insertion of the oral airway is a quick and simple procedure. Choose the proper size oral airway. The correct size is estimated by placing the proximal flange of the oral airway next to the patient's mouth. The distal tip should lie just above the angle of the mandible. An 8.0, 9.0, or 10.0 cm oral airway is appropriate for most adults.[15]

Clear the mouth and oropharynx of any blood, secretions, or vomit with a Yankauer suction catheter. Open the patient's jaw with the nondominant hand. Separate the patient's jaws with a "scissors-like" action of the thumb on the lower teeth and the index or middle finger on the upper teeth. Insert the oral airway curved side down (**Figure 10-8*A***). The tip will slide along the hard palate. Insert it until the plastic flange on the proximal end is at the patient's lips. Rotate the oral airway 180° so that its curve follows the curvature of the tongue.

An alternative method is to use a tongue depressor to depress the tongue and then insert the oral airway as described above. The oral airway may be inserted with the curve side upward if a tongue depressor is used (**Figure 10-8*B***). Begin administering supplementary oxygen or positive-pressure ventilation with a bag-valve-mask device after inserting the oral airway.

A newer device is the Dual-Air Adjustable Oropharyngeal Airway (NuZone Medical, Spokane, WA) (**Figure 10-9**). It was designed for single use, to provide greater patient comfort, and to improve ventilation. It comes in three sizes. The extra-large size adjusts from 90, 100, and 110 mm. The large adjusts from 70, 80, 90, and 100 mm. The pediatric size adjusts from 50, 60, and 70 mm. They all adjust to half-steps between the major markings. The device can be adjusted in the patient's mouth. The large central opening allows a suction catheter to bypass the tongue and teeth. Each is individually packaged and decreases the possibility of contaminating others. The large opening improves laminar flow and reduces the pressure required for ventilation.

Insertion of an oral airway is not a benign procedure. It can push the tongue posteriorly and further obstruct the oropharynx if the oral airway is not inserted properly. Significant lacerations can occur if the lips or tongue is caught between the teeth and the oral airway. An oral airway that is too long can force the epiglottis closed against the vocal cords and produce a complete airway obstruction. Too small of an oral airway will force the tongue against the pharynx and produce an obstruction.

FIGURE 10-7. Oropharyngeal airways. ***A.*** Noncentered, vented, and color-coded Berman. ***B.*** Centered, vented, and color-coded Berman. ***C.*** Color-coded Guedel. ***D.*** Williams oral airways.

A B

FIGURE 10-8. Insertion of the oropharyngeal airway. ***A.*** It is inserted with the curve toward the tongue and rotated 180° after insertion. ***B.*** It is inserted with the curve toward the palate with a tongue depressor to depress the tongue and facilitate insertion.

MASK VENTILATION

The likelihood of success in airway management is often predictable.[16] The ease of mask ventilation and intubation is directly related to anatomy. Anesthesiologists rely on a series of evaluations and classification criteria to help predict the success of airway management. The most widely used classification is the Mallampati classification (**Figure 9-6**).[17] This evaluation is combined with an examination of the ability to fully open the mouth, body habitus, cervical range of motion, dental structures, neck thickness, temporomandibular joint function, and thyromental distance to help predict the ease or difficulty of airway management.[16-22]

A distinction must be made between ease of mask ventilation and ease of oral endotracheal intubation. The two are often correlated but can, at times, be completely unrelated. For example, a patient with a normal body habitus who is in a cervical halo may be very easy to ventilate by mask but impossible to intubate orally via direct laryngoscopy. The obese patient with sleep apnea and a Mallampati class I airway may be very easy to intubate but virtually impossible to ventilate by mask.

Evaluate the presence of spontaneous respirations after achieving the proper positioning. Initiate positive-pressure ventilation if the patient is not breathing and there is no evidence of a foreign body. The Heimlich maneuver (Chapter 224) is the method of choice in the awake patient with complete airway obstruction due to a foreign body.[2] Remove the foreign body if it is visible and the patient has become unconscious. **Use caution to prevent forcing the object further into the airway.** Instrument removal of airway foreign bodies with an airway forceps is possible if the foreign body is visible and within reach of the forceps (Chapter 207).

FIGURE 10-9. The Dual-Air Adjustable Oropharyngeal Airway. (Courtesy of NuZone Medical, Spokane, WA.)

The options for positive-pressure ventilation include mouth-to-mouth, mouth-to-mask, and bag-valve devices (i.e., mask ventilation) once the airway is patent. The remainder of this section will review the latter, as the other two options are not used in Emergency Departments or hospitals.

The key to effective mask ventilation is ensuring a continually patent airway. This is initially achieved by placing the patient in the sniffing position coupled with a combination of chin-lift and jaw-thrust. Neglect of this key maneuver leads to excessive use of positive-pressure ventilation in an attempt to compensate for an obstructed upper airway. Improper patient positioning associated with positive-pressure ventilation will force gas into the stomach, increase intraabdominal pressure, and result in the need for ever-increasing positive pressure on the airway. Rising intraabdominal pressure will eventually make ventilation difficult, impossible, or significantly increase the risk of gastric aspiration. **Always keep in mind the importance of proper positioning and the use of an appropriately sized oral or nasal airway as an adjunct.**

Face masks are made of clear plastic and/or silicone, have a soft seal, and have an anatomic shape that conforms to the contours of the patient's face (**Figures 10-10, 10-11, and 10-12**). They are compatible with bag-valve devices, leak-proof, and lightweight; often include a color-coded ring; are single use; and are transparent to observe the patient. A soft inflatable cushion seals to the contours of the patient's face. Typical adult sizes are 3, 4, and 5. **The mask must be large enough to completely cover the nose, mouth, and chin but not so large as to allow a leak.** This allows effective ventilation through the mouth and nose. **It should not cover any part of the patient's eyes.**

There are ergonomically designed face masks in a variety of sizes. The ErgoMask (Touren Medical, Beijing, China) is one example (**Figure 10-11**). It is designed asymmetrically with the ventilation port being off-center. It was meant for efficient ventilation. The ErgoMask has contoured ridges and a colored marker for grip

A

B

FIGURE 10-10. Examples of face masks. ***A.*** Standard face masks with color-coded size rings. ***B.*** Standard face masks without color-coded size rings.

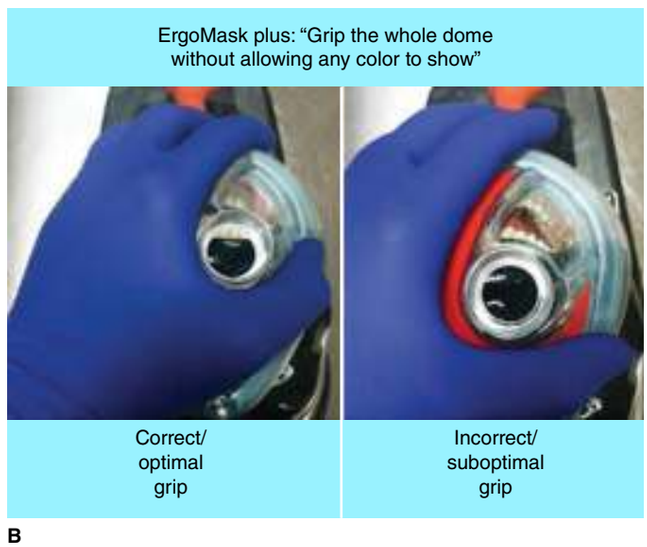
placement. The Premium ERGO Mask (SunMed, Grand Rapids, MI) is another example (**Figure 10-12**). It was designed with an ergonomic shape and finger grips to allow a secure grip. It is color-coded for size selection and available in six sizes.

There are two ways to properly hold a face mask. The one-handed technique is performed with the nondominant hand holding the mask (**Figure 10-13**). Stand at the top of the bed looking down at the patient's head. Place the little, ring, and middle fingers under the patient's mandible. Place the index finger and thumb on the bottom and top portions of the mask. **This technique allows the simultaneous jaw-thrust of the mandible and extension of the atlanto-occipital joint while applying enough downward pressure on the face mask to create an airtight seal.** An elastic head strap is a very helpful device to aid in sealing the mask tightly. The dominant hand is used to ventilate the patient through the bag-valve device.

The face mask may sometimes not form a good seal against the face. This results in poor ventilation and increased pressure to move air. Adjust the air in the cushion, apply water-soluble lubricant to the cushion, or apply a clear dressing (e.g., Tegaderm) to the patient's face.[23-25] These techniques overcome a poor seal.

A two-handed technique may be necessary in patients with facial hair and those who are edentulous, elderly, or obese.[26,27] The two-handed technique is more effective.[27,28] Two people are required to perform this technique. The Emergency Physician applies both hands to the face mask to aid in the creation of a tight seal and align the airway properly (**Figure 10-14**). Place the face mask on the patient's face. Place the index, middle, ring, and small fingers of the left hand on the body of the left side of the patient's mandible. Position the right hand similarly on the right side of the patient's mandible. Apply both thumbs to the mask and apply pressure to create a seal (**Figure 10-14**). Anteriorly elevate the mandible to perform the jaw-thrust maneuver. The two-person technique and makes it necessary to have an assistant apply positive pressure through the bag-valve device attached to the face

A



B

FIGURE 10-11. The ErgoMask. ***A.*** The mask. ***B.*** Holding the mask. (Courtesy of Tauren Medical, Beijing, China.)

FIGURE 10-12. The Premium ERGO Mask. (Courtesy of SunMed, Grand Rapids, MI.)

mask. It is preferable, when possible, to use the two-person technique because this method results in improved bag-valve-mask ventilation.[29]

A face mask can present difficulties in its use.[30,31] Difficult patient indicators include edentulousness, facial hair, male sex, Mallampati class III and IV, neck irradiation, obesity, obstructive sleep apnea, older age, and snoring. Make sure the face mask fits properly. Consider using a two-handed technique if the one-handed technique is unsuccessful.

THE NUMASK

A device can be used to assist in ventilation instead of a traditional face mask. The NuMask (NuMask Inc., Woodland Hills, CA) solves the problem of having to create an airtight face mask seal.[32,33] This device is an intraoral mask for teenagers and adults

FIGURE 10-13. The one-handed, one-person mask ventilation technique.

FIGURE 10-14. The two-handed, two-person mask ventilation technique.

that connects to a bag-valve device and eliminates the need for a face mask (**Figure 10-15*A***). It is translucent to visualize the oral contents and the patient. There is an indentation at the bottom and top that allows insertion in any direction and to protect the frenulum from damage. It is especially useful in patients in whom it is difficult to get a good face mask seal (e.g., obese, facial hair, facial trauma) or in the one-person bagging technique. The company makes a retention shield that wraps around the patient's head to secure the device and seal the patient's nostrils. This allows for easy one-person bagging.

The NuMask is simple to insert into the patient's mouth. Place the device into the patient's mouth. Ensure the soft wings sit between the patient's teeth/gums and the cheeks/lips (**Figure 10-15*B***). Use the thumb and index finger of the nondominant hand to pinch the patient's nostrils closed (**Figures 10-15*C* and *D***). Use the hand and remaining fingers to wrap around the external tube portion and to seal the patient's lips over the intraoral portion of the device (**Figures 10-15*C* and *D***). The moisture in the patient's oral cavity maintains the airtight seal. Attach the bag-valve device and begin ventilations. Remove the NuMask, insert an oral airway, replace the NuMask, and begin ventilations if ventilation is difficult.

There are numerous advantages to using the NuMask over the traditional face mask.[32-34] It is ideal for operators with small hands who have difficulty grasping and maintaining a seal with a face mask. It is easier to maintain a seal than a face mask. Ventilation is easier when only one person is available to bag and ventilate the patient. It can be used in patients in whom a good face mask seal is difficult. The device can be used in conscious and unconscious patients as it does not cause a gag reflex. The one size available will fit most teenagers and adults. A pediatric version is not available. It can be used with an oral airway (**Figure 10-15*E***).

THE BAG-VALVE DEVICE

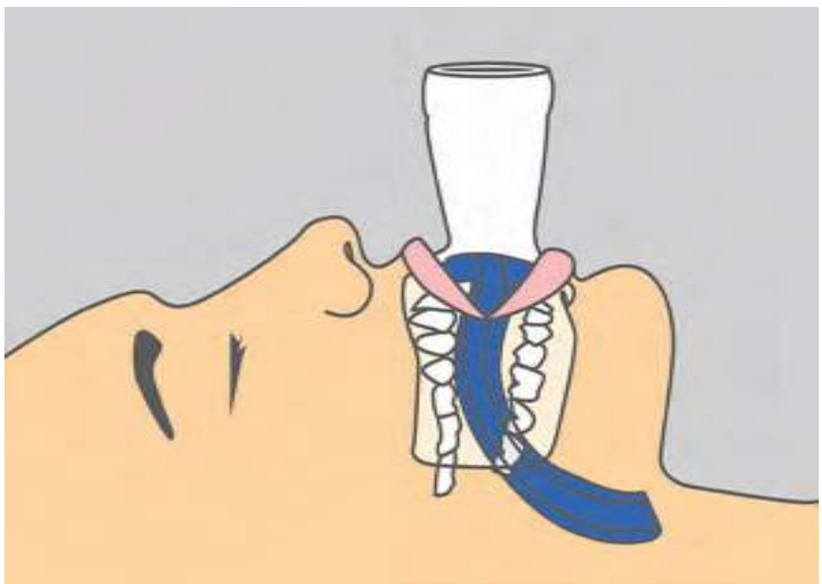
A bag-valve device is used to provide positive-pressure ventilation. It consists of a self-inflating bag connected to oxygen on one end and a one-way nonrebreathing valve on the other. They are available in several sizes depending on the age of the patient (**Figure 10-16**). The valve end is connected to the face mask or other airway device to allow one-way flow of oxygen. The other end has tubing to attach the bag to an oxygen source. This device can also force air into the esophagus and stomach and place the patient at risk for aspiration if not used properly.

A

B

C

D



E

FIGURE 10-15. The NuMASK intraoral mask. ***A.*** The device. ***B.*** The device is inserted between the patient's teeth/gums and their lips/cheeks. ***C.*** Proper hand positioning to seal the patient's nostrils and lips. ***D.*** An alternative hand positioning. ***E.*** Use with an oral airway. (Parts *A* and *E* courtesy of NuMask Inc., Woodland Hills, CA.)

It may be difficult to provide adequate ventilatory volumes through the bag-valve device attached to a face mask. This is often due to an inadequate seal of the face mask on the patient while maintaining an open airway. It can result from inadequate squeezing of the bag to generate an appropriate volume of air flow. Consider using the two-person technique to resolve these issues. Change the face mask from the standard teardrop-shaped mask to a circular-shaped face mask if ventilation is difficult in a child.

FIGURE 10-16. The bag-valve-mask device. From top to bottom: adult size, child size, and infant size.

Stand above the patient's head. Place the patient in the sniffing position. Apply a face mask. Attach the bag-valve device to the face mask and begin positive-pressure ventilation. Begin ventilations at a rate of 10 to 12 per minute or squeeze the bag every 5 to 6 seconds. Apply the jaw-thrust and/or chin-lift maneuvers if ventilation is difficult. Insert an oral or nasal airway if ventilation is still difficult. The patient requires an invasive airway device immediately if ventilation is still difficult.

The LiteSaver (Mercury Medical, Clearwater, FL) overcomes many problems associated with bag ventilation (**Figure 10-17**). The built-in red light is activated by pulling a tab. It blinks every 6 seconds or 10 times a minute to administer a breath and prevent hyperventilation and hypoventilation. Breaths at the blinking light allow sufficient expiration to avoid breath stacking. The manometer can show how much pressure is being applied with each breath. Pressures lower than 20 to 25 cmH_2O decrease aspiration. Having the manometer and timing light built in to the bag eliminates the need for additional equipment.

A B

FIGURE 10-17. The LiteSaver bag-valve device. ***A.*** Ready to use. ***B.*** The tab pulled and the light blinking red when the device is activated. (Courtesy of Mercury Medical, Clearwater, FL.)

The American Society of Anesthesiologists has published an algorithm to facilitate decision making in the face of airway management problems. A detailed discussion of the various intubation options is contained in later chapters.

IMPEDANCE THRESHOLD DEVICES

The ResQGARD and ResQPOD (Advanced Circulatory Systems Inc., Roseville, MN) were designed as adjuncts to ventilation (**Figure 10-18**).[35,36] They increase diastolic and systolic blood pressures while improving cerebral blood flow. Both devices can be used with endotracheal tubes, airway devices, or a standard face mask.

The ResQGARD is a noninvasive device to improve blood pressure in hypotensive patients (e.g., blood loss, dehydration, overdose, and sepsis). It is used in spontaneously breathing patients and attaches to the face mask. It creates a resistance when the patient inhales to enhance the vacuum in the chest. The vacuum draws blood back to the heart, increases preload, improves cardiac output, and lowers intracranial pressure. It results in improved organ blood flow in addition to the increased blood pressure.

The ResQPOD is used during cardiopulmonary resuscitation. It impedes airflow into the lungs until the threshold of –10 cmH_2O is reached but not out of the lungs during exhalation. It increases the negative intrathoracic pressure to enhance circulation. The integrated timing light flashes at a rate of 10 times per minute to as a reminder to administer ventilations only with the lights.

PEDIATRIC CONSIDERATIONS

Significant differences exist between the adult and pediatric airway (Chapter 9 and **Table 10-2**).[37] The ratio of head to body size is greater in infants and young children. Care must be taken to achieve proper positioning for optimal airway angulation. A towel may be placed under an infant's shoulders, while younger children may be optimally positioned while lying flat on the stretcher. Older children may require a towel under the head to achieve ideal positioning.[37,38] Loss of tone in the muscles supporting and protecting the upper airway may still result in an airway obstruction despite proper head positioning. This may be corrected by the use of a chin-lift, jaw-thrust, nasal airway, or oral airway.

Infants and children have a higher minute ventilation and cardiac output than adults and higher basal oxygen consumption. Infants and children have a lower functional residual capacity, which leads to oxygen desaturation more rapidly than in adults when apnea occurs. Hypoxic bradycardia can be prevented or reversed when oxygenation and ventilation are provided in an effective and efficient fashion.[38]

Properly sized equipment remains crucial to the success of airway management and can be addressed by the use of the Broselow tape. It should be noted that there is insufficient evidence for or against the routine use of supraglottic or extraglottic airway devices during pediatric cardiac arrest. A meta-analysis of in-hospital use of laryngeal mask airways cited aspiration as an infrequent complication in pediatric patients. Their use is an acceptable adjunct in the hands of experienced providers but is associated with a higher incidence of other complications in young children (e.g., failed insertion, need for repositioning, and poor seals).[39] Nasal airways are

FIGURE 10-18. Impedance threshold devices. ***A.*** The ResQGARD. ***B.*** The ResQPOD. (Courtesy of Advanced Circulatory Systems Inc., Roseville, MN.)

underused in pediatric patients. Oral airways are often not helpful but do not harm the patient if the right size is used properly.

COMPLICATIONS

The most serious complication of basic airway management is aspiration of gastric contents. Positive pressure can force air into the stomach and result in gastric distension. This can cause regurgitation and aspiration. The aspiration of acidic gastric contents can result in an acute chemical pneumonitis. This phenomenon is known as the Mendelson syndrome and has an associated 50% mortality.[40] It is a particularly significant risk when airway management is needed emergently or routinely in the diabetic, obese, pregnant, or trauma patient. Incorrectly sized oral airways can force the tongue back and cause an airway obstruction.

Less serious complications include soft tissue trauma to the eyelids, lips, oral cavity, and tongue. Tooth fractures or avulsions are uncommon but possible injuries. A dermatitis or allergic reaction to the plastic material is rarely seen. Facial nerve dysfunction due to pressure effects of the mask are transient. Corneal abrasions, conjunctival chemosis, and increased intraocular pressure are common with masks that are too large. Epistaxis can result from a nasal airway.[41] Incorrect insertion of a nasal airway can damage the turbinates.[41] This is troublesome and can sometimes result in blood aspiration. Lifting of the mandible in difficult face mask ventilation can result in bruising, pain, and mandibular dislocations.

Traumatic injuries can result in complications from using a face mask. Pharyngeal mucosal injuries can cause subcutaneous emphysema. Patients with basilar skull fractures can experience a pneumocephalus with the use of positive-pressure ventilation.

TABLE 10-2 Characteristics of Pediatric Airways

Bigger epiglottis
Different dimensions
Laryngospasm more prone to develop
Larynx more cephalad
Longer palates
More reactive airways
Narrowest at cricoid cartilage
Relatively larger head in infants and young children
Relatively larger tongue in children
Shorter jaw
Tissues more easily injured
Vocal cords situated more anteriorly

SUMMARY

Basic airway management is a fundamental skill that must be mastered by all Emergency Physicians. It is usually possible to prevent hypoxia and hypercarbia in the apneic patient with an oxygen source, a means to deliver positive-pressure ventilation, attention to detail in positioning, and airway adjuncts. The ultimate measure of the efficacy of oxygenation and ventilation is a normal $PaCO_2$ and PaO_2. The various methods of securing the airway are discussed elsewhere in this text.

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11 Noninvasive Airway Management

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INTRODUCTION

The term noninvasive ventilation (NIV) refers to delivering positive-pressure ventilation through an interface (i.e., face mask, nasal mask, or nasal plugs) rather than intubating the trachea.[1,2] It may be used to prevent acute respiratory failure (ARF) as a prophylactic treatment, to treat ARF, or as a curative treatment to avoid reintubation.[3,4] There are different types or modes of NIV. The most frequently used are continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BIPAP). CPAP is a method to deliver constant positive airway pressure during the inspiratory and expiratory phase of breathing.[5] BIPAP refers to the association of two positive airway pressures (i.e., pressure support ventilation [PSV] during inspiration and lesser pressure value during expiration).[5] The pressure applied during expiration is commonly incorrectly referred to as positive end expiratory pressure (PEEP). The correct term is expiratory positive airway pressure (EPAP), or the pressure delivered throughout the expiratory phase.

Some Emergency Physicians consider high-flow nasal cannula (HFNC) as a method to deliver NIV. NIV should be well known to all Emergency Physicians caring for patients with acute respiratory failure.[6] It is used in the Emergency Department for NIV and preoxygenation prior to intubation.[7-12] It is used as NIV by some prehospital providers.[13-17] This chapter discusses CPAP and BIPAP in detail with some reference to HFNC. Other methods to deliver oxygen therapy are discussed briefly at the end of this chapter.

ANATOMY AND PATHOPHYSIOLOGY

CPAP works mainly by increasing intrathoracic pressure. It prevents airway and alveolar collapse. CPAP leads to decreased atelectasis and maintaining functional residual capacity (FRC). CPAP reduces left ventricular afterload, which may lead to an increase in cardiac output. CPAP helps to decrease the work of breathing in chronic obstructive airway disease by decreasing the inspiratory threshold load caused by intrinsic PEEP.[5] CPAP may improve gas exchange by improving the ventilation-perfusion ratio and by increasing intraalveolar oxygen partial pressure. The tidal volume is dependent on respiratory muscles without unloading of inspiratory muscles.[6] The increase in intrathoracic pressure can result in decreased venous return to the heart, decreasing left-sided output, and decreased blood pressure.[18]

The PSV portion of BIPAP is triggered by the patient's inspiratory effort. This trigger will induce positive-pressure support that helps to unload the inspiratory muscles and decrease work of breathing. Tidal volume depends on inspiratory muscle effort, respiratory compliance, and pressure support provided by the ventilator. PEEP

FIGURE 11-1. An example of the high-flow nasal cannula (HFNC).

works during expiration by the same mechanisms that CPAP works and achieves the same goals.[19] BIPAP provides pump function in addition to improving gas exchange.

HFNC is used to provide oxygen therapy (**Figure 11-1**).[20-24] It has humidification built into the circuit and oxygen flow of 10 to 60 L/min. HFNC looks like a regular nasal cannula with bigger prongs. It provides a small positive pressure. HFNC helps to reduce the dead space by increasing the FIO_2 and displacing CO_2 at the upper airway segments.[25] The HFNC decreases heat loss, decreases moisture loss, provides PEEP, and improves oxygenation.[25] The patient controls the CPAP and PEEP by opening their mouth. HFNC allows the patient to eat, suction, and talk. It is often used in procedural sedation and as a bridge to preoxygenate prior to intubation.

INDICATIONS

NIV was considered unsafe in the past. Many Emergency Physicians thought it was more prudent to intubate the patient than use NIV.[4,26] This includes infants and children.[27] Emergency Physicians have been using NIV successfully for multiple indications over the past 20 years. The widening indications of NIV have led to improvement and advancement of ventilation techniques and equipment.

NIV is used as a prophylaxis or a curative treatment for ARF. NIV led to a decreased need for intubation in patients with chronic obstructive pulmonary disease (COPD).[28,29] NIV also decreases mortality in patients with COPD.[28,29] Use NIV as the first line of ventilatory support for patients with severe COPD exacerbation based on clinical practice guidelines.[30,31] NIV significantly decreased intubation rates and mortality in cardiogenic pulmonary edema. CPAP and BIPAP are equal effective treatment for these patients.[30] NIV reduces left ventricular transmural pressure and increases cardiac output. NIV has been used successfully in some cases of hypoxemic respiratory failure for reasons other than cardiogenic pulmonary edema.[32,33] However, some studies showed increases in intubation rates and hospital mortality rates.[34] A reason for this conflicting data may be related to the underlying cause of hypoxemic respiratory failure.

Use of NIV was beneficial in patients with hypoxemic respiratory failure resulting from pneumonia and respiratory failure after abdominal surgery.[33,35] However, it failed to show additional benefit in cases of acute respiratory distress syndrome (ARDS).[36]

Several studies have been conducted to investigate the benefit of NIV in severe asthmatic exacerbations.[37-39] These studies showed that NIV added to conventional therapy led to decreases in intubation rates, decreases in the mean bronchodilator therapy dose, and reductions in the need for hospitalization. However, the number of patients enrolled in these studies was small, and the recommendations concerning use of NIV in severe asthma remain controversial.[40]

NIV used in patients with obesity hypoventilation syndrome admitted with acute respiratory failure was as effective as NIV in COPD exacerbations.[41] NIV has been used in outpatient settings for the treatment of obstructive sleep apnea (OSA) and obesity hypoventilation syndrome.

CONTRAINDICATIONS

NIV is contraindicated in agitated patients; patients with cardiac arrest, copious secretions, facial surgery, facial trauma, the inability to protect the airway, multiple organ failure, severe hematemesis, or severe hemoptysis; patients who refuse NIV or are uncooperative; and patients who are vomiting. Apnea or respiratory arrest is an absolute contraindication for NIV because the patient should completely control the respiration in CPAP or trigger the ventilator in BIPAP. Avoid NIV in patients at high risk of aspiration or who are not able to protect the airway. Other contraindications include an elevated PCO_2, hemodynamic instability, hypotension, and lack of trained staff.

Significantly altered mental status is considered an absolute contraindication except in patients with hypercapnic encephalopathy.[42] Closely monitor patients with impaired consciousness due to hypercapnia being treated with NIV and intubate if further deterioration or no improvement is noted.

Avoid NIV in respiratory acidosis, severe hypoxemia, and shock because most patients will fail NIV. The observed mortality was higher than the predicted mortality scores.[36]

Recent esophageal suturing or anastomosis surgery is a relative contraindication because high positive pressure may lead to suture disruption and leakage.[43] It is preferable to use CPAP over BIPAP in these cases or to use low insufflation pressure below 6 to 8 cmH_2O for PSV.[3]

EQUIPMENT

- Interface (see below)
- Ventilator
- Pulse oximeter
- Capnogram monitor
- Cardiac monitor
- Noninvasive blood pressure monitor
- Face masks, various sizes
- Bag-valve device
- Endotracheal tubes, various sizes
- Oxygen source, tubing, and regulator
- Advanced life support (ALS) medications
- Advanced airway equipment
- Surgical airway equipment
- Suction system

The interface is the main difference that distinguishes NIV from invasive ventilation. Available interfaces include oronasal masks, nasal masks, nasal pillows, mouth pieces, hybrid oral interface–nasal pillows, total face masks, and helmets (**Figure 11-2**).

Choosing the interface is an important step. Make every effort to try different types and sizes of interfaces to achieve optimal therapeutic results with the smallest leak possible. No difference was found between different types of interfaces in regard to minute

FIGURE 11-2. Commonly used CPAP interfaces. ***A.*** Oronasal mask. ***B.*** Nasal mask. ***C.*** Nasal pillow. ***D.*** Hybrid oral interface–nasal pillow. ***E.*** Total face mask. ***F.*** The Boussignac CPAP System.

ventilation, PCO_2, and work of breathing.[44] The ideal interface will be the one with the lowest leakage and that provides an adequate seal with minimal pressure on the face, is well tolerated by the patient, and is easy to secure and easy to remove. The interfaces are transparent to aid in identifying any regurgitation or vomiting. Most commercially available interfaces have anti-asphyxia valves that prevent asphyxiation should any ventilator malfunction occur. Use a swivel adaptor if the patient has an orogastric or nasogastric tube (**Figure 11-3**) or special endoscopy mask to minimize the leak. Start with an oronasal mask in the Emergency Department. Have different types of interfaces immediately available for use if the patient does not tolerate the oronasal mask.

Ventilators are being used in the Emergency Department to provide NIV. The different NIV modes were added to the new-generation ventilators. Older generation ventilators were only able to provide invasive ventilation modes. Ventilators detect and compensate for any leakage. Ventilators can be adjusted to provide different oxygen concentrations (i.e., FIO_2) up to 100% oxygen. An alarm system helps to detect asynchrony and leakage. Different types of portable ventilators are used to provide CPAP for OSA.

Commonly used ventilator modes include CPAP and BIPAP. Proportional assist ventilation (PAV) and neurally adjusted ventilatory assist (NAVA) are new modes created to improve patient-ventilator synchrony.[45]

CPAP can be delivered by the Boussignac CPAP System (Vygon; **Figure 11-2*F***). This does not require a special ventilator. The Boussignac CPAP System uses a high-flow gas source (e.g., 30 L/min O_2 flowmeter) to create a virtual valve through turbulent flow. The Emergency Physician can build up CPAP into the system by adjusting the flow. The advantage of this method is having an effective, lightweight, and economic system.[46]

FIGURE 11-3. A swivel adaptor can be used to fit a nasogastric tube for a patient on CPAP.

PATIENT PREPARATION

The decision to use NIV implies the patient does not need emergent intubation. Most patients will need urgent management. Use the available time wisely and efficiently. **Delaying the initiation of NIV may lead to deterioration and increase the possibility of failure.**[47] Perform a quick history and physical examination to detect whether the patient will benefit from using NIV and to exclude any contraindications. Inquire about the NPO (nothing by mouth) status. **Assess the airway to predict a possible difficult intubation.** Apply cardiac monitoring, noninvasive blood pressure cuff, and pulse oximetry. Obtain and secure a well-functioning intravenous catheter.

NIV requires the full cooperation from the patient with the exception of hypercapnic encephalopathy. Thoroughly explain the process and expectations to help the patient better tolerate the mask and positive airway pressure. Obtain consent from the patient for NIV and possible invasive ventilation in case of NIV failure. Clarify advanced directives. Place the patient seated in a 30° to 90° position guided by the patient comfort. Leave any dentures in place to help with fitting the interface.

TECHNIQUE

Apply supplemental oxygen (e.g., through a nasal cannula, face mask, or nonrebreather mask) until replaced by the NIV interface. Set the ventilator to the correct settings and be ready to connect to the interface. Select the mode and adjust the initial settings (**Table 11-1**). Start with low pressure to help the patient tolerate and synchronize with the ventilator. Consider starting with CPAP/PEEP of 3 to 5 mmHg and PSV of 3 to 5 mmHg if using BIPAP. Start with an FIO_2 of 50% to 60%. Adjust the FIO_2 after connecting the ventilator to keep the saturation within an acceptable range. This will vary between patients depending on their baseline. A value of > 92% is commonly used. Start with initial trigger of –1 to –2 L/min or –1 to –2 cmH_2O, and adjust it to reach the lowest level that prevents auto-triggering.

Ensure the patient is positioned properly. Remove the supplemental oxygen device and replace it with the NIV interface. The patient may know which interface fits the best. Apply the interface without connecting the ventilator. Avoid excessive pressure or strap tension on the patient. Encourage the patient to hold the interface while applying it and to take a few breathes before connecting the ventilator. Connect the ventilator and allow the patient to synchronize with the ventilator. Gradually increase the pressure settings (i.e., CPAP/PEEP and PSV) as tolerated by the patient to achieve an adequate tidal volume of 6 to 10 mL/kg with good synchronization and without major leak. **Never exceed a total pressure (i.e., PSV + PEEP) of 25 cmH_2O because this may lead to gastric insufflation.** It is best to connect a nasogastric tube, if the patient has one in place, to a bag rather than suction to detect gastric insufflation.

The success of NIV depends mainly on improving synchronization and minimizing the leak. Frequently check the device. Keep reassuring the patient. Continue to adjust the interface position and pressure setting as required. Monitor the patient for the need for orotracheal intubation.

TABLE 11-1 Initial Ventilator Settings for NIV

Ventilator mode	**Respiratory rate**
Assist control (AC)	8–14 breaths/min
Breath type	Beware of settings too high
Pressure	Patient must be breathing on their own
IPAP	**Inspiratory time**
12–20 cmH_2O	Adjust to patient
Titrate to respiratory rate	**Oxygen concentration**
Titrate to work of breathing	FIO_2 room air to 100%
Titrate 2 cmH_2O every 5 minutes	Adjust to pulse oximetry > 92%
EPAP	
6–12 cmH_2O	
Start with 6–8 cmH_2O for asthma	
Start with 6–8 cmH_2O for chronic obstructive pulmonary disease	
Start with used CPAP for obstructive sleep apnea	
Start with 10–12 cmH_2O for pulmonary edema	
Titrate 2 cmH_2O every 5 minutes	

ASSESSMENT

NIV requires close monitoring and assessment of the patient. Continuous cardiac monitoring and pulse oximetry are mandatory to assess proper oxygenation. Use capnography to assess ventilation. Obtain an arterial blood gas (ABG) within 1 to 2 hours of NIV initiation to assess the patient's response. Improvement of pH, PO_2, and/or PCO_2 can be used as a sign to predict NIV success.[48] **The early identification of asynchrony is crucial since proper management will decrease failure of NIV.** Asynchrony can be related to the underlying disease or the presence of a leak.[49]

Steps can be taken to minimize the leak. This includes adjusting the interface position, switching between different interfaces, adjusting the ventilator settings, and gradually increasing the pressure support from the initial settings. Some ventilators provide a leakage compensation feature. PAV mode was used to improve patient tolerance and synchrony with the ventilator. It is not superior to intubation rates or overall mortality.[50,51]

One of the most important points in management of NIV is rapid recognition of failure.[52] The reported failure rate varies from 5% up to 40%.[53] It is difficult to predict which patients will fail NIV. Failure risk factors may encourage the Emergency Physician to closely monitor the patient to identify failure early (**Table 11-2**).[54,55] A patient is considered to have failed NIV if the ABG revealed worsening of the pH, PO_2, and/or PCO_2. **Terminate the NIV if the patient is exhibiting worsening of hemodynamics, worsening of agitation, or worsening of consciousness or is unable to clear secretions, unable to protect their airway, or unable to tolerate NIV despite all steps to help with synchronization, and perform immediate intubation.**[48]

AFTERCARE

Take every possible step to prevent pressure ulcers from excessive pressure by the interface. Multiple studies suggest that applying humidification may improve patient comfort and increase

TABLE 11-2 The Risk Factors for Failure of NIV

Age > 40 years	Initial high CO_2
Agitation	Initial high FIO_2 requirement
Air leak	Low patient PO_2/FIO_2
Asynchrony with ventilator	No improvement after 2 hours of NIV
Decreased consciousness (e.g., Glasgow coma scale score < 11)	pH < 7.25
	Respiratory distress
Diagnosis of acute respiratory distress syndrome (ARDS)	Severe tachycardia
	Severe tachypnea > 35 breaths/min
Edentulous	Underlying ARDS
Elevating CO_2	Underlying immunocompromise
Elevating FIO_2 requirements	Underlying oncologic problem
Excessive secretions	Underlying sepsis
Failure to wean FIO_2	Ventilator intolerance
Hemodynamic instability	

tolerance by decreasing dryness of the upper airway.[56] This is controversial.

The use of sedation during NIV is poorly studied. Some Emergency Physicians advocate administering sedation to improve patient tolerance and NIV success. Others are concerned that sedation may worsen the level of consciousness or depress respiration. Various agents have been used for sedation during NIV (e.g., benzodiazepines, dexmedetomidine, ketamine, and opioids).[57-59] Dexmedetomidine has the advantage of having a minimal effect on the respiratory center. Combination therapy with several sedatives may have deleterious effects and may lead to NIV failure.[60] **The decision to use sedation must be individualized for each patient. Use a single agent and titrate it cautiously with close monitoring of the level of consciousness and respiratory parameters.**

Where and when to admit the patient is a very important question once NIV is initiated. NIV is typically initiated in the Emergency Department followed by transfer of the patient to an intensive care unit or step-down unit. It is crucial to have the proper monitors and trained personnel available 24 hours a day.[61] Admit and transfer the patient after the initial assessment of NIV success and stabilization of the patient's condition. Transfer the patient with NIV applied and with monitors. NIV is usually applied in 60- to 90-minute durations at 2- to 3-hour intervals. Weaning is performed by increasing the disconnection time, decreasing the ventilator positive pressure parameters, or a combination of both.

COMPLICATIONS

NIV is generally safe, and serious complications are rare. Minor complications include interface discomfort or a minor leak. This can be resolved by adjusting the interface, minimizing the strap tension while maintaining a minimal leak, or switching interfaces. Dryness of the upper airway can cause discomfort. Dryness is managed by adding humidification. Other minor complications include eye dryness and minor gastric distension.

Serious complications may have a detrimental effect on the patient. Nasal bridge skin ulceration is a result of excessive pressure while trying to minimize leaks. Steps to prevent this include decreasing the strap tension, using masks with soft cushions on the nose, and using the interface provided with a forehead cushion. Switching masks (e.g., nasal pillows or total face mask) will minimize the possibility of nasal ulceration. The application of artificial skin (e.g., Duoderm) is an additional step taken by some Emergency Physicians to protect against nasal ulceration. Increased intrathoracic pressure can result in hypotension, hemodynamic compromise, and deterioration. Attempt to lower the pressure to resolve this.

Gastric distension results from excessive pressure support. It may lead to vomiting or regurgitation. **The total pressure support (i.e., PSV + PEEP) should not exceed 25 cmH_2O.** Connect any nasogastric tube to a bag to recognize gastric distention.

The inability to recognize NIV therapy failure may delay necessary intubation and lead to increased morbidity and mortality.[62] Guard against delays in intubation by identifying patients who would benefit from NIV, excluding the patients with contraindications to NIV, and closely monitoring to identify the early signs of NIV failure.

OTHER NONINVASIVE DEVICES TO DELIVER OXYGEN

A wide variety of devices are being used to provide supplemental oxygen (i.e., FIO_2 > 21%). They can be used as a primary oxygen treatment or as a transient bridging measure until more advanced airway management is prepared.

FIGURE 11-4. An example of the nasal cannula.

NASAL CANNULA

The nasal cannula is one of the most widely used devices to deliver supplemental oxygen (**Figure 11-4**). Adjust the oxygen flow between 1 and 6 L/min. There is an increase of 4% in FIO_2 for each L/min of oxygen flow. The delivered FIO_2 ranges from 25% for 1 L/min to 45% for 6 L/min. Further increases in the oxygen flow will have no additional effect on FIO_2. The FIO_2 delivered by nasal cannula is affected by the minute ventilation so that an increase in minute ventilation will result in a decrease in the FIO_2. Nasal dryness may ensue and lead to patient discomfort if the flow increases beyond 4 L/min. This can be alleviated by adding humidification to the oxygen.

SIMPLE FACE MASK

The face mask is another frequently used device to deliver oxygen (**Figure 11-5**). The FIO_2 will increase by 4% for each L/min of oxygen flow, similar to a nasal cannula. The minimum oxygen flow should be 5 L/min to prevent rebreathing of exhaled CO_2. Adjust the oxygen flow to between 5 and 10 L/min. The FIO_2 delivered by a simple oxygen mask ranges from 40% to 60%. The increase in minute ventilation will decrease the delivered FIO_2. A face mask is less likely to cause nasal mucosa dryness compared to a nasal cannula.

VENTURI MASK (AIR-ENTRAINMENT MASK)

This device depends on the Venturi effect to entrain air that mixes with oxygen to deliver a fixed FIO_2 (**Figure 11-6**). The Venturi effect occurs when a fluid or gas flows through a narrowing, leading to an increased velocity and creation of negative pressure.

FIGURE 11-5. An example of the simple face mask.

FIGURE 11-6. The Venturi mask. The indicator identifies how much oxygen flow is being delivered.

FIGURE 11-7. An example of the nonrebreather face mask.

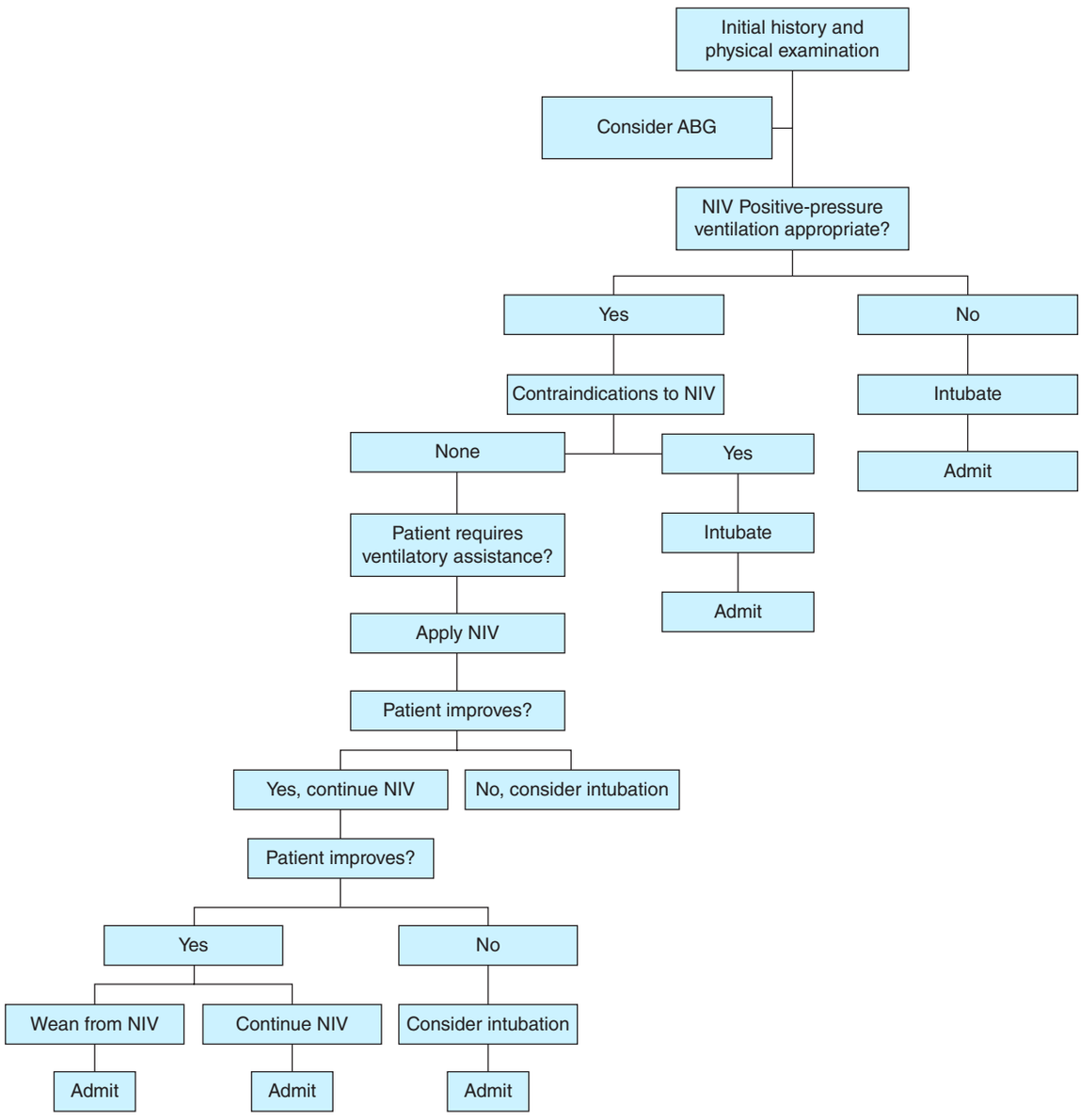

FIGURE 11-8. An algorithm for NIV. ABG, arterial blood gas;

The negative pressure will help to entrain air. The delivered FIO_2 ranges from 24% to 50%. The Venturi device will indicate how much oxygen flow is needed to deliver certain FIO_2 concentrations (**Figure 11-6**). FIO_2 delivered by a Venturi mask is not affected by minute ventilation and is usually used when a precise FIO_2 is desired.

NONREBREATHING FACE MASK

A nonrebreather mask is a face mask that contains a 1-L reservoir bag (**Figure 11-7**). There are two valves in a nonrebreather mask. One prevents entrainment of room air. The other prevents mixing the oxygen in the reservoir bag with exhaled air. Adjust the oxygen flow to between 8 and 15 L/min to ensure filling of the reservoir bag and prevent the bag from collapsing during inspiration. The delivered FIO_2 can range from 60% to 90%.

PARTIAL REBREATHING FACE MASK

The design of a partial rebreathing mask is similar to the nonrebreathing mask except it lacks the valve between the mask and the reservoir bag. The first portion of the exhaled air (i.e., 150 mL that represents the dead space) enters the reservoir bag. The dead space contains very little CO_2. The delivered FIO_2 can reach up to 80%. Oxygen flow ranges between 8 and 15 L/min with a target to prevent the reservoir bag from collapsing during inspiration.

SUMMARY

NIV is an effective management that has been used in a wide variety of acute clinical conditions to treat or prevent acute respiratory failure (**Figure 11-8**). It is used in the resuscitation of the critically ill patient. The benefit of avoiding endotracheal intubation encouraged many Emergency Physicians to try NIV as a first line of ventilatory support. Be familiar with the contraindications to using NIV. Monitor the signs of NIV failure. Delaying intubation when it is indicated may lead to increased morbidity and mortality. Emergency Physicians should be aware of the FIO_2 values that can be delivered by the supplemental oxygen devices. The use of NIV may prevent intubation, admission to the intensive care unit, the use of intensive care unit resources, and increased costs to the patient. Consider the use of NIV by a helmet interface in end-of-life care (**Figure 11-9**).

FIGURE 11-9. An example of the NIV helmet.

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12 Pharmacologic Adjuncts to Intubation

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INTRODUCTION

Reserve oral endotracheal intubation without pharmacologic assistance for the unresponsive and apneic patient. Intubate unconscious patients capable of resisting laryngoscopy or those with spontaneous respiratory effort with the assistance of pharmacologic adjuncts. Rapid sequence intubation (Chapter 16) optimizes conditions while minimizing the risk of aspiration. It can be performed with a high rate of success and minimal complications.[1] Rapid sequence intubation requires the use of several pharmacologic adjuncts (**Tables 12-1, 12-2, and 12-3**). This includes a potent anesthetic agent to induce unconsciousness and a neuromuscular blocking agent to produce paralysis.

TABLE 12-1 Recommended Anesthetic Doses of Pharmacologic Agents Used for Rapid Sequence Intubation

Medication	Adult dose (mg/kg)	Pediatric dose (mg/kg)	Onset (sec)	Duration (min)
Dexmedetomidine	0.001	0.0005–0.0010	15–45	12–20
Etomidate	0.2–0.3	0.2–0.3	15–45	3–12
Fentanyl	0.005–0.015	0.005–0.015	15–45	30–60
Ketamine	1–2	1–3	45–60	10–20
Methohexital	1–3	1–2	< 30	5–10
Midazolam	0.2–0.4	0.5–1.0	30–90	10–30
Propofol	1.5–2.5	2.5–3.5	15–45	5–10
Thiopental	2–5	2–6	< 30	5–10

TABLE 12-2 Recommended NMB Agents for Intubation and Rapid Sequence Intubation

Medication	Histamine release	Active metabolites	Adult dose (mg/kg)	Pediatric dose (mg/kg)	Time to maximum onset (min)	Duration (min)	Infusion (mcg/kg-min)	Elimination	Action
Atracurium	Yes	No	0.5	0.5	3	20–35	5–20	Hoffman, 5–10% renal	Intermediate
Pancuronium	No	Yes	0.05–0.1	0.07–0.10	2–3	40–100	0.8–1.7	40–70% renal 15% hepatic	Long
Rocuronium	No	No	0.6	1.0	4–6	15–85	8–12	33% renal < 75% hepatic	Intermediate
Rocuronium for RSI	No	No	0.9–1.2	1.0–1.2	1.0–1.5	30–110	N/A	33% renal < 75% hepatic	Intermediate
Succinylcholine	No	No	0.6–1.0	1–2	1	2–3	N/A	Plasma cholinesterase	Short
Succinylcholine for RSI	No	No	1.5	2	1	3–5	N/A	Plasma cholinesterase	Short
Vecuronium	No	Yes	0.1	0.05–0.10	3	30–40	0.8–1.7	10–50% renal 35–50% hepatic	Intermediate
Vecuronium for RSI	No	Yes	0.3–0.5	0.3–0.5	1	45–60	N/A	10–50% renal 35–50% hepatic	Intermediate

N/A, not applicable; RSI, rapid sequence intubation.

INDUCTION AGENTS

The ideal induction agent has an extremely rapid onset of action, produces predictable deep anesthesia, has a short duration of action, and has no adverse effects.[2] Such an agent does not yet exist. There are at least six drugs that can safely be used for induction of anesthesia and intubation (e.g., etomidate, fentanyl, ketamine, methohexital, midazolam, propofol, and thiopental). Midazolam and fentanyl may be used alone or in conjunction with the above agents.[2] **The decision as to which induction agent is the most suitable is largely dependent on the Emergency Physician's experience and their understanding of each drug's properties.** Each of these drugs is briefly detailed as to its pharmacokinetics, mechanism of action, pharmacodynamics, administration, and adverse effects.

TABLE 12-3 Drug Interactions of NMB Agents

Causes NMB prolongation
Aminoglycosides
Beta-blockers
Calcium channel blockers
Clindamycin
Corticosteroids
Cyclosporine
Enflurane
Furosemide
Halothane
Isoflurane
Lithium
Local anesthetics
Magnesium
Methylxanthines
Methoxyflurane
Nitrous oxide
Potassium
Procainamide
Quinidine
Tetracycline
Vancomycin
Causes NMB resistance
Caffeine
Calcium
Carbamazepine
Phenytoin
Ranitidine
Theophylline

BARBITURATES

Barbiturates have been a mainstay in the induction of anesthesia for more than 50 years. **Barbiturates rapidly produce sedation and hypnosis in a dose-dependent fashion.** They are less expensive than many of the newer induction agents.[2] The most commonly used barbiturates are thiopental (Pentothal) and methohexital (Brevital) because of their high potency, rapid onset, and short duration of action.

PHARMACOKINETICS

These ultra-short-acting barbiturates can produce effects in one arm-brain circulation time or less than 30 seconds when injected by the intravenous (IV) route.[3] The onset of central nervous system (CNS) depression is primarily due to the rapid distribution of thiopental and methohexital to the well-perfused and low-volume central compartment (i.e., mainly the brain and liver). Approximately 15% of these lipid-soluble drugs remain unbound and free to diffuse across the blood-brain barrier at high initial concentrations. Brain levels peak in about 1 minute. The duration of a single dose of thiopental is approximately 5 to 10 minutes, while an equipotent dose of methohexital lasts approximately 4 to 6 minutes.[4] The short duration of action is due to the redistribution of the drugs from the small-volume central compartment to the large-volume peripheral compartment, which is predominantly lean muscle.

Thiopental and methohexital are metabolized in the liver and excreted by the kidneys. Methohexital has a substantially higher hepatic extraction ratio, which may account for its shorter duration of action. Clearance of the drugs has little to do with the cessation of CNS effects in single or multiple small doses. The concentration in the peripheral tissue approaches the plasma concentration, and the rate of redistribution is greatly diminished if barbiturates are given in multiple high doses or in an infusion.[5] This prolongs their anesthetic effects.[5]

MECHANISM OF ACTION

Barbiturates, along with other common IV anesthetic agents, are postulated to act on the γ-aminobutyric acid (GABA) receptor complex. These drugs work on the $GABA_A$ receptor.[6] **GABA is the**

principal inhibitory neurotransmitter in the CNS. The GABA receptor complex forms a transmembrane chloride channel when activated. The influx of chloride ion causes hyperpolarization and functional inhibition of the postsynaptic neurons. Barbiturates can act on the GABA receptor in two ways. Barbiturates at lower doses may potentiate the action of endogenously produced GABA by decreasing the rate of its dissociation from the receptor complex. Barbiturates at higher doses may directly activate the chloride ion channels and inhibit neuronal activity.[2] **It is important to note that barbiturates do not inhibit sensory impulses and therefore have no analgesic effect.** They may produce hyperalgesia (i.e., increased response to painful stimuli) when given in subhypnotic doses.[2]

PHARMACODYNAMICS

Barbiturates produce dose-dependent depression of cerebral oxygen metabolism ($CMRO_2$), cerebral blood flow (CBF), and electroencephalogram (EEG) activity. A flat EEG tracing correlates to a maximal barbiturate suppression of $CMRO_2$ to 55% of normal.[2] The diminished $CMRO_2$ and CBF lead to a decrease in intracranial pressure (ICP). Barbiturates lower mean arterial pressure less than they lower ICP. Cerebral perfusion pressure (CPP) is usually enhanced. Barbiturates are commonly used in neuroanesthesia and in treatment of acute brain injury.

The cardiovascular effects of barbiturates include decreased cardiac output, decreased systemic arterial pressure, and a direct negative inotropic effect on the myocardium. Barbiturates decrease cardiac output primarily by depressing the vasomotor center, cause peripheral vasodilatation, and decrease venous return to the heart. Thiopental and methohexital have a positive chronotropic effect on the heart. Methohexital produces a greater increase in heart rate, which may explain why an equipotent dose of methohexital produces significantly less hypotension than does thiopental.[2] Myocardial oxygen demand is increased while coronary vascular resistance is decreased, although the increase in heart rate mitigates the decrease in blood pressure. Coronary blood flow will increase to meet the increased demand if the aortic pressure remains stable. **Barbiturates must be used cautiously in any patient whose condition is sensitive to tachycardia or a decrease in preload (e.g., hypovolemia, congestive heart failure, ischemic heart disease, or pericardial tamponade).** Methohexital has less cardiovascular depressant effects and a shorter duration of action, which makes it more useful than thiopental in the Emergency Department.

Barbiturates cause dose-dependent central respiratory depression characterized by diminished tidal volume and minute ventilation. The rate and depth of respiration may be suppressed to the point of apnea. The physiologic response to hypercarbia and hypoxemia may be blunted after the hypnotic effects have dissipated. All these effects may be greatly exaggerated with the concomitant use of opioids and in patients with chronic obstructive pulmonary disease.[7]

ADMINISTRATION

Thiopental and methohexital are available as sodium salts and should be dissolved in 0.9% saline. The recommended dose of thiopental is 3 to 5 mg/kg in adults, 5 to 6 mg/kg in children, and 6 to 8 mg/kg in infants IV given over 1 minute. The recommended dose of methohexital is 1 to 3 mg/kg IV over 30 seconds. **Reduce the induction dose in patients premedicated with fentanyl or midazolam.** Doses may have to be adjusted in patients with known hepatic or renal disease because decreased plasma albumin levels leave a greater fraction of barbiturate available to cross the blood-brain barrier. Make a 30% to 40% reduction in the geriatric population since their diminished muscle mass slows the rate of redistribution and lengthens the duration of CNS effects.[2]

ADVERSE EFFECTS

The most significant complications of barbiturate therapy stem from their cardiopulmonary depressant effects. Use barbiturates with caution in patients who are hypovolemic, have significant cardiovascular disease, or have reactive airway disease. Thiopental can raise plasma histamine levels, which may be associated with a transient skin rash and bronchospasm. Its use should be avoided in patients with a history of hypersensitivity to barbiturates. Severe anaphylactic reactions are extremely uncommon.[2] Laryngeal reflexes appear to be more active with thiopental than with propofol. Avoid thiopental in people with asthma.[2] Laryngospasm following induction with thiopental is more likely the result of airway manipulation in a "lightly anesthetized" patient.[2] Methohexital has known epileptogenic effects, causes myoclonic tremors, and can causes other CNS excitatory side effects (e.g., hiccups).[2] Barbiturates stimulate the production of porphyrins and are contraindicated in patients with acute intermittent porphyria, variegate porphyria, and hereditary coproporphyria.[2] Pain at the site of IV injection is more common with the administration of methohexital than thiopental. Extravascular or intraarterial injection of these drugs may cause severe pain, tissue necrosis, and thrombosis, leading to potential nerve damage and gangrene due to their high alkalinity with a pH of 10.

Treat intraarterial injection promptly by initially diluting the barbiturate with saline injected through the catheter in the artery. Consider the administration of intraarterial heparin, lidocaine, papaverine, or phenoxybenzamine to cause vasodilation and prevent vessel thrombosis.[2] Sympathectomy of the involved upper extremity by a stellate ganglion or brachial plexus block can be performed by an Anesthesiologist to relieve the vasoconstriction.

An additional concern relating to the high alkalinity of these solutions is the rapid injection through the same IV line with highly acidic drugs (e.g., neuromuscular blockers), which results in precipitation. This may lead to permanent blockage of the IV line at a time when access is crucial. It is recommended to flush the IV line thoroughly following the administration of barbiturates and before the administration of the next agent.

ETOMIDATE

Etomidate (Amidate) is an ultra-short-acting hypnotic unrelated to any other IV anesthetic agent. It is highly potent and produces a rapid onset of anesthesia. It lacks the barbiturates' cardiac depressant side effects. Etomidate has become a popular induction agent and is now the induction agent of choice in many Emergency Departments for rapid sequence intubation given its favorable profile.[8]

PHARMACOKINETICS

Etomidate is a carboxylated imidazole agent that undergoes a molecular rearrangement at physiologic pH to grant it greater lipid solubility. Approximately 75% of the drug is plasma protein bound. The free fraction accumulates readily in the CNS. Unconsciousness is produced within one arm-brain circulation. Peak brain concentration is achieved within 1 minute. Redistribution of the drug is quite rapid. A single bolus dose produces hypnosis in 10 seconds and lasts approximately 3 to 5 minutes. Clearance of etomidate is dependent on hepatic blood flow since it has a high extraction ratio and undergoes rapid hydrolysis by the liver with inactive metabolites excreted in the urine.[9]

MECHANISM OF ACTION

Etomidate (like barbiturates, propofol, and benzodiazepines) produces dose-dependent CNS depression by modulating GABA receptors to produce hyperpolarization and functional inhibition of

the postsynaptic neurons. GABA antagonists (e.g., flumazenil) may attenuate its effects. **Etomidate has no analgesic properties.**[10]

PHARMACODYNAMICS

Etomidate produces dose-dependent depression of $CMRO_2$, CBF, and EEG activity analogous to that of the barbiturates. **Etomidate decreases ICP with minimal effect on CPP since it does not affect mean arterial pressure.** It is useful in patients with elevated ICP, especially those who are hemodynamically unstable.[11] **Etomidate causes minimal cardiovascular depression, even in the presence of significant cardiac disease.** Heart rate, blood pressure, and cardiac output are adequately maintained. The respiratory depressant effects of etomidate appear to be substantially less than those of thiopental or propofol. This makes etomidate a safer choice than barbiturates in patients with diminished pulmonary function. **Etomidate is therefore considered to be the induction agent of choice in patients with severe cardiopulmonary disease and high-risk patients in whom maintenance of blood pressure is crucial**.[12] Combine etomidate with an opioid analgesic in patients who would be at risk from a transient elevation of blood pressure or heart rate because it does not blunt the sympathetic response to laryngoscopy and intubation.[13] **Etomidate is the only available IV anesthetic that does not induce the release of histamine and is safe for patients with reactive airways.**[14]

ADMINISTRATION

Etomidate is formulated in a 0.2% solution with 35% propylene glycol. The standard induction dose is 0.3 mg/kg IV based on total body weight. There is virtually no accumulation of the drug. Emergence time is dose-dependent but remains short even after repeated boluses.[15] Dose adjustments for elderly patients, hepatic disease, or renal disease may be required.

ADVERSE EFFECTS

The most common side effects of etomidate are injection site pain, myoclonus, nausea, and vomiting during the induction phase.[16] Myoclonic activity occurs in about one-third of cases and is attributed to interruption of inhibitory synapses in the thalamocortical tract rather than CNS excitation.[2] Pretreatment with a benzodiazepine, low-dose etomidate, magnesium sulfate, or an opioid analgesic diminishes the frequency of myoclonic movements.[17-19] Vein irritation and pain at the IV site can be attributed to the propylene glycol diluent. Use of a large vein with simultaneous analgesic and saline infusion reduces the incidence of injection pain.[20]

Etomidate causes a dose-dependent suppression of adrenal corticosteroid synthesis by inhibiting the enzyme 11-β-hydroxylase.[21] A single induction dose of etomidate temporarily suppresses adrenocortical hormone synthesis.[22,23] Increased mortality has been attributed to this reduction of endogenous steroids leading to acute adrenocortical insufficiency in critically ill patients on an etomidate infusion.[24] **There is currently conflicting evidence as to whether a single induction dose of etomidate affects overall mortality in critically ill patients.**[25-31] Analysis of some studies showed worse outcomes with etomidate use,[32-34] whereas other studies showed no difference in mortality with the use of etomidate.[23,25,26,35-38]

KETAMINE

Ketamine (Ketalar, Ketaject) is a phencyclidine derivative that is unique among induction agents. **It produces a dissociative anesthetic state characterized by profound analgesia and amnesia.** Patients may appear awake with their eyes open and reveal a nystagmic gaze. They may make spontaneous nonpurposeful movements. **Protective reflexes are usually maintained.** This is fortunate considering that increased salivation is a common side effect. Ketamine is fast-acting and has a brief duration of action. It has been widely used since 1970 despite emergence delirium limiting the usefulness of this drug.[39]

PHARMACOKINETICS

Ketamine has a p*K*a of 7.5. Approximately 12% is plasma protein bound. Roughly half of the unbound fraction is available to accumulate rapidly in the CNS. A single induction dose produces anesthesia within 30 seconds and brain concentration peaks at 1 minute. Ketamine follows the three-compartment model with rapid redistribution to the peripheral tissues.[39] Its CNS effects last approximately 10 to 15 minutes. Recovery of full orientation and function may take an additional 60 minutes. Ketamine is readily metabolized by hepatic microsomal enzymes to norketamine. Norketamine is one-fourth as potent as its precursor. The active metabolite may explain the prolonged recovery time. Norketamine is hydroxylated and excreted by the kidneys. Ketamine has a high extraction ratio, and its clearance is dependent on hepatic blood flow.[2]

MECHANISM OF ACTION

Ketamine acts by binding to the *N*-methyl-D-aspartate (NMDA) receptors on postsynaptic neurons. This receptor is a gated ion channel that allows depolarization and initiation of an action potential when activated by glutamate or NMDA. Ketamine blocks the flux of ions through this channel and inhibits the stimulatory effects of these neurotransmitters. Ketamine depresses neuronal activity in the cerebral cortex, depresses the thalamus, and stimulates the limbic system. Analgesia is produced by interrupting the association pathways responsible for the interpretation of painful stimuli that run from the thalamocortical and limbic systems.[40]

PHARMACODYNAMICS

Ketamine produces dose-dependent CNS depression. It increases $CMRO_2$ and CBF, leading to an increase in ICP. Ketamine has a potent sympathomimetic effect. Its use often produces an increase in heart rate and arterial blood pressure. It has a direct negative inotropic effect on the myocardium, which is evident in the critically ill patient with depleted catecholamines. Ketamine produces minimal to no respiratory depression and has a potent bronchodilatory effect. It increases both bronchial and oral secretions. Ketamine is likely to preserve protective airway reflexes in contrast to other anesthetic agents. Skeletal muscle tone is increased and results in random movements.[39]

ADMINISTRATION

Ketamine is available in 1% and 5% aqueous solutions for IV administration and a 10% aqueous solution for intramuscular (IM) injection. The induction dose of ketamine is 1 to 2 mg/kg IV over 1 minute or 5 to 10 mg/kg IM.[2] A lower dose can be used for procedural sedation.[41]

Ketamine is indicated for the intubation of asthmatic patients due to its bronchodilatory properties. Ketamine may be therapeutic for these patients because the increase in bronchial secretions may decrease the incidence of mucous plugging.[42] Ketamine may be useful in the hemodynamically unstable patient due to its cardiac stimulatory effects. **Ketamine does not adversely affect CPP, ICP, intraocular pressure (IOP), mortality, or neurologic outcomes.**[43-50] **It can be used with suspected head trauma or intracranial pathology. Do not use ketamine in patients with ischemic heart disease because it can increase blood pressure, heart rate, and myocardial oxygen demand.**

ADVERSE EFFECTS

The most significant side effect of ketamine is the occurrence of postanesthetic emergence reactions.[51] Up to 30% of patients treated with ketamine have reported agitation, aggression, confusion, delirium, hallucinations, hysteria, nightmares, restlessness, and unpleasant sensations.[2] Those most affected were the elderly, females, and patients receiving more than 2 mg/kg. Children seem less adversely affected than adults. These reactions can be attenuated or eliminated by the coadministration of a benzodiazepine (i.e., midazolam) or propofol.[52] Other side effects include increased ICP, increased IOP, hypersalivation, nystagmus, and random movements.[51] The hypersalivation may be limited by the administration of atropine or glycopyrrolate.[53,54] Ketamine may activate epileptogenic foci in patients with a seizure disorder.[39] Ketamine can rarely cause an allergic reaction.[55]

PROPOFOL

Propofol (Diprivan) is a sedative-hypnotic agent used for conscious sedation, induction, and maintenance of anesthesia. It has a profile similar to that of thiopental. It is unrelated to any of the other induction agents. The use of propofol has greatly expanded despite its cardiopulmonary depressant effects. It is well suited for ambulatory surgery performed on relatively healthy outpatients. Recovery after propofol anesthesia is rapid and is accompanied by less residual sedation, fatigue, and confusion than any other induction agent. Propofol is associated with a low incidence of postanesthetic emesis.[56] Propofol as an adjunct to emergency airway management is commonly used for procedural sedation or postintubation sedation.

PHARMACOKINETICS

Propofol is an alkyl-phenol compound that is insoluble in water. It is 98% plasma protein bound. Any unbound drug rapidly accumulates in the brain and liver since it is highly lipophilic. A single bolus produces hypnosis in 15 to 45 seconds. Its duration of action is 5 to 10 minutes and reflects a rapid redistribution to lean muscle. Propofol is cleared from the central compartment by hepatic metabolism. Its metabolites are water soluble and excreted in the urine.[2] It can cause green discoloration of the urine.[57,58]

MECHANISM OF ACTION

Propofol produces CNS depression by modulating the $GABA_A$ receptor by the same mechanism as barbiturates.[2]

PHARMACODYNAMICS

Propofol produces a dose-dependent CNS depression without analgesia. It has a strong amnestic effect. It decreases $CMRO_2$, CBF, and ICP. Its cardiac depressant effects are greater than those of thiopental. CPP may be affected at high doses. Propofol is a myocardial depressant and potent vasodilator that lowers blood pressure and cardiac output. These effects attenuate the hemodynamic pressor response to laryngoscopy and intubation. Propofol blunts the baroreflex so that heart rate does not increase in proportion to a decrease in blood pressure. Decreased respiratory rate and tidal volume are seen with propofol. The ventilatory response to hypercarbia is diminished. Propofol may produce bronchodilation in patients with chronic obstructive pulmonary disease (COPD).[59] Propofol appears to have a strong anticonvulsant effect and may be used to terminate status epilepticus.[60]

ADMINISTRATION

Propofol is prepared as a 1% oil-in-water emulsion that contains egg lecithin, soybean oil, glycerol, and EDTA. The adult dose for induction is 2.0 to 2.5 mg/kg IV. An infusion rate of 25 to 75 μg/kg/min is titrated to effect for sedation. Dosages should be reduced to 1.0 to 1.5 mg/kg IV in the elderly, in high-risk patients, and in anyone premedicated with an opioid or a benzodiazepine.[59] Propofol supports bacterial growth, and any unused portion should be discarded.

ADVERSE EFFECTS

Propofol produces pain on injection in up to 75% of patients. This may be reduced or prevented by pretreatment with an opioid or the addition of 0.01% lidocaine to the emulsion. Propofol may cause mild CNS excitation in the form of myoclonus, tremors, or hiccups.[2] Propofol can rarely cause diabetes insipidus and pulmonary edema.[61,62]

FOSPROPOFOL

Fospropofol is a prodrug or precursor of propofol used for sedation.[63,64] It comes as a water-soluble disodium salt. It is dosed at 6.5 mg/kg with additional doses at 1.6 mg/kg. The dosing is based on total body weight. **Propofol is hydrolyzed and rapidly released from the action of alkaline phosphatases on fospropofol.** The hydrolysis of fospropofol also forms phosphate and formaldehyde from the action of the alkaline phosphatases. The formaldehyde is converted into formate. The metabolites (i.e., formaldehyde, formate, and phosphate) do not accumulate to any degree. Fospropofol produces a lower peak plasma concentration of propofol than lipid emulsion propofol. The elimination kinetics of lipid emulsion propofol and fospropofol-derived propofol are the same.

The use of fospropofol has been demonstrated to be safe in clinical studies. The most common adverse effects were self-limited paresthesias and pruritus. It had a different pharmacodynamic and pharmacokinetic profile than lipid emulsion propofol. Fospropofol has a longer elimination half-life of 2 hours and a delayed onset of action. It peaks in 8 minutes. The slower onset of action may cause less hypotension but a longer recovery. The use of water-soluble fospropofol may eliminate the risk of bacterial contamination and lipid-related side effects.

BENZODIAZEPINES

Benzodiazepines are a large class of drugs with anxiolytic, sedative, hypnotic, and amnestic properties without any analgesic properties. Diazepam (Valium), lorazepam (Ativan), and midazolam (Versed) are most often used to facilitate intubation. They have a relatively short time of onset when given IV. Diazepam and lorazepam have longer times of onset, less predictable dose-effect relationships, and longer elimination half-lives when compared to midazolam. Diazepam and lorazepam are insoluble in water and formulated in a propylene glycol diluent. The propylene glycol can produce a high rate of venous irritation and unpredictable absorption after intramuscular injection.[65] Midazolam is water soluble at a low pH and does not require propylene glycol. This decreases the incidence of erratic absorption after intramuscular injection and pain on injection.

Midazolam is the newest of the three drugs. It has become the standard choice for preinduction anxiolysis, sedation, and amnesia. Midazolam is well suited and widely used as an induction agent.[66] It may be administered alone or in combination with an opioid. There is considerable hypnotic synergy when midazolam is used in combination with an opioid. The opioids provide excellent analgesia, which is a property lacking in midazolam.[2] Recovery of consciousness takes longer when midazolam is used as the sole induction agent than when it is used with other single agents (e.g., etomidate, methohexital, propofol, or thiopental). Midazolam is often used as a coinduction agent with ketamine or propofol as it facilitates the onset of anesthesia without prolonging emergence times.[2]

Midazolam attenuates ketamine's cardiac stimulatory side effects as well as the incidence, severity, and recall of emergence reactions when combined with ketamine.[67]

PHARMACOKINETICS

Midazolam is formulated at a pH of 3.5. It is water soluble in an acidic environment. It undergoes a molecular rearrangement that makes it highly lipophilic at a physiologic pH. It is 94% protein bound in the plasma. Unbound midazolam rapidly accumulates in the CNS where it produces dose-dependent sedation or unconsciousness in 30 to 90 seconds. The drug redistributes less rapidly than many of the other hypnotics. This is reflected in the 10- to 30-minute duration of its CNS effects.[68] Midazolam is oxidized by the liver and excreted in the urine. Changes in hepatic blood flow can influence the clearance of midazolam.[69] Age has little effect on the elimination half-life.[69] Fentanyl has been shown to competitively inhibit the hepatic metabolism of midazolam in vitro and to decrease its clearance.[70]

MECHANISM OF ACTION

Benzodiazepines bind to a specific site on the alpha subunit of the $GABA_A$ receptor and enhance inhibitory neurotransmission. Midazolam has the greatest affinity for the receptor when compared to other benzodiazepines.[2] It has been proposed that the percentage of benzodiazepine receptor occupancy accounts for its effects. A 20% occupancy provides anxiolysis. A 30% to 50% occupancy causes sedation. Greater than 60% occupancy produces unconsciousness. It is unknown how the benzodiazepines produce amnesia.[2]

PHARMACODYNAMICS

Midazolam decreases $CMRO_2$ and CBF like thiopental and propofol. Midazolam has a ceiling effect with respect to cerebral metabolism. The cerebrovascular response to carbon dioxide is unaffected by midazolam. It is a potent anticonvulsant. Midazolam is a mild cardiac depressant and can decrease systemic vascular resistance.[68] The cardiac output and coronary blood flow are usually not affected by midazolam.[71] The decrease in blood pressure is often masked by the sympathetic response to laryngoscopy. Hypotension may be pronounced in hypovolemic patients or in those who were given large doses.

Midazolam's respiratory effects have a ceiling. It produces a mild and dose-dependent respiratory depression. The respiratory depression is insignificant in relatively healthy patients. The respiratory depression is enhanced in patients with pulmonary disease. Hypoxemia is more frequent in patients who receive midazolam in combination with an opioid than in patients who receive either drug alone.[72]

ADMINISTRATION

An induction dose of midazolam is 0.2 to 0.4 mg/kg IV. The dose of midazolam is reduced to 0.1 to 0.2 mg/kg when used as a coinduction agent. The dose for sedation is 0.04 to 0.1 mg/kg IV. It can be administered intramuscularly at a dose of 0.07 to 0.1 mg/kg when rapid onset is not required.[2] Doses may have to be lowered in older patients because sensitivity to the hypnotic effects of midazolam increases with age independent of pharmacokinetic factors.[73]

ADVERSE EFFECTS

Midazolam has relatively few adverse effects. It is a mild cardiorespiratory depressant. A synergistic respiratory depressant effect may result in hypoxemia, apnea, and death when it is combined with an opioid. Monitor patients receiving this combination with pulse oximetry while being given supplemental oxygen.[72] Midazolam produces little venous irritation and pain upon injection. It may precipitate a psychotic episode in patients taking valproate. It does cross the placenta and is associated with birth defects when administered in the first trimester.[68] Paradoxical reactions (e.g., aggression, agitation, confusion, delirium, hysteria, and restlessness) can be treated and reversed with flumazenil.[74-76]

OPIOIDS

Opioids produce dose-dependent analgesia, sedation, and respiratory depression by mimicking the effects of endogenous opiopeptins.[77] Morphine is the standard to which all opioids are compared. Fentanyl (Sublimaze) is the opioid of choice due to its rapid onset and short duration of action.[78] It has been in use since 1968, and its effects are well documented. **Fentanyl is rarely associated with a significant release of histamine.**[79] Fentanyl has a remarkable hemodynamic stability profile.[79] Fentanyl will produce anesthesia adequate for intubation if a large dose is given. It is more often used as a sedative-analgesic or as a pretreatment adjuvant with one of the previously mentioned induction agents. Fentanyl is a synthetic opioid that is 50 to 100 times more potent than morphine. It is structurally related to the phenylpiperidines. Fentanyl is highly lipophilic and produces excellent short-term analgesia and sedation.[2]

Alfentanil (Alfenta) is a structural derivative of fentanyl. It has a more rapid onset of action than fentanyl and half the duration of effect. Alfentanil is between one-sixth and one-ninth as potent as fentanyl. Its uses are analogous to those of fentanyl. It has been in use in the United States since 1982.[80] Alfentanil is safe and effective for use in emergent rapid sequence intubation.[81] It was associated with greater cardiovascular depression than fentanyl in cardiac patients.[82] Alfentanil is associated with a greater incidence of nausea and vomiting than fentanyl.[83] There are no data to suggest that alfentanil is more efficacious than fentanyl.

PHARMACOKINETICS

Plasma fentanyl is 85% protein bound. Its lipophilic nature allows it to enter highly perfused tissues rapidly (e.g., the brain, heart, and lungs). Single bolus dose effects may be seen in as little as 10 seconds and peak in 3 to 5 minutes. Morphine's effects peak in 20 to 30 minutes. Fentanyl has a high affinity for adipose tissue, and redistribution accounts for the cessation of effects, which can take up to 30 to 60 minutes. An equipotent dose of morphine has a duration of 3 to 4 hours. Redistribution of fentanyl from peripheral tissues to the central compartment after large or repeat doses may prolong its effects. Clearance of morphine and fentanyl is by hepatic metabolism. These drugs have a high extraction ratio, and changes in hepatic blood flow can influence the clearance of fentanyl.[80]

MECHANISM OF ACTION

Fentanyl binds to the μ (mu) opioid receptor found throughout the CNS. Activation of the opioid receptor causes hyperpolarization and inhibition of neurotransmitter release.[77] It has been suggested that fentanyl may be a low-affinity NMDA receptor antagonist.[84]

PHARMACODYNAMICS

Fentanyl decreases CBF and cerebral oxygen consumption. A relatively small dose of 3 μg/kg produced an elevation of ICP in patients with head trauma.[85] Fentanyl may produce a mild reduction in heart rate with little effect on cardiac contractility. Systemic vascular resistance and blood pressure may be slightly reduced. They are usually unaffected in patients without cardiac pathology.[86] Respiratory depression induced by fentanyl is dose dependent. The respiratory rate decreases, followed by the tidal volume and subsequent apnea. The patient's response to hypercarbia is blunted when sedated with fentanyl.[87]

ADMINISTRATION

Fentanyl can be used in several ways to facilitate emergency intubation. As little as 3 to 5 μg/kg IV over 2 minutes may allow for an awake intubation when given purely for analgesia. Incremental doses of fentanyl from 25 to 50 μg in adults can be titrated to produce the desired effect.[2] Fentanyl has a more stable hemodynamic profile during rapid sequence intubation than either thiopental or midazolam.[78] Between 5 and 15 μg/kg of fentanyl may be used as the sole induction agent in hemodynamically unstable patients and those with poor cardiac reserve.[78]

The most prudent role for fentanyl is as an adjunct to an induction agent. Premedication 3 minutes prior to intubation with 2 to 4 μg/kg of fentanyl IV over 2 minutes provides excellent analgesia, attenuates the transient hypertension, and attenuates tachycardia associated with laryngoscopy and intubation.[88] Compared with lidocaine and beta-blockers, opioids are more effective at blunting the pressor response, are more reliable, and do not produce rebound hypotension and bradycardia.[89,90]

ADVERSE EFFECTS

Respiratory depression is common to all opioids. Fentanyl has relatively few adverse side effects. Nausea and vomiting are relatively uncommon when compared to other opioids.[2] Fentanyl is not associated with a significant release of histamine, which is reflected in its hemodynamic stability. Muscular rigidity involving the chest wall and diaphragm may occur.[91] This makes ventilation difficult if it occurs. This happens more often at doses greater than 15 μg/kg. It may be prevented or relieved by neuromuscular blockade or by opioid antagonism with naloxone.[91,92] Myoclonic movements may occur and do not reflect seizure activity on the EEG. Biliary colic and urinary retention are associated with fentanyl administration.[2]

NEUROMUSCULAR BLOCKING AGENTS

Neuromuscular blockade is an integral part of the rapid sequence induction and intubation protocol (Tables 12-2 and 12-3). The combination of a paralytic agent and a sedative or analgesic is superior to the use of any single agent. The use of a neuromuscular blocking (NMB) agent to facilitate intubation provides for control of the airway and better visualization of the vocal cords than does sedation without paralysis.[93,94] It is well documented that rapid sequence induction with an NMB agent allows for faster intubations with fewer complications than sedation alone.[1] **Reserve the use of a sedative without an NMB for the awake oral intubation of a patient with a difficult airway.**

NMBs are classified as either depolarizing or nondepolarizing depending on their action at the nicotinic acetylcholine receptor of the motor end plate. **The optimal NMB agent has a rapid onset of action, a predictably short duration of action, and no side effects. The optimal NMB agent has yet to be found.** Succinylcholine (Anectine) and rocuronium (Zemuron) remain the standard NMB agents for Emergency Department intubations.[95,96]

SUCCINYLCHOLINE

Succinylcholine is the only depolarizing NMB agent in clinical use. It was introduced in 1952 and is a chemical combination of two acetylcholine molecules. **It is one of the most widely used NMB agents in the Emergency Department because its onset of action is faster and its duration of action is shorter than that of any other NMB agent. This is particularly important in patients who cannot be intubated after neuromuscular blockade and when the resumption of spontaneous respirations is vital.**

PHARMACOKINETICS

Adequate intubating conditions are usually achieved in 60 seconds after a paralytic dose of succinylcholine.[97] The duration of apnea following a single dose of succinylcholine is 3 to 5 minutes and reflects the rapid degradation of the drug by pseudocholinesterase (i.e., also known as plasma cholinesterase or butyrylcholinesterase).[97] Repeated doses or infusions of succinylcholine may produce tachyphylaxis, prolonged paralysis, and repolarization of the neuromuscular membrane (i.e., phase II block).[97] A phase II block can be partially reversed by administration of an anticholinesterase agent similar to the reversal of a nondepolarizing block. **A nondepolarizing agent can be administered after the patient is intubated if paralysis of greater than 3 to 5 minutes is desired.**

MECHANISM OF ACTION

The structure of succinylcholine allows it to bind noncompetitively to acetylcholine receptors and cause depolarization of the postjunctional neuromuscular membrane. The initial depolarization is a brief period of muscle fasciculation following the administration of the drug. Acetylcholine is hydrolyzed within milliseconds. Succinylcholine remains intact for several minutes. It produces paralysis by occupying the acetylcholine receptors and makes the motor end plates refractory so that muscle contraction cannot occur. Muscle relaxation proceeds from the distal muscles to the proximal muscles. The diaphragm is one of the last muscles to become paralyzed.[98]

PHARMACODYNAMICS

Succinylcholine is rapidly hydrolyzed by plasma pseudocholinesterase to succinylmonocholine. Only a small fraction of the IV administered dose reaches the neuromuscular junction. Succinylmonocholine has some neuromuscular blocking properties and is further hydrolyzed to succinic acid and choline. These end products are rapidly taken up by cells and reused in various biochemical molecules. The rapid degradation of succinylcholine provides a concentration gradient that causes the diffusion of succinylcholine away from the acetylcholine receptors and allows repolarization of the myocyte membrane.

Some patients have an atypical pseudocholinesterase enzyme. This condition occurs with an incidence of 1 in 3200. This is a genetic variant with autosomal semidominant transmission. The duration of action of succinylcholine is markedly prolonged due to decreased enzyme activity. A single intubating dose of succinylcholine may last up to 8 hours in patients who are homozygous for the atypical allele.[98]

ADMINISTRATION

The recommended dose of succinylcholine to produce optimal intubating conditions is 1.0 to 1.5 mg/kg IV bolus in adults and 1.5 to 2.0 mg/kg in infants with the increased dose explained by their higher volume of distribution.[97,99] These doses are based on total body weight and not ideal body weight like rocuronium. Increase this dose by 50% in patients who have received a defasciculating dose of a nondepolarizing NMB prior to the administration of succinylcholine.[53] IM administration is possible and is important for use in an emergency when control of the airway is necessary and the patient has no IV access. The IM dose is two to four times the IV dose.[100,101]

ADVERSE EFFECTS

Numerous potential adverse effects are associated with the administration of succinylcholine but occur relatively uncommonly. These include anaphylaxis, autonomic stimulation, elevated ICP and IOP, histamine release, hyperkalemia, malignant hyperthermia,

muscular fasciculations and myalgia, and prolonged apnea.[97,102] **The use of succinylcholine is recommended for rapid sequence induction and intubation because the risk of a compromised airway far outweighs the potential harm from these side effects.**

The fine, chaotic muscle contractions that are often observed at the onset of paralysis are associated with several side effects. They are most commonly associated with myalgia, increased IOP, increased ICP, and increased intragastric pressure. Muscle pain 24 to 48 hours after the administration of succinylcholine is most prominent in young muscular men. Muscle pain is unlikely in children, the elderly, and those with undeveloped or diminished muscle mass. Muscle fasciculations may be prevented by the administration of a defasciculating dose (i.e., 10% of the paralytic dose, a phenomenon known as "precurarization") of a nondepolarizing NMB agent given 3 to 5 minutes prior to the succinylcholine.[103]

Increased ICP may occur with the use of succinylcholine. The magnitude and clinical significance of this increase remain unclear. Defasciculation with a nondepolarizing NMB agent has been shown to prevent this rise in ICP.[104]

Omitting the use of a paralyzing agent entirely during the rapid sequence induction of patients with penetrating eye injuries or intracranial pathology for fear of the potential increase in IOP or ICP is a decision that must be made for each individual patient. Any attempt to intubate a nonparalyzed and lightly anesthetized patient could result in gagging or "bucking." This patient action has been shown to increase IOP and ICP far more than would be achieved from an intubating dose of succinylcholine. **Muscle fasciculations are benign and precurarization is unnecessary in most patients.** It is prudent to use a nondepolarizing NMB agent or use precurarization with succinylcholine to prevent worsening of an injury in patients with eye injuries or suspected intracranial pathology.[105] Rocuronium is a nondepolarizing agent that has been shown to significantly decrease IOP during rapid sequence induction.[106] Its use may be indicated in patients with penetrating eye injuries.[106]

Increased intragastric pressure is lessened by precurarization. Increased intragastric pressure may increase the risk of aspiration. Succinylcholine favorably increases the tone of the lower esophageal sphincter and may mitigate the risk of aspiration.[107] Regurgitation of stomach contents during intubation is more likely the result of distention from overzealous mask ventilation.

Succinylcholine binds to acetylcholine receptors throughout the body including those of the autonomic ganglia. Succinylcholine may have direct muscarinic effects on the heart. It is difficult to characterize a specific cardiovascular effect typical of succinylcholine. It may produce tachycardia, bradycardia, or dysrhythmias.[97] Children are particularly susceptible to bradycardia following succinylcholine administration. Consider giving children 0.01 mg/kg IV of atropine prior to administration of succinylcholine.[100]

Prolonged apnea following succinylcholine administration is a sign of decreased plasma pseudocholinesterase levels. This may occur in patients with hepatic disease, anemia, renal failure, cancer, connective tissue disorders, pregnancy, cocaine intoxication, or genetically deficient enzyme activity or patients taking cytotoxic drugs. Most cases of prolonged apnea rarely exceed 20 minutes.[108]

Succinylcholine may produce an increase in serum potassium level, which is typically less than 0.5 meq/L. Use it with caution in patients with significant hyperkalemia. Its use is not contraindicated in patients with renal failure, but caution must be taken if their serum potassium level is elevated. It has been associated with cases of massive hyperkalemia (e.g., > 5 meq/L) and cardiac arrest in patients who have had digoxin toxicity, myasthenia gravis, massive muscle trauma, crush injuries, severe burns, and major nerve or spinal cord injury at least 1 week prior to receiving succinylcholine.[95,97] Do not use succinylcholine starting 24 hours after the insult in these patients.

MALIGNANT HYPERTHERMIA

Malignant hyperthermia (MH) is an extremely rare and life-threatening autosomal dominant condition that can develop following exposure to certain inhaled anesthetics and/or succinylcholine in genetically susceptible individuals. It is characterized by intense, sustained skeletal muscle contraction leading to severe acidosis, rhabdomyolysis, hyperthermia, hyperkalemia, arrhythmias, and death if left untreated. The triggering exposure is not dose dependent. It can occur following any single dose or combination of offending agents. It may occur in individuals who have been exposed to the agents in the past without an adverse effect. The incidence of MH during anesthesia is estimated to be 1 in 15,000 in children and 1 in 50,000 in adults.[109] **MH is most notoriously characterized by a rapid elevation in temperature. The earliest signs of the condition are usually profound tachycardia, tachypnea, and generalized skeletal muscle rigidity.** Laboratory blood analysis reveals severe respiratory and metabolic acidosis, hyperkalemia, and elevation of serum creatine kinase.

Early and aggressive treatment is the key to patient survival. This involves active cooling measures, volume resuscitation, correction of acid-base and electrolyte disturbances, and rapid administration of dantrolene in a 2 to 3 mg/kg IV bolus with additional increments up to 10 mg/kg.[97,109] Dantrolene sodium is a muscle relaxant that is supplied as a lyophilized powder in 20 mg vials. Each vial must be reconstituted with 60 mL of water.[109] **Early administration of dantrolene is important to abort the reaction and greatly increases the chance of survival. Continue dantrolene administration until all signs of MH have stabilized.** Admit the patient to the intensive care unit for observation of any recurrence following the acute phase.

Isolated masseter muscle rigidity is a benign side effect in most cases. It has been reported mainly in children receiving succinylcholine. Masseter muscle rigidity was the first sign of an abnormal reaction in several reported cases of fatal MH.[110] **Masseter rigidity following the administration of succinylcholine requires the patient to be closely monitored for other signs of MH.**

Patients of families with MH, those with suspected reactions, and other selected high-risk patients can undergo a muscle biopsy test known as the caffeine-halothane contracture test. The test carries a 100% sensitivity and 85% to 90% specificity for MH susceptibility.[109] Refer patients who develop suspected MH reactions immediately to their primary physicians for testing and follow-up. Documented positive susceptibility and patient education could prevent a future potentially lethal exposure to offending agents.

The benefits of intubation with a rapidly acting and short-lasting paralytic agent such as succinylcholine provide the safest conditions for intubation despite the lengthy list of potential adverse effects. Administer a nondepolarizing agent for maintenance following intubation with succinylcholine if prolonged paralysis is required.

NONDEPOLARIZING AGENTS

Nondepolarizing NMB agents act by competitive inhibition of the acetylcholine receptors at the motor end plate. They weakly bind to the receptor and block the binding site for acetylcholine without producing any effect on the postsynaptic neuromuscular membrane. This blockade is dependent on the relative concentrations of acetylcholine and NMB available in the synaptic cleft. It follows the kinetics of competitive inhibition. Normal neuromuscular transmission is restored as the ratio returns in favor of acetylcholine. Return of muscle function can be hastened with a cholinesterase inhibitor (e.g., neostigmine or edrophonium) after some muscular contraction is observed. **Nondepolarizing agents have**

the potential for reversal and fewer side effects than succinylcholine. Their longer time to onset and much longer duration of action make them less useful for rapid sequence intubation.[97]

Nondepolarizing NMB agents can be grouped by chemical structure. The steroid-based agents include pancuronium, vecuronium, and rocuronium. The oldest nondepolarizing agent is d-tubocurarine. Atracurium, cisatracurium, d-tubocurarine, and mivacurium are benzylisoquinolines.[97] Pancuronium, vecuronium, and rocuronium have been extensively studied for use in rapid sequence intubation.[111] Rocuronium (Zemuron) has become established as the nondepolarizing NMB of choice for rapid sequence intubation in situations where succinylcholine is contraindicated. Rocuronium is discussed separately from the other nondepolarizing NMB agents.

Rocuronium

Rocuronium has been used at a dose of 1.2 mg/kg based on ideal body weight when the avoidance of succinylcholine is desired during rapid sequence intubation. This dose is twice the standard intubating dose to yield an onset of action in approximately 60 seconds and intubating conditions that are comparable to succinylcholine.[112,113] Dosing by total body weight will result in a longer duration of action.[114] This has been demonstrated in adult and pediatric populations.[112,115] The increased dosage of 1.2 mg/kg prolongs the duration of action to greater than 1 hour, which far exceeds the 3 to 5 minutes provided by succinylcholine.[97,116] The nondepolarizing competitive blockade produced by rocuronium cannot be immediately reversed by anticholinesterases (e.g., neostigmine) until partial competitive antagonism by acetylcholine has taken place naturally at the motor end plate. This may take an average of 20 minutes to occur. It must be measured by the return of muscular twitch using a neuromuscular twitch monitor often used by Anesthesiologists in the Operating Room to monitor the level of intraoperative motor blockade.[115] **Selective reversal of the dense rocuronium paralysis with the newly Food and Drug Administration-approved agent sugammadex if unexpected difficulties with intubation or ventilation are encountered should be considered. Staff experienced in a surgical airway must be immediately available if intubation and ventilation fail and complete reversal to the point of return of adequate spontaneous ventilation proves unsuccessful.**

PHARMACOKINETICS

Rocuronium is eliminated primarily by the liver, and < 10% is eliminated by the kidneys.[97] Its duration of action is significantly prolonged in liver failure and only slightly in renal failure. There are no active metabolites. Nondepolarizing NMB agents as a class are highly ionized, water-soluble compounds that cannot easily cross lipid membranes (e.g., the blood-brain barrier and the placenta). They have no CNS effects and do not affect the fetus when administered to pregnant women.[97]

MECHANISM OF ACTION

Rocuronium binds nicotinic acetylcholine receptors at the neuromuscular end plate. It is unable to induce the conformational change necessary to open the ion channels and allow subsequent depolarization. This results in a competitive and antagonistic block.

PHARMACODYNAMICS

Several drugs (e.g., aminoglycosides, certain antiarrhythmics, dantrolene, lithium, magnesium, and volatile anesthetics) will augment the neuromuscular blockade produced by nondepolarizers. Corticosteroids, certain anticonvulsants, and calcium will diminish the effect of NMBs. Patients with myasthenia gravis, Lambert-Eaton myasthenic syndrome, and Duchenne's muscular dystrophy exhibit varying sensitivity to nondepolarizing NMBs. Burn patients are resistant to their effects. Careful titration of dosages, dosing intervals, and infusion rates is necessary in these patient populations.

ADMINISTRATION

Rocuronium is administered at a usual intubating dose of 0.6 mg/kg. It provides a clinical duration of roughly 45 minutes. Rapid sequence intubation with rocuronium is most rapidly achieved with a dose of 1.2 mg/kg based on ideal body weight and is associated with an accompanying increase in duration when compared to the 0.6 mg/kg dose.[114] Dosing by total body weight can increase the duration of action.[114] Maintenance of paralysis following intubation can be achieved with intermittent 0.1 mg/kg boluses or by infusion rates of 5 to 12 mcg/kg/min titrated to effect.

ADVERSE EFFECTS

Rocuronium is essentially devoid of any serious side effects other than the potential for a severe anaphylactic reaction in allergic patients.[102,117,118] This should not be taken lightly. NMBs are responsible for > 50% of all life-threatening anaphylactic or anaphylactoid reactions occurring during anesthesia administration.[97] Although succinylcholine is the most common offending agent, the nondepolarizers are second.

The steroidal nondepolarizers possess varying degrees of vagolytic activity and are not associated with histamine release. The vagolytic action of rocuronium is weak and rarely of clinical significance, in contrast to pancuronium, another steroidal nondepolarizer, which can cause significant tachycardia following administration. Patients may report pain on injection of rocuronium due to venous irritation. This can be reduced by prior administration of IV lidocaine but is usually not a problem because patients are unconscious or sedated at the time of rocuronium injection.

Other Nondepolarizing Agents

Pancuronium is classified as a long-acting, bisquaternary steroidal nondepolarizing agent. It produces paralysis in 2 to 5 minutes and lasts approximately 60 to 90 minutes at a dose of 0.08 to 0.12 mg/kg. Supplemental doses of 0.02 mg/kg following intubation can be given intermittently to maintain paralysis.[97] Its use is associated with an increased heart rate, blood pressure, and cardiac output through a postganglionic vagolytic effect. It is not associated with the release of histamine. An estimated 80% to 85% of a dose of pancuronium is eliminated unchanged in the urine. Patients with renal failure may finds its duration significantly prolonged.[97] The low cost of pancuronium and familiarity have made it popular. Its slow onset of action and extended duration limit its usefulness in facilitating emergent endotracheal intubation.

Removing a quaternary methyl group from pancuronium yields the monoquaternary steroidal nondepolarizer vecuronium. This small change does little to affect potency. It favorably alters the side effect profile of the drug, most notably the vagolytic action. The vagolytic effects of vecuronium are similar to those of rocuronium, are negligible, and do not cause histamine release. Vecuronium can cause anaphylaxis.[102] This molecular change alters the metabolism and excretion of the drug when compared with pancuronium. Vecuronium is metabolized to a small extent by the liver and depends primarily on biliary excretion and secondarily on renal excretion. Vecuronium is unstable in solution. It is prepared as a lyophilized powder that must be hydrated prior to use.

The normal intubating dose of vecuronium at 0.1 mg/kg takes 3 minutes to produce adequate paralysis for intubation. The patient will remain apneic for 30 to 35 minutes after an intubating dose of vecuronium. It can produce good intubating conditions within

1 minute at 2.5 times the normal dose. This results in paralysis for 1 to 2 hours. Maintenance of paralysis can be achieved with boluses of 0.02 mg/kg. The most troublesome side effect of this drug is prolonged paralysis up to several days following an infusion. This prolonged action is possibly a result of accumulation of its active 3-hydroxy metabolite.[97]

Rapacuronium is a newer nondepolarizing NMB agent with onset times and duration of action similar to those of succinylcholine. It was developed as a replacement for succinylcholine in the rapid sequence protocol. Rapacuronium was voluntarily withdrawn from the U.S. market by its manufacturer in 2001 due to reported incidences of fatal bronchospasm.

Succinylcholine and rocuronium are the muscle relaxants of choice for rapid sequence emergency intubations.[119]

MISCELLANEOUS AGENTS

LIDOCAINE

Lidocaine is a local anesthetic agent that has an antiarrhythmic effect on the heart. A dose of 1 to 2 mg/kg IV attenuates the increases in blood pressure, heart rate, ICP, and tachycardia associated with laryngoscopy.[120-123] It does not affect myocardial contractility in therapeutic doses. Lidocaine is usually administered to patients in whom the increases in blood pressure, heart rate, and ICP associated with direct laryngoscopy are undesirable.

ATROPINE

Atropine is a rapidly acting muscarinic acetylcholine receptor antagonist with significant vagolytic effect. It is commonly used as part of the Advanced Cardiac Life Support (ACLS) protocol during resuscitation of patients in cardiac arrest and those with symptomatic bradycardia at doses of 1.0 mg and 0.50 mg IV, respectively. Atropine is given during rapid sequence intubation of children using succinylcholine to prevent significant bradycardia and possibly asystole, which may occur much more frequently than in adults.[124] The dose is 0.01 mg/kg IV with a minimum dose of 0.10 mg IV and a maximum dose of 0.40 mg IV. Atropine has a secondary antisialagogue effect of reducing secretions produced in the respiratory tract and the salivary glands.

Atropine crosses the blood-brain barrier, and overdoses may lead to central atropine intoxication (i.e., central anticholinergic syndrome). This is characterized by agitation, restlessness, confusion, and hallucinations that may progress to stupor, seizures, and coma. Provide supportive treatment (e.g., seizure prophylaxis and mechanical ventilation). Reversal of this life-threatening condition can be achieved with IV physostigmine (i.e., an anticholinesterase agent with the ability to cross the blood-brain barrier).

GLYCOPYRROLATE

Glycopyrrolate is a quaternary amine of the muscarinic anticholinergic class.[125] It possesses significantly less of a vagolytic effect but greater antisialagogue effects when compared to atropine. **This makes it the drug of choice for premedication to reduce pharyngeal and tracheobronchial secretions.** Glycopyrrolate does not easily cross the blood-brain barrier due to its quaternary ammonium structure and makes the occurrence of central anticholinergic side effects much less likely. Do not administer glycopyrrolate to neonates due to the benzyl alcohol component, which can cause significant adverse effects. The recommended antisialagogue dose of glycopyrrolate is 0.004 mg/kg IM given 30 to 60 minutes prior to the intubation. It may be administered IV in boluses of 0.1 mg every 2 to 3 minutes as needed until the desired effect. It usually requires one or two doses during procedural sedation as an adjunct to prevent ketamine's effect of increasing respiratory tract secretions.

Glycopyrrolate is routinely administered with anticholinesterase medications during reversal of nondepolarizing neuromuscular blockade to counteract the unwanted side effects of bradycardia, bronchoconstriction, and intestinal hypermotility that accompany the ensuing increase in cholinergic activity.[125] It is administered along with neostigmine and pyridostigmine at a dose of 0.2 mg IV of glycopyrrolate for each 1 mg of neostigmine or 5 mg of pyridostigmine.[125] The onset is usually within 1 minute when given IV. The duration of the vagolytic and antisialagogue effects are 2 to 3 hours and up to 7 hours, respectively, when given IV.

DEXMEDETOMIDINE

Dexmedetomidine is a centrally acting alpha$_2$-adrenergic agonist similar to clonidine but with more selectivity for the alpha$_2$ receptor.[126] It causes dose-dependent sedation and anxiolysis and blocks the sympathetic response to stress and airway manipulation. It is increasingly being used with much success as an adjunct to awake fiberoptic intubation.[127] Its main indication is for sedation of intubated and mechanically ventilated patients.[128] Patients remain calm and sedated when left alone but are readily arousable when stimulated and follow commands.[126] Dexmedetomidine causes little to no respiratory depression and tachycardia.[129] The main side effects are hypotension and bradycardia.[130,131] Dosing consists of an initial loading dose of 1 mcg/kg IV in adults and 0.5 to 1 mcg/kg in children over 10 minutes followed by an infusion of 0.2 to 0.7 mcg/kg/hr titrated to the desired level of sedation.[132,133] Its half-life is about 6 minutes due to rapid redistribution. This short half-life has the potential for its use in awake intubations. It can be used for sedation in the magnetic resonance imaging scanner at 2 mcg/kg IV followed by an infusion of 1 mcg/kg/hr or 1 to 2 mcg/km IM.[134,135]

SUGAMMADEX

Sugammadex (Bridion) is a novel agent that can terminate a dense neuromuscular block due to rocuronium or vecuronium.[136-142] The recommended dose of 16 mg/kg based on total body weight can completely reverse a single dose of 1.2 mg/kg of rocuronium in as little as 2 to 2.5 minutes.[139,143-150] The drug is a cyclodextrin compound or a cyclic oligosaccharide carbohydrate that is formed from the degradation of starch.[151] It has a hydrophobic core surrounded by a hydrophilic outer rim. The hydrophobic core can trap other hydrophobic substances, encapsulate them, and render them inactive. Cyclodextrins administered IV are not metabolized and are excreted substantially by the kidney. The usual elimination half-life of 2 hours can be greatly prolonged up to 19 hours in patients with severe renal impairment, and use of the drug is not recommended in this patient population.[144,149,150]

Sugammadex is the first agent of its class. It was recently approved by the Food and Drug Administration for use in the United States. It has been used in Europe for several years. It has an affinity for the aminosteroid nondepolarizing NMB agents and has no interaction with succinylcholine or the benzylisoquinolone NMB agents (e.g., cisatracurium). Its affinity is strongest for rocuronium, followed by vecuronium then pancuronium.[151] It binds rocuronium in a 1:1 fashion to decrease rocuronium plasma concentrations.[151] Bound rocuronium cannot gain access to the motor end plate and cause paralysis. The reversal of the neuromuscular block results from the binding of plasma rocuronium and the rapid movement of rocuronium from peripheral sites to the plasma to maintain an equilibrium. The sugammadex-rocuronium complex is excreted in the urine.

A variety of adverse events have been reported with the use of sugammadex. The most significant are anaphylactic reactions

(0.3%), prolonged QT interval, and marked bradycardia occasionally resulting in cardiac arrest.[144] Sugammadex results in more hemodynamic stability than neostigmine.[152] The most common adverse reactions in > 10% of patients are vomiting, pain, nausea, hypotension, and headache.[144] Increases in the prothrombin time and international normalized ratio of up to 25% for up to 1 hour have been seen following a dose of 16 mg/kg. The clinical significance of this is uncertain. Studies in surgical patients have not demonstrated an increased risk of bleeding due to this effect.[144,149,150] It is recommended that coagulation parameters be carefully monitored in patients at risk for bleeding and in those being treated with anticoagulation. It is recommended that close monitoring of respiratory function be performed following reversal with sugammadex, although recurarization is unlikely following a dose of 16 mg/kg.[144,149,150] Sugammadex can cause bronchospasm in patients with an underlying pulmonary disease.[153] This drug exerts no action at nicotinic or muscarinic receptor sites. Muscarinic side effects (e.g., bradycardia and bronchoconstriction) are less likely.[144,149] Sugammadex binds to bone and teeth. The effect of this is unknown in growing children. The safety and efficacy of sugammadex have not been established in patients < 18 years of age. Despite this, it is safer than neostigmine.[154]

Sugammadex binds to and decreases plasma levels of other drugs with a steroid-based structure (e.g., hormones and oral contraceptives), rendering them ineffective. It is recommended that patients using hormonal contraceptives use an additional nonhormonal method of contraception for 7 days following administration of sugammadex.[144] Another drug interaction worth noting is with the oral estrogen receptor modulator toremifene. It may delay recovery with sugammadex in patients taking this medication.[144,149] There is no effect of sugammadex on cortisol and progesterone.[155] Sugammadex temporarily increases aldosterone and testosterone.[155]

Sugammadex has the potential for significant use in the Emergency Department. The biggest concern with using rocuronium for intubation is the length of time a patient is paralyzed if they cannot be intubated or ventilated. The development and availability of sugammadex provide a "safety net" to reverse rocuronium's effects in these situations.[154] **Sugammadex may eventually allow rocuronium to become the NMB agent of choice for rapid sequence intubation in the Emergency Department.**

Sugammadex can reverse rocuronium. The patient may still have the indications for airway management and ventilator management. Use of aminosteroid NMBs (i.e., pancuronium, rocuronium, and vecuronium) after sugammadex is very difficult. The use of isoquinoline NMBs (i.e., atracurium, cisatracurium, and mivacurium) and succinylcholine after sugammadex works well.

SUMMARY

Numerous pharmacologic agents are available to sedate, relax, and paralyze a patient in preparation for intubation. These same agents are used in lesser doses to maintain postintubation sedation and paralysis as well as for procedural sedation. There is no ideal sedative or paralytic agent. The combination of lesser doses of several agents will maximize the positive effects and minimize the adverse effects of each individual drug. This requires Emergency Physicians to become familiar with several drugs in each class so they may choose the appropriate combination for each patient.

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13 Endotracheal Medication Administration

Megan Johnson and Shoma Desai

INTRODUCTION

Health care providers are charged with the primary goal of optimizing the oxygenation, ventilation, and hemodynamic status of the patient during a resuscitation. In most cases, the first definitive intervention is to secure the airway through endotracheal (ET) intubation. The establishment of access to the systemic circulation soon follows. **Vascular access in certain patients can be problematic, and the ability to administer medications endotracheally can be lifesaving.**

The ET route of medication administration was first reported in 1857 for the alveolar absorption of curare in dogs.[1] This was followed in 1884 by the observation that ET instilled salicylates appeared in the urine.[2] The ET application of strychnine, atropine, chloral hydrate, and potassium iodide was studied in 1897.[2] The first suggestion to use the ET route for the therapy of pulmonary disease was made in 1915.[3] This idea resulted in a study in 1937 that recommended the use of inhaled epinephrine in asthmatics.[4] The rapidity of pulmonary absorption was eventually used for resuscitation purposes. In 1967, the equality of the ET, intravenous (IV), and intracardiac routes of epinephrine administration in the resuscitation of hypoxia-induced cardiorespiratory arrest in dogs was demonstrated.[5] This study prompted the routine use of the ET route for medication administration in emergent clinical situations.

The ideal drug delivery system to the lungs does not yet exist. It would have the following characteristics. Medications are aerosolized to a fine mist to increase the absorption through the alveoli. Avoid adherence of medication to the ET tube. Delivery to the lungs and not the bronchi, trachea, or ET tube. No risk of splash-back. No need to stop compressions or ventilations of cardiopulmonary resuscitation (CPR). We have achieved some of these characteristics.

ANATOMY AND PATHOPHYSIOLOGY

Most experiments involving ET medication administration have been conducted on subjects with normal cardiovascular function. There are still many questions remaining about the utility of this route in patients with cardiopulmonary arrest. The alveolar-capillary membrane is a highly absorptive surface. Numerous factors can undermine this potential. These include reduced pulmonary blood flow (e.g., less than 30% of normal during CPR), ventilation-perfusion mismatch, and compromised alveolar absorption (e.g., from pulmonary edema) in cardiopulmonary arrest.[6-8]

ET-administered medications are absorbed in a protracted manner during a cardiopulmonary arrest. This is termed the "depot effect" and is observed in laboratory and clinical experiments.[6] A study of 2 mg/kg ET lidocaine in nonarrest patients revealed a biphasic pattern of absorption, with an initial immediate peak and a second higher peak approximately 24 minutes later.[9] Plasma lidocaine levels were still measurable 120 minutes after instillation.[9] The relatively low initial and subsequent extended plateau of plasma medication levels is also exhibited by the ET administration of atropine, epinephrine, and vasopressin.[6,10] Local vasoconstriction induced by epinephrine may further contribute to this "depot effect."[11]

MEDICATION DOSE

The American Heart Association (AHA) recommends an ET medication dose of 2 to 2.5 times the recommended IV dose (**Table 13-1**).[12] The true optimal dose of ET-administered medications is unknown. There is a consensus that IV doses given endotracheally result in subtherapeutic plasma levels.[13-43] The equipotent dose ranges from 3 to 10 times the IV dose.[13-43]

Many studies have noted the inadequacy of the currently recommended dose of epinephrine and atropine in cardiopulmonary arrest models.[28,32,37] The rates of return of spontaneous circulation were significantly greater in the IV medication group than in the ET medication group and in the no therapy group.[28]

A discussion in the literature has drawn attention to the behavior of epinephrine when given at suboptimal doses. Epinephrine is used in arrest situations for its α-adrenergic effects. However, the β-adrenergic effects predominate when epinephrine is administered

TABLE 13-1 Recommended Endotracheal Medication Doses

Medication	Adults	Pediatrics	Neonates
Atropine	2–2.5 mg	0.03 mg/kg	Unknown (use pediatric dose)
Epinephrine	2–3 mg (1:10,000)	0.1 mg/kg (1:1000) (or 0.1 mL/kg)	0.01–0.03 mg/kg (1:10,000)
Lidocaine	2–3 mg/kg	2–3 mg/kg	Unknown (use pediatric dose)
Naloxone	0.8–2 mg	< 5 years or ≤ 20 kg: 0.1 mg/kg ≥ 5 years or > 20 kg: 2 mg	0.1 mg/kg
Vasopressin	80 U	Unknown	Unknown

in small doses. This can transiently result in hypotension, decreased coronary perfusion pressure, and a diminished likelihood of return of spontaneous circulation.[12] An early drop in mean arterial blood pressure with prolonged tachycardia in healthy dogs was documented after ET epinephrine at doses of 0.02, 0.035, 0.1, and 0.2 mg/kg.[29] Only the 0.3 mg/kg dose was effective in achieving an increase in blood pressure without a transient drop.[29] Findings such as these have prompted investigations on pretreatment with beta-blockers (e.g., propranolol) prior to ET epinephrine administration to blunt the early beta-receptor effects.[30,31] **These studies do question the efficacy of the current recommended ET epinephrine dose and call for higher ET doses.**

Concerns regarding the "depot effect" have kept the current ET medication dose recommendation at 2 to 2.5 times the IV dose. This is to avoid the prolonged side effects in the critical postresuscitation period.[6] This compromise in dosage leads to uncertainty regarding the effectiveness and reliability of the ET route for medication administration.[10,32,33]

DILUENT

A primary goal of ET medication administration is to achieve rapid absorption. This goal can be reached with larger volumes of diluent but is tempered by the potentially detrimental effects on pulmonary function.[5] **The current recommended volumes for the ET route are dilutions to a total volume of 10 mL in adults, 5 mL for children, and 1 mL for neonates.**[12,27]

Another controversy arises with respect to the type of diluent used. Distilled water and 0.9% saline have both been used for ET medication administration and are currently recommended by the AHA.[12] Water creates a greater osmotic gradient in theory and potentially speeds up medication absorption. Water also leads to a greater disruption of pulmonary surfactant and hinders gas exchange.[6] The latter was confirmed in nonarrest experiments using dogs.[34] It was later disputed using much smaller volumes of instilled medication. These studies found that distilled water provides more rapid and more effective absorption (i.e., higher peak serum medication levels) than 0.9% saline without a physiologically significant drop in PaO_2.[35,36]

PEDIATRIC CONSIDERATIONS

Obtaining vascular access in children, especially in neonates, can be extremely difficult. This makes the ET medication administration route an important consideration in early resuscitative efforts.[38] There are no published pediatric or neonatal studies examining the type or volume of diluent, only consensus recommendations.[12,27]

A major difference between adults and children in arrest is the absorptive capacity of the lung. The immature lung's surface area is smaller and the diffusion coefficient is larger. There exists the potential for right to left cardiac shunts in children.[10,39] These conditions greatly affect an already unreliable absorptive process.

The indications, procedure, and complications are much the same in pediatrics as in adults. There are very few published studies focused on ET medication administration in pediatrics. Those involving epinephrine in pediatric arrest agree that it takes 10 times the IV medication dose to achieve appropriate serum epinephrine levels, responses in heart rate, and responses in blood pressure.[27,39,40] The AHA recommends 2 to 3 times the dose of lidocaine, atropine, or naloxone and 10 times the dose of epinephrine (i.e., 0.1 mg/kg of 1:1000 concentration) when using the ET route.[12]

A retrospective study of neonates who received epinephrine in the delivery room underscores the difficulty of obtaining vascular access.[41] Almost all the neonates received their first dose of epinephrine via the ET route. Almost one-third of the neonates obtained return of spontaneous circulation, while the rest required additional epinephrine through their IV line once it was established. The authors concluded that the currently recommended ET epinephrine dose of 0.01 to 0.03 mg/kg is ineffective. In the neonatal setting, the AHA states that ET administration of higher dose epinephrine (i.e., 0.05–0.1 mg/kg of 1:10,000 concentration) can be considered, but cautions that the safety and efficacy have not been evaluated.[12]

INDICATIONS

The ET administration of medication is reserved for resuscitations in which vascular access (i.e., IV, intraosseous, or central line) is delayed.[12] The ET route may be more frequently used in certain patient populations traditionally associated with difficult vascular access such as neonates, morbidly obese patients, IV drug abusers, burn patients, and dialysis patients.[13] **The goal is to increase the probability of successful resuscitation despite a delay in obtaining definitive systemic access. This may soon be outdated with the widespread use of intraosseous access (Chapter 70).**

The ET route is recommended for the administration of lipid-soluble lidocaine, epinephrine, atropine, naloxone, and vasopressin. There are no data to address the ET administration of amiodarone.[12] Other medications that can be administered but are not widely used include flumazenil, diazepam, midazolam, penicillins, sulfonamides, and aminoglycosides.[14-18]

There is minimal discussion in the literature about the use of ET epinephrine for decompensating and near-fatal asthma. This practice may be considered in moribund patients unresponsive to conventional treatment.[42-44]

CONTRAINDICATIONS

The absorption of medication given through an ET tube is unpredictable during a cardiopulmonary arrest. The only contraindication to ET medication administration is the establishment of venous access (i.e., IV, intraosseous, or central line).

EQUIPMENT

- ET tube
- Bag-valve-mask
- Medication to be administered
- Diluent, 0.9% normal saline or distilled water
- 10 mL syringe
- 18 gauge needle
- IV adapter lock
- Water-soluble lubricant
- Optional catheters (i.e., suction, feeding, or central venous)

The patient may be intubated with a standard ET tube, an ET tube with a monitoring lumen, or a specialty ET tube with a medication injection port.[19] The EDGAR (Endobronchial Drug and Gas Application during Resuscitation, Rusch, Germany) tube has a separate injection channel that terminates at the tip of the tube (**Figure 13-1*A***). This ET tube is currently available in Europe but not in the United States. The Stat-Med ET tube (Hudson RCI, Temecula, CA) has a separate injection port and channel that terminates distal to the inflated cuff (**Figure 13-1*B***). Both ET tubes allow the ET administration of medications without interrupting CPR compressions or ventilation. The LITA ET tube (Hudson RCI, Temecula, CA) is a modification of the Stat-Med ET tube (**Figure 13-1*C***). It has a separate injection port and channel. The channel has eight openings above the cuff and two below the cuff (**Figure 13-1*C***). This ET tube was designed for use with local anesthetic solutions to

A

B

C

FIGURE 13-1. Medication administration ET tubes. ***A.*** The EDGAR tube. ***B.*** The Stat-Med tube (Hudson RCI, Temecula, CA). ***C.*** The LITA tube. (Used with permission from Andrzejowski J, Francis G. The efficacy of lidocaine administered via the LITA tracheal tube in attenuating the extubation response in beta-blocked patients following craniotomy. *Anesthesia* 2002;57:399-401.)

anesthetize the tracheal mucosa. It should not be used to administer resuscitation medications. They will mostly pool above the cuff in the trachea and not get properly absorbed. These specialized ET tubes are rarely available in U.S. Emergency Departments.

Other equipment may be used to administer the medications. A suction, feeding, or central venous catheter may be introduced into the ET tube to administer medications more distally. An IV adapter lock is required on the proximal end of these catheters to allow the syringe to attach to the catheter. There are no formal guidelines regarding proper catheter size; however, it is recommended to use a 16 French (Fr) suction catheter in adults, an 8 Fr feeding catheter in children, and a 5 Fr feeding catheter in neonates.[20-22]

The administration of drugs through laryngeal mask airways (LMAs; Chapter 26) and Combitubes (Chapter 21) as compared with ET tubes has been studied. Medication instilled via a catheter inserted in an LMA and into the trachea achieved the equivalent blood concentrations as through an ET tube.[45] It is often difficult to successfully pass a catheter through an LMA and into the trachea.[23] Administration of lidocaine through LMAs was unreliable when compared with ET tubes in nonarrest patients.[23] Subtherapeutic plasma medication levels were noted when comparing lidocaine administration via Combitubes placed in the esophagus versus ET tubes.[24]

The intubating LMA (ILMA) is a modification of an LMA. It is designed to allow the blind passage of an ET tube through it and into the trachea. Insertion of a catheter through an ILMA was only successful in 92% of attempts.[46] It also required a mean time of 20 seconds (range of 11 to 44 seconds), which is too long by AHA guidelines to halt CPR.[12]

An interesting device is the LMA MADett or the Endotracheal Tube Mucosal Atomizer Device (Wolfe Tory Medical, Morrisville, NC; **Figure 13-2**). This device allows ET medication administration without stopping compressions and ventilations of CPR and without splash-back. The MADett must be used only with an ET tube size ≥ 7.0 mm inner diameter and a length of at least 28 cm.

TECHNIQUES

PROXIMAL MEDICATION ADMINISTRATION

The simplest method for ET medication administration is direct instillation. Prepare the medication or use a prefilled syringe with a needleless adapter. Use a syringe with an 18 gauge needle to draw up the medication. Remove the 18 gauge needle from the syringe. Draw up sterile water or 0.9% normal saline to dilute the medication to the appropriate volume. Remove the bag-valve device from the ET tube. Briefly interrupt external chest compressions. Inject the

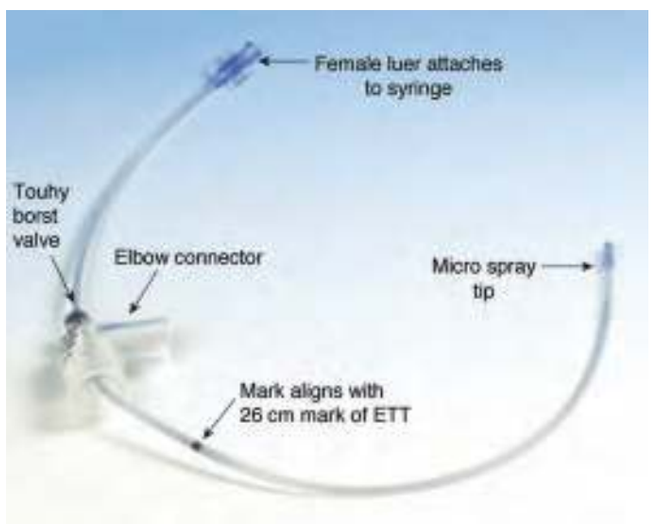

FIGURE 13-2. The MADett. ETT, endotracheal tube. (Courtesy of Teleflex Medical.)

diluted medication into the proximal end of the ET tube. Replace the bag-valve device and manually ventilate five times in quick succession. The forced hyperventilation results in bilateral and distal distribution of the medication.[13] Resume CPR.

Always remove the needle from the syringe when injecting medication into the ET tube. Some medical personnel may choose to leave the needle attached to the syringe for convenience or to decrease any delays in medication administration. The needle can fall off the syringe and into the ET tube. It will then "ride the tube" and can become a foreign body in the distal trachea or main bronchus. The needle slows down medication injection into the ET tube and delays the resumption of CPR.

PROXIMAL NEEDLE INJECTION INTO THE ENDOTRACHEAL TUBE

An alternative approach is to piercing the proximal end of the ET tube with an 18 gauge needle attached to the medication-filled syringe.[25] Care must be taken to assure the needle does not damage the ET cuff inflation tube and that the tip of the needle is completely within the lumen of the ET tube. **Instill the medication during inspiration when the bag is squeezed.** Detach the syringe from the needle. Attach a new medication-filled syringe or refill the used syringe to instill other medications without removing the needle from the ET tube. The remaining hole in the ET tube after the needle is removed results in a negligible air leak that may be closed with a piece of tape.

DISTAL MEDICATION ADMINISTRATION

Medications may be administered deeper through a catheter inserted into the ET tube. This technique minimizes medication adherence to the walls of the ET tube. Prepare the medication and catheter. Draw up the appropriate medication and diluent into a syringe or use a prefilled syringe. Choose the appropriate type and size of catheter. Attach an IV adapter lock to the proximal end of the catheter. The proximal end of the catheter may have to be cut off if it is flared, tapered, or too big for the IV adapter lock to fit.[26]

Lightly lubricate the catheter with water-soluble lubricant so it will advance easily through the ET tube. Remove the bag-valve device from the ET tube. Briefly interrupt external chest compressions. Insert the catheter (i.e., feeding, central line, or suction) into the ET tube until its tip extends just beyond the distal end of the ET tube by approximately 1 to 2 cm. Attach the medication-filled syringe to the IV catheter lock if using a feeding or suction tube or the Luer lock if using a central venous catheter. Inject the medication into the catheter followed by 5 mL of air to clear the tube of any residual medication. Replace the bag-valve device and manually ventilate five times in quick succession. The forced hyperventilation results in bilateral and distal distribution of the medication.[13] Resume CPR.

TECHNIQUES TO MINIMIZE COMPRESSION AND VENTILATION INTERRUPTION

The goal of CPR is to minimize interruptions of chest compression and ventilation. This can be accomplished using one of the specialized ET tubes discussed in the equipment section of this chapter. These ET tubes may be placed initially in a patient with a cardiopulmonary arrest or exchanged for a standard ET tube. Prepare the medication. Attach the medication-filled syringe to the injection port of the ET tube. **Inject the medication during inspiration with the bag-valve device.** The diluted drug solution may be followed by the injection of 5 mL of air to clear the ET tube of any residual medication.

An alternative to using these specialty ET tubes is to attach the MADett to a standard ET tube. The MADett is quick, simple, and easy to use (**Figure 13-3**). The patient should already be intubated. Stop ventilations, but not compressions, and remove the bag-valve device from the ET tube. Attach the MADett adapter onto the ET tube (**Figure 13-3*A***). Resume ventilations by attaching the bag-valve device to the side port of the adapter. Advance the MADett catheter into the ET tube until the black mark on the catheter lines up with the 26 cm mark on the ET tube (**Figure 13-3*B***). Tighten the lock nut on the MADett adapter to hold the catheter in place so it will not be advanced or withdrawn. Attach the medication-filled syringe to the Luer lock. **Inject the medication while ventilating the patient (Figure 13-3*C*).** Injecting the medication while bagging the patient will allow the atomized medication (**Figure 13-3*D***) to penetrate deeply into the respiratory tract.

WHICH IS THE BEST TECHNIQUE?

Mielke et al.[26] studied the time involved to perform each technique by Paramedics and Emergency Physicians. These included direct injection into the ET tube, use of a suction catheter, use of a central venous catheter, and use of an EDGAR tube with an injection channel. The direct injection into the ET tube and use of the EDGAR tube were significantly faster than the catheter techniques. A theoretical disadvantage of using a catheter was the instillation of medication unilaterally down one of the main stem bronchi if the catheter was advanced too far down the ET tube. This would thereby decrease the potential absorptive surface by half. This potential negative was offset by larger medication volumes and the application of postinstillation hyperventilation.[26]

No difference was found in nonarrest patients in the pharmacokinetic response to lidocaine administration with the same three techniques (i.e., direct ET instillation, suction catheter, and EDGAR tube).[20] There was no significant difference in serum lidocaine concentration, heart rate, blood pressure, end-tidal PCO_2, and oxygen saturation among the three groups. These findings disputed any previous assertion that using a catheter provided an advantage by instilling medications more peripherally and closer to the absorptive surface. Catheter use led to a significantly longer interruption in ventilation compared to the direct ET instillation group.[20]

These techniques were evaluated in a porcine model simulating pediatric respiratory arrests.[21] Radio-labeled epinephrine was instilled through an ET tube, a feeding catheter, and an ET tube with a monitoring lumen. There was no significant difference in hemodynamic response or in the rate of successful resuscitation. The medication adherence to the ET tube was minimal. There was no difference in bilateral pulmonary distribution between the three groups.

ASSESSMENT

Large volumes of fluid in the lung may interfere with oxygen exchange or cause pneumonitis or acute pulmonary edema. Direct the assessments to the resuscitation itself (e.g., chest compression, positive-pressure ventilation, defibrillation). There is no method by which plasma levels of these medications could be obtained rapidly enough to impact clinical care.

AFTERCARE

There is no indication to remove one of the specialty ET tubes if used. These can be left in place and used as one would use a standard ET tube. Place a piece of tape over the hole made by the needle in the ET tube if the needle injection technique was used.

A

B

C

D

FIGURE 13-3. Using the MADett. ***A.*** The MADett is attached to the ET tube. ***B.*** The black mark on the catheter lines up with the 26 cm mark on the ET tube where the finger points. ***C.*** Medication is injected while ventilating the patient. ***D.*** Medication is atomized into the airway. (Photos courtesy of Teleflex Medical.)

COMPLICATIONS

The main complication of administering medications by the ET route arises from the "depot effect." Epinephrine, atropine, vasopressin, and lidocaine have been observed to be absorbed in a prolonged fashion, much like a continuous IV infusion. This phenomenon has been attributed to local vasoconstriction (i.e., epinephrine), poor lung perfusion, vascular congestion due to diminished cardiac output, and comorbid conditions (e.g., pulmonary edema, atelectasis, chronic obstructive pulmonary disease) present at the time of the arrest. This sustained drug effect has also been observed in nonarrest patients.[9] The result of epinephrine administration is prolonged hypertension, malignant arrhythmias, and tachycardia in the postresuscitative period.[37,45] ET atropine can cause sustained tachycardia.[6] ET vasopressin can cause sustained bradycardia.[10] A canine study suggested that pretreatment with an IV beta-blocker prior to ET administration of epinephrine or norepinephrine protected against tachycardia and mildly enhanced the increase in diastolic blood pressure associated with epinephrine.[47] Another nonarrest canine model suggested that endobronchial rather than ET epinephrine may result in less of the unwanted hemodynamic effects.[48] We do not have a reliable way to deliver endobronchial medications in the Emergency Department.

A drawback to ET medication administration is the transient impairment of gas exchange. Many studies describe an early reduction in PaO_2 that is directly correlated with the volume of liquid instilled. There is a greater drop in PaO_2 after instilling water than after saline. In nonarrest dogs using large volumes of diluent (2 mL/kg), there was a decrease in PaO_2 to 61% of baseline with distilled water, which was a substantially greater reduction than the decrease in PaO_2 seen with saline.[34] In nonarrest humans with instilled volumes of 10 mL through the ET tube,

there was a drop in PaO_2 from 157 to 95 mmHg with saline and from 157 to 103 mmHg with water.[35] Hypoxia, to a much greater extent than hypercarbia, has been reported in nonarrest models. Alterations in pulmonary gas exchange in arrest are less well understood. **Adherence to recommended volumes and the use of hyperventilation after instillation has the potential to offset this complication.**

SUMMARY

Despite concerns regarding the efficacy of ET medication administration, this route remains a practical consideration for instances in which vascular access is not yet established for the instillation of resuscitation medications. The ET route will increase the chances of successful return of spontaneous circulation when venous access is delayed. Attempts at vascular access should be continued during the administration of ET medications, and the use of the ET route should be discontinued once access is achieved. Despite its inherent issues, knowledge of the ET medication administration procedure can be lifesaving.

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14 Management of the Difficult Airway

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INTRODUCTION

The definition of a difficult airway has remained nebulous despite ongoing international effort due to the complexity and variety of factors constituting this phenomenon. The latest practice guidelines by the American Society of Anesthesiologists (ASA) simply define a difficult airway as an airway that, to a conventionally trained provider, presents a challenge in terms of intubation, ventilation, or both.[1] The reported incidence of a difficult airway in the literature varies. Difficult mask ventilation occurs in approximately 2% of cases, with impossible mask ventilation occurring in 0.15% of cases.[2,3] Difficult intubation occurs in approximately 5% of cases, with impossible intubation (i.e., failed intubation after multiple attempts at direct laryngoscopy) occurring in 0.3% to 0.5% of cases.[4] The incidence of both occurring in combination is very low, at less than 2 in 10,000 intubations.[2]

Failure to control an airway in a timely and reliable manner has been associated with a high degree of morbidity and mortality.[5,6] Attempts to predict difficulty with airway management and mitigate its impact have met limited success. The ASA proposes 11 anatomic "predictors" of difficulty for intubation and five for mask ventilation.[1] No specific group of these "predictors" has been able to reliably identify problematic airways in advance.[6-8] Complex algorithmic models leave a wide "gray area" of uncertainty.[9] Most difficult airways appear to be unanticipated, even with use of these models.[7] This leaves the Emergency Physician with a critically important dilemma while under a significant time constraint.

The focus has slowly shifted away from prediction and toward standardized algorithmic approaches. The ASA difficult airway algorithm and the Difficult Airway Society (DAS) algorithms were groundbreaking developments. They were criticized for containing multiple decision points that added complexity without improving outcomes.[10] The favored approach has been extensive training in an institutionally defined, forward-only algorithm for management of the difficult airway.[10,11] This is often combined with a dedicated team of providers proficient in its use.[12,13] This works for Anesthesiologists but not for Emergency Physicians working in Emergency Departments with limited backup.

No single method has been endorsed by airway societies. The above approach remains promising due to several key advantages. It offers clear steps in a timely progression to minimize harm and maximize success.[14,15] An "open-box" approach allows for substitution of locally available tools and techniques within the chain of progression.[13] Standardized simulation training using such algorithms has demonstrated improved compliance and patient outcomes.[15-17] **Availability of the necessary tools and proficiency in their use are the keys to maximizing success in managing the difficult airway.[18,19]** However, the tools and proficiency are often lacking in many instances.[18,19]

ANATOMY OF THE DIFFICULT AIRWAY

A full overview of airway anatomy and its evaluation is contained in the on orotracheal intubation and essential anatomy of the airway (Chapters 9 and 18). **Successfully anticipating the difficult airway may allow the Emergency Physician to select an approach most likely to succeed on the first attempt and to prepare alternative approaches if needed.** Numerous potential predictive factors of difficulty have been identified in the literature. The most common predictive factors are those put forth by the ASA and are listed in **Table 14-1**.[1] An acceptable and less cumbersome alternative in the Emergency Department are the LEMON criteria (**Table 14-2**).[20] The LEMON criteria provide an effective and rapid way of assessing an airway for potential difficult.

TABLE 14-1 ASA Predictors of a Difficult Airway

A high, arched palate
A short, thick neck
Limited cervical mobility
Limited mandibular protrusion
Long upper incisors
Mallampati class III or IV
Prominent overbite
Short thyromental distance
Small mouth opening

Source: From reference 1.

These risk factors individually have a poor positive predictive value.[21] **A greater number of suggestive factors increases the likelihood of encountering a difficult airway.** No strict cutoff for the number or combination of predictors has been established. Multiple risk factors should raise suspicion for potential difficulty and the need for flexible endoscopic (Chapter 28) or video-assisted techniques (Chapter 20).

Difficult mask ventilation may result from any factor interfering with proper formation of the face mask seal or excessive resistance to flow of gas. Difficult mask ventilation traditionally has five independent risk factors.[3] These are age > 55 years, body mass index > 26 kg/m^2, presence of a beard that interferes with formation of a tight seal, edentulous state, and history of snoring. The edentulous state has been a commonly cited risk factor because the teeth contribute to the rigid framework that braces facial soft tissue from behind and allows for the creation of a tight seal. More recent work has questioned its contribution to difficult ventilation.[3] Edentulous effects may be mitigated by maneuvers (e.g., the jaw-thrust), and limited mandibular protrusion is a more appropriate risk factor for inclusion.[3]

INDICATIONS

The indications for the application of difficult airway management techniques are the same as those outlined in the orotracheal intubation chapter (Chapter 18). **Difficult airway management techniques are specifically indicated in situations where the initial attempt or technique proves unsuccessful or if difficulty in airway management is anticipated based on the airway examination.**

CONTRAINDICATIONS

The contraindications for application of these techniques and algorithms are listed in the chapters that discuss the specific techniques.

TABLE 14-2 The LEMON Criteria

L – Look externally. Note any craniofacial abnormality and pathology, an edentulous oral cavity, obesity, grossly excessive soft tissue, buck teeth, or a narrow mouth.
E – Evaluate the patient using the 3-3-2 rule. The interincisor distance and hyomental distance should be > 3 fingerbreadths. The thyrohyoid distance should be > 2 fingerbreadths.
M – Mallampati score. Class III (i.e., soft palate and base of the uvula is visible) and IV (i.e., only the hard palate is visible) are nonreassuring findings.
O – Obstruction. Note any pathology within or surrounding the upper airway.
N – Neck mobility. Note if the range of motion is less than 35° at the atlantooccipital joint.

Source: From reference 21.

TABLE 14-3 Items Recommended for Inclusion in a Difficult Airway Management Cart or Kit

Airway anesthesia equipment
Antifog solution
Bag-valve devices, various sizes
Bougies or introducers
Devices to confirm proper endotracheal tube placement
Endotracheal tube guides (e.g., semirigid stylets and ventilating tube changer)
Endotracheal tubes, assorted sizes
End-tidal CO_2 detectors, colorimetric
Face masks, various sizes
Fiberoptic bronchoscope and associated equipment
Flexible fiberoptic intubation equipment
Forceps, various sizes and styles
Lighted stylets
Lubricant
Positive end expiratory pressure valves
Rigid laryngoscope blades of alternate design and size
Scalpels, various sizes
Supraglottic airways (e.g., laryngeal mask airway [LMA] or intubating LMA of assorted sizes)
Surgical airway equipment suitable
Syringes, various sizes
Video laryngoscope

EQUIPMENT

The basic equipment for management of the difficult intubation is similar to that recommended in the orotracheal intubation chapter (Chapter 18). Items recommended for inclusion in a difficult airway management cart or kit are listed in **Table 14-3**.

PATIENT PREPARATION

Preoxygenation or the provision of supplementary oxygen prior to airway manipulation is an important step in airway management. It raises the patient's peripheral capillary oxygen saturation as close to 100% as possible, denitrogenates the blood, and denitrogenates the residual volume of the lungs.[22,23] **Use the method of delivery that allows for the highest FiO_2 possible under the circumstances.**[24] Patients with an adequate respiratory drive should receive 3 minutes of preoxygenation or take eight vital capacity breaths with maximal exhalation and inhalation. Noninvasive positive-pressure ventilation (e.g., continuous positive airway pressure [CPAP]) via mask connected to a bag-mask device with a positive end expiratory pressure valve may be superior for those unable to achieve acceptable saturations (e.g., > 93% to 95%) with standard techniques.[24] Positioning the patient in a head-elevated position, especially for the obese, may be beneficial compared to laying them supine during preoxygenation. The provision of apneic oxygenation with maximal flow of oxygen delivered via nasal cannula with or without nasopharyngeal airway insertion may further extend the duration of safe apnea.[24] These modalities may be combined in high-risk patients.

The remainder of the preparation for airway management is similar to that described in the orotracheal intubation chapter (Chapter 18). Device-specific preparation is described in the appropriate chapter. The Emergency Physician should have the equipment necessary for rapid and sequential progression from one technique to the next in case of failure to successfully control the airway (**Figures 14-1, 14-2, 14-3, and 14-4**).[23]

Difficult airway carts arranged in a sequential manner that reflects the order in which they would be used have been recommended by the ASA and the DAS. A designated kit or cart should be used given that equipment availability is a known impediment in airway management.[25]

The recommended medications are noted in Chapters 12, 16, 18, and 28. Propofol provides superior conditions for airway management and suppresses laryngeal reflexes. Neuromuscular blocking agents can facilitate ventilation, abolish laryngeal reflexes, and increase tissue compliance. Succinylcholine at 1.0 to 1.5 mg/kg intravenously is commonly used in rapid sequence intubation.[26] Rocuronium may be preferable in patients at high risk of desaturation as succinylcholine appears to decrease the time to desaturation due to fasciculations, increasing oxygen utilization.[24] The essential components of awake airway management include psychological

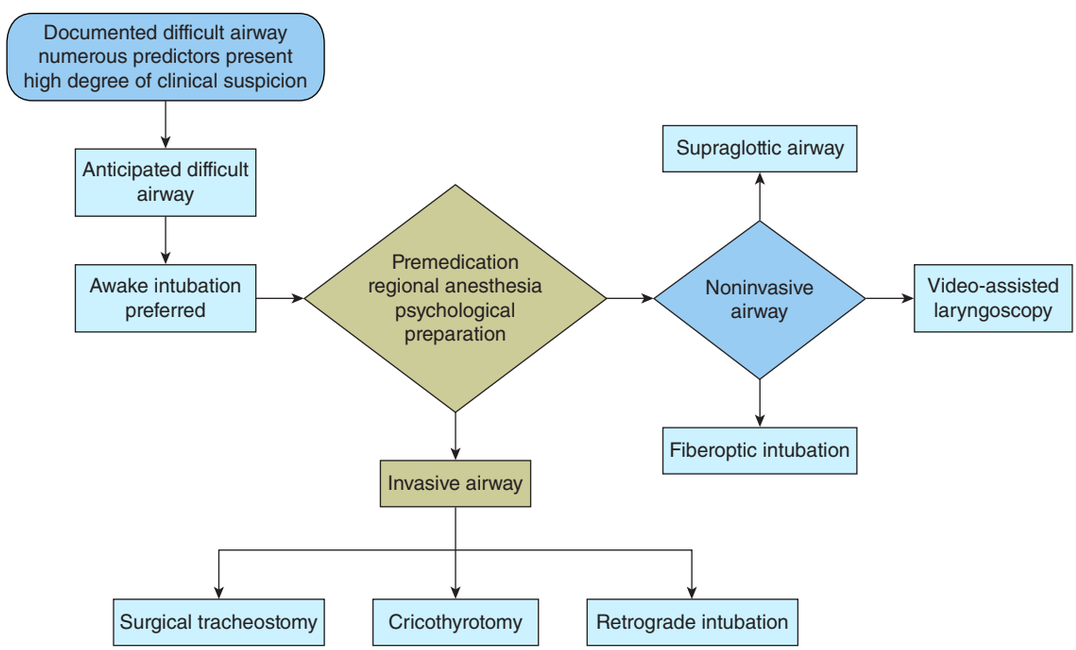

FIGURE 14-1. Basic algorithm for progression through the anticipated difficult airway.

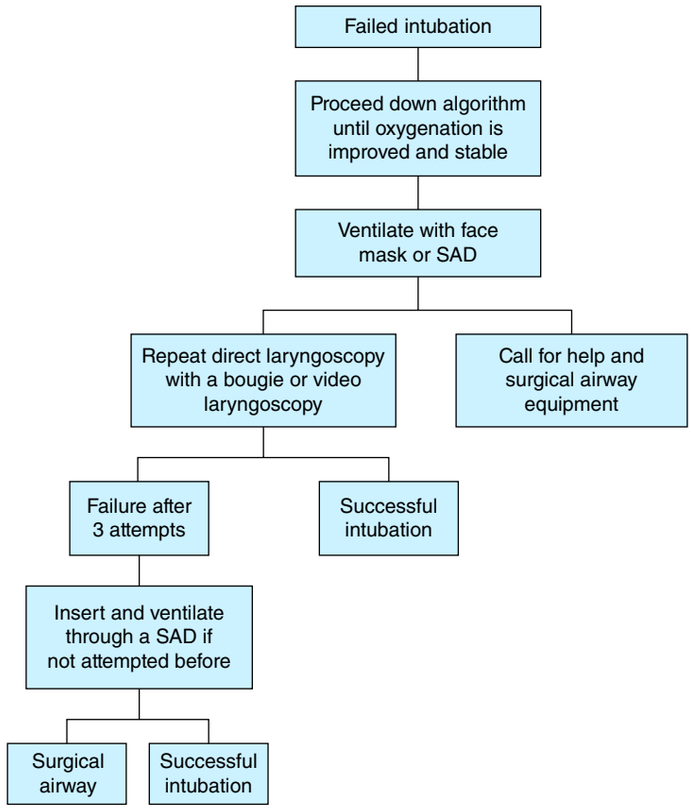

FIGURE 14-2. Example algorithm for progression through an unanticipated difficult airway. SAD, supraglottic airway device.

preparation of the patient, the use of antisialagogues (e.g., glycopyrrolate), provision of anxiolysis and sedation (e.g., midazolam and dexmedetomidine), and topical and/or regional anesthesia with lidocaine. This applies to fiberoptic bronchoscopic intubation, video laryngoscopy intubation, supraglottic airway placement, and surgical airway placement.

OPTIMAL PATIENT POSITIONING

The default position during direct laryngoscopy is the "sniffing position." It is achieved by 35° cervical flexion and maximal extension of the atlantooccipital joint.[26] This position allows for theoretical alignment of the oral, laryngeal, and pharyngeal axes for optimal visualization of the vocal cords. It has become the standard of practice in direct laryngoscopy since its detailed description in 1944.[27] Some recent efforts have called the scientific basis and superiority of this technique into question.[28,29] The sniffing position reliably reduces intubation difficulty.[30] The sniffing position is an appropriate initial choice for patients with limited cervical mobility and the obese.[28,31] Simple head extension may be sufficient to obtain a satisfactory view for the average-sized patient.[28]

The increased anteroposterior diameter of the chest in the obese population makes the standard sniffing position virtually impossible to attain without anterior displacement of the shoulders. The "ramped" position is achieved by using a specialized wedge-shaped device or a stack of blankets to elevate the upper torso and align the sternal notch with the external auditory meatus in the horizontal plane. **The ramped position has been demonstrated to improve laryngeal views in obese patients and should be routinely used.**[32-35] **Consider the ramped position for all obese patients undergoing direct laryngoscopy given that obesity is associated with difficult mask ventilation, faster desaturation, lower tolerance for apnea, an increased risk of difficult intubation, and difficult emergency tracheotomy.**[3,23,36-41] Further description of the management of the obese airway is contained in Chapter 15.

TECHNIQUES

The Emergency Physician may default to an approach most likely to successfully navigate the anatomic and physiologic complexities if a difficult airway is accurately anticipated and encountered under controlled conditions. A simple outline for approaching the anticipated difficult airway is presented below.

THE ANTICIPATED DIFFICULT AIRWAY

NONINVASIVE AIRWAY ACCESS

It is important to realize that there are numerous options for management of the anticipated difficult airway (**Figure 14-1**). Selection of the technique most likely to be successful based on the Emergency Physician's comfort and familiarity and the patient characteristics is appropriate if adequate time is available. Many of the techniques can be performed while the patient is awake given the proper preparation for the anticipated difficult airway. Awake intubation allows the Emergency Physician to maintain spontaneous oxygenation and ventilation, minimize the hemodynamic impact of sedating medications, preserve normal muscle tone, and navigate the anatomic architecture of the upper airway, while allowing the patient to maintain protective airway reflexes.[42] Noninvasive and invasive techniques may be appropriate for a complicated airway. Fiberoptic intubation via flexible bronchoscopy (Chapter 28) is often the approach of choice and may be considered the gold standard for expected difficult airway management.[43] Fiberoptic intubation may

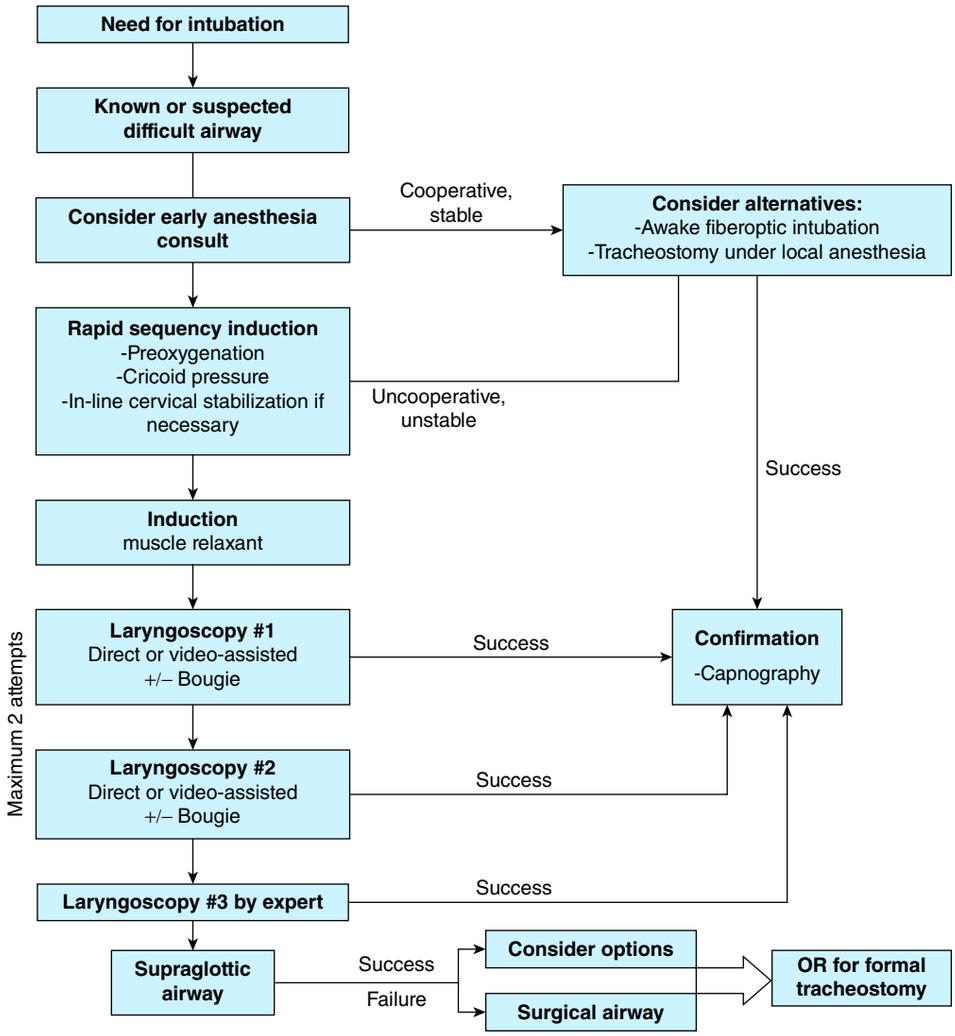

FIGURE 14-3. An institutional difficult airway algorithm. OR, Operating Room.

not always be a practical or readily available technique because it requires a dedicated tower to provide an external light source and monitor. Alternatives are using the single-use aScope (Ambu, Ballerup, Denmark) or rigid bronchoscopy, which permits simultaneous ventilation.

Video-assisted orotracheal intubation devices (e.g., GlideScope, Bonfils, Shikani, and Airtraq) represent a simpler alternative to fiberoptic bronchoscopy that circumvents the limitations of direct laryngoscopy (Chapters 20 and 21). These may be channeled (i.e., with the device and endotracheal tube moving as a single unit) or nonchanneled (i.e., with the endotracheal tube directed with the assistance of a stylet). Video laryngoscopes usually have self-contained light emitters, dedicated monitors, and power sources; require less dedicated equipment and space than fiberoptic bronchoscopes; and are easy to incorporate into clinical practice.[44] **Current literature suggests their usefulness, although not their superiority, in difficult airway management.**[43,45] A wide variety of video laryngoscopes are available.

The supraglottic airway device (SAD) is useful in the anticipated difficult airway (Chapters 25 and 26). A SAD may be used for the awake and sedated patient. The intubating laryngeal mask airway (e.g., ILMA or LMA-Fastrach) is a type of SAD that allows for ventilation and oxygenation while serving as a conduit for passage of an endotracheal tube.[46,47] The ILMA has become an important part of difficult airway management due to its high success rate in the adult and pediatric population.[48,49] Numerous alternatives to the ILMA exist (e.g., LMA CTrach with built-in fiberoptic imaging and the ProSeal, which supports higher seal pressures and provides access to the esophagus).[43] **SADs may be used when fiberoptic intubation fails.**[50] Fiberoptic intubation and ILMA demonstrate a high success rate and can be complementary to each other.[51] They can be used in conjunction or sequentially if the first attempt is unsuccessful.

INVASIVE AIRWAY ACCESS

Invasive airway placement may be appropriate. Retrograde intubation involves cannulation of the trachea via the cricothyroid membrane and the insertion of a guidewire to serve as a conduit for intubation (Chapter 30). The technique can be performed under local anesthesia. It negates the need for visualization and navigation of the laryngeal inlet. It can be very useful in overcoming significant anomalies and distortions in airway anatomy, circumventing excessive blood and secretions, and as a rescue technique after other techniques (e.g., direct laryngoscopy, SADs, and flexible bronchoscopy) have failed.[52-54] Surgical airways (e.g., cricothyrotomy or

FIGURE 14-4. An example of a difficult airway cart. Note the drawers are clearly labeled.

tracheostomy) may be performed if clinically warranted, if there is availability of the necessary equipment, and if a trained Emergency Physician is present.[54]

THE UNANTICIPATED DIFFICULT AIRWAY

NONINVASIVE AIRWAY ACCESS

Most encountered difficult airways are unanticipated and often encountered under emergent conditions.[7] The emergent nature of airway management may preclude elaborate evaluation, planning, and utilization of unfamiliar and labor-intensive techniques. Numerous algorithms and approaches exist in literature. They commonly begin with direct laryngoscopy followed by SAD insertion and terminate in invasive airway placement (**Figure 14-2**). **Memorization of a specific sequence is potentially helpful but not as important as understanding the logic behind it. The focus is on reestablishment and optimization of ventilation by rapid progression through available techniques from the least invasive to the most invasive.**

DIRECT AND VIDEO LARYNGOSCOPY

The initial approach to orotracheal intubation is rapid sequence intubation (RSI) via direct laryngoscopy (Chapter 16). Conventional direct laryngoscopy may be replaced with video-assisted laryngoscopy (Chapter 20).[55-59] Limited evidence suggests it may offer superior views and higher success rates in less experienced providers.[45,60] Video laryngoscopy may be the most successful rescue technique for failed laryngoscopy.[61]

The ASA guidelines no longer include a strict numerical attempt value in their definition of difficult intubation. Multiple attempts (e.g., three or more) at vocal cord visualization via direct laryngoscopy have been associated with a significant increase in airway adverse events and hemodynamic compromise (e.g., aspiration, hypoxemia, and cardiac arrest).[46] **It is prudent to limit the number of attempts at direct laryngoscopy, with timely progression to alternative modes of airway management and a call for help should the first one or two attempts fail.** The DAS allows for a fourth attempt by an experienced provider before proceeding to alternative methods of securing the airway.

LARYNGOSCOPY IMPROVEMENTS AND ADJUNCTS

Manual external manipulation of the larynx by the Emergency Physician or an assistant may improve the laryngoscopic view. The "BURP" maneuver (i.e., external application of *b*ackward *u*pward *r*ightward *p*ressure to the larynx with the right hand) is a well-described manipulation known to be helpful but may worsen the view in some cases.[42] Cricoid pressure (i.e., Sellick's maneuver), which involves the application of pressure to the cricoid ring to occlude the upper esophagus and prevent aspiration, may interfere with vocal cord visualization.[26] The process is often one of trial and error due to the anatomic variations and dynamic nature of airway management.

INTRODUCERS, BOUGIES, AND STYLETS

Stylets are malleable rods that permit shaping of the endotracheal tube. They allow the user to direct the endotracheal tube for greater placement precision but carry the risk of airway trauma. These should be molded to have the same 30° to 40° angle at the tip with the endotracheal tube extending past the distal end of the stylet.[43]

Introducers are rigid or semi-rigid rods inserted into the airway, and then an endotracheal tube is passed over them (Chapter 22).[62] Introducers have an angled tip and can be bent to some degree, and an endotracheal tube can be passed over them with ease. The Eschmann endotracheal tube introducer (frequently referred to as a "bougie") is a reusable, 60 cm long introducer. The Eschmann flexed tip permits obstacle avoidance and lifting of the epiglottis and provides tactile feedback in the form of "clicking" as it passes over the tracheal rings to confirm its proper location.[43] It has a high success rate and low complication rate. The Frova intubating introducer is a single-use alternative of similar design. The Frova is improved by the presence of a hollow lumen that permits ventilation and CO_2 detection while having a similar success rate to that of the Eschmann introducer.[63,64] Light wands (i.e., Trachlight) can be used to intubate and/or confirm endotracheal tube placement by transillumination of the anterior neck (Chapter 24).[65]

ALTERNATIVES AND RESCUE TECHNIQUES FOR FAILED INTUBATION

Measures to restore ventilation and oxygenation must be implemented if initial attempts at intubation are unsuccessful. Use temporizing measures (e.g., mask ventilation) whenever possible.

SUPRAGLOTTIC AIRWAY DEVICE (SAD)

Consider the insertion of a SAD. Have several SADs of varying sizes immediately available in the setting of an unrecognized difficult airway, when the vocal cords cannot easily be visualized, or the airway cannot be intubated.[1,23] **The SAD serves an essential role in difficult airway management for adult and pediatric patients.** The SAD and is typically the "plan B" that follows direct laryngoscopy due to its unique advantages and high success rates of 95% to 99%.[1,23,66] It may be inserted swiftly and blindly to create a low-pressure seal over the laryngeal inlet that permits effective

ventilation when the vocal cords are impossible to visualize. The SAD bypasses soft tissues that may be obstructing the view and limits the risks associated with direct laryngoscopy.[26] The SAD tube may serve as a conduit for flexible bronchoscopic intubation, a bougie, or an intubating stylet.[66]

SADs have drawbacks inherent to their design. They are unable to protect the airway from aspiration and laryngospasm. They cannot generate the high pressures necessary to overcome poorly compliant airways. They carry a risk of dislodgement and gastric insufflation, although later models have features that minimize this risk.[26]

A maximum of three attempts are allowed to successfully insert the SAD and ventilate the patient per the DAS guidelines. Make two attempts with a preferred second-generation SAD (e.g., LMA Supreme or Proseal LMA) and one attempt with an alternative; further attempts contribute to increased risk without demonstrating a clear improvement in intubation success.[23] Cricoid pressure may interfere with SAD placement.[23] **Do not use cricoid pressure after direct laryngoscopy has proven unsuccessful.[23]**

EMERGENCY INVASIVE AIRWAYS

Progression to establishing a surgical airway is warranted in the rare event of a "cannot intubate, cannot ventilate" (CICV) scenario. **Do not delay the progression to an invasive airway.** Options include the percutaneous transtracheal jet ventilation (Chapter 31), cricothyroidotomy (Chapter 32), and tracheostomy (Chapter 33). Reserve these techniques for when all other techniques have been exhausted due to the invasiveness of these procedures and the associated risks (e.g., barotrauma, subcutaneous and mediastinal emphysema, hemorrhage, perforation, nerve injury, and infection).[26] **Emergency Physicians should be adequately trained in these techniques to mitigate their complications and high rate of failure.[67]**

PEDIATRIC CONSIDERATIONS

A higher minute oxygen consumption and a smaller respiratory reserve result in a low tolerance for apnea, with hypoxemia and bradycardia rapidly following the cessation of breathing in the pediatric patient.[68] The pediatric difficult airway is complicated by anatomic and physiologic differences from its adult counterpart. The approach to the difficult airway remains largely unchanged. Awake intubation remains a possible and adequate choice if the patient is cooperative.[69] Direct laryngoscopy is best achieved with a Miller blade. The curved Macintosh blade may not adequately displace the tongue. Video laryngoscopy using pediatric blades is an acceptable approach.[70] SADs have demonstrated excellent success rates in children with difficult airways, and success rates approach 96%.[48] Invasive techniques remain a possibility but present a considerable challenge given the smaller airway structures and higher risk for injury.[69]

ASSESSMENT

Successful placement of an airway device must be confirmed (Chapter 19). Visual confirmation of endotracheal tube passage through the vocal cords, continuous capnography, bilateral chest expansion, and auscultation for presence of breath sounds can be used to confirm endotracheal intubation. Waveform analysis of continuous capnography with appropriate and stable end-tidal CO_2 values remains the gold standard. **Absence of exhaled CO_2 detection signifies malposition of the airway device or airway obstruction.[23]**

AFTERCARE

The device needs to be secured so control of the airway is not lost. The specific aftercare depends of the technique used. See the appropriate chapter that discusses the technique.

COMPLICATIONS

Difficult airways contribute to a large proportion of airway-related adverse events. There are numerous complications associated with the difficult airway (e.g., aspiration pneumonitis, brain damage, death, emotional distress, Intensive Care Unit admission, need for a surgical airway, and nerve injury).[5,67] Device-specific complications are discussed in the appropriate chapter with the technique.

SUMMARY

The keys to the management of a difficult airway include early recognition, selection of a technique most likely to succeed based on patient characteristics and Emergency Physician training, and a clearly delineated plan of progression if the initial intubation plan fails. Training in management of difficult airways with timely initiation and progression of alternate/rescue techniques is paramount. Creation of a standardized difficult airway kit or cart and presence of a well-trained Emergency Physician may further reduce the risks and improve outcomes associated with this challenging phenomenon.[13]

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15 Obese Airway

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INTRODUCTION

Airway control is one of the most critical actions taken during an emergency response. It is not always straightforward and can present with many variations, creating challenges for the Emergency Physician. Many factors contribute to a difficult airway, with obesity becoming one of the most prevalent in the United States.[1] The negative effects of obesity are seen in nearly every organ system through anatomic, physiologic, and metabolic changes. The Emergency Physician must take these changes into account when attempting an endotracheal intubation in an obese patient, including a thorough understanding of the anatomy, physiology, equipment necessary, and complications. This chapter will aid in providing a better understanding of what many consider the most daunting challenge to an Emergency Physician.

ANATOMY AND PATHOPHYSIOLOGY

We will discuss the important variances seen in obesity and how they affect management. There is an overall increase in fat deposition within soft tissue structures in the oropharynx in a morbidly obese patient. These fat depositions can lead to enlarged lateral pharyngeal walls and a soft palate, a decrease in the caliber of the upper airway, and an increase risk of airway collapse.[2] **Neck circumference > 40 cm, a short neck, decreased mobility of the cervical spine, and a Mallampati class of III or IV are the best predictors of difficult ventilation with a mask and difficult intubation.**[3-6] A study performed by Stanford University showed that neck circumferences of 40 cm and 60 cm were associated with 5% and 35% probabilities of difficult intubation, respectively.[7] A large neck circumference does not indicate where fat is distributed along specific areas of the anterior neck. More fat is deposited around collapsible pharyngeal segments in patients with obstructive sleep apnea (OSA) syndrome (**Table 15-1**).[8-11] Obesity is a clear risk factor for developing OSA.[12-14] Further investigation is needed to clarify the conflicting evidence associating OSA with difficult intubations. **Positioning plays a key role in alleviating the difficulties provided by anatomic variations and soft tissue distributions.**[1]

TABLE 15-1 The STOP-BANG Screening Questions for OSA

Snoring	Do you snore loudly? Louder than talking? Can it be heard through a closed door?
Tired	How often do you feel fatigued, sleepy, or tired during the day? Do you fall asleep in the day?
Observed	Has someone observed you to choke, gasp, or stop breathing during sleep?
Blood **P**ressure	Do you have high blood pressure? Are you treated for high blood pressure?
BMI	Is the BMI greater than 35 kg/m^2?
Age	Is the age greater than 50 years?
Neck	Is the circumference around the level of the laryngeal prominence greater than 43 cm (17 inches) for a male or greater than 41 cm (16 inches) for a female?
Gender	Is the patient a male?

1 point is given for each positive answer.
A score ≥ 5 signifies a risk for OSA.
Source: Modified from references 10 and 11.

The physiologic differences found in obesity offer many considerations for the Emergency Physician when approaching the airway. Obesity increases the risk of other medical problems (e.g., hypertension, type 2 diabetes, OSA, and atherosclerosis). These can negatively affect the patient's health. These diseases require the Emergency Physician to alter the approach, preparation, and execution of tracheal intubation.[15]

Respiratory changes in obesity occur due to physical impairment of the chest wall. The increased body mass restricts diaphragmatic excursion, leads to hypoventilation, and decreases respiratory compliance.[16] Increased fatty tissue causes elevated metabolic requirements and increased oxygen consumption.[17] This results in the obese patient having an increased respiratory rate. The most important lung volumes for intubating, the functional residual capacity and the expiratory reserve volume, are decreased in obesity. These changes lead to increased atelectasis, worsening ventilation-perfusion mismatch, and increased right-to-left shunt.[18] The supine position and induction of anesthesia lead to these changes. Obesity worsens the effect to a much greater degree.[19] Obese patients may need a higher tidal volume for respirations to move the chest wall.[20] **These physiologic differences can drastically shorten the time to desaturation once the patient is apneic after induction.[12,21] This further emphasizes the importance of preparation, positioning, and preoxygenation.**

EQUIPMENT

- Equipment for orotracheal intubation (Chapter 18)
- Rubber head strap
- Equipment for noninvasive positive-pressure ventilation (NIPPV)
- Folded clean sheets to stack under the patient for positioning
- Laryngeal mask airways, various sizes (Chapter 26)
- Endotracheal tube introducer or bougie (Chapter 22)
- Video laryngoscope (Chapter 20)
- Fiberoptic bronchoscope (Chapter 28)
- Fiberoptic intubating oral airway, various sizes
- Cricothyroidotomy supplies (Chapter 32)
- Additional medications (**Table 15-2**)

Many institutions have airway bags or carts with standard equipment. The above listed equipment is most likely in the difficult airway cart.

TABLE 15-2 Weight-Based Dosing Recommendations for Commonly Used Medications

Medication	Induction dosing by weight	Induction dose (IV)
Cisatracurium	IBW	0.1 mg/kg
Etomidate	IBW	0.2–0.3 mg/kg
Fentanyl	LBW	1.5–3.0 mcg/kg
Ketamine	IBW	1.0–2.0 mg/kg
Propofol	LBW	1.0–2.5 mg/kg
Remifentanil	LBW	1.0–3.0 mcg/kg
Rocuronium	IBW	0.6–1.2 mg/kg
Succinylcholine	TBW	1.0–1.5 mg/kg
Sugammadex	IBW	16 mg/kg
Thiopental	LBW	3.0–5.0 mg/kg
Vecuronium	IBW	0.1 mg/kg

Abbreviations: IBW, ideal body weight; IV, intravenous; LBW, lean body weight; TBW, total body weight.

LARYNGEAL MASK AIRWAYS

The laryngeal mask airway (LMA) is a generic term used to describe many devices. The LMA is a device used as an alternative to mask ventilation and to endotracheally intubate. LMAs are an integral part of the most current difficult airway algorithm. They are devices that can be inserted into the pharynx to allow for ventilation, oxygenation, and a conduit for endotracheal intubation. They can be used as a primary airway or a backup airway.

The LMA consists of a hollow tube attached to a mask-like cuff designed to rest in the hypopharynx with the opening facing the glottis and the tip in the esophageal opening. LMAs come in different shapes, constructions, and sizes with unique features for various functions. The authors recommend managing an emergent difficult airway with a second-generation LMA designed with additional ports for orogastric tube and blind endotracheal tube insertion. Examples include the i-Gel (Chapter 25), Air-Q (Chapter 26), and LMA Fastrach (Chapter 26). Each type comes in different sizes, with some having half sizes. Each manufacturer recommends sizes based on patient weight. Predicting a proper fit can be difficult. Have available one to two sizes larger and smaller than predicted for the patient. Intubating LMAs allow an endotracheal tube through the ventilation port, the glottis, and into the trachea. The size of the endotracheal tube able to pass through the LMA varies with the size of the LMA. The Emergency Physician must be sure to check the manufacturer's recommendations.

ENDOTRACHEAL TUBE INTRODUCER OR BOUGIE

The endotracheal tube introducer is commonly referred to as a bougie (Chapter 22). **Introducers are underused devices that can be invaluable during a difficult intubation.** This device is a long, flexible, nonmalleable stylet with the distal tip fixed at a 30° angle. The bougie is used when encountering an anterior airway, a grade III Cormack-Lehane view, or a grade IV Cormack-Lehane view on direct laryngoscopy. It can be difficult to pass an endotracheal tube regardless of how much curvature is placed with a regular stylet. The 30° curve at the end of a bougie allows for anterior direction and passage through the vocal cords, even when they cannot be seen. The tracheal rings can be felt as the curved end of the bougie passes over them. An bougie can be used as a conduit through the vocal cords to pass an endotracheal tube over it and into the trachea. **Have a bougie readily available when intubating an obese patient. The threshold for its use should be low to avoid unnecessary pharyngeal and laryngeal trauma caused by multiple attempts with a regular endotracheal tube and stylet.**

VIDEO LARYNGOSCOPE

Video laryngoscopes (Chapter 20) consistently achieve a better view of the glottis when used in difficult airways compared with direct laryngoscopy and improve intubating conditions.[12,22-26] A small video camera is at the end of a fiberoptic handle and wired to a video screen to provide a wide-angle view of the entire glottic aperture. A specialized stylet with a fixed anterior curvature is typically used to pass the endotracheal tube. Some video laryngoscopes mimic a Macintosh laryngoscope blade, which the Emergency Physician is already comfortable using. Pass a video laryngoscope blade different from the direct laryngoscope by inserting it in the midline.

Insert the video laryngoscope and endotracheal tube until the cuff is seen at or just past the vocal cords. A second provider or assistant should carefully remove the stylet while the Emergency Physician firmly holds the endotracheal tube in place. As the stylet is removed, the endotracheal tube is carefully rotated in a clockwise fashion under continuous video monitoring until it passes completely into the trachea. The blade and handle are removed once the endotracheal tube is seen going into the trachea on the video laryngoscope. **It is of utmost importance to not advance the endotracheal tube balloon beyond the vocal cords with the rigid stylet in place to prevent tracheal injury.**

PATIENT PREPARATION

Preparation is of the utmost importance due to the frequently encountered difficulties. Take a brief history if the patient is conscious and able to communicate. Information includes past medical history, current medications, previous surgeries, allergies, last food or drink intake, illicit drug use, and a history of difficult intubations.[12] Perform a brief physical examination, and focus on the airway, cardiac system, pulmonary system, and neurologic status.

The airway examination is one of the most important aspects of the patient assessment. The highest predictor of a difficult intubation is a history of a difficult intubation. Important features to note are a thick or short neck, the thyromental distance, presence of facial hair, and the presence of cervical collars.[27] These features are predictive of difficult ventilation. Assess and estimate mouth opening. An opening less than 6 cm or three finger breadths is considered limited and can predict difficulty with direct laryngoscopy. Assess the length and health of the patient's dentition. Teeth that are very long, cracked, missing, capped, or loose should be noted prior to instrumentation of the airway. Note the presence of dentures and remove them prior to direct laryngoscopy. Assess the oral and pharyngeal structures and describe the view using the Mallampati class. This is estimated with the patient's mouth open, tongue protruding, and without phonation. Thyromental distance is estimated using finger breadths. A distance less than 6 cm is considered limited. This is the area that accommodates the tongue during direct laryngoscopy. Neck mobility is assessed with the patient sitting upright and moving their head from full flexion to full extension. Limited range of motion predicts difficulty aligning the external auditory meatus with the sternal notch and achieving an unobstructed view of the glottis during direct laryngoscopy.[28] This motion is not assessed in the patient wearing a cervical collar.

POSITIONING

Positioning is a key element of preparation that is frequently overlooked and underappreciated.[12] **The supine position is disadvantageous for respiratory mechanics and oxygenation. A ramped or head-elevated position improves respiratory mechanics and improves the glottic view.**[29-32] The head elevated 25° improved the glottic view by 50% when compared to a flat supine position.[33] The ramp position maintained or improved the view in 100% of patients with a body mass index (BMI) > 45 kg/m^2.[34] The ramped position can be achieved by two common methods that have been shown to be equally effective.[35] The first method uses folded sheets placed under the patient's upper body with an increasing elevation (**Figure 15-1**). The second is to elevate the head of the bed where the goal is to align the patient's external auditory meatus with the sternal notch (**Figure 15-2**). Use reverse Trendelenburg if the patient cannot be placed in the ramped position. **This position uses gravity to move the abdominal contents down and allow the diaphragm to fully expand.**

PREOXYGENATION

Equally as important as positioning is adequate preoxygenation.[36] The most effective technique for preoxygenation is achieved with the patient in the upright or sitting position and spontaneously

FIGURE 15-1. The patient in the ramped position using folded sheets.

breathing normal tidal volumes for 3 to 5 minutes.[37] The average time to saturations of 90% was increased by 1 minute over the supine position with the patient in the full upright position.[38] A possible approach could be to preoxygenate the patient in the sitting position and lower the gurney to an elevated position during intubation. This allows for maximum apneic time benefiting both the patient and Emergency Physician. A small randomized trial proved that providing the patient with 5 L of oxygen via nasal cannula during intubation added 90 seconds to the apneic period.[38]

MEDICATION DOSING

Obese patients have alterations in the pharmacokinetics and pharmacodynamics of medications. Obese patients have increased total body weight, with an increase in both fatty tissue weight and lean body weight. These changes lead to an increase in cardiac output, tidal volume, and glomerular filtration rate (GFR). An increase in fatty tissue causes more volume of distribution for lipophilic drugs (e.g., benzodiazepines and fentanyl). Base the dosing of lipophilic medications on total body weight or ideal body weight (**Tables 15-2 and 15-3**).[39] Obese patients often receive the incorrect dosage.[39]

FIGURE 15-2. The patient in the ramped position using elevation of the head of the bed.

HOWLAND LOCK

Consider using a Howland Lock (SunMed, Grand Rapids, MI) to intubate the obese patient. This piece of equipment attaches between the laryngoscope handle and blade (**Figure 15-3**). It can prevent the angle of an obese patient's chest from interfering with intubation. It reduces the chance of levering the laryngoscope on the patient's teeth and causing any damage. It was developed for the difficult airway and helps if the patient has an anterior larynx, decreased jaw mobility, facial contractures, protruding teeth, a receding chin, or a short neck, or is obese.

TECHNIQUE

RAPID SEQUENCE INDUCTION

Proper execution of an obese intubation with minimal or no complications almost entirely depends upon excellent patient and provider preparation. The Emergency Physician often has ample time to complete the task and all resources available to manage unexpected complications, thus providing a less stressful atmosphere to work. The head of the bed should be 2 to 3 feet from the wall to allow maneuvering room. Place the patient all the way to the head of the bed in a head-elevated or ramped position (**Figures 15-1 and 15-2**). Consider the application of a nasal cannula to provide oxygen during the rapid sequence intubation to prevent the patient from desaturating. Adjust the height of the bed so that the forehead of the patient is at the level of the Emergency Physician's umbilicus. Respiratory personnel should be to the right of the Emergency Physician and ready to assist. Position an assistant of equal or greater experience (i.e., another Emergency Physician) and able to assist in any way needed to the left of the Emergency Physician who is intubating. This person can attempt intubation if difficulty is encountered. Prepare the endotracheal tube. There is no correlation of a larger BMI to a larger airway.[40] The obese patient often has a decreased tracheal width.[40] **Avoid larger endotracheal tubes due to greater BMI. Be prepared to perform a cricothyroidotomy.**

Use a rapid sequence intubation technique (Chapter 16). The obese airway is often a difficult airway. The oral intake status is often unknown in the Emergency Department. The apneic time is decreased in obese patients, so securing the airway quickly is vital. Adequate preoxygenation for intubation is determined by an expiratory O_2 concentration of greater than 80%. A normal tidal volume of breathing for 3 minutes on 100% FiO_2 in at least a 45° head-elevated position is sufficient if real-time analysis is not immediately available. Hold the face mask gently against the patient's face with a hand or rubber face mask strap to prevent leaks. Prepare the endotracheal tube and place it within reach. Check that the laryngoscope is working and place it next to the patient's head. **The "best" blade is whichever blade the Emergency Physician has the most experience with and is most confident using.** Have the Nurse prepare and draw up the medications used in the intubation and immediately afterward.

Preoxygenate the patient and proceed. Instruct an assistant to place light pressure over the cricoid cartilage. Increase the cricoid pressure when the patient is asleep. This is done as a measure to prevent regurgitation and aspiration of gastric contents. Administer a small dose of a fast-acting opioid (e.g., 50 mcg fentanyl) due to its respiratory depressing side effects. Propofol is often used due to its fast onset and offset. Propofol can cause hypotension. Etomidate can be used as an alternate sedative in situations where the patient's hemodynamics are unstable. The oxygen mask should remain sealed over the patient's mouth for roughly 30 seconds or until fasciculations have ceased.

TABLE 15-3 The Definitions Used for Dosing

Weight description	Definition	Description	Alternative description
Total body weight (TBW)	The patient's weight		
Ideal body weight (IBW)	What the patient should weigh	IBW (kg) = height (cm) – X X = 100 for males X = 105 for females	Male IBW (kg) = X + 2.3 for every inch over 5 feet = X = 50.0 for males X = 45.5 for females
Lean body weight (LBW)	The patient's weight excluding fat (usually does not exceed 100 kg in men and 70 kg in women)	Male LBW (kg) = $\frac{9270 \times \text{TBW (kg)}}{6680 + (216 \times \text{BMI [kg/m}^2\text{]})}$ Female LBW (kg) = $\frac{9270 \times \text{TBW (kg)}}{8780 + (244 \times \text{BMI [kg/m}^2\text{]})}$	Male LBW (kg) = $(1.10 \times \text{weight [kg]}) - \frac{128 \times \text{weight}^2\text{ (kg)}}{(100 \times \text{height [m]})^2}$ Female LBW (kg) = $(1.07 \times \text{weight [kg]}) - \frac{148 \times \text{weight}^2\text{ (kg)}}{(100 \times \text{height [m]})^2}$
Adjusted body weight (ABW) or current body weight (CBW)	Obese patients have an increased lean body mass and volume of distribution	ABW (kg) = IBW (kg) + 0.4 (TBW [kg] – IBW [kg])	None
Morbid obesity	Severely overweight	BMI > 40 kg/m^2	BMI > 35 kg/m^2 with associated comorbidities

Follow the sedation with the depolarizing muscle relaxant succinylcholine. Succinylcholine should not be used in certain instances (**Table 16-2**). Use a nondepolarizing muscle relaxant (e.g., rocuronium) at a rapid sequence intubation dose if succinylcholine is contraindicated. **The Emergency Physician must have sugammadex available with a 16 mg/kg dose drawn at the bedside in case a "cannot intubate, cannot oxygenate" situation is discovered.**

Proceed with direct laryngoscopy. This will not be discussed here. Refer to the orotracheal intubation chapter (Chapter 18) for details.

DIFFICULT AIRWAY ALGORITHM

Difficult airways are common in obese patients. It can be a life-threatening situation when an Emergency Physician cannot successfully intubate an airway and has no systematic plan in place to prioritize the management. The Difficult Airway Society produced guidelines commonly known as the "Difficult Airway Algorithm."[41] The most recent revision was published in 2015. An algorithm for management of the obese airway is provided in **Figure 15-4**.

The Difficult Airway Society's Difficult Intubation Guidelines consist of four separate plans in which the Emergency Physician proceeds from one to the next if difficulty is encountered intubating or ventilating the patient. Plan A is focused on maximizing the likelihood of successful intubation on the first attempt while limiting total attempts to prevent airway trauma. It calls for proper patient positioning and preoxygenation. The recommendation is for three attempts at direct laryngoscopy by a single provider with one additional attempt by a more experienced person. Video laryngoscopes may be used.[25] Cricoid pressure should be applied throughout rapid sequence intubation unless there is difficulty intubating. **Repeated attempts at intubation should only occur if a change has been made to improve the chance of success (e.g., change of position, a different blade, neuromuscular blockade depth, or different personnel).**

The Emergency Physician should proceed to Plan B if attempts at Plan A have been exhausted. **Plan B emphasizes the importance of maintaining oxygenation using a supraglottic airway device (SAD).** Use a SAD to buy time and allow the Emergency Physician to assess the situation while the patient can be adequately ventilated. Use second-generation SADs due to various design improvements (e.g., the ability to intubate through the device).[12,42-44] A total of three attempts at placing a SAD is recommended, with the third attempt using a

A B

FIGURE 15-3. The Howland Lock. ***A.*** The Howland Lock. ***B.*** The Howland Lock attached to a laryngoscope.

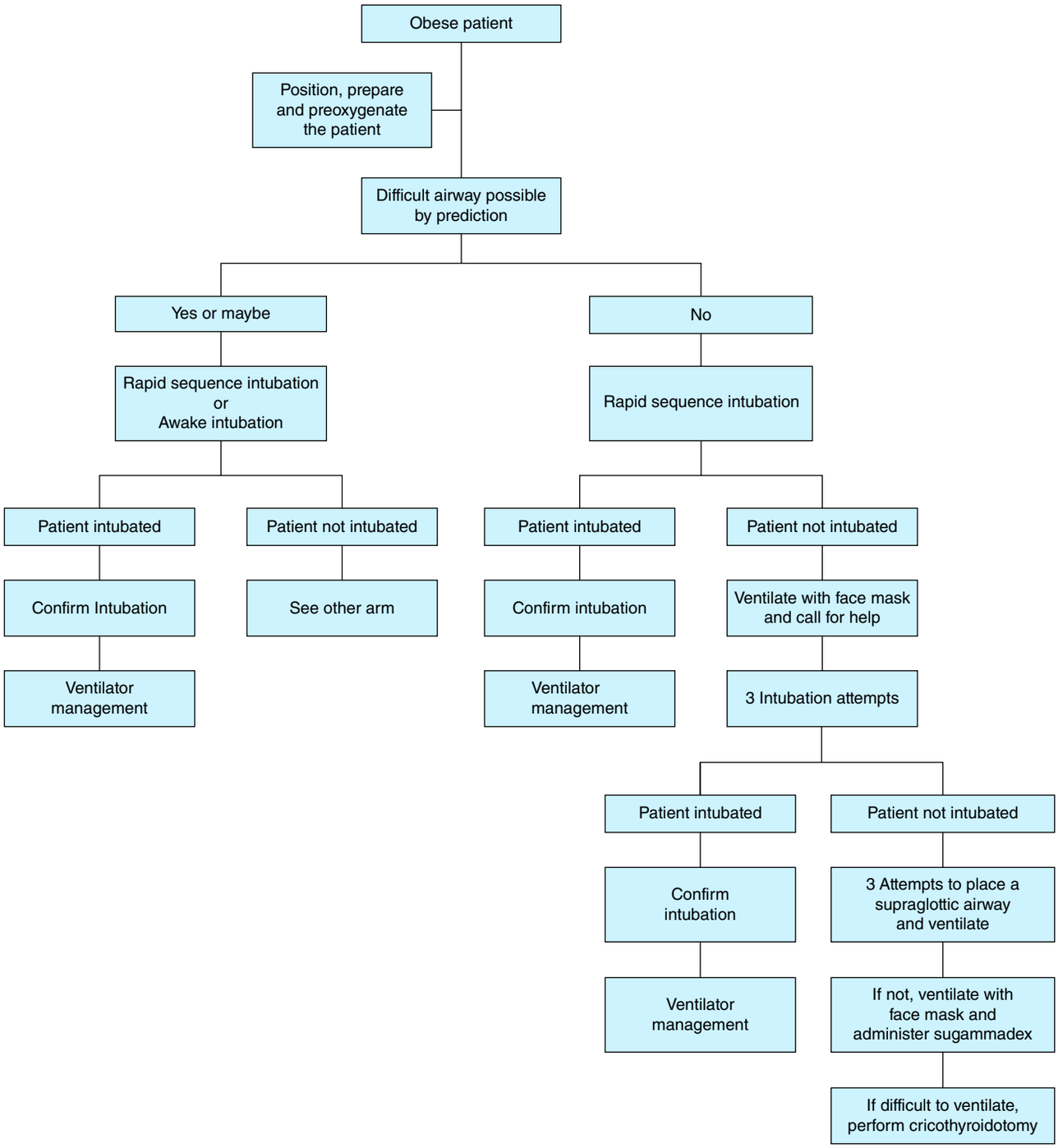

FIGURE 15-4. A suggested algorithm for the management of the obese airway.

different type or size. **Additional attempts are not recommended because SAD placement can induce airway trauma and swelling.** Successful SAD placement requires further reflection. The Emergency Physician should decide whether to wake the patient, attempt to intubate through the SAD, or proceed with a surgical airway.

The Emergency Physician should proceed to Plan C if attempts at Plan B have been exhausted. **Plan C is a final attempt to ventilate the patient with a face mask.** Maintain oxygenation and wake the patient up if mask ventilation is possible. The patient's airway can be traumatized, making mask ventilation much more difficult. Administer sugammadex at a dose of 16 mg/kg if mask ventilation is possible for a full reversal in the "cannot intubate, cannot oxygenate" scenario.[45]

The Emergency Physician should proceed to Plan D if attempts at Plan C have been exhausted. **Plan D is officially declaring the patient "cannot intubate, cannot oxygenate" and proceeding with a surgical airway. Hypoxic brain injury will ensue and potentially patient death if the situation is not rapidly changed.** Proceed with a surgical airway (Chapter 32) while two-person ventilation attempts are continued. One person uses two hands to apply the jaw-thrust and extend the patient's head, while another person delivers breaths with the bag-mask device.

ASSESSMENT

Evaluation of a successful endotracheal intubation should be fast and accurate. The time to desaturation in the obese patient is significantly shorter than the nonobese patient. Only relevant topics will be discussed.

The most common mistake made is removing eye contact of the glottis prematurely following what was thought to be a successful intubation. The intubator has the best ability to confirm intubation with direct visualization of the endotracheal tube passing through the vocal cords. It is common to see the tip of the endotracheal tube at the vocal cords and simultaneously advancing it while removing the laryngoscope. This is often performed to quickly to attach the bag-mask device. The ability to see the laryngeal aperture clearly in obese patients is poor. Their crowded pharynx allows less room to manipulate the endotracheal tube while maintaining an unobstructed view. **Eye contact should never be taken off the vocal cords from the first moment they are brought into view. Visualize the endotracheal tube cuff advancing completely past the vocal cords.** If only the portion of the endotracheal tube distal to the cuff is seen at the vocal cords, it can become caught in the arytenoid cleft and proceed into the esophagus. The Emergency Physician is likely to believe in and reassure the team of a successful intubation without direct visualization.

Confirm the placement of the endotracheal tube. Give three to four medium-sized breaths and watch for continuous and complete color change of the CO_2 colorimetric device. Instruct an assistant to auscultate the left subcostal region for gastric breath sounds and both sides of the chest for lung sounds. Watch for bilateral chest rise and condensation on the inside the entire endotracheal tube, and listen for an air leak with the application of breaths. Chest rise may not always be seen in obese individuals, but any movement should be symmetrical. Condensation can be mistaken if air from the esophagus is projected back onto the outside of the endotracheal tube. An air leak is important to assess because an intubated esophagus commonly gives a "gurgling" sound with each breath. Refer to Chapter 19 for a more complete discussion of the confirmation of endotracheal intubation.

This entire assessment should take no more than 10 seconds. **Look with the laryngoscope immediately to try to visualize the endotracheal tube position if an esophageal intubation is suspected.** Remove the endotracheal tube immediately and proceed with mask ventilation or Plan B of the difficult airway algorithm if the patient's saturations continue to fall or esophageal placement is confirmed.

AFTERCARE

There is very little to guide the Emergency Physician on ventilator management of the obese patient. Attach the endotracheal tube to a ventilator (Chapter 36). The Emergency Physician tends to lie the patient down. **Keep the head of the bed elevated 30° to 45° to displace the abdomen and improve ventilation.** This position also decreases the aspiration risk. Apply positive end expiratory pressure (PEEP) so the chest wall does not collapse the airways.[20] Keep the tidal volume based on the patient's ideal body weight to decrease the chance of inappropriate ventilator settings.[20] **Remember to sedate the patient after intubation. Do not paralyze the patient without sedation.**

COMPLICATIONS

Many of the complications encountered while intubating an obese patient are similar to those of the nonobese patient. Refer to Chapter 18 for a complete discussion of the complications associated with orotracheal intubation. Obese patients are at an increased risk for aspiration, which is discussed in greater detail below.[1,21] **Quick and efficient handling of aspiration is vital to the patient's survival.**

Obese patients are at an increased risk of aspiration, especially with full stomachs. **Perform an awake fiberoptic or awake indirect laryngoscopy to minimize the risk of aspiration.** Succinylcholine is the paralytic of choice for multiple reasons. It has a very rapid onset and is short acting. Succinylcholine increases the tone of the lower esophageal sphincter and further minimizes the risk of emesis. Cricoid pressure has been shown to decrease the tone of the lower esophageal sphincter. Several reports and studies have been performed weighing the risk versus benefit of cricoid pressure. The use of this maneuver is controversial, but it is used widely and the risks versus benefits should be known to all Emergency Physicians.[46] The common practice is to use cricoid pressure for rapid sequence intubation, with adjustments directed by the intubating provider.

Immediately apply cricoid pressure if aspiration occurs at any point, even if cricoid pressure has been released. **Direct the Yankauer suction catheter toward the posterior pharynx to clear as much emesis as possible**. Instruct an assistant to position the bed in Trendelenburg to minimize the flow of emesis into the trachea. Do not remove the laryngoscope if it is in the patient's mouth as the best protection from aspiration is to quickly intubate. Inflate the cuff immediately once intubated. Apply breaths if the saturation is below 80%. Proceed with endotracheal suctioning if the saturation is above 92% to remove tracheal aspirate prior to positive-pressure breaths. Perform a fiberoptic examination to completely assess the airway once the patient is properly oxygenated. Consult an Anesthesiologist or Pulmonologist if large particulate matter is seen or deep bronchial lavage is needed.

SUMMARY

Obesity is becoming is an exceedingly common problem in the United States. It adds many challenges for the Emergency Physician when managing the airway. Changes are seen in anatomic variations and physiologic variations. This further stresses the importance of experience and knowledge needed to safely manage these patients. Obese patients are at higher risk of having a difficult airway. Advanced airway equipment must be available along with the capability for a surgical airway. Special emphasis must be given to proper patient positioning and technique because the time to desaturation is significantly decreased in the obese patient. Awareness of medication pharmacodynamic and pharmacokinetic differences in obesity is necessary for accurate and safe intubation. Assessments of successful intubations must be fast and thorough with obese patients to avoid the increased risk of morbidity and mortality.

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16 Rapid Sequence Intubation

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INTRODUCTION

Rapid sequence intubation (RSI) of anesthesia is sometimes referred to as a "crash" intubation. The definition of RSI is the near simultaneous administration of a neuromuscular blocking drug and a sedative to induce unconsciousness and paralysis for endotracheal intubation. It has become a safe and effective method of establishing emergent airway control in patients with suspected life-threatening emergencies. It ensures optimal patient compliance in the best possibly controlled environment that can be achieved in the Emergency Department (ED). RSI in its classical description involves preoxygenation followed by the near simultaneous administration of a potent sedative-hypnotic agent and a neuromuscular blocking agent, application of cricoid pressure, avoidance of positive-pressure ventilation by mask, and intubation with a cuffed endotracheal tube.[1-21] Various pretreatment drug regimens have been advocated to prevent the potentially deleterious side effects of aspiration of gastric contents, cardiovascular excitation or depression, and intracranial pressure elevation.

The first endotracheal tubes were developed for the resuscitation of the newborn and victims of drowning in the 19th century but were not used in anesthesia until 1878.[13] Muscle relaxants were not prepared until some 60 years later. Succinylcholine was prepared by the Nobel Laureate Daniel Bovet in 1949, after which it gained the widespread usage it still enjoys today. The RSI technique did not come into modern-day practice until the end of World War II.

Patients can be hypoxic, confused, uncooperative, unstable, and unknowing of their medications or medical conditions and can require airway control within minutes of arrival at the ED. **RSI is**

the preferred method for securing the airway in the ED, as these patients are at risk for aspiration. These risks include vomiting from gastrointestinal obstruction, opioids, or hypotension; regurgitation from diabetic gastroparesis, gastroesophageal reflux, increased gastric pressure, or decreased lower esophageal sphincter tone; impaired laryngeal protective reflexes; and difficult airway management.[7] Conditions such as recent meal ingestion, pain, obesity, and pregnancy place patients at higher risk as well.

The procedure of RSI has many steps. It is sometimes easier to remember the 10 P's of RSI: plan, preparation (of drugs, equipment, and people), protect the cervical spine (if indicated), positioning (can be done after paralysis), preoxygenation, pretreatment (e.g., atropine, fentanyl, lidocaine), paralysis, protection (i.e., Sellick's maneuver), placement (endotracheal tube and confirmation of its position), and postintubation management. The controversy surrounding the technique (e.g., cricoid pressure effectiveness and ventilation prior to intubation) and the role in preventing aspiration has been questioned.[22-25] This has contributed to the lack of standardization.[22-25] Despite this, RSI has currently achieved a status close to being a standard of care for ED intubation in patients with full stomachs.[24]

INDICATIONS

The primary indication for RSI is to quickly protect and secure the patient's airway, preventing regurgitation of gastric content, and preventing aspiration. The rationale behind RSI is to create an environment in which the trachea can be intubated as quickly and with as little difficulty as possible. The clinical conditions occurring at the time of attempted intubation are therefore of great importance. The drugs used to produce hypnosis and muscle relaxation interact together to produce intubating conditions individualized to the patient and their condition(s). A complete list of the indications for RSI appears in **Table 16-1**. Other indications for RSI include to control ventilation in the intubated patient, to control agitation, to control seizures, and to facilitate the patient workup.

The drugs are used attenuate the response to intubation, blunt perception, blunt recall, decrease adverse effects, make intubation easier, and make intubation more controlled. Some patients inadequately respond to paralysis or have a prolonged paralysis. A decrease in neuromuscular blockade with rocuronium and succinylcholine is seen with hypercalcemia. Chronic use of carbamazepine or phenytoin may show resistance to rocuronium. A prolonged neuromuscular blockade with rocuronium and succinylcholine is seen with acidosis, aminoglycoside antibiotics, hypermagnesemia, hypokalemia, and hypothermia. Sever hepatic dysfunction may decrease the metabolism of rocuronium and result in a more prolonged paralysis.

Nondepolarizing neuromuscular blocking agents are usually used in the ED for RSI. A few specific situations deserve mention. These agents are safe in epilepsy, Parkinson's disease, and stroke. More of the agent is required if the patient has myasthenia gravis and its effects last longer. Less of the agent is required with Eaton-Lambert syndrome, hypothyroidism, and organophosphate poisoning.

TABLE 16-1 Indications for RSI in the Emergency Department

Airway protection and risk for pulmonary aspiration (e.g., full stomach, pregnancy, and obesity)
Altered mental status
Application of advanced cardiac life support and administration of drugs
Clinically declining patient
Definitive maintenance of airway patency
Depressed level of consciousness and questionable ability to maintain a patent airway
Emergency surgery and requirement for general anesthesia
Facilitation of fiberoptic bronchoscopy
Full stomach or unknown stomach contents
Head trauma with a decreased Glasgow Coma Scale score
Head trauma with the need for airway control
Head trauma with the need for ventilation
Hypothermia management
Hypoventilation
Impending airway obstruction (e.g., burns or penetrating neck injury)
Increased oxygen consumption
Increased oxygen delivery
Increased work of breathing
Potentially difficult intubation after airway evaluation
Prevention of brain hypoxemia
Pulmonary toilet or secretion management
Respiratory failure, actual or impending
Status epilepticus
Transfer of patient between facilities
Uncontrolled seizure activity requiring airway control
Uncooperative or combative patient with compromised airway
Unresponsiveness

Source: Adapted from references 2 and 7.

CONTRAINDICATIONS

There are few contraindications to RSI. It should not be performed by an inexperienced intubator. Consider an awake intubation or surgical airway if the Emergency Physician has doubts about their ability to intubate the patient. The unavailability of equipment, contraindications to muscle relaxants, and critically ill patients in whom the airway can be secured by other methods (e.g., fiberoptic intubation, topical anesthesia, or minimal sedation with a benzodiazepine and/or narcotic) are also contraindications to RSI. Any contraindication to the use of succinylcholine is also a contraindication to RSI. **Table 16-2** lists the many common contraindications to the use of succinylcholine. Patients in cardiopulmonary arrest do not require RSI as they should not have any muscle tone to overcome. A relative contraindication is a patient in whom bag-valve-mask

TABLE 16-2 Contraindications to the Use of Succinylcholine

Cardiac arrhythmia
Children and adolescents, unless no other option exists
Exaggerated hyperkalemia in susceptible patients
Crush injuries
Denervation beyond 48–72 h after injury[14]
Disuse atrophy
More than 24 hours after major burns and trauma
Metastatic rhabdomyosarcoma[14]
Muscular atrophy
Paraplegia or hemiplegia
Prolonged immobilization
Severe abdominal infection
Guillain-Barré syndrome
History of malignant hyperthermia in the patient or in their family history
Hypersensitivity to the drug
Increased intracranial pressure (relative)
Increased intragastric pressure (relative)
Increased intraocular pressure (relative)
Masseter muscle spasm or rigidity
Motor neuron disease
Muscular dystrophy
Plasma cholinesterase deficiency (relative)
Skeletal muscle myopathies in the patient or in their family history
Spinal cord injury

Source: Adapted from references 1, 2, 8, and 14.

ventilation is difficult or anticipated to be difficult.[26,27] Other relative contraindications are a team with poor skills or that functions poorly, the anticipated difficult airway, children less than 5 years of age, patients with distorted upper airways (e.g., congenital or acquired), situations when a surgical airway is not possible (e.g., lack of equipment or lack of experience), and patients with an upper airway obstruction.

EQUIPMENT

- Supplemental oxygen with appropriate tubing and connectors
- Laryngoscope handle with extra batteries
- Laryngoscope blades, various sizes and types
- Video laryngoscope
- Endotracheal tubes, various sizes
- Wire stylet, malleable type
- Nonrebreather oxygen masks, various sizes
- Oropharyngeal airways, various sizes
- Nasopharyngeal airways, various sizes
- Alternative airway devices
- Suction source with appropriate tubing
- Suction catheters for endotracheal tubes
- Yankauer suction catheter
- Bag-valve-mask devices, various sizes
- Face masks, various sizes
- Stethoscope
- Water-soluble lubricant or anesthetic jelly
- Tape
- Benzoin adhesive
- Syringes, 10 and 20 mL
- Medications drawn up and labeled
- Pulse oximeter
- Cardiac monitor
- Automatic sphygmomanometer
- End-tidal carbon dioxide (CO_2) monitor/device
- Crash cart
- Resuscitation medications
- Personnel (respiratory technician, medication nurse, recorder, in-line stabilization assistant)

The equipment required for RSI is the same as that for any intubation.[10,13] Complete details regarding the selection of properly sized equipment can be found in Chapters 10 and 18. **Backup equipment should be readily available if the patient cannot be intubated**. This can be a laryngeal mask airway, cricothyroidotomy tray, retrograde guidewire kit, or percutaneous jet ventilation system, to name a few.

PATIENT PREPARATION

RSI can be performed with little or no preparation. A few steps can be performed while the patient is being evaluated. Administer supplemental oxygen to the patient with a nonrebreather mask, continuous positive airway pressure (CPAP) mask, or noninvasive nasal positive-pressure ventilation (NINPPV). Apply a noninvasive blood pressure cuff, continuous pulse oximetry, and cardiac monitoring. Obtain intravenous (IV) or intraosseous access.[28] If time permits, pharmacologic agents can be used to increase gastric pH and motility (e.g., antacids, H_2-receptor blockers, and metoclopramide). An antisialagogue (e.g., atropine or glycopyrrolate) may also be administered to decrease excessive oral and respiratory tract secretions.

What is the ideal RSI agent? It has the following characteristics: provides analgesia and amnesia, short onset, predictable length of time to work and last, maintains hemodynamics, maintains cerebral perfusion pressure, causes unconsciousness and unresponsiveness, and has few side effects. **No single agent exists. We use multiple agents to minimize side effects and maximize the effects we want**.

Evaluate the airway. Long teeth and buck teeth are problematic and affect the angle of the laryngoscope blade. The mandibular teeth should move in front of the maxillary teeth to test the motion of the temporomandibular joint (TMJ). There should be 3 cm between the mandibular teeth and maxillary teeth upon mouth opening. Look at the size of the tongue to see if it is enlarged (i.e., Does the tongue obscure the pharyngeal structures?). Ensure the palate is not too narrow and there is room for the laryngoscope blade and endotracheal tube. Check to see if there are three finger breadths between the chin and the thyroid cartilage. Determine if the neck is short, long, and/or thick. The patient should be able to assume the sniffing position. Finally, is there any resistance to movement of the front of the neck? **Any deviation from the "normal" can signify a potentially difficult airway. If, after evaluation of the airway, there is sufficient doubt as to the possibility of intubating the patient successfully, a neuromuscular relaxant should not be administered. Consideration should be given to securing the airway in another fashion (e.g., awake intubation).**

TECHNIQUE

The three main characteristics of RSI are preoxygenation, application of cricoid pressure, and avoidance of positive-pressure ventilation if possible prior to securing the airway with a cuffed endotracheal tube. In addition, RSI requires the presence of ancillary equipment and experienced assistance. The details of timing, drug choice, and dosage are not rigidly defined. An RSI protocol is described below from start to finish.

Preoxygenate the patient with 100% O_2 by nonrebreather mask, or ventilate with a bag-valve-mask device using cricoid pressure. This will build an oxygen reserve and prevent hypoxemia during intubation. Preoxygenation for 5 minutes is the routine practice. If this is not practical, attempt to preoxygenate the patient for 3 minutes.[6] However, four maximal inspirations are equally effective in the cooperative patient.[6] A recent study has shown that eight vital capacity breaths in 1 minute provide a better safety margin with almost double the apneic time without hypoxia compared with 3 minutes of tidal volume breathing.[29] Oxygen administration via noninvasive positive-pressure ventilation can improve oxygenation more rapidly than by a face mask.[30] Utilization of CPAP or NINPPV during the preoxygenation has been shown to increase the duration of the apneic period without hypoxemia.[31-33] In addition, head elevation of 20° to 25° has been shown to improve oxygenation as well.[34] Preoxygenation often eliminates the need for positive-pressure ventilation in RSI.[35]

Consider apneic oxygenation during the period of preoxygenation and paralysis.[36-39] This can be provided by using a nasal cannula at 15 L/min. The diaphragm does not have to move for gas exchange at the alveoli. Its use has shown mixed results in terms of eliminating hypoxemia during RSI.

While an assistant is preoxygenating the patient, evaluate the airway to anticipate any difficulties during the intubation. Assemble all required equipment. Connect the bag-valve device to a mask and

TABLE 16-3 Pharmacologic Adjuncts to Intubation in the Premedication Phase

Agent	Standard dose	Trauma dose	Blood pressure	Cerebral perfusion pressure
Fentanyl*	2.0–8.0 µg/kg	1.0–3.0 µg/kg	Stable	Stable
Sufentanil†	0.25–µg/kg	0.1–0.2 µg/kg	Stable	Stable
Alfentanil‡	5.0–25.0 µg/kg	5 µg/kg	Stable	Stable
Remifentanil¶	0.25–1.0 µg/kg/min	0.05–0.5 µg/kg/min	Stable	Stable
Lidocaine§	1.0–1.5 mg/kg	1.0–1.5 mg/kg	Stable	Stable or increased

*Minimal hemodynamic or cerebrovascular effects. Useful agents for blunting the noxious stimuli of direct laryngoscopy or intubation. The half-time of equilibration between the effect and plasma is relatively slow (5 to 6 minutes). May cause central vagal stimulation with resultant bradycardia and occasionally hypotension in patients with high sympathetic tone.

†Similar to fentanyl, but more potent and faster offset.

‡Similar to fentanyl, but faster onset and duration of action. Half-time of equilibration between the effect site and the plasma is 1.5 minutes, making this opioid a very appropriate drug to provide a transient peak effect after a single bolus dose. May prevent the increase in intraocular pressure caused by succinylcholine.[8]

¶Similar to alfentanil in terms of fast onset. Extremely rapid clearance (3 to 4 L/min) due to esterase metabolism, resulting in a rapid and predictable recovery.

§Useful adjuvant agent for blunting airway reflexes. Also blunts blood pressure, intracranial pressure, and intraocular pressure responses to intubation, involuntary muscle movements after etomidate, and injection site pain from propofol and etomidate. Topical lidocaine is also effective in blunting reflexes.

Source: Adapted from Chapter 12.

an oxygen supply. Lubricate and place the malleable stylet into the endotracheal tube. Attach a syringe to the inflation port of the endotracheal tube cuff. Inflate the cuff to look for any air leaks. Deflate the cuff and leave the syringe attached to the inflation port. Attach the laryngoscope blade to the handle and make sure that the light is functional.

Simultaneously, the nurses should apply a noninvasive blood pressure cuff, continuous pulse oximetry, and cardiac monitoring to the patient. They should also draw up and label the required medications, establish intravascular access, set up the suction, record all events, and continuously observe the noninvasive blood pressure readings, cardiac monitor, and pulse oximeter.

If there is no suspicion of a cervical spine injury, position the patient in the optimum "sniffing" position. If there is suspicion of a cervical spine injury, an assistant should provide manual in-line axial stabilization of the head and neck during the intubation sequence. Remove the anterior aspect of the cervical spine collar to allow for maximal mouth opening and access to the neck.

Premedicate the patient (**Table 16-3**). The mnemonic **LOAD** has been used to indicate the pretreatment drugs for RSI.[15] The mnemonic stands for **l**idocaine, **o**pioid (specifically, fentanyl), **a**tropine, and **d**efasciculation. Lidocaine (1.0 to 1.5 mg/kg) can be given to blunt the intracranial pressure response, transient hypertension, bronchospasm, and tachycardia associated with intubation. Fentanyl (2 to 3 µg/kg) or one of its derivatives can be given to also blunt the intracranial pressure response, transient hypertension, and tachycardia associated with intubation. Atropine (0.01 to 0.02 mg/kg; minimum 0.1 mg, maximum 1.0 mg) should be given to children less than 1 year old to prevent bradycardia in response to direct laryngoscopy. Administer a defasciculating dose of a nondepolarizing neuromuscular blocking agent. This is one-tenth of the intubating dose (**Table 16-4**). Phenylephrine (50 µg) can be given to attenuate the hypotensive response to intubation. Administer an appropriate induction agent as indicated by the clinical setting and patient's hemodynamic status (**Tables 16-5 and 16-6**). Flush the intravenous line with 10 to 20 mL of 0.9% normal saline solution after each drug to ensure delivery.[40]

Administer a neuromuscular blocking agent.[1,2,5] Numerous agents are available (**Table 16-4**). The most commonly used medications are succinylcholine (1.0 to 1.5 mg/kg) or rocuronium (1.0 to 1.2 mg/kg). **Some consider succinylcholine the preferred agent. Its effects are short-lasting (i.e., 4 to 6 minutes). This is especially useful if the patient cannot be intubated, as he or she will need to be ventilated with a bag-valve-mask device until the succinylcholine wears off.** Rocuronium allows the same intubating conditions as succinylcholine except that it lasts for 30 to 60 minutes, causes no fasciculations, and causes no histamine release. This long-lasting time frame is problematic if the patient cannot be intubated. Rocuronium (1.2 mg/kg) delivers a rapid onset of paralysis similar to succinylcholine.[41] A Cochrane Review concluded that succinylcholine was superior to rocuronium in achieving excellent and clinically acceptable intubating conditions.[42]

TABLE 16-4 Selected Pharmacologic Properties of the Neuromuscular Relaxants

Agent	Intubation dose (mg/kg)	Average intubating time (min)	Clinical duration (min)	Comments
Succinylcholine	Children: 2 mg/kg Adults: 1 mg/kg[16] IM: 4 mg/kg	1	4–6	Agent used for RSI.[1,2] Associated with side effects such as exaggerated hyperkalemia in susceptible patients (> 24 hours after major burns and trauma, crush injury, denervation, prolonged immobilization, paraplegia, hemiplegia, muscular dystrophy) and malignant hyperthermia. Elevates intraocular, intracranial, and intragastric pressures. Use the total body weight (not the lean weight) even in the morbidly obese or pregnant patient.
Rocuronium	0.6–1.2 Children 2 to 12 years old: 0.9–1.2[17]	0.7–1.1	31–67	An alternative to succinylcholine provided there is no anticipated difficulty in intubation.[4]
Vecuronium	0.08–0.10	2.5–3.0	25–40	Cardiovascular effects unlikely. Alternative to succinylcholine.
Cisatracurium	0.15–0.20	1.5–2.0	55–65	Stereoisomer of atracurium. No cardiovascular effects. Organ-independent elimination.
Atracurium	0.4–0.5	2.0–2.5	35–45	Elimination independent of liver and kidney. Releases histamine.
Pancuronium	0.06–0.10	2.0–3.0	56–100	Tachycardia and sympathetic nervous system activation.

Source: Adapted from references 1, 2, 4, 5, and 9.

TABLE 16-5 Pharmacology of the Anesthetic Induction Agents

Agent	Dose (mg/kg)	Steady-state volume of distribution (L/kg)	Clearance (mL/min/kg)	Elimination half-life (h)
Thiopental	Adults: 3.0–5.0 Children: 3.0–5.0 Infants: 7.0–8.0[18]	2.5	3.4	11.6
Etomidate	0.2–0.3	2.5–4.5	10.0–20.0	2.0–5.0
Propofol	Adults: 1.5–2.5 Children 3.0–6.0	2.0–10.0	59.4	4.0–7.0
Midazolam	0.1–0.2	1.0–1.5	7.5	1.0–4.0
Ketamine	1.0–2.0	2.5–3.3	16.0–18.0	1.0–2.0
Methohexital	1.0–3.0	0.4	6.56	22 days
Fentanyl	0.002–0.02	4.0	4.8–10.5	3.5–4.0

Source: Adapted from references 3, 7, 8, and 18.

The U.S. Food and Drug Administration (FDA) approved sugammadex in the United States in 2015. It has been available in other parts of the world for several years. It binds to rocuronium and prevents the rocuronium from binding to nicotinic receptors and inducing neuromuscular blockade. Sugammadex works in approximately 30 minutes. It is not metabolized and is excreted unchanged in the urine in about 8 hours. The argument of succinylcholine being the preferred agent due to its short-lasting action no longer exists. Sugammadex not only more predictably antagonizes muscle relaxation induced by rocuronium than spontaneous recovery from succinylcholine, but the reversal time is faster too.[33,43-45] The typical dose is 2 to 4 mg/kg. Up to 16 mg/kg can be given if quick reversal of rocuronium is needed. The use of sugammadex has not been studied in pregnancy and lactation. A recent survey of Anesthesiologists in the United Kingdom showed that succinylcholine is still the most common used agent for RSI.[25]

An assistant should apply cricoid pressure with a force of 20 to 30 Newtons as soon as the patient loses consciousness and maintain it until successful oral endotracheal intubation has been confirmed. How much force is a Newton? It is difficult to translate from mechanical devices to the human hand. A discussion of the use of cricoid pressure is in Chapter 18. Avoid mask ventilation if possible. If hypoxemia or hypercarbia ensues, begin mask ventilation to a maximum pressure of 20 cmH_2O while maintaining cricoid pressure.

Intubate the trachea 60 to 90 seconds after the succinylcholine (or rocuronium) has been administered and the patient's muscles are relaxed, as noted by apnea and jaw relaxation. **Confirm the correct position of the endotracheal tube by visualizing the tube passing through the vocal cords, observing sustained presence of end-tidal CO_2 on the capnograph, and auscultating breath sounds at the midaxillary lines.**[11,12] Auscultate over the epigastric area to ensure that ventilation is not audible over the stomach. Release the cricoid pressure. If indicated, administer a long-acting paralytic agent (**Table 16-4**). After successful intubation, administer additional sedative hypnotics and analgesics as dictated by clinical need. Obtain a chest radiograph to confirm proper placement of the endotracheal tube.

PEDIATRIC CONSIDERATIONS

RSI is often used in the Emergency Department for securing the airway of pediatric patients. The indications and equipment, except for being smaller, are essentially no different than those of the adult patient. Infants and young children have developmental differences in head and neck anatomy. These differences make the Miller blade the preferred laryngoscope blade for intubation in this group. Infants and young children have a higher volume of distribution, which is why they require different doses of induction agents, as reflected in **Tables 16-4** and **16-5**.

Preoxygenation is particularly important for infants and children. Compared with adults, these young patients have a higher oxygen consumption rate with lower functional residual capacity. Consequently, oxygen desaturation occurs much more rapidly (i.e., 1 to 2 minutes in healthy infants and 2 to 4 minutes in healthy children). Perform careful bag-mask ventilation with small tidal volumes while maintaining cricoid pressure to achieve adequate preoxygenation if the child is desaturating or is apneic.

The use of succinylcholine remains controversial in pediatrics. RSI and laryngospasm are perhaps the last remaining indications for the use of succinylcholine in pediatrics. In 1994, the FDA recommended that the use of succinylcholine in children be reserved for emergency intubation and instances where the immediate securing of the airway is necessary due to the risks of hyperkalemia. This includes patients with laryngospasm, difficult airways, or full stomachs, or for intramuscular use. If a child has no vascular access, succinylcholine (4 mg/kg) can be administered intramuscularly. It will provide a maximum onset of blockade in 3 to 4 minutes and last approximately 20 minutes. If there is a contraindication to using succinylcholine, rocuronium can be used for RSI. Children between

TABLE 16-6 Cardiovascular and Central Nervous System Effects of Anesthetic Induction Agents

Agent	Blood pressure	Cardiac contractility	Cerebral blood flow	$CMRO_2$	Intracranial pressure	Cerebral perfusion pressure
Thiopental	Decrease	Decrease or no change	Decrease	Decrease	Decrease	Decrease or no change
Etomidate	Slight decrease or no change	No change	Decrease	Decrease	Decrease	Increase
Propofol	Decrease	Decrease	Decrease	Decrease	Decrease	Decrease or no change
Midazolam	Slight decrease	No change	Decrease	Decrease	Decrease	No change
Ketamine	Increase	Increase	Increase	Increase	Increase	Increase or no change
Methohexital	Decrease	Decrease	Decrease	Decrease	Decrease	Increase
Fentanyl	Slight decrease	No change	Decrease	Decrease	Increase or no change	No change

Source: Adapted from Chapter 12.

TABLE 16-7 Complications of RSI

Airway trauma
Awareness
Bradycardia
Cerebral anoxia
Complication specific to the medications administered
Corneal injury
Death
Dental damage
Hypertension
Hypotension
Hypoxia
Increased intracranial pressure
Increased intraocular pressure
Myocardial ischemia
Pulmonary aspiration
Tachycardia

2 and 12 years old require more rocuronium than adults. The recommended doses are 0.9 to 1.2 mg/kg in this age group.

Atropine is commonly used in young children. Atropine (20 μg/kg) administered as a premedication is indicated in all children < 1 year old and in all ages if a second dose of succinylcholine is required to intubate the patient.[19] There is a risk of bradycardia with the use of succinylcholine in children. This can be attenuated by premedicating the patient with atropine (20 μg/kg) or glycopyrrolate (10 μg/kg).[20]

TABLE 16-8 Mnemonics for RSI Preparation

SOAPME
- Suction
  - At least one working suction
  - Two suctions for gastrointestinal bleeds, vomiting, or lots of secretions
  - Yankauer suction catheter placed between mattress and head of bed
- Oxygen
  - Nonrebreather mask and bag-valve device attached to 15 L/min O_2
  - ? Nasal cannula for apneic oxygenation
- Airways
  - Oral and nasal airways
  - Endotracheal tubes with stylets, balloon tested
  - Stylet inside endotracheal tube and bent 30°
  - Laryngoscope handles and blades, light tested
  - Video laryngoscope
  - Cricothyroidotomy kit as backup
  - Backup devices (e.g., bougie, laryngeal mask airway, supraglottic airway, etc.)
- Preoxygenate and positioning
  - Nonrebreather mask at 15 L/min O_2
  - Sniffing or ear to sternal notch
  - Ramp or blankets for obese patients
- Monitoring equipment and medications
  - Cardiac monitor, noninvasive blood pressure cuff, pulse oximeter, IV line
  - Medications drawn into syringes and labeled for RSI and postintubation sedation
- End-tidal CO_2 and other equipment
  - End-tidal CO_2 monitor or colorimetric device

O_2 MARBLES
- Oxygen
- Masks (e.g., nonrebreather and bag-valve-mask) and medications
- Airway adjuncts, ask for help, and airway cart for difficult airways
- RSI drugs and resuscitation drugs
- Bag-valve-mask and bougie
- Laryngoscopes (e.g., direct and video) and laryngeal mask airway
- Endotracheal tubes and end-tidal CO_2 detection
- Suction and state plan

ASSESSMENT

Confirm that endotracheal intubation is successful. Success is indicated by auscultation, fogging of the endotracheal tube with ventilations, and the presence of persistent end-tidal CO_2. End-tidal CO_2 can be confirmed using a capnograph or a chemical colorimetric indicator device, which consists of a pH-sensitive indicator where the dye changes its color in the presence of CO_2. A minimum of six breaths should be administered before a determination is made regarding successful endotracheal intubation. This will eliminate false-positive readings obtained by CO_2 forced into the stomach during mask ventilation, antacids in the stomach, or carbonated drinks in the stomach. False-negative results may be seen with very low tidal volumes and low end-tidal CO_2 concentrations during severe hypotension or cardiac arrest. During cardiopulmonary resuscitation (CPR), a negative result requires an alternative method of confirming the position of the endotracheal tube because compromised circulation causes low end-tidal CO_2.[12] Other methods used to confirm endotracheal intubation include fiberoptic endoscopy with direct visualization of the tracheal rings and carina, pulse oximetry (low saturation is a late sign for esophageal intubation), blood gas analysis, chest radiography, and a variety of detection devices. Please refer to Chapter 19 for more information regarding the confirmation of endotracheal intubation.

AFTERCARE

The aftercare is the same as for any intubated patient. Secure the endotracheal tube with tape or a commercially available

TABLE 16-9 A Sample RSI Protocol

- Zero minus 10 minutes
  - Preparation
  - Assess airway for difficulty
  - Plan approach
  - Assemble drugs, equipment, and personnel
- Zero minus 5 minutes
  - Preoxygenation
  - ? Nasal cannula for apneic oxygenation also
- Zero minus 3 minutes
  - Pretreatment with medications if applicable
  - Consider LOAD mnemonic
    - Lidocaine IV and local anesthesia if awake intubation or nasotracheal
    - Opioid
    - Atropine
    - Defasciculation
- Zero
  - IV bolus of sedative agent
  - IV bolus of neuromuscular blocking agent
- Zero plus 30 seconds
  - No bag-valve-mask unless saturations are ≤ 90
  - Sellick's maneuver
  - Position patient if no contraindications exist
- Zero plus 45 to 90 seconds
  - Check jaw for relaxation
  - Intubate
  - Confirm placement of endotracheal tube
  - Release Sellick's maneuver
- Zero plus 90 seconds
  - Secure the tube
  - Administer sedation
  - Administer long-acting paralysis if indicated
  - Attach and set ventilator
  - Obtain a chest radiograph

TABLE 16-10 A Sample Checklist for RSI

Prepare Patient
- ☐ Preoxygenation
- ☐ Apneic oxygenation
- ☐ Assess airway for potential difficulty
- ☐ Call for help if difficulty is determined
- ☐ Vascular access obtained
- ☐ Vascular access flushes
- ☐ Patient positioning
- ☐ How will anesthesia be maintained?
- ☐ Explain process to patient

Prepare Equipment
- ☐ Electrocardiogram applied
- ☐ Blood pressure cuff applied
- ☐ Blood pressure cuff set for 2-minute cycles
- ☐ Pulse oximetry applied and working
- ☐ End-tidal CO_2 monitor or colorimetric device
- ☐ Bag-valve-mask, correct size for patient
- ☐ Suction set up from wall to Yankauer
- ☐ Suction turned on and working
- ☐ Multiple endotracheal tubes and stylets
- ☐ Endotracheal tube cuff tested
- ☐ Multiple laryngoscope handles and blades
- ☐ Video laryngoscope and working
- ☐ Multiple syringes, various sizes
- ☐ Bougie
- ☐ Difficult airway cart at bedside
- ☐ List of drugs available
- ☐ Syringes with drugs and labeled
- ☐ Confirmed drug doses

Prepare the Team
- ☐ Identify team leader
- ☐ Identify intubator
- ☐ Identify Sellick's holder
- ☐ Identify in-line cervical stabilizer, if applicable
- ☐ Identify drug preparer
- ☐ Identify drug administrator
- ☐ Identify recorder of record
- ☐ Describe the plan

The Procedure
- ☐ Administer premedication
- ☐ Administer sedation
- ☐ Administer neuromuscular blocker
- ☐ Allow neuromuscular blocker to work
- ☐ Laryngoscopy
- ☐ Insertion of endotracheal tube
- ☐ Abort laryngoscopy if time runs out, pulse oximetry drops, or unsuccessful
- ☐ Call for help
- ☐ Secure endotracheal tube
- ☐ Ventilate the patient
- ☐ Confirm proper tube position
- ☐ Insert nasogastric tube
- ☐ Obtain chest radiograph
- ☐ Administer postintubation drugs

Debriefing Team
- ☐ What went right
- ☐ What went wrong
- ☐ What to change next time
- ☐ Additional training needed
- ☐ Equipment issues
- ☐ Other things to discuss

device. Administer adequate sedation and neuromuscular relaxation as necessary. Place a nasogastric tube to decompress the stomach.

COMPLICATIONS

The complications associated with RSI are numerous (**Table 16-7**), ranging from minor airway trauma to cerebral anoxia and death. **The technique of RSI should be performed only by experienced Emergency Physicians. The availability of alternate invasive airway devices and techniques can often prevent complications if oral endotracheal intubation is unsuccessful.** A more complete discussion of the complications is contained in Chapter 18.

The procedure of RSI has many steps. It is sometimes easier to remember the 10 P's of RSI: plan, preparation (of drugs, equipment, and people), protect the cervical spine (if indicated), positioning (can be done after paralysis), preoxygenation, pretreatment (e.g., atropine, fentanyl, lidocaine), paralysis, protection (i.e., Sellick's maneuver), placement (endotracheal tube and confirmation of its position), and postintubation management (**Table 16-8**). A sample checklist and protocol can simplify the procedure and decrease the complication rate (**Tables 16-9 and 16-10**).

SUMMARY

Rapid sequence induction is the preferred method to secure an airway on an emergent basis and when there is a risk of aspiration of gastric contents. In experienced hands, it is a relatively safe procedure with few complications. The choice of pharmacologic agents used will vary by physician experience, physician preference, the clinical condition of the patient, and the pharmacology of the agents.

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17 Delayed Sequence Intubation

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INTRODUCTION

Delayed sequence intubation (DSI) is an alternative technique in optimization of preoxygenation for noncompliant patients with altered mental status. The technique is designed to improve preoxygenation by providing controlled sedation.[1] The current evidence consists of data from small studies of patients sedated with ketamine and successfully preoxygenated and intubated.[1-6] DSI evidence is based on a small series of cases. This technique can be successful for many patients, including those with asthma.

ANATOMY AND PATHOPHYSIOLOGY

DSI separates the administration of induction drugs from the administration of paralytic agents to allow preintubation oxygenation and denitrogenation of the patient.[1] It has been described as procedural analgesia and sedation (Chapter 159) for the procedure of preoxygenation and denitrogenation. Denitrogenation makes the functional residual capacity of the lungs to be filled with oxygen to provide an oxygen reservoir. Ketamine is used as the induction agent (Chapter 12). The ketamine allows the patient to maintain airway reflexes and spontaneous respiration. DSI allows a longer time for intubation, increases the apnea time, decreases the risk of gastric insufflation, and decreases the risk of aspiration. DSI may prevent peri-intubation acidosis, carbon dioxide elevations, cardiac arrest, hypotension, hypoxemia, morbidity, and mortality.[1] A bag-valve device is not used to ventilate the patient in DSI. This may result in a decreased gastric insufflation and the subsequent risk of aspiration.

INDICATIONS

The primary indication for DSI is to provide controlled sedation to achieve adequate preoxygenation and denitrogenation in the patient intolerant of desaturating (i.e., oxygen saturation < 93%) when emergent rapid sequence intubation (RSI) would be otherwise unsafe due to the risk of severe hypoxemia.[1] Some patients may not need to be intubated once the sedation has led to dramatic increases in oxygen saturation. Improved cooperation along with the adequate oxygen saturation levels may allow the Emergency Physician to observe the patient before immediately continuing to intubation. Safe and controlled endotracheal intubation is performed to secure the patient's airway once the oxygen reserve and saturation level have reached an

appropriate value. DSI allows essential procedures (e.g., nasogastric tube placement) to be performed prior to intubation.

CONTRAINDICATIONS

Contraindications include situations in which definitive airway control is needed in a non–spontaneously breathing patient. Patients who present with increased aspiration risk are not candidates for DSI. A relative contraindication is in the patient with hypertension, tachycardia, and increased intracranial pressure when the sympathomimetic effects of ketamine will be undesirable (Chapter 12). Contraindications to the administration of ketamine (Chapter 12) are contraindications to DSI. Patients under the influence of illicit substances or medications or with psychiatric disorders that cause delirium or hallucinations may experience additive effects with the administration of ketamine. Emergency Physicians with minimal to no experience with providing ketamine sedation should not provide DSI.

EQUIPMENT

The equipment required for DSI is the same as that for any intubation (Chapters 16 and 18). Difficult airway equipment should be readily available if the patient cannot be intubated (Chapter 14). This can include laryngeal mask airways (Chapter 26), a cricothyroidotomy tray (Chapter 32), a retrograde guidewire kit (Chapter 30), or a percutaneous transtracheal jet ventilation system (Chapter 31).

PATIENT PREPARATION

Provide supplemental oxygen via nasal cannula or nonrebreather mask. **Be prepared with the equipment, trained personnel, and monitoring systems before initiating DSI.** Apply a noninvasive blood pressure cuff, continuous pulse oximetry, and cardiac monitoring. Obtain intravenous (IV) access in two locations if possible. Use the intraosseous route if an IV cannot be obtained (Chapter 70).[7] Flush the IV line and angiocatheter with sterile saline to ensure it is patent. Prepare suction and ensure it is working in case it is needed. Set the IV line to keep open with a saline bag. Safely secure the patient using restraints or extra personnel to avoid harm to the patient or others in the area. Consider administering an antisialagogue (e.g., atropine or glycopyrrolate) if time allows to decrease excessive oral and respiratory tract secretions. Evaluate the patient's airway and devise a detailed plan for the intubation following the RSI or DSI technique. Have additional ketamine and the medications for RSI prepared and drawn up at the bedside. Have difficult airway equipment at the bedside.

TECHNIQUE

Consider DSI in the patient with unsuccessful attempts at preoxygenation secondary to agitation, combative behavior, confusion, delirium, or any other state resulting in an oxygen saturation of less than 93%. The procedure is summarized in **Figure 17-1**. Administer 1 to 2 mg/kg of ketamine over 10 to 20 seconds.[8] The slow intravenous push will result in a calmed and cooperative patient within 30 to 45 seconds. **Ketamine will not blunt the patient's respirations or airway reflexes.** Administer a second dose of 0.5 to 1.0 mg/kg of ketamine if the patient remains agitated and is not tolerating the preoxygenation. Place the patient in over 30° of head-up position for preoxygenation. The dissociative state produced by the ketamine will allow the application of preoxygenation.[9]

Preoxygenation can proceed with the use of a nonrebreather mask, nasal cannula, or, in some cases, noninvasive continuous positive airway pressure (Chapter 11) of 5 to 15 cmH_2O. Continue preoxygenation for another 2 to 3 minutes once the patient reaches an oxygen saturation > 95%. Intubate the patient with or without paralytics while maintaining adequate oxygen saturation (Chapters 16 and 18). Neuromuscular blocking agents can be used for paralysis (Chapter 12).

PEDIATRIC CONSIDERATIONS

Sedation for the pediatric population requires expertise. The Emergency Physician handling the pediatric airway must be familiar with pediatric anatomy, medication dosage, and respiratory physiology.[10] The noncompliant pediatric patient just before sedation is very similar to the agitated adult patient who does not tolerate preoxygenation.[11] The described technique of DSI for adults can be applied to children with appropriate weight-based dose adjustment of the medications. Intramuscular injection of ketamine is described but reserved for children without IV access due to the unpredictable time and depth of sedation. DSI has not been clinically studied in the pediatric population. Future studies and clinical trials have to confirm the effectiveness and safety of DSI in this group.

ALTERNATIVE TECHNIQUES

Dexmedetomidine is an alpha-2 agonist that provides sedation with no blunting of respiratory drive or airway reflexes.[12] A dose of 1 μg/kg administered over 10 minutes will lead to sedation. The use of dexmedetomidine will require a longer waiting period before most patients allow preoxygenation when compared with ketamine, which requires 30 to 45 seconds.[13] Other agents include droperidol, etomidate, and fentanyl derivatives.[1,14] These medications have not been adequately studied in the setting of DSI to make any recommendations.

ASSESSMENT

Monitor oxygen saturation levels, blood pressure, good chest rise and fall with ventilation, and adequate IV medication access. Evaluate the increase in oxygen saturation level with ketamine administration. **Confirm that endotracheal intubation is successful (Chapter 19).** This includes auscultation, fogging of the endotracheal tube with ventilation, and the presence of persistent end-tidal CO_2 (i.e., capnograph or a chemical colorimetric indicator).[15]

AFTERCARE

Some patients may not ultimately need to be intubated once the sedation has led to dramatic increases in oxygen saturation.[1] Improved cooperation along with the adequate oxygen saturation levels may allow the Emergency Physician to observe the patient before immediately continuing to intubation. The aftercare is the same as for any intubated patient (Chapters 16 and 18). Secure the endotracheal tube with tape or a commercially available device. Administer adequate sedation and neuromuscular relaxation as necessary. Monitor oxygen saturation levels and airway pressures.

COMPLICATIONS

Complications of DSI are rare and similar to those orotracheal intubation (Chapters 16 and 18).[1] DSI can lead to complications; thus, a rapid back-up plan is needed in case of an impediment. DSI goes against the technique described as RSI. The amount of time allowed for preoxygenation with sedation medication can lead to gastric contents entering the oropharynx and significant aspiration. Do not overlook the risk of pulmonary aspiration despite the use

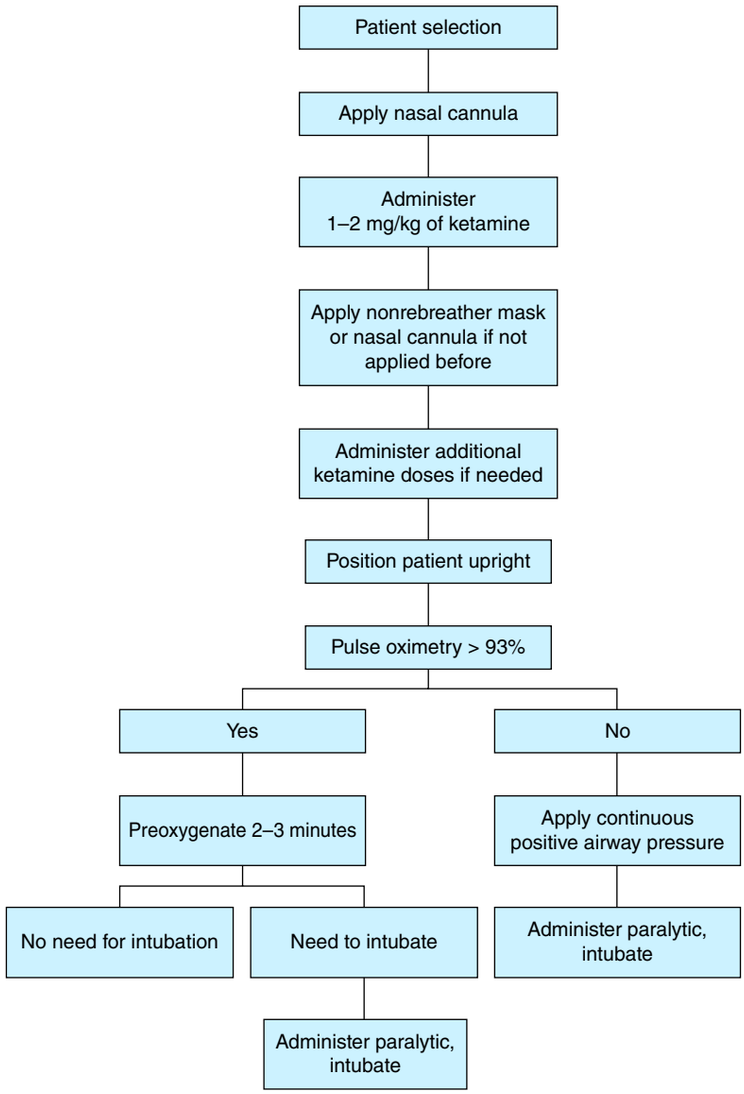

FIGURE 17-1. DSI algorithm.

of ketamine or dexmedetomidine to maintain spontaneous respirations and reflexes.[16]

Side effects of ketamine include excessive agitation, delirium, hallucinations, increased cardiac demand with tachycardia and hypertension, and increased airway secretions (Chapter 12). These side effects can lead to serious complications while attempting to sedate and intubate the patient. Obtaining a complete medical history and drug history before administering these drugs is crucial for safe outcomes.

SUMMARY

Effective preoxygenation and denitrogenation during the peri-intubation period allows the Emergency Physician to intubate the patient with adequate oxygen reserve and sufficient time before desaturation. Patients often arrive to the Emergency Department confused, combative, delirious, or uncooperative. These patients pose a great risk for desaturation and hypoxia during induction and intubation. The experienced and knowledgeable Emergency Physician can apply oxygen supplementation that is tolerated and effective. This rise in oxygen saturation and oxygen reserve volume in the alveoli can improve intubating conditions and overall patient oxygenation.

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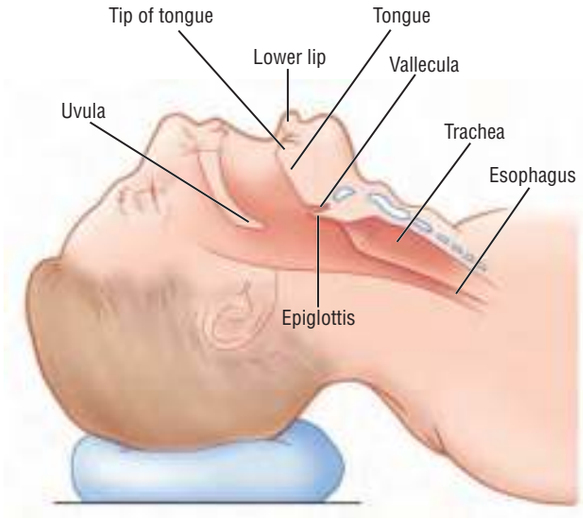


FIGURE 18-1. Schematic representation of the airway. The patient is in the "sniffing" position.

18 Orotracheal Intubation

Hyangwon Paek, Gennadiy Voronov, and Ned Nasr

INTRODUCTION

Airway control is the first and most critical action of the Emergency Physician. The "A" in the ABC's demands that no other action may take place until the airway is secure. Endotracheal (ET) intubation inserts an artificial airway connecting the respiratory system to the outside world and provides definitive control of the airway. All methods of support can be applied once the ET tube is in place. Nothing can help the patient if the airway is not secure. ET intubation can be accomplished by a variety of methods. The method of choice will be dictated by physician preference and experience, the patient's condition, and the available equipment. The most common method of ET intubation is orotracheal intubation. **There are no good alternatives to intubation when oxygenation and ventilation are threatened. All actions should be focused on the objectives of getting the ET tube placed quickly and in the right location.** The proper preparation, practice, and personnel can assure that the "nightmare airway" is an extremely rare event.[1]

ANATOMY AND PATHOPHYSIOLOGY

The discussion of anatomy starts at the lips and travels inward to end at the right mainstem bronchus. Visualize the normal structures expected and match them with what is seen as you approach the patient. Distortion occurs from edema or trauma. Structures may be hidden by vomit or blood. Airway structures are viewed upside down from the position of standing over the head of the supine patient. The potential for disorientation is multiplied due to the upside-down view.

Begin at the face and move inward (**Figures 18-1 and 18-2**). The philtrum of the upper lip will be located at the 6 o'clock (i.e., bottom) position. Symmetrical swelling, carbon deposits, blistering, or signs of trauma to the lips can indicate that the inner anatomy of the airway may be altered and the intubation more difficult. Open the patient's mouth and check the teeth for fractures, size, and the presence of removable dental devices. Large upper incisors (i.e., buck teeth) and/or limited jaw opening will make orotracheal intubation more difficult. The tongue hangs down from the floor of the mandible and ends with the tip against the maxillary incisors (**Figure 18-2**). Visualize the tongue as a hanging oval of tissue with two "tips." The first is the anterior tip of the tongue proper. The second is the posteriorly located epiglottis. The anatomic floor of this view is formed by the hard and soft palates, which end at the palatopharyngeal arch (**Figure 18-2**). The uvula is located inferiorly and in the midline. The palatoglossal arch and palatopharyngeal arch form twin vertical pillars that lie posterior to the molars of the upper teeth (**Figure 18-2**). All of these structures are potential sources of obstruction and must be evaluated for swelling, deformity, or trauma. The back wall is the posterior wall of the pharynx (**Figure 18-2**).

The airway bends 90° at the posterior wall of the pharynx to run almost parallel to the gurney. The root of the tongue and the

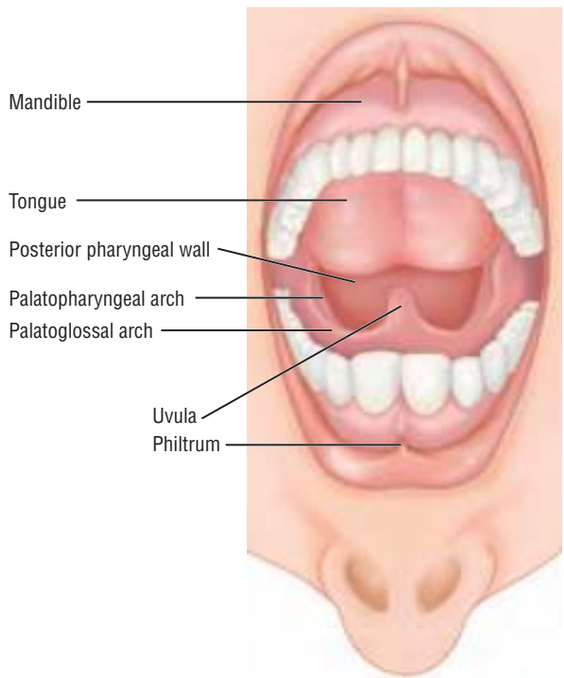


FIGURE 18-2. The oral structures as viewed from above the supine patient's head.

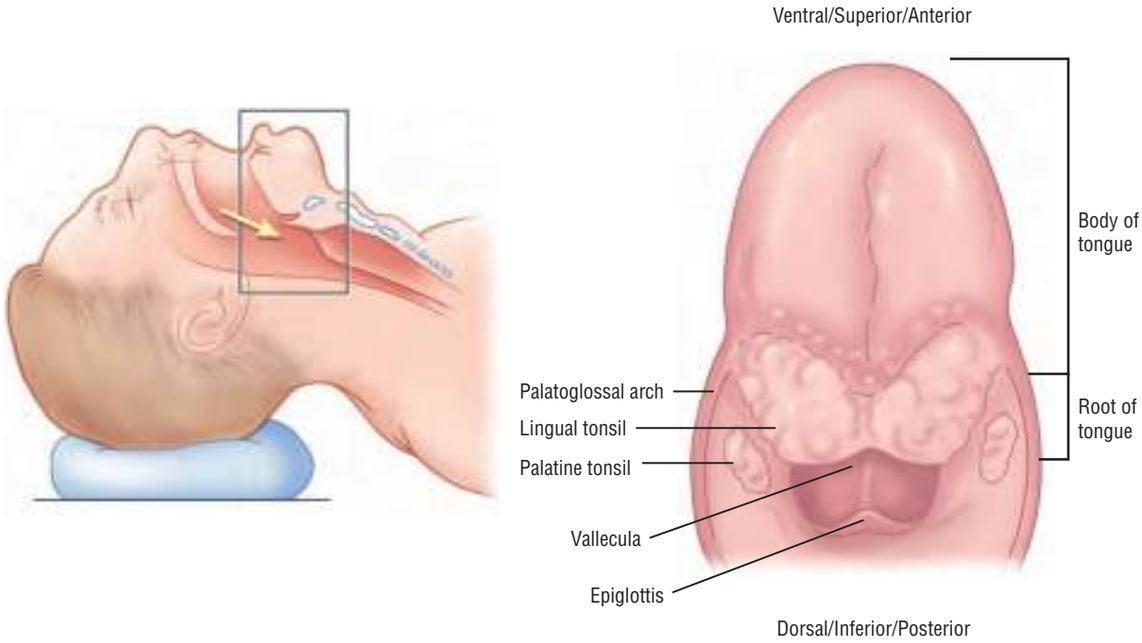


FIGURE 18-3. The tongue and adjacent structures as viewed from above the supine patient's head.

lingual tonsils are located at the 12 o'clock position when visualized from the perspective of the top of the patient's head (**Figures 18-3 and 18-4**). The tongue continues into a blind pocket known as the vallecula. The vallecula is continuous with the epiglottis. The epiglottis hangs with its tip pointing downward. Directly behind and protected by the epiglottis is the entry to the remainder of the airway (**Figure 18-4**). The esophagus lies at the 6 o'clock position. The hypopharynx appears like the number "8" from the viewpoint of the intubator. The top half is the airway and the bottom half the esophagus (**Figure 18-4**).

Under the epiglottis is the larynx (**Figure 18-5).** The vocal cords are located in the midline and form an "A" shape, with its apex superior and toward the epiglottis. **Identifying the vocal cords is important since the visualization of the ET tube passing between the vocal cords is proof of a successful ET intubation.** The arytenoid cartilages are paired structures (**Figure 18-5**). One lies at the posterior aspect of each vocal cord. The aryepiglottic folds are paired structures that span from the lateral edge of the epiglottis to the arytenoid cartilages (**Figure 18-5**). They contain the muscles that move the arytenoid cartilages and subsequently move (i.e., open and close) the vocal cords. The trachea bifurcates at the carina into the right and left mainstem bronchi. The cartilaginous tracheal rings are sometimes visible through the vocal cords.

INDICATIONS

Any threat to oxygenation and/or ventilation is a relative indication for orotracheal intubation. Remove the threat if it is simple and easily removed. **Orotracheal intubation is required if there is uncertainty that the patient's airway patency, respiratory drive, or oxygenation cannot be maintained without intervention. Time is of the essence. The decision to intubate early can make**

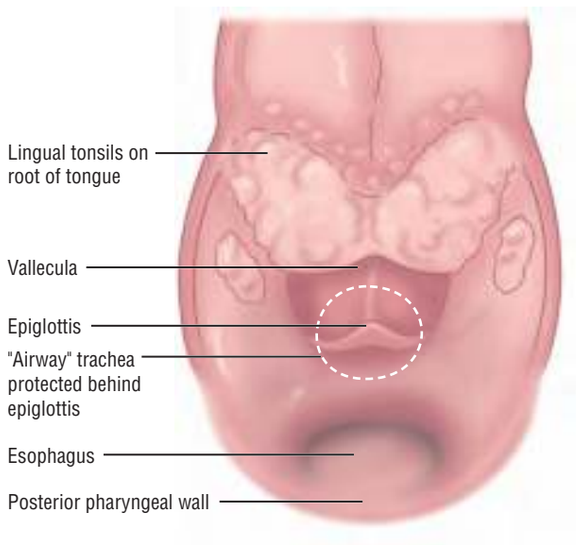


FIGURE 18-4. Structures of the hypopharynx as viewed from above the supine patient's head.

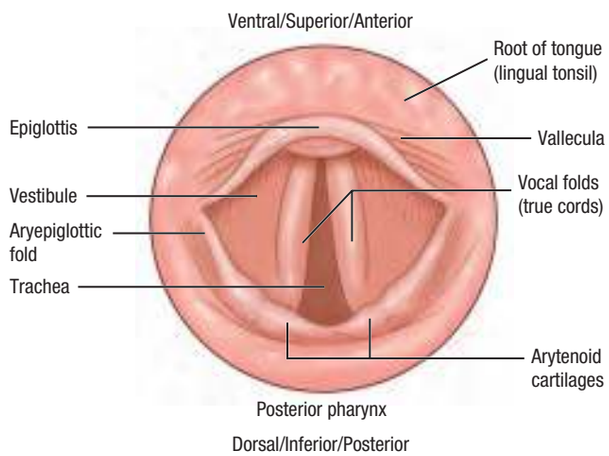


FIGURE 18-5. The structures of the glottis as viewed from above the supine patient's head.

the difference between a controlled, successful procedure and a chaotic, "crashing" nightmare. Orotracheal intubation can be performed to administer resuscitation medications, ensure a patent airway, deliver oxygen, isolate the airway, reduce the risk of aspiration of gastric or oral contents, suction the trachea, ventilate the patient, and apply positive-pressure ventilation. Other indications for orotracheal intubation include altered mental status, head injury requiring hyperventilation, hypoxemia, hypoventilation, apnea, lack of a gag reflex, shock, and unconsciousness.

CONTRAINDICATIONS

Orotracheal intubation is relatively contraindicated in patients who do not need it, who are likely to be injured by the procedure, or whose injuries make success unlikely. Spontaneous breathing with adequate ventilation and normal mental status may allow less invasive techniques such as continuous positive airway pressure (CPAP) in patients whose medical conditions are likely to respond quickly to interventions (e.g., cardiogenic pulmonary edema or pneumonitis).[2] Trauma patients with likely cervical spine injury or anterior neck wounds and severely immobile arthritis patients may be injured by the manipulation required during orotracheal intubation.[3] Successful orotracheal intubation may be impossible with severe orofacial injuries, bleeding, deep airway obstruction, or gross deformity of the head and neck. A quickly changing obstruction (e.g., edema or an expanding hematoma) may require a surgical airway if orotracheal intubation is delayed. **Choose a surgical airway if the manipulation or time required for orotracheal intubation puts the patient at risk for spinal injury or hypoxia.**[4] Orotracheal intubation should not be performed by individuals unfamiliar with the equipment and technique.

EQUIPMENT

- ET tubes, various sizes (**Table 18-1**)
- 10 mL syringe
- Water-soluble lubricant or anesthetic jelly
- Wire stylet, malleable type
- Laryngoscope handle
- Fresh batteries for the laryngoscope
- Laryngoscope blades, various sizes and shapes
- Supplemental oxygen with appropriate tubing and connectors
- Nonrebreather oxygen masks, various sizes
- Wall suction with appropriate tubing
- Yankauer suction catheter
- Bag-valve device, various sizes
- Oral airways, various sizes
- Nasal airways, various sizes
- Benzoin adhesive
- Tape
- Pulse oximeter
- Cardiac monitor
- Automatic sphygmomanometer
- End-tidal carbon dioxide (CO_2) monitor or device
- Cricothyrotomy tray as backup
- Crash cart
- Resuscitation medicines
- Personnel (respiratory technician, medication nurse, in-line stabilization assistant, recorder)
- Medications (premedications, induction, anesthetics, paralytics) (**Table 18-2**)

Many institutions make their own intubating/airway kit. It contains all the commonly used equipment in a portable container or cart that can be moved wherever required in the Emergency Department. While differences will exist between institutions, the kit commonly includes the following: adult and pediatric laryngoscope handles, various sizes and types of laryngoscope blades, various sizes of oropharyngeal airways, various sizes of nasopharyngeal airways, tongue blades, malleable stylets, various sizes of ET tubes, syringes, tape, and commercially available devices to secure the ET tube. Some institutions may have a single kit or separate kits for adult and pediatric patients.

TABLE 18-1 Oral ET Tube Sizes and Positioning Based on Patient Age

Patient's age	Size* (French)	Internal diameter (mm)	External diameter† (mm)	Distance inserted from lips (cm)
Premature	10	2.5	3.3	9–10
Full-term/newborn	12	3.0	4.0–4.2	11
1–6 months	14	3.5	4.7–4.8	11
6–12 months	16	4.0	5.3–5.6	12
1–2 years	18	4.5	6.0–6.3	13
3–4 years	20	5.0	6.7–7.0	14
5–6 years	22	5.5	7.3–7.6	15–16
7–8 years	24	6.0	8.0–8.2	16–17
9–10 years	26	6.5	8.7–9.3	17–18
11–13 years	28–30	7.0	9.3–10.0	18–20
Female ≥ 14 years	28–30	7.0	9.3–10.0	20–22
Male ≥ 14 years	32–34	8.0	10.7–11.3	22–24

*Calculated as follows: External diameter (mm) × π.
†Varies by manufacturer.
Source: Modified from reference 6.

ET TUBES

The ET tube is a clear polyvinyl chloride disposable tube that is open on both ends (**Figure 18-6**).[5] The proximal end contains a standard size 15 mm connector that will attach to the bag-valve device, a ventilator, and other sources of positive-pressure ventilation. The distal end is beveled. It has a perforation located approximately 0.5 to 0.75 cm from the tip and opposite the bevel. This perforation is known as the Murphy eye. Printed on the tube are the size, a radiopaque line to aid in radiographic visualization, and 1 cm incremental marks beginning at the tip. An inflatable cuff or balloon is positioned proximal to the Murphy eye. A pilot balloon with an inflation port to inflate the cuff hangs from the proximal third of the ET tube. An air-filled syringe attaches to the inflation port to inflate and deflate the cuff.

The ET tube cuff is a high-volume, low-pressure balloon. **The cuff is designed to accommodate a high volume of air before the intracuff pressure rises.** This is an extremely important feature. Intracuff pressure is transmitted to the delicate tracheal mucosa where it can cause pressure necrosis and ischemia.

The Parker Flex-Tip tracheal tube is curved, designed to facilitate rapid intubation, flexible, and nontraumatic, with a distal tip geometry that is different from a traditional ET tube (Parker Medical, Highlands Ranch, CO).[6] The tube is designed to flex and slide past airway structures rather than getting caught and causing trauma to these structures. The tip of the Parker tube lies close to the stylet and

TABLE 18-2 Rapid Sequence Induction Medications for Specific Patient Profiles

Patient type	Premedication*	Induction and paralysis†
"Normal adult"	Rocuronium (0.06 mg/kg)	Etomidate (0.3 mg/kg) or propofol (1.5–2.5 mg/kg) and succinylcholine (2 mg/kg)
"Normal child"	Rocuronium (0.06 mg/kg) and atropine (0.02 mg/kg, min dose 0.1 mg)	Propofol (2–3 mg/kg) and succinylcholine (2–3 mg/kg)
Asthma, adult	Lidocaine (1.5 mg/kg) and glycopyrrolate (0.1–0.2 mg)	Ketamine (1–2 mg/kg) or propofol (1.5–2.5 mg/kg) and succinylcholine (2 mg/kg)
Asthma, child	Lidocaine (1.5 mg/kg) and atropine (0.02 mg, min 0.1 mg)	Ketamine (1–2 mg/kg) or propofol (2–3 mg/kg) and succinylcholine (2–3 mg/kg)
Head injury, adult	Rocuronium (0.06 mg/kg) and lidocaine (1.5 mg/kg)	Etomidate (0.3 mg/kg) or propofol (1.5–2.5 mg/kg) and succinylcholine (2 mg/kg)
Head injury, child	Rocuronium (0.06 mg/kg) and atropine (0.02 mg/kg, min 0.1 mg) and lidocaine (1.5 mg/kg)	Etomidate (0.3 mg/kg) or propofol (1–3 mg/kg) and succinylcholine (2 mg/kg)
Head injury, adult, hypotensive	Rocuronium (0.06 mg/kg) and lidocaine (1.5 mg/kg)	Etomidate (0.2 mg/kg) and succinylcholine (1.5 mg/kg)
Head injury, child, hypotensive	Rocuronium (0.06 mg/kg) and atropine (0.02 mg/kg, min 0.1 mg) and lidocaine (1.5 mg/kg)	Midazolam (0.05–0.2 mg/kg) or fentanyl (1–3 µg/kg) and etomidate (0.3 mg/kg) and succinylcholine (2 mg/kg)
Hyperkalemia or renal failure, adult	None	Etomidate (0.2 mg/kg) or propofol (1–2 mg/kg) and rocuronium (0.6 mg/kg) or vecuronium (0.01 mg/kg)
Hyperkalemia or renal failure, child	None	Etomidate (0.2 mg/kg) or propofol (1–2 mg/kg) and rocuronium (0.6 mg/kg) or vecuronium (0.01 mg/kg)
Status epilepticus, adult	None	Etomidate (0.3 mg/kg) or propofol (1.5–2.5 mg/kg) and succinylcholine (2 mg/kg)
Status epilepticus, child	None	Etomidate (0.3 mg/kg) or propofol (1–3 mg/kg) and succinylcholine (2–3 mg/kg)
Pregnancy	Aspiration prophylaxis: ranitidine (50 mg) and metoclopramide (10 mg) Rocuronium (0.06 mg/kg) and glycopyrrolate (0.1–0.2 mg)	Ketamine (1–2 mg/kg) or propofol (1.5–2.5 mg/kg) and succinylcholine (1 mg/kg)

*Given 3 minutes before intubating ($T - 3$).
†Given simultaneously at the beginning of intubation ($T = 0$) and wait for 45 to 60 seconds for onset of paralysis.

minimizes this gap that could result in the ET tube getting caught. The Parker tube is available in many different styles.

The choice of ET tube size will vary based on the patient's age, anatomic anomalies, body habitus, and airway anatomy (**Table 18-1**). **The ET tube is sized based on the internal diameter (ID) measured in millimeters.** The size is printed onto the surface of the ET tube for reference. The sizes begin with 2.5 mm and increase in 0.5 mm increments. Some generalities hold true in most patients. Adult males usually require a size 7.5 to 8.0 cuffed ET tube. Adult females usually require a size 7.0 to 7.5 cuffed ET tube.

ET tube selection in children can be made by one of several methods. A Broselow tape will identify the proper size tube. Visually select a tube with an ID that matches the size of: the width of the nail of the patient's little finger, the width or diameter of the fifth finger, the diameter of the distal phalanx of the third finger, or the external nares luminal diameter. All of these visual methods will approximate the same size ET tube. The following formula may be used to confirm the uncuffed ET tube size: (16 + child's age in years) ÷ 4. Tables based on the child's age, length (e.g., Broselow tape), or weight may be used to estimate the proper ET tube size. An uncuffed ET tube was recommended to be used in children under 28 days of age to prevent the complications of subglottic and tracheal stenosis. Select and prepare a second ET tube that is one size smaller in case the patient's airway is smaller than expected.

FIGURE 18-6. A variety of ET tubes. (Photo courtesy of Eric F. Reichman, PhD, MD.)

Traditional teaching holds that cuffed ET tubes increase the risk of ischemic damage to the tracheal mucosa due to compression between the cuff and the cartilaginous rings. This results in the old mandate to use uncuffed ET tubes in children younger than 8 years of age. There have been numerous advances in modern ET tubes that are changing this orthodoxy.[7] Some current guidelines recommend, but do not require, a cuffed ET tube for children older than 28 days of age. In the first 28 days of life, the cricoid narrowing functions as a cuff. Use of cuffed ET tubes in infants and neonates less than 28 days of age has been demonstrated to be safe.[8-13] For children over 28 days of age, the cuffed ET tube is just as safe as an uncuffed ET tube.[14,15] The high-volume, low-pressure cuffs found on new ET tubes allow the cuff to produce a seal at much lower pressures. The use of cuffed ET tubes is becoming more common in pediatric Intensive Care Units and Emergency Departments. Several studies have shown no increase in postintubation stridor or reintubation when cuffed ET tubes are used in controlled settings with regular cuff pressure monitoring.

Potential benefits in children from the use of cuffed ET tubes include some protection from aspiration, allowance of ventilation at higher pressures, maintenance of more consistent ventilatory parameters, and fewer changes of inappropriately sized ET tubes.[16] Cuffed ET tube size can be calculated using the following equation: (age in years/4) + 3; cuffed ET tube size can also be determined by using an ET tube one-half size smaller than the calculated uncuffed ET tube size.[17,18]

All ET tubes should be examined for defects before use. Attach a 10 mL syringe filled with air to the pilot balloon inflation port. Inject the air to inflate the cuff. The cuff should inflate symmetrically and have no air leak. Deflate the cuff completely. Leave the syringe attached to the pilot balloon in order to inflate the cuff after the ET tube has been inserted into a patient's airway. Discard any defective ET tube and prepare a new ET tube.

FIGURE 18-7. A variety of laryngoscope handles. (Photo courtesy of Eric F. Reichman, PhD, MD.)

FIGURE 18-9. The Miller laryngoscope blades. (Photo courtesy of Eric F. Reichman, PhD, MD.)

LARYNGOSCOPES

The laryngoscope is a handheld device that is used to elevate the tongue and epiglottis to expose the glottis. **It is a device that is held in the left hand regardless of which hand of the user is dominant.** It consists of a handle (**Figure 18-7**) and a blade (**Figures 18-8 and 18-9**). The handle contains the battery for the light source. The distal end of the handle has a fitting where the handle connects to the blade. A transverse bar indicates where the indentation on the proximal blade attaches to the handle. There are many types of laryngoscope handles. They all have the same basic design and are available in a variety of diameters and lengths (**Figure 18-7**). Smaller diameter (i.e., thinner) laryngoscope handles may be better suited for use with the smaller sized pediatric laryngoscope blades. Shorter "stubby" laryngoscope handles may offer an advantage when proceeding with intubation of obese or barrel-chested patients, especially in cases where the neck cannot be manipulated. The shorter handle will not catch on the chest wall during attempts to place the laryngoscope blade in the patient's mouth.

The laryngoscope blade may have a removable bulb that is attached to its distal third. A fiberoptic bundle within the blade transfers power from the handle to the bulb. Other laryngoscope blades only contain fiberoptic bundles which transmit light from the light source located within the handle. The choice of the type and size of laryngoscope blade will vary with physician experience and preference. **The best blade is one that the intubator feels comfortable and confident using.** The curved Macintosh blade is most commonly used (**Figure 18-8**).[19] It is the easier blade to use for those with little experience with orotracheal intubation. Many feel that it requires less forearm strength to use as compared to the straight blade. The large flange allows for easier control of the tongue, and the flat curved shape of the spatula fits the natural curve of the tongue. The straight Miller blade is often used in patients with a floppy epiglottis, large tongue, prominent incisors (i.e., buck teeth), or overriding teeth (**Figure 18-9**).[20] Children can be intubated with either a Macintosh or Miller blade.[21,22]

The tip of the curved Macintosh blade fits into the vallecula and indirectly lifts the epiglottis to expose the vocal cords (Figure 18-10). A size 2 blade is used for children 3 to 6 years of

FIGURE 18-8. The Macintosh laryngoscope blades. (Photo courtesy of Eric F. Reichman, PhD, MD.)

FIGURE 18-10. Use of the Macintosh blade. It is inserted into the vallecula to elevate the mandible, tongue, and epiglottis as a unit.

FIGURE 18-11. Use of the Miller blade. It is inserted below the epiglottis to elevate the mandible, tongue, and epiglottis as a unit.

age. A size 3 blade is used for children starting at about age 6, for women, and for small to average-size males. A size 4 blade is usually reserved for large males. The size 4 blade requires more power to lift the handle. This is because as the distance from the tip of the blade to the handle increases, the harder it becomes to lift the tongue and epiglottis as well as to maintain the blade straight. **Choosing the shortest blade that is long enough to reach the vallecula will allow the best intubation attempt.** The Macintosh Extended Flange (i.e., the English blade) has a spatula of the blade that is smooth and has an extended gentle curve. It is widely used because some contend that it allows better visualization due to the continuous natural curve of the spatula that is not present in the standard curved Macintosh blade.[23]

The tip of the straight Miller blade goes directly under the epiglottis to lift it and the tongue to expose the vocal cords (Figure 18-11).[19] A straight blade makes controlling the epiglottis and tongue easier than with a curved blade. It also makes visualization of the vocal cords easier due to its smaller flange profile. A size 0 blade is used for premature babies and neonates up to approximately 1 month of age. A size 1 blade is used for children from approximately 1 month of age to toddlers up to 2 years of age. A size 2 blade is used for children 3 to 6 years of age. Children between 6 and 12 years of age may require either a size 2 or 3 blade depending on their body size. A size 3 blade is used for adolescents, women, and average-size males. A size 4 blade is rarely used, and then primarily for large males. If one cannot remember the proper blade size, it can be determined based on patient anatomy.[24] Place the base of the blade, excluding the handle insertion block, at the level of the patient's upper incisor teeth. The tip of the blade should be located within 1 cm proximal or distal to the angle of the patient's mandible. Correct blade size allows for approximately 90% of first attempt intubations to be successful versus 57% if the blade is too small.[24]

There are a wide variety of laryngoscope blades commercially available. They tend to be variations of the curved Macintosh or straight Miller blades (**Figure 18-12**). The McCoy blade is a curved blade with a hinged tip (**Figure 18-12*A***). The tip can be flexed by depressing a lever on the laryngoscope handle. This flexion augments indirect elevation of the epiglottis by stretching the hypoepiglottic ligament. The Flexiblade (Arco-Medic Ltd., Omer, Israel) laryngoscope also has a levering blade (**Figure 18-12*B***). The extra lift provided by these blades may improve visualization of the vocal cords.[25,26] The Propper Flip-Tip laryngoscope blade (Propper Manufacturing Co., Long Island, NY) has a lever that elevates its tip up to 90° to lift the epiglottis (**Figure 18-12*C***). Other variations of the Macintosh blade include the incorporation of a variety of prism or mirror systems. These modifications allow for indirect visualization of otherwise obscured vocal cords. The Belscope (International Medical Inc., Burnsville, MN), Truvue EVO2 (Truphatek International Ltd., Netanya, Israel) (**Figure 18-12*D***), Lee-Fiberview (Anesthesia Medical Specialties, Beaumont, CA), and Viewmax (Rusch, Duluth, GA) blades are examples of these types of modifications.

There have also been many variations of the straight laryngoscope blade. Today, the Miller is the most popular straight blade. Other variants include the Phillips (**Figure 18-13**) and Henderson blades. These modify components such as the cross section, the blade channel width, tip style, and light source placement. These modifications represent efforts to avoid such problems as dental trauma, laceration of the ET tube cuff, tongue displacement, tip trauma, and obscuration of the light source by secretions.

In addition to traditional metal laryngoscope blades, plastic single-use blades are also available (**Figure 18-14**). These single-use plastic blades were developed, in part, because of concerns regarding possible transmission of infectious agents by incompletely sterilized metallic blades. A study comparing plastic versus metallic Macintosh laryngoscope blades in 1177 patients found that metallic blades had higher first attempt intubation rates, had fewer cases of difficult intubation, and used alternative airway interventions less often than when intubation was attempted with a plastic blade.[27] Plastic blades cause less dental trauma when used on dental models.[28] The rates of dental trauma in patients when compared to metal blades are not known. The use of single-use disposable plastic blades cannot be recommended at this time unless circumstances do not allow proper cleaning of metallic laryngoscope blades.[29]

STYLETS

The stylet is a semirigid piece of metal that is usually bendable (**Figure 18-15 and Table 18-3**). It is often plastic coated. It inserts into the lumen of the ET tube. It should be lubricated with water-soluble lubricant or anesthetic jelly prior to insertion into the ET tube. **The tip of the stylet should be 1 cm proximal to the tip of the ET tube to prevent injury to the patient's airway.** The ET tube, with a stylet, can be bent to maintain a specific shape. The stylet is used to facilitate passage of the ET tube through the vocal cords. It is commonly bent into a "hockey-stick" or "J" shape for most intubations. A greater curvature is often used for intubations when the larynx is "anterior," in difficult intubations, and in "blind" intubations.

A modification of the traditional stylet is the Parker Flex-It Directional Stylet (Parker Medical, Highlands Ranch, CO). This is a plastic articulating stylet that requires no prebending (**Figure 18-15*B***). The stylet has a built-in gentle curve. It has a button on its proximal end that extends from the ET tube. When pushed with the thumb, it allows the curvature of the ET tube to be continuously adjusted during intubation attempts.

Another modification of the traditional stylet is the Rapid Positioning Intubation Stylet or RPiS (Airway Management Enterprises LLC, Wellesley, MA). The RPiS was designed for difficult airways when the vocal cords are seen but the ET tube does not bend to go into the airway (**Figure 18-15*C***). It is a hand-controlled intubating stylet with a tip that can flex and retroflex. The RPiS can accommodate ET tubes with internal diameters between 6.0 and 9.0 mm.

FIGURE 18-12. Some of the variations of the Macintosh. ***A.*** McCoy blade. ***B.*** Flexi blade. ***C.*** Propper Flip-Tip blade. ***D.*** Truvue EVO2 blade.

FIGURE 18-13. The Phillips variation of the Miller blade.

FIGURE 18-14. An example of a plastic, single-use blade.

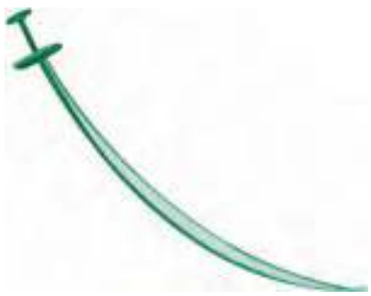
FORCEPS

The McGill forceps is the standard used by most institutions (**Figure 18-16*A***). The forceps are used to introduce the ET tube into the patient's trachea from the hypopharynx under direct visualization. It is available in infant, child, and adult sizes. The forceps tips twin blades for gripping are offset with serrated tips. It is curved at an oblique angle between the handles and the blades to prevent the view of the patient's airway from being obscured. The ends of the blades are rounded to minimize trauma.

The Boedeker forceps (Karl Storz Endoscopy, El Segundo, CA) is an alternative to the McGill forceps (**Figure 18-16*B***). It is also curved but does not put the working end into the view of the intubator.

The Tylke forceps (Rodinia LLC, Jupiter, FL) is a new device that is simple to use, safe, and an effective alternative to the McGill forceps (**Figure 18-16*C***). It allows nasotracheal intubations 25% of the time and reduces the possibility of torn cuffs and vocal cord trauma. The circular tip of the Tylke forceps prevents losing control of the ET tube, without having to grip the ET tube firmly, and allows the aiming of the ET tube in the desired direction. The twist and bend of the Tylke forceps prevent the vocal cords from being visually obstructed and provide improved access to the trachea. It is available in three sizes (i.e., adult, adolescent, and child).

A



B

C

FIGURE 18-15. The intubating stylet. ***A.*** It may be bent into any required shape. When inserted into an ET tube, it will form the ET tube into the desire shape. The most common shapes are the "J" and the "hockey stick." ***B.*** The Parker Medical Flex-It. ***C.*** The Rapid Positioning Intubating Stylet.

TABLE 18-3 Characteristics of Some Stylets Available in the Emergency Department

Name	Manufacturer	Comments
CoPilot VL Rigid Stylet	Magaw Medical	Reusable Fits ET tube ≥ 6.0 mm inner diameter
GlideRite Rigid Stylet	Verathon	Reusable Fits ET tube ≥ 6.0 mm inner diameter Matches GlideScope angulated blade
Muallem ET Tube Stylet	VBM Medizintechnik	Atraumatic soft Coude tip
Optishape Stylet	Teleflex	4 sizes Fits ET tube with inner diameter (mm) of 2.5–3.5, 4.0–5.5, 5.0–6.5, and 7.0–9.0
Rapid Positioning Intubating Stylet (RPiS)	Airway Management Enterprises	Atraumatic soft tip Tip allows 180° of flexion and retroflexion Fits ET tube ≥ 6.0 mm inner diameter
Standard ET Tube Stylet	Various	Malleable Single-use and disposable Available in multiple lengths and diameters
Truflex Flexible Stylet	Teleflex	30–60° lift of flexible tip Fits ET tube 6.5–8.5 mm inner diameter Adjustable stopper for different length ET tube

Use of the Tylke forceps is simple (**Figure 18-16*D***). Insert the Tylke forceps into the mouth. Grab the ET tube either from the right side or slide it over the tip of the ET tube with the Tylke forceps vertical. Another method is to tilt the Tylke forceps left 90° to encircle the tube from the top and then return the Tylke forceps to a vertical position. Aim the Tylke forceps at the vocal cords and have an assistant advance the tube through the vocal cords and into the trachea. Pull the Tylke forceps back to the oropharynx while loosely wrapped around the tube. Rotate the hand to the right, open, and disengage the Tylke forceps. Carefully remove the Tylke forceps from the mouth.

PREPARATION

PHYSICIAN

Once the decision to intubate has been made, the Emergency Physician must use their training and experience to begin leading the team toward a successful intubation. Although the process must move quickly, the Emergency Physician must, by example, ensure a calm and orderly environment. Making the decision to intubate earlier allows the team to follow a shorter and easier time line. The Emergency Physician must visualize this time line and identify actions and potential problems before they occur. **A backup plan should also be available in case orotracheal intubation is impossible.** Any Emergency Department patient about to be intubated is a high priority, so do not be afraid to use resources liberally. Obtain assistants to help as soon as the decision to intubate is made.

PERSONNEL

Shortly before the procedure begins, assemble the entire team near the bed and go over "the game plan" calmly and quickly. All personnel involved with the procedure should be gloved, gowned, and masked. Eye protection should be worn by all personnel to protect against splash injury from blood and secretions. **Explicitly identify**

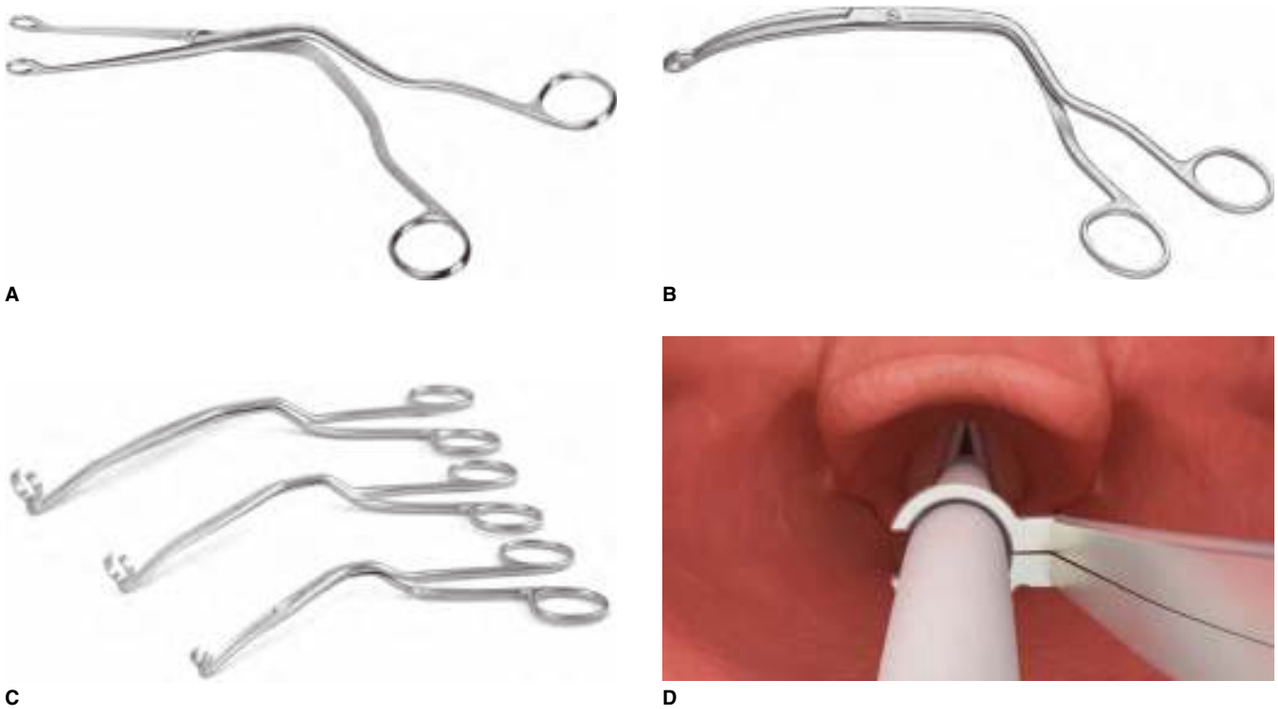


FIGURE 18-16. Forceps used to aid intubation. ***A.*** The McGill forceps. ***B.*** The Boedeker forceps. (Photo courtesy of Karl Storz Inc.) ***C.*** The child, adolescent, and adult versions of the Tylke forceps. (Photo courtesy of Rodinia LLC.) ***D.*** The Tylke forceps easing an ET tube through the vocal cords. (Photo courtesy of Rodinia LLC.)

assistants and assign their roles early. Give instructions clearly and calmly before the procedure begins. It is helpful to write down medications and doses in the order that they will be given and to review them quickly with the medication nurse. A designated nurse should draw up, label, and administer the medications followed by a saline flush. It should be emphasized that during the procedure this will be a particular nurse's only role. The respiratory assistant has three important tasks: helping to ventilate the patient, applying cricoid pressure, and handing the ET tube to the intubator so that visual contact with the vocal cords is not lost. A third assistant should be explicitly instructed as to how and when the team leader would like the patient's neck secured if cervical spine immobilization is needed.

EQUIPMENT

The mnemonic **SOAPME** (Chapter 16 and **Table 16-8**) can be used to help review the equipment required for intubation (**s**uction, **o**xygen, **a**irway, **p**harmacology, **m**onitoring, and **e**quipment).[30] Check that the room is ready and all equipment is within arm's reach. Turn on the suction and the oxygen. Confirm that both systems work. Attach the suction tip and check to see whether there is a small finger hole in the barrel that must be covered for the suction to work. If so, close it with a piece of tape so that the suction is always on. This is not a concern if a Yankauer suction catheter is being used. Ensure that the suction tubing is long enough to reach the center of the bed. Place the suction catheter under the mattress to the right of the patient's head and within easy reach. Place the spare suction tip nearby. Set the oxygen flow regulator to 15 L/min. Apply a nonrebreather mask to the oxygen and the patient. Place the bag-valve device near the head of the bed and within easy reach. Confirm that the noninvasive blood pressure cuff, cardiac monitor, and pulse oximeter are working and attached to the patient. Confirm that the end-tidal CO_2 monitor is nearby and working. If such a monitor is unavailable, a disposable in-line monitoring colorimetric device should be available. Ensure that the patient has at least one working intravenous line.

Assemble the intubation equipment. Place the proper size laryngoscope blade on the handle. Open the blade and confirm that the light works. Close the blade into the ready position, flat against the handle to keep the bulb cool and not drain the batteries. Take the ET tube and the backup smaller one and prepare them. Insert the 10 mL syringe into the inflation port of the pilot balloon for the ET tube cuff. Inject enough air to inflate the balloon. Deflate the balloon until it is completely flat against the ET tube if there is no leak. **Leave the syringe attached.** Liberally lubricate the stylet with water soluble lubricant. Insert the stylet into the ET tube until its tip is 1 cm proximal to the distal tip of the ET tube. Place a bend in the stylet as it enters the proximal end of the ET tube to keep the stylet from advancing. Bend the stylet-ET tube assembly into a curve roughly approximating a "hockey stick" or "J" (**Figure 18-15**). Lubricate the tip of the ET tube and the collapsed cuff. This prevents the ET tube from getting caught on the epiglottis and making its advancement through the vocal cords difficult. Place the assembly back into the ET tube package. Place the ET tubes, laryngoscope, backup laryngoscope handle and blades, oral airways, and tape on a tray within easy reach of the bed. Check the room lighting. Raise the bed to minimize excessive bending and better visualize the patient's airway.

PATIENT PREPARATION

If the patient is competent and awake, explain the procedure, clarify advance directives, and obtain consent. If time permits, a history is especially helpful. The mnemonic **AMPLE** can help to provide quick information on **a**llergies, **m**edications, **p**ast medical history, **l**ast meal, and **e**vents leading to the current problem. Evaluate the patient's airway (Chapter 16).[31-34]

Confirm again that the appropriate monitoring sources are working and attached to the patient. Confirm adequate intravenous access. Place the patient, with a normal neck, in the "sniffing" position with the head extended at the atlantooccipital joint while the neck is relatively flexed.[35-39] A folded towel under the occiput helps to gently raise and tilt the head back into the proper position (**Figures 10-1, 18-1, and 18-17**). **Correct positioning is probably the most important preparation of the patient.** Place the obese patient into the head-elevated position using a ramp or pile of sheets under their head and shoulders (**Figure 10-2**). This position facilitates spontaneous ventilation, mask ventilation, and laryngoscopy.[40-42] It also prolongs the patient's oxygen saturation during and after rapid sequence induction.

Begin preoxygenation for 5 minutes before the procedure if the patient is breathing spontaneously and if time permits. Use a well-fitting nonrebreather mask with the oxygen flow regulator set at 15 L/min. This displaces nitrogen from the lungs and gives the patient a physiologic reservoir of oxygen for approximately 5 minutes while apneic. **Remember, 5 minutes of preoxygenation provides 5 minutes of protection.**[43] If bag-valve-mask ventilation is required, have an assistant apply posteriorly directed cricoid pressure to minimize gastric distention and decrease the chance of vomiting and aspiration. Have assistants ready to turn the patient onto their left side to minimize the risk of aspiration if vomiting occurs. Monitor the pulse oximeter to assure good oxygenation and ventilation. It should rise to the high 90s and remain there. If not, check the O_2 circuit from the wall to the patient and confirm that spontaneous breathing is still occurring.

TECHNIQUE

The evaluation and preparation for orotracheal intubation are complex and essential. The intubation will hopefully be quick and anticlimactic if the preparation is done well. Position the respiratory assistant to the right side near the patient's head. The intubator should stand at the head of the bed. Adjust the bed to place the mattress level with the intubator's umbilicus. Pull the bed away from the wall at least 2 feet and clear a "maneuvering space" of tubes, lines, and equipment to prevent distractions. The assistant should stand at the intubator's left hip and be ready to remove the cervical collar and hold the patient's neck in position if in-line cervical immobilization is needed.

Grasp the laryngoscope with the left hand. **It is a left-handed instrument regardless of the handedness of the intubator.** Pull it open and lock the blade onto the handle. Confirm that the light is functioning. The tip of the laryngoscope blade should be pointed toward the patient's chin. Pass the prepared ET tube and suction catheter to the respiratory assistant, who will place them into your right hand when asked. This allows the intubator to maintain constant visual contact with the patient's airway during the procedure.

Induction of anesthesia is the final preparation for orotracheal intubation. The choice of drug sequence is based on the physician's experience and the patient's condition (**Table 18-2**). A typical sequence begins with a defasciculating dose of a nondepolarizing neuromuscular blocking drug (**Table 16-9**). After 2 to 3 minutes, induce anesthesia with a sedative followed immediately by a paralytic agent (i.e., succinylcholine). Apply cricoid pressure. Once the patient's muscles are relaxed, perform the intubation as described below. Please refer to Chapters 12 and 16 regarding the complete details of the pharmacology of the induction agents and rapid sequence intubation. **Some patients, especially the old and sick, may stop breathing earlier than anticipated. Be prepared to intubate before the expected time of drug onset.**

Observe the patient's chest. Watch it rise. Note the time when chest rise stops. Place your right thumb on the patient's jaw. Gently pull down the lower lip and open the mouth. Reinspect the oral cavity. Remove any dentures or foreign bodies. Compare what you see with what you expect to see. Fix any problems as you go further into the airway. If blood or vomit is seen, ask for the suction catheter. **Apply the suction catheter without removing your gaze from the patient's airway.** When done suctioning, hold the suction catheter up for the assistant to take.

INTUBATING WITH THE (CURVED) MACINTOSH BLADE

Firmly grasp the laryngoscope in the left hand (**Figure 18-17*A***). Insert the tip of the Macintosh laryngoscope blade into the right side of the patient's mouth. Smoothly advance the blade inward while keeping slight upward pressure against the tongue. Use the blade to trap and push the tongue to the left as the blade is simultaneously moved to the midline, "clearing a path" for your gaze. Keep the left wrist firm. **Use the forearm, wrist, and hand as a single unit and avoid bending or flexing the wrist. It is essential to move the patient's tongue up and to the left.** The tongue will protect the mandibular teeth from being injured by the laryngoscope blade. It allows the laryngoscope blade to be moved away from the maxillary teeth. It also opens a path to visualize the patient's airway.

When the blade has been inserted all the way, lift the patient's airway up and forward exactly along the long axis of the laryngoscope handle, which is aimed toward a point directly above the patient's chin (**Figures 18-10, 18-11, and 18-17*B***). **Do not "cock" or "crank back" on the laryngoscope handle with your wrist or the back of the laryngoscope blade may break the patient's incisors.** The epiglottis should be seen at the base of the tongue.

A variable amount of force is required to lift the mandible, tongue, and soft tissues to visualize the vocal cords. More force is required to visualize the vocal cords in patients who have a large tongue, are obese, have redundant pharyngeal soft tissue, or have trismus. Grasp the laryngoscope handle as close to its base as possible in these patients. **The lower grasp provides more control of the laryngoscope and a mechanical advantage to apply more force without dental trauma.**

Advance the tip of the laryngoscope blade into the vallecula—the space between the base of the tongue and the body of the epiglottis (**Figures 18-10 and 18-17*B***). Lift the laryngoscope handle to raise the tongue, jaw, and epiglottis as a unit (**Figure 18-10**). Observe carefully as the epiglottis pivots upward and uncovers the glottis (**Figure 18-5**). The vocal cords should be visualized. Cricoid pressure (i.e., Sellick's maneuver) may make intubation more difficult. If the cricoid pressure is adversely impacting the view, it should be relaxed or even removed.[44] The application of pressure by an assistant in the sequence of pressing the thyroid cartilage **b**ack, **u**pward, **r**ightward, and **p**osteriorly can help bring the vocal cords into view when the intubator cannot apply more lifting force due to lack of strength or reluctance to lift the airway (e.g., a suspected neck injury).[45] **This is known as the BURP maneuver.**[46]

Other anterior neck manipulation maneuvers known as optimal external laryngeal manipulation (OELM) and bimanual laryngoscopy may be applied.[47-49] They differ from the BURP maneuver in that the intubator uses their right hand to manipulate the larynx into optimal position while simultaneously viewing the patient's airway and controlling the laryngoscope with their left hand. An assistant then assumes control of the larynx, maintaining the same position as the intubator proceeds to insert the ET tube. Alternatively, to limit laryngeal movement during the hand off, the intubator can use their right hand to manipulate an assistant's hand on the patient's larynx.[49] When properly positioned, instruct the

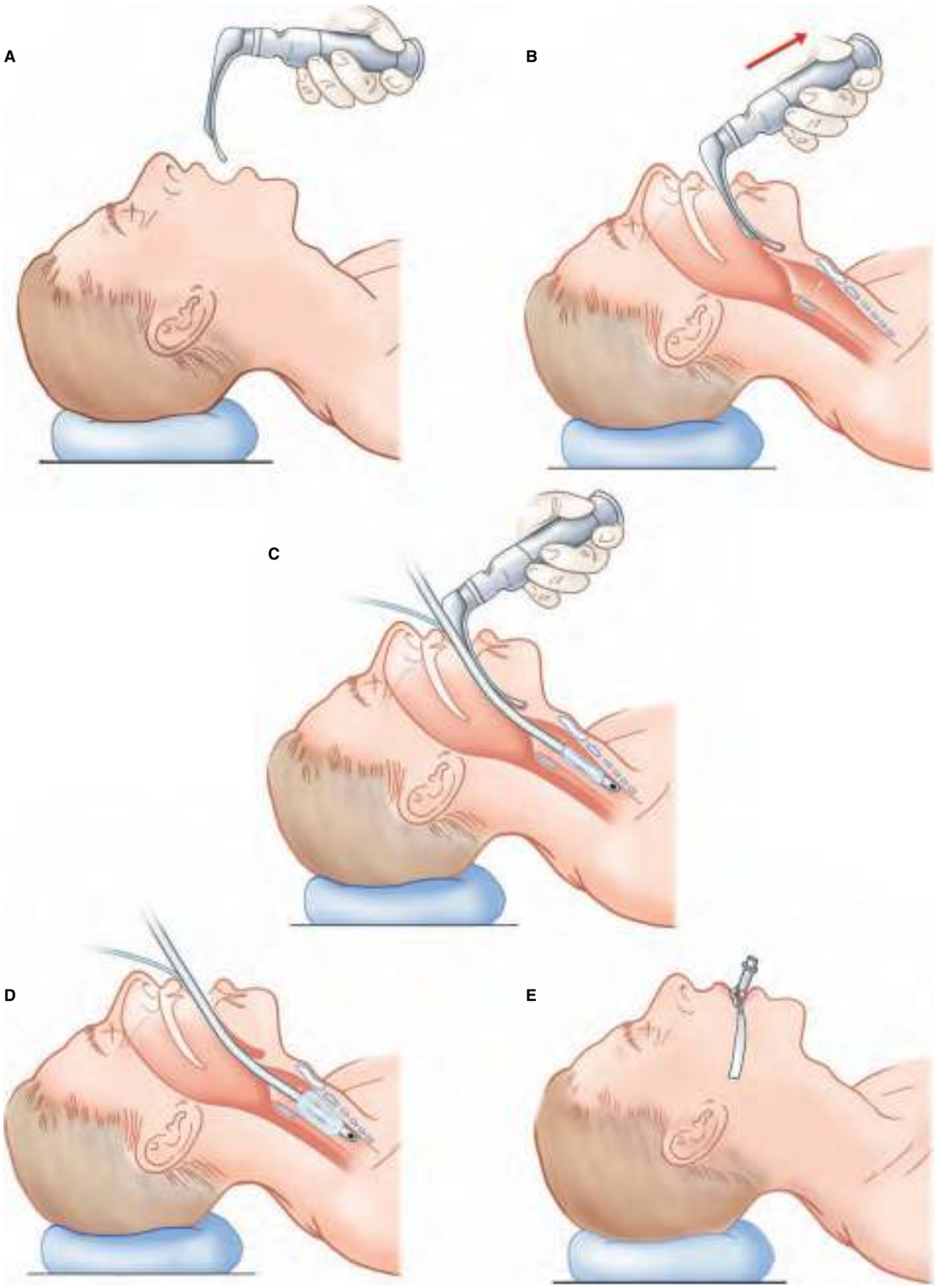


FIGURE 18-17. Orotracheal intubation with the Macintosh blade. The patient is in the "sniffing" position. ***A.*** Proper positioning of the laryngoscope blade above the patient's mouth. ***B.*** The blade is inserted into the vallecula. The handle is lifted anteriorly and inferiorly to elevate the mandible, tongue, and epiglottis (*arrow*) to visualize the glottis. ***C.*** The ET tube is inserted into the trachea until the cuff is below the vocal cords. ***D.*** The laryngoscope has been removed and the cuff inflated. ***E.*** The ET tube is secured.

assistant to keep their hand still while the intubator removes their hand off the assistant's hand. A third variation uses the assistant to manipulate the patient's larynx while the intubator verbally directs them.

When the vocal cords are visualized, instruct the assistant to pass the ET tube into your right hand. This allows you to keep a "visual lock" on the vocal cords. Insert the ET tube into the right side of the patient's mouth. Advance the ET tube so that the tip reaches the vocal cords without letting the body of the tube block the view. Continue to advance the ET tube through the vocal cords until the cuff passes through them and into the trachea (**Figure 18-17*C***). Advance the tube an additional 2 to 3 cm. **The tip and cuff of the ET tube must be visualized passing through the vocal cords to assure placement in the trachea.**

The fit of the ET tube through the vocal cords always seems to be tight, even in larger patients. **A well-lubricated tip with the cuff completely collapsed is essential.** Rolling the tube gently between the thumb and index finger at the moment of insertion can also help pilot the tip between the vocal cords. Ask the assistant to pass the already prepared smaller ET tube if the first ET tube is too large or the vocal cord opening is narrow.

The average depth of insertion (in cm) of the ET tube starting from the patient's lips is listed in **Table 18-1**. It can also be calculated using one of the following formulas: ET tube ID (in mm) × 3, (age in years ÷ 2) + 12 for patients over 2 years of age to a maximum of 18 years of age, or age + 10 for children less than 8 years of age.

Once the ET tube has been inserted, the intubator's right hand must hold the tube in place continuously until it is properly secured. The assistant should inflate the ET tube cuff with the attached 10 mL syringe of air (**Figure 18-17*D***) and then remove the syringe and stylet. **The intubator must hold the ET tube firmly to make sure that it does not become dislodged when the stylet is removed.** The assistant should attach the end-tidal CO_2 monitor and the bag-valve device to the ET tube. Secure the ET tube in position in the right corner of the patient's mouth if symmetrical lung sounds are heard on auscultation and if the pulse oximetry and CO_2 monitoring appear appropriate (**Figure 18-17*E***).

INTUBATING WITH THE (STRAIGHT) MILLER BLADE

Intubating with the straight blade is similar to the curved blade with a few differences. **Insert the laryngoscope blade gradually.** Insert the tip directly under and slightly beyond the epiglottis if it is seen. Lift the epiglottis and airway by raising the left hand along the long axis of the laryngoscope handle toward a point above the patient's chin (**Figure 18-11**). **The tip of the laryngoscope blade is in the esophagus if neither the epiglottis nor the vocal cords are seen.** Locate the airway by lifting as above while slowly withdrawing the laryngoscope blade. As the tip slides back, it will "catch" the epiglottis and the airway should "fall down" into view. The BURP or OELM maneuver may now be applied if necessary. Some physicians use a variation of this to localize the epiglottis by inserting the blade deeply, lifting the soft tissue, withdrawing the laryngoscope blade while feeling for the "give" as the epiglottis tip falls off of the retreating blade, and then readvancing a small distance to "scoop up" the epiglottis and expose the airway. However, when using the Miller blade on pediatric patients, a careful insertion and advancing of the laryngoscope blade is required, while constantly visualizing from the surface of tongue to the vallecula or epiglottis. Some have advocated for the technique of advancing deep into the esophagus and then visualizing laryngeal structure during withdrawal. We contend that this should be avoided because this practice may cause laryngeal trauma with the blade tip damaging the arytenoid cartilage or aryepiglottic fold, as well as increasing the incidence of esophageal intubation.[16] It may be difficult to distinguish the glottic opening from the esophagus in the pediatric patient.

ASSESSMENT

Simple commonsense methods will quickly and accurately assess ET tube placement. The confirmation of ET intubation is briefly described in this section. Please refer to Chapter 19 for a more detailed discussion. **The assessment must be made quickly! An ET tube in the wrong place (i.e., the esophagus) is as quickly dangerous as a properly placed one is lifesaving. Was the ET tube visualized passing through the "A frame" of the vocal cords? This is the most important assessment.** If it was directly visualized being placed and continuously held in place, it is properly positioned. Is the pulse oximeter reading in the high 90s and steady or rising? Is the CO_2 monitoring appropriate?[50] Be familiar with the monitor in your institution. Electronic monitors will show a respiratory waveform and a numerical value.[51] In-line colorimetric devices connected between the ET tube and the respiratory circuit will change color with inspiration and expiration to indicate the flow of CO_2 past the device.[51]

Evaluate the patient. Symmetrical upper chest rise without increasing abdominal size suggests proper placement. Persistent "fogging" or condensation inside the ET tube with each breath for at least six ventilations will also confirm proper placement. Auscultate lateral to the nipples for strong and symmetrical breath sounds during positive-pressure breaths. Avoid auscultating in the midline, where "normal" breath sounds can be heard from a misplaced ET tube in the esophagus. Auscultate at the lateral apices and bases of the lungs. Auscultate over the epigastrium. Correct placement will give strong, symmetrical breath sounds except in the epigastrium. **Assume incorrect ET tube placement if epigastric sounds are strongest, if "gurgling" is heard, or if vocalization is heard.** Breath sounds that are asymmetrical and stronger on the right indicate a right mainstem intubation. Deflate the cuff and gently withdraw the ET tube in 1 cm increments while auscultating. Continue to withdraw the ET tube until equal breath sounds are heard. Secure the tube and reinflate the cuff.

Obtain a chest radiograph after clinically confirming the placement of the ET tube.[52] The tip of the radiopaque stripe of the ET tube should end over the third or fourth thoracic vertebra and 3 to 4 cm above the carina of the trachea. Always inspect the radiograph for any signs of a pneumomediastinum, pneumothorax, or hemothorax.

Clinical assessment of the ET tubes position should take less than 15 seconds. If you are unsure of the ET tube position, leave the first tube in place while applying cricoid pressure. Look to see if the ET tube is passing between the vocal cords if the patient's pulse oximetry reading is in the mid- to high 90s. Alternatively, the ET tube can be removed and intubation reattempted. If the pulse oximetry is low, remove the ET tube and ventilate the patient with a bag-valve-mask device for 30 to 60 seconds to allow the pulse oximetry to rise into the high 90s before making a second attempt at intubation. Some physicians prefer to leave the misplaced ET tube in place.[53] Leaving the first tube might seem to complicate subsequent intubation attempts but can serve to vent gastric vomit out of the oropharynx as well as to locate the esophageal entrance during the next attempt at direct visualization of the airway.[54]

As long as ventilation is possible with the bag-valve-mask device and pulse oximetry can be maintained above 92% (i.e., PO_2 = 60 mmHg), two or three attempts can be made at orotracheal intubation.[55-57] **Stop the intubation attempt and ventilate the patient for 30 to 60 seconds if 30 seconds of time have elapsed or pulse**

oximetry falls to 92% or below. In training programs, the third attempt should be made by the most skilled person available. Three failed attempts define a "failed airway" and call for rescue intubation by an alternative method. **Any patient in whom bag-valve-mask ventilation becomes impossible must be given a surgical airway.**

AFTERCARE

The ET tube must be secured to prevent it from migrating distally or proximally. The traditional method of wrapping tape around the ET tube and then around the patient's head is rarely used today. If used properly, tape functions just as well or better than commercially available ET tube holders.[58] A variety of disposable, relatively inexpensive, and single patient use ET tube holders are commercially available. They are easy to apply, adjust, and reposition if necessary. The basic unit is a nonlatex plastic ET tube holder that positions over the patient's mouth, a cushioned neckband, and Velcro closures for a snug fit. There are many variations of this basic unit.

A nasogastric tube or orogastric tube is often placed after orotracheal intubation to remove air, fluid, and gastric juices from the stomach. This tube must also be secured. It is either taped to the patient's face or to the ET tube to prevent it from migrating distally or proximally. One specific ET tube holder is a novel device that deserves mention. The Intubix (Intubix LLC, Houston, TX) is an ET tube holder with a built-in bite guard and orogastric tube side port (**Figure 18-18**). It allows the blind passage of the orogastric tube through the side port as well as securing it in position.

Postintubation management should include pain control, sedation, and paralysis if indicated. The patient should be sedated and possibly paralyzed depending on the situation, so they do not fight the ET tube and extubate themselves. **The patient should never be paralyzed and not sedated so they are aware and but unable to respond.[59] This is considered cruel.** Administer an opioid analgesic as required for pain control. The patient may require repeated bolus dosing of these medications or a continuous infusion for analgesia, sedation, and/or paralysis depending on their condition.

COMPLICATIONS

HYPOXEMIA, MULTIPLE ATTEMPTS, AND MISPLACED ET TUBES

Hypoxia is the most destructive complication. It often results from prolonged intubation attempts, with or without proper preoxygenation, and unrecognized misplaced ET tubes. Without adequate oxygenation, irreversible brain injury begins to occur within 2 to 3 minutes. Hypoxia can result in cardiac arrhythmias.

Neonates and infants desaturate quicker than adults for many reasons. Their metabolic rate is twice that of an adult. The lungs are a smaller volume and have less alveoli. The neonate and infant minute ventilation is dependent on the rapid respiratory rate. The amount of dead space is greater in the neonate and infant than in the adult. The chest wall is more compliant and easily collapses. Hypoxia of the neonate and infant brain causes bradycardia. Cardiac output is rate dependent.

The complications of orotracheal intubation increase with more than one attempt.[60] Complications were found to increase with over two intubation attempts.[61,62] Some studies confirmed that complications increase when more than one intubation attempt is made.[63-66] Regardless of one or two attempts, the authors agree that complications increase when more attempts at intubation are made. Orotracheal intubation should be accomplished by the most skilled person available.

An unrecognized esophageal intubation will result in significant morbidity and mortality. After intubation, the proper ET tube placement should be confirmed by auscultation, chest rise, fogging in the ET tube, end-tidal CO_2 monitoring, and chest radiography. **Any manipulation or movement of the ET tube or the patient's upper body (i.e., head, neck, and torso) should be followed by an assessment of the ET tube position. It can easily become dislodged and migrate into the hypopharynx and esophagus.** Other methods to confirm ET tube placement include inserting a fiberoptic bronchoscope through the ET tube and visualizing the tracheal rings and carina (Chapter 28) or inserting a

A B

FIGURE 18-18. The Intubix ET tube holder. ***A.*** The unit. ***B.*** The unit with an ET tube and an orogastric tube attached.

lighted stylet and following the illumination into the trachea (Chapter 24).

The cuff of the ET tube can be malpositioned in the airway. Mainstem intubation can be detected on physical examination and chest radiography. The ET tube cuff can be located between the vocal cords. This is visualized on the chest radiograph, and the ET tube is easily advanced. The cuff can be located above the vocal cords with its tip at the vocal cords. The ET tube softens with the patient's body heat over time, and simple advancement might be impeded. The cuff and tip of the ET tube can be above the vocal cords (i.e., a complete extubation). Advancement can result in esophageal intubation.

POSTINTUBATION HYPOTENSION OR HEMODYNAMIC COLLAPSE

Orotracheal intubation may precipitate cardiac arrest, cardiac arrhythmias, or hypotension in patients with comorbidities or underlying heart disease. Cardiac arrest after intubation is associated with a poor outcome.[67] Consider resuscitating the patient before intubation if time allows.[68] Hypotension or cardiac dysrhythmias before intubation can result in postintubation cardiac arrest.[69-73] Interventions for hypotension include intravenous (IV) fluids, vasopressor infusion, press-dose vasopressors, and checking to ensure the patient is oxygenated.

The mnemonic **DOPES** can be used to troubleshoot. It stands for **d**isplacement of the ET tube, **o**bstruction of the ET tube, a **p**neumothorax, **e**quipment failure, and breath **s**tacking. A displaced ET tube may be in the esophagus, right mainstem, or oropharynx. Check the ET tube and reintubate if required. Obstruction is usually due to mucous plugs or biting of the ET tube. This is relieved with suctioning or sedation, respectively. Equipment failure refers to all of the equipment from the wall air sources and to the patient. Use a bag-valve device attached to the ET tube to ventilate the patient while a second person checks and replaces the equipment. Finally, the stacking of breaths can result in gas trapping, high plateau and peak pressures, and incomplete gas return. Disconnect the breathing line and allow the patient to fully exhale.

Bradycardia can be produced by pharyngeal manipulation. It may be especially pronounced in children because of their higher vagal tone. Pretreatment with atropine (0.02 mg/kg with a minimum dose of 0.15 mg) in children under 6 years of age can avoid this. It will also serve to decrease airway secretions.

Increased intracranial pressure can occur as a result of the direct laryngoscopy. The exact cause of this transient rise is unknown. Lidocaine has been postulated as being of benefit in blunting this but is so far unproven. A dose of 1.5 to 2.0 mg/kg IV may be used as a premedication if time allows and the patient's condition warrants its use.

MECHANICAL COMPLICATIONS

Direct mechanical complications from the laryngoscope include lacerations of the lips, trauma to the pharyngeal wall, broken teeth, or dentures that may be aspirated and require later removal.[74] Vomiting can cause subsequent chemical and bacterial pneumonitis. A pneumothorax is a rarely seen complication of laryngoscopy. It is more often associated with positive-pressure ventilation. Laryngoscopy may cause apnea, bronchospasm, and/or laryngospasm due to prolonged stimulation of the pharynx.

Laryngospasm may result from insertion of the laryngoscope blade or attempts to advance the ET tube through the vocal cords.[75] This occurs more often in patients who are awake, semiconscious, not paralyzed, and not anesthetized. It is sometimes preceded by high-pitched inspiratory stridor followed by complete airway obstruction. It can occur without any warning. Laryngospasm should be suspected in an airway obstruction. It is characterized by the inability to manually ventilate the patient, loss of end-tidal CO_2, no breath sounds, no chest wall movement, and no stridor. Laryngospasm can lead to bradycardia, cardiac arrhythmias, end-organ injury, hypoxia, and death.

Laryngospasm may be prevented by the application of nebulized lidocaine, topical anesthetic spray, transtracheal injection of lidocaine, or laryngeal nerve blocks.[76] **Remove the laryngoscope and begin positive-pressure ventilation if laryngospasm occurs during intubation. Positive-pressure ventilation through a face mask will often overcome the laryngospasm.[75] Inward and anterior pressure applied to the mandible at Larson's point or the laryngospasm notch can break the laryngospasm. Consider paralyzing the patient immediately with succinylcholine or performing a surgical airway if the laryngospasm is not relieved.[75]**

The ET tube and stylet can be a source of mechanical complications.[77-80] A sore throat is a common and self-resolving nuisance. Uvular necrosis occurs when the uvula is compressed between the ET tube and palate. This rare complication is self-limited and often heals within 2 weeks. Treatment includes antihistamines, steroids, and possibly antibiotics. The stylet protruding from the distal end of the ET tube can cause soft tissue contusions and lacerations, perforation of the vocal cords, hemorrhage into the airway, and perforation of the trachea.[81] **Proper stylet placement with its tip within the ET tube will prevent these complications.** ET tube cuff overinflation can result in mucosal sluffing, pressure necrosis, and hemorrhage. Tracheal rupture is a rare but life-threatening complication.[79,80] It may be due to cuff overinflation, a protruding stylet, intubation using more rigid and less pliable stylets, pushing through a weakness or defect, or multiple intubation attempts. The complications associated with cuff overinflation can be prevented by using seldom used manometry.

AIR LEAKS

A "leak" of air out from the patient's mouth or nose during ventilation signifies a mechanical problem with the ET tube. The ET tube must be removed and replaced if the cuff is damaged. Check the position of the ET tube by direct laryngoscopy. The ET tube will not secure the airway properly if the cuff is located between or above the vocal cords. Deflate the cuff, advance it through the vocal cords, and reinflate the cuff. There may be a leak in the pilot balloon or its tubing if the cuff slowly deflates. Reinflate the cuff and apply a hemostat to the tubing attached to the pilot balloon or attach a closed stopcock to the inflation port.

ASPIRATION

The risk of aspiration increases in patients with difficult airways or full stomachs. This includes obese and pregnant patients. The use of an awake ET intubation or rapid sequence intubation with cricoid pressure may minimize the risk of aspiration. **A properly placed ET tube with the cuff inflated will decrease, but not totally eliminate, the risk of aspiration.**

THE UTILITY OF SELLICK'S MANEUVER

The use of cricoid pressure was introduced in 1774 to prevent gastric distention during artificial ventilation of drowning victims.[82] Sellick reintroduced the application of cricoid pressure in 1961.[83] Cricoid pressure theoretically causes an occlusion of the upper esophagus by trapping it between the cricoid cartilage and the vertebral bodies. It has since been used on a daily basis for rapid sequence intubation by Anesthesiologists and Emergency Physicians. This maneuver

was thought to prevent regurgitation and gastric insufflation during positive-pressure ventilation.[84,85] The application of cricoid pressure was later termed "Sellick's maneuver."

Sellick's original publications were small observational studies self-reporting on his technique. These studies were not blinded, controlled, or randomized. Several subsequent studies determined the amount of force required to occlude the esophagus on cadavers and patients. It is not practical or possible to measure the cricoid force applied during clinical care in the Emergency Department.

Multiple studies have found adverse effects associated with Sellick's maneuver.[47,84,86-97] It can worsen the laryngoscopic view of the airway. Other complications include bruising, airway obstruction, cricoid cartilage fracture, goiter hemorrhage, subconjunctival hemorrhage, and esophageal rupture.[95] Cricoid pressure decreases lower esophageal sphincter tone and may explain in part the cases of pulmonary aspiration prior to intubation.

Sellick's maneuver is considered by some to be the standard of care.[98] Very little evidence supports its use to prevent aspiration.[88-90] No studies have shown that the application of Sellick's maneuver improves patient outcomes.[85,99] Even Anesthesiologists do not agree on its use.[100] **This does not mean Sellick's maneuver should not be performed!** It may be of benefit to prevent aspiration, especially in the Emergency Department where patients are considered to have "full stomachs." Slowly release cricoid pressure in an attempt to improve ventilation or airway visualization if intubation or ventilation is difficult using Sellick's maneuver.[101]

ENDOTRACHEAL CUFF PRESSURE

Inflation of the ET tube cuff is required to adequately ventilate a patient without an air leak, provide positive-pressure ventilation, and prevent aspiration. There can be a fine line between proper cuff inflation and overinflation.[102,103] One of the goals of any procedure is to prevent complications from the procedure itself. Underinflation of the ET tube cuff can result in aspiration, extubation, and inadequate ventilation. Overinflation of the ET tube cuff can result in bloody phlegm, cough, dysphagia, hemorrhage, hoarseness, mucosal necrosis, mucosal sloughing, nerve palsy, subsequent tracheal stenosis, tracheoesophageal fistula, tracheal rupture, tracheal wall damage, and tracheitis. **Physicians have traditionally palpated the pilot balloon to estimate cuff pressure and prevent any complications from overinflation. This method is not accurate.**[104,105]

Measurement of the cuff pressure is simple, quick, and inexpensive. It should be considered whenever a patient is intubated.[102,106-108] Cuff pressure should be measured if an intubated patient is moved.[108,109] **Be careful because connecting a manometer can lower the cuff pressure.**[110] Cuff pressure should be measured if a patient is transported by air because the cuff pressure changes at elevated altitudes.[108,111-114] A pressure of up to 30 cmH_2O provides an adequate seal without compromising mucosal blood flow and lowers the risk of subsequent subglottic stenosis.[115] Tracheal mucosal blood flow shows a decline at 30 cmH_2O, and is completely blocked at 45 to 50 cmH_2O.[116] An ET tube cuff pressure manometer is needed to measure these pressures accurately.

There are numerous methods to quickly assess cuff pressure. **Palpation is not accurate and should not be used**. The simplest method is to use a manometer. These devices (Cufflator, Posey Corp., Pasadena, CA, or Rusch Endotest, Teleflex Medical, Research Triangle Park, NC) allow the simultaneous inflation of the cuff while monitoring the cuff pressure on a dial (**Figures 18-19*A* and *B***). These devices function similarly to those used to inflate a car tire. While simple to use, they can be cumbersome due to their shape and weight. An in-line device that attached to the cuff inflation port is available (PressureEasy Cuff Pressure Controller, Smiths-Medical, Dublin, OH). The device has an indicator window that signals when the cuff pressure is maintained between 20 and 30 cmH_2O (**Figure 18-19*C***). A third novel device is the Pressure Alert Endotracheal Tube. This device is not yet commercially available. It incorporates a "pop up" button into the pilot balloon that alerts the user when the cuff pressure is too high. Automated cuff pressure devices are available. These include the Pressure Eyes (formerly known as the Pyton, Bay State Anesthesia, Bristol, CT), the IntelliCuff (Hamilton Medical, Reno, NV), and the Cuff Sentry (Outcome Solutions, Mocksville, NC) (**Figures 18-19*D* through *F***).

A

B

FIGURE 18-19. ET tube cuff pressure measurement devices. ***A.*** The Posey Cufflator. ***B.*** The Rusch Endotest. ***C.*** The PressureEasy Cuff Pressure Controller. (Photo courtesy of Smiths-Medical.) ***D.*** The Pressure Eyes. (Photo courtesy of Bay State Anesthesia.) ***E.*** The Intellicuff. (Photo courtesy of Hamilton Medical.) ***F.*** The Cuff Sentry. (Photo courtesy of Outcome Solutions.)

C

D

E

F

FIGURE 18-19. (*Continued*)

SUMMARY

Orotracheal intubation is both common and lifesaving. It is the primary and preferred method of airway management. Every Emergency Physician must master this skill. With proper preparation, definitive control of the airway can be obtained. This assures that patients can be oxygenated and ventilated when they cannot do this on their own. Good team leadership skills are nearly as important as physical dexterity and assure an orderly and quick procedure. Rapid patient assessment is important to prevent complications. If orotracheal intubation is unsuccessful, another form of intubation or a surgical airway should be performed.

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19 Confirmation of Endotracheal Intubation

Tarlan Hedayati

INTRODUCTION

This chapter will review the various methods used to confirm appropriate endotracheal (ET) intubation. **Direct visualization of the ET tube passing through the vocal cords is the preferred method for the initial assessment of a properly placed airway.** This is not always feasible. Rates for incorrect ET tube placement have been noted to be up to 25%.[1,2] **The verification of correct ET tube placement is as important or more important than the intubation procedure. Lack of proper confirmation of ET tube placement has the potential for serious patient harm and catastrophic outcomes if unrecognized and uncorrected.**[3-5]

For over 20 years, there has been ongoing research and development to improve upon the basic techniques of physical examination confirmation of ET intubation. Physical examination with auscultation has been found to be inadequately sensitive (94%) and specific (83%) as an independent method for confirmation of correct ET tube placement.[6]

This chapter discusses the use of physical examination findings, esophageal detection devices (e.g., syringe and bulb), carbon dioxide (CO_2) detection devices (i.e., qualitative detector and continuous quantitative monitor), and imaging techniques (i.e., radiography and ultrasound). Each method is described for a patient with normal anatomy and the absence of any pathology (e.g., chest or neck trauma). Certain patient conditions or pathology may affect the accuracy of some methods. **No single method is universally or completely reliable. The confirmation of ET intubation requires a multiple method approach**. This multiple method approach to confirmation of ET intubation is now the accepted practice according to the American College of Emergency Physicians (ACEP) Board of Directors policy statement as of January 2016.[7]

PHYSICAL EXAMINATION

The use of physical examination methods has been the mainstay for the initial evaluation of proper ET tube placement. Direct visualization of the ET tube insertion through the vocal cords and into the trachea is the first method to confirm proper ET tube placement. Direct postintubation visualization of the ET tube using laryngoscopy or bronchoscopy and noting tracheal rings past the end of the ET tube is the next best method of assessing correct ET tube placement.

Secondary methods for confirmation of ET intubation are an absolute requirement. Auscultate the chest and abdomen to assess for delivery of air to the lungs via either a bag-valve device or a mechanical ventilator (**Figure 19-1**). First auscultate over the epigastrium to assess for the absence or presence of sounds in the stomach. **The presence of an enlarging abdomen or audible air inflation into the stomach with each positive-pressure ventilation may be the initial sign of an esophageal intubation.** The next auscultation points are located at the chest wall lateral to the nipples. Auscultate bilaterally from top to bottom for the presence and equality of breath sounds. **Avoid auscultating over the central portion of the chest. This may lead to the misinterpretation of the transmission of esophageal or gastric inflation.[8] These sounds may mimic breath sounds and lead to failure in detecting an esophageal intubation.**

The final note pertaining to auscultation involves equality of breath sounds. The anatomy of the left and right mainstem bronchi allows for a preferential right mainstem bronchial intubation when the ET tube is placed too deep. Insertion in centimeters to a depth three times the diameter size of the ET tube (e.g., 21 cm for a 7.0 mm size ET tube) at the level of the incisor teeth generally is preferred. This will place the ET tube approximately 3 to 4 cm above the carina in an adult.[9] Deflate the ET tube cuff if breath sounds are heard better on the right side and gradually withdraw the ET tube in 1 cm increments until equal bilateral breath sounds are auscultated. This will prevent complications from hyperinflation of the ventilated lung, inadequate ventilation and oxygenation, and pulmonary edema in the nonventilated lung.[10] Please refer to Chapter 18 for a more complete discussion of ET tube size, insertion depth, and placement for adults and children.

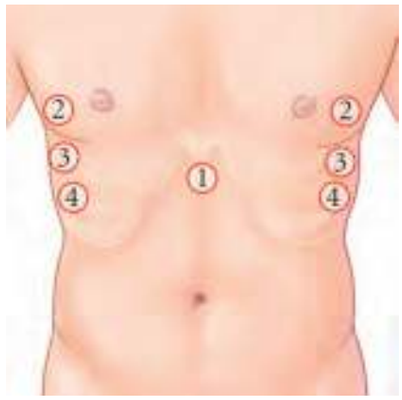


FIGURE 19-1. The order of auscultation after intubation to confirm ET tube placement begins at the epigastrium (1) listening for absence of air sounds, then down both sides of the chest wall, just lateral to the nipple line (2–4).

The next assessment involves visualizing the chest wall and ET tube characteristics with each breath. **Unequal or a lack of chest rise and fall with each instilled breath may indicate a misplaced ET tube in a mainstem bronchus or the esophagus, respectively.** Condensation and/or fogging in the ET tube with each breath has been used frequently in the past to confirm proper ET tube placement. This has been proven to be an unreliable source of confirmation.[11] **Use fogging in the ET tube in conjunction with auscultation and visualization of bilateral chest wall movement.** The presence of vomit in the ET tube may be a sign of aspiration or esophageal intubation.

The final postintubation physical assessment is an evaluation of oxygenation via skin signs and, more reliably, pulse oximetry. Cyanosis and a down-trending oxygen saturation on the pulse oximeter are delayed findings. The patient may experience significant hypoxemia before cyanosis or a significant drop in the pulse oximetry appears. **Do not rely upon cyanosis and pulse oximetry as a first-line assessment to confirm proper ET tube placement.**

ESOPHAGEAL DETECTOR DEVICES

Esophageal manometer devices use the structural differences between the trachea and esophagus in determining correct ET tube placement. The semicircular tracheal cartilaginous rings provide a constant open passage for the flow of air. The tubular esophagus with its lack of a luminal support structure collapses and prevents movement of air when suction is applied.[12] The two types of ET tube confirmation devices that rely on this anatomic variance are the syringe and the manometer or bulb methods (**Figures 19-2 and 19-3**).

The syringe method relies on a gradual and constant retraction of the plunger while its tip is attached to the proximal end of

FIGURE 19-2. Examples of the bulb and syringe esophageal detector device. (Photo courtesy of Wolf Tory Medical, Salt Lake City, UT.)

the ET tube using a manufactured connector (**Figure 19-3**).[13] The plunger will draw back easily and without resistance if the ET tube is correctly placed in the trachea (**Figure 19-3*A***). The volume of aspirated air should be greater than 30 mL in less than 4 seconds.[14] Resistance when drawing back the plunger is seen when the distal end of the ET tube is incorrectly placed in the esophagus (**Figure 19-3*B***). **An airtight seal must be created between the syringe and ET tube to prevent any air leak that would lead to a false-positive test and result in the incorrect assumption that the ET tube is in the trachea.**

The manometer or bulb method relies on the collapse of the esophageal wall. Compress the bulb to remove any air it contains. Firmly and securely apply the bulb to the proximal end of the ET tube while still compressing the bulb. Release compression on the bulb. The ET tube is presumably in the trachea if the bulb inflates fully and easily. The ET tube is presumably in the esophagus if the bulb does not inflate fully and easily.

There are numerous circumstances in which an esophageal detector device has repeatedly failed to identify an improperly placed ET tube.[12-17] Most notably is after bag-valve-mask ventilations in the absence of cricoid pressure. The stomach and esophagus can inflate with air and allow for easy flow of air into the device simulating air flow from the airway. The device may fail to detect a misplaced ET tube when the distal end of the ET tube is just above the vocal cords. There is no resistance to airflow when the distal end of the ET tube is above the vocal cords and the bulb or syringe device easily fills with air.

This method has been shown to falsely identify an esophageal intubation when the ET tube is placed into a mainstem bronchus or the tip of the ET tube is pressed against the tracheal wall. The subsequent resistance to back flow of air into the syringe or bulb mimics that of the collapsed wall of the esophagus. Withdraw the ET tube 1 cm and attempt using the esophageal detector device again if this occurs. Repositioning will continue to result in resistance to aspiration or bulb inflation if the ET tube is in the esophagus. The presence of heavy or thick pulmonary secretions or fluid in the lungs can plug the airways and reduce the flow of air into the syringe or bulb.[15] Use care not to rapidly aspirate and potentially draw the tracheal mucosa into the ET tube yielding a false-negative result due to obstruction.[14] The posterior trachea lacks cartilage and may collapse into the trachea in obese patients and prevent the flow of air.[16-19]

Consider the limitations of esophageal detection devices and use them as an adjunct to other methods as they can provide a rapid and

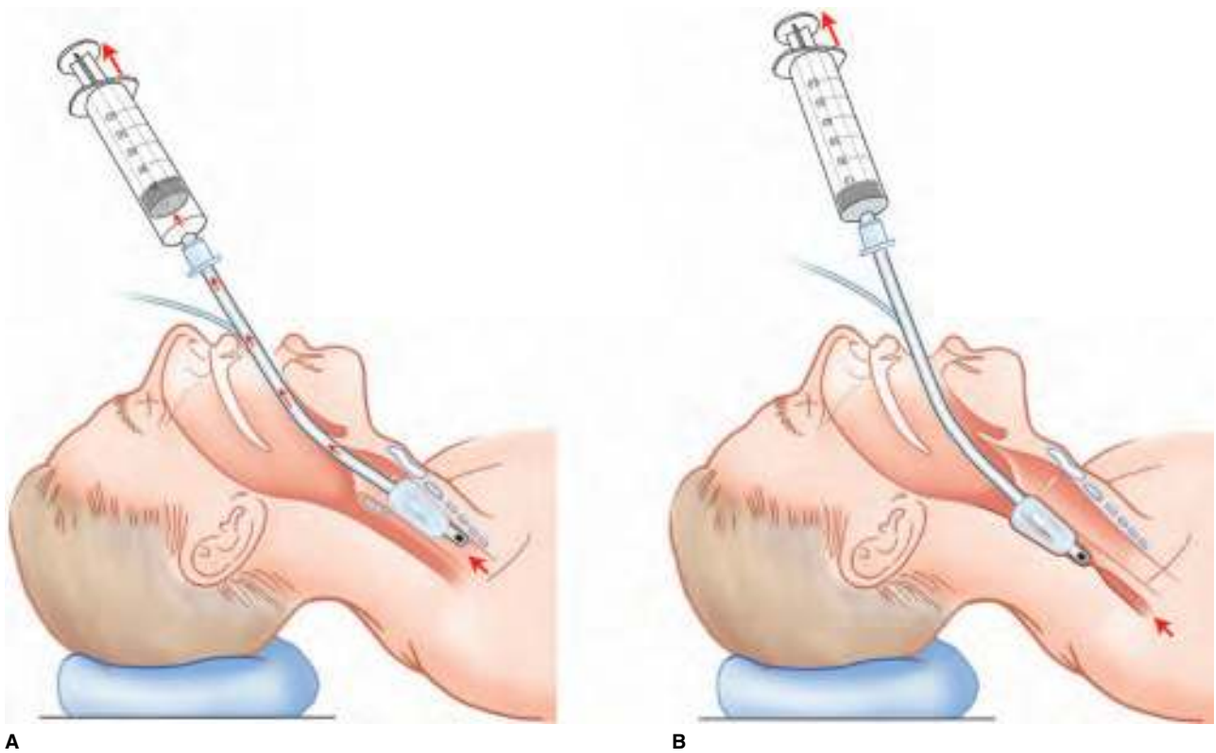


FIGURE 19-3. Using the syringe esophageal detector device. ***A.*** Correct placement of the ET tube in the trachea allows for aspiration of air. ***B.*** Placement of the ET tube in the esophagus will result in resistance to aspiration due to collapse of the esophageal wall.

inexpensive method for confirming ET tube placement.[20-22] This is especially evident in the patient with cardiac arrest, circulatory collapse, hypotension, or a pulmonary embolism where other devices are limited.[21]

CARBON DIOXIDE DETECTORS AND CAPNOGRAPHY

End-tidal carbon dioxide ($ETCO_2$) detection as a means for confirming proper ET intubation has been studied and recently confirmed to be the "most accurate" method.[23] The premise of $ETCO_2$ detection devices requires the presence of exhaled CO_2 passing through and exiting the ET tube. Adequate circulation is required for CO_2 to be produced and transported from the lung parenchyma. The accuracy of this method relies on its use in the noncirculatory collapsed and noncardiac arrest patient. $ETCO_2$ detection using qualitative or quantitative methods approaches 100% sensitivity and specificity in the patient with an inflated cuffed ET tube and spontaneous circulation. Nearly every case of a false-negative detection of a correct ET tube placement (i.e., correct ET tube placement in the absence of $ETCO_2$ detection) has been discovered in the cardiac arrest patient.[18,19,24-28]

$ETCO_2$ detection devices can be used qualitatively as a one-time spot check using a colorimetric detector or monitored continuously via capnography. Both are used in a similar fashion and are in-line devices that have two connectors. One connector attaches to the proximal end of the ET tube and the other attaches to the oxygen delivery device (i.e., bag-valve device or mechanical ventilator tubing).

The colorimetric $ETCO_2$ detector uses a piece of pH-sensitive material that lies beneath a clear plastic window. The presence of CO_2 flowing through the device, typically using six manual breaths as the maximum number needed and the minimum amount to clear any gastric CO_2, causes a color change from purple to yellow that then changes back to purple in the absence of CO_2 (**Figure 19-4**). The minimum concentration of $ETCO_2$ required for a color change is 0.5%. Literature has brought into question whether newborns and children produce sufficient quantities of $ETCO_2$ for accurate detection using this method.

Capnography, or continuous quantitative graphical demonstration of $ETCO_2$ detection, involves placement of an infrared detector in-line between the end of the ET tube and the oxygen delivery device (i.e., bag-valve device or ventilator tubing). The infrared detector is then connected with a cable to an electronic monitor that interprets the reading and generates a waveform representation of the $ETCO_2$ levels. One of the advantages of this method over the qualitative approach is the ongoing monitoring of CO_2 production.

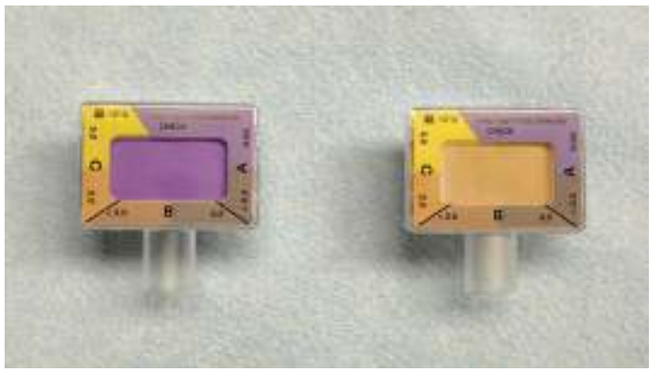


FIGURE 19-4. Colorimetric $ETCO_2$ detectors before (left) and after (right) exposure to exhaled CO_2. Note the change in color of the pH paper from purple to yellow when exposed to CO_2.

This allows a continuous assessment of the airway and ventilation as well as the overall quality of resuscitative efforts with some determination of outcome.[6] Capnography is recommended to be used on all patients for confirming ET intubation and monitoring ventilation.[29-32]

The main disadvantage of CO_2 detectors for confirmation of ET intubation is false-negative results (e.g., airflow obstruction, cardiopulmonary arrests, hypotension and other poor perfusion states, low-amplitude waveforms with low CO_2 emission, the presence of secretions on the device, and pulmonary embolism). Sensitivities approach 100% in the non–cardiac arrest patient with adequate cardiac output and pulmonary flow and 76% for patients in cardiac arrest for ruling out esophageal intubation in patients of all ages.[33-35]

A false-positive result may lead the Emergency Physician to believe the airway is secure if the distal end of the ET tube is located just above the vocal cords in the hypopharynx. The device may detect adequate levels of CO_2 without a properly secured and definitive airway in the trachea. False-positive results from an improperly placed ET tube may occur when the device is used a short time after the patient has consumed a carbonated beverage. The device cannot differentiate between gastric and pulmonary CO_2 sources.

The universal presence of capnography is not yet a reality. Its utility in most circumstances with few exceptions is undeniable. The colorimetric $ETCO_2$ detector is as equally reliable as capnography. It is a disposable, rapid, less expensive, and more widely available means to assess proper ET tube placement in the non–cardiac arrest patient.[23]

RADIOGRAPHY

The lack of availability of radiographs or ultrasonography is far less of an issue.[36] These methods are unique in that they rely on anatomic relationships for confirmation of ET tube placement. Radiographs and ultrasonography may be particularly useful in the cardiac arrest patient.

The main utility for radiographic confirmation of ET intubation lies in its ability to detect whether the ET tube is placed too deeply into the trachea (**Figure 19-5**).[37] Locating the end of the ET tube approximately 4 cm above the carina may prevent some of the complications associated with a right mainstem intubation. The postintubation chest radiograph can assist in identifying any complications that may have resulted during the intubation (e.g., aspiration, a pneumomediastinum, a pneumothorax, or tracheal injuries). **The postintubation chest radiograph does little to distinguish between esophageal versus tracheal intubation. The delay in obtaining a radiograph to confirm ET tube placement puts the patient at significant risk.**

ULTRASONOGRAPHY

The use of ultrasound (US) in the Emergency Department has gained significant popularity in the diagnosis of various diseases and pathology. US is readily available, can be used at the bedside, and uses no ionizing radiation. US can identify conditions before and after intubation (e.g., pneumothorax or tracheal injury). It can be used during intubation to see the ET passing the vocal cords. US is used after intubation to confirm the presence of the ET tube in the trachea and its lack of residing in the right mainstem bronchus.

There are multiple methods for using US guidance to assess for proper ET tube placement.[38-47] Practice in advance of needing US to become familiar with the US anatomy of the airway (**Figure 19-6**).[48] **Table 19-1** notes some of the definitions related to airway US and the confirmation of intubation. The trachea is an air-mucosal interface with posterior reverberation or comet tail artifact (**Figure 19-6*D***).

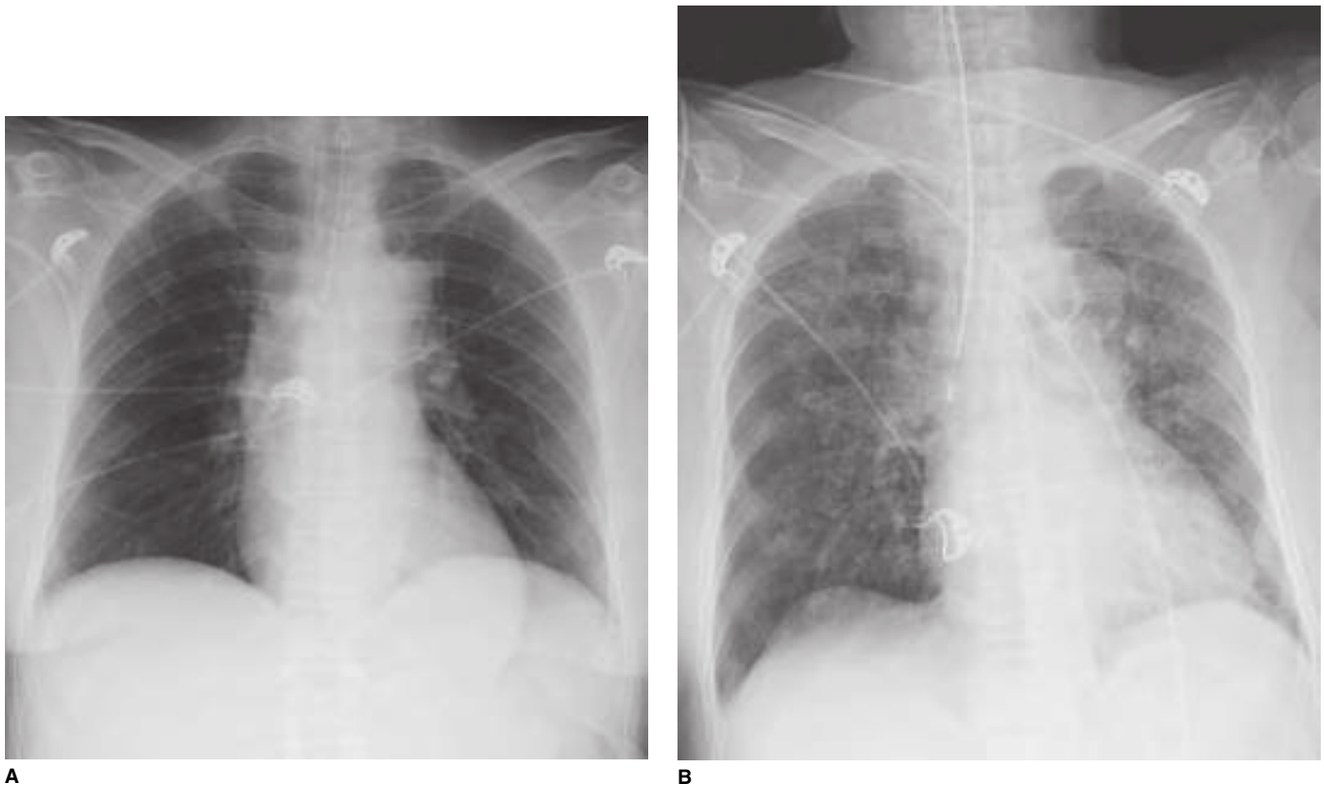


FIGURE 19-5. Chest radiographs after intubation. ***A.*** The ET tube is in the trachea and above the carina. ***B.*** The ET tube is in the right mainstem bronchus.

Two common methods of confirming proper ET tube placement are described below. The benefits of these two methods is that ventilation is not required for ET tube placement confirmation. There is no risk of inflating the stomach or its resultant emesis and aspiration of gastric contents in the case of an accidental esophageal intubation. A meta-analysis of transtracheal US accuracy in confirmation of ET intubation had a pooled sensitivity and specificity of 98%.[49]

The first method uses a 7 to 10 MHz, high-frequency linear US transducer. Place the US transducer horizontally at the level of the suprasternal notch (**Figure 19-7**). A properly placed ET tube in the trachea will demonstrate shadowing of the ET tube posteriorly (i.e., the "bullet sign") without direct visualization of the esophagus (**Figures 19-8*A*, 19-9, and 19-10*A***). The trachea appears similarly to the previously described image, but the esophagus will be directly visualized to the left of the trachea (i.e., the "double track

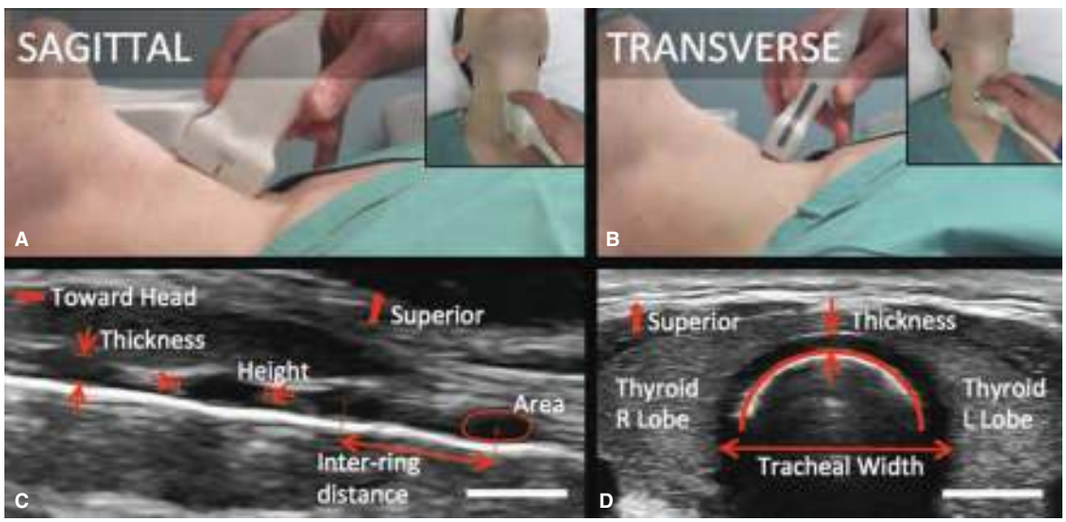


FIGURE 19-6. US of the trachea using a linear transducer. ***A.*** Positioning of the transducer vertically. ***B.*** The labeled US image visualized in part A. ***C.*** Positioning of the transducer horizontally. ***D.*** The labeled US image visualized in part C. (Used with permission from reference 48.)

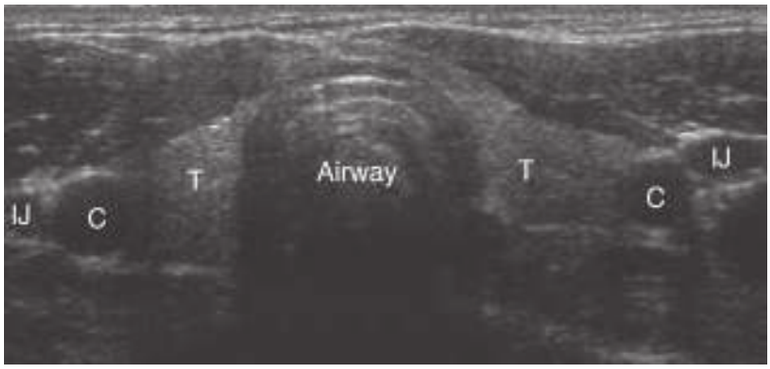
TABLE 19-1 Definitions of Terms Used When US Is Performed for Intubation

Terms	Definition
Bullet Sign	A properly placed ET tube in the trachea will demonstrate shadowing of the ET tube posteriorly
Double-tract sign	An esophageal intubation is considered if there are two air-mucosal interfaces visible, both having comet tail artifacts
Enhanced tracheal posterior shadow	An ET is considered if there is one air-mucosal interface visible with comet tail artifacts and enhanced posterior shadowing
Lung sliding sign	Pleural movement of the parietal and visceral pleura on chest US
Snowstorm sign	US image through the cricothyroid membrane with correct ET tube positioning
Triangular sign	Change in vocal cord shape when the ET tube passes them

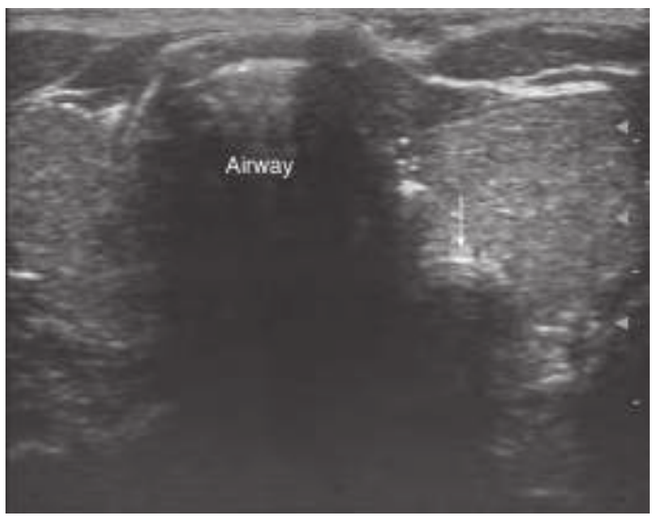
FIGURE 19-7. US transducer placement horizontally just above the sternal notch.

sign") with posterior shadowing due to the presence of the ET tube in the esophagus (**Figures 19-8*B* and 19-10*B***). A small, prospective, randomized, controlled study found 100% sensitivity and 100% specificity in the accuracy of US as a method to confirm ET tube placement.[50]

The second technique uses similar methodology over the cricothyroid membrane. Place the US transducer longitudinally over the cricothyroid membrane to identify the hyperechoic anterior and posterior laryngeal walls (**Figure 19-11**). The appearance of



A



B

FIGURE 19-8. Ultrasound images at the level of the sternal notch to confirm proper ET tube placement. ***A.*** Endotracheal intubation. The signature double echo of the plastic ET tube can be seen in the airway or trachea. (C, carotid artery, IJ, internal jugular vein, T, thyroid gland.) ***B.*** Esophageal intubation. The ET tube (*arrow*) is in the esophagus. It is positioned deep and lateral to the airway. (Ultrasound images courtesy of Sam Hsu, MD.)

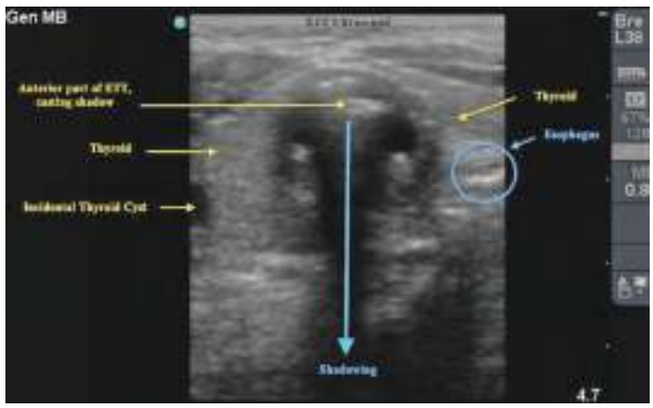


FIGURE 19-9. US image of an ET intubation with the transducer placed horizontally just above the sternal notch. (Photo courtesy of Ultrasound Section, Emory University Department of Emergency Medicine.)

FIGURE 19-11. US transducer placement longitudinal or vertically over the cricothyroid membrane.

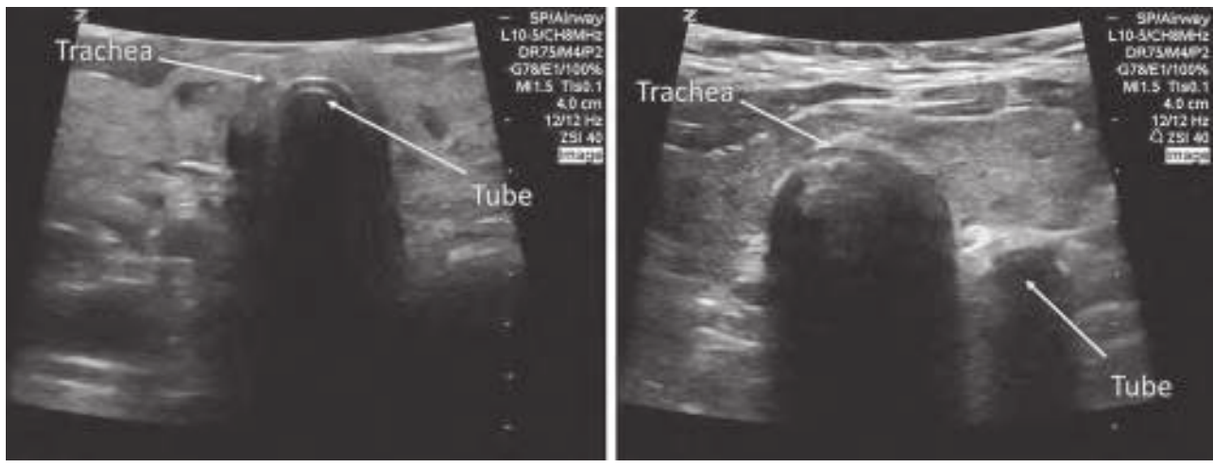
a "snowstorm" pattern between the two lines indicates correct ET tube placement (**Figure 19-12**).[51]

Foreign bodies are often hyperechoic and appear bright white on US. One study looked at using US to confirm ET tube placement with and without a stylet within the ET tube.[52] The investigators hypothesized that the addition of a stylet would increase the sensitivity and specificity of identifying correct ET tube placement by adding an extra hyperechoic shadow to those of the ET tube. The use of a stylet did not improve US localization of the ET tube within the trachea.[52]

US of the chest wall has been studied as a method of confirming ET tube placement.[53-55] This method does require ventilation of the patient and uses the visceral-parietal pleural interface of the lungs to confirm inflation of the lung. Apply the US probe to the anterior-superior chest wall. Orient the US transducer vertically over the second and third ribs in the midclavicular line. The visceral and parietal pleural interface can be seen and appreciated as the so-called "sliding lung sign" (**Figure 19-13**). The hyperechoic interface will be seen moving back and forth with each respiration.[53-55] Lung sliding can be used with other methods to confirm ET intubation.[56]

US can be used to see the ET tube pass through the vocal cords (**Figure 19-14**).[56] Place the US transducer transversely and visualize the vocal cords (**Figure 19-14*A***). View the triangular vocal

FIGURE 19-12. US image through the cricothyroid membrane demonstrating the snowstorm associated with correct ET intubation. The posterior laryngeal wall (*large arrow*) and the anterior laryngeal wall (*small arrow*) are visible. (Used with permission from reference 51.)



A **B**

FIGURE 19-10. US images of ET intubation (left) and esophageal intubation (right). (Photo courtesy of Jordan Chenkin, MD.)

FIGURE 19-13. Ultrasound through the chest wall demonstrating the bright interface (*solid arrows*) of the visceral and parietal pleura generating the "sliding lung sign" during ventilation. The *dashed arrows* demonstrate echogenic bands moving side-to-side with ventilations. The asterisks (*) identify the ribs. (Ultrasound image courtesy of Sam Hsu, MD.)

cords turn to a curved shape with the ET tube in the trachea (**Figure 19-14*B***). This method is hard to perform as the vocal cords are thin and difficult to visualize using US.

TRACHEAL TUBE INTRODUCERS

The tracheal tube introducer (TTI) or gum elastic bougie has long been used to intubate in difficult conditions (Chapter 22). The TTI is a flexible rod-like device whose distal end is slightly angled. It is inserted through the patient's vocal cords with the angled tip facing anteriorly during direct laryngoscopy. It is then advanced into the trachea. Advancement of the TTI causes the tip to slide along the anterior tracheal cartilage rings. A "click" is palpable as the tip crosses each tracheal ring. The TTI will eventually stop or "hang up" as it passes into smaller bronchi. The ET tube is then advanced over the TTI and into the trachea.

Intubate the patient. Insert the lubricated TTI into the ET tube with the angled tip facing anteriorly. Advance the TTI through the ET tube. The tip will catch on each tracheal ring and "click" to verify proper ET tube position. The lack of any "clicks" or "hang up" suggests an esophageal intubation. This method is not 100% perfect like all other methods of confirming ET intubation.[57] Use the TTI in conjunction with other methods to confirm intubation.

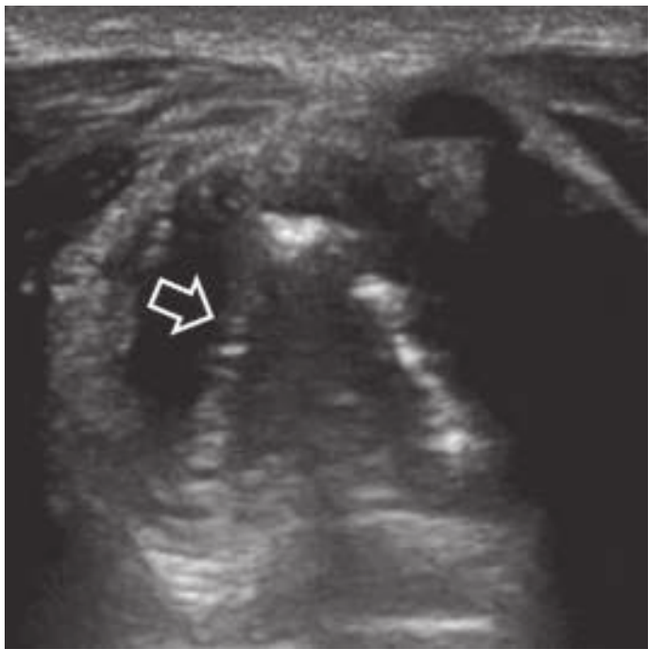
DOCUMENTATION

Physicians are poor at documentation of ET tube confirmation.[5] **Confirmation of the ET tube in the airway is critical.** This should be documented in the medical record. Document the location of the ET tube using positive or negative results and the corrective actions if taken. The lack of documentation can be the result of just not being written down in the busy Emergency Department, or it may not have been performed. This can be and is incorporated into some templates.

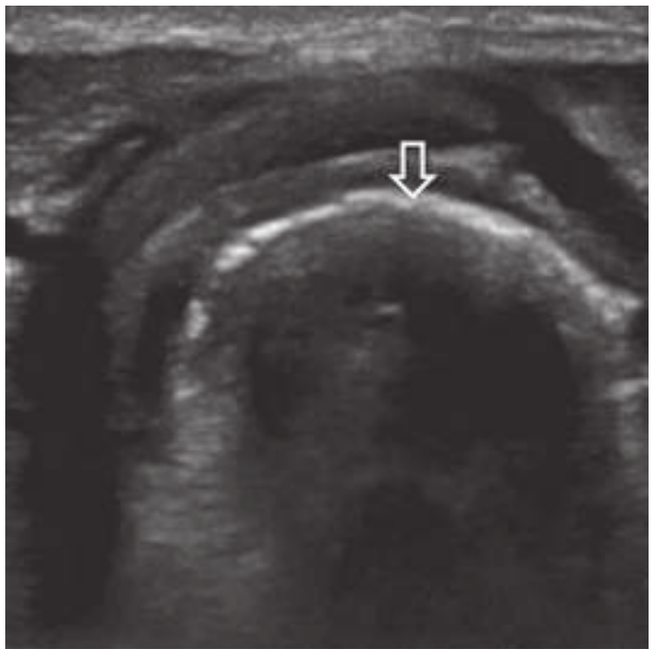
The documentation serves many purposes. The primary reason it is done is to document the medical care received and any decisions made. It provides information to consultants and others involved with the care of the patient. It is used for quality assurance and continuous quality improvement. The documentation is later used for billing and in medical malpractice cases to provide defenses.

SUMMARY

Insertion of an ET tube always requires verification of its proper placement. No one tool or technique is sufficient for any or all situations. The best method to confirm proper ET tube placement involves using multiple methods, keeping limitations in mind, and performing ongoing repeat assessments. Any change in a patient's



A



B

FIGURE 19-14. US images through the cricothyroid membrane. ***A.*** The vocal cords are triangular shaped without the ET tube. ***B.*** The vocal cords are round with the correct positioning of the ET tube. (Used with permission from reference 56.)

clinical condition requires reverification that the ET tube is still properly positioned. The ongoing research and development of improved methods note the critical importance of rapid and accurate confirmation of ET intubation.

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20 Video Assisted Orotracheal Intubation Devices

Pholaphat Charles Inboriboon and Curtis Blake Buchanan

INTRODUCTION

Direct laryngoscopy or the direct laryngoscope (both referred to as DL) was introduced in 1895 by Alfred Kirstein. Physicians have since developed instruments to improve visualization of the larynx while limiting tissue trauma. The Macintosh and Miller laryngoscope blades were developed in the 1940s and have been the primary tool for endotracheal (ET) intubation.[1] They have been effectively used for most ET intubations. There are limitations to their ability to allow direct visualization of the glottis and surrounding structures. Numerous adjuncts have been developed to assist in ET intubation.[1]

The development of video laryngoscopy or the video laryngoscope (both referred to as VL) marks a new era in airway management (**Tables 20-1 and 20-2**).[2-4] Traditional DL requires alignment of the oral, pharyngeal, and laryngeal axes to visualize the glottis (Chapter 9). It is not always possible to align these three axes with mechanical manipulation. **The major advantage of VL is that it does not require the Emergency Physician to align the three airway axes, reducing the need for manipulation and potential traumatic forces on the airway.[5] VL provides a superior view of the glottis when compared to traditional DL.** The eye of the VL camera is within centimeters of the glottis and provides a wider angle of vision than the 15° of traditional DL. The video monitor magnifies the view of the airway, making structures easier to visualize. The American Society of Anesthesiologists recommends having VL available as a tool for their Difficult Airway Algorithm.[6] This chapter reviews a representative number of device types currently available and used in Emergency Departments (**Table 20-3**). A recent study reviews commonly used VLs (**Table 20-4**).[7,8]

PENTAX AIRWAY SCOPE

The Pentax Airway Scope (Pentax Medical Co., Montvale, NJ), known as the Pentax AWS or the AWS, is a rigid VL (**Figure 20-1**). It incorporates the blade, camera, and an ET tube targeting mechanism into one device. It is designed for prehospital, Emergency Department, and Operating Room use.[9,10]

TABLE 20-1 The Advantages of Video Laryngoscopy

Alignment of oral, pharyngeal, and laryngeal axes not needed
Appropriate for prehospital, Emergency Department, and in-hospital use
Can be viewed by others
Cricoid pressure not needed
Decreased dental trauma with less lifting
Decreased esophageal intubation
Decreased use of bougies
Decreased use of external manipulation
High resolution
Improved first-pass success
Improved laryngeal view (Cormack-Lehane grades 1 and 2)
Less soft tissue lifting compared to DL
Minimal image distortion
Minimal cervical spine movement
Rescue device after failed DL
Sniffing position not needed
Teach airway anatomy
Teach DL
Use in known or suspected difficult airways
Use in morbidly obese
Use in "routine" intubation
VL-guided ET tube exchange

INDICATIONS AND CONTRAINDICATIONS

The AWS can be used for elective and emergent intubations. Patients with their head and neck immobilized can benefit from intubation with this device. It provides better visualization of the glottis, decreased cervical movement, and a higher success rate compared with traditional DL.[11,12] Intubation with the AWS produces less airway stimulation than either the Glidescope or traditional laryngoscopes, thus minimizing the hemodynamic changes associated with intubation.[12,13] There are no contraindications to using the AWS.

EQUIPMENT

The AWS incorporates an imaging system and a targeting system into one portable tool. It is ergonomically designed to minimize tissue trauma. Its use does not require the alignment of the three airway axes. The imaging system provides an illuminated 90° field of view in the original AWS-S100L model and 80° field of view in the AWS-S200. The S200 is lighter weight.[9] The AWS produces better glottic visualization than traditional DL.[14] The external shell is composed of water-resistant plastic, making it suitable for prehospital and Emergency Department use.

TABLE 20-2 The Disadvantages of Video Laryngoscopy

Adequate mouth opening required
Cost
Different device characteristics
Difficulty passing ET tube despite good airway view
Few comparisons between devices
Increased success in those familiar with device
Injury during ET tube advancement at blind spot
Learning curve with each device
Maintenance
Many different models available
May take longer to intubate than DL
Mucosal trauma
Skills required

TABLE 20-3 Some of the Available Video Intubation Devices and Their Characteristics

Name	Manufacturer	Comments
Airtraq Avant	Prodol Meditec	Disposable blade and eyecups Fits ET tube (ETT) sizes 6.0–7.5 and 7.0–8.5 inner diameter Guiding channel for ETT Low-temperature light source May be used in magnetic resonance imaging (MRI) Optional camera Partly disposable Regular and small adult versions
Airtraq SP	Prodol Meditec	Fits ETT sizes 2.5–3.5, 4.0–5.5, 6.0–7.5, and 7.0–8.5 inner dimeter Fully disposable Low-temperature light source May be used in MRI No guiding channel Regular and small adult, pediatric, and infant versions Six color-coded sizes
Berci-Kaplan DCI Video Laryngoscope	Karl Storz Endoscopy	80° field of view from blades Large size of system Numerous blades available (Miller 0, 1, 4; MAC 2–4, Doerges, and D-blade) Requires components (monitor, light source, control unit) System with interchangeable laryngoscope blades Uses multiple airway devices
Clarus Video System	Clarus Medical LLC	4.0 inch color LCD screen Can be used as lighted stylet Malleable stylet Optional oxygen port Optional tube stop USB connection attaches to monitor or recording device Uses batteries
ClearVue Video Laryngoscope	Infinium Medical	3.5 inch color monitor 66° field of view 2 megapixel camera Anti-shatter screen Disposable and reusable models
C-MAC Pocket Monitor	Karl Storz Endoscopy	2.4 inch monitor that folds and moves in several directions Numerous blades available (Miller 0–1; MAC 2–4, S-blade, & D-blade) Rechargeable battery Works in sunlight Waterproof
C-MAC Video Laryngoscope	Karl Storz Endoscopy	7 inch color monitor 80° field of view from blades Built-in memory card Numerous blades available (Miller 0–1; MAC 2–4, S-blade, and D-blade)
CoPilot VL	Magaw Medical	Adult sizes 3 and 4 C-shaped channel for a bougie Rechargeable battery
Glidescope Advanced Video Laryngoscope (Glidescope AVL)	Verathon Inc.	Color monitor Light, impact resistant, and portable Numerous blades available (4 reusable blade sizes and 6 disposable blade sizes) Real-time video recording Reusable and single-use blades
Glidescope Ranger and Glidescope Ranger Single-use Video Laryngoscopes	Verathon Inc.	Built to military specifications Compact, rugged, and portable Rechargeable battery Reusable blade sizes 3 and 4 Single-use blade sizes 0–4
Glidescope Titanium	Verathon Inc.	6.4 inch color monitor Compatible with Glidescope AVL pediatric blades Durable and lightweight titanium Low-profile blade designs Numerous blades available (4 reusable, LoPro angled 3 and 4, MAC 3 and 4) Real-time video recording Reusable

(*Continued*)

TABLE 20-3 Some of the Available Video Intubation Devices and Their Characteristics (*Continued*)

Name	Manufacturer	Comments
Intubrite Video Laryngoscope	Intubrite LLC	3.5 inch color monitor 2 models available Records images and video Removable SD card Use in adults and children
King Vision aBlade Video Laryngoscope	Ambu USA	2.4 inch screen Attaches to an external display Battery powered (3 AAA) Channeled and nonchanneled blades Fits ETT sizes 6.0–8.5 inner diameter in channeled blade One size corresponds to MAC 3 Reusable video adapter
McGrath MAC Enhanced Direct Laryngoscope	Physio-Control Inc.	EMS version available Flat screen monitor on handle Numerous blades disposable available (2, 3, 4, X3) Screen displays battery usage Uses MAC-shaped blades
McGrath Series 5	Physio-Control Inc.	Blade adjusts to many sizes (adult and pediatric) Blade separates from handle Flat screen monitor on handle Single-use disposable MAC-shaped blade
Pentax AWS	Pentax Medical Co.	2 models 4 blade sizes (neonate, pediatric, standard adult, thin adult) Alcohol or Cidex sterilization Disposable blades Micro USB port Video output to monitor
TotalTrack VLM Video Laryngoscope Mask	Medcom Flow	2.5 inch color screen 2 adult versions for ET tubes 6.0–8.5 Allows gastric and laryngeal aspiration Ideal for prehospital use Inserts from any intubator position Only mask currently available Output jacks SD slot
TruView PCD Video Laryngoscope	Teleflex	5.5 inch monitor Adult and pediatric models Records images and video Soft-feel handles
UE Scope	UE Medical Devices	2.5 inch monitor rotates and tilts Can be used on neonates through adults Designed for anterior airways Disposable and reusable models Rechargeable battery
Venner APA Video Laryngoscope	Venner Medical Intl.	3.5 inch monitor attached to reusable handle Attaches to external monitor Numerous blades disposable available (MAC 3 and 4, difficult airway blade)
VividTrac	Vivid Medical Inc.	Adult and pediatric versions Adult and pediatric sizes Attaches to computer or monitor with USB port Channeled blade Fits ETT sizes 4.0–6.0 and 6.0–8.5 inner dimeter

The body is an integrated 12 cm long cable attached to a charge-coupled device camera, and both models have a 2.4 inch full-color LCD monitor (**Figure 20-1**). The monitor is hinged and allows a 0° to 120° angle for optimal viewing on the original S100L. The S200 is a fixed monitor on top of the device. The monitor screen incorporates a target symbol used to facilitate intubation. External monitoring and recording can be used with the external micro USB output with both devices. The device is battery operated using two AA alkaline batteries for 1 hour of continuous operation.

The PBLADE is a plastic, single-use, disposable, and transparent laryngoscope blade that attaches to the body of the AWS (**Figure 20-1**). It is designed to follow the natural curvature of the upper airway and minimize the need for manipulation of the three airway axes. The blade is composed of Lexan plastic that resists fogging, but not to the extent of the Glidescope antifogging mechanism.[15] The PBLADE comes in four styles (i.e., standard adult blade, thin adult blade for micrognathia or bases where the oral opening is limited, pediatric blade, and neonate blade). The fiberoptic cable

TABLE 20-4 Six Commonly Used Video Laryngoscopes

	Unchanneled devices			Channeled devices		
	C-Mac*	Glidescope*	McGrath*	Airtraq*	King vision*	Venner APA*
Ease of ET tube insertion	4	5	2	1	3	6
First attempt success rate	2	4	1	5	3	6
Insertion into oropharynx	2	3	1	4	5	6
Quality of view	4	2	1	3	5	6
Time of successful intubation (median)	4	5	3	1	2	6
Time to advance ET tube (median)	1	3	2	4	5	6
Time to view vocal cords (median)	1	3	2	4	5	6

*Numbers represent best (1) to worst (6).
Source: Modified from references 7 and 8.

and CMOS camera rest inside the cavity of the PBLADE. An ET tube is loaded into a track located on the right side of the blade. It accommodates a size 6.0 to 8.5 ET tube. Intubation is performed without the use of a stylet through the guided track. Suctioning can be performed under direct visualization with a 4.0 mm (i.e., 12 Fr) or smaller suction catheter inserted through the blade's suction port.

PREPARATION

Prepare and check the AWS before each use. Turn on the device. The video image and target symbol should appear on the monitor. The power lamp below the monitor should illuminate. Immediately replace the batteries if the monitor displays a flickering battery image. Check the light source. Place a hand below the tip of the AWS to ensure that it is illuminating. **Do not look directly at the light source.**

Attach the PBLADE to the AWS. Loosen the lock ring on the scope by rotating it in a leftward direction. **Align the triangular marks on the PBLADE and on the AWS body to ensure proper positioning prior to connection.** Insert the flexible fiberoptic tube through the scope insertion port on the blade. Realign the triangular marks. Keep the connector ring pressed in the direction of the scope body and push in the connector of the PBLADE. Release the connector ring. Confirm that the PBLADE is firmly attached and that the tip of the AWS comes into close contact with the scope window. Secure the connection by turning the lock ring in the rightward direction.

Load the ET tube. Liberally lubricate the ET tube with a water-soluble lubricant. Insert the ET tube by sliding it along the ET tube guide groove. Fix the ET tube onto the hooks located on the proximal end of the PBLADE. Adjust the ET tube so that the tip is aligned with the inferior edge of the PBLADE tip (**Figure 20-1**). Apply medical-grade antifogging to the outside of the PBLADE's scope window.

A B

FIGURE 20-1. The Pentax AWS. ***A.*** AWS-S100L. ***B.*** AWS-S200 and PBLADES. (Photos courtesy of Pentax Medical Co., Montvale, NJ.)

TECHNIQUE

Gently insert the PBLADE into the patient's mouth as when performing DL. Insert it along the right side of the mouth. Gently and slowly advance the PBLADE. **Always visually observe the blade as it is inserted to prevent damage to the patient's lips, teeth, and other soft tissues during insertion. Do not put pressure on the teeth, and make sure that the tongue is not pushed inward while inserting the PBLADE.** Reposition the device to the midline of the mouth once inserted.

Observe the monitor to visualize the airway and epiglottis. Suction any secretions with a 12 French suction catheter inserted through the suction catheter insertion port. Perform suctioning while observing the location of the suction catheter tip on the monitor. Visualize the epiglottis. Slide the epiglottis lifting blade underneath the epiglottis. Gently elevate the device to elevate the epiglottis. Gently manipulate the device until the glottis is aligned with the target symbol on the monitor screen. Gently advance the ET tube through the glottis until the ET tube marker line reaches the glottis. Inflate the ET tube cuff. Detach the ET tube from the hooks. Gently remove the PBLADE from the patient's mouth while securely holding the ET tube in place. Confirm proper ET tube position.

ALTERNATIVE TECHNIQUES

The Emergency Physician cannot be positioned above the patient's head in rare circumstances. In these cases, an inferior or lateral approach can be used. Insert the PBLADE as described above. Connect the AWS to a video monitor, or if using the S100L, adjust the position of the screen using the hinge to obtain an optimal viewing angle. Visualize the epiglottis. The remainder of the technique is exactly as described previously. It may feel awkward to manipulate the device and ET tube from a position inferior or adjacent to the patient's head.

Insertion of the fully assembled AWS may not be possible in obese patients and others with large chests.[16] Separate the PBLADE loaded with the ET tube from the AWS. Insert the PBLADE into the oropharynx under direct visualization. Gently and carefully reattach the AWS to the PBLADE. **Be careful not to traumatize the patient's lips, teeth, or airway soft tissues.** Reassemble the device and proceed with intubation as described above.

This technique can be used for patients in which deep sedation and induction are contraindicated. An off-label technique for awake intubation has been described.[17] Assemble the device. Attach a Bodai connector to the proximal end of the ET tube. Insert a 14 French suction catheter into the lumen of the ET tube via the Bodai connector. Attach the breathing circuit to the other branch of the Y-connector to administer oxygen at a rate of 10 L/min. Insert a tracheal spray tube, smaller than 12 French, through the original suction channel. Attach a syringe loaded with topical anesthetic solution to the proximal end of the tracheal spray tube. Suctioning can be performed through the ET tube as the PBLADE is advanced. The topical anesthetic agent can be delivered via the spray tube. Visualize the glottis. Administer the local anesthetic solution to and through the vocal cords prior to advancing the ET tube through the glottic opening.

COMPLICATIONS

Placement of the AWS may not be possible in patients with limited mouth opening when using the standard PBLADE, which is 25 mm wide.[10] Use the thin PBLADE, which is 9 mm, or a surgical airway. Visualization of the vocal cords can be limited by secretions.[18] **Have a suction catheter always available.** Insert the PBLADE slowly to prevent it from being inserted into pooled secretions. Visualization may not be possible despite aggressive suctioning with particulate matter or severe bleeding in the airway. **Be prepared to use an alternative airway device if this occurs.** Incorrect or forceful insertion can result in dental trauma and soft tissue injuries. The other complications associated with this device are the same as those of DL (Chapter 18).

The ET tube maneuverability is limited when loaded onto the PBLADE. Use a bougie in cases where visualization of the glottis is possible but intubation is not successful. Align the PBLADE and ET tube as much as possible with the glottis. Pass a bougie through the ET tube. Advance and direct the bougie through the vocal cords. Slowly and gently advance the ET tube over the bougie under video guidance.[18]

VL offers a quicker time to visualization and insertion of the ET tube. The time from initiation of intubation to ventilation is not significantly different from traditional DL.[15] It is speculated that this is due to the increased time it takes to remove the ET tube from the AWS after intubation. The time to ventilation can be reduced by beginning manual ventilation before removal of the PBLADE from the oropharynx. Consider this technique in patients who are hypoxic after intubation.[10]

McGRATH LARYNGOSCOPE

The McGrath Laryngoscope (Physio-Control Inc., Redmond, WA) is a VL that is designed to provide a clear video view of the glottis with minimal change in the traditional DL technique. It is a portable VL and modeled after the traditional Macintosh laryngoscope blade (**Figure 20-2*A***). It is a refinement of the "smart scope" laryngoscope developed by Matt McGrath. He won the 1999 Royal Society of Arts student design competition as a British university student for outstanding design achievement for his portable laryngoscope with a small video monitor attached to the handle.[15]

INDICATIONS AND CONTRAINDICATIONS

The McGrath laryngoscope is indicated for elective and emergent intubations. It is useful in difficult intubations in which the three airway axes may be difficult to align (e.g., cervical spine immobilization, high Cormack and Lehane grade airways, limited mouth openings, and tongue edema).[2,19-21] It is indicated for use in pediatric patients weighing 15 kg or more.[22] The McGrath laryngoscope is not indicated for use in awake intubations.[23] The McGrath provides clearer glottic views, greater ease of intubation, and less complications than DL with a Macintosh blade when used by inexperienced providers.[23] There are no contraindications using the VL.

EQUIPMENT

The Series 5 McGrath consists of the handle module, the camera stick, and the blade (**Figure 20-2*A***). The handle module houses the power source and the video monitor. The nonslip rubberized handle contains a single AA battery and the power switch. At the crown of the handle module rests the 1.7 inch color LCD monitor. The monitor can rotate around the handle 360° and can be tilted to adjust the viewing angle. The camera stick houses the camera and light source. Its length is adjustable to produce a blade length ranging from a Macintosh size 3 to 5 blade. The disposable single-use plastic blade fits over the distal camera stick to protect the camera and to assist in lifting of the epiglottis. The McGrath MAC is a newer version without the adjustable length. The McGrath EMS is similar but built tough for field use and orange colored.

PREPARATION

Preparing the Series 5 McGrath for use is quite simple. Unscrew the cap on the top of the handle and insert the AA battery. The top of the handle is opened by turning it counterclockwise and closed by turning it clockwise. Apply the camera stick onto the handle. Pull

A B

FIGURE 20-2. The McGrath Laryngoscope. ***A.*** The Series 5 (left) and the MAC (right). ***B.*** The blade has been inserted and the ET tube is advanced through the vocal cords. (Photos courtesy of Physio-Control Inc., Redmond, WA.)

out the release safety catch at the base of the handle and rotate it so that it is aligned parallel to the long axis of the camera stick. Slide the camera stick into the base of the handle. Return the safety catch to its original position. Firmly attach the disposable sterile laryngoscope blade to the camera stick. Slide it over the camera stick until it firmly latches. Adjust the blade length by sliding the camera stick through the clamp handle. A click will be heard for each adjustment in blade size. Turn the power on by pressing the power switch on the top of the handle. The LED light on the LCD monitor will be continuously lit if there is adequate battery power. The battery power is low if it blinks and the battery should be changed prior to using the device. The McGrath MAC and EMS models have a fixed length and are not adjustable like the Series 5.

TECHNIQUE

The intubation technique using any version of the McGrath is similar to DL.[23] Insert the tip of the blade in the midline and superior to the tongue (**Figure 20-2*B***). Slowly advance the blade and rotate its tip toward the larynx in the sagittal plane until the epiglottis is visualized via direct visualization or via indirect visualization using the camera monitor. Advance the blade further until its tip rests in the vallecula. Gently lift the McGrath until the glottis is visualized. Gently elevate the device to elevate the epiglottis. The monitor should show the vocal cords and surrounding structures (**Figure 20-2*B***). Slightly withdraw the blade until the desired view is obtained if only a portion of the vocal cords are visible. Gently manipulate the device until the glottis is centered on the monitor screen. Insert and advance the proper size ET tube through the vocal cords with the assistance of either a malleable stylet or a bougie. Make the ET tube with a stylet hockey stick shaped 5 cm from the tip to optimize maneuverability.[19] Gently advance the ET tube through the glottis until the ET tube marker line reaches the glottis. Inflate the ET tube cuff. Withdraw the stylet or bougie. Gently remove the blade from the patient's mouth while securely holding the ET tube in place. Confirm proper ET tube position.

ALTERNATIVE TECHNIQUES

An alternative technique can be performed in those with limited mouth opening or chest anatomy that prevents placement of the device into the oropharynx using the traditional DL technique.[20] Disarticulate the Series 5 camera stick from the handle to facilitate placement of the blade into the oropharynx. Insert the camera stick and blade similar to introducing a tongue depressor into the oral cavity. Attach the handle to the camera stick. Intubate the patient as previously described.

COMPLICATIONS

Apply greater upward force to lift the epiglottis if the epiglottis obstructs visualization of the glottic opening. The plastic blade may be used in the same manner as a Miller blade. Redirect the blade to lift the epiglottis, visualize the glottis, and intubate as previously described. Incorrect or forceful insertion can result in dental trauma and soft tissue injuries. The other complications associated with this device are similar to those of DL (Chapter 18).

Check for fogging if the monitor image is unclear. Apply an antifog solution as needed.[15] A blurred image can result from the blade not being latched onto the camera stick. Ensure the video lens is flush with the viewing window of the blade. Remove the blade from the device and clean the camera lens with a gentle soft wipe if neither of these maneuvers is successful.

A grade I view may be obtained of the glottis yet the ET tube cannot be advanced into the trachea. This may be due to the acute bend and long flange of the blade.[24] The device provides a view of the glottis without the alignment of the three airway axes. This requires the ET tube to have a more acute bend than normally used for DL. Place an acute bend in the styletted ET tube approximately 7 to 10 cm from the tip. Another option is to advance the ET tube into the glottis while simultaneously withdrawing the stylet and not inserting the styletted ET tube into the glottis.

BERCI-KAPLAN DCI VIDEO LARYNGOSCOPE

The Berci-Kaplan DCI VL is a video Macintosh intubating laryngoscope developed by Drs. Kaplan, Ward, and Berci (Karl Storz Endoscopy-America, El Segundo, CA).[25] It incorporates micro video imaging technology into a traditional laryngoscope blade. This reduces the learning curve in operating this device for those experienced with traditional DL. It is ideal for use when training

FIGURE 20-3. The Berci-Kaplan DCI Video Laryngoscope. (Photo courtesy of Karl Storz Endoscopy-America, El Segundo, CA.)

the novice in the traditional DL technique.[25,26] A fiberoptic bundle extends from the proximal end of the laryngoscope blade allowing for a larger angle of viewing and a magnified view of the airway. This VL has been marketed for used as part of an "all-in-one" video intubation system (**Figure 20-3**).

INDICATIONS AND CONTRAINDICATIONS

The VL is indicated for normal and difficult intubations in pediatric and adult patients. The large video screen makes this device particularly useful for difficult intubations requiring external manipulation of the airway by an assistant. There are no contraindications to using the VL.

EQUIPMENT

The VL device consists of various laryngoscope blades, the DCI camera head, and the control unit (**Figure 20-3**). The VL blade houses the camera optics. It is available in Macintosh sizes (3 and 4), Miller sizes (0, 1, and 3), and the Doerges universal blade style. The lens provides a viewing angle of 60° to 80° depending on the type of blade. The laryngoscope blade is integrated into the ergonomic handle (**Figure 20-3**). The handle attaches to the camera head, the interface between the control unit and the VL. Other interchangeable airway devices can be attached to the control unit including the Bonfil's scope, Brambrinck scope, and fiberoptic scope (**Figure 20-3**). The control unit incorporates the light source, image processing module, imaging memory, power supply, keyboard, and color LCD monitor.

PREPARATION

Attach the selected laryngoscope blade to the camera head. Turn on the control unit. The laryngoscope blades can be changed with the control unit powered on. Apply medical-grade antifogging solution onto the camera lens.

TECHNIQUE

An advantage of this VL is that the intubation technique does not differ from that of traditional DL. **The added benefit of indirect visualization is that less force is needed to visualize the glottis.** An assistant can view the monitor to facilitate external manipulation of the larynx to bring the glottis into view. The ET tube can be passed through the glottic opening under direct or video-assisted visualization. Gently advance the ET tube through the glottis until the ET tube marker line reaches the glottis. Inflate the ET tube cuff. Withdraw the stylet or bougie. Gently remove the blade from the patient's mouth while securely holding the ET tube in place. Confirm proper ET tube position.

ALTERNATIVE TECHNIQUES

A limitation of this VL is its relatively large handle with cables protruding from its proximal end. This can impede intubation in patients with cervical spine immobilization, large chests, or short necks.[26] Insert and advance the laryngoscope blade into the patient's mouth from the side. Gently rotate and return the laryngoscope handle into the midline once the blade is deep enough and the handle can clear the chest. **Be careful to not push the tongue posteriorly.** Proceed with intubation as previously described.

COMPLICATIONS

A known issue with this VL is lens fogging. This is particularly problematic in patients who are not fully paralyzed. Prevent this by applying a medical-grade antifogging solution, a thin layer of water-soluble lubricant, or the patient's saliva on the lens.[26] Blood and other secretions can obscure the view. Reduce this by not inserting the laryngoscope blade into pooled secretions and by using suctioning.[26] Incorrect or forceful insertion can result in dental trauma and soft tissue injuries. The other complications associated with this device are similar to those of DL (Chapter 18).

Common barriers to purchasing this device are its cost and its large size. The large size of the control unit limits its portability unless it is placed on a rolling cart. The control unit can be used in conjunction with several other Storz airway devices. This makes it a worthwhile investment in Emergency Departments that use other compatible airway devices.[26]

C-MAC VIDEO LARYNGOSCOPE

The C-Mac (Karl Storz Endoscopy-America, El Segundo, CA) is a VL system released in 2009 (**Figure 20-4*A***). It replaces the original VL by Karl Storz. It has numerous advantages including being more compact and portable, a decreased blade width, and improved video technology. The original VL incorporated a fiberoptic camera and video system into a traditional laryngoscope blade. The C-Mac abandons this technology for a CMOS micro video camera. This allows the video system to be incorporated within the laryngoscope blade. The CMOS micro video camera provides an enhanced area of view and does not have the issue of fogging.

INDICATIONS AND CONTRAINDICATIONS

The C-Mac VL is indicated for normal and difficult intubations in pediatric and adult patients. There are no contraindications using the VL.

EQUIPMENT

The handheld portable C-Mac consists of the blade, the electronic module, and the monitor (**Figure 20-4*B***). The blade reproduces the curvature of the traditional Macintosh blade and is composed of

A

B

C

D

FIGURE 20-4. The C-Mac Video Laryngoscope. ***A.*** The C-Mac system. ***B.*** The blades available for the C-Mac system. ***C.*** The C-Mac. ***D.*** The C-Mac PM. (Photos courtesy of Karl Storz Endoscopy-America, El Segundo, CA.)

stainless steel. The proximal end has been flattened to reduce the amount of mouth opening required for intubation and to reduce the risk of oral trauma. The lens is located approximately one-third of the distance from the tip of the blade and provides a 60° field of vision. The CMOS chip housed in the blade provides lens antifogging and an optimal image quality. The blade is available in several Macintosh sizes (2, 3, and 4) and Miller sizes (0 and 1), and there is a hyperangulated adult and pediatric D-Blade that is similar to traditional Glidescope blades.

The electronic module is the interface between the laryngoscope blade and the monitor unit. The module permits easy operator-controlled video documentation. Images can be recorded as still shots or video sequences using the incorporated key pads.

The 7 inch, high-resolution monitor is housed in an impact-resistant and splash-protected plastic body. The top of the monitor has an integrated secure digital (SD) memory card and USB port for video recording and transfer. The monitor automatically white balances when it is turned on. The monitor image can be further modified using the touch key controls to the right of the monitor screen. The monitor device houses a lithium ion battery with a 2 hour operating time when fully charged.

Several new attachments make the C-Mac quite versatile (**Figure 20-4*A***). The C-Mac PM incorporates a 2.4 inch LCD monitor that inserts into the laryngoscope handle (**Figure 20-4*C***). This eliminates the electronic module, cord, and base unit. The LCD monitor unit can be used with all the C-Mac laryngoscope blades. The C-Mac S is a single-use version of the original C-Mac that can tolerate temperatures up to 65°C for sterilization. The C-Cam is a camera head that attaches to the monitor unit. Through this, numerous other airway devices can be attached including all Storz airway devices, the Bonfil's scope, and other fiberoptic scopes (**Figure 20-4*A***).

PREPARATION

Preparing the C-Mac for use is quite simple.[27] Insert the electronic module into the VL blade receptacle. The blade can later be changed while the monitor is on. Insert the yellow connection cord of the electronic module into the yellow socket on the back of the monitor. Connect the power cord into the blue socket on the back of the monitor and plug in the power supply if the battery symbol turns red. The battery sign will indicate that the unit is charging. Insert an SD card into the monitor for recording and image capture. An alternative is to connect an external video recording source into the USB port.

Turn on the C-Mac using the power switch located at the left lower corner of the display monitor. Check the camera for proper functioning by focusing the lens under the laryngoscope blade on a hand. The camera's light source will be visible on the hand. The light intensity, color saturation, and contrast can all be adjusted using the key pads located on the monitor. Wipe the lens with a soft cloth or lens tissue if the video image is blurry. Clean the contacts of the electronic module if wiping the lens does not improve the image. Recording can be performed by pressing the record button located on the laryngoscope blade handle.

TECHNIQUE

The intubation using the C-Mac is exactly the same as using traditional DL (Chapter 18).[27] Insert the laryngoscope blade into the right side of the oral cavity under direct visualization. Move the VL to the midline and catch the tongue on the flange of the blade. Advance the blade past the oropharynx and into the vallecula. **Be careful to not push the tongue posteriorly.** Lift the laryngoscope handle upward to improve visibility. Visualization can be performed by direct visualization or via the monitor. Gently advance the ET tube through the glottis until the ET tube marker line reaches the glottis. Inflate the ET tube cuff. Withdraw the stylet or bougie. Gently remove the blade from the patient's mouth while securely holding the ET tube in place. Confirm proper ET tube position.

The C-Mac PM is similar to the McGrath. Turn on the device. Insert it into the midline of the patient's mouth and advance it into the vallecula. Adjust the monitor if needed. Insert, verify the location, and secure the ET tube.

COMPLICATIONS

Incorrect or forceful insertion can result in dental trauma and soft tissue injuries. The other complications associated with this device are similar to those of DL (Chapter 18).

GLIDESCOPE

The Glidescope (Verathon Inc., Seattle, WA) combines a video camera with a patented antifog system into a portable laryngoscope blade and monitor system. The blade design modifies the traditional Macintosh blade curvature to provide a more anterior view of the larynx. The Glidescope was developed by a Canadian surgeon.[13] Modifications in the basic design have resulted in several models (**Figure 20-5**). The Glidescope AVL uses a single-use disposable blade for a rapid turnaround without sterilization between uses.[28] The Ranger Glidescope models provide a compact, high-impact device originally intended for military and prehospital use.[29] The Glidescope Titanium provides two different blade style options, the original low-profile angulated design that the previous Glidescope models are known for and a Macintosh-style blade that can allow VL and DL.[29]

INDICATIONS AND CONTRAINDICATIONS

The Glidescope is indicated for routine and difficult intubations. It is particularly useful for patients requiring cervical spine immobilization. It has been demonstrated to have a significant advantage over the Macintosh blade in the patient with tongue edema.[30] It can be used in pediatric patients who weigh as little as 1.8 kg. There are no reported contraindications to using this device.

EQUIPMENT

There are three current Glidescope models (i.e., Titanium Glidescope, Glidescope AVL, and Ranger Glidescope). Some Emergency Departments may use an older version known as the Glidescope GVL. The primary components of the Glidescope are the blade and the video monitor. All the blade models incorporate an autofocusing CMOS camera, LED light source, and patented antifogging mechanism housed inside of a medical-grade plastic shell. The original Glidescope blade begins with the traditional Macintosh blade and adds a 60° curvature at the midpoint. This allows for a more anterior view of the airway with less lifting force required.[15] The camera lens lies at the distal aspect of the blade's curve to protect it from secretions while providing a close-up view of the glottis. The Glidescope produces better glottic visualization than traditional DL.[31-34]

The traditional Glidescope blade is a reusable integrated blade and handle. It is available in four sizes. The traditional Ranger blade consist of a reusable integrated blade, handle, and video cable that cradles into the body of the Glidescope monitor. It is currently available in two sizes. The Ranger single-use blades are available in six sizes (i.e., 0, 1, 2, 2.5, 3, and 4). These are available in both LoPro and MAC blade styles.

The Glidescope AVL and Ranger single-use blade consists of a video baton and a STAT blade. The video baton houses the CMOS camera, LED light, and antifogging mechanism. The baton is inserted into a one-time use STAT blade constructed of medical-grade plastic. The STAT blade is shaped like the traditional Glidescope blade and provides the protective cover for the baton, reducing the risk of transmitting an infection.[30] This dramatically reduces turnaround by eliminating the 30 minutes required to clean traditional blades prior to reuse.[28,30] The baton is available in two sizes and four blade sizes.

The blades are connected to the video monitor with a video cable. The traditional Glidescope, the Cobalt Glidescope, and the Titanium Glidescope have a 7 inch, nonglare, color LCD monitor. Housed within the monitor is the battery and power device. The monitor includes the power switch and control pads that allow adjustment of the video image. The Ranger versions have a rugged shell for field use and a 3.5 inch color, nonglare LCD. The body of the monitor incorporates a cradle for the blade. The Ranger Glidescope uses a rechargeable lithium ion battery. The Ranger can be used for 90 continuous minutes or approximately 20 intubations.[29] The Titanium Glidescope video monitor has video output and recording capabilities to allow it to be connected to a larger monitor or have videos of intubations saved (e.g., for educational or quality-improvement purposes).

Many of the Glidescope models come with the GlideRite rigid stylet, although it is not required for intubation (**Figure 20-5**). It is a rigid stylet designed to complement the angle of the Glidescope blades. Initial studies indicate that intubation with the GlideRite rigid stylet offers no advantages over the standard malleable stylet.[35] More recent literature demonstrates an increased success rate in first-pass and successful intubation in the Emergency Department with the Glidescope when the rigid stylet is used instead of a malleable stylet.[36]

A

B

C

D

E

FIGURE 20-5. The Glidescope Video Laryngoscope. ***A.*** The Glidescope GVL system. ***B.*** The Glidescope AVL system. ***C.*** The Glidescope Ranger system. ***D.*** The Glidescope Titanium. ***E.*** Single-use titanium blades. (Photos courtesy of Verathon Inc., Seattle, WA.)

PREPARATION

Preparing the Glidescope for use is quite simple for all models. Attach the Glidescope blade to the monitor using the video cable. Turn on the device, and it is ready for use. For the Cobalt and single-use Ranger models, insert the baton into the STAT blade. A click will be heard to confirm that the blade has been securely attached. It is not required to use the GlideRite rigid stylet. It is recommended when using a traditional malleable stylet to curve it to model the shape of the Glidescope blade to facilitate manipulation of the ET tube.[15,28,29,37-39]

TECHNIQUE

The intubation technique using the Glidescope is similar to that of traditional DL (Chapter 18).[28,29] Insert the blade into the midline of the oral cavity. Advance the blade under direct visualization until its tip reaches the pharynx. **Be careful to not push the tongue posteriorly.** Continue to advance the blade while observing the monitor to identify the epiglottis. Advance the blade into the vallecula. Lift the blade, if necessary, to elevate the epiglottis. Insert the ET tube under direct visualization until its tip nears the tip of the laryngoscope blade. Guide the ET tube toward the glottic opening. Pull the GlideRite stylet back approximately 2 cm and advance the ET tube through the vocal cords. This will facilitate passage of the ET tube through the vocal cords while reducing the potential for injury. Continue to gently advance the ET tube through the vocal cords until the ET tube marker line reaches the glottis. Inflate the ET tube cuff. Withdraw the stylet. Gently remove the blade from the patient's mouth while securely holding the ET tube in place. Confirm proper ET tube position. The option of intubating with direct visualization and confirming via video is available when using the Titanium Glidescope.

There are several troubleshooting tips when problems arise using the Glidescope. Positioning the blade to the left of midline makes room for the ET tube in small or limited-opening mouths. The view may be improved by backing up the blade a little. Trouble advancing the ET tube is eliminated with the thumb advancing it. A limited or restricted view (i.e., not a Cormack-Lehane grade 1) may aid in advancing the ET tube into the airway.[40-42]

COMPLICATIONS

Use of a Glidescope for intubation can increase the time to advance the ET tube through the vocal cords when compared to intubation using DL.[12] This difference in time decreases with increased use and familiarity with the device.[43] It is strongly recommended that novice operators use the Glidescope on mannequin models and for "routine" intubations prior to using it as a rescue device for difficult intubations.

Intubation with the Glidescope can be difficult in patients with small mouths or large tongues due to the limited area for the blade and ET tube. It may be difficult to pass the ET tube into the larynx. **Do not blindly insert the ET tube as it may cause injury to the oropharyngeal tissues.**[44] Insert the Glidescope under direct visualization and then move it to the left if space is a problem. The ET tube can then be inserted under direct visualization. The view on the monitor of the larynx may appear deviated.[44] Manipulate the Glidescope to obtain an optimal view on the monitor. An additional technique is to shape the ET tube into a hockey stick or J-curve to facilitate advancement and manipulation from the lateral aspect of the mouth.[12] Incorrect or forceful insertion can result in dental trauma and soft tissue injuries.[45,46] The other complications associated with this device are similar to those of DL (Chapter 18).

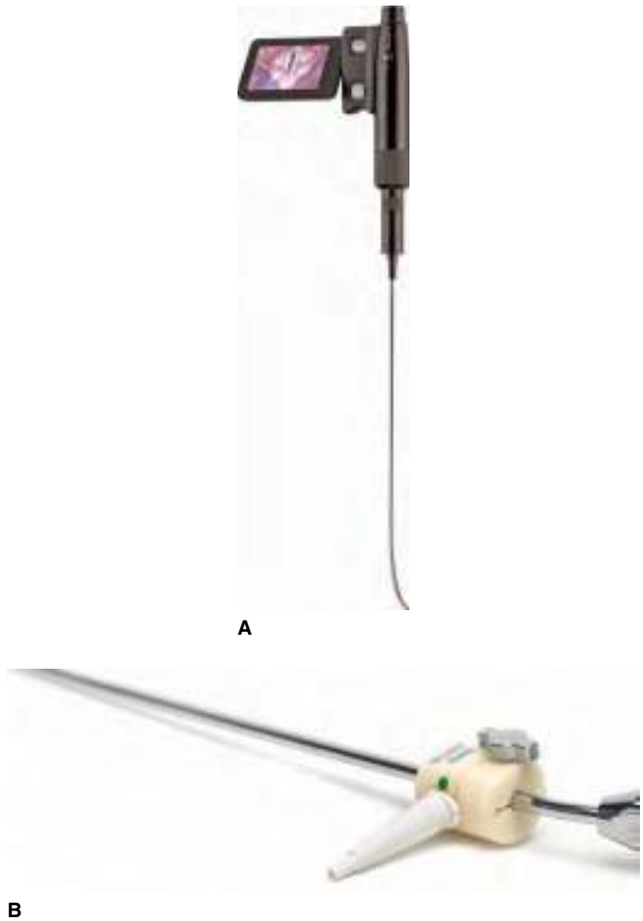

FIGURE 20-6. The Clarus Video System. ***A.*** The system. ***B.*** The disposable tube stop and oxygen port. (Photos courtesy of Clarus Medical LLC, Minneapolis, MN.)

CLARUS VIDEO SYSTEM

The Clarus video system (Clarus Medical LLC, Minneapolis, MN) is a device that can be modified with various accessories for use as a VL, ET tube exchange catheter, flexible scope, and malleable stylet.[47-50] It incorporates the video, lighting, power, and monitor system into one handheld product (**Figure 20-6**). The malleable video stylet shaft and the disposable VL blade are designed for intubation.[47] The optional flexible scope is detachable for use as an ET tube exchange catheter.

INDICATIONS AND CONTRAINDICATIONS

The Clarus Video System is intended for use in performing and confirming placement of an ET tube. It is indicated for use alone or as a video stylet in conjunction with a DL. It can be used for routine and difficult intubations. There are no contraindications to using this device.

EQUIPMENT

The handle of the video system houses the rechargeable power unit. The color LCD display monitor attaches to the handle. The monitor viewing angle can be adjusted with an easy to reach thumb control. The light intensity can be adjusted using controls on the handle. The handle contains the external video connector and the battery charger port. The distal lens, light LED, and camera are housed in the distal tip of the video stylet shaft.

A disposable tube stop fits on the malleable video stylet to hold the ET tube at the desired height on the stylet (**Figure 20-6*B***). An oxygen port can be connected to the tube stop to supply supplemental oxygen through the ET tube. A detachable and flexible video scope is available for use during ET tube exchange.

PREPARATION

Preparing the Clarus video scope for use requires the device to be assembled. Place the disposable tube stop over the stylet shaft. Attach the oxygen port to the matching circular receptacle on the tube stop. Attach oxygen tubing to this port if desired. The oxygen flow rate and pressure must be monitored to prevent barotrauma.[47]

Apply an ET tube with a minimum diameter of 5.5 mm and a maximum length of 34.3 cm over the distal tip of the stylet and fitted to the tube stop. Consider applying water-soluble lubricant onto the stylet to facilitate this process.[47] Consider applying a medical-grade antifogging agent to the lens on the stylet tip. Adjust the tube stop so that the stylet tip rests just proximal to the distal end of the ET tube to provide optimal visualization and reduce the potential for tissue trauma. Tighten the clamp screw on the tube stop to fix the position of the ET tube onto the stylet shaft. The image on the monitor screen will automatically focus when the device is turned on. The illumination can be adjusted using the dial on the monitor.

TECHNIQUE

The device can be used for intubation with a traditional DL. Insert the laryngoscope and attempt to visualize the vocal cords and other surrounding structures. Insert the ET tube on the Clarus stylet if the vocal cords are visualized similar to performing traditional DL (Chapter 18). Visualization of the airway structures may not be possible in patients with difficult airways or those requiring cervical spine immobilization. Use the monitor on the Clarus Scope to direct, manipulate, and advance the styletted ET tube through the glottis. Gently advance the ET tube through the glottis until the ET tube marker line reaches the glottis. Inflate the ET tube cuff. Withdraw the Clarus stylet. Gently remove the laryngoscope blade from the patient's mouth while securely holding the ET tube in place. Confirm proper ET tube position.

COMPLICATIONS

Incorrect or forceful insertion can result in dental trauma and soft tissue injuries. The other complications associated with this device are similar to those of DL (Chapter 18).

KING VISION VIDEO LARYNGOSCOPE

The King Vision Video Laryngoscope (Ambu USA, Columbia, MD) and the King Vision aBlade Video Laryngoscope are battery operated, rigid, and portable digital VLs (**Figure 20-7**). They are simple to use and understand. The King Vision is significantly less expensive than most other VLs. The device uses organic light emitting diode (OLED) technology.

INDICATIONS AND CONTRAINDICATIONS

The King Vision is indicated for routine and difficult intubations in adolescents and adults. The laryngoscope blade is equivalent to a Macintosh #3 size blade. There are no contraindications to using this device if a Macintosh #3 blade is appropriate for intubation. The King Vision aBlade can be used for any type of airway, whether routine or complicated. It is small and lightweight enough to be kept in an airway cart as a primary intubation tool or as a backup.

FIGURE 20-7. The King Vision Video Laryngoscope. (Photo courtesy of Ambu USA, Columbia, MD.)

EQUIPMENT

The King Vision consists of the color OLED monitor base unit that attaches to a disposable laryngoscope blade (**Figure 20-7**). The two pieces snap together. The monitor unit houses three AAA batteries as the power source, the on/off switch, and a mini USB port for video output to a display monitor or recording device. The batteries provide at least 90 minutes of "on time." The OLED monitor offers increased contrast at a lower cost than LCDs and LEDs. The monitor is brighter, faster, lighter, thinner, and uses less power than LCDs.

The blades are plastic, single-use, and disposable, and are available in two styles (i.e., with and without an ET tube channel). The channeled blade accommodates a 6.0 through 8.0 ET tube. The blade is only available in a Macintosh #3 size. It differs from the standard Macintosh #3 blade in that it is slightly shorter and wider. The blade is smaller than most VLs. It requires a minimum mouth opening of 13 mm for the standard blade and 18 mm for the channeled blade. A CMOS camera and LED light source are mounted on the disposable blade. The lens has an antifog coating.

PREPARATION

Preparation of the King Vision is quick and simple. Ensure that the power is off. **Applying a blade with the unit powered on will result in image distortion.** Choose a laryngoscope blade with or without a channel. Slide the blade onto the monitor piece and snap it in place. The front and back of the blades and the monitor unit are color coded to facilitate proper orientation. The blade with a channel can be used with or without an ET tube inserted into the channel. Load an appropriate-sized ET tube, if desired, without a stylet into the well-lubricated channeled blade. Use only water-soluble lubricant. Avoid placing lubricant over the lens. Align the distal tip of the ET tube at the end of the channel. Turn on the device. Replace the batteries if the battery indicator light is flashing red. Observe the monitor to ensure that an image is present and clear. The image will be distorted if the unit was powered on when the blade was attached. Simply turn the unit off and then back on to obtain a clear image. The tip of the ET tube, if using the channeled blade, should not be visible on the monitor. If it is visible, pull the ET tube back until the tip is aligned with the tip of the laryngoscope blade and no longer visible in the monitor.

TECHNIQUE

The technique for intubation is similar to traditional DL if using the standard blade or the channeled blade without loading an ET tube (Chapter 18). Insert the blade in the midline and advance it into the oropharynx. **Be careful to not push the tongue posteriorly.** Continue to advance the blade toward the vallecula while observing the monitor. The blade can be placed into the vallecula like a Macintosh blade or can be used to elevate the epiglottis like a Miller blade. View the monitor while directing, manipulating, and advancing the styletted ET tube through the glottis. Gently advance the ET tube through the glottis until the ET tube marker line reaches the vocal cords. Inflate the ET tube cuff. Withdraw the device. Gently remove the laryngoscope blade from the patient's mouth while securely holding the ET tube in place. Confirm proper ET tube position.

A slight technique modification is required if mounting an ET tube into the channeled blade. Insert the blade as described previously. Center the glottic opening on the screen. Slide and advance the ET tube along the channel and through the vocal cords. Minor manipulation of the blade may be required to align and advance the tip of the ET tube through the vocal cords. A bougie can be loaded through the ET tube to facilitate intubation.

COMPLICATIONS

Imaging can be compromised by excessive secretions or blood in the oropharynx. Incorrect or forceful insertion can result in dental trauma and soft tissue injuries. The other complications associated with this device are similar to those of DL (Chapter 18).

ALTERNATIVE DEVICES

The above devices are representative devices used in the Emergency Department. Other devices are listed briefly below and used like the devices already described.

COPILOT VL

The Copilot VL (Magaw Medical, Fort Worth, TX) is small and portable (**Figure 20-8**). It consists of the display monitor, handle video blade, and disposable sheaths.[51] A cable attaches the handle to the monitor. It is lower in cost than other VLs. The side channel can be used to guide a bougie toward the screen image. The lithium battery is rechargeable and can be used for 2 hours continuously before the charge runs out. The monitor has a battery status indicator. The

A

B

FIGURE 20-8. The Copilot Video Laryngoscope. (Photos courtesy of Magaw Medical, Fort Worth, TX.)

FIGURE 20-9. The Intubrite Video Laryngoscope. (Photo courtesy of Intubrite LLC, Vista, CA.)

Copilot VL has an antifog mechanism that requires the device to be on for 30 seconds prior to laryngoscopy. The proprietary rigid stylet can be used in the ET tube instead of a standard stylet. The Copilot VL is only available in adult sizes 3 and 4. The contaminated disposable sheath can be removed from the device hands-free. The Copilot VL is appropriate for prehospital, Emergency Department, and in-hospital use.

INTUBRITE VL

The Intubrite VL (IntuBrite LLC, Vista, CA) has a 3.5 inch monitor attached to the laryngoscope (**Figure 20-9**).[52] It records images and video to the removable SD card. It is available in two models (i.e., VLS 8800 and VLS Edge), is durable, and is made of stainless steel. The Intubrite VL contains a high-resolution camera, data output port, and a video output port. The VLS Edge uses reusable blades or sheaths in six sizes. The VLS 8800 blades come in sizes 2, 3, and 4A for anterior airways.

FIGURE 20-10. The VividTrac Video Laryngoscope. (Photo courtesy of Vivid Medical Inc., Palo Alto, CA.)

VIVIDTRAC VL

The VividTrac (Vivid Medical Inc., Palo Alto, CA) is available in an adult and pediatric version (**Figure 20-10**).[53] The adult version uses ET tube sizes 6.0 to 8.5, and the pediatric version uses ET tube sizes 4.0 to 6.0. The shape is anatomically designed. The VividTrac contains a stainless steel blade, ET tube channel, and a rounded tip. It contains an antifog mechanism. The 3 foot USB cord is integrated and attaches to a computer or monitor. The device is single-use, contains and needs no battery, is maintenance-free, and requires no cleaning.

VENNER APA VL

The Venner APA VL (Venner Medical Intl., Saint Helier, Jersey) is a battery-powered device with a CMOS camera (**Figure 20-11**).[54] A 3.5 inch high-resolution monitor is mounted on an ergonomic handle. The light source is mounted on the distal tip and increases the intensity. Four blades are available (i.e., Mac 3, Mac 4, difficult airway blade [DAB], and an unchanneled difficult airway blade [U-DAB]). A video port connects the device to an external monitor.

FIGURE 20-11. The Venner APA Video Laryngoscope. (Photo courtesy of Venner Medical Intl., Saint Helier, Jersey.)

FIGURE 20-12. The TruView PCD Video Laryngoscope. (Photo courtesy of Truphatek Intl. Ltd., Netanya, Israel.)

TRUVIEW PCD VL

The TruView PCD VL (Truphatek Intl. Ltd., Netanya, Israel) is available in an "R" model and a pediatric model (**Figure 20-12**).[55] The "R" model uses blades in sizes 0, 1, 2, 3, and 4. The pediatric model uses blades in sizes 0, 1, 2, and 3. It can be used in adults with restricted mouth opening. The blades have a built-in jet oxygen cleaning system. The devices have soft-feel rechargeable power handles with LED illumination. The devices have a 5.5 inch monitor and allow for recording videos and photos.

UE SCOPE

The UE Scope (UE Medical Devices, Newton, MA) can be used in neonates through adults (**Figure 20-13**).[56] It contains a rechargeable battery for over 90 minutes of operation, an adjustable grip, an adjustable 2.5 inch monitor, and an antifog mechanism. The angulated blades are meant for anterior airways and are available in five reusable sizes (i.e., 0, 1, 2, 3, and 4) and three disposable sizes (i.e., 2, 3, and 4). The camera points upward to provide a 60° field of view. A proprietary rigid stylet can be used.

AIRTRAQ VL

The AirTraq VL (Prodol Meditec SA, Las Arenas, Spain) is available in an all-in-one design (**Figure 20-14**).[57-62] The Avant model has disposable blades/sheaths, a flip screen that rotates, and is compatible with magnetic resonance imaging (MRI). The SP model is completely disposable and MRI compatible. Both models are equipped with a Wi-Fi camera that allows viewing on the device, an attachable

FIGURE 20-13. The UE Scope. (Photo courtesy of UE Medical Devices, Newton, MA.)

A

B

C

FIGURE 20-14. The Airtraq Video Laryngoscope. ***A.*** Avant model. ***B.*** SP model. ***C.*** Wi-Fi camera. (Photos courtesy of Prodol Meditec SA, Las Arenas, Spain.)

camera, or an attachable monitor. A channel guide does not require a stylet and allows for a bougie. They can be used on infants through adults (i.e., ET tube sizes 2.5 to 8.5). Adults with restricted mouth opening can use the pediatric version with the adult-sized ET tube. It can be used on morbidly obese patients. The Airtraq causes less blood pressure and heart rate alterations than DL.

TOTALTRACK VLM

The TotalTrack VLM (Medcom Flow SA, Barcelona, Spain) is the only available VL mask (**Figure 20-15**).[63-67] It allows intubation and ventilation while directly visualizing the airway. It is available in two adult versions. One uses ET tubes sized 7.5 to 8.5, and the other uses size 6.0 to 7.5 ET tubes. There are two aspiration ports (i.e., gastric and laryngeal). The device contains a 2.5 inch monitor screen and an SD memory card slot and is battery operated. The output jack allows attachment to a monitor. The TotalTrack can be inserted from any intubator position.

CLEARVUE VL

The ClearVue VL (Infinium Medical, Largo, FL) comes with a 3.5 inch display monitor, rechargeable lithium ion battery, and an anti-shatter screen (**Figure 20-16**).[68] It is available with disposable and reusable blades. The ClearVue VL has antifog technology, a quick-shot camera button, and a 66° field of view.

SUMMARY

There is a growing array of video intubation devices available with varied designs. This includes those that embed the camera into the laryngoscope blade, those that embed the video camera into a scope

FIGURE 20-15. The TotalTrack VLM. (Photos courtesy of Medcom Flow SA, Barcelona, Spain.)

or stylet for use inside an ET tube, and those that use an ET tube delivery device. They represent a simple method for routine intubation and a promising rescue device for the difficult airway. These devices have varied learning curves. It is important to use these devices in simulations and routine intubations prior to using them as a rescue devices. These devices are useful for training healthcare personnel in the technique of orotracheal intubation in the prehospital and hospital setting. The use of a VL to intubate is much easier than using traditional DL, especially for novices.

FIGURE 20-16. The ClearVue Video Laryngoscope. (Photo courtesy of Infinium Medical, Largo, FL.)

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21 Fiberoptic-Assisted Endotracheal Intubation Devices

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INTRODUCTION

Recent years have seen a rapid expansion in optical devices used to aid in endotracheal (ET) intubation. The devices that use fiberoptics begin near the distal end and transmit an image to be viewed at the proximal end. Some of these devices are variations on the "optical stylet" concept (**Table 21-1**). They consist of an eyepiece or other viewing mechanism attached to stylet of varying degrees of flexibility. A standard ET tube can be jacketed onto each device. The stylet can then be used as an adjunct to standard ET intubation or as a stand-alone device. These devices are being replaced by video laryngoscopes that are more versatile, easier to operate, and easily learned by a novice user. Most fiberoptic stylets and their variations are falling out of favor and are less often used today.

Traditional direct laryngoscopy requires alignment of the oral, pharyngeal, and laryngeal axes to visualize the glottis. It is not always possible to align these three axes despite mechanical manipulation. The major advantage of fiberoptic laryngoscopy devices is that they do not require the Emergency Physician to align the three airway axes, reduce the need for airway manipulation, and reduce potential traumatic forces on the airway (**Table 20-1**). Fiberoptic devices provide a superior view of the glottis when compared to traditional direct laryngoscopy. The lens of fiberoptic devices is within centimeters of the glottis and provides a wider angle of vision than the 15° of traditional direct laryngoscopy. The viewing port magnifies the view of the airway, making critical structures easier to visualize.

A generalized approach to using optical stylet-type devices will first be discussed, followed by the unique features of selected devices. This chapter reviews a representative number of device types currently available and commonly used in Emergency Departments (**Table 21-1**). A detailed discussion of video laryngoscopy is contained in Chapter 20. The intent of this chapter is not to describe every single product on the market.

TABLE 21-1 Some of the Commercially Available Fiberoptic Devices

Name	Manufacturer	Comments
AincA Video Stylet	Anesthesia Associates	2.4 inch rechargeable color monitor Can be used with a laryngoscope handle Fits ET tube (ETT) sizes ≥ 6.0 mm inner diameter Malleable
Air-Vu Plus Fiberoptic Stylet	Cookgas	Adjustable tube stop Can be used with a laryngoscope handle or battery light source Designed for use with Air-Q laryngeal mask Fits ETT sizes ≥ 5.5 mm inner diameter Oxygen insufflation port Rigid
Bonfils Retromolar Intubation Endoscope	Karl Storz Endoscopy	Can be used with battery-powered or portable light source Comes in two sizes (3.5 and 5.0 mm outer diameter) ETT must be 0.5 mm or greater in inner diameter size to fit oxygen insufflation port Rigid with a 40° curve Rugged and able to lift soft tissues out of the way Use with eyepiece or connect to DCI Video System
Clarus 30000-V Intubation Video System	Clarus Medical	5.0 mm outer diameter Attached LCD screen Can be used as a lighted stylet Fits ETT sizes ≥ 5.5 mm inner diameter Malleable rigid scope Red LED illumination provides better visualization
Levitan Scope	Clarus Medical	Fits ETT sizes ≥ 5.5 mm inner diameter Malleable rigid scope Oxygen insufflation port Preformed shape ("hockey stick") Tube stop Use in direct laryngoscopy or alone
Pocket Scope	Clarus Medical	Deflected tip allows nasal intubation Fits ETT sizes ≥ 4.0 mm inner diameter Flexible stylet Stylet wraps around to limit size
SensaScope	Acutronic Medical Systems	3 cm of tip steers Fits ETT sizes ≥ 6.5 mm inner diameter Lever moves tip 75° in sagittal plane Semirigid-shaped
Shikani Optical Stylet	Clarus Medical	Can use multiple light sources Comes in two versions (adult and pediatric) Fits ETT sizes 2.5–5.0 and 5.5 mm inner diameter Malleable rigid scope Oxygen insufflation port Preformed shape ("hockey stick") Tube stop Use in direct laryngoscopy or alone

INDICATIONS AND CONTRAINDICATIONS

Fiberoptic intubation devices can be used for adult and pediatric difficult (i.e., actual or anticipated) intubation, elective intubation, emergent intubation, and routine intubation. These devices can be used as "rescue devices" in cases of failed direct laryngoscopy. Patients with their head and neck immobilized, limited mouth opening, or morbid obesity or patients requiring awake intubation or intubation while in a sitting position can benefit from intubation with these devices. These devices provide better visualization of the glottis, decreased cervical movement, and higher success rates in comparison to traditional direct laryngoscopy. These devices can be used to aid in the localization and removal of airway foreign bodies. There are no contraindications to using fiberoptic devices.

USING FIBEROPTIC INTUBATION DEVICES

There are some general guidelines to using fiberoptic intubation devices that will improve the Emergency Physician's success in their implementation. It is useful to understand the limitation of these devices. Fogging is a common problem. Fogging can be minimized by applying a medical-grade antifog solution to the lighted end of the instrument or warming the distal tip by placing it in a warm blanket or warmed saline solution. Any fluid (e.g., blood, secretions, or vomitus) in the oropharynx will limit the usefulness of these devices. Suction any fluid and debris from the oropharynx prior to device insertion. Use care to avoid touching the mucosal surface with the distal tip of the device. Encountering the mucosa can cause "pink out" and promote fogging of the device, both of which will impair visualization. Fiberoptic intubation devices cannot be used to separate tissues. They should follow an open channel created by a standard laryngoscope or by manual distraction of the patient's jaw.[1] Small movements are magnified by these devices. Proceed slowly and identify known landmarks as the device is navigated from the mouth to the glottic opening. Getting lost in a field of pink mucosa is best overcome by slowly backing the device out until known landmarks are visualized and identified, and then slowly re-advancing the device along a path of familiar anatomy.

LEVITAN SCOPE

The Levitan Scope (Clarus Medical, Minneapolis, MN) was created by Dr. Richard Levitan (**Figure 21-1**). He is an Emergency Physician, a noted airway educator, and an innovator. The Levitan Scope differs from other devices in this chapter in that it is shorter and requires the ET tube to be cut to a length of approximately 28 cm. Cutting the ET tube is an inconvenient step. Doing so makes the working length of the scope shorter and wielding the device easier. The scope is equipped with a side port that allows for oxygen insufflation. The distal end of the device is flexible and allows it to be bent to the specific needs of the scenario. It is powered by a detachable battery-operated light source or with a standard green line fiberoptic laryngoscope handle. It is relatively affordable compared to other

FIGURE 21-1. The Levitan Scope. (Photo courtesy of Clarus Medical, Minneapolis, MN.)

devices. Much of the intubation strategy described for the Levitan Scope is generalizable to other optical stylets. The techniques and strategies are summarized from a publication by the inventor.[2]

PREPARATION

Prepare the scope for use. Liberally lubricate the scope with water-soluble lubricant. **Do not get lubricant on the lens.** Apply an ET tube of 6.0 mm or larger over the shaft. **The tip of the scope should be approximately 1 cm from the end of the ET tube.** Connect the side port to oxygen tubing. Turn on the oxygen source to provide a flow rate of 5 to 10 L/min. This flow rate allows for oxygen insufflation and aids in keeping the lens clear. The manufacturer recommends bending the distal tip to approximately 35° in the straight-to-cuff fashion. Attach the light source, either a miniature LED light source or a green line fiberoptic laryngoscope handle.

TECHNIQUE

The Levitan Scope was designed for incorporating the option of fiberoptic assistance into every intubation attempt. The scope will simply act as a stylet in most cases where landmark visualization is feasible with standard laryngoscopy. Additional confirmation can be made by visualizing the tracheal rings via the eyepiece once ET intubation is achieved.

The Levitan Scope jacketed with an ET tube can be used for fiberoptic intubation when ET intubation is not possible by direct laryngoscopy. Obtain the best visualization of the airway anatomy possible using the traditional laryngoscope. At least the tip of the epiglottis should be visible. Introduce the scope into the mouth under direct vision. Advance the scope until its tip is positioned approximately 1 cm superior to the epiglottis. It may be helpful to rest the device against the patient's dentition at the right corner of the mouth to maintain this position. Avoid touching the distal tip of the scope against the mucosa to prevent "pink out" and fogging.

Switch from direct visualization to fiberoptic viewing via the device's eyepiece. Identify the airway structures. Slowly advance and direct the tip of the scope under the epiglottis. Continue to advance the tip of the device into the glottic opening. Pivoting the device toward the Emergency Physician will direct the tip of the scope anteriorly toward the glottic opening if the device is resting at the right corner of the patient's mouth. Confirm proper placement by visualizing the tracheal rings through the eyepiece.

Remove the laryngoscope once the tip of the Levitan Scope with the jacketed ET tube has been advanced through the vocal cords and into the trachea. Advance the ET tube by rotating it off the stylet in a counterclockwise fashion. Rotating the ET tube while advancing it helps to prevent the beveled edge from catching on the tracheal rings. Securely hold the ET tube. Remove the Levitan Scope and secure the ET tube.

The Levitan Scope can be used without a laryngoscope, although it may require more practice to become adept at this technique. Prepare the scope as described above, with the exception that the distal 3 to 4 cm should be bent to approximately 70°. Grasp the patient's jaw and tongue in the left hand and lift to create an open channel for the scope. Insert and advance the scope in the midline while avoiding contact with the mucosal surfaces. Look through the eyepiece and advance the device with the ET tube through the glottic opening.

VIDEO RIFL

The Video Rigid Intubating Fiberoptic Laryngoscope or Video RIFL (AI Medical Devices Inc., Williamston, MI) is unique among the optical stylets in that it has a flexible tip that can be manipulated with a trigger at the handle-end of the device (**Figure 21-2**). There were originally two models available, the Airway RIFL and the Video RIFL. The Video RIFL replaced the eyepiece of the Airway RIFL with a small LCD screen. The Nasal RIFL stylet attaches to the device and allows nasal intubation. The high-resolution LCD screen rotates 180° to provide visualization from any angle. The Video RIFL has a unique tip that articulates up to 135° to navigate obstructions. The device can be used as a stand-alone device or as an adjunct to standard laryngoscopy. It can be used to facilitate intubation through many supraglottic airway devices (e.g., the Air-Q or I-gel). The primary disadvantages of this device are the cost, the relative length of the device, and that it is top-heavy. Being top-heavy and the length may prove cumbersome for users of shorter stature.

FIGURE 21-2. The Airway RIFL. (Photo courtesy of AI Medical Devices Inc., Williamston, MI.)

The two-sided RIFL Blade can be used in conjunction with the Video RIFL. It is a disposable, single-use, plastic blade the use of which is optional. The RIFL Blade is inserted into the midline of the patient's mouth and lifted to elevate the tongue, open the airway, and provide a path to the glottis.

PREPARATION

Inspect the Video RIFL for any damage. Turn it on. Liberally lubricate the stylet portion of the device with a water-soluble lubricant. Place a 6.5 mm or larger ET tube onto the stylet. The current model does not accommodate ET tubes less than 6.5 mm. A smaller pediatric version is being developed. Set the tube stop so that the tip of the stylet is just inside the distal end of the ET tube.

TECHNIQUE

The device can be used as a stylet to introduce the ET tube into the trachea as an adjunct to standard direct laryngoscopy if the glottic opening can be easily visualized using a standard laryngoscope.

Insert and advance the device as described for the Levitan Scope if the laryngeal structures are not well visualized with direct laryngoscopy. Attempt to visualize the epiglottis through the eyepiece or the LCD screen of the Video RIFL. Advance the device under the epiglottis and through the vocal cords. Squeeze the trigger-like handle of the Video RIFL to direct the tip of the instrument. Release the handle once the tip of the device passes through the vocal cords to facilitate advancement of the ET tube from the device. Advance the ET tube into the trachea. Withdraw the device and secure the ET tube. Confirm proper ET placement.

The Video RIFL can be used as a stand-alone device without the aid of a laryngoscope. Grasp the patient's mandible and tongue in the left hand and manually distract it. Insert the Video RIFL into the patient's mouth with the long axis of the device parallel to the palate. Look through the eye piece or at the LCD screen to visualize the airway structures. Flex the tip of the device to visualize the vocal cords. Advance the Video RIFL through the vocal cords. Complete the remainder of the procedure as described above.

AIR-VU PLUS FIBEROPTIC SCOPE

The Air-Vu Plus Fiber Optic Scope (Mercury Medical, Clearwater, FL) is an optical stylet designed specifically for aiding in fiberoptic ET tube placement through the Air-Q supraglottic airway (Mercury Medical, Clearwater, FL). It is a rigid device with a curved tip designed to fit the shape of the Air-Q (**Figure 21-3**). The Air-Vu Plus can be used with a compact LED light source or a standard green line fiberoptic laryngoscope handle. The device can be used as an optical stylet or as a stand-alone device like the Levitan Scope. The primary advantage of the Air-Vu Plus versus other devices is its compatibility with the Air-Q. The primary disadvantage is the rigidity of the device and the fixed curve of the shaft, both of which can make advancement of the device and the ET tube difficult.

SHIKANI OPTICAL STYLET

The Shikani Optical Stylet or S.O.S. (Clarus Medical, Minneapolis, MN) is unique among the optical stylets in that it comes in both pediatric and adult sizes (**Figure 21-4**). The S.O.S. can be used with a variety of light sources (e.g., its own specific handle, a green line laryngoscope handle, or a fiber optic cable attached to a remote light source). The S.O.S. is malleable and can be shaped into a 70° to 80° angle at the tip for use as a stand-alone device or a 30° angle when used as an adjunct to standard direct laryngoscopy. The S.O.S. can be attached to oxygen tubing to allow insufflation of oxygen at 5 to 10 L/min. Intubation is achieved like other optical stylets. Advantages of the S.O.S. include the availability of adult and pediatric sizes, its malleability, less dental trauma, and the option of providing oxygen insufflation.[3] There are several case reports of its successful use in children.[4,5] The disadvantages are primarily related to its length, which makes coordination of intubation difficult for some users.

FIGURE 21-3. The Air-Vu Plus Fiber Optic Scope. (Photo courtesy of Mercury Medical, Clearwater, FL.)

FIGURE 21-4. The Shikani Optical Stylet. (Photo courtesy of Clarus Medical, Minneapolis, MN.)

BONFILS RETROMOLAR INTUBATION ENDOSCOPE

The Bonfils Retromolar Intubation Endoscope (Karl Storz Endoscopy-America, El Segundo, CA) is simple in appearance (**Figure 21-5**).[6] It can accommodate a 6.5 mm or larger ET tube and has an adjustable tube stop. It can be equipped with either an eyepiece or an adapter to couple with a monitor. It allows for oxygen insufflation via a side port. The curvature at the tip of the scope is fixed.

Intubation with the Bonfils Retromolar Intubation Endoscope is like other optical stylets with or without the aid of a standard laryngoscope. In one study, 103 of 107 patients with unanticipated difficult airways were successfully intubated with this device, with 80% intubated without the aid of a laryngoscope.[7] The other 20% of patients were intubated with a Macintosh blade and the Bonfils scope. A study reports the device's success in the awake intubation of five patients with challenging airways.[8] It can be difficult to maneuver this device into proper position under the epiglottis despite having higher success rates than traditional direct laryngoscopy.[9]

BULLARD LARYNGOSCOPE

The Bullard laryngoscope (Gyrus ACMI, Southborough, MA) is a device that is a fusion product between video laryngoscopes and optical stylets (**Figure 21-6**). It is an old device that is being replaced by more advanced video laryngoscopes. It can still can be found in clinical practice. The Bullard laryngoscope provides superior views with less cervical spine movement than other laryngoscopes and video laryngoscopes.[10]

FIGURE 21-5. The Bonfils Retromolar Intubation Endoscope. (Photo courtesy of Karl Storz Endoscopy-America, El Segundo, CA.)

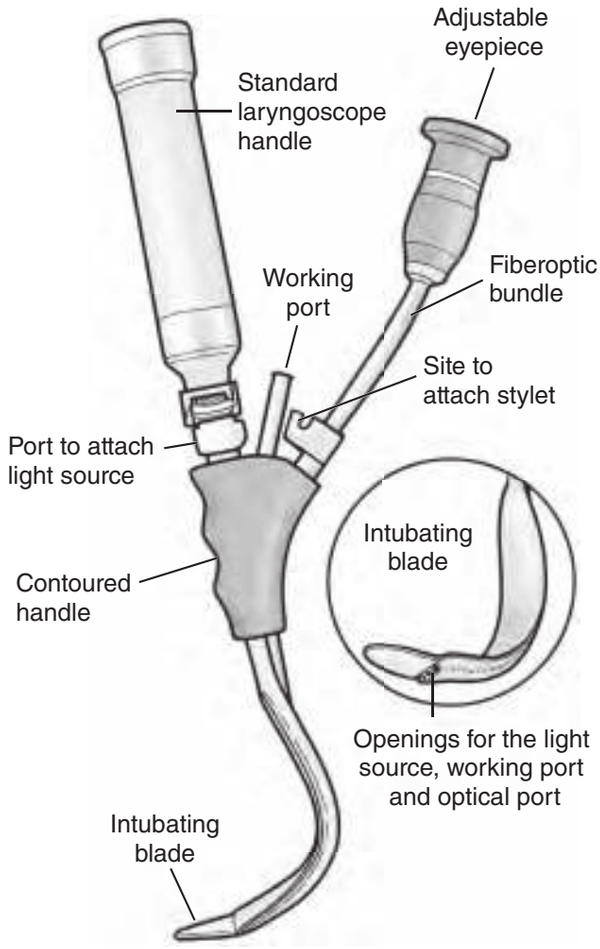

FIGURE 21-6. Anatomy of the Bullard laryngoscope.

FIGURE 21-7. Three versions of the Bullard laryngoscope. From left to right: the pediatric model, the pediatric long model, and the standard model.

The Bullard is a rigid laryngoscope that combines a curved blade with fiberoptic visualization into a simple and easy-to-use handheld unit (**Figures 21-6 and 21-7**). The proximal handle contains an 11 French or 3.7 mm working port, a port to attach the light source (e.g., traditional laryngoscope handle or a fiberoptic laryngoscope handle), and a visualization port (**Figure 21-7**). The third or working port allows oxygen insufflation, suctioning, administration of pharmaceuticals, or the passage of a guidewire to promote tracheal intubation.[11,12] The curved blade is similar in shape to a Macintosh blade. A fiberoptic bundle allows visualization of the vocal cords and tracheal intubation without a direct line of sight.

The Bullard laryngoscope is available in three sizes (**Figure 21-7**). The device is handheld, readily portable, self-contained, and operated as quickly as a traditional laryngoscope with a Macintosh blade. The ability to visualize the vocal cords without aligning the oral, pharyngeal, and laryngeal axes allows successful intubation with a minimum of cervical spine movement.[11-16]

PREPARATION

Assemble the Bullard laryngoscope when the possible need for airway intervention is recognized. The working port can be fitted with a three-way stopcock to provide intermittent suction and oxygen insufflation. Attach a traditional laryngoscope handle as the light source. A fiberoptic light source with the required adapter may be used to provide illumination. Lubricate the lower half of the intubating stylet with a water-soluble lubricant. Attach the stylet to the fiberoptic bundle, between the eyepiece and the handle, on the right side of the laryngoscope. Select and load the appropriate-size ET tube onto the intubating stylet. **The tip of the stylet should extend 0.5 cm past the distal end of the ~~ET~~ tube.** Place the tip of the stylet beneath the flange of the blade. It is recommended, but not required, that a disposable plastic blade extender be placed on the tip of the metal blade for adult intubations.

TECHNIQUE

Stand at the head of the bed. Open the patient's mouth to a minimum of 0.6 cm between the upper and lower incisors. Grasp the handle of the Bullard laryngoscope parallel to the patient and toward the patient's feet (**Figure 21-8*A***). Insert the blade into the midline of the mouth. Lift the laryngoscope handle upward (**Figure 21-8*B***). Slightly elevate the Bullard laryngoscope to lift the tongue (**Figure 21-8*C***). The blade will follow the contour of the tongue and pharynx with minimal effort.

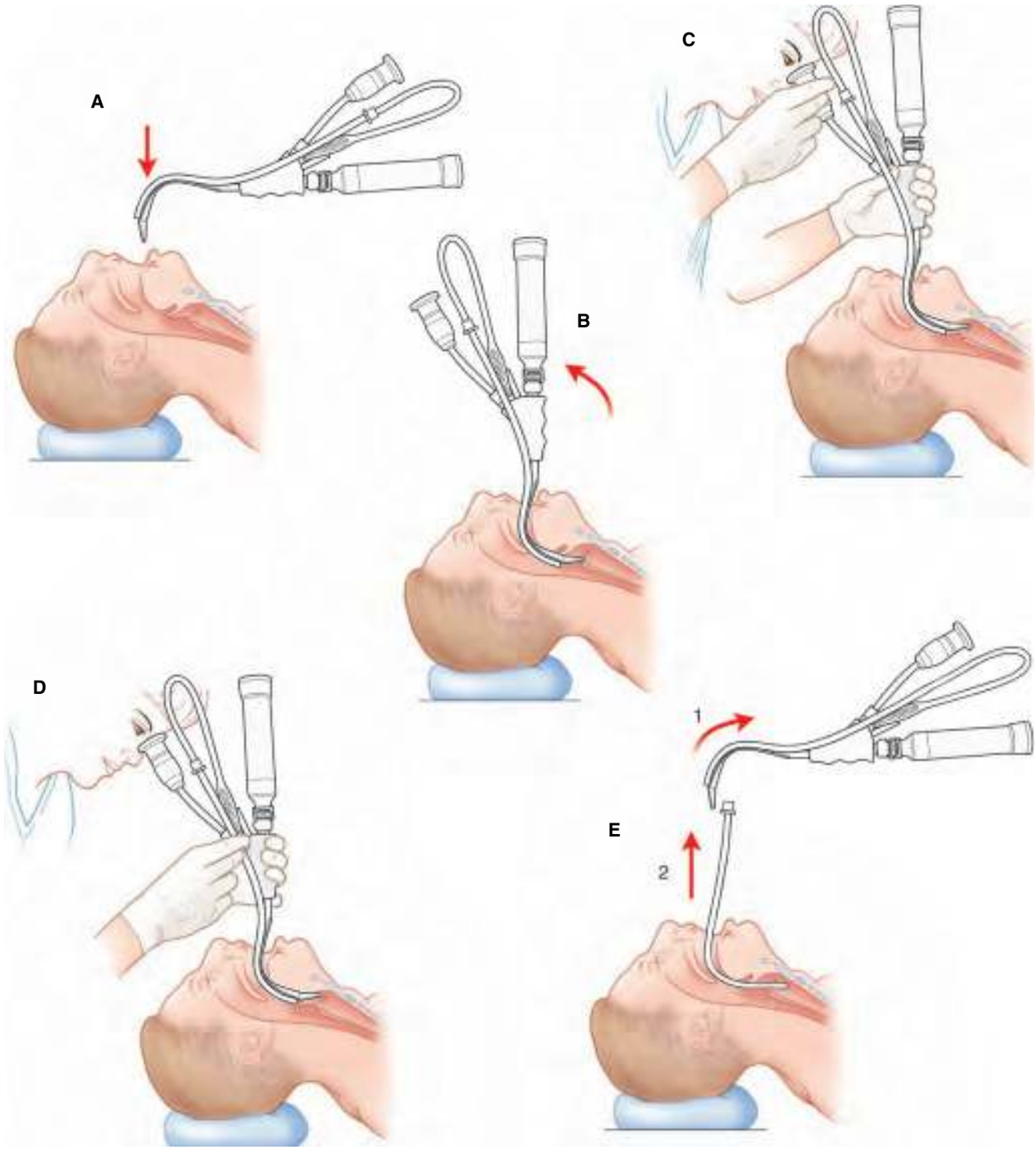

FIGURE 21-8. Intubating with the Bullard laryngoscope. ***A.*** Inserting the laryngoscope. ***B.*** Rotation of the handle 90° properly positions the laryngoscope. ***C.*** Slight elevation of the laryngoscope moves the tongue and epiglottis out of the visual axis. ***D.*** Advancement of the endotracheal tube under direct visualization. ***E.*** Removal of the Bullard laryngoscope. It is first rotated 90° toward the patient's feet (*curved arrow*), then lifted out of the mouth (*straight arrow*).

The blade of the Bullard laryngoscope will elevate the epiglottis (**Figures 21-8*C* and *D***). Visualize the airway through the fiberoptic port (**Figure 21-8*D***). The view can be focused by turning the eyepiece. The blade can be repositioned by backing it out toward the posterior pharynx to catch the epiglottis if the blade is not beneath the epiglottis. Suction through the working port if blood, debris, or secretions limit the view. Advance the ET tube under direct visualization when the vocal cords are visualized (**Figure 21-8*D***). Secure the ET tube at the patient's teeth with the nondominant hand. Remove the Bullard laryngoscope with the dominant hand by reversing the technique of insertion (**Figure 21-8*E***).

The Bullard laryngoscope allows for other strategies to aid in ET intubation. The stylet has a central opening of 4.5 mm in the adult model and 3.6 mm in the pediatric long model, which can be used to pass an intubating guidewire through the vocal cords.[12] An intubating guidewire may be passed through the working port and through the vocal cords. In either case, insert the guidewire, remove the Bullard laryngoscope, and pass the ET tube over the guidewire

and into the trachea. It is possible to pass an ET tube with a standard malleable stylet. The styletted ET tube can be bent into a shape approximating that of the Bullard laryngoscope.

Problems specific to the Bullard laryngoscope include the inability to visualize the vocal cords. Confirm that the laryngoscope is in midline. Reposition it by moving the blade into the posterior pharynx and attempt to capture the epiglottis if the blade is above the epiglottis and the view obscured. Use a disposable plastic blade extender to avoid this difficulty. Suction through the working port if the view is obscured by debris. Visualize the stylet with the loaded ET tube through the scope. It may have slipped underneath the blade if not visualized. Reposition the stylet and ET tube without removing the laryngoscope.[12] Extension of the stylet too far beyond the ET tube may cause abrasions, bleeding, and lacerations to the walls of the oral cavity, oropharynx, and laryngopharynx. These can be prevented with proper assembly of the Bullard laryngoscope.

OTHER DEVICES

Other fiberoptic devices are available (**Table 21-1** and **Figures 21-9 through 21-11**). These devices are used like the above devices. They also are not used as often as video laryngoscopes.

COMPLICATIONS

Most of the complications associated with fiberoptic devices mirror those of ET intubation (Chapter 18). These include failure to intubate, esophageal intubation, right mainstem bronchus intubation, and the hemodynamic consequences of intubation. The most common difficulty encountered in ET tube passage is impaction of the ET tube on the right arytenoid cartilage. This obstacle can be overcome by directing the device slightly toward the patient's left side. Alternatively, rotate the ET tube until the bevel is facing the viewing channel.

VIDEO LARYNGOSCOPES

Video laryngoscopes have received wide acceptance in clinical practice over the past 8 to 10 years (Chapter 20). They allow relatively stress-free intubation on challenging airways by even fairly inexperienced clinical personal. Many of these devices allow recording capabilities that might be important due to the increased malpractice litigations related to substandard airway management. They are becoming more and more common. These devices are considered the next-generation scopes that followed the optical stylets and the

FIGURE 21-9. The Acutronic Sensascope. (Photo courtesy of Acutronic Medical Systems AG, Switzerland.)

FIGURE 21-10. The AincA Video Stylet. (Photo courtesy of Anesthesia Associates, San Marcos, CA.)

FIGURE 21-11. The Clarus Pocketscope. (Photo courtesy of Clarus Medical LLC, Minneapolis, MN.)

Bullard. They do not incorporate a stylet as part of their integral design. Video laryngoscopes may fall under standard blade styles, angulated blade styles, and anatomically shaped channeled design.[17]

The design allows the video laryngoscope to be inserted in the midline without sweeping the tongue laterally. Video laryngoscopes allow the Emergency Physician an option of using them as a direct laryngoscope without the video display. The benefits of these devices over standard laryngoscopes are limited, but they present good teaching capabilities since several providers can observe the patient's airway and ET tube placement.[17] Video laryngoscopes that have reusable blades are usually more difficult to maintain than the conventional direct laryngoscopes. Their maintenance requires extra time and costs that cannot always be justified. The user essentially pays "per use" with disposable blades, which makes them expensive. These extra costs may be justified in anticipated difficult airway cases.

Video laryngoscopes are largely very safe. Injury to airway structures is due to blind insertion of ET tubes with visualization on the screen only, in other words blind insertion. There is a blind spot from the time the tube is orally inserted until the tip is visualized on the screen. This seems to be the most vulnerable time for injury. This can largely be avoided by what has been described the "look up–look down" approach.[17] The basic premise is to directly visualize the scope and ET tube (i.e., look down) enter the oral cavity, indirectly visualizing the vocal cords and the entry of the ET tube into the glottic opening (i.e., look up). Another mechanism of airway injury has been attributed to the stylet itself. This can be partially avoided by withdrawing the stylet soon after entering the glottis and before advancing the ET tube further.

SUMMARY

A variety of fiberoptic devices are available to help indirectly visualize the vocal cords and aid in intubating the trachea. The devices have a variety of features including malleable stylets, articulating tips, and differing lengths. The devices can be shaped for differing applications (e.g., used with and without a laryngoscope or to be inserted through a supraglottic airway). The devices feature different visualization capabilities (e.g., fixed eyepiece or optional video screen). Fiberoptic devices offer alternative techniques for managing the airway. Every Emergency Department should have at least one of these devices available to aid in intubation when direct laryngoscopy is difficult or fails.

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22 Endotracheal Tube Intubating Introducers and Bougies

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INTRODUCTION

The endotracheal tube introducer (also known as an introducer) and a tracheal tube bougie (also known as the bougie) have been effective, easy-to-use, and inexpensive adjuncts to difficult airway management for many years (**Figure 22-1**).[1] Although newer devices such as the fiberoptic bronchoscopes and the video laryngoscopes are becoming more frequently used in the Emergency Department (ED) in difficult airway situations, skills pertaining to bougie insertion should be developed and maintained.[2] They can be valuable if other intubating modalities are not readily available or the airway cannot be secured with direct laryngoscopy. It is recommended that a bougie be readily available in every ED.

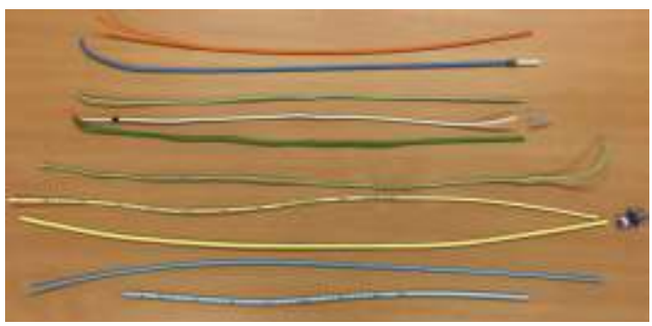


FIGURE 22-1. Tracheal tube introducers and bougies. From top to bottom: Eschmann introducer, Frova intubating introducer, Muallem endotracheal tube stylet, S-guide with universal connector, Muallem endotracheal tube introducer, Rivier airway introducer, Cook airway exchange introducer 14 French, Cook airway exchange introducer 8 French, Cook tube exchanger, and Aintree intubation catheter. (Used with permission from reference 1.)

Airway management in the ED often occurs in an unpredictable and uncontrolled environment, sometimes with the patient arriving unannounced.[3] Difficult intubation should be always anticipated and prepared for.[3] The American Society of Anesthesiology defines a difficult intubation as an inability to properly insert an endotracheal tube with traditional direct laryngoscopy in three attempts or if intubation takes longer than 10 minutes.[4] Difficult intubations usually reflect poor glottic visualization during direct laryngoscopy. A four-grade classification system by Cormack and Lehane describes the views of the laryngeal inlet during laryngoscopy.[5] The exact incidence of difficult-to-intubate patients in the ED is difficult to extrapolate, with estimates ranging from 6% to 11%.[6,7]

Difficulties arise when the vocal cords cannot be fully visualized due to airway distortion (e.g., edema, expanding hematomas, radiation, surgery, or trauma), airway masses, anatomic variations, limited neck mobility due to cervical spine fractures and cervical collars, deformities of the head and neck, orofacial injuries, or oropharyngeal blood and secretions. One study reported that the vocal cords could not be visualized in 22% of patients wearing a cervical collar.[8] **This failure to visualize the glottis can make intubation difficult or impossible. The intubating introducer, tracheal tube introducer, or bougie can be a good rescue device in these situations.** The main advantage of these devices is the angled or coudé tip that can be aimed anteriorly and advanced under the epiglottis and into the trachea. Intubation with one of these devices was first described by MacIntosh in 1949.[9] The device he used was a 60 cm long, 15 French, elastic catheter with a J or coudé tip that was bent 40° at the distal end.

The bougie or introducer is used to facilitate difficult intubation. It is a thin, long, cylindrical rod composed of rubber or plastic. Despite sometimes being called "gum elastic bougies," they are not composed of gum, not composed of elastic, and not true bougies (i.e., dilators).[10] They are inexpensive, with most costing less than $20.00 each. They are available in numerous adult and pediatric sizes, various degrees of flexibility, and are with a variety of ports and/or devices. They are flexible enough to allow an endotracheal tube to freely pass over them or to be inserted through a supraglottic airway device, yet stiff enough to be easily maneuvered and advanced into the airway.

The bougie or introducer should not be confused with the more rigid malleable stylet, which is inserted into an endotracheal tube and used to alter its shape prior to intubation. Unlike the stylet, a bougie or introducer is usually inserted independently of the endotracheal tube and is used as a guide. The bougie is considerably softer, more malleable, and blunter than a stylet. The insertion of a bougie is a relatively atraumatic procedure.[11]

This chapter reviews the general principles for using tracheal tube introducers, intubating introducers, and bougies and reviews some of the more commonly available devices. The terms *tracheal tube introducers*, *intubating introducers*, and *bougies* are often used interchangeably. The remainder of this chapter uses the general term *bougie* unless some other term is specific to a manufacturer's device.

INDICATIONS

The bougie is intended to facilitate endotracheal intubation in patients when the epiglottis is visible but visualization of the glottis is difficult or inadequate (i.e., grade 3 Cormack-Lehane view) despite external laryngeal manipulation and optimal patient positioning.[12] The most frequent indication for the use of a bougie is the inability to intubate endotracheally using traditional direct laryngoscopy. It can also be used for "routine intubations." The narrower and more flexible bougie, compared to an endotracheal tube, can easily be inserted into the trachea when the glottis is visualized during direct laryngoscopy and the endotracheal tube is advanced over the bougie. A bougie can be inserted directly into the trachea or through a supraglottic airway device to facilitate endotracheal intubation. A bougie may be inserted when the glottic opening is visible but the endotracheal tube will not pass through the vocal cords. The bougie serves as a placeholder in these cases and avoids the need to remove the laryngoscope and re-perform direct laryngoscopy when the bougie is available. The use of a bougie to intubate the trauma patient may result in less cervical spine motion than traditional direct laryngoscopy.[13] Long bougies, at least 70 cm, can also be used as an endotracheal tube exchanger.

CONTRAINDICATIONS

There are no absolute contraindications to the use of a bougie. Bougies should be used with caution, if at all, in patients with airway trauma because they can result in additional injury and hemorrhage to the airway structures, perforation (e.g., piriform fossa, trachea, bronchus, or esophagus), or barotrauma.[14-16] Bougies are unlikely to be beneficial when no part of the airway is visible (i.e., grade 4 Cormack-Lehane view) and are relatively contraindicated. Another relative contraindication is its use in children. The lack of calcification and mineralization of the tracheal rings makes it difficult to sometimes differentiate between the trachea and esophagus.

EQUIPMENT

- Endotracheal tubes, various sizes
- 10 mL syringe
- Water-soluble lubricant or anesthetic jelly
- Wire stylet, malleable type
- Laryngoscope handle
- Fresh batteries for the laryngoscope
- Laryngoscope blades, various sizes and shapes
- Supplemental oxygen with appropriate tubing and connectors
- Nonrebreather oxygen masks, various sizes
- Wall suction with appropriate tubing
- Yankauer suction catheter
- Bag-valve device, various sizes
- Oral airways, various sizes
- Nasal airways, various sizes
- Benzoin adhesive
- Tape
- Pulse oximeter
- Cardiac monitor
- Automatic sphygmomanometer
- End-tidal carbon dioxide (CO_2) monitor or device
- Cricothyrotomy backup tray
- Crash cart
- Resuscitation medicines
- Personnel (e.g., respiratory technician, medication nurse, in-line stabilization assistant, recorder)
- Medications (i.e., premedications, induction, anesthetics, paralytics)
- Endotracheal tube introducer or bougie

Some of the more commonly used devices in the ED are described below. Other commercially available bougies also exist (**Table 22-1**).

TABLE 22-1 Some of the Commercially Available Intubating Introducers and Bougies

Name	Manufacturer	Length(s) (cm)	Size(s) (French)	Comments
Aintree Intubation Catheter	Cook Medical	56	19	Hollow with two holes at distal end Allows passage of fiberoptic bronchoscope Rapi-Fit adapters allow bag-valve and jet ventilation Allows ventilation while exchanging endotracheal tube (ETT) and supraglottic airway (SGA)
Arndt Airway Exchange Catheter	Cook Medical	50 65 78	8 14	Hollow with multiple side holes at distal end Allows passage of fiberoptic bronchoscope Rapi-Fit adapters allow bag-valve and jet ventilation Allows ventilation while exchanging ETT and SGA
Cobra Introducer	Occam Design	60 (telescopes to 73)	15	Straight tip Fits ETT sizes 6–11
Cobralet	Occam Design	60	15	Coudé tip Hollow with side holes at distal end Allows ventilation Fits ETT sizes 6–11
Cook Airway Exchange Catheter	Cook Medical	43 83 100	8 11 14 19	ETT exchanger but can be used as bougie Rapi-Fit adapters allow bag-valve and jet ventilation Soft-tip model minimizes trauma to airway
CoPilot VL Single-Use Bougie	Magaw Medical	60	14	Coudé tip Fits ETT ≥ 6.0 mm inner diameter
Frova Intubating Introducer	Cook Medical	35 65	8 14	Angled tip Fits ETT ≥ 3.0 mm inner diameter Hollow with side holes at distal end Comes with metal stiffening cannula Rapi-Fit adapters allow bag-valve and jet ventilation
Introes Pocket Bougie	BOMimed	60	14	Malleable Coudé tip Comes curved with shape memory Fits ETT ≥ 5.0 mm inner diameter
Muallem ET Tube Stylet	VBM Medizintechnik	40 65	8 12 14	Atraumatic soft coudé tip
Pocket Introducer	VBM Medizintechnik	65	15	Folded to 20 cm to save space Unfolds fast
Portex Venn Tracheal Tube Introducer	Smith's Medical	60	15	Coudé tip Sterilizable and reusable
Rhinoguard	Davis Medical	25.4 35.5	N/A	Designed for nasal intubation Can be used as a bougie Fits sizes 3–8 ETT
Single-Use Bougie	Smith's Medical	70	15	Coudé tip Hollow allows bag-valve and jet ventilation
VBM Introducer	VBM Medizintechnik	65	15	Coudé tip Hollow allows bag-valve and jet ventilation
VBM S-guide	VBM Medizintechnik	65	15	Atraumatic coudé tip Hollow allows bag-valve and jet ventilation
VBM Tube Exchanger	VBM Medizintechnik	80	11 14 19	ETT exchanger but can be used as bougie Hollow allows oxygenation

ESCHMANN TRACHEAL TUBE INTRODUCER

The Eschmann tracheal tube introducer was formerly known as the gum elastic bougie. Reusable, or multiple-use, and single-use disposable versions are available. The Eschmann multiple-use tracheal tube introducer was introduced into clinical practice in 1973. The Portex single-use introducer (Smiths-Medical, Dublin, OH) became available in 1997 and replaced the Eschmann Introducer. It is a 60 cm long, 15 French, flexible device with a J-angle at its distal tip (**Figure 22-2**). During use, the multiple-use introducer forms a curve toward its distal end. The single-use introducer is more rigid and is thus more likely to cause trauma.[17] It does not maintain a curved shape when bent, and it has a significantly lower tracheal placement rate in simulated grade 3 Cormack-Lehane views.[18] The multiple-use device retained the curved shape for longer compared with the single-use device.

FROVA INTUBATING INTRODUCER

The Frova Intubating Introducer (Cook Medical Inc., Bloomington, IN) was introduced into clinical practice in 1998. The Frova is a single-use device able to maintain the desired curvature—a feature shared with the Eschmann multiple-use introducer.[19] It is a flexible, hollow, radiopaque polyethylene plastic catheter with centimeter markings and a blunt curved tip (**Figure 22-3**). It is available in two sizes, the 14 French version for 6.0 mm and larger endotracheal

FIGURE 22-2. The Portex Tracheal Tube Introducer. (Photo courtesy of Smiths Medical, Dublin, OH.)

tubes and the 8 French version for 3.0 mm and larger endotracheal tubes. It is packaged with a removable metal cannula that stiffens the device except for the distal tip. The advantage of the metal cannula is that it can be bent to change the shape of the Frova and make it easier to advance. The disadvantages of the metal cannula are that

FIGURE 22-3. The Frova Intubating Introducer with Rapi-Fit adapter. From left to right: the proximal end with the Rapi-Fit adapter, the body with centimeter markings, and the distal end. (Photo courtesy of Cook Medical Inc., Bloomington, IN.)

FIGURE 22-4. The SunMed Bougie. From top to bottom: 10 French coudé tip pediatric version, 15 French straight tip adult version, and the 15 French coudé tip adult version. (Photo courtesy of SunMed, Largo, FL.)

it makes the Frova more rigid and harder to maneuver and potentially increases airway soft tissue trauma. Products with the suffix "-FII" also include a stiffening cannula and two Rapi-Fit adapters for connection to a ventilatory device. Products with the suffix "-FI" include two Rapi-Fit adapters for connection to a ventilatory device, but no stiffening cannula. The adapter can be used to ventilate the patient and confirm proper tracheal placement before advancing the endotracheal tube. It takes only 15 seconds to attach an aspirating esophageal detector device to the adapter and confirm its position.[20]

SUNMED BOUGIE

The SunMed Bougie (SunMed, Largo, FL) is made of a blend of low- and high-density polyethylene for optimal stiffness and has depth calibration markings. It is 70 cm long and available as a 10 French pediatric size or a 15 French adult size (**Figure 22-4**). Three versions of these devices are available: the original, the malleable, and the ported version. The original adult version is available with a straight or curved tip and fits 4.0 to 11.0 mm endotracheal tubes. The original pediatric version is only available with a curved tip. The malleable version has a color-coded stopper and fits 6.0 to 11.0 mm endotracheal tubes. The ported version allows for the insufflation of oxygen and gas sampling. It is available only in the 15 French size. It uses a blend of low- and medium-density polyethylene for optimum firmness, and its depth is calibrated in centimeters. A study comparing the SunMed, Portex, Greenfield, and Eschmann bougies demonstrated that Emergency Physicians had better success rates using the SunMed and Greenfield devices, but they had a low preference for the Greenfield bougie.[21]

GREENFIELD FLEX-GUIDE ET TUBE INTRODUCER

The Flex-Guide ET Tube Introducer (Greenfield Medical Sourcing Inc., Austin, TX) is a 60 cm long polyethylene tube that is available in a 15 French (i.e., 5.0 mm) diameter for adults and a 10 French (i.e., 3.3 mm) diameter for children (**Figure 22-5**). The ends of the Flex-Guide are smooth and rounded with a bend 2 cm from the distal end, resulting in a 30° coudé tip. The tubing is flexible and can be straightened or bent as required. The adult Flex-Guide has a black band marking 37 cm from its proximal end (**Figure 22-5**) to help determine correct placement depth. The pediatric Flex-Guide has markings every 10 cm (**Figure 22-5**). The Flex-Guide is similar to the Eschmann or Portex bougie, only more economical. This device has been used in the ED to intubate difficult airways.[22]

FIGURE 22-5. The Greenfield Flex-Guide Endotracheal Tube Introducer adult version (above) and pediatric version (below). (Photo courtesy of Greenfield Medical Sourcing Inc., Austin, TX.)

FIGURE 22-6. The Aintree Intubation Catheter. From left to right: the proximal end with the Rapi-Fit adapter, the body with centimeter markings, and the distal end. (Photo courtesy of Cook Medical Inc., Bloomington, IN.)

AINTREE INTUBATION CATHETER

The Aintree Intubation Catheter (Cook Medical Inc., Bloomington, IN) was designed to be used with a fiberoptic bronchoscope (**Figure 22-6**). It is packaged with a Rapi-Fit Adapter that allows ventilation through the device and air exchange through the multiple distal side ports. The Aintree is only available in one size and for use with endotracheal tubes greater than 7.0 mm in diameter. It may also be used as a bougie without the fiberoptic bronchoscope. It is generally not used as a bougie due to it being hollow, flexible, straight, and lacking the flexed tip of a typical bougie.

POCKET BOUGIES

These are single-use catheters that can be folded and placed in an emergency bag. The Introes Pocket Bougie (BOMImed, Winnipeg, Manitoba, Canada) is a 60 cm long malleable bougie. It is made of a special blend of Teflon to balance the rigidity and offer soft tissue protection (**Figure 22-7**). The diameter of 14 French (i.e., 4.7 mm) accommodates size 5.0 endotracheal tubes and greater. It can be used with direct laryngoscopy or video laryngoscopy. It has a unique curvature that follows the natural airway. Its flexibility allows manipulation of the distal coudé tip for anterior airways. The Tactiglide technology facilitates tactile sensation.

Another pocket introducer is manufactured by VBM Medizintechnik in Germany. It is 65 cm long and 15 French in diameter with a coudé tip. It can be folded to only 20 cm and unfolds to 65 cm within seconds.[23]

ENDOTRACHEAL TUBE EXCHANGERS

The use of an endotracheal tube exchanger to facilitate orotracheal intubation similar to a bougie is not recommended. These devices

FIGURE 22-7. The Introes Pocket Bougie.

are straight, very flexible, thin-walled, and hollow tubes. Their flexibility makes it difficult for them to be advanced into the trachea and they more often enter the esophagus. These thin-walled hollow tubes become even more flexible when inserted and warmed by the patient's body temperature.

PATIENT PREPARATION

The patient preparation is the same as that for orotracheal intubation (Chapter 18). The patient should be appropriately monitored with electrocardiography (ECG), end-tidal CO_2 monitoring, noninvasive blood pressure cuff, and pulse oximetry. As for any situation where airway manipulation is to occur and the patient's protective airway reflexes are blunted or ablated, a fully functioning suction apparatus with a variety of catheters must be immediately available. Place the patient supine with their head in a neutral position.

Place the patient, with a normal neck, in the "sniffing" position, with their head extended at the atlantooccipital joint while the neck is relatively flexed. A folded towel under the occiput helps to gently raise and tilt the head back into the proper position in adults. An appropriate-size shoulder roll should be placed in pediatric patients. **Correct positioning is probably the most important preparation of the patient.** Dentures should be left in place temporarily, as they help to stabilize the mouth and prevent occlusion during preoxygenation and bag-valve-mask ventilation.

TECHNIQUES

OROTRACHEAL INTUBATION

The bougie can be used in anticipation of a difficult airway or when a difficult airway is encountered and direct laryngoscopy is not successful. Instruct an assistant to ventilate the patient with a bag-valve-mask device. Liberally lubricate the bougie with water-soluble lubricant if there is a tight fit of the endotracheal tube because it allows the latter to slide easily over the bougie. If there is no tight fit, the lubricant can make the bougie difficult to grasp and control. A silicone spray, if available, may be used instead of the water-soluble lubricant. Insert the laryngoscope loaded with either a Macintosh or Miller blade. Elevate the laryngoscope and attempt to visualize the

epiglottis and vocal cords. Insert the bougie through the vocal cords and into the trachea if the vocal cords are visualized in part or completely. **Care should be taken to keep the tip of the bougie pointing anteriorly and in the midline. The tracheal rings may not be appreciated if the bougie rotates significantly. Forceful insertion must be avoided as this can result in an iatrogenic airway injury such as bleeding and perforation.**

Attempt to pass the bougie under the epiglottis with the curved distal tip facing anteriorly if the vocal cords are not visualized (**Figures 22-8*A* and *B***). **Slowly and carefully advance the bougie distally toward the trachea.** The bougie will slightly jump or bounce as its tip is advanced over each tracheal ring (**Figure 22-8*C***). This is sometimes referred to as the "palpation of a click" as the tip passes over each tracheal ring. Advance the bougie approximately 10 to 15 cm into the trachea (**Figure 22-8*D***). Securely hold the bougie in place. **Do not remove the laryngoscope after the bougie is placed within the trachea. The laryngoscope will continue to elevate the tongue and allow easier passage of the endotracheal tube.**

There are several signs that signify proper bougie placement within the trachea. If the bougie is unable to be advanced more than 40 cm from the patient's lips in an adolescent or adult, or more than 24 cm in the child, it is likely caught at the carina or bronchus as the airway narrows. This is sometimes referred to as the "hold-up" sign. The "hold-up" sign in addition to the palpation of "clicks" as it is inserted confirms proper tracheal positioning of the bougie.[24] If the bougie freely advances more than 40 cm from the adolescent

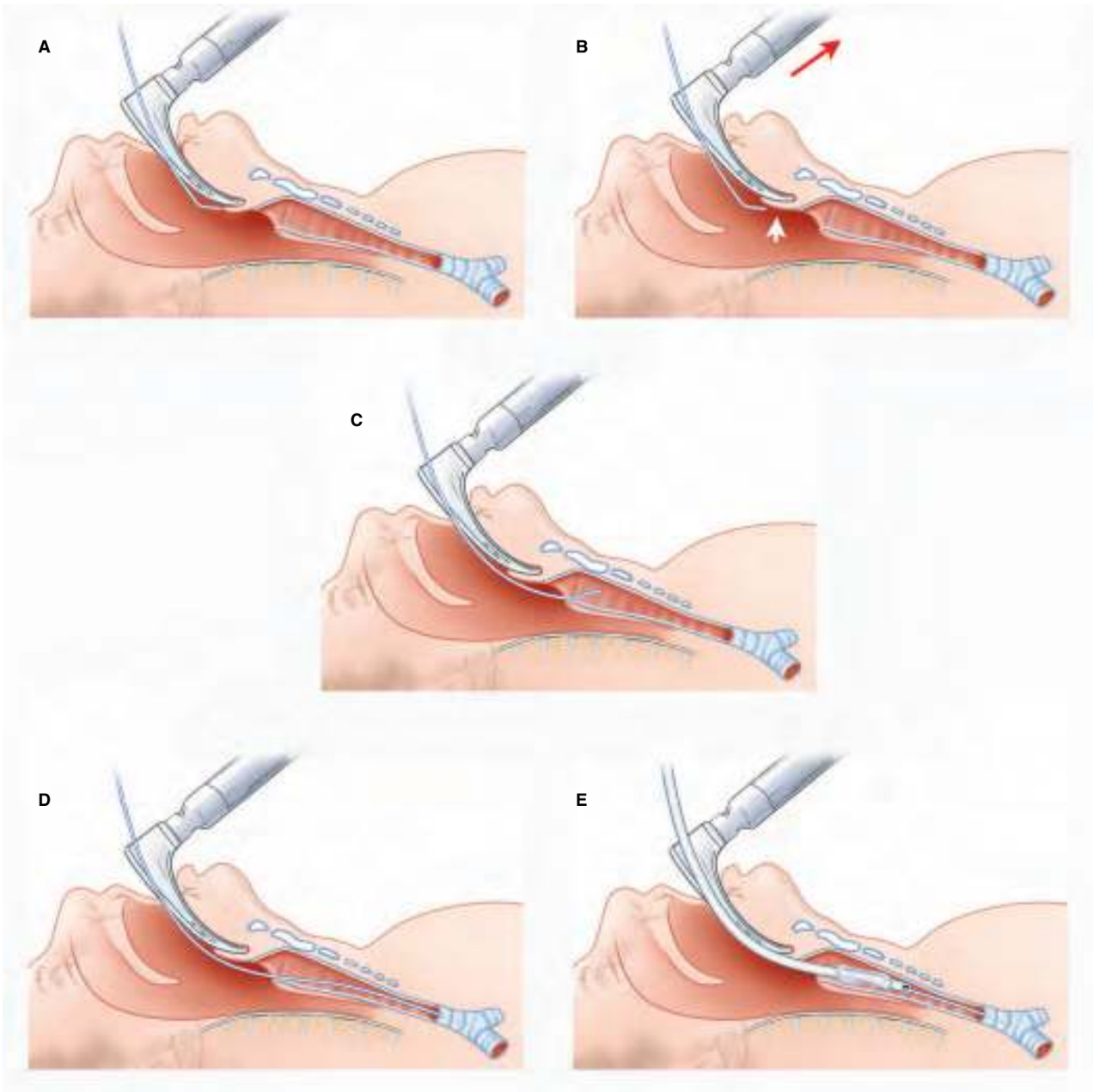


FIGURE 22-8. Intubation using a bougie. ***A.*** The laryngoscope has been inserted and the bougie advanced into the hypopharynx. ***B.*** The bougie is advanced under the epiglottis. ***C.*** The bougie is inserted into the trachea and advanced over the tracheal rings. ***D.*** The bougie is advanced until its tip is at the carina or mainstem bronchus. ***E.*** The endotracheal tube is advanced over the bougie and into the trachea.

or adult patient's lips, or 24 cm in the child, it is most likely in the esophagus or stomach. One study noted the bougie getting caught and the palpation of "clicks" to be a less reliable indicators of tracheal placement than in some other studies.[25] A 2014 study recommends against using the hold-up sign with single-use bougies.[26] The same study cautions clinicians when trying to elicit this sign with reusable bougies.[26]

An assistant performing the Sellick's maneuver should feel the tip of the bougie passing under their fingertips as it is advanced down the trachea. The bougie will rotate as the tip passes the carina and enters a mainstem bronchus. It will rotate clockwise if it enters the right mainstem bronchus and counterclockwise if it enters the left mainstem bronchus. Correct positioning can be checked by using an end-tidal CO_2 monitor or detector attached to the Rapi-Fit adaptor of the bougie.

Instruct an assistant to load the endotracheal tube over the bougie and slide it to the level of the patient's lips. **The assistant should hold and advance the endotracheal tube until its tip is at the patient's lips while the Emergency Physician controls the bougie, controls the laryngoscope, and provides instruction to the assistant. The Emergency Physician must now grasp and control the endotracheal tube with their right hand while the assistant holds the bougie securely in place.** Advance the endotracheal tube over the bougie and into the trachea (**Figure 22-8*E***). Rotate the endotracheal tube 90° counterclockwise under direct visualization so that the bevel is facing posteriorly as the endotracheal tube passes through the vocal cords. This maneuver allows the endotracheal tube bevel to not get caught on the arytenoid cartilage and to gently spread the arytenoids with a minimum of force, thus avoiding any injury to the vocal cords.[27] **Do not try to advance the endotracheal tube if resistance is encountered.** Instruct the assistant to slightly withdraw the endotracheal tube, rotate it in a slightly more counterclockwise direction, and re-advance it again.

Hold the endotracheal tube firmly in place. Remove the laryngoscope. Remove the bougie. Secure the endotracheal tube, confirm its proper position, and begin ventilation of the patient.

Little training is needed before successful intubation using a bougie. This may be due to the similarity to the standard intubating technique and the familiarity with the Seldinger technique that is used during the intubation process. An observational prehospital study showed that bougies were used to successfully intubate 78% of difficult airways that could not be managed by standard techniques.[28]

ALTERNATIVE OROTRACHEAL INTUBATION TECHNIQUE

An assistant is not always available to load the endotracheal tube and advance it over the bougie. Likewise, an airway may turn out to be difficult when it is not anticipated. An alternative is for the Emergency Physician to preload the endotracheal tube on the bougie (**Figure 22-9**) and insert them as a unit.[29-31] Load the endotracheal tube over the bougie so that the bougie projects approximately 10 cm from the distal end of the endotracheal tube (**Figure 22-9*A***). Form a loop with the endotracheal tube and proximal bougie and grasp it with the right hand (**Figure 22-9*A***). A second technique is to load the endotracheal tube on the bougie so that the bougie projects approximately 6 to 10 cm from the distal end of the endotracheal tube and grasp it with the right hand (**Figure 22-9*B***).

Insert the laryngoscope into the patient's mouth and attempt to visualize the glottis. Grasp the bougie loaded with the endotracheal tube in the right hand. Insert the distal end of the bougie through the vocal cords and into the trachea. Remove the laryngoscope. Advance the endotracheal tube over the bougie and into the trachea. Securely hold the endotracheal tube. Remove the bougie. Secure the endotracheal tube, confirm its proper position, and begin ventilation of the patient.

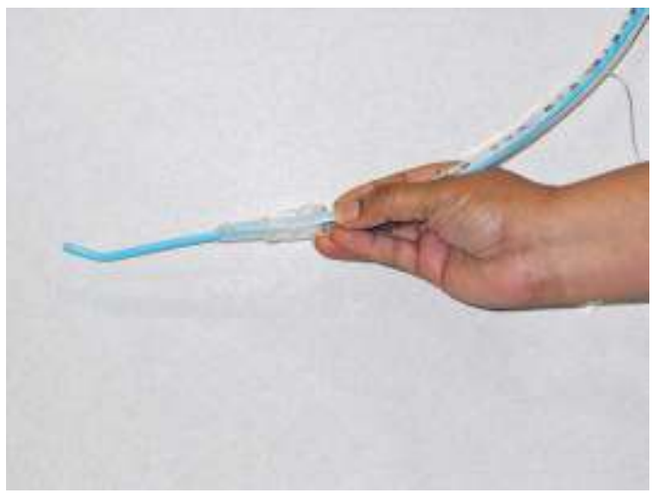
BOUGIE-GUIDED SUPRAGLOTTIC AIRWAY INSERTION

Bougies can be used to guide supraglottic airway devices.[32-34] A bougie was used as a guide through the ProSeal LMA.[32,35-37] The esophageal channel of the laryngeal mask airway (LMA) was used to load the bougie and place it in the esophagus. This resulted in a better success rate than using the LMA introducer tool. The bougie-guided insertion caused less trauma than the LMA insertion tool after failed digital insertion of the ProSeal LMA. A similar technique has been used in the pediatric population.[33] Other studies have used bougies as guides through a supralaryngeal device for rescue intubation.[34,38]

BOUGIE-ASSISTED RETROGRADE INTUBATION

Retrograde intubation with a bougie may be considered as an emergent airway management option.[39] A patient was successfully intubated using a modified retrograde technique through

A



B

FIGURE 22-9. Two methods to preload an endotracheal tube onto a bougie. ***A.*** The curved hold. ***B.*** The straight hold.

a tracheal defect with a bougie. The bougie is often more conveniently available than a retrograde guidewire kit. A bougie can be inserted through the skin and airway incision, laceration, or defect and advanced superiorly to exit the patient's mouth. The endotracheal tube can then be loaded over the bougie and advanced into the trachea. Refer to Chapter 30 for the complete details of retrograde intubation.

BOUGIE-ASSISTED CRICOTHYROIDOTOMY

Various medical and surgical specialties have used the bougie as an aid when performing a cricothyroidotomy.[40-42] Using a bougie as an aid was found to be easier and faster to teach how to perform a cricothyroidotomy in an animal lab than a traditional cricothyroidotomy.[43] The military describes this as the preferred technique of combat airway management.[44] The three-step technique using the bougie can be quickly taught to combat medics and performed in complete darkness using night vision goggles. A bougie placed through the incision maintains the open tract, provides tactile confirmation of tracheal placement, and serves as a guide for the endotracheal tube insertion.

ALTERNATIVE USES

Various other applications of the bougie have been described.[45-56] The bougie can be used as an adjunct in nasal intubation in the adult and pediatric populations.[45,46,49] The bougie has been successfully used to aid in correct placement of a double-lumen airway tube in a difficult airway.[47] It has also been described and tested as a back-up device during extubation to allow for a quick reintubation in case of the need to resecure the airway.[48] A bougie can be used as an endotracheal tube exchanger, which is performed mainly in the Operating Room or Intensive Care Unit if direct visualization of the vocal cords by a repeated direct laryngoscopy is difficult to achieve. Bougies can be used to facilitate intubation in conjunction with a video laryngoscope or a fiberoptic bronchoscope.[50,51] It can also be used as an adjunct with blind digital intubation.[52] A bougie can be used during chest compressions of cardiopulmonary resuscitation without stopping.[53,54] A bougie can be used to intubate and ventilate one lung in cases of severe hemoptysis.[55] Finally, the bougie has been inserted through an endotracheal tube to confirm its proper placement.[56]

COMPLICATIONS

There are some complications specific to the use of a bougie. The bougie can cause trauma to the airway soft tissues.[14-16,57-59] The Frova causes significantly more complications than other bougies (5% incidence versus rare).[57] This difference is most likely due to using the metal cannula, making the Frova quite stiff. The Frova with the metal cannula exerts more force on the airway tissues than other bougies.[19,58] Using the Frova without the metal cannula will reduce the incidence of tissue trauma. Single-use bougies may be more prone to cause soft tissue trauma when compared to multiple-use bougies, especially if they are held close to the distal end.[17,58] A false passage can be created by the tip of the bougie penetrating the soft tissues.[14] Insertion of an endotracheal tube over the bougie and into a false passage can increase the amount of soft tissue damage and hemorrhage. Insufflation of air into the surrounding soft tissues can compress the trachea and cause an external airway obstruction. A bougie can perforate the airway and result in a tension pneumothorax.[60] Advancement of the bougie into the trachea or a mainstem bronchus can induce bronchospasm.[57] The remainder of the complications are similar to those of orotracheal intubation (Chapter 18).

SUMMARY

Bougies are widely used to aid in intubation with traditional direct laryngoscopy when the glottic opening cannot be adequately visualized. They have been successfully used both in the adult and pediatric populations, with devices available for use with endotracheal tubes as small as 3.0 mm in size. They are also used to aid in placement of supralaryngeal devices, blind nasotracheal intubations, placement of double-lumen airway tubes, retrograde intubation, cricothyroidotomies, video laryngoscopy, and fiberoptic bronchoscopes. A bougie should be a required device in any emergency or difficult airway cart.

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23 Digital (Tactile) Orotracheal Intubation

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INTRODUCTION

Digital orotracheal intubation is an alternative advanced airway technique that has been demonstrated to be effective when performed by trained prehospital, aeromedical, combat, and hospital providers.[1-18] The index and middle fingers are used to direct the endotracheal tube into the patient's larynx. This technique is "blind" in that the airway is not visualized at any point during the procedure. Use this technique in the Emergency Department as a secondary or tertiary method. It may be particularly useful when oral secretions or blood inhibit direct visualization of the upper airway.[1] There is evidence in hospitalized neonatal patients that digital intubation may be more effective than direct laryngoscopy as a primary method of orotracheal intubation.[2-4,14]

ANATOMY AND PATHOPHYSIOLOGY

The significant anatomic structures that the Emergency Physician will encounter are the patient's tongue and epiglottis. The epiglottis is the cartilaginous structure that is located at the root of the tongue and serves as a valve over the superior aperture of the larynx during the act of swallowing.[6] Refer to Chapters 9 and 18 for a more complete discussion of the airway anatomy.

INDICATIONS

Digital orotracheal intubation is an alternative technique for intubating the comatose or chemically paralyzed patient. There are three major indications for digital intubation. The equipment required to perform direct or video laryngoscopy is either not available or not functioning. The physical positioning of the patient does not permit the provider to intubate using direct or video laryngoscopy.[7] Visualization cannot be achieved using conventional techniques due to trauma, excessive secretions, or blood in the airway.[1]

This technique involves minimal movement of the head and neck. It may be a suitable method for intubating patients with known or suspected cervical spine injuries. Digital intubation may be a useful procedure for paramedics and aeromedical personnel in the out-of-hospital setting, when trapped patients require intubation but are not in a position for more conventional methods.[7] It is an alternative technique for out-of-hospital intubation where other techniques and equipment are unavailable or limited.

This procedure may be considered a primary method of intubation in neonatal patients.[2,4,5] One small study demonstrated that digital intubation of the neonate was more successful and faster than direct laryngoscopy when performed by a very experienced neonatal intubator.[3] The first-pass success rate was 90.5% for the digital

intubation versus 50% for the direct laryngoscopy, with a mean time to intubation of 8.2 seconds and 13.1 seconds, respectively.

Digital intubation may be a life-saving technique for difficult airways.[16,17] Adequate skill training and maintenance of competency are critical to achieve successful outcomes. Medical flight crewmembers and Emergency Medicine residents in a simulated tactical setting were found to require more attempts and take longer to provide advanced airway management with digital intubation compared to direct laryngoscopy and King LT tube placement.[13] Digital intubation only had a 14% success rate in a large prospective prehospital difficult airway study.[14] Adequate training for digital intubation is paramount, and the Emergency Physician should choose airway techniques with which they are most familiar before considering secondary or tertiary methods in a patient with a difficult airway.

CONTRAINDICATIONS

There are no absolute contraindications to digital intubation. The main danger of this procedure is to the Emergency Physician who is at risk for having their fingers bitten by the patient. **Do not perform this technique on any patient who is awake or semiconscious.** It should be performed only on patients who are paralyzed or unconscious. Relative contraindications would be performing this procedure on a patient with multiple fractured teeth that may abrade or cut the fingers or a patient whose ingestion may present a biochemical hazard.

EQUIPMENT

- Endotracheal tubes, various sizes
- 10 mL syringe
- Water-soluble lubricant or anesthetic jelly
- Bag-valve device
- Oxygen source and tubing
- Gloves
- Gauze 4×4 squares
- Wire malleable stylet (optional)
- Bite block (optional)
- Bougie (optional)
- Supplies for orotracheal intubation (Chapter 18)

PATIENT PREPARATION

Digital intubation in the Emergency Department is commonly performed on an emergent or urgent basis. Explain the risks, benefits, and complications of the procedure to the patient and/or the patient's representative if there is time.

The use of gloves, a bite block, and gauze over the teeth as guards is recommended when performing this procedure. Place the patient supine. Immobilize the cervical spine if the patient has sustained a concerning mechanism of injury. An assistant can help hold the patient's head to maintain in-line immobilization. Place the patient on continuous cardiac monitoring, pulse oximetry, and supplemental oxygen. The remainder of the preparation is the same as for orotracheal intubation (Chapter 18).

Prepare the endotracheal tube. Attach a 10 mL syringe to the cuff's inflation port. Inflate the cuff and inspect it for any air leaks. Deflate the cuff and leave the syringe attached to the inflation port. The use of a stylet is optional, but one is commonly used by most practitioners in this procedure.[10] A stylet may be used if the patient's larynx is anterior, the intubator has short fingers, or it is the Emergency Physician's preference. Lubricate and insert the stylet until it is 1 cm proximal to the distal end of the endotracheal tube. Bend the malleable stylet just as it enters the endotracheal tube. This will prevent it from migrating distally and injuring the patient. Gently bend the distal end of the endotracheal tube into a "J."[1] Liberally lubricate the distal end of the endotracheal tube. Induce anesthesia if the patient is conscious.

FIGURE 23-1. The index and middle fingers are placed in the right side of the patient's mouth and advanced until the epiglottis is palpated. The endotracheal tube is inserted into the patient's mouth between the two fingers.

TECHNIQUE

Stand at the patient's right side, facing the patient. Open the patient's mouth. Insert the index and middle fingers of the right or dominant hand into the right angle of the patient's mouth (**Figure 23-1**).[1,3] Slide the fingers along the surface of the tongue until the epiglottis is palpated. The metacarpophalangeal joints of the index and middle fingers will usually be at the level of the patient's incisors in an adult. The tip of the epiglottis is approximately 8 to 10 cm from the incisors. Elevate the epiglottis with the index finger (**Figure 23-2**). An assistant or the thumb of the nondominant hand may be used to provide cricoid pressure if needed.[11]

Insert the endotracheal tube with the left or nondominant hand along the left side of the patient's mouth and between the two fingers

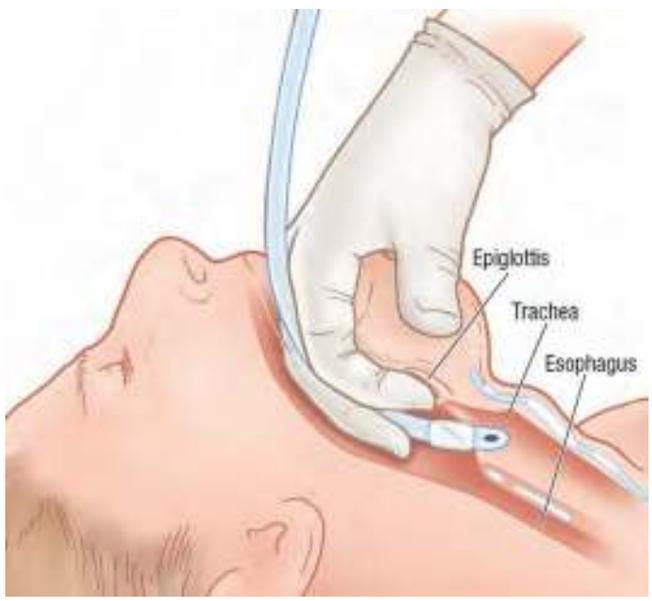

FIGURE 23-2. Advancing the endotracheal tube. The epiglottis is elevated with the index finger. The endotracheal tube is advanced between the fingers and into the larynx.

(**Figures 23-1 and 23-2**).[1,3] Alternatively, the endotracheal tube can be advanced between the two fingers and the tongue. Gently advance the tip of the endotracheal tube into the patient's trachea (**Figure 23-2**).

Do not advance the endotracheal tube if any resistance is encountered. Partially withdraw the endotracheal tube and attempt to reinsert it into the patient's trachea. An alternative is to withdraw the endotracheal tube completely, bend it more acutely, and attempt reinsertion.

Have an assistant withdraw the stylet if one was used. Advance the endotracheal tube approximately 3 to 4 cm.[1,12] Securely hold the endotracheal tube with the left or nondominant hand and carefully withdraw the other hand from the patient's mouth. Inflate the cuff of the endotracheal tube. Begin ventilating the patient while securely holding the endotracheal tube and confirming proper placement (Chapter 19).

The technique of digital orotracheal intubation may require some modification under certain circumstances. **A patient's mouth may appear too small to allow an endotracheal tube and two fingers.** Use the fingers in the patient's mouth to push their jaw and tongue forward. If the patient's mouth still appears too small, apply gauze squares over their mandibular incisors and push their mouth open further using the bases of the fingers in the patient's mouth.

It may be difficult to blindly identify the patient's epiglottis. Insert the fingers in the midline of the patient's mouth and slowly push them posteriorly. The fingers will roll off the posterior portion of the tongue and allow palpation of the epiglottis. **Do not push the epiglottis posteriorly.** Once identified, move fingers to the right side of the patient's mouth while the fingertips are in constant contact with the epiglottis.

Some may be concerned their fingers are not long enough to palpate the epiglottis. Push the soft tissue in the right angle of the patient's mouth posteriorly while inserting the fingers posteriorly in their mouth. This will allow the fingers to reach more posteriorly. This may not work in tall thin patients. Try a supraglottic device (Chapter 25) or bougie (Chapter 22) if using the fingers does not work.

This technique can be used in patients of all ages, including neonates, with only minor variations in technique.[3-5] Use only the index finger for neonates and young children when performing this procedure.[3] Use the appropriate-size suction catheter or endotracheal tube as would be used to orally intubate the child. **Do not insert the catheter or endotracheal tube using this technique if the child is spontaneously breathing because their vocal cords may be damaged.**

An alternative approach involves introduction of a bougie into the trachea using the technique described above followed by the passage of an endotracheal tube over the bougie (**Figure 23-3**).[13,17,18]

FIGURE 23-3. The bougie-aided digital intubation. ***A.*** The fingers have been inserted into the mouth to elevate the tongue and epiglottis. ***B.*** The bougie is advanced into the trachea. ***C.*** The endotracheal tube is inserted over the bougie. ***D.*** The bougie has been removed and the endotracheal tube remains. (Used with permission from reference 18.)

Advantages of this approach include the smaller diameter and malleability of the bougie; tracheal clicking with the bougie, which can help confirm proper placement; and the ability to easily carry a bougie into tactical environments.

ASSESSMENT

The placement of an endotracheal tube should be followed by an assessment to ensure its proper positioning (Chapter 19). This includes visual inspection of chest rise with ventilation, lack of abdominal movement with ventilation, fogging in the endotracheal tube for at least six breaths, auscultation, and end-tidal CO_2 monitoring. This should be followed by a chest radiograph to confirm proper positing of the endotracheal tube within the trachea.

AFTERCARE

The steps of ensuring proper placement of the endotracheal tube and securing the tube are the same as for any patient who has undergone orotracheal intubation (Chapter 18).

COMPLICATIONS

No significant complications to the patient have been identified with digital intubation. One study involving a small number of cadavers found that digital intubation predisposes to left mainstem bronchial intubation.[9] The investigators concluded that decreased right-sided breath sounds after tactile intubation may represent an easily corrected left mainstem intubation rather than more concerning pathology. An awake or semiconscious patient may gag with subsequent vomiting, aspirate, and injure the Emergency Physician's fingers. Insert a bite block for added safety between the patient's molars if its placement still allows enough space in the patient's mouth for two fingers and the endotracheal tube. The possibility of esophageal intubation is significant, especially if the intubator has small fingers or the airway is anterior. Hypoxemia can result from numerous attempts at intubation without any intervening ventilations. Insert the endotracheal tube gently to prevent traumatic injury to the patient's hypopharynx, vocal cords, or trachea.[10]

SUMMARY

Digital (tactile) intubation is a viable alternative technique for the management of the difficult airway. Prehospital, aeromedical, combat, and Emergency Physician providers should receive training in this technique. It is an expedient method for intubating a comatose or chemically paralyzed patient if the upper airway cannot be visualized because of trauma, secretions, or blood. This technique may be the only method available in certain environments due to patient positioning and lack of advanced airway equipment.

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24 Lighted Stylet Intubation

Jaroslav Tymouch, Gennadiy Voronov, and Ned F. Nasr

INTRODUCTION

Direct laryngoscopy is the most common method of tracheal intubation in the Emergency Department. Tracheal intubation in critically ill patients is a high-risk procedure. The risk of complications increases with repeated or prolonged attempts. **Make expedient first attempt success the goal for airway management in these patients.** Patient-related factors often make visualization of the airway and placement of the tracheal tube difficult. Physiologic derangements reduce the patient's tolerance for repeated or prolonged attempts at laryngoscopy. This results in hypoxemia and hemodynamic deterioration. Operator-related factors (e.g., experience, device selection, and pharmacologic choices) affect the odds of a successful intubation on the first attempt. Direct laryngoscopy will be difficult or impossible in approximately 1% to 3% of patients requiring intubation.[1-4] This may be due to many different causes (e.g., excessive airway bleeding, limited cervical spine mobility, or limited cervical spine mobility). Blind intubation using a lighted stylet in these situations is a proven valuable technique.[5-14]

Light-guided intubation relies on the transillumination of the soft tissue of the neck to indicate intratracheal endotracheal (ET) tube placement. A bright and well-defined glow is seen in the anterior neck when the light is in the trachea (**Figures 24-1 and 24-2A**). A diffuse and less intense glow is seen with esophageal intubation (**Figure 24-2B**). Lighted stylet intubation is a relatively easy technique to learn and rapid to perform.

The concept of using a light-guided introducer for orotracheal intubations first appeared in print in the late 1950s.[1,2,5,6] Several authors described ingenious devices to tunnel a light bulb attached to an introducer through the ET tube and power it with a pocket battery, penlight, or laryngoscope handle.[1,2,5,6] These primitive devices were simple and effective. Further development of this technology was stagnant until the late 1970s when several authors described use of the Flexilum surgical light as a lighted stylet or "light wand."[7,8] The makers of the Flexilum light revised the design after a few problems

FIGURE 24-1. A bright and well-circumscribed glow (*arrow*) is seen below the thyroid prominence when the lighted stylet enters the glottis. (Used with permission from Hung O, Murphy MF (eds): *Management of the Difficult and Failed Airway*, 2nd ed. New York: McGraw-Hill; 2012.)

with bulb dislodgment and marketed the first lighted stylet known as the Tube-Stat.[9,15] A similar design is still available as the Tube-Stat lighted intubating stylet (Medtronic Xomed, Jacksonville, FL). Laerdal introduced the more sophisticated Trachlight in the mid-1990s (**Figure 24-3**). It was an improvement over previous lighted stylets with a more intense light source, a more flexible light wand, and a separate retractable stylet that adjusts to a variety of ~~ET~~ tube lengths.[10] There are numerous lighted stylets today (**Figures 24-3 through 24-6** and **Table 24-1**).

ANATOMY AND PATHOPHYSIOLOGY

The trachea lies anterior in the neck and is covered only by the skin, subcutaneous tissue, and pretracheal fascia. The esophagus lies posterior to the trachea surrounded by much more soft tissue (Chapter 9). **A light source positioned within the trachea will transilluminate a bright and discrete glow that can easily be**

A **B**

FIGURE 24-2. Use of a lighted stylet. ***A.*** The tip of the ET tube is in the glottic opening and below the thyroid prominence at the glottic opening. ***B.*** The tip of the ET tube is in the esophagus. (Used with permission from Hung O, Murphy MF (eds): *Management of the Difficult and Failed Airway*, 2nd ed. New York: McGraw-Hill; 2012.)

FIGURE 24-3. The Laerdal Trachlight (Laerdal Medical Inc., Wappingers Falls, NY).

FIGURE 24-4. The Vital Signs Light Wand (Vital Signs Inc., Totowa, NJ).

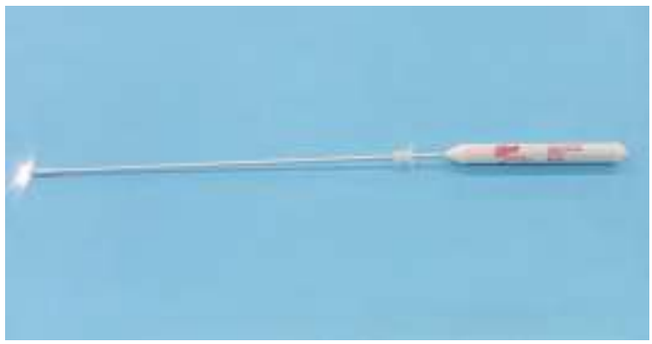

FIGURE 24-5. The Aaron Medical Surch-Light Lighted Stylet (Bovie Medical Corp., Purchase, NY).

FIGURE 24-6. The Medtronic Tube-Stat Lighted Intubation Stylet (Medtronic Xomed Inc., Minneapolis, MN).

TABLE 24-1 Some of the Commercially Available Lighted Stylet Devices

Name	Manufacturer	Comments
Aaron Surch-Lite	Bovie Medical Corporation	Adult sizes Malleable stylet Single-use
AincA Lighted Stylet	Anesthesia Associates	Fits ET tube sizes ≥ 3.0 mm and ≥ 5.0 mm inner diameter Malleable stylet Removable handle Reusable Two sizes (adult/pediatric and infant) Xenon bulb keeps cool
Tube-Stat Lighted Intubation Stylet	Medtronic	Malleable stylet Reusable Two sizes (nasotracheal 33 cm and orotracheal 25 cm)
Vital Signs Light Wand	GE Healthcare	Adult sizes Bulb keeps below 42°C or 108°F Malleable stylet Reusable

seen on the surface of the neck (Figures 24-1 and 24-2A). A light source directed within the esophagus will be diffused by the surrounding tissue and appear dull (Figure 24-2B). The Emergency Physician can easily discriminate between the dull, diffuse transillumination of an esophageal light source and the more discrete, intense signal transmitted from within the trachea. **A submental, superior to the hyoid bone, glowing light indicates that the tip is positioned in the vallecula. A lateral glowing light indicates placement in the pyriform sinus.**

Lighted stylet tracheal intubation requires practice and is easily learned. Intubation training influenced the speed of intubation but not the success rate on the first attempt.[16] The average time for a successful intubation was 42 seconds in the first 25 attempts. This time decreased to an average of 32 seconds for the second 25 attempts. All lighted stylet intubations were successful by the third attempt. Videographic study of 125 children tracheally intubated by anesthesia residents with little or no lighted stylet experience had an overall success rate of 83%.[16] Failures were due to a too large tracheal tube or persistent entry into the vallecula or esophagus.

Traditional methods of estimating the degree of difficulty in achieving tracheal intubation under direct laryngoscopy may not apply when a lighted stylet approach is undertaken. In two studies, 200 patients who were predicted to be easy intubations with direct laryngoscopy were intubated with a lighted stylet.[17,18] Approximately 87% were intubated on the first attempt with a lighted stylet and 99% within three attempts. There was no correlation between the time to intubate and any of the airway prediction variables in the Trachlight group in a series of 950 patients.[10] This contrasted with the direct laryngoscopy group, in which some of the airway variables (e.g., the Mallampati score and the circumference of the neck) did correlate with the time to intubate the trachea.

Laryngoscopy and ~~ET~~ intubation are intensely stimulating procedures. Both are associated with varying degrees of sympathetic activity, which may be detrimental in patients with coexisting conditions. Several investigations of the possibility that lighted stylet intubation may result in less stimulation than direct laryngoscopy and may offer some protective effects from sympathetic hyperactivity did not confirm this to be true. There is some tendency for lower blood pressure and heart rate in a lighted wand group. A study that compared two direct laryngoscope blades (i.e., the Macintosh 3 and the Miller 2) with lighted stylet intubation showed no significant difference in hemodynamic changes among the three groups.[19]

The difficult airway is one of the most common indications for the use of the lighted stylet. This is especially true in situations with acute oropharynx-tracheal angles, increased secretions, or bleeding causing failure of intubation with direct or fiberoptic laryngoscopy. It can be used as a back-up technique or primarily if direct laryngoscopy is predicted to be difficult. There are limited data comparing lighted stylet intubations to other rescue techniques.[17,20-24] Lighted stylet intubations are faster and have fewer complications than blind nasal tracheal and bronchoscopy-assisted intubations.[13,17,24] Lighted stylets are easier to use, less expensive, and more portable than fiberoptic bronchoscopes.[17,22] Experience must be gained from intubating normal airways before it can be relied on for the difficult case.

INDICATIONS

There are numerous indications for lighted stylet intubation (**Table 24-2**). Lighted stylet intubation is a valuable rescue technique in patients who can be ventilated and oxygenated but are unable to be intubated with direct laryngoscopy. It is particularly useful in patients who have cervical spine immobilization, extensive blood in the hypopharynx, extensive oral trauma, extensive secretions in the hypopharynx, limited jaw opening, limited neck movement, or loose teeth that limit visibility.[16,20-22,25-27] Lighted stylet intubations can be performed as part of rapid sequence intubation. They can be used on spontaneously breathing patients who are sedated and have topical airway anesthesia. Lighted stylets can be used for oral or nasal intubations.[25,28] This technique can also be used for pediatric patients and infants.[16,22,25-27]

There are some cases of emergency tracheal intubation using a modified lighted stylet technique in patients with clenched jaws in whom nasal intubation was contraindicated.[29] The lighted stylet was inserted behind the third molars as the cheek was retracted. The tip of the ET tube was then advanced toward the larynx in a paramedian approach and was guided by the typical transillumination. A lighted stylet can be used for intubation of patients with cervical spine injuries.[26] This allows the administration of cricoid pressure while keeping the cervical spine in a neutral position. There is no influence from blood and secretions in the airway.

CONTRAINDICATIONS

Intubation with lighted stylets is relatively safe and simple. There are few contraindications to this technique. **Patients with laryngeal trauma should have direct laryngeal visualization for intubation rather than a blind technique that may cause additional trauma.** Do not use a lighted stylet if there is any active infection or known tumor of the posterior pharynx or upper airway. The presence of an airway foreign body, epiglottitis, a laryngeal polyp or tumor, a

TABLE 24-2 Indications for Intubating with a Lighted Stylet

Anatomic abnormalities
Congenital head and neck anomalies
Midface hypoplasia
Pierre Robin syndrome
Treacher Collins syndrome

Excessive secretions
Blood
Vomitus

Limited cervical spine movement
Burn strictures of the neck
Cervical arthritis
Known or suspected cervical spine injury

Limited jaw mobility
Temporomandibular immobility
Trismus

Trauma
Dental trauma
Maxillofacial trauma

Miscellaneous
Unable to intubate by other methods

retropharyngeal abscess, or tracheal stenosis precludes the use of this technology. It may be less successful in bright sunlight, obese patients, and patients with very dark skin.[18,20,21]

Lighted stylets are relatively contraindicated in patients who cannot be oxygenated and ventilated. It can be used in this situation by very experienced Emergency Physicians while simultaneous preparations are under way for a cricothroidotomy (Chapter 32). Lighted stylets should be used only by Emergency Physicians who have sufficient experience and training with the equipment and the technique.

Sometimes the lighted stylet meets resistance when it is advanced. It should be withdrawn, redirected, and advanced only if it passes with ease. **Abandon the technique and use an alternative method to intubate the patient if unexpected difficulty occurs during its passage.**

EQUIPMENT

- Lighted stylet
- ET tubes, various sizes
- 10 mL syringe
- Water-soluble lubricant
- Bag-valve device
- Equipment for orotracheal intubation (Chapter 18)

The basic unit of a lighted stylet is quite simple. It consists of a handle, a malleable stylet, and a light on the end of the stylet (**Figure 24-7**). Numerous variations exist on the basic unit. The handle contains the battery, on-off switch, and sometimes an on-off indicator light. The stylets may be reusable or single-use disposable devices. The stylet is malleable and available in numerous sizes. Longer stylets allow for nasal and oral intubation in adults. Stylets are also available in smaller lengths and widths to allow for use in small ET tubes for infants and children. The stylet is inserted into the ET tube instead of a standard stylet.

Numerous devices are available (**Table 24-2**) Examples include the Trachlight (**Figure 24-3**), the Light Wand (**Figure 24-4**), the Surch-Lite (**Figure 24-5**), and the Tube-Stat (**Figure 24-6**). There are slight differences in design. They all rely on using a lighted stylet inside the ET tube to transilluminate the anterior neck and guide blind intubation of the trachea. The Tube-Stat (Medtronic Xomed, Jacksonville, FL) is an example of the simplest lighted stylet (**Figure 24-6**). It incorporates a stylet, power source, and light bulb all in a single piece. The unit is inserted into the ET tube in place of the stylet. A switch on the handle activates the light. The Trachlight (Laerdal Medical Corporation, Wappingers Falls, NY) consists of a battery-powered handle, the light wand, and a separate stylet that inserts into the light wand (**Figures 24-3 and 24-7**). It is a simple device to operate. It is likely that further developments will continue to make these and similar models attractive options in airway management.

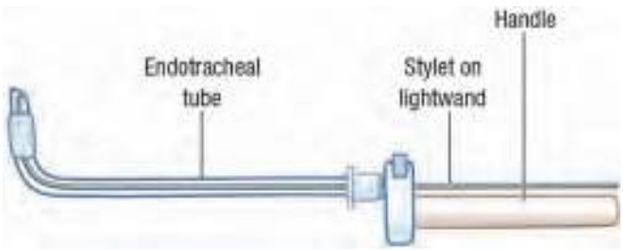

FIGURE 24-7. The Trachlight lighted stylet.

PATIENT PREPARATION

Prepare the patient as for any other intubation (Chapter 18). Obtain intravenous access and hemodynamic monitoring (e.g., an automatic blood pressure monitor, cardiac monitor, and continuous pulse oximetry). Prepare suction and have it immediately available. Rapid sequence intubation (Chapter 16) with an induction agent and paralytic agent is the most common technique used in the Emergency Department. Lighted stylet intubations can be performed with mild sedation and topical anesthesia in cooperative patients.

Preoxygenate the patient prior to airway manipulation. The position of the neck can be neutral, unlike with other techniques. The "sniffing" position is not required for this technique. The anterior half of a cervical collar must be opened or the entire collar removed to be able to visualize the glowing light. An assistant can maintain in-line stabilization of the cervical spine when the collar is opened or removed.

TECHNIQUE

OROTRACHEAL INTUBATION

The exact technique will depend on the type of lighted stylet or light wand used. This technology varies by manufacturer. Be familiar with the equipment and manufacturer's instructions prior to adopting lighted stylets for use clinically. This text describes the general guidelines for the lighted stylets available. The reader is urged to take advantage of the teaching videos supplied by some manufacturers.

Check that the light source is working and apply a water-based lubricant to the stylet. Attach a 10 mL syringe to the ET tube cuff inflation port and ensure the integrity of the cuff. Insert the lighted stylet through the ET tube. **Ensure that the tip of the lighted stylet is just inside the ET tube so that the lighted stylet does not damage soft tissues.**

Bend the tip of the ET tube and lighted stylet just proximal to the cuff or about 3 to 6 cm from the distal end (**Figure 24-8**). The bend may have to be a little more proximal or distal depending on the length of the patient's neck. Measure the mandibular-hyoid distance in the patient. Place the index finger in the submental space below the chin and determine the number of finger breadths between the mandible and the hyoid bone.[12] Typical measurements are 1 to 3 finger breadths. Bend the tip of the ET tube and lighted stylet sharply at a site that approximates the mandibular-hyoid distance between the bend and the junction of the lighted tip of the stylet. This is usually 3 to 6 cm from the distal end of the ET tube and just above the cuff. Avoid making the bend at the cuff, if possible, to prevent damaging the cuff. **Be sure the bend is about 90° to allow the maximal light intensity to be directed anteriorly.**

Stand above or to the side of the patient's head. The lighted stylet, unlike the traditional laryngoscope, can be held in either hand. Lower the bed to facilitate insertion of the lighted stylet. Grasp the patient's jaw with the nondominant hand. Place the nondominant thumb on the mandibular molars and the nondominant fingers under the body of the mandible. Lift upward and inferiorly to open the jaw, elevate the tongue, and elevate the epiglottis. Grasp the lighted stylet with the dominant hand and turn it on. It is best held with a "pencil grip" over the proximal ET tube.

Introduce the ET tube from the side of the patient's mouth and bring it to the midline. The handle will project toward the patient's feet as the hockey stick–shaped tip is placed over the tongue (**Figure 18-8*A***). Advance the tip by moving the handle in a vertical arc toward the patient's head (**Figure 18-8*B***). This will bring the ET tube tip toward the vocal cords. A bright light will be seen

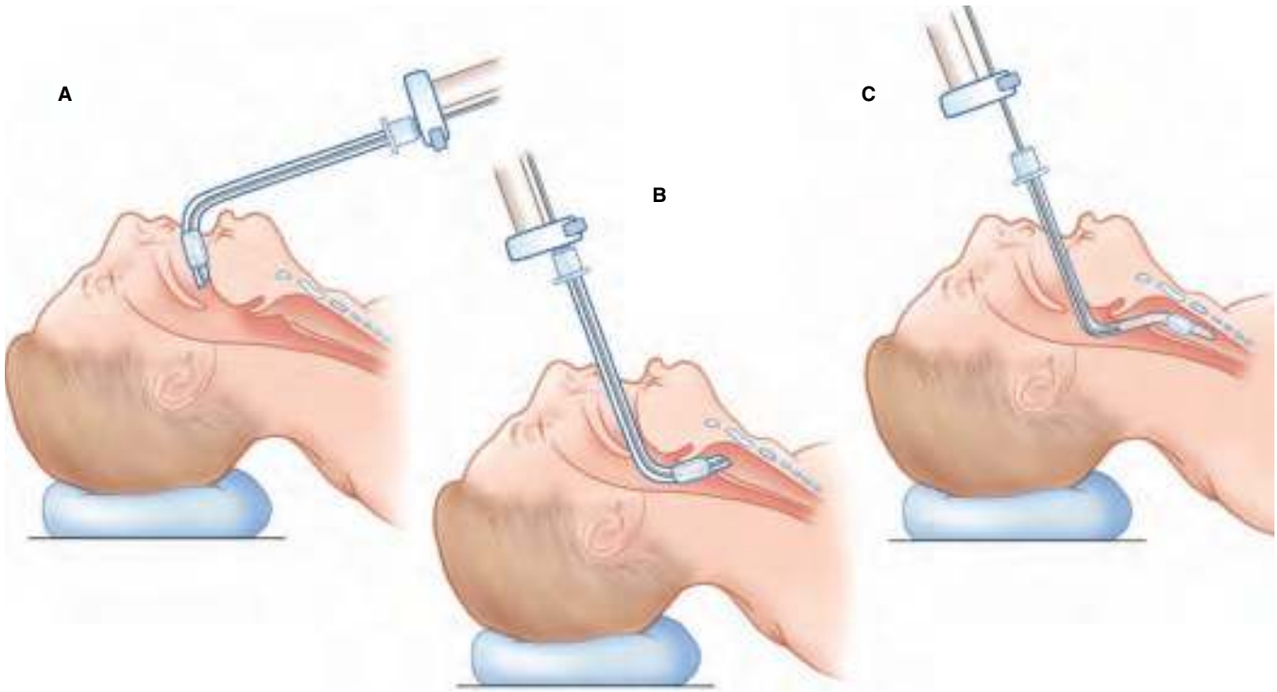

FIGURE 24-8. Insertion of the lighted stylet. ***A.*** Insert the hockey stick–shaped ET tube containing the lighted stylet over the tongue. ***B.*** Move the ET tube in a vertical arc toward the patient's head. The bright light transilluminates the anterior neck as the lighted stylet approaches the trachea. ***C.*** The ET tube is advanced into the trachea.

in the midline of the neck just below the hyoid bone (**Figures 24-1 and 24-9*A***). **The tip of the ET tube is in the vallecula if the light is in the submental space (Figure 24-9*B*). The tip of the ET tube is lodged in the pyriform sinus if the light is lateral (Figure 24-9*C*). A dull, faint light in the midline signifies that the tip of the ET tube is in the esophagus (Figures 24-2 and 24-9*D*)**. Simply withdraw the malpositioned ET tube, reposition it in the midline, and advance it again.

It is safe to advance the ET tube once a bright and discrete light is detected in the midline at the level of the thyroid cartilage (i.e., Adam's apple). **Advance the ET tube while observing the transilluminating light march down the neck to the suprasternal notch (Figure 24-9A).** The light will disappear as the tube passes behind the suprasternal notch. The epiglottis is obstructing its advancement if the glowing light is in the midline and the ET tube is resistant to advancement. Slightly rock the unit in the sagittal plane (i.e., from the patient's head to their feet) to slip the tip of the ET tube under the epiglottis. Remove the lighted stylet if resistance is still encountered. Ventilate the patient with a bag-valve-mask device. Load a smaller ET tube onto the lighted stylet and try again. The tip of the ET tube is appropriately positioned midway between the vocal cords and carina at the point the light is lost.[30] Remove the lighted stylet, inflate the ET tube cuff, confirm proper ET tube placement, secure the ET tube at the lips, and begin ventilating the patient.

Use caution in very thin or very obese patients. A bright light may be visible when the stylet is in the esophagus in the thin patient. Gently rock the light off midline to compare the diffuse dull light to that seen when the light is truly midline. The extra soft tissue in the obese patient may dull the light. Dim the room lights to facilitate adequate visualization.

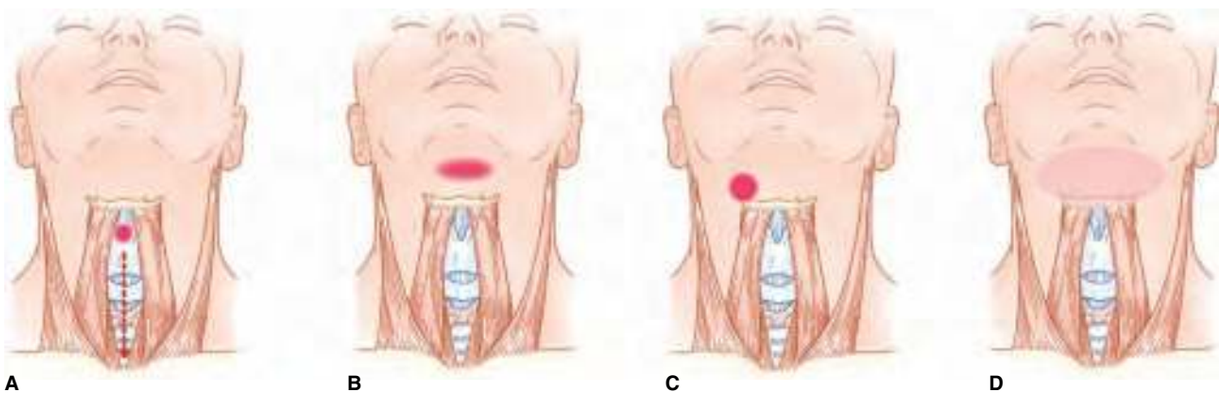

FIGURE 24-9. Appearance of the transilluminated light of the lighted stylet based on the location of the tip. ***A.*** Proper placement in the larynx with a bright distinct light in the midline at the level of the thyroid cartilage. The light moves down the anterior neck and disappears behind the sternal notch (*dashed arrow*) with advancement of the ET tube. ***B.*** Incorrect placement in the vallecula causes a submental glow superior to the hyoid. ***C.*** Incorrect placement in the pyriform sinus causes a glow off the midline. ***D.*** Incorrect esophageal placement causes a diffuse, dull, or absent light.

FIGURE 24-10. Nasotracheal intubation using a lighted stylet. A bright and well-circumscribed glow (*arrow*) is seen below the thyroid prominence when the lighted stylet enters the glottis. (Used with permission from Hung O, Murphy MF (eds): *Management of the Difficult and Failed Airway*, 2nd ed. New York: McGraw-Hill; 2012.)

The glowing light must maintain a continual brightness to demonstrate tracheal intubation. The ET tube has been misplaced in the esophagus if the glowing light is briefly lost or dulls and then returns (Figure 24-2). The brief loss or dulling of the glowing light corresponds to its passage behind the larynx. The return of the bright glowing light corresponds to the ET tube advancing past the larynx and into the esophagus. This is commonly seen in infants, small children, and very thin adults. Gently withdraw the lighted stylet while applying anteriorly directed traction to the tip of the stylet. Stop withdrawing the lighted stylet when the glowing light suddenly intensifies after it exits the esophagus. Readvance the lighted stylet, as previously described, while applying anterior traction on the unit to help it enter the larynx.

NASOTRACHEAL INTUBATION

The procedure for nasotracheal intubation is similar to orotracheal intubation with the lighted stylet with a few differences (**Figure 24-10**). Treat the nasal passages with a topical anesthetic and a topical decongestant to vasoconstrict the mucosal tissue (Chapter 29). The nasal passage may need to be dilated to accommodate the ET tube. The lighted stylet should have a gentle curve of about 100° to 120°. The bend-to-tip length should correspond to the distance from the posterior nasopharynx to the cricothyroid membrane.

ASSESSMENT

Assess the patient continuously at every step of the procedure to assure adequate oxygenation and ventilation. Confirm the position of the ET tube by end-tidal CO_2 monitoring, esophageal detector device, fogging in the ET tube for at least six ventilations, auscultation, and chest radiograph (Chapter 19). Secure the ET tube.

AFTERCARE

The ongoing care of the patient should proceed as with any other intubation technique. Routine care of the ET tube is no different. The manufacturer's guidelines should be followed for maintenance of the equipment.

COMPLICATIONS

Intubation with a lighted stylet is a very safe procedure with a very low complication rate.[13,21] There are only a small number of reported complications in over 30 years of use. These include two reports of accidental bulb dislodgment, two arytenoid dislocations, one case of stylet fracture, a lacerated frenulum, and varied reports of mild soft tissue trauma.[9,10,15,31-33] Dysphagia, hoarseness, and a sore throat occur less frequently with lighted stylets than with direct laryngoscopy.[14] The cases of equipment failure have been in older models of lighted stylets. There are data to suggest that lighted stylet intubations are less traumatic than direct laryngoscopy.[14] No complications were noted in 253 patients intubated with lighted stylets.[21] Intubation with lighted stylets may cause less cervical spine motion compared with direct laryngoscopy.[30]

The newer models have been revised to minimize problems and have been remarkably free of adverse events. Complications with this technique are unusual in experienced hands. Weis and Hatton reported no complications in 253 patients intubated with a lighted stylet.[21] Success rates with lighted stylet intubation are comparable to those with other techniques. Success rates on first attempts are reported to be as low as 70% with the Tube-Stat and as high as 92% with the Trachlight.[10,11,13,14,17,27,34] Success rates approach 100% when multiple attempts are allowed.[12,17] Success rates of 88% have been reported in the prehospital setting under adverse and emergent conditions.[18] The rates of successful intubation remain high for difficult airways. Hung and colleagues reported the successful intubation in 95 of 96 known difficult airways.[20] This included patients with prior difficult or failed intubations by direct laryngoscopy, patients with unstable cervical spines, patients with ankylosing temporomandibular joints, and the morbidly obese. Holzman and colleagues reported successful intubation in 30 of 31 children with anatomic airway abnormalities.[22]

Intubation times with lighted stylets are comparable to those with other techniques and range from 15.7 to 45 seconds.[10-12,17,18,20-22,35] Recent evidence suggests that intubation with a lighted stylet may cause fewer hemodynamic alterations when compared to direct laryngoscopy.[36,37] The skill level of the intubator is an important factor in success rates, time to intubation, and possibly complications.[22] The best success rates are reported by those who have devoted a great deal of time to the technique and use it regularly. There is a definite learning curve, and time to intubation improves with experience.[18,23,26] Skills may improve with practice in a cadaver lab.[12,23] Manikins designed as intubation teaching models can be modified for use with lighted stylets.[34] Proper training, use of teaching videos, didactics, formal practice with training manikins and in cadaver labs, and supervised clinical experience are all important means of developing and maintaining skills and decreasing complication rates.

SUMMARY

Lighted stylet intubation is an easily learned technique that is a valuable adjunct for securing difficult airways. It is particularly useful in patients with limited jaw opening, limited neck movement, and marked airway bleeding. The American Society of Anesthesiologists includes use of lighted stylets in its recommendations for the management of difficult airways.[11] The American Heart Association encourages instruction in alternative airway approaches, including lighted stylets, for difficult airways.[38] The use of lighted stylets has proven to be rapid, safe, and effective in emergent and difficult settings.

The success of lighted stylet techniques is determined by the experience of the Emergency Physician. This technique is likely to be of greatest benefit in the occasional unexpected airway emergency.

Maintaining sufficient skills to use it in the emergent setting is a challenge. Emergency Physicians will need to devote time and effort to developing and maintaining the skills they will need to cope with an airway emergency when it presents.

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25 Supraglottic Airway Devices

Fred A. Severyn

INTRODUCTION

Airway management and the subsequent provision of oxygenation and ventilation remains one of the foundational principles in the clinical practice of Emergency Medicine in the Emergency Department and the prehospital environment. The majority of advanced airway interventions in the prehospital environment occur in the cardiac arrest or major trauma patient with significant altered mentation. A growing body of evidence questions actual survival benefit for the practice of endotracheal (ET) intubation. A baseline proficiency in airway management via intubation requires on the order of 50 to 100 intubations, a clinical experience not commonly found in the prehospital training environment.[1] Even more disheartening are numerous studies demonstrating a significant percentage of cases of unrecognized esophageal airway placement or ET tube migration out of the airway upon Emergency Department arrival. Such therapeutic misadventures guarantee bad outcomes.[2] Most of these studies describe relatively busy prehospital systems with clearly defined medical oversight.

Evidence-based cardiopulmonary resuscitation (CPR) science has shown that limiting "no-flow time" associated with compression-ventilation cycles is directly related to the efficiency of resuscitation as measured by coronary perfusion pressure. Most supraglottic airway devices (SADs) can be placed with a very high first-pass success rate in under 20 seconds. This allows for progression to continuous uninterrupted compression cycles with interposed ventilations. Multiple prehospital systems have placed SADs into their protocols for the use of alternative airway devices in preference over ET intubation.[3,4]

The spectrum of airway management begins with the Basic Life Support (BLS) skill set using airway positioning, suctioning, and ventilation support with the use of a bag-valve-mask device. The next decision is to progress to the Advanced Life Support (ALS) skill set for definitive long-term airway management via cuffed ET tube placement or a surgical airway. ALS management has revolved

around the concept of placing a cuffed ET tube. The concept of the "rescue airway device" has risen as a back-up technique after unsuccessful ET tube placement using a SAD as a conduit for oxygenation and ventilation to "buy some time." This life-saving practice is included in difficult airway and CPR algorithms aimed at maximizing uninterrupted chest compressions.[5-11] **It is a reasonable expectation that the Emergency Physician develop a baseline expertise and experience with a variety of SADs.** This is for receiving patients who have had them placed and when ALS procedures do not go exactly as planned in the Emergency Department.

The original creation of SADs as airway adjuncts occurred with an initial aim at the operative airway management of patients in the Operating Room. Simplistically, SADs function as a conduit between the mouth and laryngeal inlet at the vocal cords, bypassing tongue-induced airway obstruction in the sedated and paralyzed patient during an operation.[12] The classic laryngeal mask airway (cLMA) is the original example of a SAD. It was created in 1981 and is used daily in Operating Rooms across the world with several hundred million cumulative cases of patient care experience.[12,13]

Most Emergency Medicine residents will get a baseline exposure to the use of SADs from operative exposure.[13] The logical migration from the Operating Room to the Emergency Department has occurred as we train the current generation. The device migration from the Emergency Department to the prehospital arena has begun. An explosion of new devices in a very competitive marketplace could easily overwhelm the Emergency Physician as to the nuances of devices and brands. Each SAD comes with manufacturing claims as to their device superiority over the competition in a very competitive marketplace. It is difficult for Emergency Physicians to keep up with the industry explosion of these airway adjuncts. The disadvantage of SADs is that they are only temporary airway devices and must be removed or replaced with another airway device within a few hours. This chapter aims to describe this group of airway adjuncts in broad basic terms, with LMA devices (Chapter 26) and dual-lumen supraglottic ventilation devices (Chapter 27) described in later chapters.

ANATOMY AND PATHOPHYSIOLOGY

ET intubation relies on the placement of a ventilation tube between the vocal cords and inferiorly into the trachea to provide a low-resistance bridge between the trachea and resuscitation/ventilation circuit. This bypasses the most commonly encountered anatomic airway obstruction, the posteriorly directed lax tongue in the supine patient. **SADs offer low resistance to gas flow, provide a variable degree of protection to the trachea from gastric and oropharyngeal secretions, and are suitable to provide positive-pressure ventilation in the apneic patient while limiting potential adverse events.[13] SADs are not designed to pass through the vocal cords.** They rely on the close anatomic relationships between the laryngeal inlet and the upper esophagus for proper device placement and facilitation of gas movement. Some SADs allow for blind ET tube placement through the device lumen with varying degrees of success. Others are too narrow to allow ET tube placement but allow for fiberoptic visualization of vocal cord structures and direct placement of an Aintree introducer.[14] The Aintree introducer can then be used as a stylet for subsequent ET tube placement following SAD removal.[14]

The close anatomic relationships between laryngeal inlet and esophageal orifice help explain some observed complications with SADs. The tip of the SAD was designed to go posterior to the glottis opening at the root of the esophagus. The SAD may impinge into the laryngeal orifice and obstruct airflow to some degree. The device may still "buy some time" but will not function as a definitive airway. The epiglottis may become folded over upon SAD insertion, or it may fold into the aperture meant to sit just outside the vocal cords. This results in varying degrees of airflow obstruction and requires the removal of the SAD.

INDICATIONS

The use of SADs is reserved for the unconscious patient without an intact gag reflex, much like the use of the oropharyngeal airway.[15] Use SADs in the unconscious patient who is difficult to ventilate with a face mask while preparations are being made for ET intubation. Consider a SAD in the "can't ventilate, can't intubate" patient when time is of the essence. The patient's mouth must be able to open at least 1.5 to 2 cm to allow for the insertion of the SAD. SADs can be used as a primary airway device or as a rescue airway device after unsuccessful ET tube insertion.[16-18] High success rates coupled with low experience reinforcement allows their use in the prehospital setting. The use as a rescue device can "buy time" until additional airway assistance, equipment, and/or expertise can be brought to the patient. Consider using SADs during the initial resuscitation of the sudden cardiac arrest victim in the Emergency Department, as they are quick to insert and allow for progression to uninterrupted cardiac compressions. The SAD can be removed or transitioned to a cuffed ET tube. The primary advantage over ET intubation in a mass toxicology event is time and ease of insertion while wearing a protective garment.[19]

CONTRAINDICATIONS

The presence of an intact gag reflex is an absolute contraindication to the use of SADs. The inability to open the patient's mouth 1.5 to 2 cm will prevent the insertion of most SADs. Trauma to the upper airway is a relative contraindication to placement of these devices. These devices are contraindicated in the patient with a caustic ingestion or airway burns. Many SADs require the patient to be over a certain height and/or weight to be used. Refer to the specific manufacturer recommendations when choosing a SAD and the appropriate size. Dual lumen devices meant to be placed into the esophageal lumen had esophageal disease as a contraindication. Newer SADs fit just into the esophageal entrance and do not traverse the lumen. The dangers of SADs in esophageal diseases and injury are probably overemphasized.

EQUIPMENT

- SADS, various sizes and brands
- Water-soluble lubricant
- Device-appropriate syringe, sized to inflate the cuff
- Oxygen source, tubing, and regulator
- Face mask
- Bag-valve-mask device
- Suction source, tubing, and Yankauer catheter
- Cardiac monitor
- Noninvasive blood pressure cuff
- Pulse oximeter
- End-tidal capnography
- ALS medications
- Advanced airway equipment
- Surgical airway equipment

There are approximately 20 variations of SADs available for clinical use today. They are conceptually divided into three broad categories based on the primary separation between the respiratory and

FIGURE 25-1. The SLIPA.

gastrointestinal tracts. These include cuffless preshaped sealers, cuffed perilaryngeal sealers, and cuffed pharyngeal sealers.[20,21]

The cuffless anatomic preshaped sealers are flexible molded devices whose shape is based on spiral computed tomography (CT) scan investigations of normal subjects of varying heights and weights.[22] These studies created a design template for a device that when inserted blindly will accommodate the "normal" airway in most cases.[22] Without an inflatable separation between the respiratory and gastrointestinal tracts there is less flexibility in their design, fit, and use. **These devices offer less potential protection from aspiration of gastric and oropharyngeal contents than other SADs, although some devices do have a gastric sump channel that allows for removal of gastric fluids and air insufflation.** SADs in this category include the Single-Use Liner of Pharyngeal Airway (SLIPA; Hudson RCI, Research Triangle Park, NC) and the i-Gel (Intersurgical Ltd., Berkshire, England). The SLIPA is more commonly used in the Operating Room (**Figure 25-1**).[23] The i-Gel can often be found in the prehospital and Emergency Department airway equipment (**Figure 25-2**). The i-Gel performs as well as the LMA-Supreme in both ventilation and protecting the airway from aspiration.[24]

Cuffed perilaryngeal sealers include the LMA (LMA North America, San Diego, CA) and its many variations, and other laryngeal mask devices. These balloon sealers impact the mucosa of the airway above the laryngeal inlet itself. The narrowest part of the inflatable lumen is meant to wedge into the upper esophagus, theoretically limiting passive gastric distension during positive-pressure ventilation. Design advances have allowed for device inclusions and upgrades to include a potential gastric channel in the tip of the laryngeal mask that allows for placement of a narrow (typically 14 French or smaller) gastric sump tube for gastric suction and decompression. Refer to Chapter 26 for a more complete and detailed discussion of LMA devices.

Cuffed pharyngeal sealers use a design in which a balloon seals the hypopharynx upstream at the base of the tongue, preventing passive air escape through the mouth and nose. Downstream from the balloon occluding the hypopharynx are numerous ventilation apertures that allow for gas to pass into the tracheal and esophageal lumens. This category of SADs can be further subdivided into those devices with and without a second balloon for the esophagus. Those devices without distal esophageal balloons have a less specific and precise anatomic placement site. This may require more device manipulation to allow for easy ventilation with a theoretical lower aspiration protection. Devices in this category of airways include the Cuffed OroPharyngeal Airway or COPA (Covidien, Mansfield, MA; **Figure 25-3**), PAexpress or PAX (Vital Signs Inc., Totowa, NJ), and the CobraPLA and CobraPlus (Pulmodyne Inc., Indianapolis, IN; **Figure 25-4**). The CobraPLA is the only one of these that may be found in the Emergency Department difficult airway cart.

Cuffed pharyngeal sealers with esophageal balloons not only occlude the airway proximally at the base of the tongue, but the esophageal balloon theoretically isolates the esophageal lumen from the respiratory tract. Devices in this category include the Combitube (Kendall Sheridan, Mansfield, MA), the EasyTube (Teleflex Medical, Kernen, Germany), and the King Laryngeal Tubes (King

FIGURE 25-2. The i-Gel.

FIGURE 25-3. The COPA.

FIGURE 25-4. The CobraPLA. (Photo courtesy of Pulmodyne Inc., Indianapolis, IN.)

Systems, Noblesville, IN). All these devices may be found in the Emergency Department difficult airway cart. Refer to Chapter 27 for the complete details of the Combitube and EasyTube. These two SADs are more commonly used in the prehospital environment for a primary or secondary airway and are available in several models.

The King Laryngeal Tubes are made in a variety of sizes and styles (**Figure 25-5**). They require a single large syringe to fill the esophageal and hypopharyngeal balloons with a preestablished amount of air. Newer models incorporated a gastric sump channel that allows for gastric decompression. An additional advantage is the ability to advance a gastric sump tube in troubleshooting the device with potential kinking of the end of the tube.

The specific equipment for the various SADs will vary slightly with the device used and the size of the device. All SADs are relatively similar. Most of the airway devices are made from silicone and require the liberal use of a water-soluble lubricant to help ease airway placement without mucosal trauma and abrasion. Size-appropriate airway adjuncts must be chosen for the individual patient's anatomy.

PATIENT PREPARATION

The patient should be appropriately monitored with electrocardiography (ECG), end-tidal CO_2 monitoring, noninvasive blood pressure cuff, and pulse oximetry. The patient preparation is the same as that for orotracheal intubation (Chapter 18). As with any situation where airway manipulation is to occur and the patient's protective airway reflexes are blunted or ablated, a fully functioning suction apparatus must be immediately available. Insertion of the SAD requires an anesthetic depth or degree of unconsciousness similar to that which allows placement and acceptance of an oropharyngeal airway. Successful placement of the SAD is much more likely if the patient is unconscious, premedicated, or sedated. The optimal induction agent should produce jaw relaxation and attenuation of airway reflexes, permitting insertion of the SAD within 30 to 60 seconds of induction and loss of consciousness. A variety of agents are available for induction (Chapter 16). The Emergency Physician experience coupled with the patient's condition will dictate these agents.

Select the appropriate-size SAD based on patient length or weight. Inflate the cuff(s), if present, to ensure that there are no air leaks in the device. Deflate the cuff(s) and leave the air-filled syringe(s) attached to the SAD. Liberally lubricate the SAD with a water-soluble lubricant to aid in its insertion and seating.

TECHNIQUE

The basic insertion technique begins with choosing the appropriate SAD. Gently access the airway. Use the "triple airway maneuver" with head extension, mouth opening, and a jaw thrust to maximize insertion success. Use the nondominant thumb and forefinger in a scissors-like manner to open the patient's teeth, allowing for device insertion. Insert the liberally lubricated SAD along the roof of the mouth and follow the pharyngeal curve at the back of the throat until resistance is noted, markers on the airway tube are at the incisor level, or the airway sealing balloon is situated in the hypopharynx and cannot be advanced any further.[25] A rotational technique has been described with either a 90° or 180° rotation that might be more helpful after initial insertion failure by provider.[26]

Most SADs use a balloon cuff surrounding the air lumen that limits leakage of ventilated gas retrograde into the oropharyngeal cavity and into the environment. The cuffless preshaped pharyngeal sealers do not have a cuff requiring inflation, so no additional syringe is needed. Each cuffed SAD has a range of air required for proper cuff inflation. **Inflate the cuff with the recommended volume of air. Injection of higher volumes of air over recommendations risks SAD migration out of position, airway or esophageal trauma, balloon herniation over the gas passage ports on the device, cuff rupture, and eventual device failure.**

First-pass insertion success for the various SADs depends on the device, the indication for placement, the setting (e.g., prehospital, Emergency Department, or Operating Room), and the provider level performing the procedure. Success rates between 72% and 95% are common when researching the literature. One study showed an 81% first-pass success rate when used as a primary airway and an 84% first-pass success rate when used as a rescue airway.[27]

A B

FIGURE 25-5. The King Laryngeal Tubes. ***A.*** King LTD models. ***B.*** King LTS-D models. (Photos courtesy of King Systems Inc., Noblesville, IN.)

Begin ventilating the patient. Attach a bag-valve device to the SAD airway tube and begin ventilations. Minor manipulations of the SAD may be required to maximize the ease of ventilation in each patient. Slightly rotate or withdraw the SAD with the nondominant hand while ventilating through the device until ventilations are smooth and without resistance. Repositioning of the airway device during its use over time may be required in up to 18% of patients.[25]

PEDIATRIC CONSIDERATIONS

A SAD can be used for pediatric patients.[28-36] The SAD is mostly used in adults in the Emergency Department. There are limited studies showing the use in pediatric patients in the Emergency Department. Most studies are based on use in the Operating Room or on simulations. Manufacturers make many of the SADs in pediatric sizes. There is no good reason to not use a SAD as a rescue airway device if an appropriate-sized SAD is present in the Emergency Department. A Combitube or an EasyTube can be used in adolescents (Chapter 27).

ASSESSMENT

The appropriately placed and positioned SAD allows for air movement into the trachea, with varying degrees of potential air leakage into the digestive system during positive-pressure ventilation. Successful placement will allow for relatively effortless gas movement between the patient and the bag-valve device. Successful placement of the SAD is most accurately confirmed by auscultation of bilateral breath sounds, chest wall rise and fall movement with ventilation, and continuous end-tidal CO_2 capnography waveforms. One may gain a sense of correct placement by inserting the gastric sump tubing. The successful insertion of the sump tubing will help verify that the tip of the inserted device has not folded back upon itself, potentially obstructing gas flow.[37] The sump tubing has the added benefit of decompressing the stomach.

There does exist the possibility of failure of insertion, migration of the device after correct initial placement, and failure to maintain an adequate internal seal, which prevents effective patient ventilation via positive-pressure ventilation.[4] **If the patient cannot effectively be ventilated or oxygenated, remove the SAD and return to the BLS skills until additional expertise arrives or another airway option is available and initiated.**

AFTERCARE

Secure the SAD to the patient similar to the process used for ET tubes. This may require device-specific tube ties or straps, twill tape, or adhesive tape. **Periodically reevaluate the SAD function and placement to ensure its position has not changed and it is still providing appropriate oxygenation and ventilation. The SAD may need an occasional position adjustment.** Place the patient on a ventilator. Use a flexible elbow between the SAD and ventilator circuit tubing to prevent any potential for airway displacement during unsupervised use.

The SAD does not protect against aspiration as well as an ET tube. Use the gastric suctioning channels, if present, to decompress the stomach and limit the potential for subsequent aspiration. Replace the SAD with an ET tube or surgical airway once the airway management has migrated from emergent to urgent. The actual method of replacement will be determined by the patient's condition, presence of a "difficult airway," available equipment, and experience of the Emergency Physician.[38-42] Some SADs allow for the blind passage of an ET tube through the airway tube. Other SADs are designed with slit-like apertures that will prevent the passage of an ET cuff.

COMPLICATIONS

There are numerous documented complications associated with the use of SADs.[43] One of the most obvious and potentially devastating is the failure to place the device successfully or to obtain a satisfactory seal to allow for efficient oxygenation and ventilation. The incidence of ventilation failure is quite low. Failure to place a SAD appropriately has been estimated to occur in up to 5% of all insertions. The SAD may become displaced during transport or patient movement. Studies on the i-Gel show that device rotation may be required in up to 45% of insertions after correct placement, some minor mandibular manipulation in 23%, and some element of cervical extension to maintain seal in up to 20%. Additionally, up to 5% of patients may require manual securing of tube insertion to correct depth.[44] Herniation of the epiglottis tip is a documented complication associated with SAD use. It is increasingly noted in anesthesia fiberoptic evaluation of device use and may be of varying clinical significance. Up to 50% of insertions evaluated with fiberoptics may show the tip of the epiglottis in the bowl of the device.[45-47] The tip of the King tube may be malpositioned in the airway instead of the esophagus.[47]

There is some controversy in the medical literature as to potential of carotid artery compromise, even with correct anatomic placement. Porcine studies have demonstrated reductions in carotid blood flow and vessel diameter during imaging of use in cardiac arrest. Reduction in carotid blood flow has been demonstrated during routine use in the Operating Room, but the actual clinical impact is uncertain.[48] There are imaging studies with SADs demonstrating no carotid compression when used during sedation for magnetic resonance imaging procedures.[49,50]

The insertion of a SAD into the airway can result in mucosal damage and abrasions, especially if not properly lubricated or forcefully inserted. Unilateral vocal cord paralysis can occur secondary to traumatic insertion. Bilateral vocal cord paralysis has not been reported. Dental trauma may occur during the insertion or during maintenance of the airway.

The large and thin cuffs may be torn during use.[51,52] Overinflation of balloons and cuffs can result in tissue ischemia, erosion, and rupture. Herniation of pharyngeal and airway structures into the SAD has been documented using fiberoptic airway evaluation.[46] Trapping of the epiglottis in the distal aperture of the SAD may result in edema of the epiglottis. The oropharyngeal balloons have been linked to the development of tongue swelling secondary to venous congestion as a potential complication.[51,53,54] Overinflation of balloons and cuffs can result in mucosal ischemia, erosion, and lumen rupture. Soft tissue complications (e.g., sore throat, abrasions, and dysphagia) are present after use in 15% to 30% of cases.[43,55]

Cranial nerve palsies are rare.[43,55,56] They are being increasingly reported with awareness of their existence. The associated dysphagia, dysphonia, and hoarseness are thought to be related to airway manipulation and soft tissue complications following surgery. Not until a careful evaluation of the patient does one find a cranial nerve deficit. Lingual nerve injury is the most common cranial nerve injury. It is typically self-limiting and may take up to 6 months for resolution. This is thought to be secondary to pressure just below the mucosa of the inner mandible below the roots of the third molar, resulting in tongue numbness and dysgeusia. Hypoglossal nerve damage is related to cuff position. Neurapraxia due to overinflation of the cuff at the level of the greater horn of the hyoid bone results in nerve compression. Recurrent laryngeal nerve dysfunction is most devastating. The recurrent laryngeal nerve is typically compressed bilaterally against the cricoid cartilage where it enters the larynx passing under the inferior constrictor muscles. The resulting nerve dysfunction allows for vocal cord paralysis with the vocal cords stuck in the paramedian position.[55]

TABLE 25-1 Some of the Commercially Available SADs

Name	Manufacturer	Comments
CobraPLA	Pulmodyne	Available in adult and pediatric sizes 0.5–6 Accommodates ET tube ≤ 8.0 mm inner diameter Disposable single use High-volume, low-pressure oropharyngeal cuff Improved distal curve Large inner diameter Soft and pliable Softer tube and head
CobraPLUS	Pulmodyne	Distal gas sampling Same as CobraPLA except contains a temperature monitor
i-Gel	Intersurgical	Accommodates ET tube 6.0–8.0 mm inner diameter Available in adult and pediatric sizes 1–5 Can use fiberoptic bronchoscope through device Cuff matches the anatomy of laryngeal inlet Gastric channel Integrated bite block Noninflating cuff Oral cavity stabilizer Wide airway channel
i-Gel O_2	Intersurgical	Accommodates ET tube 6.0–8.0 mm inner diameter Available in adult sizes 3–5 Color-coded hook to attach airway strap Similar to i-Gel except supplementary oxygen port
King LT	Ambu	Available in adult and pediatric sizes 0–5 Blind distal tip Insertion marks printed on tube Oropharyngeal and esophageal low-pressure balloons Reusable and disposable single-use versions
King LTS	Ambu	Second lumen allows gastric suctioning with ≤ 18 Fr catheter Second lumen is esophageal lumen on posterior Similar to King LT except double-lumen tube

Inappropriate SAD insertion or cuff overinflation in the patient with an intact gag reflex can induce emesis and aspiration.[57] Both are associated with decreased survival.[1,58,59] **Always be vigilant to suspect, prevent, and identify aspiration in these patients.** When the SAD permits, provide gastric decompression via suction tube prior to device removal or additional airway manipulation.[60]

SUMMARY

SADs can be effectively used for primary and secondary airway management in the Emergency Department and the prehospital environment. A highly competitive marketplace has allowed for a wide variety of constructions. These devices are relatively simple and quick to insert. They provide an effective bridge between the tracheobronchial tree and the ventilating device. The Emergency Physician must be familiar with their use and limitations.

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26 Laryngeal Mask Airways

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INTRODUCTION

The term supraglottic airway (SGA) or supraglottic airway device (SAD) describes a heterogeneous group of airway devices that allow ventilation and oxygenation by sitting above the glottis. The first device of this kind was the laryngeal mask airway (LMA). SGAs are classified according to their characteristics into first-generation and second-generation devices. First-generation devices include the LMA Classic, LMA Unique, LMA ProSeal, LMA Supreme, and the first version of the Air-Q. Second-generation devices generally have an additional conduit for orogastric tube insertion and higher oropharyngeal leak pressures, and some of them allow SGA-guided fiberoptic intubation.[1,2] SADs result in less cervical spine movement than traditional laryngoscopes.[3] This can be useful for prehospital providers and the Emergency Department patient with potential cervical spine injury.

The LMA is a device that fills the gap in airway management between that of endotracheal (ET) intubation and the use of a face mask. It was introduced in the United Kingdom in 1983 by British anesthesiologist A. I. J. Brain. His goal was to develop an airway apparatus that could rapidly overcome an obstructed airway, is simple to use, and is atraumatic to insert. The LMA was approved for use in the United States by the Food and Drug Administration in 1991.

The LMA was designed primarily as a means of providing ventilatory support while avoiding the fundamental disadvantage of the need to visualize and penetrate the vocal cords with an ET tube.[4] The LMA is introduced into the hypopharynx without direct visualization. It forms a low-pressure seal around the laryngeal inlet and permits positive-pressure ventilation. Pressures of up to 30 cmH_2O may be administered safely with the introduction of the LMA ProSeal (A. I. J. Brain, MD, personal communication). The LMA may be used as a conduit for fiberoptically guided ET intubation or to place an ET tube blindly.[5] The LMA has come to be viewed as a viable method of airway management, with over 1000 articles and case reports describing the advantages and disadvantages of the device.[6] A more recent Medline search for articles involving the use of LMAs yielded over 6000 results.

Many disadvantages of the standard LMA became apparent with widespread use of the device. More than 10 years after its introduction, Dr. Brain and colleagues began to work on a new airway system with better intubation characteristics than the standard LMA. The intubating laryngeal mask airway (ILMA) was developed through the aid of analysis of magnetic resonance images of the human pharynx and laboratory testing of ET tubes.[7] The new and more "anatomically correct" ILMA effects more precise placement. The design of the ILMA also avoids head and neck manipulation and insertion of the intubator's fingers into the patient's mouth, both of which occur during the placement of the standard LMA.[7,8]

There are approximately nine different models of the LMA (**Table 26-1**). The term "Laryngeal Mask Airway" is specific to one brand of laryngeal mask devices produced by Teleflex (Research

TABLE 26-1 Some of the LMAs Available Commercially

Name	Manufacturer	Comments
AES Laryngeal Masks	AES Inc.	Similar to LMAs Reusable and single-use versions Made of 100% silicone or silicone cuff and flexible PVC tubes Some models come with a cuff manometer to monitor pressure Available in adult and pediatric sizes depending on model
Air-Q Blocker Laryngeal Mask	Cookgas	Disposable Soft and flexible guide tube next to airway tube Guide tube allows decompression and suction of esophagus and stomach Available in adult sizes 2.5, 3.5, and 4.5 Accommodates ET tube (ETT) ≤ 8.5 mm inner diameter Uses blocker tube to suction pharynx and block upper esophagus
Air-Q Laryngeal Mask	Cookgas	Hypercurved Larger mask Resists kinking of mask upon insertion Integrated bite block Reusable and single-use versions Available in adult and pediatric sizes 1.0, 1.5, 2.0, 2.5, 3.5, and 4.5 Accommodates ETT ≤ 8.5 mm inner diameter Removal after ET intubation with removal stylet
Air-Q SP Laryngeal Mask	Cookgas	Reusable and single-use versions Self-pressurizing mask requires no inflation port and line Mask inflates with ventilations Mask deflates upon exhalation to positive end-expiratory pressure level Available in adult and pediatric sizes 1.0, 1.5, 2.0, 2.5, 3.5, and 4.5 Accommodates ETT ≤ 8.5 mm inner diameter
AuraFlex Laryngeal Mask	Ambu	Disposable Wire reinforced Integrated pilot tube Extra soft cuff Available in adult and pediatric sizes 2–6
AuraGain Laryngeal Mask	Ambu	Anatomic curve Allows decompression and suction of esophagus and stomach Allows ≤ 14 French gastric tube for suction Can intubate with ETT through device Allowable size of ETT printed on device Available in adult sizes 3, 4, and 5
Aura-I Laryngeal Mask	Ambu	Disposable Preformed anatomically correct curve Integrated bite block Available in adult and pediatric sizes 1–6 Specifically designed for ETT through device
AuraOnce Laryngeal Mask	Ambu	Disposable MRI safe Preformed anatomically correct curve Molded in one piece with integrated inflation line No epiglottic bars on cuff Allows access of fiberoptic bronchoscope Recommendation to intubate over Aintree device Available in adult and pediatric sizes 1–6
AuraStraight Laryngeal Mask	Ambu	Disposable MRI safe Extra soft cuff Molded in one piece No epiglottic bars on cuff Available in adult and pediatric sizes 1–6
Aura40 Laryngeal Mask	Ambu	Same as AuraOnce except reusable Available in adult and pediatric sizes 1–6
Aura40 Straight Laryngeal Mask	Ambu	No epiglottic bars on cuff Available in adult and pediatric sizes 1–6
LMA Classic	Teleflex	Reusable Available in adult and pediatric sizes 1–6

(*Continued*)

TABLE 26-1 Some of the LMAs Available Commercially (*Continued*)

Name	Manufacturer	Comments
LMA Classic Excel	Teleflex	Reusable Same as LMA Classic except improved design facilitates ET intubation Available in adult sizes 3–5 Accommodates ETT ≤ 7.5 mm inner diameter
LMA Fastrach	Teleflex	Disposable and reusable versions ET intubation through device Available in adult sizes 3–5 Allows ventilation between intubation attempts Accommodates proprietary ETT from company 6.0–8.0 mm inner diameter
LMA Flexible	Teleflex	Reusable and disposable single-use versions Reinforced tube Available in adult and pediatric sizes 2–6
LMA ProSeal	Teleflex	Double cuff; second cuff allows tighter seal Minimizes posterior pharyngeal wall damage Drain tube separates airway and esophageal tracts Available in adult and pediatric sizes 1–5
LMA Protector	Teleflex	Disposable single use Preformed fixed curve Gastric channel moves regurgitated gastric fluid away from airways Integrated cuff manometer Can use fiberoptic bronchoscope through device Available in adult sizes 3–5
LMA Supreme	Teleflex	Increased safety and ease of use Preformed fixed curve allows easy of insertion Allows higher seal pressures Gastric channel moves regurgitated gastric fluid away from airways Available in adult and pediatric sizes 1–5
LMA Unique EVO	Teleflex	Disposable single use Integrated cuff manometer Integrated bite block Available in adult sizes 3–5 Soft cuff and tube minimize airway trauma
LMA Unique with Cuff Pilot	Teleflex	Disposable single use Integrated cuff manometer Soft cuff and tube minimize airway trauma Available in adult and pediatric sizes 1–5
LMA Guardian	Teleflex	Disposable single use Integrated cuff manometer Integrated bite block Not available in United States
Portex Soft Seal Laryngeal Mask	Smiths Medical	Disposable single use Softer cuff than LMAs No epiglottic bars on cuff Wider ventilation orifice than LMAs Integrated inflation line Can use fiberoptic bronchoscope through device Accommodates ETT ≤ 7.5 mm inner diameter Available in adult and pediatric sizes 1–5
Solus satin Laryngeal Mask	Intersurgical	Disposable single use Softer airway tube than LMAs to provide flexibility and ease insertion User information on exposed airway tube Available in adult sizes 3–5
Solus Standard Laryngeal Mask	Intersurgical	Disposable single use Softer airway tube than LMAs to provide flexibility and ease insertion User information on exposed airway tube Available in adult and pediatric sizes 1–5

Triangle Park, NC). Several other manufactures also make laryngeal mask devices. Some of these will also be described in this chapter. The LMA Classic (LMA-C) is the original and most commonly used version. The LMA Classic Excel (LMA-CE) is more durable than the original LMA-C and can be reused up to 60 times. The LMA Unique (LMA-U) is a single-use disposable version of the LMA-C. The LMA Flexible is a wire-reinforced version of the LMA that is more flexible than the original version and resists kinking. It is used by Anesthesiologists for patients undergoing head and neck procedures. It is not used in the Emergency Department. The LMA Fastrach is a modified version of the LMA that allows ET intubation through the unit. It is also referred to as the ILMA. It allows ventilation during

intubation attempts. Its advantages include the following: it requires no manipulation of the head and neck, it can accommodate up to a size 8 ET tube, it facilitates one-handed insertion, it can be inserted from the patient's side or from above the head, and it can be used in conjunction with fiberoptic intubation. The LMA ProSeal (LMA-PS) features a cuff deflator, modified cuff design, dual tubes, and a bite block. Like the LMA-PS, the LMA Supreme (LMA-S) has a built-in drain tube and bite block. However, the LMA-S is intended for a single use and has a more curved airway tube than the LMA-PS. The LMA CTrach (LMA-CT) includes an insertion site for an ET tube and allows for direct visualization of the larynx using built-in fiberoptics. The Ambu Laryngeal Mask (Ambu LM) has an airway tube that is curved more acutely than that of the LMA-C. This curve follows the anatomy of the upper airway and allows for easier insertion without having to manipulate the head or neck.

The standard LMA, ILMA, LMA-PS, LMA-CT, or Ambu LM may be available in the Emergency Department. The techniques for inserting these devices are discussed in this chapter. The anatomic differences between these devices produce subtle differences in their insertion methods. The indications, contraindications, assessment, and complications associated with these devices are largely identical.

ANATOMY AND PATHOPHYSIOLOGY

The anatomy of the airway is briefly reviewed below (**Figure 26-1**). Refer to Chapter 9 for the complete details of the airway anatomy. The oral cavity is bounded by the hard palate, soft palate, the anterior portion of the tongue, and the reflection of the tongue mucosa onto the floor of the mouth below. The mouth opens into the oropharynx through the oropharyngeal isthmus. The pharynx is a U-shaped tube extending from the base of the skull to the level of the cricoid cartilage, at which point it becomes continuous with the esophagus.[9] The larynx extends from its oblique opening bordered

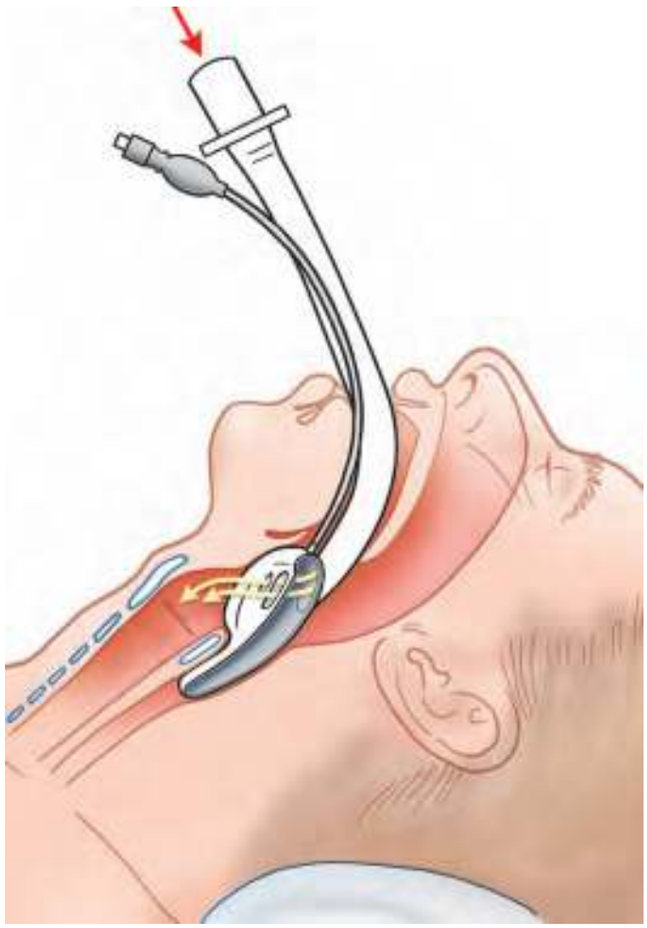

FIGURE 26-2. Sagittal view of the airway demonstrating correct placement of the LMA.

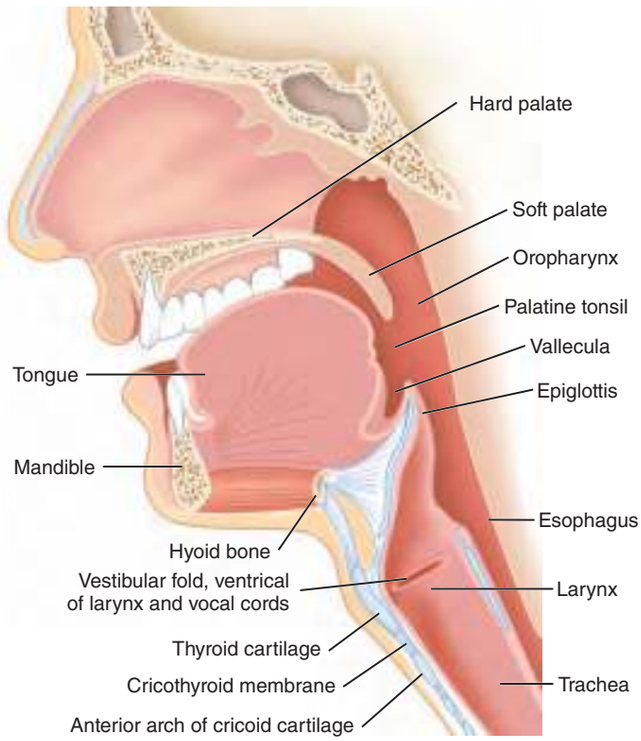

FIGURE 26-1. Midsagittal section of the head and neck demonstrating the airway anatomy.

by the aryepiglottic folds, the tip of the epiglottis, and the posterior commissure to the base of the cricoid cartilage.[9] The esophagus lies posterior to the airway. The tip of the device will lie in the esophagus at the level of the upper esophageal sphincter and directly posterior to the cricoid cartilage when inflated and properly positioned (**Figure 26-2**).

INDICATIONS

The indications for the use of an LMA device parallel the general indications for active airway management.[10,11] These include the correction of hypoxemia or hypercarbia, the provision of controlled hyperventilation, the provision of a secure airway in the presence of obstruction, and the provision of airway access for pulmonary hygiene and bronchoscopy. An LMA may aid in supporting airways that are difficult to manage as well as being an invaluable aid to blind and fiberoptic intubation.[12] The success rate of correct placement in inexperienced hands approaches 90%.[13] The LMA is superbly suited for use by medical personnel who have had only a minimal amount of training in airway management.[14]

Airway control also facilitates emergent radiographic investigations (e.g., computed tomography [CT] or magnetic resonance imaging [MRI] scans without motion artifact).[9] The standard LMA contains no ferromagnetic components and is a suitable alternative to an ET tube in many situations. It is ideal for use in patients emergently requiring diagnostic MRI scans. Use of the LMA does not require a metal laryngoscope, which is contraindicated if a patient requires airway management in the MRI suite.

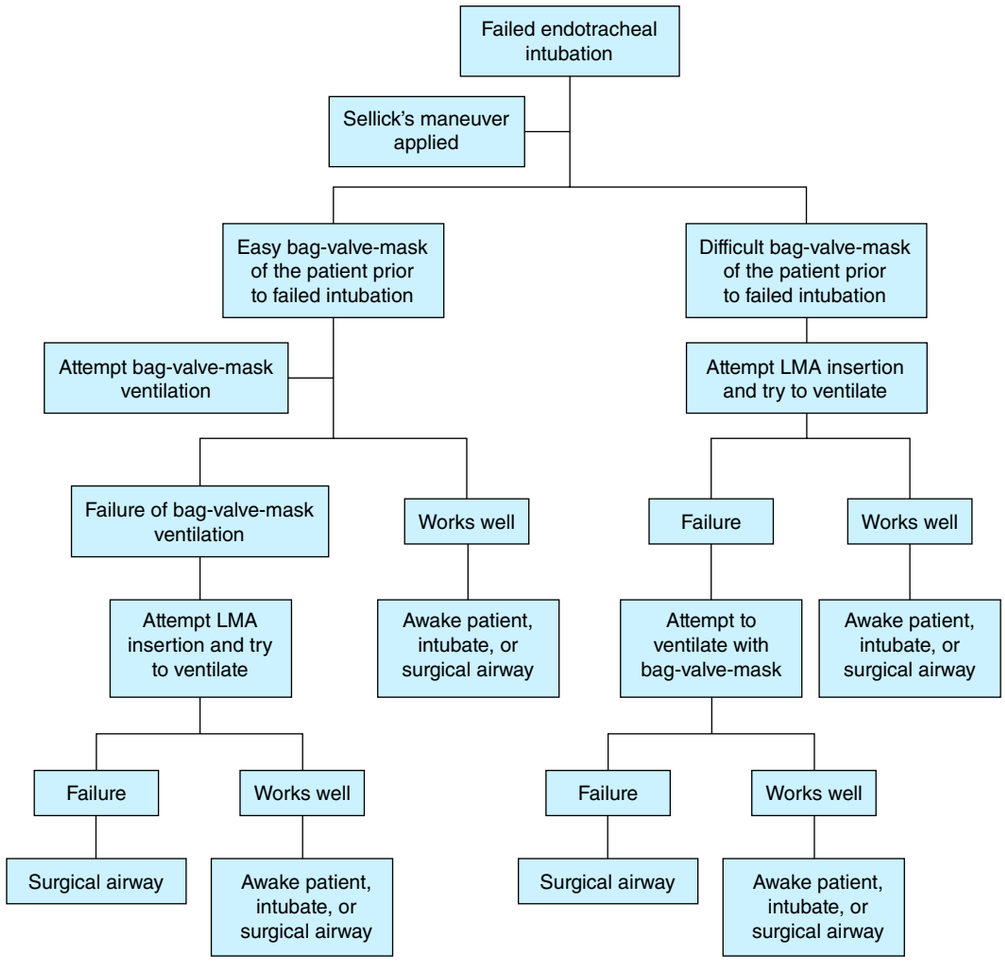

FIGURE 26-3. An algorithm for the use of a LMA in failed orotracheal intubation.

LMA devices may be used in the event of a failed ET intubation (Figure 26-3).[10,15,16] They have a role in securing the airway presumptively in patients with an "anteriorly" situated larynx, a situation whereby direct laryngoscopy and ET intubation are historically difficult. The LMA is a safe alternative to the esophageal obturator airway, King tube, Combitube, and EasyTube. The LMA has proved to be useful in burn patients requiring repeated dressing changes. The LMA may well be the airway technique of choice for professional singers who require short-term airway management since there is less likelihood of causing vocal cord or laryngeal nerve injury.[13]

There is a conspicuous absence of airway protection from aspiration.[17] The LMA-PS and LMA-S are being marketed as superior to the LMA-C for use in nonfasting patients. These versions feature an improved laryngeal seal for permitting positive-pressure ventilation at pressures up to 30 cmH_2O and a drain tube in tandem with the airway tube. The drain tube facilitates blind insertion of a gastric tube for decompressing the stomach. An introducer aids insertion of the LMA-PS while obviating the need to introduce fingers into the patient's mouth.

CONTRAINDICATIONS

There are no absolute contraindications to the use of LMAs. However, there are several relative contraindications. These devices should not be used in individuals who are at an increased risk of regurgitation or aspiration unless the benefit of securing an airway outweighs the risk of aspiration (e.g., when other techniques for securing the airway have failed).[18] This is supported by uncontrolled studies using fiberoptic bronchoscopy, which have shown that the esophagus is visible within the LMA mask in 6% to 9% of patients.[13] Patients at high risk for aspiration include those with previous upper gastrointestinal surgery, known or symptomatic hiatal hernia, gastroesophageal reflux disease, women more than 10 weeks pregnant, patients with intestinal ileus or peptic ulcer disease, obese patients, or those individuals who are not fasted.[8,19,20]

The LMA is not an appropriate airway device in emergent situations where cricoid pressure is needed to prevent active or passive regurgitation of stomach contents prior to airway placement. It will not prevent aspiration of stomach contents as efficiently as will a cuffed ET tube. The act of placing an LMA after the application of cricoid pressure (i.e., the Sellick maneuver) has been shown to have a significantly high failure rate.[13] The LMA-PS and LMA-S feature dual tubes (i.e., airway and drain) as well as a modified cuff designed to provide separation of the respiratory and alimentary tracts. They may prove invaluable in negating this "Achilles' heel" of the standard LMA.

These devices should not be used in individuals with severe respiratory diseases.[5,19,20] Patients with airway obstruction at or below the larynx and those with low pulmonary compliance or high airway resistance (e.g., morbid obesity, bronchospasm, pulmonary edema, pulmonary fibrosis, or thoracic trauma) are not appropriate

candidates for LMAs. **The only possible exception is that it can be inserted as a temporary airway rescue device in preparation of another method of ET intubation or a surgical airway.**

Patients must be able to assume the "sniffing" position, analogous to that of individuals positioned for direct laryngoscopy prior to ET intubation. Patients who cannot passively or actively extend their head and flex their neck are not candidates for the LMA-C, LMA-CE, LMA-U, or LMA-S. The ILMA, LMA-CT, LMA-PS with an introducer, and Ambu LM are more appropriate for these patients. Patients should not receive these devices if they cannot open their mouth at least 1.5 cm due to anatomic limitations (e.g., ankylosing spondylitis, severe rheumatoid arthritis, and cervical spine instability).[13] This is one situation where blind nasotracheal intubation or fiberoptically guided nasotracheal tube placement has an advantage over the LMAs.

The LMA is relatively contraindicated in cases of pharyngeal pathology (e.g., abscesses, caustic ingestions, hematomas, and tissue disruptions). These processes make the use of the LMA difficult. The device may rupture an abscess or a hematoma and cause the patient to aspirate. The use of an LMA after a caustic ingestion can result in perforation of the eroded upper esophageal or hypopharyngeal walls from the pressure of the inflated cuff.

There are numerous cautions that go with the SAD. It should not be used for routine airway management in nonfasted individuals. Trismus or limited mouth opening limits its use. Do not allow high peak ventilatory pressures. Do not use force to place the SAD or a nasogastric tube. Ensure appropriate anesthesia levels. Do not leave the SAD in place for extended times. Either perform an orotracheal intubation, awaken the patient, or perform a surgical airway. Do not reuse an airway meant for single use.

EQUIPMENT

- LMAs, various types and sizes (**Table 26-1**)
- ET tubes
- Water-soluble lubricant
- Syringes, 10 and 20 mL
- Oxygen source, tubing, and regulator
- Face masks
- Bag-valve device
- Pulse oximeter
- Cardiac monitor
- Noninvasive blood pressure monitor
- Advanced Cardiac Life Support (ACLS) medications
- Advanced airway equipment
- Surgical airway equipment

Many Emergency Physicians have difficulty in picking the appropriate-sized LMA for children. Two formulas have been proposed[21]: (1) the LMA size equals the log of the patient's weight in kilograms divided by 0.6; round this result up or down to the nearest half number; or (2) the cuff inflation volume is five times the LMA size. These formulas are only guides and the LMA size and cuff volume may need to be adjusted to the size of the patient and the air leak.

LMA CLASSIC (LMA-C) AND UNIQUE (LMA-U)

The standard LMA preceded the ILMA by more than a decade. The prototype of the LMA was constructed by forming a shallow mask with an inflatable rubber cuff joined to a tube communicating with the lumen of the mask at an angle.[4,5] The modern LMA is made of flexible silicone, is completely latex free, and has a more tapered

FIGURE 26-4. The LMA-C. (Photo courtesy of Teleflex, Research Triangle Park, NC.)

appearance. It has a variably sized, internally ridged tube fused at a 30° angle to a spoon-shaped mask with a flexible rim (**Figure 26-4**).

The LMA-C is a disposable unit that can be used multiple times. It must be sterilized between uses following specific manufacturer recommendations. The LMA-U is similar in construct to the LMA-C. However, the LMA-U is a single-use device. These LMAs are designed to conform to the contours of the hypopharynx with the lumen facing the glottic opening (**Figure 26-2**).

They consist of an airway tube, inflation line, and mask (**Figure 26-4**). Overall, they resemble a giant spoon. The airway tube has a large bore and is clear.[5] The proximal end contains a standard 15 mm airway adapter that can connect to a bag-valve device or a ventilator. A black line along the posterior border is used as a marker for proper positioning. The distal end of the airway tube connects to the mask.

The mask is elliptical in shape (**Figure 26-5**). The outer rim of the mask contains an inflatable cuff. The tip of the LMA will lie in the esophagus at the level of the upper esophageal sphincter and directly posterior to the cricoid cartilage when inflated and properly positioned (**Figure 26-2**). The lateral edges of the mask rest in the pyriform fossae. The upper edge rests against the base of the tongue. The LMA provides a seal against the upper esophageal sphincter, aryepiglottic folds, and distal epiglottis to direct air into the trachea and avoid insufflation of the stomach (**Figure 26-2**).[5,6]

The distal end of the airway tube opens into the mask. This opening is covered by two vertical aperture bars that prevent the epiglottis from obstructing the lumen of the airway tube (**Figure 26-4**). **The aperture bars should be cut off prior to inserting the LMA if an ET tube is to be inserted through the LMA.**

A

B

FIGURE 26-5. The distal end or mask of the LMA-C. ***A.*** The deflated cuff. ***B.*** The inflated cuff.

The inflation line is used to inflate and deflate the cuff (**Figure 26-5**). The distal end of the inflation line attaches to the upper border of the cuff. The proximal end contains an inflation port and balloon like an ET tube. An air-filled syringe attaches to the inflation port to inflate the cuff.

The correct size of the LMA is based on the patient's weight and is crucial to ensure a proper seal and reduce complications (**Table 26-2**).[18] The distal end assumes a different shape when the cuff is inflated and deflated. The distal end is pentagon-shaped when deflated (**Figure 26-5*A***). It is oval-shaped when inflated (**Figure 26-5*B***).

LMA CLASSIC EXCEL (LMA-CE)

Although similar in construct to the standard LMA, the LMA-CE has been designed as an "enhanced" version of the LMA-C (**Figure 26-6**). The airway tube has been reinforced, allowing the LMA-CE to be used up to 60 times. It must be sterilized between uses following specific manufacturer recommendations. The inflatable cuff is made of soft silicone to minimize throat stimulation and irritation. The LMA-CE is available in three sizes (**Table 26-3**).

Several modifications have been made to facilitate ET intubation with the aid of a fiberoptic bronchoscope. The airway tube has a 15 mm connector that can be easily removed to allow for access. The airway tube has been designed to accommodate up to a 7.0 cuffed ET tube. It is short enough that the cuff of the ET tube can pass through the LMA-CE and beyond the vocal cords. The mask aperture includes a vertically oriented epiglottic elevating bar, also known as the EEB (**Figure 26-6**). The EEB is free at the caudal end and fixed at the cephalad end, effectively creating a hinge mechanism. An ET tube passed through the mask aperture swings the EEB backward and elevates the epiglottis away from the path of the advancing ET tube.

LMA FASTRACH (ILMA)

The LMA Fastrach is also known as the intubating LMA or ILMA (**Figures 26-7 and 26-8**). The form of the ILMA was derived from head and neck sagittal MRI studies in normal subjects whose heads were held in a neutral position.[7] The convex radius of the curve of the silicone-covered steel tube represents a value close to the best-fit curve derived from the MRI studies.[7] The new prototype consists of an anatomically curved steel tube connected to standard LMA cuff sizes 3, 4, and 5.[8,19]

The ILMA has several significant modifications that make it different from the LMA-C (**Figures 26-7 and 26-8**). It is available in a reusable stainless steel version and a plastic single-use disposable version (**Figure 26-7*A***). Both versions are available in three sizes (**Table 26-4**). The airway tube is stainless steel covered with silicone rubber. The proximal end has a handle fused to the airway tube to facilitate insertion, manipulation, and removal of the ILMA. It is curved to follow the curve of the hypopharynx and position the mask aperture over the glottic aperture. It has a larger diameter of 13 mm versus 9 mm with the LMA.[19] This allows the

TABLE 26-2 LMA-C and LMA-U Size Selection

Patient's weight (kg)	LMA-C and LMA-U size	Maximum cuff inflation volume (mL)	Largest ET tube size*	Fiberoptic bronchoscope size (mm)
< 5	1.0	4	3.5	2.7
5–10	1.5	7	4.0	3.0
10–20	2.0	10	4.5	3.5
20–30	2.5	14	5.0	4.0
30–50	3.0	20	6.0 (cuffed)	5.0
50–70	4.0	30	6.0 (cuffed)	5.0
70–100	5.0	40	7.0 (cuffed)	5.0
> 100	6.0	50	7.0 (cuffed)	5.0

*The inner diameter in millimeters.

FIGURE 26-6. The LMA-CE. (Photo courtesy of Teleflex, Research Triangle Park, NC.)

TABLE 26-3 LMA-CE Size Selection

Patient's weight (kg)	LMA-CE size	Maximum cuff inflation volume (mL)	Largest ET tube size*
30–50	3.0	20	7.0 (cuffed)
50–70	4.0	30	7.5 (cuffed)
70–100	5.0	40	7.5 (cuffed)

*The inner diameter in millimeters.

TABLE 26-4 LMA Fastrach or ILMA Size Selection

Patient's weight (kg)	ILMA size	Maximum cuff inflation volume (mL)	Largest ET tube size*
30–50	3.0	20	7.0 (cuffed)
50–70	4.0	30	7.5 (cuffed)
70–100	5.0	40	8.0 (cuffed)

*The inner diameter in millimeters.

A

B

FIGURE 26-7. The ILMA. ***A.*** The reusable device (left) and the single-use device (right). (Photo courtesy of Teleflex, Research Triangle Park, NC) ***B.*** The mask.

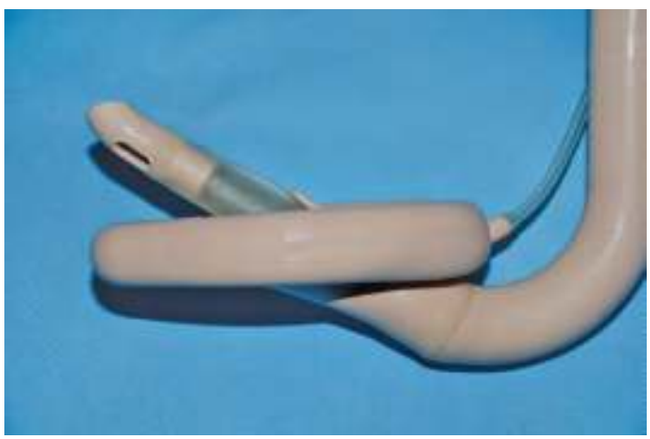

FIGURE 26-8. An ET tube is inserted through the ILMA. The tip of the ET tube is guided by the V-shaped EEB.

ILMA to accommodate a cuffed ET tube with an inner diameter up to 9.0 mm.[8,22] It is significantly shorter at 14.5 cm versus 20 cm for the LMA-C.[19]

The mask of the ILMA is like that of the LMA with two major modifications. The ILMA contains a ramp inside the distal airway tube as it meets the mask and continues into the mask aperture. It is designed to direct the ET tube into the center of the aperture and into the patient's airway. It also has a large, single, and stiff EEB designed to lift the epiglottis out of the way of the advancing ET tube (**Figures 26-7*B* and 26-8**). The ILMA avoids hyperextension of the head and neck.[23] This is advantageous in the patient with a potential cervical spine injury.

The ILMA was designed to be used with a wire-reinforced cuffed silicone ET tube with an 8.0 mm inner diameter (**Figure 26-9**).[8] The ILMA ET tube is available in 6.0, 6.5, 7.0, 7.5, and 8.0 mm sizes. The molded tip allows atraumatic insertion through the vocal cords. It has a transverse black line along its posterior surface. This line serves as a marker to let the intubator know when the tip of the ET tube is positioned at the EEB. This occurs when the ET tube is inserted through the ILMA and the transverse bar is located at the proximal end of the airway tube. While it is ideal to use the wire-reinforced silicone ILMA ET tube, a standard ET tube may also be used.[24] The LMA can be inserted in a difficult airway and the patient intubated using the retrograde guidewire technique (Chapter 30).[25]

LMA CTRACH (LMA-CT)

The LMA-CT is basically an ILMA with built-in fiberoptics that allow direct visualization of airway anatomy (**Figure 26-10**). Two fiberoptic channels lie along the lateral edges of the airway tube.

FIGURE 26-9. The silicone ET tube and pusher used with the ILMA. (Photo courtesy of Teleflex, Research Triangle Park, NC.)

FIGURE 26-10. The LMA-CT. (Photo courtesy of Teleflex, Research Triangle Park, NC.)

These channels merge and exit under the EEB. The properly positioned LMA-CT fiberoptics project an image of the laryngeal inlet directly in front of the mask aperture. A portable color display, the LMA-CT Viewer, attaches magnetically to the top of the LMA-CT airway tube. This allows the intubator to monitor the passage of the ET tube through the vocal cords. The LMA-CT Viewer lies just above the patient's chin and in line with the actual airway anatomy, making intubation easier from a hand-eye coordination standpoint. Images may be recorded and downloaded onto a computer for later documentation and teaching purposes. The use may result in longer intubation times, more maneuvering, and more sore throats than the use of the ILMA, especially in morbidly obese patients.[26] One study showed insertion and ventilation rates of 100% and an intubation rate of 90%.[27]

It is easy to use a LMA CT. A clear view of the vocal cords allows intubation. If the epiglottis is folded on the EEB, perform the up-down maneuver by pulling the LMA out approximately 6 cm and reinserting it with the cuff inflated. The view can be foggy or blurry. Ventilate the patient gently and several times to clear the view. Turn on the light source if the view is dark.

LMA PROSEAL (LMA-PS)

The LMA-PS has been designed to be inserted manually or with the aid of a metal introducer (**Figure 26-11**). The use of the introducer obviates the need for the intubator to place their fingers in the patient's mouth. The LMA-PS has been modified to allow for ventilation at higher airway pressures. A combination of a softer silicone cuff, an additional rear cuff in larger sizes, and a deeper bowl mask permits higher seal pressures up to 30 cmH_2O. This is approximately 50% higher than with the LMA-C. The LMA-PS provides a tighter seal without increasing mucosal pressure or the risk of complications. It is a reusable device that is available in numerous sizes (**Table 26-5**).

The LMA-PS features a dual tube (i.e., drain tube and airway tube) system that decreases the risk of rotational dislodgement of the device. The drain tube communicates with the upper esophageal sphincter, allowing for blind insertion of an orogastric tube and for

FIGURE 26-11. The LMA-PS. (Photo courtesy of Teleflex, Research Triangle Park, NC.)

venting of gastric gases and liquids. The drain tube is positioned in the cuff to prevent the epiglottis from blocking the airway tube. The LMA-PS has been designed to decrease the risk of aspiration. Despite this, the manufacturer cautions that the LMA-PS does not provide complete protection against aspiration.

LMA SUPREME (LMA-S)

The LMA-S, like the LMA-PS, is a dual tube system that provides some separation of the respiratory and digestive tracts. The LMA-S is a single-use device and has an airway tube that is more curved and anatomically shaped (**Figure 26-12**). It has a more rigid airway tube than the LMA-PS; thus, it requires no digital or metal introducer for placement. It can be used for the blind passage of an orogastric tube. The drain tube can help monitor correct positioning of the LMA-S. Gases will audibly leak from the drain tube if the LMA-S does not have a tight airway seal. The LMA-S incorporates a bite block. It is available in numerous sizes (**Table 26-6**). One study has shown the LMA-S to be equivalent to ET intubation.[28]

TABLE 26-5 LMA-PS Size Selection

Patient's weight (kg)	LMA-PS size	Maximum cuff inflation volume (mL)	Largest ET tube size*	Largest size OG tube/Salem Sump
5–10	1.5	7	4.0	10 French/8 French
10–20	2.0	10	4.5	10 French/8 French
20–30	2.5	14	5.0	14 French/12 French
30–50	3.0	20	5.0	16 French/14 French
50–70	4.0	30	5.0	16 French/14 French
70–100	5.0	40	6.0 (cuffed)	18 French/16 French

*The inner diameter in millimeters.

FIGURE 26-12. The LMA-S. (Photo courtesy of Teleflex, Research Triangle Park, NC.)

FIGURE 26-13. The Ambu LM.

AMBU LARYNGEAL MASK (AMBU LM)

The Ambu LM (Ambu Inc., Glen Burnie, MD) consists of an airway tube, spoon-shaped mask, and inflatable cuff (**Figure 26-13**). However, the airway tube of the Ambu LM is bent into a smooth curve of approximately 90° to conform to the anatomy of the upper airway and to facilitate easier insertion. Because of this anatomically correct curve, the device can be placed without manipulating the patient's head or neck. The airway tube is D-shaped and easier to grip than other tubes. The distal tip of the Ambu LM is reinforced so that the cuff does not fold over during insertion. The Ambu LM comes in multiple sizes and styles (**Table 26-7**).

KING LARYNGEAL AIRWAY DEVICE (KING LAD)

The King LAD (King Systems, Noblesville, IN) is a disposable, single-use, silicone device like the LMA-C. It is available in two styles (**Figure 26-14**) and a wide range of sizes in each style. The standard model has a curved airway tube, whereas the flexible model has a straight and easily bendable airway tube. The flexible model is reinforced with a wire.

AIR-Q MASKED LARYNGEAL AIRWAY (AIR-Q)

The Air-Q Masked Laryngeal Airway or Air-Q (Mercury Medical, Clearwater, FL) is another commonly used device (**Figures 26-15*A* and 26-15*B***). It is available in a full range of sizes (**Table 26-8**).

TABLE 26-6 LMA-S Size Selection

Patient's weight (kg)	LMA-S size	Maximum cuff inflation volume (mL)	Largest ET tube size*	Largest size OG tube
< 5	1	5	4.0	6 French
10–20	2	12	4.5	10 French
30–50	3	30	5.0	14 French
50–70	4	45	5.0	14 French
70–100	5	45	6.0 (cuffed)	14 French

*The inner diameter in millimeters.

The airway tube shape and mask inlet have a different shape than the LMA-S. An ET tube can be inserted through the Air-Q like the ILMA. Its shape may make head positioning irrelevant.[29]

The Air-Q Blocker Masked Laryngeal Airway or Air-Q Blocker (Mercury Medical, Clearwater, FL) has been modified from the Air-Q to include a soft guide tube along the side of the airway tube (**Figure 26-15*B***). A lubricated suction catheter or nasogastric tube (up to size 18 French) can be inserted and directed into the posterior pharynx and esophagus to suction secretions. A proprietary suction catheter with an inflatable balloon near the distal end can be inserted through the guide tube. The inflated balloon blocks the upper esophagus to prevent aspiration. The tip of this catheter has multiple holes to allow esophageal venting while the balloon prevents aspiration. The Air-Q Blocker is available in three sizes (**Table 26-8**).

The Air-Q Self-Pressurizing Masked Laryngeal Airway or Air-Q SP (Mercury Medical, Clearwater, FL) is like the Air-Q except for one major structural change. The Air-Q SP has a self-pressurizing cuff that does not require a pilot balloon, inflation line, or syringe to inflate the cuff (**Figures 26-15*D* and 26-15*E***). Positive-pressure ventilation provided by a bag-valve device or ventilator inflates and self-pressurizes the cuff. The Air-Q SP is available in a full range of sizes (**Table 26-8**).

INTERSURGICAL I-GEL

The i-Gel (Intersurgical Inc., East Syracuse, NY) is a second-generation SGA (**Figure 26-16**). It is made from a thermoplastic elastomer (i.e., styrene ethylene butadiene styrene [SEBS]) with a soft durometer and gel-like feel. The softness was designed to match the patient's soft tissues orally, maintain its shape, and facilitate insertion. The noninflating cuff creates a fit with the perilaryngeal and hypopharyngeal structures after placement.[30] It has a gastric port (i.e., the second lumen) that allows placement of an oropharyngeal tube. The i-Gel has an integrated bite block. It can also provide a channel for ET intubation.[31] The i-Gel is available for adult and pediatric patients (**Table 26-9**). Advantages of this SGA include easy insertion, less airway morbidity, and higher airway peak pressures.[32] The success of ventilation is decreased with the application of cricoid pressure.[33] The i-Gel is easy to use in infants and adults.[34,35]

TABLE 26-7 Ambu Size Selection

Patient's weight (lb)	Ambu AuraFlex size*	Ambu Aura40 Straight size*	Ambu Aura40 size*	Ambu AuraStraight size*	Ambu AuraOnce size*	Maximum cuff inflation volume (mL)
< 11	N/A	1	1	1	1	4
11–22	N/A	1.5	1.5	1.5	1.5	7
22–44	2	2	2	2	2	10
44–66	2.5	2.5	2.5	2.5	2.5	14
66–110	3	3	3	3	3	20
110–154	4	4	4	4	4	30
154–220	5	5	5	5	5	40
> 220	6	6	6	6	6	50

N/A, not applicable.*The inner diameter in millimeters.

MANOMETERS

Newer versions of the LMA contain a built-in manometer attached to the cuff inflation port.[36] This improves the cuff inflation volume administered initially as well as changes to the cuff volume over time. The older versions of the LMA can avoid cuff overinflation by insertion with half of the maximum volume or insertion with the resting cuff volume.[36]

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure if time exists, and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away because the procedure can be disconcerting to some parents.

The patient should be appropriately monitored with electrocardiography (ECG), end-tidal CO_2 monitoring, noninvasive blood pressure cuff, and pulse oximetry. The patient preparation is the same as that for orotracheal intubation. **As for any situation where airway manipulation is to occur and the patient's protective airway reflexes are blunted or ablated, a fully functioning suction apparatus with a variety of catheters must be immediately available.** Insertion of the LMA requires an anesthetic depth like that for the placement and acceptance of an oropharyngeal airway.[13] Successful placement of the standard LMA is much more likely if the patient is premedicated. **The successful placement of the ET tube is highly dependent on adequate sedation and/or muscle relaxation.** The optimal induction agent should produce jaw relaxation and attenuation of airway reflexes and permit the insertion of the LMA device within 30 to 60 seconds of loss of consciousness. A variety of induction agents may be used.

There is some controversy as to what physical examination findings represent the end point for judging when to insert the LMA. The consensus is that the first attempt at insertion should occur following the loss of the eyelash reflex (i.e., seventh cranial nerve); the jaw should be relaxed at this point.[20,37] This typically occurs 30 to 60 seconds after administration of the ultra-short-acting induction agents. Some practitioners also rely on the onset of apnea and/or loss of response to verbal stimuli as signs of adequate depth of anesthesia.[16]

FIGURE 26-14. The King LAD standard model (left) and flexible model (right).

TECHNIQUES

LMA CLASSIC, UNIQUE, AND CLASSIC EXCEL

Prior to insertion, carefully inspect the cuff for leaks with the cuff slightly overinflated. Completely deflate the cuff so that it forms a smooth wedge shape. The technique for inserting the LMA-C, LMA-U, and LMA-CE is rather simple (**Figure 26-17**). Lubricate the posterior surface of the LMA with a water-soluble lubricant. Care must be taken to avoid lubricating the anterior surface of the device, as the gel might obstruct the distal aperture or trickle into the larynx and provoke laryngospasm.[13] **Avoid using silicone-based lubricants that may degrade the cuff.[18] Avoid lubricants containing lidocaine as they may provoke an allergic reaction or decrease laryngeal protective reflexes.[18]**

Position the patient's head in the sniffing position if not contraindicated. Place the nondominant hand behind the patient's head to stabilize the occiput and slightly flex the neck (**Figure 26-17*A***). Allow the patient's jaw to fall open. An assistant may be required to help open it.

Insert the LMA into the oral cavity with the aperture facing but not touching the tongue (**Figure 26-17*A***). **It is essential that the leading edge of the cuff be smooth, wrinkle-free, and shaped like a wedge.** This facilitates passage of the cuff around the posterior pharyngeal curvature and into the hypopharynx while avoiding the epiglottis. Place the index and middle fingers of the dominant hand against the junction between the LMA and the cuff (**Figure 26-17*B***). Advance the LMA in one smooth movement following the curvature of the pharynx until it enters the hypopharynx (**Figure 26-17*B***). The fingers should lie almost horizontally

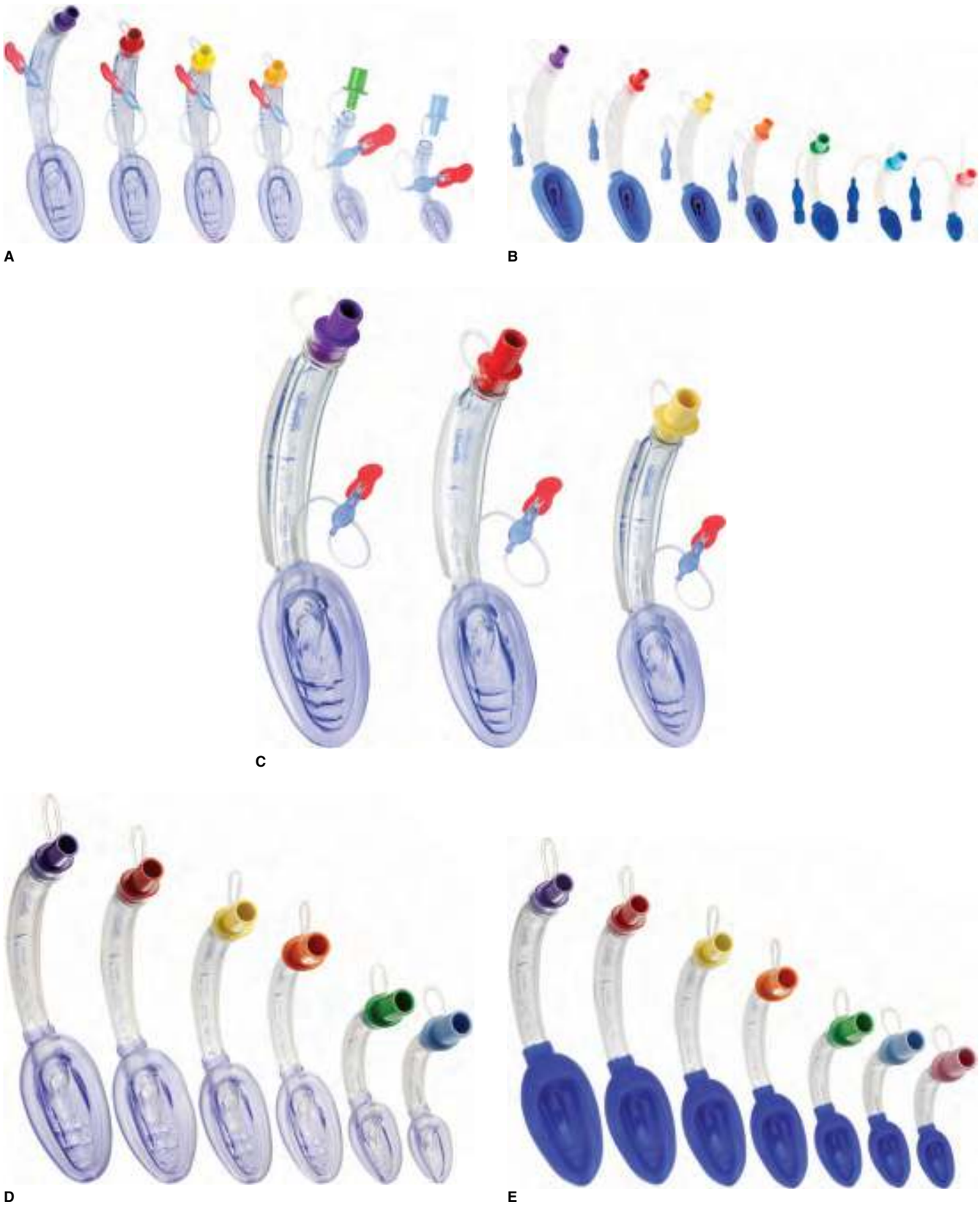

FIGURE 26-15. The Air-Q Masked Laryngeal Airway. ***A.*** The disposable Air-Q. ***B.*** The reusable Air-Q. ***C.*** The Air-Q Blocker. ***D.*** The disposable Air-Q SP. ***E.*** The reusable Air-Q SP. (Photos courtesy of Mercury Medical Inc., Clearwater, FL.)

TABLE 26-8 Air-Q, Air-Q Blocker, and Air-Q SP Size Selection

Patient's weight (kg)	Size	Maximum cuff inflation volume (mL)	Largest ET tube size*	Air-Q sizes	Air-Q Blocker sizes	Air-Q SP sizes
< 7	1.0	3	4.5	X		X
7–17	1.5	5	5.0	X		X
17-30	2.0	8	5.5	X		X
30–50	2.5	12	6.5	X	X	X
50–70	3.5	18	7.5	X	X	X
70–100	4.5	25	8.5	X	X	X

*The inner diameter in millimeters.

TABLE 26-9 i-Gel Size Selection

Patient's weight (kg)	i-Gel size	Largest cuffed ET tube size*	Type of patient	Maximum size of nasogastric tube (Fr)
2–5	1.0	3.0 mm	Neonate	N/A
5–12	1.5	4.0 mm	Infant	10
10–25	2.0	5.0 mm	Small pediatric	12
25–35	2.5	5.0 mm	Large pediatric	12
30–60	3	6.0 mm	Small adult	12
50–90	4	7.0 mm	Medium adult	12
> 90	5	8.0 mm	Large adult	14

N/A, not applicable.
*The inner diameter in millimeters.

when the LMA is properly positioned.[38] Grasp and stabilize the airway tube with the nondominant hand. Remove the index and middle fingers of the intubating hand (**Figure 26-17*C***). **Slightly advance the LMA further downward until resistance is felt. It is important to not push further.** If difficulty is encountered, a rotational movement of the tube, slight inflation of the cuff, a jaw-thrust maneuver, or the use of a laryngoscope may be helpful.[13]

Inflate the cuff with the recommended volume of air (**Figure 26-17*D***). **Do not overinflate the cuff. Inflation usually causes a characteristic outward movement of the airway tube of up to 1.5 cm as the cuff centers itself around the laryngeal inlet.** A slight forward movement of both the thyroid and cricoid cartilages will be noted. **The longitudinal black line on the shaft of the tube should lie in the midline against the upper lip. Any deviation may indicate the wrong size device was used, misplacement of the cuff, or a partial airway obstruction.**[13] The tip of the LMA cuff lies at the base of the hypopharynx against the upper esophageal sphincter, the sides lie in the pyriform fossae, and the upper border of the mask lies at the base of the tongue pushing it forward when correctly positioned.[13] The mask may still create a useful airway when grossly malpositioned.[18] Secure the LMA like an ET tube.

LMA PROSEAL

Carefully inspect the cuff for leaks with the cuff slightly overinflated. Completely deflate the cuff so that it forms a smooth wedge shape. The LMA-PS features a cuff deflator. It is a compact, portable instrument for assuring complete removal of air without causing the silicone to wrinkle. Lubricate the posterior surface of the LMA-PS with a water-soluble lubricant. Insert the LMA-PS like that described above for the LMA-C. The only difference is that the fingertip should be pushed into the introducer strap at the rear of the cuff.

FIGURE 26-16. The i-Gel. (Photo courtesy of Intersurgical, East Syracuse, NY.)

An alternative method of insertion involves a metal introducer. Head and/or neck manipulation may not be required when using the introducer. Place a properly sized introducer into the strap. Fold the tubes around the convex surface of the introducer and fit the proximal end of the airway tube into the matching slot. Insert the LMA-PS into the oral cavity with the aperture facing, but not touching, the tongue. **The back of the mask must remain in constant contact with the hard palate.** Rotate the LMA-PS inward in one smooth movement following the curvature of the introducer until it enters the hypopharynx and resistance is felt. Grasp and stabilize the airway tube with the nondominant hand. Remove the introducer. Inflate the cuff and secure the LMA-PS. The LMA-PS includes a built-in bite block.

LMA SUPREME

Carefully inspect the cuff for leaks with the cuff slightly overinflated. Completely deflate the cuff so that it forms a smooth wedge shape. Lubricate the posterior surface of the LMA-S with a water-soluble lubricant. Position the patient's head in a semi-sniffing position. The neutral position or a full "sniffing" position may preclude proper placement of the LMA-S.

Insert the LMA-S. Grasp the LMA-S by the connector end. Insert the LMA-S into the oral cavity with the aperture facing, but not touching, the tongue. Briefly rub the mask tip across the palate to lubricate the area. Rotate the LMA-S inward in one smooth movement following the curvature of the pharynx until it enters the hypopharynx and resistance is felt. Directing the distal tip toward the right or left side of the throat may facilitate placement. Grasp and stabilize the airway tube with the nondominant hand. Inflate the cuff and secure the LMA.

LMA FASTRACH (ILMA)

The technique for inserting the ILMA is not very different from that for the standard LMA. It involves a one-handed rotational movement in the sagittal plane with the patient's head supported to achieve a neutral position.[8] The ILMA may be inserted from above the patient's head or standing to the side of the patient's head. It may be inserted with the right or left hand.

Prior to insertion, slightly overinflate the cuff and check it for leaks. Completely deflate the cuff. Lubricate the posterior surface of the airway tube and the mask liberally. Grasp the ILMA by its handle. Place the patient in the sniffing position if no contraindications exist. Open the patient's mouth with the nondominant hand. Position the ILMA over the patient with the tip of the mask in the

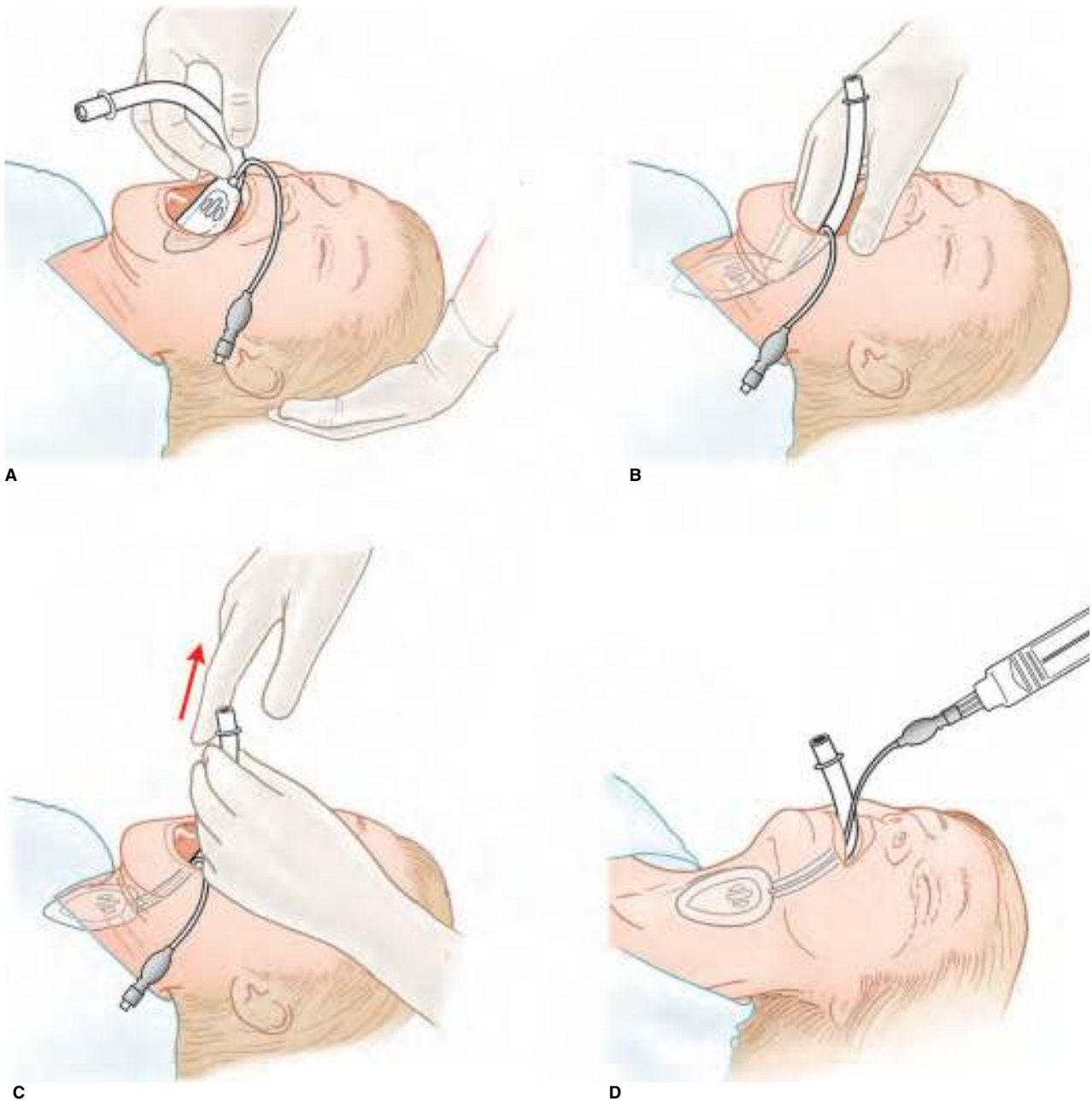

FIGURE 26-17. Insertion of the LMA. ***A.*** The patient's head is properly positioned, and the LMA is inserted into the patient's mouth. ***B.*** The LMA is advanced with two fingers. ***C.*** The LMA is stabilized while the insertion hand is removed. ***D.*** The cuff is inflated.

patient's mouth (**Figure 26-18*A***). Slowly insert the mask while the posterior aspect of the mask remains in constant contact with the hard palate. When the entire mask is inside the patient's mouth and against the hard palate, rotate the ILMA inward along the natural curve of the hard palate and pharynx (**Figure 26-18*B***). The airway tube should maintain constant contact with the upper central incisors as the unit is advanced. Stop advancing the unit when resistance is felt. This signifies that the tip of the mask is in the upper esophagus (**Figure 26-18*B***).

Inflate the cuff with the recommended volume of air (**Figure 26-18*C***). **Inflation usually causes a characteristic outward movement of the airway tube, up to 1.5 cm, as the cuff centers itself around the laryngeal inlet.** A slight forward movement of the thyroid and cricoid cartilages will be noted. The airway tube should lie in the midline against the upper central incisors. **Any deviation may indicate the misplacement of the cuff and a partial airway obstruction.** The tip of the ILMA cuff lies at the base of the hypopharynx against the upper esophageal sphincter, the sides lie in the pyriform fossae, and the upper border of the mask lies at the base of the tongue pushing it forward when correctly positioned.

Confirm proper placement of the ILMA. Have an assistant attach a bag-valve device to the proximal end of the airway tube and ventilate the patient. Observe the upper chest rise, auscultate bilateral breath sounds, and observe end-tidal CO_2 monitoring to confirm proper placement. An anterior movement, or bulging, of the cricoid and thyroid cartilages during or after cuff inflation also indicates correct positioning of the ILMA.[4]

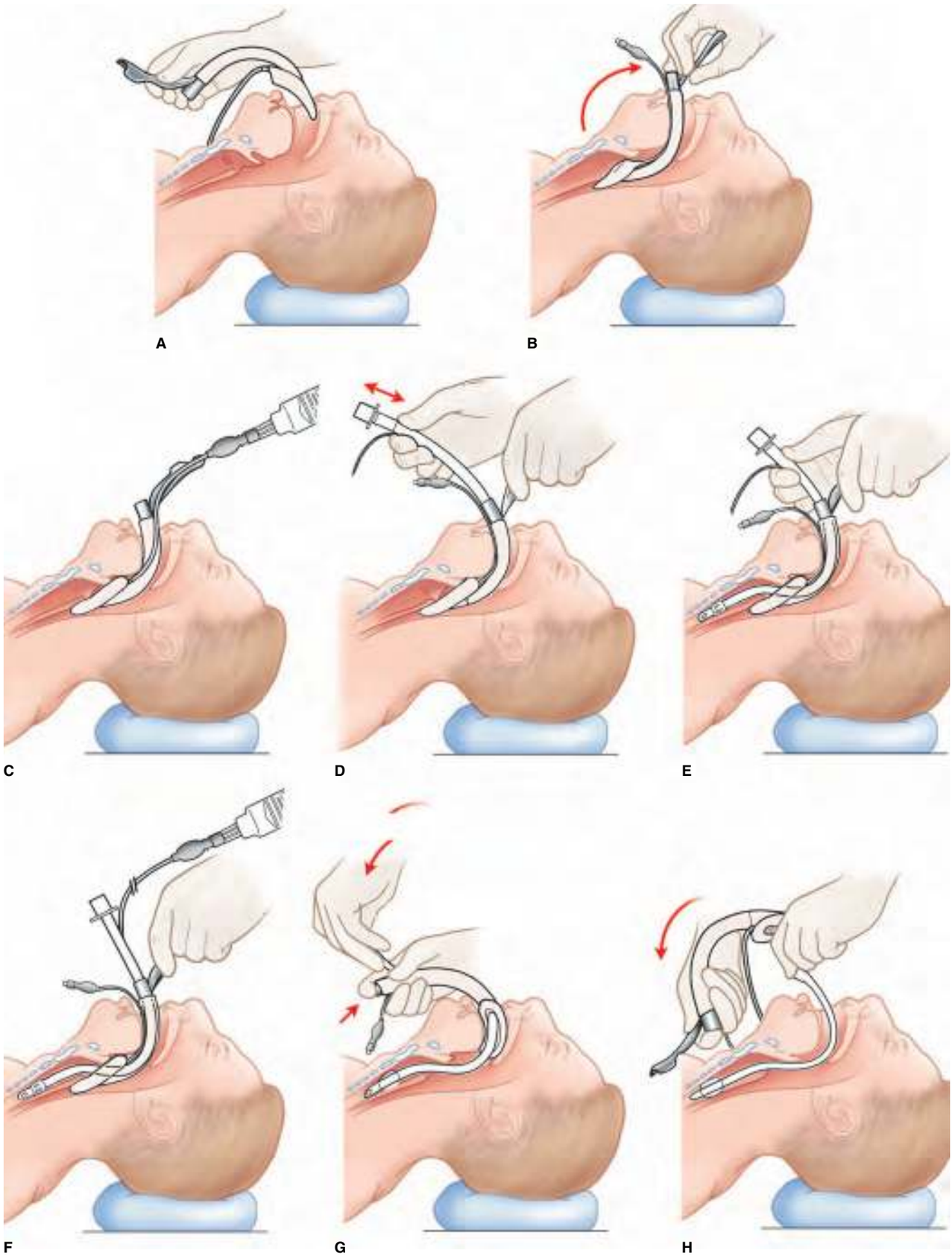

FIGURE 26-18. Insertion of the LMA Fastrach or ILMA. ***A.*** The ILMA is inserted. ***B.*** The ILMA is advanced until resistance is encountered. ***C.*** The cuff is inflated. ***D.*** The ILMA is stabilized and the ET tube is inserted. ***E.*** The ET tube is advanced into the trachea. ***F.*** The ET tube cuff is inflated. ***G.*** The ILMA is carefully removed. ***H.*** When the ILMA has exited the patient's mouth, grasp and stabilize the ET tube. Completely remove the ILMA.

Insert an ET tube. Lubricate the wire-reinforced silicone ET tube or a standard ET tube liberally. Insert the silicone ET tube into the ILMA until the transverse black line on its posterior surface is at the proximal end of the airway tube (**Figure 26-18*D***). The tip of the silicone tube will be just inside the distal end of the airway tube. An assistant can connect a bag-valve device to the silicone ET tube and ventilate the patient if necessary. Make sure that the longitudinal black line on the posterior surface of the silicone ET tube is facing upward.

Slowly and gently advance the silicone ET tube 1.5 cm beyond the transverse black line. **The tip of the silicone tube is just past the vocal cords if no resistance to its advancement is felt.** Continue to advance the silicone ET tube an additional 4 cm (**Figure 26-18*E***). The patient can be ventilated by an assistant during this procedure if necessary. Inflate the cuff of the silicone ET tube and ventilate the patient through the silicone ET tube (**Figure 26-18*F***). Confirm proper tube placement by the auscultation of breath sounds, observation of chest rise, and end-tidal CO_2 monitoring.

The ILMA should now be withdrawn. Deflate the cuff of the ILMA. Have an assistant remove the bag-valve device and the 15 mm adapter on the proximal end of the silicone ET tube. Withdraw the ILMA by gently reversing the ILMA over the silicone ET tube (**Figure 26-18*G***). **Simultaneously apply slight pressure to the proximal end of the silicone ET tube so that it does not become dislodged (Figure 26-18*G*).** Stop withdrawing the ILMA when the mask begins to exit the patient's mouth. **Grasp the silicone ET tube firmly at the patient's mouth and hold it securely.** Withdraw the ILMA in a smooth curved motion (**Figure 26-18*H***). Reattach the standard respiratory connector, ventilate the patient, and reconfirm proper placement of the silicone ET tube.

Some Physicians prefer to use a "pusher" to prevent accidental extubation while the ILMA is being withdrawn. Cut a 25 cm length from a second silicone ET tube. Insert this into the ILMA as it is being removed. Apply slight pressure so it pushes against the first ET tube and prevents it from moving proximally. Remove the ILMA and pusher as a unit when the ILMA exits the patient's mouth. Secure and assess the proper positioning of the ET tube, as mentioned previously.

LMA CTRACH (LMA-CT)

The technique for inserting the LMA-CT is similar to that for the ILMA. The LMA-CT may be inserted from above the patient's head like the LMA-C or standing to the side of their head. Place the patient in neutral position to avoid head extension. The manufacturer recommends only using the LMA-CT with straight, wire-reinforced cuffed silicone ET tubes with a 6.0 to 8.0 mm inner diameter. Standard curved plastic ET tubes may lead to an increased incidence of laryngeal trauma.

Insert the LMA-CT using the technique described for the ILMA. Inflate the cuff with the recommended volume of air and confirm proper placement of the ILMA. Have an assistant attach a bag-valve device to the proximal end of the airway tube and ventilate the patient. Observe the upper chest rise, auscultate bilateral breath sounds, and observe end-tidal CO_2 monitoring to confirm proper placement.

Attach the Viewer by placing the Viewer's socket onto the magnetic latch connector on the LMA-CT. Turn on the Viewer to visualize the glottis. Lubricate the wire-reinforced silicone ET tube. Grasp the LMA-CT by its handle and pass the ET tube back and forth through the airway tube several times to lubricate the entire airway tube. **Do not pass the lubricated silicone ET tube beyond the transverse black line on its posterior surface to avoid obscuring the fiberoptics with lubricant.** Make sure that the longitudinal black line on the posterior surface of the silicone ET tube is facing upward.

Slowly and gently advance the silicone ET tube 1.5 cm beyond the transverse black line. Gripping the handle and lifting a few millimeters optimizes the alignment of the silicone ET tube and the trachea. The EEB will be seen to rise on the Viewer display as the silicone ET tube passes the mask aperture. Continue to advance the silicone ET tube through the vocal cords. The patient can be ventilated by an assistant during this procedure if necessary. Detach the viewer. Inflate the cuff of the silicone ET tube and ventilate the patient through the silicone ET tube. Confirm proper tube placement by the auscultation of breath sounds, observation of chest rise, and end-tidal CO_2 monitoring. Withdraw the LMA-CT as described for the ILMA.

OTHER LARYNGEAL MASK DEVICES

The Ambu Laryngeal Mask (Ambu LM), King Laryngeal Airway Device (King LAD), Air-Q Masked Laryngeal Airway (Air-Q), and the i-Gel can be inserted and secured like the LMA. An ET tube can be inserted through the i-Gel and Air-Q models. The use of a fiberoptic scope to guide intubation may be more desirable.[31]

ALTERNATIVE TECHNIQUES

Difficulty in insertion of the standard LMA occurs most frequently at the point where the tip of the mask passes just behind the tongue as it changes direction toward the hypopharynx.[39] Most of the suggested alternative methods for inserting the LMA involve the negotiation of direction change from the pharynx to the hypopharynx. It has been suggested that a partially inflated mask is easier to place in the correct position.[39] Others employ a jaw-thrust maneuver. The mask is positioned firmly and flatly against the hard palate after adequate jaw relaxation has been established. Perform the jaw-thrust maneuver with the nondominant hand while firmly thrusting the mask into place with the dominant hand, in one motion.[40] The jaw thrust creates a space in the hypopharynx for the mask. The use of a laryngoscope may help facilitate LMA placement, although this reduces the inherent simplicity of the technique of LMA insertion.

One study of the LMA-PS showed that a 90° rotation would improve rates of successful placement.[41] This technique was associated with a lower incidence of mucosal bleeding and sore throat. Insert the device until the entire cuff is inside the mouth. Rotate the LMA-PS 90° counterclockwise and advance it until resistance is felt. Straighten the LMA-PS when it is in the hypopharynx.

A few alternative methods of insertion exist with the ILMA. The metal handle on the tip of the ILMA tube may be used to modify the position of the cuff within the hypopharynx.[19] Pulling back on the metal handle toward the intubator rotates the tube caudally in the sagittal plane. Pushing on the metal handle away from the intubator rotates the tube cephalad in the sagittal plane.

ASSESSMENT

Successful placement of an LMA device is most accurately demonstrated by auscultation of bilateral breath sounds, chest wall movement, and end-tidal CO_2 monitoring (Chapter 19).[8,19] One may gain a sense of accuracy of placement during insertion. A bulging of the tissues overlying the larynx may be seen during observation of the front of the neck while inserting an LMA. Visualization of this bulge and increased resistance to forward motion of the mask indicates that the device is in the correct position.[4] Obtain a chest radiograph if an ET tube has been placed through an LMA. Any LMA used for a significant time, 30 to 60 minutes, should be checked to ensure that the LMA has not changed position.[42] This is best done with a fiberoptic bronchoscope (Chapter 28).

AFTERCARE

The LMA does not protect against aspiration as well as an ET tube. It should be replaced with an ET tube or a surgical airway. The method of securing the airway with a device other than an LMA will be determined by the patient's condition, if the patient has a difficult airway, available equipment, and experience of the Emergency Physician.

COMPLICATIONS

There are numerous documented complications associated with the use of LMAs.[43-45] One of the most obvious of these and potentially the most devastating is the failure to place the device successfully or to obtain a satisfactory laryngeal seal. The incidence of failure to achieve satisfactory ventilation is quite low, even in inexperienced hands. Two large retrospective studies that reviewed 11,910 and 15,795 surgical cases of the standard LMA and unique LMA noted an overall success rate of 99.8% and 98.9%, respectively.[46,47] Success has been classified as success on one single attempt and overall success. The overall success rate allows up to three attempts to be considered for successful placement. One study demonstrated a 99.5% single-attempt success rate in a retrospective analysis of 1500 cases.[20] Most studies indicate correct LMA placement in 88% to 90% of first attempts.[13] Several studies have found success rates of 90% to 99% for ILMAs. A small study showed a first-try rate of 96% using the Ambu AuraGain LMA.[48] Success rates appear to be higher if a wire-reinforced silicone ET tube is used rather than a standard ET tube.[49,50] Risk factors associated with LMA placement failure include rotation of the patient after placement, poor dentition, male sex, and increased body mass index.[51] The patients with LMA failure were noted to have a three fold increase in the incidence of difficult mask ventilation.[46]

There are other complications besides failure.[45] These are divided into minor complications and major complications. The complication rate is defined as events not attributable to the patient's underlying condition, to surgical interventions, or to other interventions. It should be equivalent to that seen during placement of either a Guedel or Berman type oropharyngeal airway. The complication rate when using the LMA is significantly lower than that occurring during direct laryngoscopy and ET intubation. This is potentially due to the intense autonomic nervous system stimulation occurring with the latter procedure.

MINOR COMPLICATIONS

Minor complications are those that may result in significant patient morbidity but usually are not associated with mortality or extremely deleterious outcomes. Stomach inflation can occur during positive-pressure ventilation at pressures greater than 20 cmH_2O for the standard LMA. Cuff herniation secondary to overinflation may result in failure of the cuff to seal effectively. Partial airway obstruction may occur in up to 10% of adults and 25% to 50% of pediatric patients when standard LMA cuffs are examined by fiberoptic bronchoscopy.[13] Trapping of the epiglottis in the distal aperture of the LMA may result in edema of the epiglottis. Air leaks around the cuff can occur during positive-pressure ventilation at pressures greater than 20 cmH_2O for the standard LMA and 30 cmH_2O for the LMA-PS. Forceful attempts to pass the LMA around the posterior pharyngeal curvature can result in uvular bruising.[13] Lingual nerve injury, tongue numbness, parotid gland swelling, and hypoglossal nerve palsy are sometimes noted.[52] Unilateral vocal cord paralysis can occur secondary to traumatic insertion. Bilateral vocal cord paralysis has not been reported. Dental trauma may occur during the insertion or during maintenance of the airway.

MAJOR COMPLICATIONS

Major complications are those from which significant patient morbidity may be expected, including patient mortality. Major complications are exceedingly rare. The LMA may be malpositioned.[53] Aspiration occurs in a few patients (0.08%).[17] A report of 11,910 surgical cases where the standard LMA was used had 18 critical events related to the LMA, for an overall incidence of 0.15%.[47] These events included regurgitation of stomach contents in 0.03%, vomiting of stomach contents in 0.017%, pulmonary aspiration in 0.009%, laryngospasm in 0.07%, bronchospasm in 0.025%, cardiac dysrhythmias in 0.09%, and cardiac arrest in 0.06%. Laryngospasm has been noted in other studies.[54] While there appears to be a higher incidence of critical events when the device is used for controlled ventilation and positive-pressure ventilation, the incidence has not proved to be statistically significant.

The use of a SAD in swine decreases carotid blood flow.[55] No studies in humans have been shown to confirm this finding.[56] If this turns out to be true, an LMA may not be indicated for certain patients.

SUMMARY

A major advancement in airway management was made with the introduction of the LMA. It is superior to a face mask in that it prevents supraglottic obstruction and reduces the likelihood of gastric insufflation.[5] **The LMA does not provide protection from aspiration.** The LMA-PS and LMA-S, with their dual tube system, may help decrease the risk of aspiration.

The standard LMA has clearly earned a valuable place in the armamentarium of airway management. The technique is easy to learn, easy to teach, and requires no specialized equipment. The LMA causes minimal autonomic nervous system activation and less of a response from the cardiovascular system than with direct laryngoscopy. The LMA is not associated with a risk of esophageal or endobronchial intubation. Both are possible complications following use of the ILMA. The LMA has minimal effects on the intraocular pressure response to airway manipulation. The LMA may be of use in cases of suspected cervical spine injury.

The use of an LMA in place of a face mask avoids many risks, such as injury to the eyes, supraorbital and facial nerves, nose, and lips. There is less risk of hand fatigue than with the bag-valve-mask device. The LMA provides a safer and more secure airway in children and adults than does a face mask, with fewer episodes of hypoxemia as detected by pulse oximetry.[13] Risk factors for LMA failure include poor dentition, male sex, and increased body mass index.[51]

The introduction of the ILMA more than a decade after the standard LMA further defined and expanded the role of this apparatus in airway management. It allows for precise ET tube placement. It is anatomically designed to ensure more accurate placement of the cuff. The placement of the ILMA does not need for the Emergency Physician to be positioned behind the patient's head. There may be a significant place for the ILMA in future airway management algorithms.[7,57] The advent of the LMA-CT allows for direct fiberoptic visualization of the vocal cords prior to intubation. Newer SGAs such as the Air-Q and the i-Gel follow the same principle and can be effective alternatives for fiberoptic-guided tracheal intubation via an SGA.

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27 Double Lumen Airway Tube Intubation

Joseph Weber and Katie Tataris

INTRODUCTION

The Esophageal-Tracheal Combitube (ETC; Medtronics, Minneapolis, MN) and the EasyTube (EzT; Rusch, Kernen, Germany) are double lumen airway devices that can be blindly inserted into the unconscious and unresponsive patient. The ETC and EzT function to adequately ventilate and oxygenate a patient while simultaneously protecting the airway from aspiration.[1,2] They are most often used in the prehospital setting by Emergency Medical Technicians not trained in standard orotracheal intubation and by paramedic-level rescuers as an alternative airway device when standard orotracheal intubation fails.[3-6] These devices are used in the Emergency Department primarily as a rescue airway. These are the only two double lumen devices used in the prehospital setting and the Emergency Department. The Emergency Physician should be familiar with these devices so that they can be removed and exchanged with an endotracheal tube if placed in the prehospital setting or if required to manage a difficult airway.

ANATOMY AND PATHOPHYSIOLOGY

The ETC is a double-tube, double lumen, and double-cuffed device (**Figure 27-1**). The ETC starts as two distinct tubes that fuse into one but remain functionally separated by a partition. The shorter clear tube is continuous with the distal open port and is known as the tracheoesophageal lumen. At its distal end is the distal tracheoesophageal cuff, similar to that of an endotracheal tube. It is a high-volume, low-pressure cuff that is inflated through the white inflation port. The longer blue tube is continuous with the eight perforations known as the proximal ports. A large pharyngeal cuff is just proximal to the perforations. This cuff is positioned between the base of the tongue and the palate. It separates the oral and nasal cavities from the remainder of the airway. It is inflated through the blue inflation port.

The EzT is also a double-tube, double lumen, and double-cuffed device with a structure similar to the ETC (**Figure 27-2**). It has several differences in comparison to the ETC (**Table 27-1**). The shorter clear tube ends as a conventional single-lumen 7.5 mm endotracheal tube. The EzT may be used as a primary airway device under direct laryngoscopy. When placed successfully into the trachea, it functions as a 7.5 mm endotracheal tube. If unable to pass it into the trachea, it can be advanced into the esophagus and the patient ventilated through the blue tube.[7,8] The longer blue tube ends in an open aperture rather than multiple perforations. The EzT is latex free.[7]

Both the ETC and EzT may be inserted blindly into a patient's airway. If the distal tip enters the trachea, the patient is ventilated through the shorter clear tube and the distal cuff prevents aspiration of gastric contents into the trachea. If the distal tip enters the esophagus, the patient is ventilated through the longer blue tube whose proximal ports lie in the hypopharynx and the distal cuff will occlude the esophagus.

Both devices are available in two sizes. The 37 French SA model of the ETC is meant for small adults. The manufacturer recommends its use in patients with a height of 122 to 168 cm (i.e., 4 to 5.5 ft). The 41 French model of the ETC is meant for larger adults with a height of 152 cm (i.e., 5 ft) and greater. Patients in the intermediate range of 152 to 168 cm (i.e., 5 to 5.5 ft) can use either model.

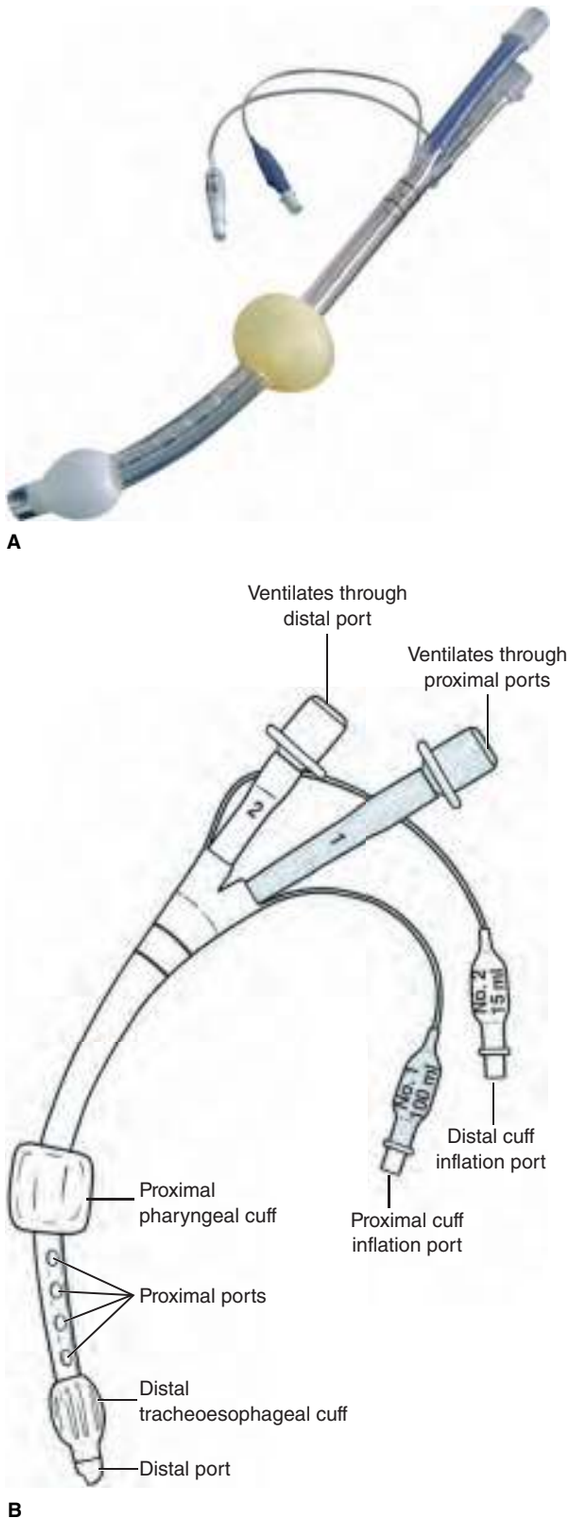
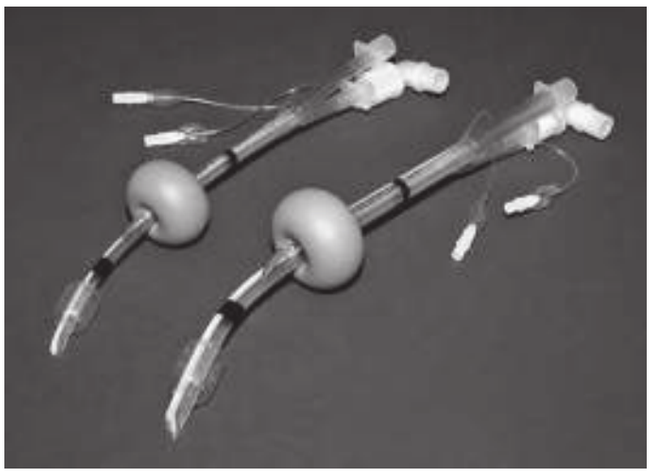


FIGURE 27-1. The Combitube. ***A.*** Photograph. ***B.*** Illustration.

A study demonstrated that the 37 French SA model can be used in patients up to 183 cm (i.e., 6 ft, 1 in) in height. The EzT is available in a 28 French model for patients with a height from 90 to 130 cm (i.e., 3 to 4.3 ft) and a 41 French model for patients over 130 cm (i.e., 4.3 ft). The lack of pediatric-sized double lumen airway tubes is a limitation.



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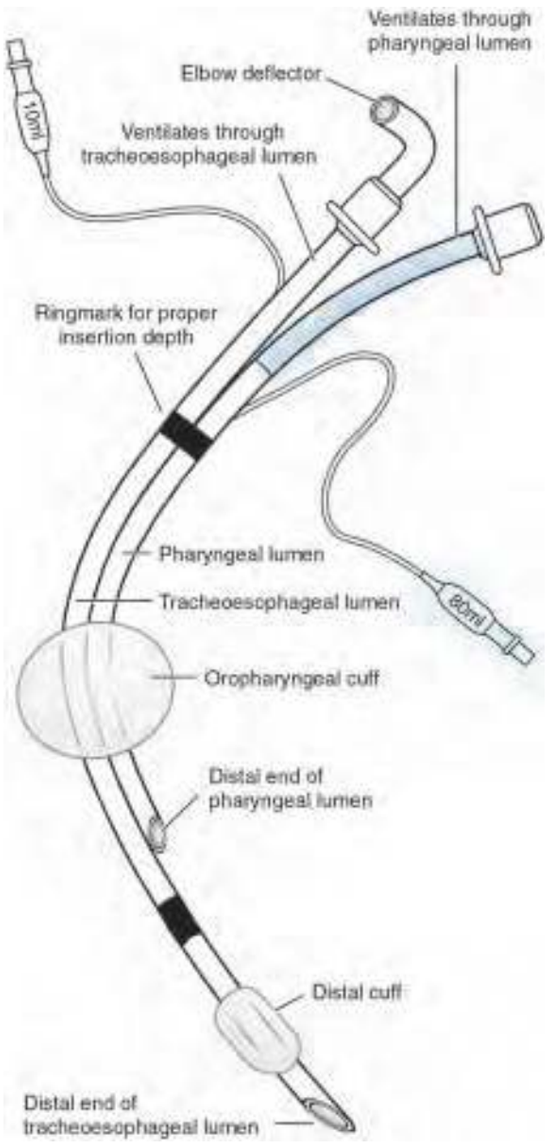

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FIGURE 27-2. The EasyTube. ***A.*** Photograph. (Used with permission from reference 12.) ***B.*** Illustration.

TABLE 27-1 Features of Double Lumen Airway Tubes

Name	Manufacturer	Comments
Combitube	Medtronic	Air-filled syringes for quick cuff inflation included Available in two sizes (37 and 41 French) Can be used as primary airway device or rescue airway device Cannot give medications through tube if placed in esophagus Can ventilate through either tube depending on placement Color-coded and numbered lumens with matching syringes Combines features of endotracheal tube (ETT) and esophageal obturator Disposable and single-use Distal esophageal low-pressure cuff Eight ventilatory holes Includes gastric suction catheter Inserted without a laryngoscope Larger proximal cuff for oropharynx Quick insertion with the head and neck in neutral position Second cuff in esophagus sealed to prevent gastric aspiration When placed in esophagus, allows for passage of gastric suction catheter
Rusch Easy Tube	Teleflex	Air-filled syringes for quick cuff inflation included Available in two sizes (28 and 41 French) Can be used as primary airway device or rescue airway device Can ventilate through either tube depending on placement Color-coded and numbered lumens with matching syringes Combines features of ETT and esophageal obturator Disposable and single-use Includes gastric suction catheter Inserted without a laryngoscope Open second proximal lumen Quick insertion with the head and neck in neutral position Second lumen allows passage of devices (fiberoptic intubation, suction catheter, or ETT exchanger) Single lumen at distal tip

Several attributes specific to these devices contribute to their usefulness in the acute setting. Both are effective as either a primary or backup airway management device. A patient can be ventilated with the tip positioned in either the esophagus or the trachea. Minimal training is necessary. There is no need for a laryngoscope. They are easily inserted with the patient's head and neck in a neutral position. The ETC and EzT are faster and easier to insert than an endotracheal tube in the cardiac arrest patient. This optimizes high-quality compressions and minimizes hands-off time.[9]

The dual lumens and cuffs offer certain advantages. The distal lumen of both allows for gastric suctioning when the tip is placed in the esophagus. The proximal lumen of the EzT is an open lumen rather than perforations, as in the ETC. This allows for the passage of a suction catheter, fiberoptic scope, or endotracheal tube changer with a maximum diameter of 3.99 mm into the trachea.[7] The cuffs firmly secure the device and make dislodgment unlikely. The proximal cuff can functionally tamponade oropharyngeal bleeding and serve to minimize the risk of aspirating oral debris.[10] These attributes make these devices suitable for rescuers of all skill levels.

The EzT was found to have easier and quicker insertion, higher oropharyngeal leak pressures, and lower peak airway pressures when it was compared to the ETC.[11]

INDICATIONS

The primary indication for an ETC or an EzT is as a backup device for airway management. It can be used for difficult or failed orotracheal intubations.[12-18] It should be placed on crash carts for use by individuals not skilled with orotracheal intubation. They can be used when standard orotracheal intubation cannot be performed. This includes limited access to the patient's head (e.g., extrication situations).[19] They should be considered in situations of potential cervical spine injury as the device can be inserted with the patient's head and neck in a neutral position. The ETC is an appropriate device for management of the airway when visualization is limited due to bleeding or secretions. All Emergency Physicians should become familiar with these devices if they are used by Emergency Medical Technicians in their region, and these devices should be considered as alternative airway devices for difficult airways in the Emergency Department.[10,20] These devices cause less cervical spine movement than direct laryngoscopy.[21] This makes these devices useful for intubation of trauma patients.

CONTRAINDICATIONS

Certain contraindications exist and should be addressed. **These devices should not be used on patients with an intact gag reflex.** There should be enough time to premedicate the patient and induce anesthesia before inserting the devices if intubation is anticipated. Size limits apply to the ETC and the EzT. The standard ETC cannot be used in patients under 5 ft tall. The smaller ETC SA model cannot be used in patients under 4 ft tall. The EzT cannot be used in patients under 3 ft tall. The ETC cannot be used in patients with a latex allergy. The EzT is latex free and can be used in all patients. Neither device can be used in small children.[22]

Their use is contraindicated if the patient has known esophageal disease or an airway obstruction. Patients with known esophageal disease (e.g., strictures, cancer, or victims of caustic ingestions) are at an increased risk of complications (e.g., perforation and failed performance). Patients with known upper airway obstruction (e.g., secondary to congenital disorders, cancer, or other anatomic abnormalities) are at increased risk for complication and performance failure.

EQUIPMENT

- Gloves, gown, and mask
- Eye protection, goggles, or a face mask with an eye shield
- Pulse oximeter
- Noninvasive blood pressure cuff
- Cardiac monitor
- Intravenous (IV) access equipment
- ETC or EzT kit
- Oxygen source and tubing
- Suction source and tubing
- Water-soluble lubricant
- Bag-valve-mask device
- Advanced Cardiac Life Support equipment and medications
- Surgical airway equipment

The ETC and the EzT are prepackaged in kit form with the dual lumen airway tube, two syringes for cuff inflation, a 90° elbow, and a flexible suction catheter (**Figures 27-3*A* and 3*B***). The ETC kit also contains a vomit deflector that is not routinely used. It may be associated with significant complications and gastric contents directed at the healthcare professional. The suction catheter included is designed to be inserted through the smaller tube and to exit the tracheoesophageal port.

PATIENT PREPARATION

Preoxygenate the patient with a bag-valve-mask device using 100% oxygen. Establish IV access. Apply the pulse oximeter, noninvasive blood pressure cuff, and cardiac monitor. Place the patient supine or in any position that may be required. Prepare the equipment while an assistant is ventilating the patient. Remove the ETC or EzT and equipment from the package. Attach the syringes to their respective ports and inflate the cuffs (**Figure 27-4**). Discard the device and open another kit if a leak of air is present or if the cuff does not inflate properly. Deflate the cuffs and leave the syringes attached to the ports and prefilled with the air needed to inflate

A

B

FIGURE 27-3. The contents of the double lumen kits. ***A.*** The Combitube kit. ***B.*** The EasyTube kit.

FIGURE 27-4. The Combitube (***A***) and the EasyTube (***B***) with their cuffs inflated. (Used with permission from reference 11)

on insertion. Liberally lubricate the tip of the device with water-soluble lubricant.

TECHNIQUE

Insert the thumb of the nondominant hand into the patient's mouth and over their tongue (**Figure 27-5*A***). Place the nondominant fingers under the chin. Depress the tongue and open the jaw simultaneously. Remove any dental devices and foreign bodies. Grasp the device with the dominant hand. The curve of the device should be positioned in the same direction as the natural curve of the pharynx (**Figure 27-5*A***). Insert the device into the midline of the patient's mouth. Advance the device in a downward curved motion until the patient's incisor teeth or alveolar ridge lies between the two printed bands of the ETC (**Figure 27-5*B***) or the black stripe of the EzT (**Figure 27-6**). **Do not insert the device forcefully as significant injury can occur.** If it does not advance easily, redirect it and then reinsert the device. Inflate the pharyngeal cuff with the volume of air marked on the blue port (i.e., 100 mL for the ETC, 85 mL for the ETC SA, or 80 mL for the EzT). The device will withdraw slightly from the patient's mouth as the pharyngeal cuff is inflated. Inflate the distal cuff with the volume of air marked on the white port (i.e., 15 mL for the ETC, 12 mL for the ETC SA, or 10 mL for the EzT).

Begin ventilation through the longer blue tube because most blind intubations follow the natural curve into the esophagus (**Figures 27-5*C* and 27-6**). This is easily remembered as "blue is best." Auscultation of breath sounds, symmetric rise of the chest, fogging in the tube for more than six breaths, and lack of gastric insufflation confirm placement within the esophagus and ventilation through the proximal ports or apertures (**Figures 27-5*C* and 27-6**). The trachea is intubated if no breath sounds are auscultated and gastric insufflation occurs (**Figure 27-5*D***). Begin ventilation through the shorter clear tube and verify by auscultation the presence of breath sounds.

The device may be too far into the pharynx if breath sounds cannot be auscultated when ventilating through either tube. Deflate the pharyngeal cuff and withdraw the device 2 to 3 cm. Reinflate the pharyngeal cuff. Ventilate through the longer tube and confirm tube placement as noted above.

AFTERCARE

Secure the device. This is accomplished using the standard method of taping or a commercially available endotracheal tube holder. Although there are reports of short-term (e.g., 4 to 6 hours) ventilator use with these devices, it should be replaced with a standard endotracheal tube for long-term ventilation.[23]

Several methods for replacing the device are available. The first is to remove the device entirely and intubate the patient orotracheally. Deflate the pharyngeal cuff. Suction the patient's mouth and oropharynx. Tilt the device to the left side of the patient's mouth. Deflate the distal cuff. Remove the device. Intubate the patient orotracheally (Chapter 18).[24]

Alternative methods of intubation are also possible. Deflate the pharyngeal cuff. Suction the mouth and oropharynx. Tilt the device to the left side of the patient's mouth. Insert the laryngoscope and visualize the tip of the device. Orotracheally intubate the patient if it is in the esophagus. Deflate the distal cuff of the device and remove the device. This method will prevent aspiration if endotracheal intubation is unsuccessful. Instruct an assistant to deflate the distal cuff of the device and remove it slowly if it is in the trachea. Immediately insert the endotracheal tube after the device clears the patient's vocal cords.

The EzT has a significant advantage over the ETC when attempting to replace it with a conventional endotracheal tube. It can be left in place as it is functioning as a 7.5 mm endotracheal tube if the distal end is in the trachea and the patient is being ventilated through the short clear tube. A fiberoptic scope, endotracheal tube changer, or a bougie can be passed through the blue tube and into the trachea if the distal end is in the esophagus and the patient is being ventilated through the long blue tube. The EzT can then be removed and an endotracheal tube passed over the endotracheal tube changer. This cannot be done with the ETC because it has perforations rather than an open aperture.[7,8]

COMPLICATIONS

Several disadvantages must be kept in mind despite the potential utility of the ETC or EzT in the acute setting. These include the high cost, bulky packaging, and the fact that the ETC detachable "vomit deflector" can expose providers to gastric contents if improperly managed. It is advised not to use the vomit deflector as it can be associated with aspiration. Several risks are also inherent to the insertion and mechanics of the devices. It should be recognized that the presence of a rigid cervical collar can cause great difficulties in the proper placement of this device.[25] The device is most frequently inserted into the esophagus. There is a risk of esophageal injury.[26]

There is no way to suction the trachea with the open distal port in the esophagus. It is important to note that resuscitation drugs that may be administered through an endotracheal tube cannot be given through the device positioned with the tip in the esophagus. Drugs will accumulate in the blind end of the tube or the hypopharynx. Significant soft tissue injury can occur due to the tip of the device or if the cuffs contain too much air.[25,27] The tip of the device can perforate the esophagus, piriform sinus, or vallecula.

The increased cuff pressure of the ETC versus the EzT can result in mucosal injury.[28] An overinflated distal cuff located in the

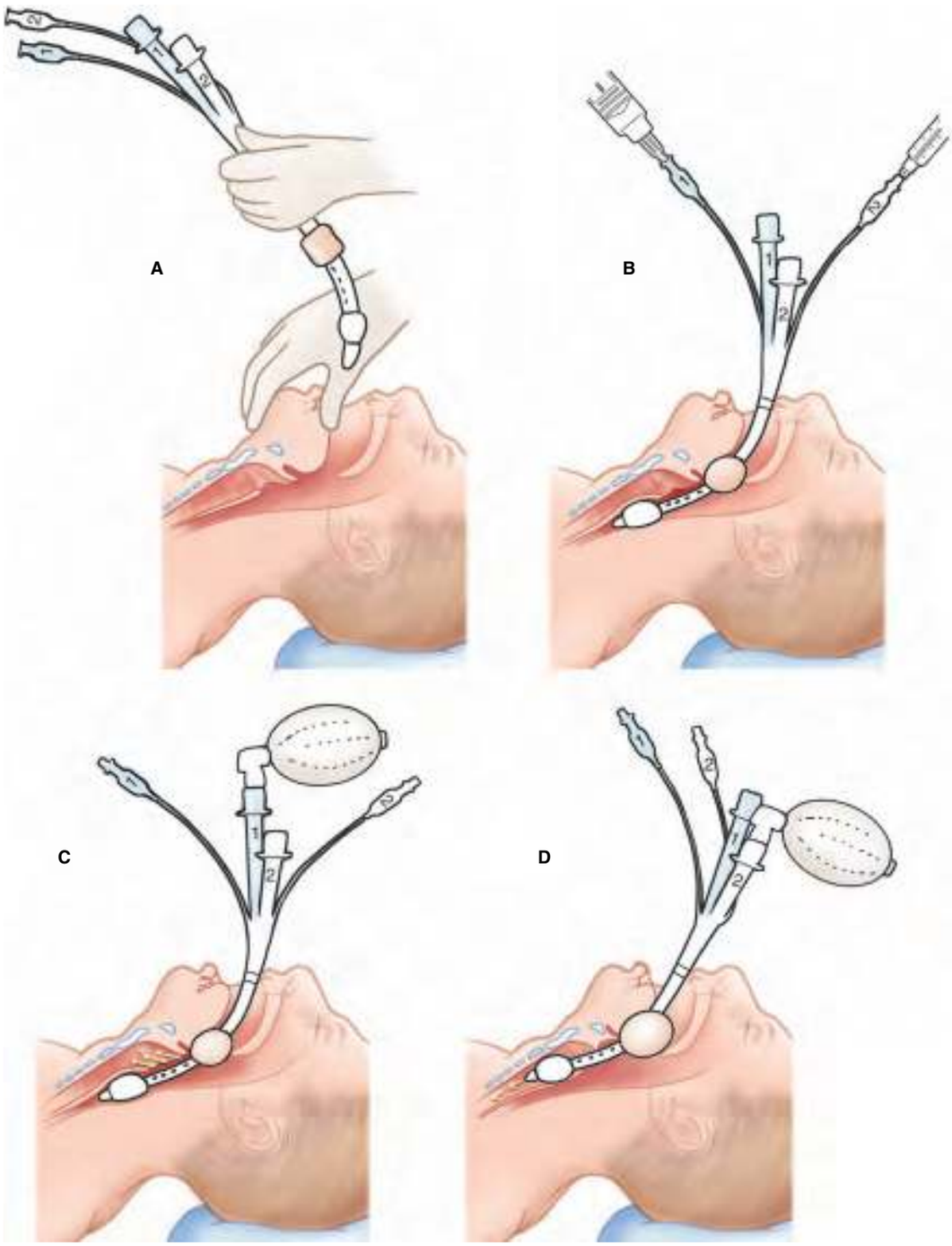

FIGURE 27-5. Insertion of the ETC. ***A.*** Positioning of the patient and the physician. ***B.*** The tube is inserted until the patient's incisor teeth are between the black lines and the cuffs are inflated. ***C.*** The tube is inserted into the esophagus. The patient is ventilated through the longer tube (1) and air is directed from the proximal ports (*arrows*). ***D.*** The tube is inserted into the trachea. The patient is ventilated through the shorter tube (2) and air is directed through the distal port (*arrows*).

esophagus can compress the trachea and cause an airway obstruction.[29] **Always inflate the cuffs with the recommended volume of air and not more.**[30-32] Prolonged use of up to 4 hours can result in the proximal cuff obstructing the lingual veins and resultant tongue engorgement.[33] This can result in a difficult intubation when exchanging the device for an endotracheal tube. Studies have demonstrated that the lengthy mean insertion speed and the need for multiple steps required has led to increased popularity of single-lumen devices such as the Laryngeal Tube (LT).[34-36]

SUMMARY

The ~~ETC~~ and the EasyTube can adequately ventilate a patient whether they are placed in the esophagus or trachea. They are relatively simple to use and require minimal training. They offer an additional technique to secure the airway in the prehospital and hospital environments. These devices should be familiar to Emergency Physicians who need to exchange these tubes for a definitively managed airway or need to use these devices as alternative tools in the difficult airway patient.

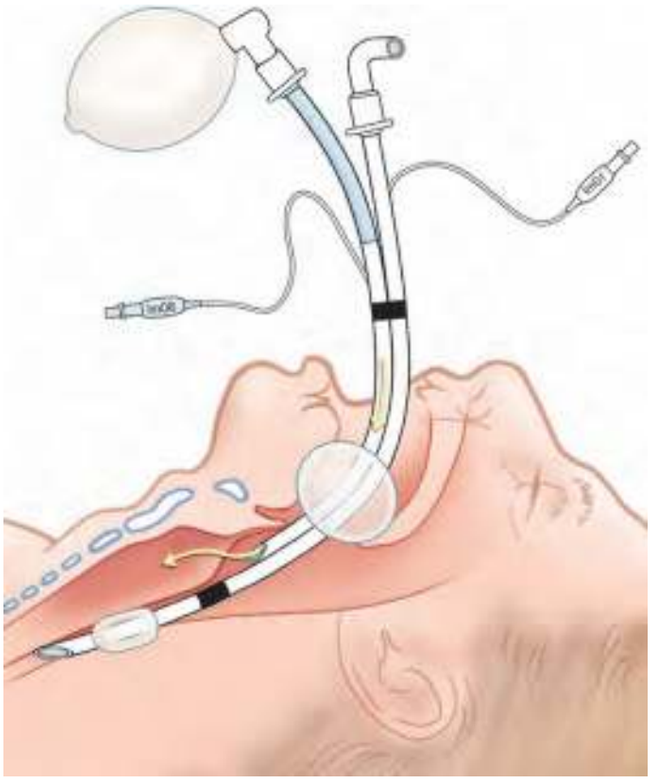

FIGURE 27-6. Insertion of the EzT with the distal end in the esophagus.

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28 Fiberoptic Endoscopic Intubation

Ruth Moncayo, Alia Safi, and Ned Nasr

INTRODUCTION

The flexible fiberoptic bronchoscope is a useful instrument for the intubation of patients with an anticipated or unanticipated difficult airway.[1] The hallmark of a difficult airway is an abnormal anatomy that precludes the use of the conventional airway management techniques (e.g., face mask and laryngoscope) or maneuverers aimed to align the airway (e.g., jaw thrust and sniffing position).

Any acquired or congenital condition that prevents successful ventilation or intubation by conventional means is a difficult airway. Alternative techniques must be considered for the successful management of these difficult airways. It is imperative in these circumstances to preserve the patient's spontaneous ventilation while safely preparing for a flexible fiberoptic intubation. A unique advantage of the flexible fiberoptic scope is that its malleability and softness allow it to conform to the patient's anatomy and to navigate through the airway with a minimal amount of trauma and discomfort. **Technical problems and failure to successfully intubate patients using this technique are usually due to a lack of familiarity and expertise with the fiberoptic bronchoscope, using it in the wrong clinical setting, or inadequate patient preparation.**

AIRWAY ANATOMY AND INNERVATION

A recognition of the airway's anatomy is of the utmost importance in the performance of a successful fiberoptic intubation by a nasal or oral approach. A more detailed description of the airway anatomy is provided in Chapters 9 (Essential Anatomy of the Airway), 10 (Basic Airway Management), and 18 (Orotracheal Intubation).

It is important to understand the anatomic innervation of the areas through which the fiberoptic bronchoscope will course during the performance of the procedure. This allows a fiberoptic intubation unhindered by the gag and cough reflexes. The fiberoptic bronchoscope passage through the oral cavity will encounter structures innervated by the glossopharyngeal nerve (e.g., the posterior third of the tongue, the vallecula, the anterior surface of the epiglottis, the walls of the pharynx and the tonsils). The glossopharyngeal nerve can be blocked by topicalization of the oropharyngeal mucosa using benzocaine- or 5% lidocaine-soaked pledgets (i.e., applied to the posterior tongue, tonsils, anterior tonsillar pillars [glossopalatal arches] and posterior tonsillar pillars [glossopharyngeal arches]), the inhalation of aerosolized 4% lidocaine, or directly injecting 5 mL of 2% lidocaine. The transoral approach of injection is achieved by using a spinal needle aimed at the base of the posterior pillar of the tonsil (**Figure 28-1**).[2] **Do not inject the local anesthetic solution into the internal carotid artery as its runs at 1 inch lateral and posterior to the tonsil.**

The fiberoptic bronchoscope next encounters structures innervated by the internal branch of the superior laryngeal nerve. This nerve originates from a branch of the vagus nerve. It provides sensory innervation to the base of the tongue, posterior surface of the epiglottis, aryepiglottic folds, and the arytenoids. These structures comprise the supraglottic area. The superior laryngeal nerve can be blocked by injecting 2% lidocaine at the cornu of the hyoid bone bilaterally. Finally, the fiberoptic bronchoscope encounters

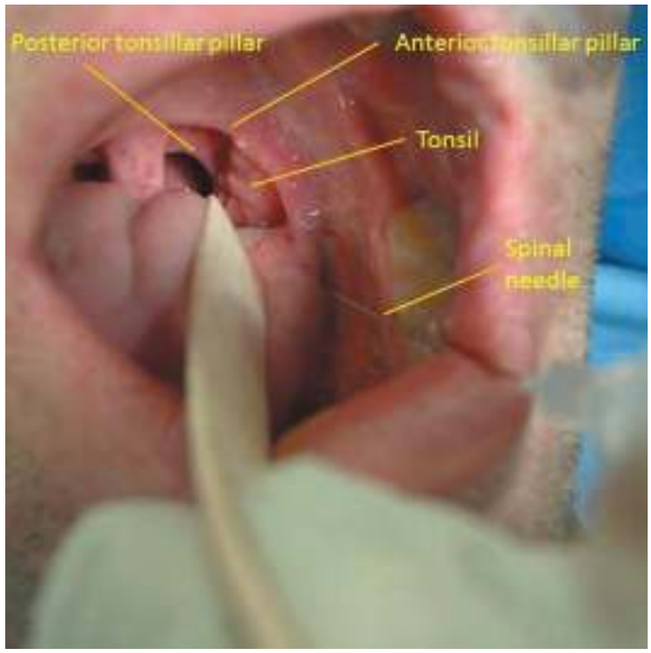


FIGURE 28-1. Intraoral approach to block the glossopharyngeal nerve. (Used with permission from reference 2).

structures innervated by the recurrent laryngeal nerve. This is also a branch of the vagus nerve. It supplies sensory innervation to the subglottic area and trachea. It can be blocked by transtracheal injection of local anesthetic solution.

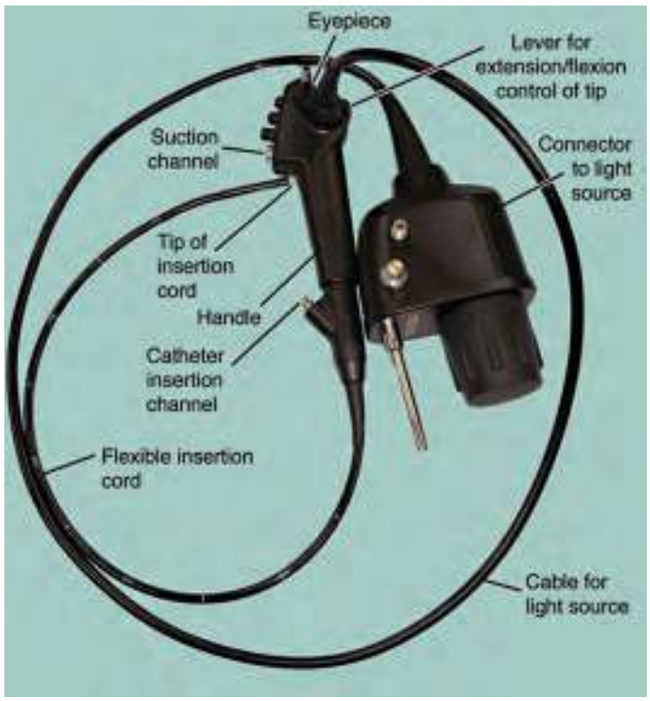
The nasal cavity is innervated by the greater palatine nerve, lesser palatine nerve, and anterior ethmoidal nerve (**Figure 203-7**). The palatine nerves arise from the pterygopalatine ganglion located posterior to the middle turbinate in the pterygopalatine fossa. They innervate the nasal turbinates and most of the nasal septum. The anterior ethmoidal nerves arise from the olfactory nerve. They innervate the nares and the anterior third of the nasal septum.

FIBEROPTIC TECHNOLOGY

A brief description of the flexible fiberoptic bronchoscope is presented in this section. There are other sources for a more in-depth description of the fiberoptic bronchoscope.[3] The principal virtue of the fiberoptic scope is its capacity to transmit an image off axis from the tip of the device to the eyepiece or video camera at the opposite end by means of internal reflections occurring along the length of flexible optic fibers.

The basic structure of the flexible fiberoptic bronchoscope is shown in **Figures 28-2*A* and 28-2*B***. The major components are the handle, the insertion cord or flexible fiberscope, and a light source. The handle contains the eyepiece for image viewing and a dial to bring the image into focus. A thumb control lever allows deflections of the tip of the fiberoptic bronchoscope in one plane up to 120° up or down.

The insertion cord is composed of thousands of glass fibers, each approximately 8 to 25 μm in diameter. The fibers in the cord transmit an image to the proximal viewing lens. There is a side port along the length the fiberoptic bronchoscope that can be used for the insufflation of oxygen, instillation of local anesthetic or saline solution, limited suction due to the small size of the port, passage



A

B

C

FIGURE 28-2. Anatomy of the flexible fiberoptic bronchoscope. ***A.*** The fiberoptic bronchoscope. ***B.*** The tip of the fiberoptic bronchoscope. (Photo courtesy of Pentax Medical, Montvale, NJ.) ***C.*** The aScope.

of a guidewire, and end-tidal CO_2 monitoring. Any fiberoptic bronchoscope used for intubation should have a length of at least 55 to 60 cm. Consider the diameter of the flexible fiberoptic bronchoscope cord when selecting an endotracheal (ET) tube that fits over it, especially when using small ET tubes (**Table 28-1**).

TABLE 28-1 Flexible Fiberoptic Scopes

	4.1–4.9 mm OD (adult)	3.1 mm OD (pediatric)	2.8 mm OD (pediatric)	2.2 mm OD (neonatal)
Smallest LMA	2	1	1	1
Working channel (mm)	2.2	1.2	1.2	None
Viewing angle	100°	95°	90°	75°
Smallest ETT	4.5	3.5	3.5	2.5

ETT, endotracheal tube; LMA, laryngeal mask airway; OD, outer diameter.

INDICATIONS

The decision to select an awake versus asleep fiberoptic bronchoscope intubation depends on the information collected during the initial assessment of the patient's airway, specifically information pertaining to the ability to mask ventilate the patient. Multiple studies have addressed the impact of certain physical features on predicting difficult or impossible mask ventilation and intubation.[4] The physical features that have been recognized as being independent predictors of a difficult airway include limited mandibular protrusion, abnormal neck anatomy, sleep apnea or snoring, and a body mass index of 30 kg/m^2 or greater. Some of the most common contributing factors to be assessed in a patient with a difficult airway are listed in **Table 28-2**.[5] An awake fiberoptic bronchoscopy has been used successfully in patients at increased risk for dental damage or with a rapidly enlarging neck mass.[6,7]

TABLE 28-2 The Most Common Contributing Factors to Assess in a Patient to Determine a Difficult Airway

Abnormal anatomy (e.g., congenital craniofacial defects, Pierre-Robin syndrome)
Acromegaly
Burns to face and airway
Cancer of the head and neck
Cervical spine disease (e.g., ankylosing spondylitis or rheumatoid arthritis)
Croup
Decreased thyromental distance
Dental abscess
Epiglottitis
History of difficult intubation
Infection or inflammation that causes airway disruption or rapidly progressing edema
Known difficult intubation
Known difficult mask ventilation
Limited mouth opening
Ludwig's angina
Macroglossia
Micrognathia
Obesity
Obstructive sleep apnea
Pathologic airways (e.g., postradiation fibrosis, stenosis, tracheal deviation)
Positional orthopnea
Risk of aspiration with potential difficult airway
Suspected difficult intubation by direct laryngoscopy
Trauma to face, neck, and/or upper airway
Unstable cervical spine

CONTRAINDICATIONS

The contraindications to fiberoptic bronchoscope intubation are similar to nasotracheal intubation (Chapter 29). Fiberoptic intubation is not recommended for patients who are actively apneic, are vomiting, or have significant oropharyngeal bleeding. Opaque fluids cover the fiberoptic port and prevent adequate visualization through the fiberoptic bronchoscope. Patients who are hypoxic or require assisted ventilation by mask are poor candidates for fiberoptic intubation as the technique may require several minutes to perform. An exception may be made if the patient can be ventilated by an endoscopy mask that has a specialized central orifice for placement of a fiberoptic bronchoscope, by using an elbow connector with a bronchoscopy port attached to a standard face mask, or by using a supraglottic device (e.g., an i-Gel or a laryngeal mask) through which fiberoptic intubation may be performed.[8,9] Contraindications specific to nasal fiberoptic intubation include coagulopathy, significant midface trauma, severe intranasal pathology, fracture of the cribriform plate, and leakage of cerebrospinal fluid.[10]

Relative contraindications to fiberoptic intubation of the airway are situations when instrumentation of the airway may further compromise airway patency (e.g., stridor resulting from airway edema, infection, or epiglottitis). **Some authors advocate fiberoptic intubation as an option to consider in these circumstances, but only by individuals extremely proficient at fiberoptic endoscopic intubation and only with a qualified physician standing by to perform an emergent tracheostomy or cricothyroidotomy if the need arises.**[11] It would probably be prudent to establish a surgical airway in the Operating Room under a more controlled setting rather than to attempt a fiberoptic intubation. An emergent tracheostomy or cricothyroidotomy cannot be successfully performed in these patients before complete airway obstruction occurs. **Always be prepared to provide oxygen emergently by another route (e.g., transtracheal jet ventilation) to prevent hypoxic brain damage.**

EQUIPMENT

NASAL ANESTHESIA

- Cotton-tipped applicators
- 4% lidocaine
- 0.05% oxymetazoline (Afrin)
- 4% topical cocaine

OROPHARYNGEAL ANESTHESIA

- 1%, 2%, or 4% lidocaine or benzocaine spray
- Nebulizer device with tubing (**Figure 28-3 and Table 28-3**)
- Cotton 4×4 swabs soaked in 4% lidocaine
- Emesis basin
- Yankauer suction
- Tongue blade

LARYNGEAL ANESTHESIA

- Alcohol swabs
- 10 mL syringes
- 21 gauge needle, 1½ inches
- 2% lidocaine for atomization
- 1% and 4% lidocaine solution
- Nebulizer device with tubing

FIBEROPTIC BRONCHOSCOPY AND INTUBATION

- An assistant
- Fiberoptic bronchoscope with working channel
- Bite block
- Oral airways
- Nasopharyngeal airways
- Light source
- ET tubes, various sizes
- Suction source and catheters
- Cuffed tracheal tubes of various sizes, especially 5 and 7 mm
- Oxygen source
- Oxygen tubing
- Bag-valve device
- Face masks
- Water-soluble lubricant or anesthetic jelly
- Gauze 4×4 squares
- Antisialagogue (e.g., 0.3 to 0.4 mg glycopyrrolate or 0.5 mg atropine)

MISCELLANEOUS SUPPLIES

- Povidone iodine solution or chlorhexidine
- Alternative intubation devices (e.g., difficult airway cart)
- Cricothyroidotomy supplies
- Rapid sequence induction medications
- Crash cart

- Cardiac monitor
- Pulse oximetry
- Noninvasive blood pressure cuff

Atomizer devices are usually not available in the Emergency Department. Many devices or available commercially. These are disposable devices that are single patient use (**Figure 28-3 and Table 28-3**). A popular device is the MAD line of mucosal atomizer devices (LMA North America, San Diego, CA). They make multiple devices for all topical needs.

Numerous companies make fiberoptic scopes. One popular device in the Emergency Department is the aScope (AMBU Inc., Glen Burnie, MD).[12,13] It is a single patient use articulating videoscope (**Figure 28-2*C***). The aScope uses small digital chips attached

A

B

C

D

E

F

FIGURE 28-3. Atomizers. ***A.*** The LMA MAD nasal. ***B.*** The LMA MADgic Laryngo-Tracheal. ***C.*** The child's LMA MADdy. ***D.*** The EZ Spray. ***E.*** The Devilbiss Model 15. ***F.*** The MADomizer refillable bottle.

TABLE 28-3 Some of the Commercially Available Atomizers

Name	Manufacturer	Comments
DeVilbiss Model 15 Medical Atomizer	DeVilbiss Healthcare	Adjustable tip Disposable and reusable versions
Enk Fiberoptic Atomizer Kit	Cook Medical	Atomizes through fiberoptic bronchoscope working channel Pressure-resistant oxygen tubing
EZ-Spray	Alcove Medical	Disposable Trigger valve system
LMA MADdy Pediatric Mucosal Atomizer Device	Teleflex	11.4 cm long Attaches to standard syringe Child friendly Comes in various colors and shapes Disposable single-use
LMA Bottle MADomizer Atomizer	Teleflex	10 cm long Disposable single-use tips Reusable bottle
LMA MADgic Laryngo-Tracheal Atomizer	Teleflex	21.6 cm long Attaches to standard syringe Disposable single-use Flexible tube
LMA MAD Nasal-Intranasal Mucosal Atomizer Device	Teleflex	Attaches to standard syringe Disposable single-use

directly at the distal end of the scope. The video signal is electronically conducted to a small portable monitor since the aScope has no eyepiece. Using the plug-and-play monitor is easier than a monocular eyepiece for supervision of others performing the intubation. The unit costs approximately $200, much less than the cost of a traditional fiberoptic bronchoscope, which costs a minimum of $8000. There are no cleaning, sterilization, maintenance, and repair costs. The image resolution of the aScope is not equivalent to that of a fiberoptic bronchoscope. However, it is more than adequate to intubate a patient. The aScope can be used as a backup device for the fiberoptic bronchoscope when it is not available.

A disposable sheath for the fiberoptic bronchoscope is available. Numerous companies make an EndoSheath. It is a sterile, single-use, disposable, and microbial barrier that covers the fiberoptic bronchoscope and prevents patient contact. The setup is quick and often takes less than a minute. The proximal end of the EndoSheath has a rubberized hub that grips the proximal end of the fiberoptic bronchoscope. The distal end of the EndoSheath contains an optical quality lens that does not alter the fiberoptic bronchoscope view. It costs approximately $10. The EndoSheath is removed by pushing the hub off the scope. The EndoSheath comes in a variety of lengths and fits over most fiberoptic bronchoscopes. The EndoSheath eliminates the need to send the fiberoptic bronchoscope out of the Emergency Department for cleaning after its use. The fiberoptic bronchoscope can be used from patient to patient by simply changing the sheath. This eliminates any delays in patient care related to cleaning, processing, and locating the fiberoptic bronchoscope.

PATIENT PREPARATION

Fiberoptic intubation is best performed on awake and spontaneously breathing patients. Rendering the patient unconscious might relax the airway anatomy, distort the airway anatomy, and place the patient at risk for more serious complications (e.g., apnea, airway obstruction, and aspiration of gastric contents).[14] **Proper patient preparation is essential to the successful completion of a fiberoptic intubation**. Proper patient preparation includes counseling the patient, clearing the airway of secretions and blood, judicious sedation, and airway anesthesia (e.g., nasopharynx, larynx, and trachea).[10] **Counseling is an important and often underestimated part of patient preparation.**[15] Thoroughly explain the necessity for the procedure and the technique. The Emergency Physician can gain the patient's confidence and cooperation, which are invaluable for the performance of a successful fiberoptic intubation.

Apply monitors for electrocardiogram, blood pressure, and pulse oximetry along with an intravenous line for the administration of drugs. Administer supplemental oxygen by nasal cannula, "blow-by," or through the side port of the fiberoptic bronchoscope. Delivering oxygen through the side port offers the additional advantage of blowing airway secretions away from the fiberoptic bronchoscope's tip and can be used to help visualize the vocal cords when redundant tissue is obscuring the view. It has the potential disadvantage of causing gastric distention and rupture.[16,17] Maintain low oxygen flow to avoid trauma related to oxygen flow through the bronchoscope side port.

The patient may be in a sitting, semirecumbent, supine, or left semilateral position during fiberoptic bronchoscopy.[18,19] The sitting position will displace redundant pharyngeal tissue anteriorly and open the pharyngeal space by gravity in morbidly obese patients.[15] The use of the sitting position requires the Emergency Physician to stand at the patient's side and inverts the image seen through the fiberoptic bronchoscope. Alternatively, the Emergency Physician can stand on a platform behind the patient to be of sufficient height to correctly perform the procedure. When performing the procedure with the patient in the supine position, the patient's head should lie flat against the table surface with the neck extended (if it is safe to do so). This head position brings the tracheal axis more in line with the nasal and oral passageways and elevates the epiglottis from the posterior pharyngeal wall. The "sniffing position" used for direct laryngoscopy increases obstruction of the glottis by the epiglottis during fiberoptic bronchoscopy and makes the passage of the fiberoptic bronchoscope more difficult.[20] Maneuvers performed by an assistant (e.g., the jaw thrust or pulling the tongue forward with a cotton swab) can help to move the pharyngeal soft issues anteriorly and allow increased maneuverability of the fiberoptic bronchoscope's tip.[10]

Instrumentation of the airway may cause the patient to produce copious secretions. This makes an otherwise straightforward fiberoptic bronchoscopy extremely difficult. Bronchoscopy via a "dry" airway devoid of secretions can be accomplished in most instances by administering 0.3 to 0.4 mg of glycopyrrolate. Administer this potent antisialagogue intravenously at least 10 minutes before or intramuscularly 30 minutes before fiberoptic bronchoscopy. Atropine is often more readily available in the Emergency Department than glycopyrrolate. It can be administered in a dose of 0.5 mg intravenously at least 10 minutes before fiberoptic bronchoscopy. The only disadvantage of atropine is that it crosses the blood-brain barrier and can cause central nervous system effects, whereas glycopyrrolate does not. Refer to Chapter 12 for a more complete discussion of these two antisialagogues.

Sedation can be extremely beneficial in gaining the patient's cooperation during the performance of a fiberoptic bronchoscopy. **Sedative drugs, if used at all, should be judiciously titrated to the desired effect with continual assessment of the patient's level of consciousness while avoiding respiratory depression.** Ketamine given in small doses (e.g., 0.5 to 1 mg/kg) and titrating to the desired level of sedation has the advantage of producing minimal respiratory depression and may be preferable to opioids in some cases. Other agents that have been successfully used for sedation during fiberoptic intubations include midazolam, propofol, fentanyl, dexmedetomidine, and remifentanil.[21-26] Refer to Chapter 12 for a more complete discussion of the pharmacologic adjuncts to intubation. **Avoid sedation if the patient has a tenuous airway, labored respirations, a distended abdomen, or is vomiting.**

A B

FIGURE 28-4. The landmarks of the airway. ***A.*** Drawn on a model. (Used with permission from reference 2.) ***B.*** Hand positioning.

AIRWAY ANESTHESIA

Adequate anesthesia of the airway is extremely important when intubating an awake patient.[27,28] This establishes a quiet larynx devoid of reflexes prior to attempting fiberoptic bronchoscopy. It is especially effective in improving the success rate of individuals who are less experienced at performing fiberoptic intubation. The regional anesthesia techniques employed to anesthetize the airway are a glossopharyngeal nerve block, a superior laryngeal nerve block, and recurrent laryngeal nerve block.

Bilateral blockade of the superior laryngeal nerves will provide effective anesthesia of the larynx and trachea. Prepare a 5 mL syringe with a 25 gauge needle and filled with 1% or 2% lidocaine. Identify the hyoid bone and its superior cornu by palpation (**Figure 28-4*A***). Place both index fingers on the neck, one on each side of a point immediately below the lower edge of the mandible and 1 inch anteroinferior to the angle of the jaw (**Figure 28-4*B***). Feel the contralateral cornua by gently pushing on one side.

Clean and prep the skin over the superior cornu bilaterally. Insert and advance the needle until it contacts the superior cornu of the hyoid bone. Alternatively, contact with the bone can be made easier by extending the patient's head and gently palpating the hyoid bone with the thumb and forefinger of one hand. By applying gentle pressure to one side, the opposite cornu comes into closer contact with the skin and is subsequently easier to contact with the needle. Walk the needle tip inferiorly and off the bone. Advance the needle 3 to 4 mm and through the thyrohyoid membrane (**Figures 28-5, 28-6, and 28-7**). **Aspirate before injecting the local anesthetic solution to confirm that the needle has not entered the external carotid artery**. Inject 2 to 3 mL of lidocaine. Repeat the procedure on the contralateral side.

The hyoid bone may not be palpated due to obesity, infections, or masses. **Do not attempt a superior laryngeal nerve block blindly.**[29] It may be possible to perform the block by using ultrasound (US) to locate the hyoid cornu, thyroid cartilage, vocal cords, cricoid cartilage, and cricothyroid membrane (**Figure 28-8**).[30] This will facilitate the performance of the superior laryngeal nerve block and the transtracheal block.[31]

It remains an unresolved controversy whether to abolish the laryngeal reflexes of a patient considered to have a "full stomach." The Emergency Physician must weigh the risk of abolishing the

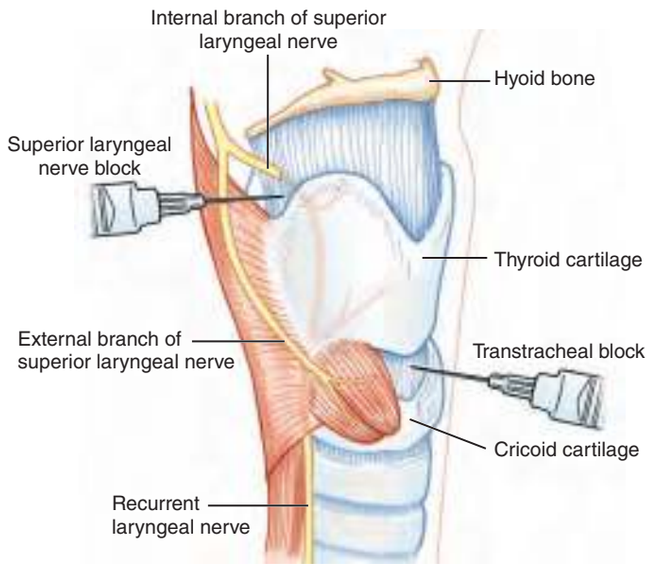


FIGURE 28-5. The anatomy of the larynx. The syringes demonstrate the superior laryngeal nerve block and the transtracheal block.

FIGURE 28-6. The superior laryngeal nerve block. (Used with permission from reference 2.)

FIGURE 28-7. The recurrent laryngeal nerve block. The needle is inserted and advanced just below the superior cornua of the hyoid bone.

laryngeal reflexes and rendering the patient potentially vulnerable to aspiration versus leaving the laryngeal reflexes intact and thus causing significant discomfort for the patient. Aspiration of gastric contents occasionally does occur in patients with intact laryngeal reflexes when deliberating to proceed with a regional block of the larynx in a patient at risk for gastric aspiration. Instrumentation of the airway in patients with an intact gag reflex can induce vomiting.

The larynx and trachea can be effectively anesthetized by performing a transtracheal injection of 4 mL of lidocaine (**Figures 28-5 and 28-9**).[32] Clean and prepare the surface of the skin over the cricothyroid membrane. Prepare a 20 gauge intravenous angiocatheter on a 5 mL syringe containing 2 to 3 mL of sterile saline. Prepare a second syringe containing lidocaine solution. Anesthetize the skin by performing a skin wheal. Insert the angiocatheter on the syringe containing sterile saline perpendicular through the skin in the midline over the cricothyroid membrane (**Figures 28-5 and 28-9**). Advance the syringe until the tip of the angiocatheter is in the trachea. The lumen of the trachea is identified by the loss of resistance. Aspirate air into the syringe, as evidenced by the presence of bubbles in the saline, to confirm that the angiocatheter is within the trachea. **Do not advance the angiocatheter too far, perforate the posterior trachea, and cause a pneumomediastinum.**[33] Securely hold the syringe in place and advance the catheter into the trachea. Securely

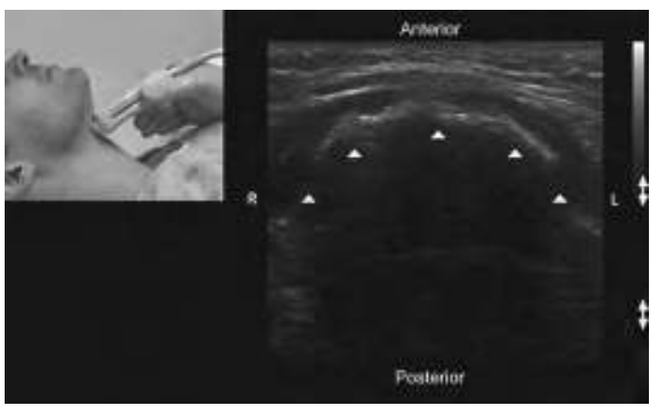


FIGURE 28-8. US to locate the hyoid bone (triangles). Placement of the transducer (*inset*). Transverse US view immediately below the line running through both angles of the mandible. (Used with permission from reference 30.)

FIGURE 28-9. Transtracheal anesthesia of the trachea and larynx. The catheter enters the trachea through the midline of the cricothyroid membrane.

hold the catheter hub at the skin. Withdraw the needle and syringe. Attach the syringe containing lidocaine to the catheter. Briskly inject 3 to 4 mL of 4% lidocaine through the catheter and into the tracheal lumen. This will cause the patient to cough and disperse the local anesthetic solution throughout the trachea and larynx. Remove the syringe and catheter. **The Emergency Physician performing this block should be wearing a gown, face mask, and eye protection to prevent exposure to the respiratory secretions when the patient coughs.**

The nasal passages, pharynx, and larynx can be anesthetized by several other techniques. If the nasal passageways are the anticipated route for intubation, they should be prepared by shrinking and anesthetizing the nasal mucosa. Cocaine (i.e., 4%) applied topically (maximum dose 200 mg) has the advantage of providing profound vasoconstriction and anesthesia to the nasal passageways. Similar effects can be provided by applying 0.25% to 1.0% phenylephrine topically to the nasal mucosa followed by 4% lidocaine via cotton-tipped applicators. Place these applicators gently one at a time through the middle meatus and back to the inferior turbinate. This will anesthetize the posterior palatine ganglion. A nasal passage that accommodates four to five single cotton-tipped applicators will usually allow passage of a 7.0 mm ET tube.[10] The use of three to four sprays of atomized oxymetazoline (e.g., Afrin) into one or both nares is effective in providing profound vasoconstriction of the nasal mucosa.

Once the antisialagogues have taken effect, the tongue and pharynx can be anesthetized. Place a tongue blade on the patient's tongue and apply topical anesthetic spray (**Figure 28-10**). Benzocaine or lidocaine spray will usually provide effective anesthesia for the posterior pharynx within 30 seconds. They can be administered from a commercially available spray container or using an atomizer device (**Figure 28-3 and Table 28-3**). There are reports of methemoglobinemia from the overzealous use of benzocaine.[34] Limit its use to several short sprays.

FIGURE 28-10. Anesthetizing the posterior oropharynx with atomized lidocaine solution.

Alternatively, the patient can swish 2% viscous lidocaine in their mouth for several minutes to provide effective anesthesia. One of the most effective methods of blocking the glossopharyngeal nerve is the "lollypop method." Create a lollypop by soaking sterile gauze in lidocaine ointment and taping it to a tongue depressor. Place the lidocaine lollypop into the patient's posterior mouth while setting up for the fiberoptic bronchoscopy. Advance the lollypop 1 cm every 2 minutes until the patient's posterior pharynx is completely anesthetized.

Nebulized 4% lidocaine can be administered as an alternative at least 20 minutes prior to fiberoptic bronchoscopy and will usually provide adequate anesthesia of the supraglottic structures. A second alternative is the "spray as you go" technique.[35] Once the epiglottis is visualized through the fiberoptic bronchoscope, instill 3 to 4 mL of 2% lidocaine through the working channel of the scope onto the epiglottis and the surface of the vocal cords. This will induce coughing and temporarily obliterate the view of the laryngeal structures. Allow 2 to 3 minutes for the anesthetic solution to exert its effect before proceeding with fiberoptic bronchoscopy.

Place a suction catheter into the oropharynx. This will clear the airway of secretions and blood that can impair the visual image. It will also determine the adequacy of the topical anesthesia for preventing coughing and gagging.

TECHNIQUES

Prepare the fiberoptic bronchoscope. Attach the light source. Check the focus of the image by holding the tip of the insertion cord 1 to 2 cm over a printed page. Adjust the eyepiece until the letters on the image are clear. Note how the image appears as you move toward and away from the page. Briefly use the angulation lever to move the tip of the insertion cord and learn its movements. **The most difficult aspect of mastering fiberoptic bronchoscopy is learning to simultaneously angle the tip, rotate the scope, and advance the insertion cord.**[36] It requires repetition and practice to develop these skills before attempting to intubate a patient.

Let the insertion cord hang toward the floor to straighten the inner fiberoptic strands before placing the fiberoptic bronchoscope insertion cord into the ET tube. Identify the plane in which the angulation lever moves the tip. Over time the insertion cord may develop a curve when fiberoptic bronchoscopes are stored coiled in a case. Slightly rotate the fiberscope to the right or left until the angulation of the tip is in the midline plane. Look through the eyepiece and note the position of the directional arrow (▼) on the anterior edge of the image that correlates with midline.

Apply an antifog solution to the insertion cord tip or place the tip in warm water before inserting the fiberoptic bronchoscope into the patient's nose to prevent fogging of the lens. Warming the ET tube with warm water just prior to placing it on the insertion cord will soften the ET tube and may make its later advancement through the mouth or nares easier.

Apply a thin film of silicone spray or water-soluble lubricant over the insertion cord to facilitate passage of the ET tube over the flexible cord. Insert the flexible insertion cord completely through the ET tube. **Use care and do not get any of the lubricant on the lens tip.** Advance the insertion cord tip until it exits the distal tip of the ET tube. **Do not place the tip of the insertion cord through the Murphy eye of the ET tube.** Lubricate the ET tube liberally.

Estimate the distance from the patient's mouth to their glottis. Place the tip of the insertion cord by the patient's ear. Mark the point the insertion cord touches the patient's mouth. The distance from the mouth to the ear is approximately the distance from the mouth to the glottic opening. Add 3 cm to this length if performing the procedure through the nose.

Hold the fiberoptic bronchoscope in your dominant hand with the angulation lever operated by the thumb and the suction port (if used) covered by the index finger. The other end of the scope should be held between the index finger and thumb of the nondominant hand. Place the nondominant hand at the patient's nose or mouth. There should be no slack in the fiberoptic bronchoscope between the two hands. The removal of slack from the insertion cord makes more precise rotary movements of the tip possible.

NASAL INTUBATION

Nasal fiberoptic intubation has several advantages over the oral route. Nasal fiberoptic bronchoscopy is usually easier to perform because less angulation of the tip is required for those less experienced at fiberoptic bronchoscopy. Nasal ET tubes are better tolerated by patients and are associated with a lower incidence of accidental extubation. Disadvantages include a higher incidence of bacteremia, middle ear infection, epistaxis, and alar necrosis.[10] **Nasal intubation, unlike oral intubation, may produce bacteremia. Provide appropriate endocarditis prophylaxis for those at risk**. Mechanical dilation with a finger or nasopharyngeal airways is not necessary.[37]

Examine the patient to determine which is the most patent nostril. The right side is better than the left and results in decreased epistaxis and faster intubation times.[38] Insert and navigate the insertion cord's tip along the posterior floor of the nares (**Figure 28-11**). Continue to advance the insertion cord and ET tube as a unit until the ET tube enters the oropharynx. This will serve to minimize patient discomfort and the risk of epistaxis early in the procedure. Loss of view and maneuverability of the insertion cord tip occasionally occurs in the oropharyngeal area as the tip encounters the pharyngeal mucosa. Pulling the patient's tongue forward with gauze, using the jaw-thrust maneuver, or simply advancing the insertion cord a few centimeters further will usually bring pharyngeal structures back into view. Continue to advance the tip of the insertion cord until the epiglottis is visualized (**Figure 28-12**). Maneuver the

FIGURE 28-11. The technique of placing the fiberoptic bronchoscope insertion cord into the nares. Note the position of the ET tube over the proximal portion of the insertion cord.

insertion cord tip with the lever until the glottis comes into view (**Figure 28-13**). Continue to advance the insertion cord tip through the vocal cords and to a point approximately 3 cm above the carina. Advance the ET tube over the insertion cord and into the trachea to the appropriate depth.

The distance from the nares to the epiglottis is approximately 15 to 17 cm. **It is very likely that the insertion cord has entered the esophagus if the 15 cm mark has been passed**. If that is the case, withdraw it to 12 cm and redirect the tip upward with a slight downward movement of the angulation lever.[36] This will usually bring the glottic opening into view (**Figure 28-13**). The epiglottis will occasionally obscure the glottic opening. Position the tip of the insertion cord just above the tip of the epiglottis, then advance it a few

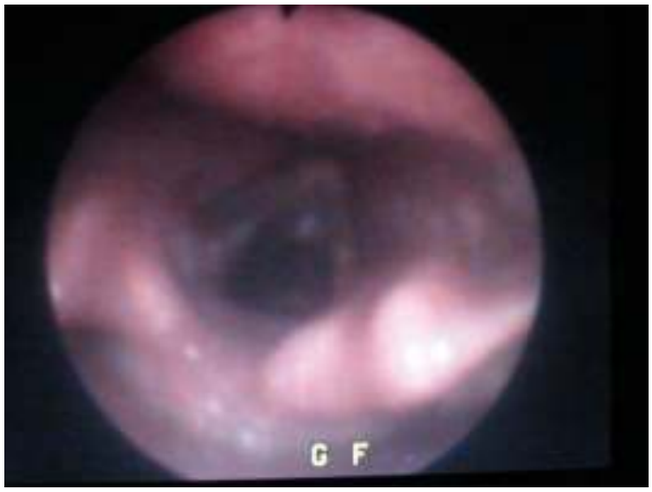


FIGURE 28-13. Visualization of the glottis through the flexible fiberoptic bronchoscope just prior to its insertion cord passing through the vocal cords.

millimeters posterior to the epiglottis while angulating the tip of the insertion cord slightly anterior by pressing down on the angulation lever. This will bring the glottic opening into view. Simultaneously rotate, angulate, and advance the insertion cord tip toward and past the vocal cords.

An alternative technique would be to advance the ET tube through the nares until the tip is just past the soft palate (**Figure 28-14**). This is usually at a depth of 10 to 12 cm. The insertion cord is then passed through the ET tube and through the vocal cords. The ET tube is then advanced over the insertion cord into the trachea. This approach has the advantage of bringing the tip of the insertion cord directly midline and toward the epiglottis. This can cause some patient discomfort early in the procedure and may decrease patient cooperation before the insertion cord has entered the trachea. There is the potential for epistaxis to make visualization of laryngeal structures difficult if not impossible. It is difficult to anesthetize the nasal passages completely despite the application of topical anesthesia to the nasal passages. Passage of the ET tube is often the most uncomfortable part of the fiberoptic intubation procedure for the awake patient.

FIGURE 28-12. Visualization of the epiglottis (at the top of the photo) through the flexible fiberoptic bronchoscope.

FIGURE 28-14. The ET tube has been inserted into the nares and advanced into the oropharynx. The insertion cord is advanced through the ET tube.

Emergency Physicians have difficulty passing the insertion cord through the vocal cords on some occasions. There are several causes for this. The tip of the insertion cord may remain angulated and abut against the wall of the trachea. The vocal cords may not be properly anesthetized and may have closed reflexively. The insertion cord tip may be abutting the arytenoid cartilages or the pyriform sinus. If inadequate anesthesia is the cause, inject 2 mL of 2% or 4% lidocaine through the working channel of the insertion cord and wait several minutes for it to take effect. Having the patient inspire deeply will bring the vocal cords into greater opposition. Once the insertion cord tip has passed the vocal cords, bring the tip into neutral position with a light downward motion of the angulation lever.

Advance the insertion cord tip further to bring the bifurcation of the trachea at the carina into view. The trachea can easily be identified anteriorly by the cartilaginous rings and posteriorly by the smooth mucosa of the posterior wall. Advance the ET tube over the insertion cord and into the trachea. The arytenoids or the interarytenoid soft tissues can impede advancement of the ET tube past the vocal cords and into the trachea. If the ET tube advancement is inhibited, withdraw it slightly, rotate it 90° counterclockwise, and reattempt intubation.[39-41] If this maneuver fails, rotate the ET tube so that its bevel faces either posteriorly or to the left and laterally. The inability to advance the ET tube occurs with greater frequency when the diameter of the insertion cord is significantly smaller than that of the ET tube or with oral fiberoptic intubation due to the greater curve that the ET tube must assume for it to enter the trachea.[36] Consider substituting a smaller ET tube, a spiral-bound ET tube, an ET tube with a flexible tip, or an ET tube that can be guided.[42,43]

Occasionally, when the trachea is not anesthetized, the patient's subsequent coughing and the associated muscular contractions of the trachealis muscle will collapse the trachea almost completely. This makes it difficult to discern whether the insertion cord tip is in the trachea or whether to advance the insertion cord or ET tube into the trachea. Wait until the trachealis muscle relaxes and then continue with the procedure.

To prevent endobronchial intubation in adults, which can occur with flexion of the head, confirm that the tip of the ET tube is 3 cm above the carina. This is accomplished by advancing the tip of the insertion cord to the carina with the thumb and forefinger of the nondominant hand. Mark the point on the insertion cord where it exits the ET tube. Withdraw the insertion cord until the distance on the insertion cord between the marked point and the tracheal tube connector is 4 cm. While looking through the eyepiece, advance the ET tube until its tip is visible. This places the tip of the ET tube at approximately 3 cm above the carina.

Difficulty is occasionally encountered while attempting to pass the ET tube through the nares and nasal cavity. This might be caused by a deviated nasal septum, enlarged turbinates, a nasal spur which can tear the ET tube cuff, or nasal polyps. Selection of an ET tube that is too large, inadequate lubrication, or failure to presoften the ET tube can be the cause. Reattempt insertion with a well-lubricated, presoftened ET tube that is 0.5 to 1.0 mm smaller. Alternatively, try placing a 5.0 mm inner diameter (ID) ET tube in a 7.0 mm ID ET tube, as discussed below.

ORAL INTUBATION

Begin by noting any loose or broken teeth. Ensure an adequate sensory block by the absence of a gag reflex. Insert an oral intubating airway into the patient's mouth. These devices are placed in the patient's mouth like any other oral airway. They allow for the midline passage of the insertion cord and protect the delicate fibers within it from the patient's teeth.

Technical problems exist in attempting oral fiberoptic intubation. Oral fiberoptic intubation requires that the insertion cord tip traverse a more acute angle to reach the vocal cords than it would by the nasal route. Maximally extending the patient's head at the atlantooccipital joint will bring the oropharyngeal and laryngeal axes more closely in line if it is safe to move the patient's neck. This maneuver will reduce the angle that the insertion cord tip must traverse.

The ET tube becomes hung up on the vocal cords more frequently than with the nasal route. A technique believed to significantly improve the first-time pass rate with oral fiberoptic bronchoscopic intubation is to pass a lubricated 5.0 mm ID ET tube through a 7.0 mm ID ET tube that has been cut to 24 cm. This should leave 2 cm of the 5.0 mm ID ET tube protruding from the distal end. It is believed that the close approximation of the diameters of the 5.0 mm ID ET tube and the fiberoptic bronchoscope allows easier passage of the scope. After the 5.0 mm ID/7.0 mm ID ET tube complex is in place, withdraw the 5.0 mm ID ET tube, and leave the 7.0 mm ID ET tube in the trachea.[44]

A face mask with a fiberoptic bronchoscope adapter can be used (**Figure 28-15**). This allows an assistant to hold the face mask and ventilate the patient during the fiberoptic bronchoscopy. High-flow nasal oxygen can be administered to those spontaneously breathing. This can be alone or in addition to the face mask.

FIGURE 28-15. A fiberoptic bronchoscopy adapter that is added to a face mask.

ALTERNATIVE TECHNIQUES

Alternative techniques that have been shown to be as effective as fiberoptic intubation for intubating patients with unstable cervical spines include the Bullard laryngoscope and the lighted stylet.[45,46] Blind nasotracheal intubation is as successful as nasal fiberoptic intubation in anesthetized patients with unstable cervical spines.[47] The Glidescope, a video laryngoscope, causes less cervical spine movement than direct laryngoscopy.[48]

A combined technique using oral fiberoptic intubation through a laryngeal mask airway (LMA) or alternative supraglottic airway can be extremely helpful in instances where there is severe oropharyngeal bleeding or when direct laryngoscopy is not possible.[8,49-51] After confirming successful placement of the LMA, place a self-sealing bronchoscopy elbow over the proximal end of a 6.0 mm ID ET tube.[52] Advance the ET tube tip through the LMA until the LMA grille is encountered and resistance is felt. Inflate just enough air into the ET tube cuff to provide a seal for positive-pressure ventilation via the ET tube. Advance the insertion cord through the ET tube and into the trachea under direct visualization. Deflate the ET tube cuff. Advance the ET tube over the insertion cord and into the trachea until the ET tube adapter meets the adapter of the LMA. Inflate the ET tube cuff and confirm ventilation through the ET tube.

Because standard ET tubes are not long enough to allow removal of the LMA, longer ET tubes have been developed. If you do not have access to a specially made ET tube, use a nasal Rae tube, which is 6 cm longer than a standard ET tube. If a longer ET tube is not available, another ET tube can be temporarily lengthened by removing the adapter of the 6.0 mm ID ET tube and placing the tip of a 5.0 mm ID ET tube into the lumen of the 6.0 mm ID ET tube. This maneuver lengthens the ET tube enough that the LMA cuff can be deflated and withdrawn, leaving the 6.0 mm ID ET tube correctly placed in the trachea. Additionally, ventilation can be maintained the entire time simply by using a bag-valve device connected to the 5.0 mm ID ET tube adapter. After removing the LMA, remove the 5.0 mm ID ET tube from the 6.0 mm ID ET tube, replace the adapter, and resume ventilation.[53] **Always confirm by fiberoptic bronchoscopy that the ET tube is correctly positioned after removing the LMA and establishing ventilation.**

ASSESSMENT

The placement of an ET tube should be followed by an assessment to ensure its proper positioning (Chapter 19). This includes visual inspection of chest rise and lack of abdominal movement with ventilation, fogging in the ET tube for at least six breaths, auscultation, and end-tidal CO_2 monitoring. This should be followed by a chest radiograph to confirm proper positing of the ET tube within the trachea.

AFTERCARE

The steps of ensuring proper placement of the ET tube and securing the tube are the same as for any patient who has undergone orotracheal intubation (Chapter 18).

COMPLICATIONS

Many of the complications associated with fiberoptic intubation are the same as those seen with direct laryngoscopy (Chapter 18). The most severe complication is hypoxemia from a prolonged procedure or delays due to inexperience. Use high-flow nasal oxygen to prevent hypoxemia.[54] Use the suction channel on the bronchoscope to provide oxygenation to the patient during the procedure. Epistaxis can be minimal or significant enough to complicate the procedure. Bleeding can be minimized by using the correct ET tube size and pretreatment with a nasal vasoconstrictor and anesthetic. Failure to intubate can be due to narrow nasal or airway passages, blood or vomitus limiting the fiberoptic field of view, or inexperience. Oxygen insufflation into the pharynx and esophagus works its way into the stomach. This can distend the stomach and cause it to rupture.[55]

While the insertion cord is passed through the glottis and into the trachea under direct vision, the ET tube is passed blindly over the insertion cord tip. It is possible to cause injury to the arytenoids resulting in permanent hoarseness if the ET tube bevel faces anteriorly. A randomized controlled trial showed no difference in the incidence of vocal cord injury for nasotracheal fiberoptic intubation versus orotracheal intubation.[56] The ET tube may become blocked in the nasal cavity or larynx, cause epistaxis, result in a nasal turbinate fracture, and tear the ET tube cuff.[10,57] Sinusitis and otitis media are known complications from nasal intubation.[10]

SUMMARY

Awake intubation under direct visualization in spontaneously breathing patients by the oral or nasal route is possible with the fiberoptic bronchoscope. Intubation by means of the fiberoptic bronchoscope is an option to consider when direct laryngoscopy is difficult or impossible. Awake fiberoptic intubation may be preferable in some instances to other techniques that render the patient unconscious and apneic. Fiberoptic intubation is associated with a high success rate when performed by appropriately trained individuals. Training and practice are required to develop and master the necessary skills.[58] Appropriate patient selection, preparation, and patience are essential for a safe and successful fiberoptic intubation.

The benefits of performing regional anesthesia of the oropharynx, larynx, and trachea prior to fiberoptic intubation are a quiet visual field and improved patient acceptance.

Oral fiberoptic intubation may be performed in conjunction with an LMA. In most instances, fiberoptic intubation is an extremely safe and effective means of securing the airway. Be prepared to implement an alternate plan for securing the airway and for providing oxygen in the case of procedural failure or sudden deterioration of the patient's condition.

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29 Nasotracheal Intubation

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INTRODUCTION

Nasotracheal intubation is a relatively simple procedure that is performed rapidly without the aid or risks of neuromuscular blockade.[1] This method of intubation is sometimes favored in difficult airway cases, especially when oral access is limited or impossible. Such conditions include trismus, oral injuries, and obstructive oral processes such as angioedema. Nasotracheal intubation is also the method of intubation preferred by some for acute epiglottitis.[2] **It is the most common method of inducing anesthesia for oral surgery cases to ensure optimal surgical access.[3]**

Nasotracheal intubation is well tolerated by most patients and produces less reflex salivation than orotracheal intubation, thus leading to fewer attempts at self-extubation. The nasotracheal tube is more easily stabilized and is generally easier to care for than an orotracheal tube. This method prevents biting of the tube by the patient and manipulation by the patient's tongue.[2,4]

INDICATIONS

Nasotracheal intubation is indicated in any patient with spontaneous respirations, especially those whose period of intubation is anticipated to be brief.[1-4] It is indicated in patients who are unable to lay supine due to respiratory distress from severe asthma, chronic obstructive pulmonary disease (COPD), or congestive heart failure. It is also indicated in patients who are unable to open their mouths due to facial trauma, mandibular trauma, or trismus. Nasotracheal intubation can be performed in patients with limited airway patency due to obstruction from neoplasm or tongue swelling. Nasotracheal intubation is an appropriate method of intubation in patients who require neck immobilization for suspected cervical spine injuries, patients unable to move their necks due to cervical kyphosis, patients with severe cervical arthritis limiting neck movement, or patients with postradiation fibrosis. Because they are often intubated for a short time, patients with severe alcohol intoxication or drug overdose whose level of consciousness is decreased are good candidates for nasotracheal intubation.[1,2,4] Nasotracheal intubation may be performed in patients who have contraindications to the use of succinylcholine (**Table 16-2**).

CONTRAINDICATIONS

Nasotracheal intubation is contraindicated in patients with apnea, severe facial or maxillofacial fractures, basilar skull fractures, head injury with an elevated intracranial pressure, penetrating neck injury, nasal obstruction, recent nasal surgery, or nasopharyngeal obstruction; patients receiving thrombolytics or parenteral anticoagulants; and in the presence of a coagulopathy.[1-5]

Nasotracheal blind intubation should not be performed in neonates, infants, or very young children. The more anterior and cephalic position of the airway in these age groups makes blind passage of an endotracheal tube (ETT) almost impossible. A patient must provide a degree of cooperation during the procedure. A crying, kicking, and struggling child who must be restrained is not a candidate for nasotracheal intubation.

EQUIPMENT

- Nasal mucosa vasoconstrictor (e.g., 4% cocaine, 0.05% oxymetazoline, 0.25% phenylephrine)
- Nasal mucosa anesthetic (e.g., viscous lidocaine, cocaine, benzocaine spray, xylocaine spray)
- Nasal atomizer (**Table 29-1 and Figure 29-1**)
- Nasopharyngeal airways, multiple sizes
- Laryngoscope handle
- Laryngoscope blades, various sizes and types
- ETTs, various sizes (average female 7.0 to 7.5 and average male 7.5 to 8.0)
- Endotrol tubes, various sizes (Mallinckrodt Medical, St. Louis, MO)
- Magill forceps or Tylke forceps (**Figure 29-2**)
- Suction apparatus

TABLE 29-1 Some of the Nasal Atomizers Commercially Available

Name	Manufacturer	Comments
DeVilbiss Model 15 Medical Atomizer	DeVilbiss Healthcare	Disposable and reusable versions Adjustable tip
Enk Fiberoptic Atomizer Kit	Cook Medical	Pressure-resistant oxygen tubing Atomizes through fiberoptic bronchoscope working channel
EZ-Spray	Alcove Medical	Disposable Trigger valve system
LMA MADdy Pediatric Mucosal Atomizer Device	Teleflex Medical	Disposable single-use Comes in various colors and shapes Child friendly Attaches to standard syringe 11.4 cm long
LMA Bottle MADomizer Atomizer	Teleflex Medical	Disposable single-use tips Reusable bottle 10 cm long
LMA MADgic Laryngo-Tracheal Atomizer	Teleflex Medical	Disposable single-use Attaches to standard syringe Flexible tube 21.6 cm long
LMA MAD Nasal-Intranasal Mucosal Atomizer Device	Teleflex Medical	Disposable single-use Attaches to standard syringe
MADomizer Caps	Wolfe Tory Medical	Disposable bottle and pump No need for compressed air

- Topical anesthetic (e.g., 4% cocaine or 2% lidocaine with epinephrine)
- Gauze strips
- Water-soluble lubricant or anesthetic jelly
- Bag-valve device
- Face mask
- Oxygen source and tubing
- Fiberoptic laryngoscope (Chapter 28)
- Video laryngoscope (Chapter 21)

Atomizer devices are usually not available in the Emergency Department. Many devices are available commercially. These are single patient use, disposable devices (**Figure 29-1 and Table 29-1**). A popular device is the MAD line of mucosal atomizer devices (LMA North America, San Diego, C). They make multiple devices for all topical needs.

The McGill forceps is the standard used by most institutions (**Figure 29-2*A***). The forceps are used to introduce the ETT into the patient's trachea from the hypopharynx under direct visualization. It is available in infant, child, and adult sizes. The forceps tip's twin blades for gripping are offset with serrated tips. It is curved at an oblique angle between the handles and the blades to prevent the view of the patient's airway from being obscured. The ends of the blades are rounded to minimize trauma.

The Boedeker forceps (Karl Storz Endoscopy, El Segundo, CA) is an alternative to the McGill forceps (**Figure 29-2*B***). It is also curved but does not put the working end into the view of the intubator.

The Tylke forceps (Rodinia LLC, Jupiter, FL) is a new device that is simple to use, safe, and an effective alternative to the McGill forceps (**Figure 29-2*C***). It allows nasotracheal intubations 25% of the time and reduces the possibility of torn cuffs and vocal cord trauma. The circular tip of the Tylke forceps prevents losing control of the ETT, without having to grip the ETT firmly, and allows the aiming of the ETT in the desired direction. The twist and bend of the Tylke forceps prevent the vocal cords from being visually obstructed and provide improved access to the trachea. It is available in three sizes (i.e., adult, adolescent, and child).

A

B

C

D

FIGURE 29-1. Some common nasal atomizers for topical nasal anesthesia. ***A.*** The LMA MAD nasal. ***B.*** The LMA MADgic Laryngeo-Tracheal. ***C.*** The child's LMA MADdy. ***D.*** The MADomizer.

A

B

C

D

FIGURE 29-2. Forceps used to aid intubation. ***A.*** The McGill forceps. ***B.*** The Boedeker forceps. (Photo courtesy of Karl Storz Inc.) ***C.*** The child, adolescent, and adult versions of the Tylke forceps. (Photo courtesy of Rodinia LLC.) ***D.*** The Tylke forceps easing an ETT tube through the vocal cords. (Photo courtesy of Rodinia LLC.)

Use of the Tylke forceps is simple (**Figure 29-2*D***). Insert the Tylke forceps into the mouth. Grab the ETT from the right side or slide the Tylke forceps over the tip of the ETT with the forceps vertical. Another method is to tilt the Tylke forceps left 90° to encircle the ETT from the top and then return the Tylke forceps to a vertical position. Aim the Tylke forceps at the vocal cords and have an assistant advance the ETT through the vocal cords and into the trachea. Pull the Tylke forceps back to the oropharynx while loosely wrapped around the ETT. Rotate the hand to the right, open, and disengage the Tylke forceps. Carefully remove the Tylke forceps from the mouth.

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative if time permits. All procedural steps should be clearly outlined, with the understanding that an orotracheal intubation may be necessary should the Emergency Physician fail to secure the airway nasotracheally. Since this is a lifesaving procedure, a signed consent may not be necessary, but a procedure note should be included in the medical record.

Prepare the patient with preoxygenation, hemodynamic monitoring, pulse oximetry, and vascular access. Place the patient supine and in the "sniffing" position if there is no suspicion of a cervical spine injury. If the patient needs to remain sitting due to respiratory distress, also place them in the sniffing position.

Examine the patient's nostrils. Choose the larger and more patent nostril for the intubation. The choice of which nostril to use is not an exact science.[6] Look at and into each nostril and determine which one is more patent. Estimate the airflow through each nostril.[7,8] Occlude one nostril and instruct the patient to exhale with their mouth closed. Repeat the process with the other nostril. Ask the patient which nostril they feel is more patent. This test can be repeated after the application of a vasoconstrictor agent. **There is some evidence that the right nares is preferred due to faster intubation times and less epistaxis**.[5,9,10]

Prepare the mucous membranes.[11] Apply a topical vasoconstrictor to shrink the nasal mucosa followed by a topical anesthetic to the nasal mucosa. It is best to perform this using a nasal atomizer (**Table 29-1 and Figure 29-1**). Use cotton soaked pledgets if an atomizer is not available. Cocaine is preferred, if not contraindicated, because it is a single agent that acts as both a vasoconstrictor and an anesthetic. Commonly, an equal mixture of 2 to 3 mL of 1% lidocaine and oxymetazoline 0.05% is sprayed or swabbed in the nasal mucosa. Dilate the nasal passage by serial dilation. Liberally lubricate a series of increasingly larger-size nasopharyngeal airways. Insert and then remove the smallest nasopharyngeal airway. Continue to insert and remove each successively larger nasopharyngeal airway until the nasal passage is dilated. This procedure can take 2 to 3 minutes. If time is an issue, insert a gloved and lubricated pinky finger into the nostril to dilate it. Serial dilation of the nasal passages can be bypassed in patients with large nostrils for routine nasotracheal intubations of healthy adults.[12] Apply a topical anesthetic spray to the palate and oropharynx.

Choose an ETT appropriate for the patient. The proper size tube should be at least 0.5 to 1.0 mm smaller than the size chosen for orotracheal intubation of the same patient. Soften the ETT by placing it in warm saline or warm water before use to reduce epistaxis and nasal damage if possible and time permits.[13] Apply a 10 mL syringe to the inflation port and inflate the cuff. Check the integrity of the cuff. Deflate the cuff and leave the syringe attached. Lubricate the ETT.

TECHNIQUES

BLIND PLACEMENT OF AN ETT

The technique of blind nasotracheal intubation was first described by Magill in 1930. The technique essentially remains the same with some modifications to increase the success rate and limit complications. This technique is technically more difficult than the placement under direct vision described below. **Its major advantages are that the patient's mouth does not have to be opened and minimal to no cervical spine movement is required.** This procedure may be performed while the patient is sitting or supine. Prepare the patient as mentioned previously.

Stand to the right side of the patient's bed and facing them. Insert the ETT into the nostril with the bevel facing the septum (**Figures 29-3 and 29-4*A***). Insert the ETT concave side down if the patient's right nostril is being used (**Figure 29-3*A***). Insert the ETT concave side up if the patient's left nostril is being used (**Figure 29-3*B***). Advance the ETT with gentle pressure along the nasal floor to pass it through the nasal cavity (**Figure 29-4*B***).

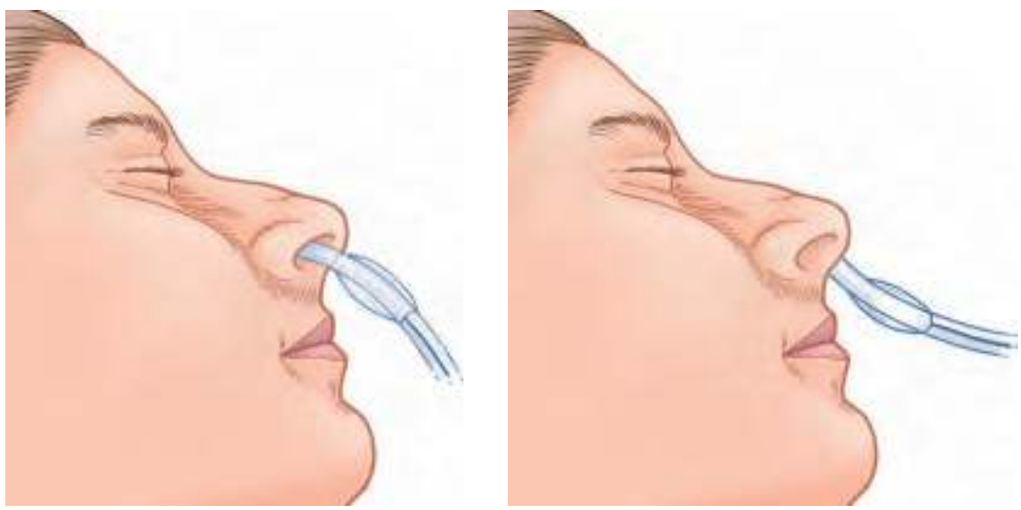

FIGURE 29-3. Insertion of the nasotracheal tube. The bevel of the ETT should face the septum. ***A.*** Placement in the right nostril with the concave side of the ETT downward. ***B.*** Placement in the left nostril with the concave side of the ETT upward. When the tip of the tube enters the nasopharynx, rotate it 180°.

Slightly withdraw the ETT if any resistance is felt. Readvance the tube with a slight twisting motion to bypass the obstruction. If resistance is still met, withdraw the ETT, prepare the other nostril, and insert the tube into the other nostril.

The tip of the ETT will be past the choana and in the nasopharynx when it is inserted approximately 5 to 7 cm (**Figure 29-4*B***). Continue advancing the ETT as resistance is met while it makes a 90° change of direction into the oropharynx. A slight twisting motion may be required to advance the ETT. A loss of resistance signifies that the ETT has made the curve. Several options are available if the ETT will not curve from the nasopharynx into the oropharynx. These include trying the other nostril, using an ETT 0.5 mm smaller, and reattempting intubation through the original nostril, or using an Endotrol tube (described in the next section). Stop advancing the ETT and rotate it so that its natural curve is concave upward and in the same curvature of the airway.

Advance the ETT through the oropharynx and into the laryngopharynx (**Figure 29-4*C***). Listen for breath sounds through the proximal end of the ETT while advancing it. The breath sounds and air movement will be maximal when the tip of the ETT is just above the glottis. As soon as an exhalation is heard, the patient will take a breath and advance the ETT. The vocal cords are opened their widest during inspiration. This will facilitate passage of the ETT.

The patient will often cough or gag as the ETT traverses the vocal cords. Breath sounds should be audible from the proximal end of the ETT, and it should fog with each breath. The esophagus has been intubated if the patient groans or speaks. Withdraw the ETT and reinsert it during inspiration. The application of posteriorly applied pressure on the trachea (i.e., Sellick's maneuver) will occlude the esophagus and may allow easier intubation.

The ETT may be caught in the hypopharynx if resistance to the advancement of the ETT is felt. Common sites for the tip of the ETT

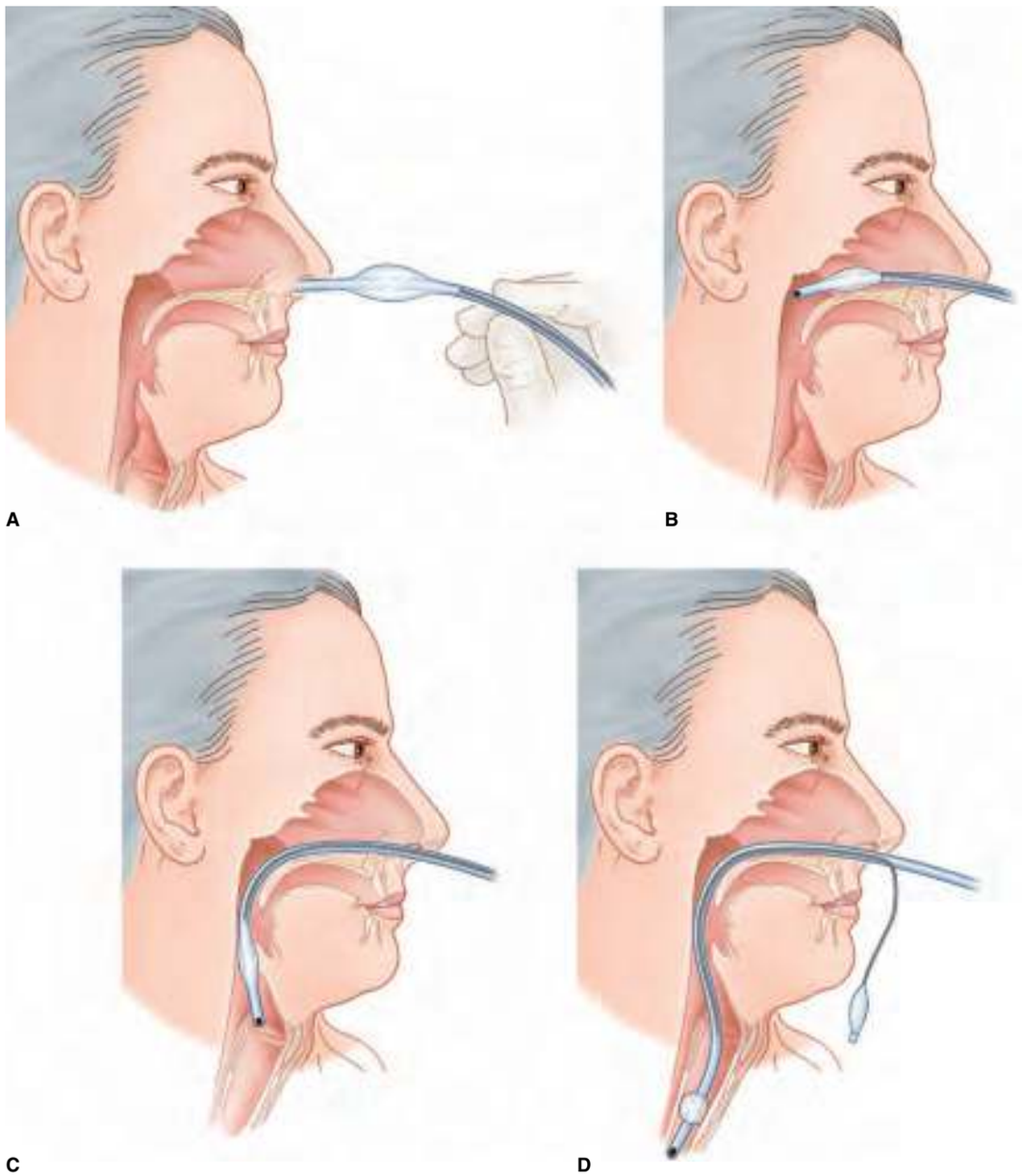

FIGURE 29-4. Blind nasotracheal placement. ***A.*** The ETT is placed within the nasal cavity. ***B.*** The ETT is advanced along the floor of the nasal cavity and into the nasopharynx. ***C.*** The ETT is advanced into the laryngopharynx. ***D.*** At the start of inspiration, the tube is advanced through the vocal cords and into the trachea.

to get caught are the arytenoid cartilage, piriform sinus, vallecula, and vocal cords. Withdraw the ETT 3 to 4 cm, slightly rotate the ETT clockwise or counterclockwise, and readvance it.

Inflate the ETT cuff. Confirmation of ETT placement should be assessed by auscultating both lungs while ventilating the patient with a bag-valve device through the nasotracheal tube. Adjust the position of the tube until both lungs are being ventilated equally and secure the tube (**Figure 29-4*D***). Continue to ventilate the patient.

PLACEMENT WITH FIBEROPTIC ENDOSCOPY

Fiberoptic bronchoscope endoscopy, also known as fiberoptic endoscopy, is an alternative to advance the nasal ETT through the nares of choice. It decreases epistaxis, results in better navigation than the blind techniques, and results in less redirection of the ETT.[14] There is no advantage between the fiberoptic bronchoscope and direct laryngoscopy in terms of the hemodynamic response to nasotracheal intubation.[15]

Prepare the nares and insert the ETT into the chosen nostril, as described above. Once the ETT is in the oropharynx, advance a prepared and appropriate-sized fiberoptic bronchoscope through the ETT (Chapter 28). The fiberoptic bronchoscope may be connected to oxygen or suction based on Emergency Physician preference. The view through the fiberoptic bronchoscope will reveal the epiglottis and vocal cords after exiting the nasal ETT distal opening. Consider the injection of 1% lidocaine through the fiberoptic bronchoscope side port to anesthetize the vocal cords. Advance the fiberoptic bronchoscope through the vocal cords and visualize the tracheal rings. **Gently advance the endotracheal tube over the fiberoptic bronchoscope.** Remove the fiberoptic bronchoscope. Connect the ETT to a CO_2 monitor or device and ventilator.

BLIND PLACEMENT OF THE ENDOTROL TUBE

The indications, contraindications, and patient preparation are the same as described previously. The Endotrol tube is an ETT whose tip can be controlled. It looks like a cuffed ETT but has a plastic ligature along the inner side that is connected to a ring on the proximal end of the ETT (**Figure 29-5**). Pulling of the ring exerts tension on the plastic ligature, leading to an increase in the curvature of the tip of the ETT. This will project the tip anteriorly and inferiorly (**Figure 29-6**). The procedure for inserting the Endotrol tube is the same as that for inserting an ETT. Changing

FIGURE 29-5. The Endotrol ETT. The curvature can be changed by pulling on the ring to facilitate intubation.

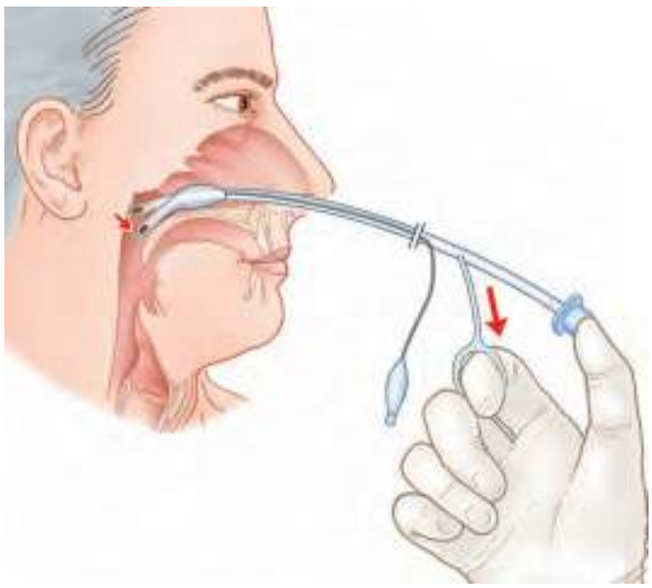

FIGURE 29-6. Blind nasotracheal placement of an Endotrol tube. Tension exerted on the ring of the tube causes the curvature of the tube to increase (*arrow*).

the curvature of the tip will aid in passage of the tube from the nasopharynx to the oropharynx and from the hypopharynx into the trachea. The tip of the Endotrol tube may be exerting continuous pressure on the anterior tracheal mucosa if the ring is sitting firmly against the nares after intubation. Cut the ligature and remove the ring.

PLACEMENT UNDER DIRECT VISION

This technique begins with nasotracheal intubation followed by direct laryngoscopy. **The placement of a nasotracheal tube using direct visualization must be performed with the patient supine.** The indications and precautions are similar to those for orotracheal intubation (Chapter 18). This method should be considered in the event of an oral injury that renders an orotracheal tube a nuisance, if blind nasal intubation is unsuccessful, or if nasal fiberoptic intubation is unsuccessful.

This procedure is initially performed as previously described. Direct laryngoscopy is performed once the ETT is inserted into the hypopharynx. Use the left hand to grasp the laryngoscope and insert the blade. A video laryngoscope may also be used.[16-21] Look into the patient's mouth and visualize the epiglottis, vocal cords, and ETT. Use a Magill forceps or Tylke forceps (**Figure 29-2**) with the right hand to grasp the ETT just above the cuff (**Figure 29-7**). **Never grasp the ETT cuff, as it is delicate and can easily be damaged by the forceps.** Have an assistant grasp the proximal end of the ETT and gently advance it while the Emergency Physician simultaneously guides the tip through the vocal cords (**Figures 29-2*C* and 29-7**). Remove the forceps and the laryngoscope. Inflate the cuff, secure the tube, and ventilate the patient. The depth of the endotracheal tube can be verified by fiberoptic bronchoscopy.

BLIND DIGITAL INTUBATION

A technique was developed that combines blind nasotracheal intubation and digital orotracheal intubation.[22] This technique starts with the procedure of blind nasotracheal intubation. If not successful, insert the index and middle fingers of the nondominant hand into the patient's mouth. Slide these fingers posteriorly over the

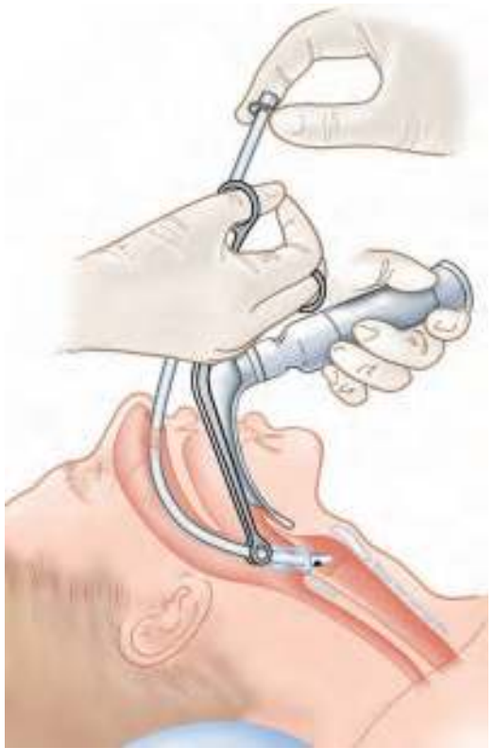

FIGURE 29-7. Nasotracheal intubation under direct visualization.

tongue to palpate the epiglottis. Grasp the tip of the ETT between the two fingers (**Figure 29-8*A***). Pull the ETT anteriorly and behind the epiglottis (**Figure 29-8*A***). Use the dominant hand to advance the ETT further into the patient's nose, advancing the tip into the trachea (**Figure 29-8*B***).

PEDIATRIC CONSIDERATIONS

Very little literature is available regarding nasotracheal intubation on children. In the past, this approach was an option for patients with epiglottitis and acute laryngotracheobronchitis.[23] The most recent data reserve nasotracheal intubation for children with congenital facial anomalies.[24] This technique cannot be recommended for children in the Emergency Department. Their small nostrils limit ETT size. Large adenoids may make passage of the ETT difficult and increase the risk of bleeding.[25] The use of an uncuffed ETT in children decreases the complications.[26] Consider the use of a feeding tube to guide the ETT and reduce complications.[27] Rotation of the ETT 90° counterclockwise during fiberoptic bronchoscope endoscopy may improve the success rate.[28]

ALTERNATIVE TECHNIQUES

Numerous alternatives have been used to facilitate nasotracheal intubation. A nasogastric tube (Chapter 75) may facilitate nasotracheal intubation.[29,30] The nasogastric tube is inserted first and the ETT is fed over the nasogastric tube. The nasogastric tube goes below the inferior turbinate and decreases the rate of epistaxis. The ETT alone usually passes between the middle and superior turbinates, causing epistaxis and turbinate trauma. Other equipment placed in the nares to form a conduit for the ETT include a bougie, red rubber catheter, and suction catheter.[31-33] **Use caution with these devices and only use them to get the ETT into the oropharynx. Advancement of these devices into the lung can result in perforation.** A video laryngoscope may be used instead of the direct laryngoscope.[14,16-19,21,34] Video laryngoscopy is quicker than using the fiberoptic bronchoscope.[34]

ASSESSMENT

The position of the ETT should be confirmed by end-tidal CO_2 monitoring, fogging in the ETT for at least six ventilations, loss of voice, auscultation over the chest and the epigastrium, and a chest

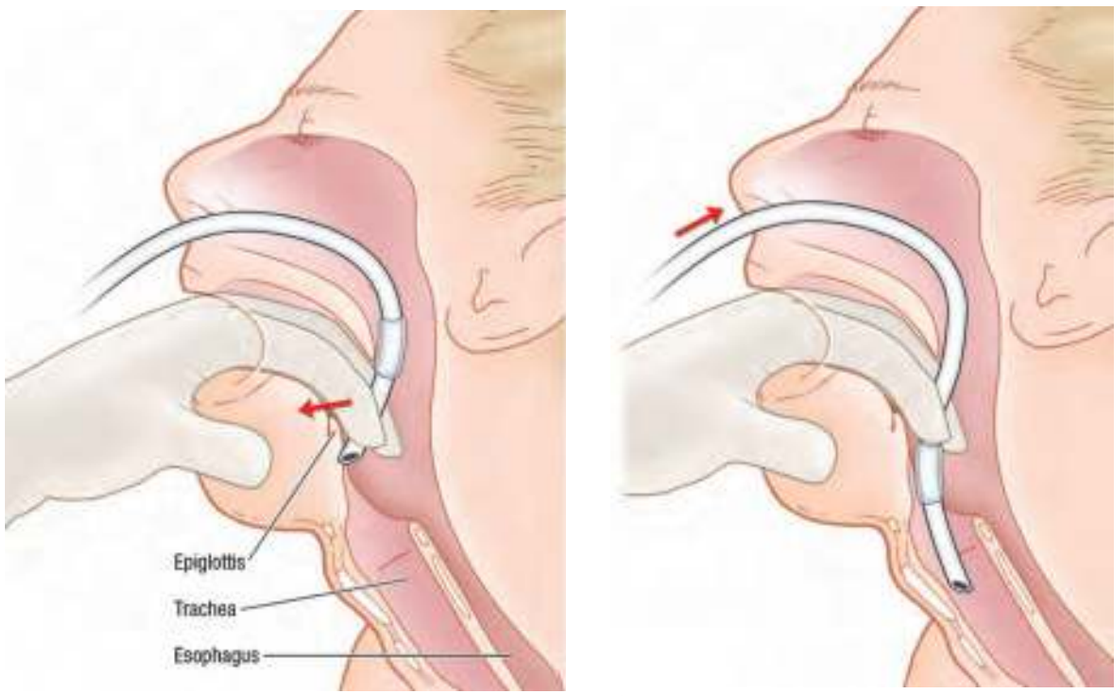

FIGURE 29-8. Digital nasotracheal intubation. ***A.*** The ETT is grasped with the fingertips and pulled anteriorly (*arrow*) behind the epiglottis. ***B.*** The ETT is advanced into the trachea.

radiograph. Please refer to Chapter 19 for a more detailed discussion regarding confirmation of endotracheal intubation.

AFTERCARE

The ongoing care of the patient should proceed as with any other intubation technique.

COMPLICATIONS

The immediate complications of nasotracheal intubation include epistaxis, laryngeal and tracheal trauma, mucosal avulsion, retropharyngeal laceration, turbinate avulsion, intracranial placement, bacteremia, esophageal intubation, and prolonged attempts to place the tube.[9,14,25,32,35-41] Many of these can be prevented by choosing the appropriate size ETT, ensuring adequate nasal mucosal vasoconstriction, and applying a liberal amount of lubricant to the ETT.[29] Risk factors for epistaxis should be assessed in every patient prior to nasotracheal intubation.[42] Long-term complications include maxillary sinusitis, retropharyngeal abscesses, mediastinitis, nasal mucosal necrosis, and cellulitis.[9,39,41]

SUMMARY

Nasotracheal intubation is an alternative to orotracheal intubation to secure an airway in the spontaneously breathing patient. It allows for the awake intubation while the patient maintains protective airway reflexes. Nasotracheal intubation avoids the risks of paralytic agents. It is a fairly simple procedure that should be considered in patients in whom an oral airway is considered difficult and in those with an anticipated short intubation period.

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30 Retrograde Guidewire Intubation

Ned F. Nasr, Anna Tzonkov, and Gennadiy G. Voronov

INTRODUCTION

Oral endotracheal intubation via direct laryngoscopy or video-assisted laryngoscopy remains the "gold standard" of airway management. Difficult situations arise in which oral endotracheal intubation is impossible, is contraindicated, or fails. **Retrograde guidewire intubation is an alternative airway management technique.** The American Society of Anesthesiology difficult airway algorithm describes retrograde intubation as an alternative airway in the nonemergent pathway when mask ventilation is adequate but multiple intubation attempts are not successful. The technique should be familiar to those involved with emergency airway management.[1,2]

Retrograde intubation was first described in 1960 by Butler and Carillo as a way to remove a tracheostomy tube in neck surgery.[3] In 1963, Waters described insertion of an epidural catheter through a cricothyroid puncture as an alternative means of establishing an airway.[4] Powell and Odzil reported a series of patients in whom retrograde intubation was employed without complications using a plastic catheter rather than an epidural catheter as a guide.[5] The current technique of retrograde intubation varies little from these original descriptions.

Retrograde intubation represents one of several alternative maneuvers for securing the difficult airway. The technique can be used in awake, sedated, or obtunded patients who have either an anticipated or unanticipated difficult airway.[6] The technique can be performed despite presence of secretions or blood in the oropharynx. Retrograde intubation has proven to be an effective method used by Emergency Physicians to establish a definitive airway.

The success rate of retrograde intubation is variable.[7] Completion times for retrograde intubation vary based on physician experience. The mean length of time to intubation was 71 ± 4 seconds among health care professionals who had no prior experience with the technique but who had just completed a mannequin-aided training course.[8] The completion of retrograde intubation within 150 seconds with a mean intubation time of 56 ± 6 seconds was seen in resident physicians after a brief instruction course.[9]

INDICATIONS

The American Society of Anesthesiologists defines a difficult airway "as the clinical situation in which a conventionally trained anesthesiologist experiences difficulty with mask ventilation, difficulty with tracheal intubation, or both."[1] **Consider performing retrograde intubation in any patient in whom endotracheal intubation may be difficult, is contraindicated, or has failed.[10-16] The technique can be used in awake, sedated, or obtunded patients who have either an anticipated or unanticipated difficult airway.[6]** It is potentially indicated when airway control is required and less invasive methods have failed. Maxillofacial trauma and cervical spine fractures represent the most common etiologies of a difficult airway.[17] Retrograde intubation was successful on the first attempt in these patients. Ankylosing spondylitis, rheumatoid arthritis, trismus, and congenital anomalies represent another group of challenging airway situations where retrograde intubation could be considered. It is also useful when bleeding obstructs visualization of the glottis or as an alternative in situations where a flexible fiberoptic scope is not available.[6,18]

Another clinically important situation arises when a patient presents with impending ventilatory failure. While retrograde intubation is generally a longer procedure than orotracheal intubation, oxygenation and ventilation can be maintained with a bag-valve-mask device during the procedure.

A less common indication includes retrograde intubation of a difficult airway in a patient ventilated with a laryngeal mask airway or an intubating laryngeal mask airway.[19,20] This indication exists because withdrawal of the laryngeal mask airway over a blindly placed catheter can result in dislodgement of the catheter, necessitating replacement of the laryngeal mask airway.[21]

CONTRAINDICATIONS

The major contraindication to retrograde intubation is the ability to control the airway with less invasive techniques. Other contraindications include an anterior neck mass overlying the cricothyroid membrane, local infection over the skin covering the cricothyroid membrane, coagulopathy, and tracheal stenosis. Apneic patients who cannot be ventilated with a bag-valve-mask device should receive a cricothyroidotomy and not a retrograde guidewire intubation. Those unfamiliar with the equipment and/or technique should not attempt this procedure. Familiarity with the procedure is required for optimum patient management.[8,22]

EQUIPMENT

- 68 to 80 cm spring guidewire with a J-tip
- 16 to 18 gauge catheter-over-the-needle (i.e., angiocatheter)
- Endotracheal tubes, various sizes
- Sterile saline
- 10 and 20 mL syringes
- 18 gauge needles
- Hemostats
- Magill forceps or Boedeker forceps
- Sterile drapes
- Povidone iodine or chlorhexidine solution
- Face mask
- Bag-valve device
- Oxygen source and tubing
- Suction source and tubing
- Yankauer suction catheter
- 1% lidocaine
- 4% viscous lidocaine (optional)
- Topical anesthetic (e.g., lidocaine or benzocaine)
- Tape or a commercially available endotracheal tube holder

Retrograde guidewire intubation can be performed using a standard commercial retrograde intubation kit (Cook Retrograde Intubation Set; Cook Medical, Bloomington, IN). It consists of an 18 gauge needle set, 68 to 80 cm spring guidewire, an 11 French introducer catheter, a hemostat, and respiratory adapters (**Figure 30-1**). The remainder of the materials must be supplied.

PATIENT PREPARATION

The success of retrograde intubation in a patient who is awake depends on the patient's collaboration. If time permits and the patient is aware of pain, anesthetize the airway. Bilateral superior laryngeal nerve blocks and a transtracheal block would provide

FIGURE 30-1. The retrograde guidewire intubation kit. (Courtesy of Cook Medical.)

anesthesia for the airway below the epiglottis.[21] Clean the patient's neck of any dirt and debris. Identify, by palpation, the hyoid bone, thyroid cartilage, cricoid cartilage, and cricothyroid membrane. Apply povidone iodine to the patient's neck followed by sterile drapes.

Anesthetize the patient's airway. A superior laryngeal nerve block is performed by locating the greater horn of the hyoid bone. Insert a 25 gauge spinal needle attached to a 10 mL syringe filled with 2% lidocaine until the anterior horn of the hyoid bone is contacted. Walk the needle off the anterior horn of the hyoid bone and toward the thyrohyoid membrane. Advance the needle 1 to 2 cm. Aspirate to ensure the tip of the needle is not within a blood vessel. Inject 2 mL of 2% lidocaine. Nebulized viscous lidocaine is an alternative that will anesthetize the airway in 15 to 20 minutes if there is time.[17] A transtracheal block is performed by locating and penetrating the cricothyroid membrane with a 20 gauge needle and injecting 2 to 4 mL of 4% lidocaine. This will elicit a vigorous cough, which will spread the lidocaine, anesthetize the trachea, and anesthetize the vocal cords. Lidocaine or benzocaine may be sprayed into the pharynx as topical anesthesia. These blocks may allow the patient to tolerate the procedure better, depress the swallowing reflex, and lead to aspiration.[2]

Prepare the equipment. Place the 16 or 18 gauge catheter-over-the-needle onto a 10 mL syringe containing 3 to 5 mL of sterile saline. Select an appropriate size endotracheal tube for the patient. Check the integrity of the cuff. Lubricate the inside and outside of the distal tip of the endotracheal tube liberally. Open the retrograde guidewire kit and/or assemble all equipment. The equipment should be preassembled, prepackaged, sterilized, and stored in an easily accessible site.

TECHNIQUE

The procedure is relatively simple in theory but difficult to perform "in the heat of battle."[8,9,23-25] Position the patient. **The ideal patient position is supine with their neck in extension.** An alternative is the sitting position or with the neck in a neutral position. Ultrasound guidance may be used if anatomic landmarks are difficult to identify.[18] Stabilize the patient's larynx with the thumb and middle finger of the nondominant hand (**Figure 30-2**). Identify the cricothyroid membrane with the index finger of the nondominant hand. Leave the index finger on the cricothyroid membrane.

Insert the catheter-over-the-needle guided along the index finger, at a 20° to 30° angle upward, and through the cricothyroid membrane (**Figure 30-2*A***).[26] Some physicians prefer to use the needle without the catheter even though this is not recommended. The sharp needle within the trachea can cause significant injury when compared to the soft catheter and can shear off the guidewire. **Care should be taken to puncture the cricothyroid membrane just above the cricoid cartilage to avoid injury to the cricothyroid arteries**. The loss of resistance signifies that the needle is in the trachea. **Aspirate air through the saline-filled syringe to confirm correct needle placement** (**Figure 30-2*A***). Advance the catheter until the hub is against the skin. Remove the needle and syringe while leaving the catheter pointed upward and through the cricothyroid membrane. If this has not already been done and the patient is awake, inject 2 mL of 2% to 4% lidocaine through the catheter to anesthetize the airway.

Advance the guidewire through the catheter and into the oropharynx (**Figures 30-2*B* and 30-3**). The guidewire may exit the mouth or nose. The preferred site of exit is the mouth, but the nose is acceptable. Insert the laryngoscope if the guidewire is not visualized and look for the guidewire. It is often in the oropharynx or hypopharynx. Retrieve it with a Magill or Boedeker forceps. Continue to advance the guidewire through the mouth (or nose) until only 4 to 5 cm of the wire is protruding from the patient's neck. **Carefully remove the catheter while firmly holding the guidewire in place**. **Place a hemostat on the guidewire where it enters the skin of the neck (Figure 30-2*C*). This will ensure that the guidewire does not pull through the skin and into the trachea.**

Select the introducer catheter if a retrograde guidewire kit is being used. Pass the introducer catheter over the guidewire that is exiting the mouth (or nose). Advance the catheter until resistance is met. This signifies that the tip of the introducer catheter is at the inside of the cricothyroid membrane (**Figure 30-2*C***). Advance the well-lubricated endotracheal tube over the introducer and guidewire (**Figure 30-2*D***). Continue to advance the endotracheal tube until resistance is met. The tip of the endotracheal tube should be at the inside of the cricothyroid membrane (**Figure 30-2*D***). While securely holding the endotracheal tube at the patient's mouth, remove the hemostat from the guidewire. Pull on the proximal end of the guidewire until the distal tip is through the skin and just into the endotracheal tube. **Simultaneously withdraw the guidewire and introducer catheter while advancing the endotracheal tube into the trachea** (**Figure 30-2*E***). Inflate the endotracheal tube cuff and confirm proper placement (e.g., auscultation, detection of end-tidal CO_2, and fogging in the endotracheal tube).

A second method can be used to insert the endotracheal tube. This follows the same technique described above to the point of the guidewire exiting the mouth (or nose), the guidewire being secured with a hemostat at the neck, and passing the introducer catheter over the guidewire. Remove the hemostat from the guidewire. **While securely holding the introducer catheter at the patient's mouth (or nose), remove the guidewire through the mouth (or nose)**. Advance the introducer catheter an additional 2 to 3 cm into the trachea. Lubricate the endotracheal tube liberally. Place the endotracheal tube over the introducer catheter. Hold the introducer catheter securely and advance the endotracheal tube into the patient's trachea. Remove the introducer catheter. Inflate the endotracheal tube cuff and confirm proper placement (e.g., auscultation, detection of end-tidal CO_2, fogging in the endotracheal tube).

ALTERNATIVE TECHNIQUES

This technique may be performed without a formal retrograde intubation kit because the introducer catheter is not required.[8,17,24] This follows the same technique described above to the point of

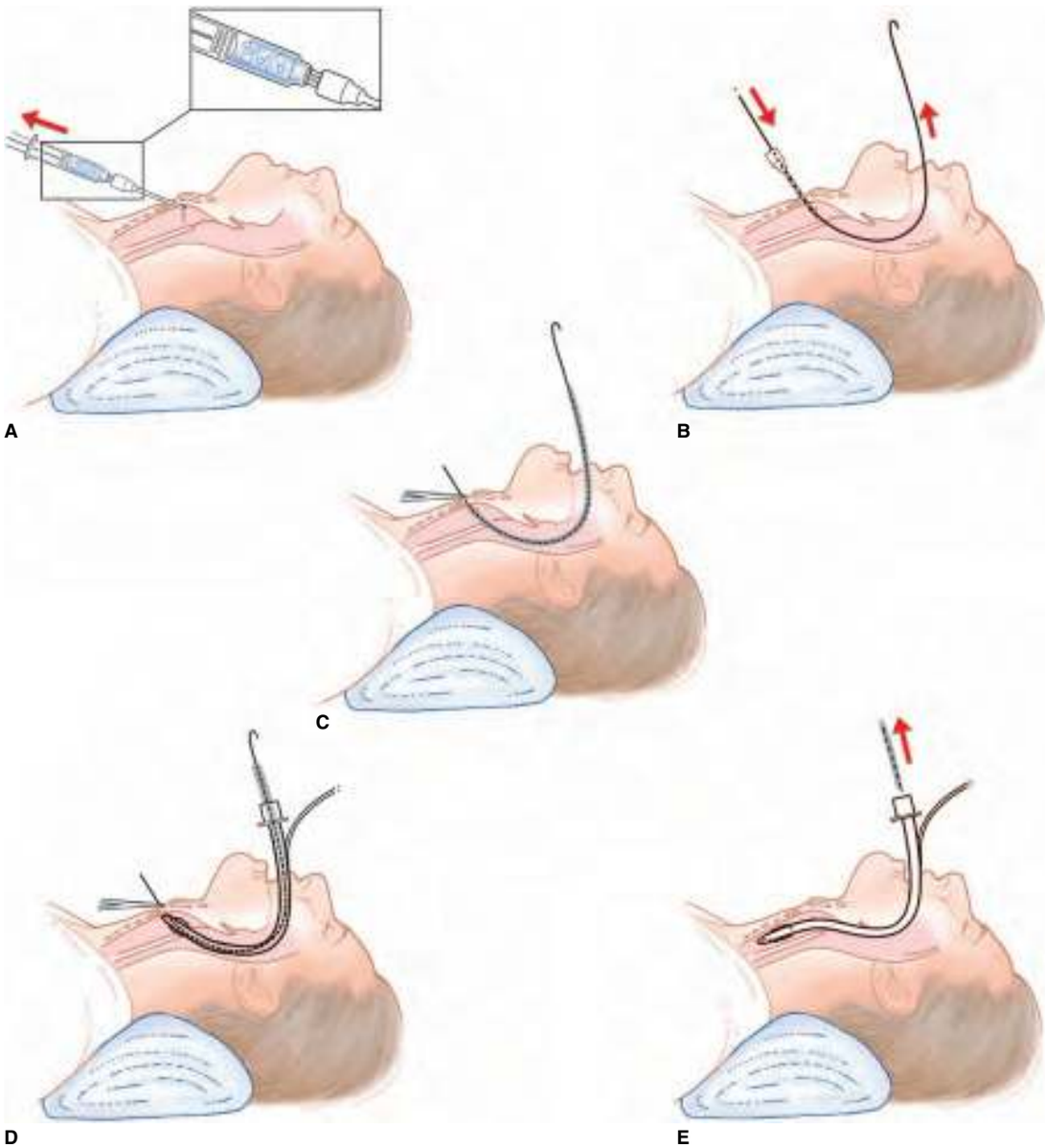

FIGURE 30-2. Retrograde guidewire intubation. ***A.*** A syringe containing saline is attached to the catheter-over-the-needle. The catheter-over-the-needle is inserted through the cricothyroid membrane at a 20° to 30° angle. The air bubbles in the syringe indicate air aspirated from the trachea. For clarity, the physician's hand and fingers stabilizing the airway and identifying the cricothyroid membrane are not seen in this illustration. ***B.*** The needle and syringe have been removed and the catheter remains. The guidewire is fed through the catheter and out the patient's mouth. ***C.*** The distal guidewire is clamped with a hemostat as it exits the skin of the neck. The introducer catheter has been fed over the guidewire and advanced to the cricothyroid membrane. ***D.*** An endotracheal tube is advanced over the guidewire and introducer catheter until its tip is at the cricothyroid membrane. ***E.*** The hemostat has been removed and the endotracheal tube is advanced as the guidewire and introducer catheter are removed.

the guidewire exiting the mouth (or nose) and being secured with a hemostat at the neck (**Figure 30-2*C***). Lubricate the endotracheal tube liberally. Insert the guidewire through the Murphy eye and into the endotracheal tube (**Figure 30-4**). This allows the distal tip of the endotracheal tube to project approximately 1 cm distal to the site at which the guidewire enters the larynx. Some physicians prefer to load the guidewire through the tip of the endotracheal tube as an alternative. **Always hold the proximal end of the guidewire to maintain control during the procedure**.

Advance the endotracheal tube over the guidewire until resistance is felt. The tip of the endotracheal tube should be at the inside of the cricothyroid membrane (**Figure 30-4**). **Hold the proximal end of the guidewire firmly**. Release the hemostat over the neck. Pull the guidewire through the skin and just into the endotracheal tube. Advance the endotracheal tube until it is at 20 to 21 cm at the teeth for an adult female or 22 to 23 cm at the teeth for an adult male. Hold the endotracheal tube securely at the patient's lips. Withdraw the guidewire through the endotracheal tube. Inflate the endotracheal tube cuff and confirm proper placement (e.g., auscultation, detection of end-tidal CO_2, and fogging in the endotracheal tube).

When the endotracheal tube is advanced over the guidewire until resistance is met, the tip should be situated against the inside of the cricothyroid membrane. **It is imperative to determine if the tip of the tube is in the trachea or caught on the epiglottis, arytenoid cartilage, pyriform recess, vallecula, or vocal cords.** Withdraw the endotracheal tube 2 cm if concern exists as to the position of the tip, rotate it 90°, and readvance it into the trachea. A laryngoscope or

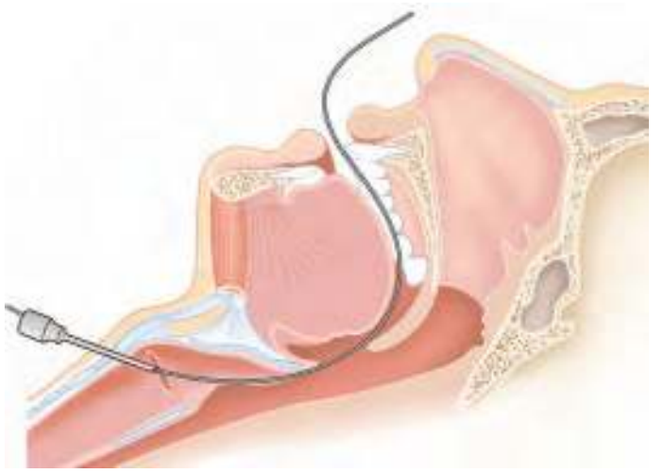

FIGURE 30-3. The guidewire is inserted through the catheter until it exits the mouth.

fiberoptic bronchoscope can be inserted to help visualize the placement of the endotracheal tube.

Another variation involves the use of the guidewire sheath as an introducer catheter.[24,25] Shorten the sheath by 3 to 5 cm using sterile scissors. The remainder of the technique is the same as described above. The only drawback to this technique is that the curvature of the sheath must be straightened before use to allow easy threading over the guidewire.

A central venous catheter or nasogastric tube can be used rather than a guidewire.[17,27,28] It allows the physician to inject air through the catheter in retrograde fashion to help locate the catheter in the mouth of the severely injured patient with significant intraoral blood or secretions. This technique requires a relatively long central venous catheter. It does allow retrograde intubation without the use of a formal retrograde intubation kit.

Another version uses a lighted stylet attached to the endotracheal tube.[29] The lighted stylet acts as a guide to indicate the tube's location. When the tip of the endotracheal tube enters the glottic opening, a bright glow is readily seen in the anterior neck below the thyroid prominence. This glow acts as an indicator of correct endotracheal tube placement.

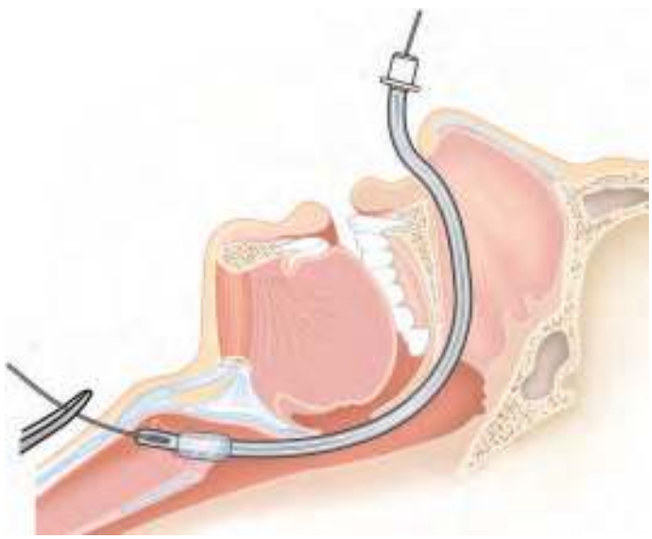

FIGURE 30-4. The endotracheal tube is advanced over the guidewire until the tip is against the cricothyroid membrane.

There are two possibilities when continuous oxygenation is required throughout the procedure.[12] A T-adapter can be connected to the needle hub with its side arm for oxygen insufflation. A swivel adapter with a fiberoptic bronchoscopic cap can be interposed between the endotracheal tube and the bag-valve device or anesthesia breathing circuit.

ASSESSMENT

Auscultation of both lungs will confirm proper placement of the endotracheal tube and minimize the risk of intubation into the right mainstem bronchus. End-tidal CO_2 has also become part of the postintubation routine. A chest radiograph will confirm the placement of the endotracheal tube tip in relation to the clavicles and carina. Please refer to Chapter 19 for a more complete discussion of the methods to confirm endotracheal intubation.

AFTERCARE

The patient should receive standard wound care and dressing of the skin at the neck entrance site. Wound checks and infection monitoring should continue as with any other surgical procedure. The risk of skin, tracheal, or pharyngeal infection is minimal if sterile technique is followed. Evaluate the wound and treat with appropriate antibiotics if an infection develops.

COMPLICATIONS

While complications may occur in association with retrograde intubation, the rate of complications is relatively low. Complications of retrograde guidewire intubation include those of standard endotracheal intubation. Complications can occur when the needle traverses the cricothyroid membrane.[23] **Hypoxia due to prolonged intubation time or incorrect endotracheal tube placement remains an important complication.** Drug reactions or side effects secondary to administered medications must always be considered.

Retrograde intubation is associated with additional complications related to the use of the guidewire. One case report discusses a patient with a past history of retrograde intubation who experienced a foreign-body sensation and bloody sputum 2 years after the procedure.[30] The patient was found to have a 10 cm segment of guidewire fixed in the soft tissue of the puncture site and extending cephalad 2 cm past the true vocal cords. The skin puncture can occur below the cricothyroid membrane. Injuries can occur to the thyroid or cricoid cartilage, the posterior wall of the larynx, the epiglottis, or the soft palate. The clinical importance of these injuries other than being a source of pain is unclear.

Three technical complications from retrograde guidewire intubation have been identified.[23,24] Difficulties inserting the guidewire can be prevented by first aspirating air into a saline-filled syringe to confirm the proper intratracheal needle tip position. Endotracheal intubation over a flexible guidewire necessitates keeping the guidewire taut to minimize the risk of kinking. This moves the guidewire anteriorly toward the narrowest portion of the glottis and may prevent passage of the endotracheal tube as the tip can become caught on the epiglottis or the vocal cords. This problem is obviated by the use of the introducer catheter in the retrograde guidewire intubation kit. The tip of the endotracheal tube may flip out of the larynx when the introducer is being removed. The distance between the vocal cords and the point where the introducer enters and anchors the larynx averages only 1.0 to 1.3 cm in adults. The guidewire can be fed caudally into the trachea due to improper angling of the needle. The use of ultrasound visualization can help to locate the needle and guidewire in the tracheal lumen and possibly aid in the successful performance of the procedure while avoiding complications.[18,22]

SUMMARY

Retrograde guidewire intubation requires little operator experience and minimal equipment and has a high level of skill retention.[31] Multiple reports suggest that this technique is safe, relatively easy to learn, and routinely successful. All Emergency Physicians involved in the airway management of critically ill and injured patients should be aware of this technique as a potential method to overcome the challenge of a difficult airway. Retrograde guidewire intubation should be given due consideration in any situation in which orotracheal intubation is impossible or contraindicated.

Numerous difficult airway management devices and adjuncts have been invented and marketed. The various video-assisted laryngoscopy devices have come to dominate the realm of alternative airway management techniques. The role of retrograde guidewire intubation as a difficult airway management approach has further diminished as a consequence. However, retrograde guidewire intubation remains an easy-to-learn and potentially lifesaving tool in the Emergency Physician's airway management armamentarium.

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31 Percutaneous Transtracheal Jet Ventilation

Gennadiy Voronov, Tamer Elattary, and Ned Nasr

INTRODUCTION

Percutaneous transtracheal jet ventilation (PTTJV) provides emergency ventilatory support in patients who cannot be adequately ventilated with a bag-valve-mask device using oral or nasal airways, a laryngeal mask airway (LMA), or endotracheal tube.[1-3] This includes patients with upper airway foreign bodies, upper airway neoplasms, maxillofacial trauma, laryngeal edema, or infection.[2,4] It is used electively with general anesthesia for surgery involving the larynx and subglottic areas.[5] PTTJV involves inserting a catheter-over-the-needle (i.e., angiocatheter) through the cricothyroid membrane and delivering oxygen to the lungs using a high-pressure oxygen delivery system.[6]

ANATOMY AND PATHOPHYSIOLOGY

Early studies of transtracheal ventilation used transtracheal catheters connected to 4 to 5 L/min of oxygen.[7] Oxygenation with this apparatus was adequate, but patients quickly developed hypercarbia due to lack of ventilation.[8] This "apneic oxygenation" also occurs in ventilation through a catheter attached to a bag-valve device.[9] The low pressure and flow of oxygen generated by the bag-valve device results in increases in $PaCO_2$ of 4 mmHg/min and the rapid development of respiratory acidosis.[1,10] Numerous studies have since demonstrated that intermittent jets of pressurized 100% oxygen at 50 pounds per square inch (psi) allow for both oxygenation and adequate ventilation.[10,11]

The anterior neck provides direct access to the airway via the trachea as it extends from the larynx into the lungs (**Figure 31-1**). At the top of the laryngeal skeleton is the thyroid cartilage. It lies at the level of the fourth and fifth cervical vertebrae. The laryngeal prominence (i.e., Adam's apple) of the thyroid cartilage is more prominent in men and is easily palpated. The cricoid cartilage lies just inferior to the thyroid cartilage at the level of the sixth cervical vertebra. It serves as the junction of the larynx and trachea. Multiple cartilaginous rings support the trachea. Between the cricoid and thyroid cartilages lies the cricothyroid membrane.

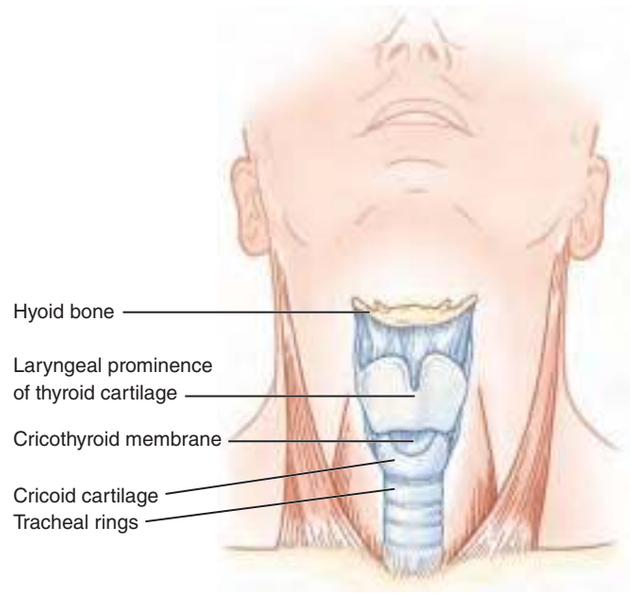


FIGURE 31-1. Airway structures of the neck.

The cricothyroid membrane is a palpable membranous depression just inferior to the laryngeal prominence and is the access site for PTTJV.[12] The cricothyroid artery is a branch of the superior thyroid artery. It travels transversely across the cricothyroid membrane just below the thyroid cartilage. **Placement of the catheter through the lower half of the cricothyroid membrane will prevent injury to this small artery**.[13]

The Emergency Physician should attempt to insert the largest possible catheter based on the limited information obtained from palpation of the landmarks. The cricothyroid membrane is found at different distances from the skin that vary based on patient weight and neck circumference. The mean of depth distance is approximately 2.3 mm.[14] Ultrasonography can be used to identify the landmarks if unable to palpate the cricothyroid membrane.[14-16] It is usually more difficult to identify the cricothyroid membrane in adult females compared to adult males irrespective of body weight.[16]

Oxygen is delivered via bulk flow through the cannula into the trachea and lungs. Entrainment of room air translaryngeally via the Venturi principle is negligible, even with minimal upper airway obstructions.[1] Near 100% O_2 is delivered with each insufflation.

Inhalation occurs through the catheter via a pressurized flow of oxygen. Exhalation occurs passively through the elastic recoil of the lungs and chest wall.[3] The minute ventilation delivered during PTTJV is proportional to the volume of air injected, the driving air pressure, and the degree of upper airway obstruction.[2,17] Animal studies demonstrate that PTTJV delivers more tidal volume than positive-pressure mask ventilation with the same tracheal and transpulmonary pressures despite delivery of oxygen from a pressurized source.[2]

INDICATIONS

PTTJV is indicated as a backup emergent airway in any patient who cannot be endotracheally intubated or ventilated with a bag-valve device despite the use of a jaw-thrust maneuver, oropharyngeal airway, nasopharyngeal airway, or LMA.[1,2,8] It serves as a simple, relatively safe, and effective alternative to cricothyroidotomy.[1,7] It can be performed at any age. PTTJV is preferable to a surgical cricothyroidotomy in infants and children up to 10 to 12 years old due to it being easier to perform and its potentially fewer complications.[18]

PTTJV is the procedure of choice in the pediatric age group for establishing an emergent airway when endotracheal intubation fails.[9] PTTJV can serve as a quick alternative to a difficult intubation. It is especially valuable in cases of maxillofacial trauma, suspected cervical spine injury, or when nasal intubation is contraindicated or unsuccessful.[7,8,19] PTTJV is occasionally performed by the Anesthesiologist as a method of airway management when a difficult intubation is anticipated.[20]

This procedure is indicated in cases of partial upper airway obstruction due to foreign bodies, laryngeal edema, neoplasm, or infection.[2,4] PTTJV serves as an emergent airway in cases of upper airway foreign bodies and can assist in dislodging the foreign body.[7,9] Animal studies have demonstrated that the expulsion of foreign bodies from the hypopharynx and upper trachea is possible with high-frequency jet ventilation, an effect similar to the Heimlich maneuver.[19] Approximately 30% of the air flow from transtracheal jet ventilation is directed cephalad and can therefore assist in the expulsion of airway foreign bodies.[21]

PTTJV has numerous advantages when compared to an emergent cricothyroidotomy.[1-12,17,19,21-28] It is easier and faster to perform. The technique is simpler to learn. The need for many instruments, surgical preparation and technique, and an assistant is eliminated. The complications of bleeding, glottic stenosis, subglottic stenosis, and tracheal erosion are significantly lessened. PTTJV causes less cosmetic disfigurement. PTTJV can also direct secretions and foreign bodies out of the proximal trachea.

CONTRAINDICATIONS

PTTJV is contraindicated in patients who can be orally or nasally intubated. Anterior neck trauma may be a contraindication to PTTJV. Damage to the larynx or cricoid cartilage is a contraindication to PTTJV. Catheter placement may result in laryngeal disruption if there is laryngeal trauma.[8,17] It should not be performed in patients with partial or complete transection of the trachea. Lower tracheal or proximal bronchial tree disruption can result in an increased risk of pneumothorax and pneumomediastinum with high-pressure ventilation.[8,9,17]

Complete airway obstruction is an absolute contraindication to PTTJV.[8,10,17] Exhalation requires passive recoil of the lungs and chest wall and a patent airway for outflow of gas. A patient with a complete upper airway obstruction is at an increased risk for barotrauma (e.g., pneumothorax and pneumomediastinum). Numerous studies have been performed to evaluate PTTJV with varying degrees of upper airway obstruction. Progressively increasing airway obstructions up to 80% did not cause barotrauma.[27] **Less air escapes and more volume is forced into the lungs with upper airway obstruction.** The tidal volume increases with increasing airway obstruction. Once a patient develops complete upper airway obstruction, auto-PEEP (i.e., end-expiratory alveolar pressure above the set level of positive end-expiratory pressure [PEEP]) will develop as air is trapped within the thoracic cavity with no outlet and insufflation of air under pressure continues. This will ultimately result in barotrauma and decreased mean arterial pressure.[26]

EQUIPMENT

- Pressurized oxygen source, wall source or tank at 50 psi
- Povidone iodine or chlorhexidine solution
- Commercially available PTTJV kit or a self-assembled kit from available components
- 12 to 16 gauge, 2 to 3 inch catheter-over-the-needle (i.e., angiocatheter)

- Noncompressible high-pressure oxygen tubing
- Valve device (manual push valve, Y connector, T piece)
- 10 mL syringe
- Sterile saline
- Sterile drapes
- Local anesthetic solution (e.g., 1% lidocaine)
- 3–0 nylon suture
- Needle driver
- Bag-valve device
- 3 mL syringe
- Adult endotracheal tube connector

A PTTJV system can be purchased from a commercial company (**Table 31-1**). It may also be assembled with individual parts.[1] Other alternative catheters include vessel dilators from central venous kits (5 to 7 French for younger children and 7 to 9 French for adults), a catheter introducer, or large angiocatheter.[29] Examples of PTTJV systems are shown in **Figures 31-2 through 31-8**. The components of a PTTJV system are quite simple (**Figure 31-9**). The oxygen source may be a wall supply or tank. It should have a pressure regulator that can provide 100% O_2 at 50 psi through noncompressible high-pressure tubing. Along the tubing, there must be a valve (e.g., Y connector or manual push valve) to allow intermittent flow of oxygen.[8] The flow into the trachea is regulated by manual control of this valve. Ventilations should be delivered at a rate of 12 to 20 breaths per minute. Oxygen flow rates will vary with catheter size. Flow rates for 20, 16, and 14 gauge catheters at 50 psi are 400, 500,

TABLE 31-1 Some of the Commercially Available Devices

Name	Manufacturer	Comments
AincA Manual Jet Ventilator	Anesthesia Associates	Reusable Thumb depression mechanism gives oxygen flow Connects to luer lock female connection For adults, children, and infants Adapters to directly connect to endotracheal tube or fiberoptic bronchoscope
AincA MRI Conditional 3.0 Tesla Manual Jet Ventilator	Anesthesia Associates	Reusable Similar to AincA Manual Jet Ventilator Magnetic resonance imaging compatible for ≤ 3 Tesla strength
Enk Oxygen Flow Modulator Set	Cook Medical	Disposable single-use Packaged as complete set 7.5 cm length catheter, 2.0 mm inner diameter For use when jet ventilator is not available
Manual Jet Ventilator	Instrumentation Industries	Disposable single-use outlet tube Reusable rest of system Adult (13 gauge) and children's (14 gauge) versions With or without adjustable pressure regulator
Manujet III	VBM Medizintechnik	Packaged as complete set with 13, 14, and 16 gauge catheters For adults, children, and infants with three catheters
Trans-Tracheal Catheter	Acutronic Medical System	Needle for tracheal puncture Adult (13 gauge) and children's (14 gauge) versions
Ventrain	Ventinova Medical	Available device or kit Uses suction to remove gases during expiration

FIGURE 31-2. The manual jet ventilation system (Instrumentation Industries).

FIGURE 31-3. The Manujet jet ventilation system (VBM Medizintechnik).

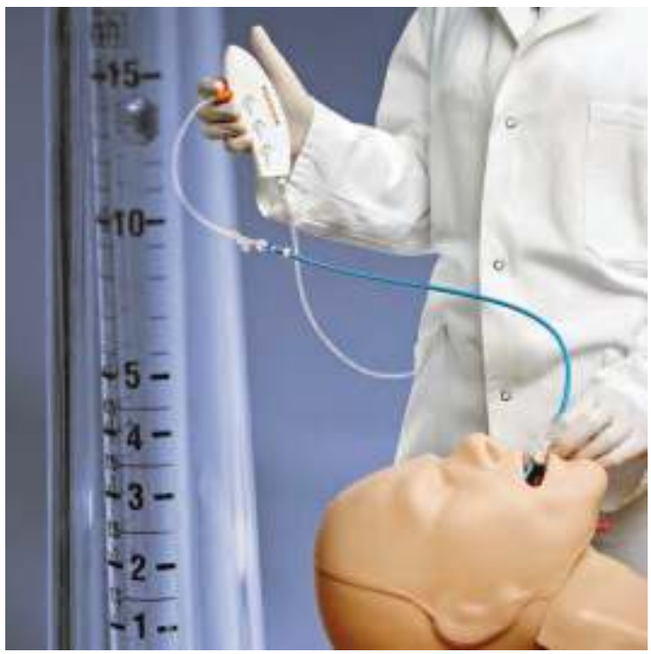
FIGURE 31-4. The Norgren jet ventilation system (Norgren Inc.).

FIGURE 31-5. The Trans-Tracheal Catheter (Acutronic Medical Systems).

FIGURE 31-6. The AincA system (Anesthesia Associates).

FIGURE 31-7. The ENK oxygen flow modular set (Cook Medical).

A



B

FIGURE 31-8. The Ventrain system (Ventinova Medical). (Photos courtesy of Ventinova Medical.)

and 1600 mL/s, respectively.[11] The inspiratory time is brief compared to the expiratory time by a ratio of 1:2 to 1:9 seconds.[2,3,22]

The patient may be temporarily oxygenated through a transtracheally placed catheter if a high-pressure system is unavailable.[30] Attach a 3 mL syringe without the plunger to the 12 to 16 gauge catheter of a catheter-over-the-needle (i.e., angiocatheter). Insert a standard endotracheal tube connector from a size 5 to 9 mm inner diameter endotracheal tube into the barrel of the syringe. Connect the bag-valve device to the connector and begin ventilation while preparing for more definitive airway control.[7,9,31,32]

The commercially available PTTJV kits are designed to provide 100% oxygen at 50 psi, which has been shown to provide both adequate oxygenation and ventilation.[10,11] Several recent studies have compared the effectiveness of a self-made apparatus with the commercial kits. They have shown that flow rates of > 15 L/min with the regulator wide open are needed to produce equivalent flow rates.[33] Using flow rates less than 15 L/min will result in failure

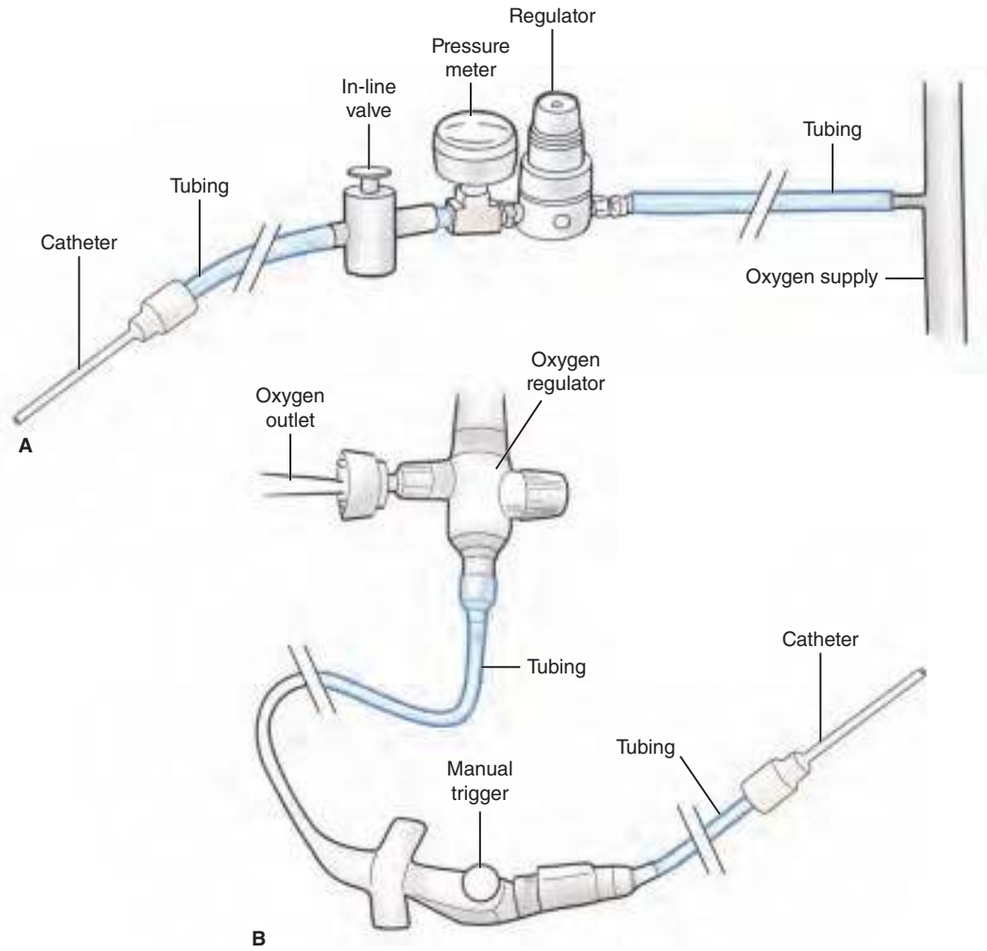


FIGURE 31-9. The components of high-pressure jet ventilation systems. ***A.*** Modified from Mittal and Baren.[28] ***B.*** Modified from Patel.[3]

of ventilation within 60 seconds. **A surgical cricothyroidotomy should be performed if a high-pressure system is unavailable and a more definitive airway cannot be obtained.**[34]

PATIENT PREPARATION

The establishment of PTTJV requires not only proper insertion of the transtracheal catheter but also proper setup of the ventilatory equipment. The required equipment should be prepackaged and placed where it is readily accessible. Ensure that the fittings are secure and the tubing is not damaged. Place the patient supine and in the sniffing position if not contraindicated. The patient is most likely already in the proper position, as this technique is most often performed on apneic patients in whom other intubation techniques have failed. Place a rolled towel behind the middle of the neck to hyperextend the neck and allow for better access. Identify by palpation the hyoid bone, thyroid cartilage, cricoid cartilage, and cricothyroid membrane. Clean the anterior neck of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the anterior neck and let it dry if there is time. Prepare the needed equipment. Attach a 12 to 16 gauge catheter-over-the-needle (i.e., angiocatheter) to a 10 mL syringe containing 5 mL of sterile saline.

TECHNIQUE

Stand at the side of the bed and adjacent to the patient's head and neck. **Reidentify the anatomic landmarks. This is crucial to perform this procedure**. Use the nondominant hand and place the thumb on one side of the thyroid cartilage and the middle finger on the other side. Use these fingers to stabilize the larynx. Use the index finger to identify the anatomic landmarks.[3,8] Start at the laryngeal prominence (i.e., Adam's apple) and work inferiorly. The soft membranous defect inferior to the laryngeal prominence is the cricothyroid membrane. Below this is the firm cartilaginous ring of the cricoid cartilage.

Insert the catheter-over-the-needle (i.e., angiocatheter) through the skin, subcutaneous tissue, and inferior aspect of the cricothyroid membrane. **The inferior aspect of the cricothyroid membrane is the preferred site as it avoids injury to the cricothyroid arteries.**[9] Direct the catheter-over-the-needle inferiorly and at a 30° to 45° angle to the upright perpendicular (**Figure 31-10*A***). Maintain constant negative pressure within the syringe as it is advanced (**Figure 31-10*B***). **Continue to advance the catheter-over-the-needle while maintaining negative pressure until air bubbles are visible in the syringe and a loss of resistance is felt.**[3,8] **These signify that the tip of the angiocatheter is within the trachea.**

Once placement within the trachea is confirmed, securely hold the needle and advance the catheter until the hub is against the skin (**Figure 31-10*C***). Remove the needle and syringe (**Figure 31-10*C***). Reattach the syringe without the needle to the catheter. **Aspirate once again to reconfirm placement of the catheter within the trachea.** The 2 to 3 cm catheter should be long enough to pass into the tracheal lumen without sitting against the posterior wall. If the

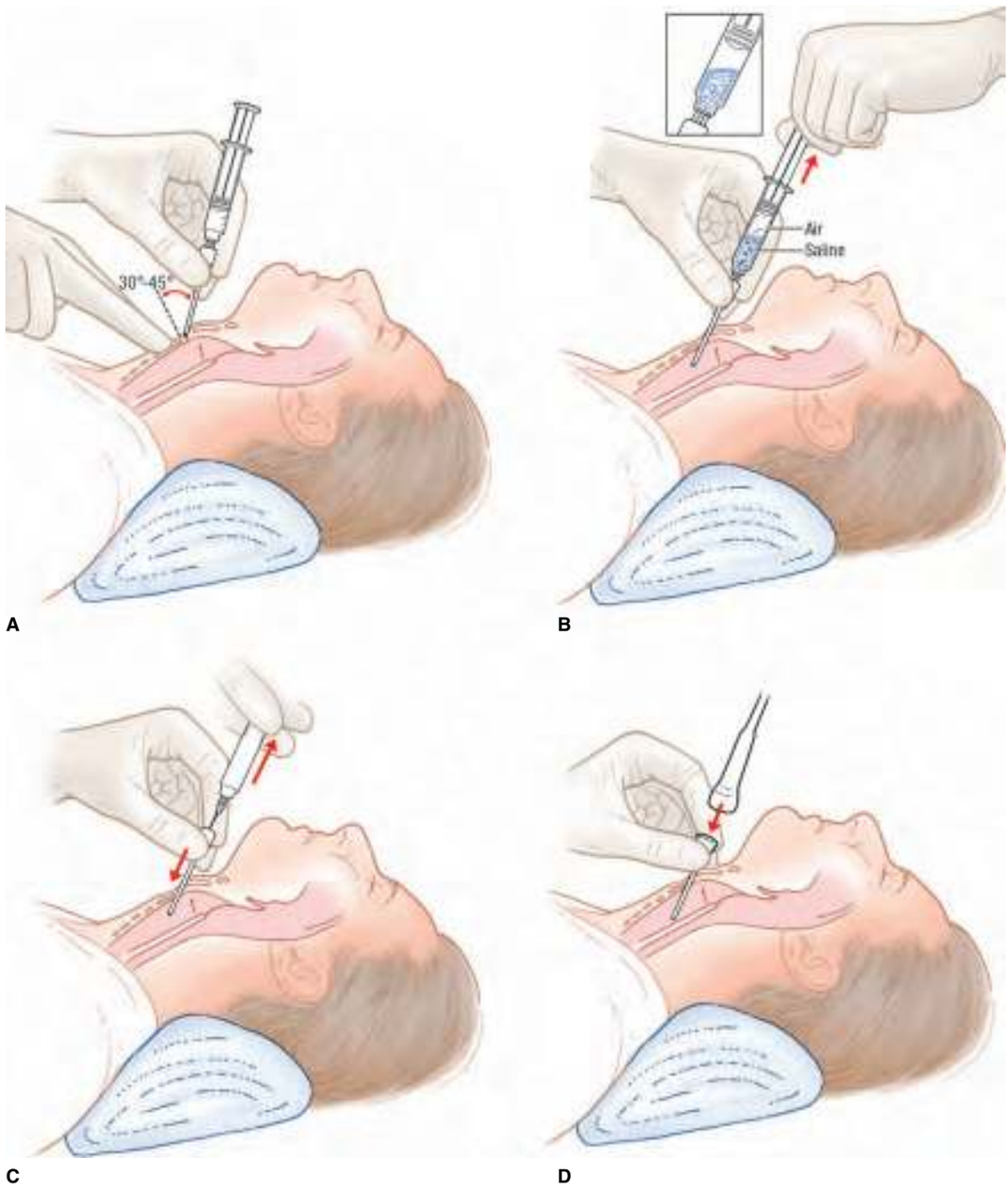


FIGURE 31-10. Insertion of the transtracheal catheter. ***A.*** The catheter-over-the-needle is inserted 30° to 45° to the perpendicular (*dotted line*) and aimed inferiorly. ***B.*** Application of negative pressure to a saline-containing syringe during catheter insertion (*arrow*). Air bubbles in the saline confirm intratracheal placement of the catheter. ***C.*** The catheter is advanced until the hub is against the skin. The needle and syringe are then removed. ***D.*** High-pressure oxygen tubing is attached to the catheter and ventilation is begun.

catheter tip directly touches or faces the posterior tracheal wall, there is the risk of forcing air submucosally.[17] **Firmly grasp and hold the catheter hub at the skin of the neck.** Remove the syringe. Attach the oxygen tubing to the catheter (**Figure 31-10*D***). Begin ventilation and continue until a more permanent and secure airway is established.[8] **Watch for adequate chest rise with ventilation and allow for complete elastic recoil of the chest wall before giving the next breath to avoid air trapping and barotrauma.**

ASSESSMENT

PTTJV requires continuous cardiac and pulse oximetry monitoring to evaluate oxygenation. Continuous capnography can be used to assure adequate ventilation. Arterial blood gas samples should be obtained periodically to look for hypoxia and hypercarbia. **Careful attention must be paid to maintaining a patent upper airway to allow for passive expiration and avoid barotrauma.** An oropharyngeal airway, nasopharyngeal airway, or a jaw-thrust maneuver is often adequate.

The catheter, oxygen tubing, and patient must be continually assessed during PTTJV. Check the catheter tubing at regular intervals for signs of dislodgement or kinking. Examine the patient for crepitus in the neck and torso. The catheter tip is most likely directly against or directed toward the mucosa of the posterior tracheal wall if there is crepitus. Oxygen is being forced into the submucosal tissues and tracking subcutaneously. Remove the catheter and reinsert a new one. Obtain a chest radiograph to assess the patient for a pneumomediastinum, pneumopericardium, or pneumothorax that

may require decompression. **Continuously monitor pulse oximetry, cardiac monitoring, and capnography.**

AFTERCARE

The catheter and tubing must be secured to prevent accidental dislodgement.[3] One person must continuously hold the hub of the catheter against the patient's skin during PTTJV while a second person secures it. There are three ways to secure the equipment. The first and preferred method is to suture it in place. Place a skin wheal of local anesthetic solution (e.g., 1% lidocaine) next to the catheter hub. Use 3–0 nylon suture to place a stitch through the skin wheal and tie it securely. Do not cut the suture. Wrap the long end of the suture around the catheter hub two or three times and tie it securely to the tail of the suture. Wrap the long end of the suture around the oxygen tubing, just above the attachment to the catheter hub, two or three times and tie it securely to the tail of the suture. Alternatively, wrap a piece of umbilical or plain tape around the patient's neck, the catheter hub, and the oxygen tubing. A second alternative is to attach a commercially available endotracheal tube holder around the patient's neck and connect it to the oxygen tubing. The catheter will still have to be secured with a suture or by being taped to the oxygen tubing and skin.

The patient should be reevaluated for possible endotracheal intubation. Studies have shown that subsequent attempts have been successful. It is possible that the positive pressure used in PTTJV increases the success rate.[3]

COMPLICATIONS

PTTJV is a relatively safe and effective means of establishing an emergent airway in a patient who cannot be intubated or ventilated by another mechanism. Complications are fewer than with a cricothyroidotomy, but they do occur and must be anticipated.

Subcutaneous emphysema occurs most commonly when the transtracheal catheter is misplaced, becomes dislodged into the soft tissues of the neck during ventilation, or is placed against the mucosa of the posterior tracheal wall.[1,7,8] It may also occur if catheter placement is unsuccessful on the first attempt, creating a port for leakage of pressurized air into the subcutaneous tissues of the neck.[8] Frequent examination of the catheter site for evidence of subcutaneous emphysema may provide the earliest clue to catheter malfunction.

Barotrauma may present as a pneumothorax, pneumomediastinum, or pneumopericardium. It may be a result of upper airway obstruction. The exhalation of air is passive and depends on a patent upper airway. **It is important to monitor chest rise and fall as evidence of continued air exchange.**[1] An oropharyngeal airway, nasopharyngeal airway, or jaw-thrust maneuver will often provide adequate upper airway patency.[8] **Assume that any sudden change in the patient's heart rate or blood pressure during PTTJV is secondary to a tension pneumothorax until proven otherwise**. There has been a case report of laryngospasm with PTTJV use during an elective surgical procedure.[23] It resulted in sudden desaturation and hypotension that were easily resolved with the administration of a paralytic agent.

Catheter obstruction is a potential complication. The catheter will most commonly kink as it traverses the soft tissues of the neck.[1,8] This may occur if the catheter is dislodged or from the high-pressure ventilation. The use of a commercially available kink-resistant catheter greatly reduces this risk.

The catheter may be inappropriately placed. Misplacement of the catheter into the submucosa of the larynx can lead to a laryngeal pneumatocele. If this is suspected or identified, remove the catheter and reinsert a new one. The pneumatocele can be aspirated with a needle and syringe after placement of a new transtracheal catheter.[8] Misplacement of the catheter posteriorly through the back of the trachea and perforation of the esophagus constitute a theoretical concern that has never been reported.[1,7,8]

Pulmonary aspiration is a potential complication of PTTJV. The epiglottis provides no airway protection. The small transtracheal catheter does not prevent aspiration of secretions or gastric contents into the lungs. Animal studies have demonstrated that pulmonary aspiration did not occur despite variable frequencies of ventilation, variable oxygen flow pressure, and cardiac compressions during cardiopulmonary resuscitation (CPR).[24,25] The pressurized flow of air through the catheter provides an adequate forceful gas outflow from the lungs, which may prevent pulmonary aspiration.[24,25] Secretions and foreign bodies have been shown to stay above the jetting catheter while PTTJV is in progress. **Great care must be taken to assure complete suctioning and cleansing of the upper airway above the catheter if PTTJV is to be discontinued.**[25]

Less serious complications include local hematoma formation at the catheter insertion site, hemoptysis, and cough.[1,7,8] The use of nonhumidified oxygen in the catheter has been reported to cause irritation and erosion of the tracheal mucosa.[1]

SUMMARY

PTTJV is an effective and easy method for establishing an emergent airway in patients who cannot be ventilated with a bag-valve device or intubated. The indications for PTTJV are the same as those for a cricothyroidotomy. Placement of a catheter through the cricothyroid membrane and attached to a high-pressure oxygen source will provide adequate oxygenation and ventilation until a more definitive airway can be established. This is an airway management technique that is rapidly performed and should be considered as a reliable backup rescue technique for the "can't intubate, can't ventilate" patient.

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32 Cricothyroidotomy

Charles Boland, Ned Nasr, and Gennadiy Voronov

INTRODUCTION

Establishment of an airway is imperative to patient survival.[1] The most predictive factor of survival from cardiac arrest is establishment of an airway.[2] The Emergency Physician is occasionally confronted with an airway that is extremely difficult or even impossible to obtain by endotracheal intubation.[3] Up to 4% of all emergent airways require a cricothyroidotomy.[4-8] Up to 7% of trauma patients who present in cardiopulmonary arrest will require a cricothyroidotomy.[6]

The technique of cricothyroidotomy has been documented in use since the early 1900s. Chevalier Jackson condemned its use in 1921 because of fears of subglottic stenosis.[9,10] Jackson's technique involved incising the cricoid cartilage, which was responsible for the resulting subglottic stenosis. The technique was popularized again in 1966 by Brantigan and Grow, but it was considered primarily an elective procedure.[9,10] **Cricothyroidotomy has since evolved into the surgical airway of choice for emergent situations in which other intubation methods have failed or are contraindicated.**[5,11] The Emergency Physician using rapid sequence induction to intubate patients must be knowledgeable and skilled in performing a cricothyroidotomy.[6] The success rate of a cricothyroidotomy ranges from 96% to 100%.[7,12]

The most difficult part of performing a cricothyroidotomy is deciding to do it. The Emergency Physician often continues to attempt intubation in hopes of gaining the airway. The decision to perform a cricothyroidotomy is often made after the patient is hypoxic and cannot be intubated, oxygenated, or ventilated. There are often multiple intubation attempts before the decision is made to perform a cricothyroidotomy. Quickly determine if intubation is unsuccessful and prevent delaying the cricothyroidotomy. Follow a difficult airway algorithm.

A cricothyroidotomy has numerous advantages over a tracheostomy.[9,13,14] A cricothyroidotomy is easier, faster, and safer to perform. It can be performed in less than 2 minutes and performed by those with little or no surgical training. It does not require the support of an Operating Room and a large amount of equipment. The anatomic landmarks are easily palpated, easily seen, and superficial. The procedure does not require a deep dissection as the structures are located superficially. The cricothyroid membrane is not covered by any structures that would interfere with the procedure. There is a minimal chance of injuring the esophagus because the cricothyroid membrane is in the anterior part of the neck. A cricothyroidotomy can be performed with the patient's neck in a neutral position rather than the extended position. This is especially important in those with potential cervical spine injuries. The procedure has fewer associated complications than a tracheostomy. The skin incision will heal with a smaller and less noticeable scar, although this is not a concern when securing an airway emergently.

ANATOMY AND PATHOPHYSIOLOGY

The cricothyroid membrane is located between the thyroid cartilage superiorly and the cricoid cartilage inferiorly (**Figure 32-1**).[15] **Identify the cricothyroid membrane by palpation of the surrounding cartilaginous structures.**[16] Use the nondominant hand. Place the thumb on one side of the thyroid cartilage and the middle finger on the other side (**Figure 32-2**). Palpate the laryngeal prominence (i.e., Adam's apple) with the index finger. **It is important to locate the hyoid bone superiorly to ensure the incision is not above the vocal cords.**[15] Just below the thyroid cartilage is a gap before reaching the cricoid cartilage. This soft tissue gap is the location of the cricothyroid membrane.[15] Ultrasound can be used to identify the cricothyroid membrane and may be more accurate than palpation.[17-21] Either perform ultrasound in advance and mark the cricothyroid membrane or use it during the procedure.

The cricothyroid membrane is a thin membrane measuring 2 to 3 cm in width and only 7.8 to 10 mm in height in the adult.[11,14] It is located approximately 1 cm below the true vocal cords.[14,15,22] **There is relatively little subcutaneous tissue and few to no vascular structures overlying the cricothyroid membrane.** The anterior

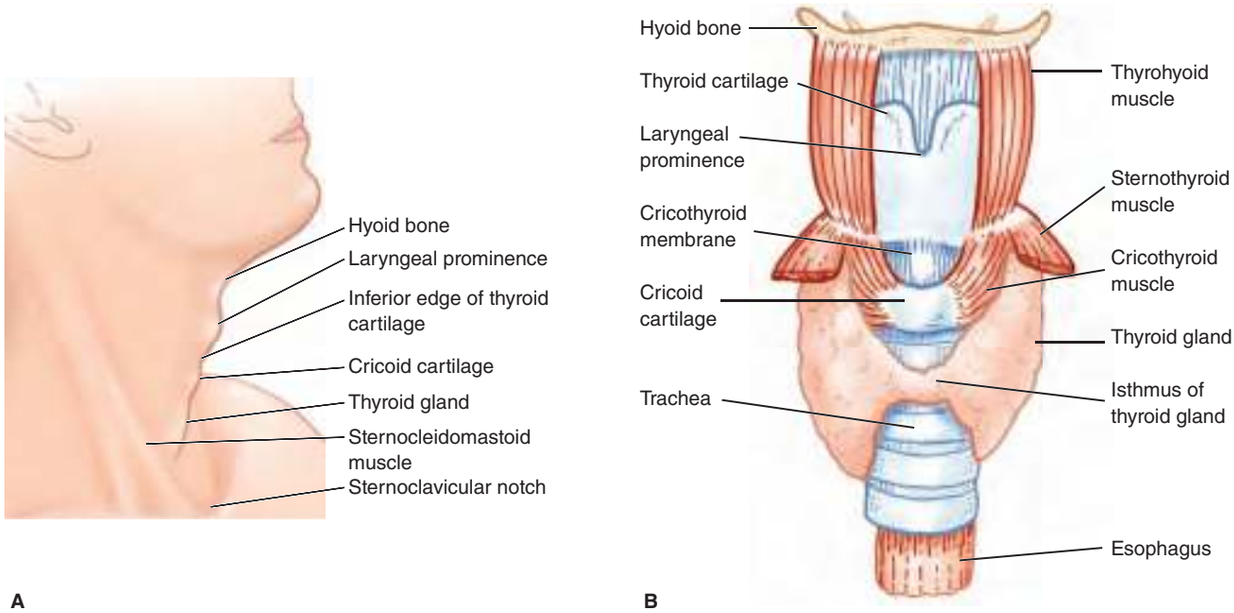


FIGURE 32-1. Anatomy of the airway in the neck region. ***A.*** Topographic anatomy. ***B.*** The framework of the airway.

cricothyroid arteries travel from lateral to medial over the superior border of the cricothyroid membrane. The anterior jugular veins may lie immediately superior and lateral to the cricothyroid membrane. Farther lateral and posterior are the great vessels of the neck. Posterior to the larynx and trachea is the esophagus. **It is important not to make the incision too deep and risk an esophageal intubation or injury.**

INDICATIONS

The most frequent indication for an emergent or urgent surgical airway is the inability to intubate orotracheally with less invasive techniques.[3] These less invasive techniques may have failed or may be contraindicated.[13] **Attempt to orotracheally or nasotracheally intubate prior to creating a surgical airway.**[7,11] Common reasons for failed orotracheal intubation include airway hemorrhage, cervical spine injuries, deformities of the mouth and/or pharynx, distortion of the normal anatomy, edema, laryngospasm, masseter spasm, obesity, severe facial trauma, severe neck trauma, upper airway hemorrhage, upper airway obstruction, and vomitus in the oropharynx.[11,12,14,23,24] **It is of the utmost importance to have the surgical airway supplies nearby if a difficult airway is suspected.** The old saying "A surgical airway is better than a deceased patient with a good-looking neck" holds quite true.

Needle cricothyroidotomy is the emergent "surgical" airway of choice in a patient younger than 8 to 10 years of age.[14,25] The cricothyroid membrane and surrounding structures are much smaller in young children and more difficult to access. It is easier to injure one of the cervical vessels or the esophagus when a standard cricothyroidotomy is performed in a child. Subglottic stenosis is a common late complication following a cricothyroidotomy in children.[25] **A needle cricothyroidotomy is a safer alternative and allows for adequate oxygenation and ventilation until a formal tracheostomy or other method of intubation can be performed.** The small caliber of the catheter does not often provide adequate oxygenation and ventilation for an adult.

FIGURE 32-2. Proper hand positioning to identify the airway structures of the neck.

CONTRAINDICATIONS

There are a few absolute contraindications to performing a cricothyroidotomy. The most important is if the patient can be intubated by less invasive methods. Partial or complete transection of the airway is a contraindication to a cricothyroidotomy. A tracheostomy is the preferred method of securing the airway in a complete airway transection. Do not perform a cricothyroidotomy with a fracture or significant injury of the cricoid cartilage, larynx, and/or thyroid cartilage.

There are some situations where the performance of a cricothyroidotomy may be less desirable. The presence of laryngeal pathology (e.g., fracture or tumor) may preclude the performance of a cricothyroidotomy and necessitate a high tracheostomy.[14,26] There is a higher incidence of long-term complications following a cricothyroidotomy if a patient has been previously intubated for a prolonged period.[26] This does not prevent the performance of a cricothyroidotomy. Consider an early revision to a tracheostomy to minimize

these complications. Other relative contraindications to performing a cricothyroidotomy are the presence of a coagulopathy, hematoma of the neck, or massive neck swelling; all of which increase the risk of bleeding and distortion of the anatomy. Unfamiliarity with the technique may lead to increased complications.[14]

EQUIPMENT

GENERAL SUPPLIES

- Sterile gloves, gowns, and drapes
- Face mask and eye protection
- 1% lidocaine local anesthetic solution
- Syringes, 5 and 10 mL
- Needles, 18 to 27 gauge
- Bag-valve-mask device
- Oxygen source and tubing
- Suction source, tubing, and catheter
- Pulse oximeter
- Noninvasive blood pressure cuff
- Cardiac monitor
- End-tidal CO_2 monitor

SURGICAL CRICOTHYROIDOTOMY

- Povidone iodine or chlorhexidine solution
- #11 scalpel blade on a handle
- Trousseau tracheal dilator or curved 6 inch hemostat
- Tracheal hook
- Hemostats, 4 small
- Needle driver
- Suture scissors
- Tracheostomy tubes, sizes #4 and #6
- Endotracheal tubes, #5 or #6
- Tracheostomy tape (i.e., twill tape)
- 3–0 sutures for hemostasis (e.g., Dexon, Vicryl, or chromic)
- 3–0 nylon sutures for skin closure
- 1 inch tape or a commercially available endotracheal tube holder
- Gauze 4×4 squares
- Iodoform gauze ribbon
- Percutaneous cricothyroidotomy kit, adult and pediatric sizes

NEEDLE CRICOTHYROIDOTOMY

- Povidone iodine or chlorhexidine solution
- 14 gauge catheter-over-the-needle (angiocatheter), 2 inches long
- 5 mL syringe
- Tape
- Percutaneous transtracheal jet ventilator (Chapter 31)

The equipment should be prepackaged in a sterile tray that is readily accessible. A minimal amount of equipment is needed to perform a cricothyroidotomy (**Figure 32-3**). Some institutions maintain separate cricothyroidotomy and tracheostomy trays. Others have one tray that contains the equipment necessary to perform both procedures. A thoracotomy tray usually contains all the required equipment to perform this procedure if a cricothyroidotomy tray is not immediately available.

A

B

FIGURE 32-3. The minimal equipment to perform a cricothyrotomy. ***A.*** A "homemade" kit (from top to bottom: an endotracheal tube, a hemostat, a scalpel, and a tracheal hook). Some kits contain a Trousseau dilator instead of the hemostat. ***B.*** The Cook Surgical Cricothyrotomy kit (top to bottom: tracheostomy tube, Trousseau dilator, scalpel, tracheal hook, syringe, tracheal ties). (Photo courtesy of Cook Medical, Bloomington, IN.)

Should the Emergency Department stock surgical cricothyroidotomy kits, percutaneous cricothyroidotomy kits, or both? The answer to this will depend on Emergency Physician preference, hospital stores, and available budget. There are many cricothyroidotomy devices commercially available (**Table 32-1**). The Melker Universal Emergency Cricothyroidotomy Catheter Set (Cook Inc., Bloomington, IN) may resolve this issue for some Emergency Departments. This single-use, disposable kit contains all the equipment required to perform both types of cricothyroidotomies (**Figure 32-4**). The convenience of having all the required equipment readily available in one kit can clearly justify the cost. Unfortunately, this kit is only available in one adult size.

PATIENT PREPARATION

A cricothyroidotomy is most commonly an emergent procedure following unsuccessful attempts at orotracheal intubation. The patient is already unconscious, so informed consent is not possible. Informed consent must be obtained if the patient is awake prior to induction and there is high suspicion of a difficult airway.

Have the nursing staff apply and monitor the electrocardiography (ECG), end-tidal CO_2, noninvasive blood pressure cuff or arterial

TABLE 32-1 Some Commercially Available Devices

Name	Manufacturer	Comments
Arndt Emergency Cricothyrotomy Catheter Set	Cook Medical	6.0 cm length 3.0 mm inner diameter Extra stiff guidewire
Emergency Transtracheal Airway Catheter	Cook Medical	6 French size 5.0 and 7.5 cm lengths For needle cricothyroidotomy Kink resistant
Melker Emergency Cricothyrotomy Catheter Set	Cook Medical	Complete set with 3.8, 4.2, and 7.5 cm lengths Corresponds to 3.5, 4.0, and 6.0 mm inner diameter Extra stiff guidewire Uses Seldinger technique
Nu-Trach Adult Emergency Cricothyrotomy Kit	Mercury Medical	Complete set with 4.5, 6.0, and 7.2 mm inner diameter airway Uses special introducer contained in set
Patil Cricothyrotomy Emergency Catheter Kit	Cook Medical	9 French catheter
Pertrach Emergency Cricothyrotomy Kit	Pulmodyne	Available in 3.9, 4.0, 4.1, 4.4, and 6.8 cm lengths Corresponds to 3.0, 3.5, 4.0, 5.0, and 5.6 mm inner diameter Splitting needle and dilator
Pedia-Trake Emergency Cricothyrotomy Kit	Mercury Medical	Complete set with 3.0, 4.0, and 5.0 mm inner diameter airway Uses special introducer contained in set
Portex Cricothyrotomy Kit (PCK)	Portex	Soft seal cuff
Quicktrach II	VBM Medizintechnik	Available in 2.0 and 4.0 mm inner diameter Conical needle tip allows smallest stoma Removable stopper prevents perforation of tracheal back wall
Rusch Quicktrach	Teleflex	Anatomic-shaped cannula Available in 2.0 and 4.0 mm inner diameter Removable stopper prevents perforation of tracheal back wall
Cric-Key	Engineered Medical Systems	Combines functions of bougie, dilator, stylet, and trach hook Hybrid surgical technique Semirigid smooth plastic introducer and cuffed 5.2 mm tube
Cric-Knife	Engineered Medical Systems	Combines a double-edge scalpel with a sliding hook Hybrid surgical technique
Cricothyrotomy Tactical Medical Model	Chinook Medical	Contains cuffed tube 6.0 mm inner diameter
H&H Emergency Cricothyrotomy Kit	H&H Medical	Contains cuffed tube 6.0 mm inner diameter
Kwik Cric Emergency Field Cricothyrotomy Kits	Boundtree Medical	Available in sizes 5.5, 6.0, and 6.5 mm inner diameter endotracheal tube
Melker Surgical Cricothyrotomy set	Cook Medical	9.0 cm length Corresponds to 5.0 mm inner diameter
Melker Universal Emergency Cricothyrotomy Catheter Set	Cook Medical	Combines contents from percutaneous and surgical kits For percutaneous and surgical cricothyroidotomy
Rusch Easycric	Teleflex	Anatomic design Available in adult size 5 Hydrophilic coating for easy insertion Uses Seldinger technique
Surgicric	VBM Medizintechnik	Available in 6.0 mm inner diameter Available in three complete sets I: rapid four-step technique II: classic surgical technique III: Seldinger technique

line, and pulse oximetry. A fully functioning suction apparatus with a variety of catheters must be immediately available.

Place the patient supine with their head in a neutral position. Place a rolled towel under the patient's upper shoulders if no contraindications exist (**Figure 32-5**). This position offers excellent exposure through mild neck extension, stabilizes and prevents the airway structures from moving, and lengthens the cricothyroid membrane. While it is not impossible to perform a cricothyroidotomy with a cervical collar on the patient, it is very difficult. It is preferable to carefully remove the collar while an assistant maintains in-line stabilization of the head and neck in a neutral position. If the patient is at risk for a cervical spine injury, have an additional assistant maintain the airway and ventilate the patient with a bag-valve-mask device.

Prep the skin of the anterior neck with povidone iodine or chlorhexidine solution and place sterile drapes to isolate a surgical field. The Emergency Physician performing the procedure should be clad in a mask, sterile gown, and sterile gloves. **The circumstances in which the procedure is usually performed often require that the airway be obtained rapidly.** A quick spray of povidone iodine or chlorohexidine and sterile gloves will suffice in an emergency.

TECHNIQUES

One or two assistants should be maintaining the airway by ventilating the patient with a bag-valve-mask device or a supraglottic airway device while a cricothyroidotomy is performed. Stand on the

FIGURE 32-4. The Melker Universal Cricothyrotomy Kit. (Photo courtesy of Cook Medical, Bloomington, IN.)

patient's right side if the Emergency Physician is right-handed.[14] The position is reversed for the left-handed Emergency Physician.

Stabilize the large thyroid cartilage in place with the thumb and middle finger of the nondominant hand (**Figure 32-2**).[11,27] **The immobilization of the larynx cannot be overemphasized. The procedure will fail and life-threatening injury can result if the larynx is not secure and the landmarks are lost. Identify the anatomic landmarks necessary to perform this procedure.[16,28] This is critical to the successful performance of a cricothyroidotomy.** Place the index finger over the laryngeal prominence (i.e., Adam's apple). Move the index finger inferiorly to identify the cricothyroid membrane, cricoid cartilage, and tracheal rings in this order. Move the index finger superiorly until it falls back into the cricothyroid membrane. Leave the index finger over the cricothyroid membrane. Infiltrate the area of the incision with local anesthetic solution after the landmarks are identified if the patient is awake and stable.

TRADITIONAL TECHNIQUE

Make a 2 to 3 cm transverse incision, centered in the midline, through the skin and subcutaneous tissue (**Figures 32-6 and 32-7*A***). Continue the incision through the cricothyroid membrane. All layers may be incised simultaneously with one incision as one gains skill with this procedure. The beginner should proceed with some caution because there is a small risk of incising through the

FIGURE 32-5. Patient positioning with a rolled towel under their neck and upper shoulders.

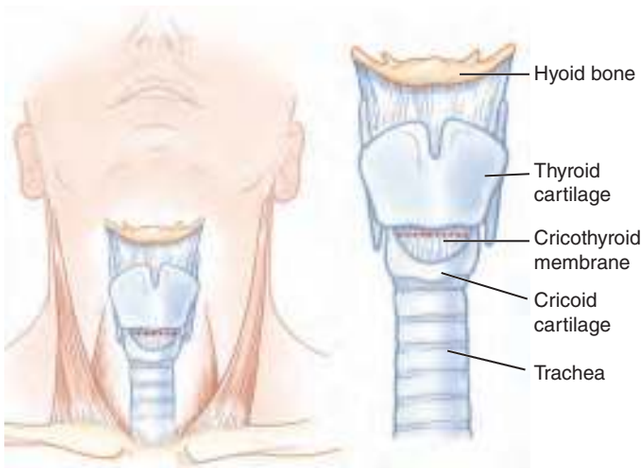


FIGURE 32-6. The cricothyroidotomy site. The dotted line represents the incision over the cricothyroid membrane.

posterior wall of the airway.[11,14] **Make the incision no longer than 3 cm or 1.5 cm on either side of the midline, as this represents the width of the cricothyroid membrane.[10,27]** Longer incisions risk injury to the anterior jugular veins that lie just lateral to the thyroid cartilage.[14]

Longitudinal incisions in the midline are not recommended. They take longer to perform and require repositioning after the skin incision. The primary indication for a longitudinal skin incision is in the patient with a suspected laryngeal injury and distortion of the anatomic landmarks or an obese patient with nonappreciable landmarks.[14,29] The longitudinal incision in these cases allows for better identification of landmarks followed by continuation of a traditional cricothyroidotomy.

A rush of air will be heard and bubbling seen from the incision once the cricothyroid membrane has been incised and the airway entered with the scalpel. This is true if the patient is ventilating spontaneously or with the assistance of a bag-valve-mask device. **Do not remove the scalpel.** Insert the tracheal hook along the scalpel until it contacts the posterior tracheal wall (**Figure 32-7*B***). Rotate the hook superiorly 90° and grasp the inferior border of the thyroid cartilage. Elevate the tracheal hook to retract the thyroid cartilage anteriorly and superiorly (**Figure 32-7*B***).[7,8] The scalpel may now be removed.

The incision site must be expanded to accommodate the passage of an endotracheal tube or a tracheostomy tube. Insert the jaws of a Trousseau dilator or 6 inch hemostat through the cricothyroid membrane in the midsagittal plane while controlling the airway with the tracheal hook (**Figures 32-7*C***). Open the jaws of the instrument to dilate the opening in the sagittal plane or vertically.[7,8,10,11] Close the jaws of the dilator. Rotate the dilator 90° within the incision. Open the jaws of the dilator to dilate the incision in the transverse plane or horizontally. Remove the dilator while continuing to maintain control of the airway with the tracheal hook.

Select a tracheostomy tube that is of an appropriate size for the patient, typically a size 6. An endotracheal tube can be used as an alternative to the tracheostomy tube. Instruct an assistant to lubricate the obturator and outer cannula. Have the assistant insert the obturator into the outer cannula. Insert the tracheostomy tube perpendicularly or 90° to the skin while controlling the airway with the tracheal hook (**Figure 32-7*D***). Continue to advance the tracheostomy tube with a semicircular motion and inferiorly until the flange is against the skin. The tracheostomy tube should pass with minimal difficulty. Remove the tracheal hook. Securely hold the outer cannula.

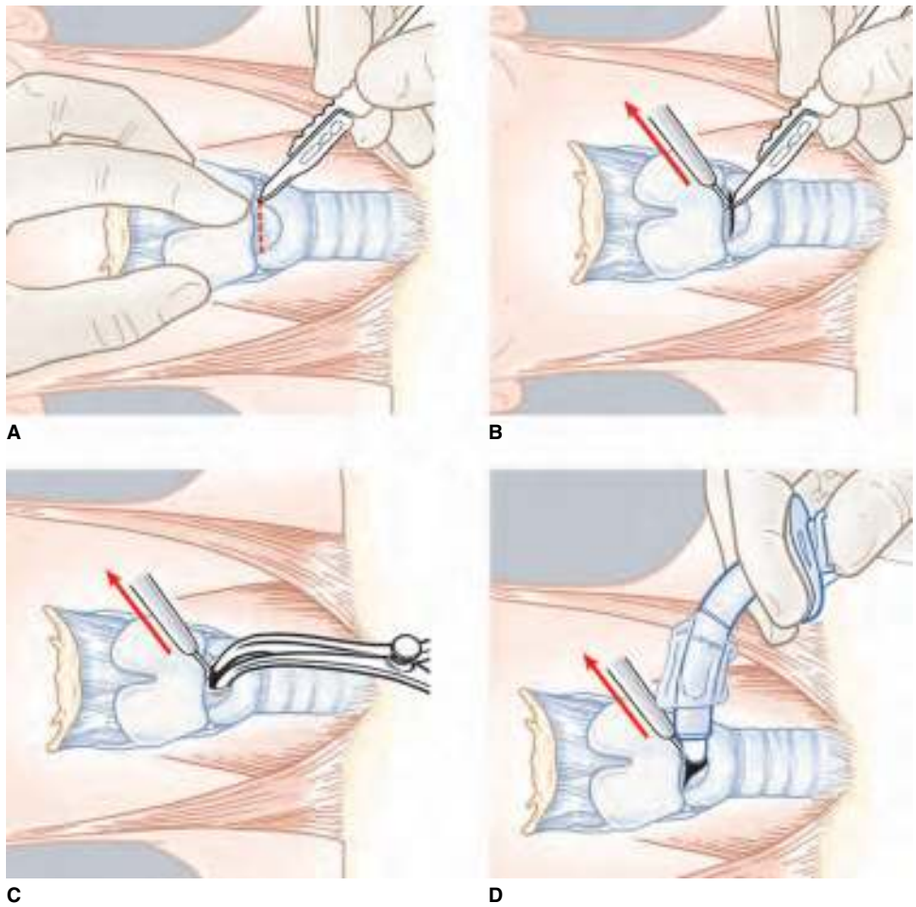


FIGURE 32-7. A surgical cricothyroidotomy. ***A.*** The nondominant hand stabilizes the cricothyroid membrane. A transverse incision is made through the skin, subcutaneous tissue, and cricothyroid membrane. ***B.*** A tracheal hook has been inserted over the scalpel blade to grasp the inferior border of the thyroid cartilage. The hook is lifted anteriorly and superiorly to control the airway (*arrow*). ***C.*** A Trousseau dilator is inserted into the incision and opened to dilate the incision site. ***D.*** A tracheostomy tube is inserted through the cricothyroid membrane.

Remove the obturator, insert the inner cannula, inflate the cuff of the tracheostomy tube, connect the bag-valve device, and ventilate the patient.[7] Confirm the intratracheal position of the tube by auscultating bilateral breath sounds, noting the absence of breath sounds over the stomach, and performing a colorimetric or quantitative end-tidal CO_2 assessment. Secure the tube.

ALTERNATIVE SURGICAL TECHNIQUE

An alternative surgical approach to a cricothyroidotomy was first developed by Oppenheimer.[30] It is simpler, more rapid, and easier to perform than the traditional technique described above (**Figures 32-8 and 32-9**). The technique has been modified from the original description.[31]

Clean, prepare, and drape the neck. Position the nondominant hand with the thumb on one side of the thyroid cartilage and the middle finger on the other side (**Figures 32-8*A* and 32-9*A***). Identify the anatomic landmarks. Leave the nondominant index finger over the cricothyroid membrane. Infiltrate local anesthetic solution subcutaneously over the cricothyroid membrane if the patient is awake.

Guide a #11 blade along the nondominant index finger and into the cricothyroid membrane making a stab incision (**Figures 32-8*A*, 32-8*B*, and 32-9*B***). **Do not insert the scalpel blade more than 1.5 to 2.0 cm to prevent it from injuring the posterior tracheal wall and esophagus.** It is recommended to hold the scalpel just above the blade with the thumb and index finger like a pencil while stabilizing your hand with the fifth digit against the patient's skin to prevent it from incising too deep.[31] Air or bubbles from the incision signify entrance into the trachea. **Do not remove the scalpel blade.** Extend the incision laterally 0.5 to 0.75 cm to extend the incision (**Figures 32-8*C* and 32-9*C***). Rotate the scalpel blade 180° and extend the incision 0.5 to 0.75 cm in the opposite direction (**Figures 32-8*D* and 32-9*D***). **Do not remove the scalpel blade. Removing the scalpel from the incision will result in losing the landmarks and the location of the incision through the cricothyroid membrane.**

With the scalpel blade in place, guide the tracheal hook down the blade and into the trachea until the posterior wall of the larynx is encountered. The blunt end of the hook will not cause damage to the tracheal mucosa. This will help confirm proper placement inside the trachea and avoid creation of a false passage. Rotate the hook superiorly 90° until the thyroid cartilage is grasped and secured (**Figures 32-8*E* and 32-9*E***). Lift the tracheal hook superiorly and then anteriorly to elevate and expose tracheal lumen (**Figures 32-8*F* and 32-9*F***). **Remove the scalpel from the incision only after the airway is controlled with the tracheal hook.**

The incision site must be expanded to accommodate the passage of an endotracheal or tracheostomy tube. Insert the jaws of a Trousseau dilator or a 6 inch hemostat through the cricothyroid membrane in the midsagittal plane while controlling the airway with the tracheal hook (**Figures 32-8*G* and 32-9*G***). Open the jaws of the instrument to dilate the opening in the sagittal plane or vertically (**Figure 32-9*H***). Close the jaws of the dilator. Rotate the dilator 90°

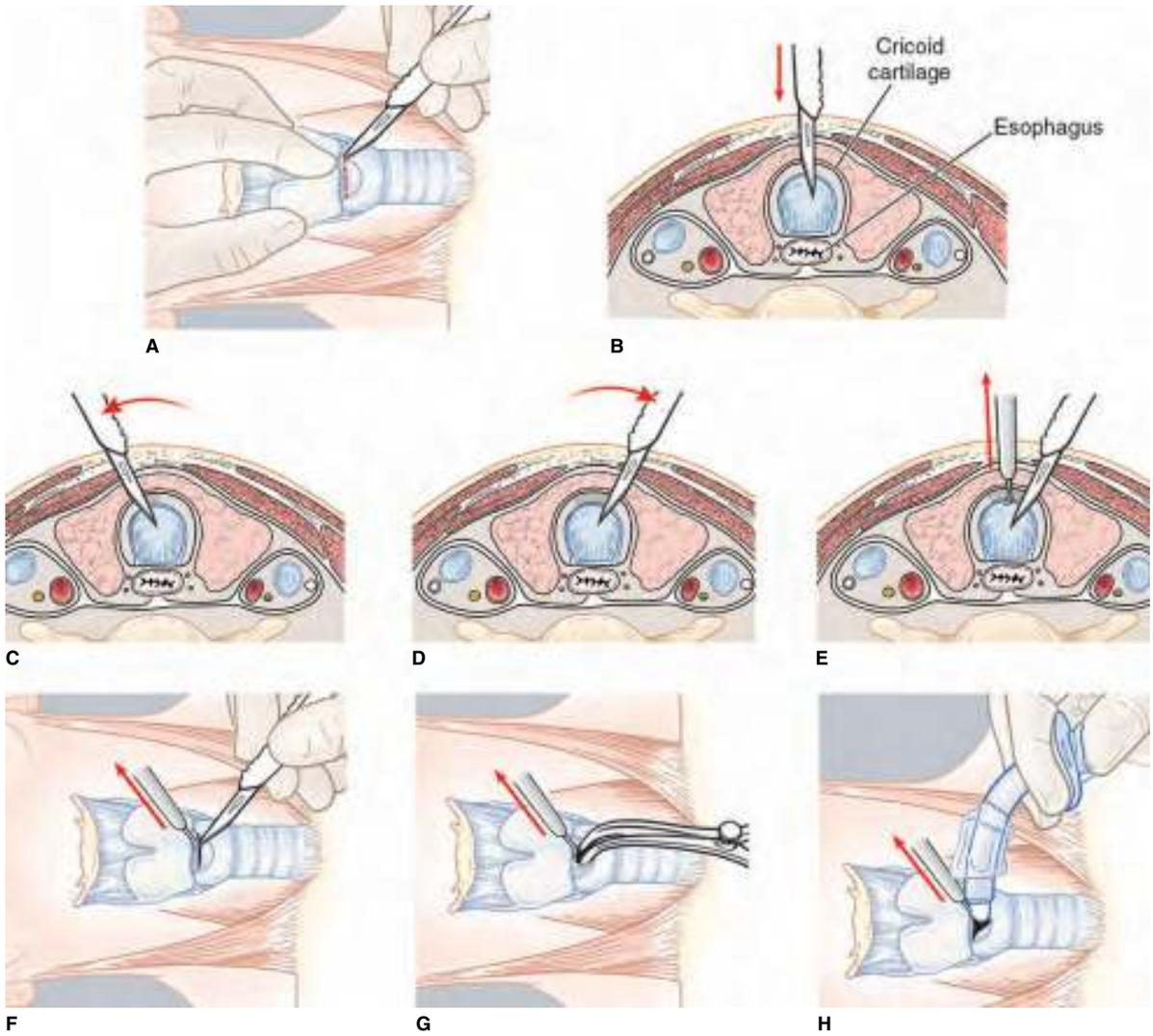


FIGURE 32-8. An alternative method to perform a surgical cricothyroidotomy. ***A.*** The thyroid cartilage is secured while a stab incision is made in the cricothyroid membrane. ***B.*** The scalpel blade penetrates the midline and enters the airway. ***C.*** The incision is extended laterally from the midline. ***D.*** The scalpel is rotated 180° and extends the incision to the other side. ***E.*** A tracheal hook is inserted in the midline and grasps the inferior border of the thyroid cartilage. ***F.*** The thyroid cartilage is lifted anteriorly and superiorly to control the airway (*arrow*). After the airway is controlled, the scalpel is removed. ***G.*** A Trousseau dilator is inserted into the incision and the jaws are opened to dilate the incision. ***H.*** A tracheostomy tube is inserted into the trachea using a semicircular motion before the tracheal hook is removed.

within the incision (**Figure 32-9*I***). Open the jaws of the dilator to dilate the incision in the transverse plane or horizontally (**Figure 32-9*J***). Insert an endotracheal or tracheostomy tube through the incision and caudally into the trachea (**Figures 32-8*H* and 32-9*K***). If using an endotracheal tube, be sure not to insert it entirely as bronchial intubation and trauma can occur.

Hold the tube securely at the skin and remove the tracheal hook. Inflate the cuff of the endotracheal tube, connect the bag-valve device, and ventilate the patient.[7] Confirm the intratracheal position of the tube by auscultating bilateral breath sounds, noting the absence of breath sounds over the stomach, and performing a colorimetric or quantitative end-tidal CO_2 assessment. Secure the tube.

PATIENTS WITH MASSIVE NECKS DUE TO SWELLING OR OBESITY

Patients may present with massive neck swelling secondary to edema, hemorrhage, hematomas, obesity, or subcutaneous emphysema after trauma.[32] These patients often have no palpable anatomic landmarks in the neck, making it difficult to create a surgical airway.[33] The traditional surgical methods used to perform a cricothyroidotomy are not practical due to difficulty in identifying the anatomic landmarks and hemorrhage. A technique has been developed to perform a cricothyroidotomy in these patients.[34-36]

The location of the hyoid bone must be determined to use this technique (**Figure 32-10*A***). A piece of suture, string, or tracheal tie is required. Place one end of the suture at the angle of the patient's mandible. Run the suture along the mandible and note where it contacts the tip of the chin (**Figure 32-10*A***, line 1). Cut the suture at the point where it contacts the tip of the chin. Fold the suture in half. Place one end of the folded suture on the tip of the chin. Pull the other end of the folded suture tight to make a 90° angle to line 1 (**Figure 32-10*A***, line 2). Draw an imaginary line from the free end of the folded suture to the angle of the patient's mandible (**Figure 32-10*A***, line 3). This third line is the line used to identify the hyoid bone.

Insert a #11 scalpel blade through the midline of the neck in an upward and posterior direction along line 3 (**Figure 32-10*B***).

Advance the scalpel blade until it meets resistance when it contacts the hyoid bone. Alternatively, a spinal needle can be inserted along line 3 until it contacts the hyoid bone (**Figure 32-10*C***). Insert the #11 scalpel along the track of the spinal needle until the hyoid bone is contacted. **Do not remove the scalpel.** Insert a tracheal hook along the scalpel blade until the hyoid bone is contacted. Move the tip of the tracheal hook under the hyoid bone (**Figure 32-10*D***). Lift the tracheal hook anteriorly and superiorly to elevate and control

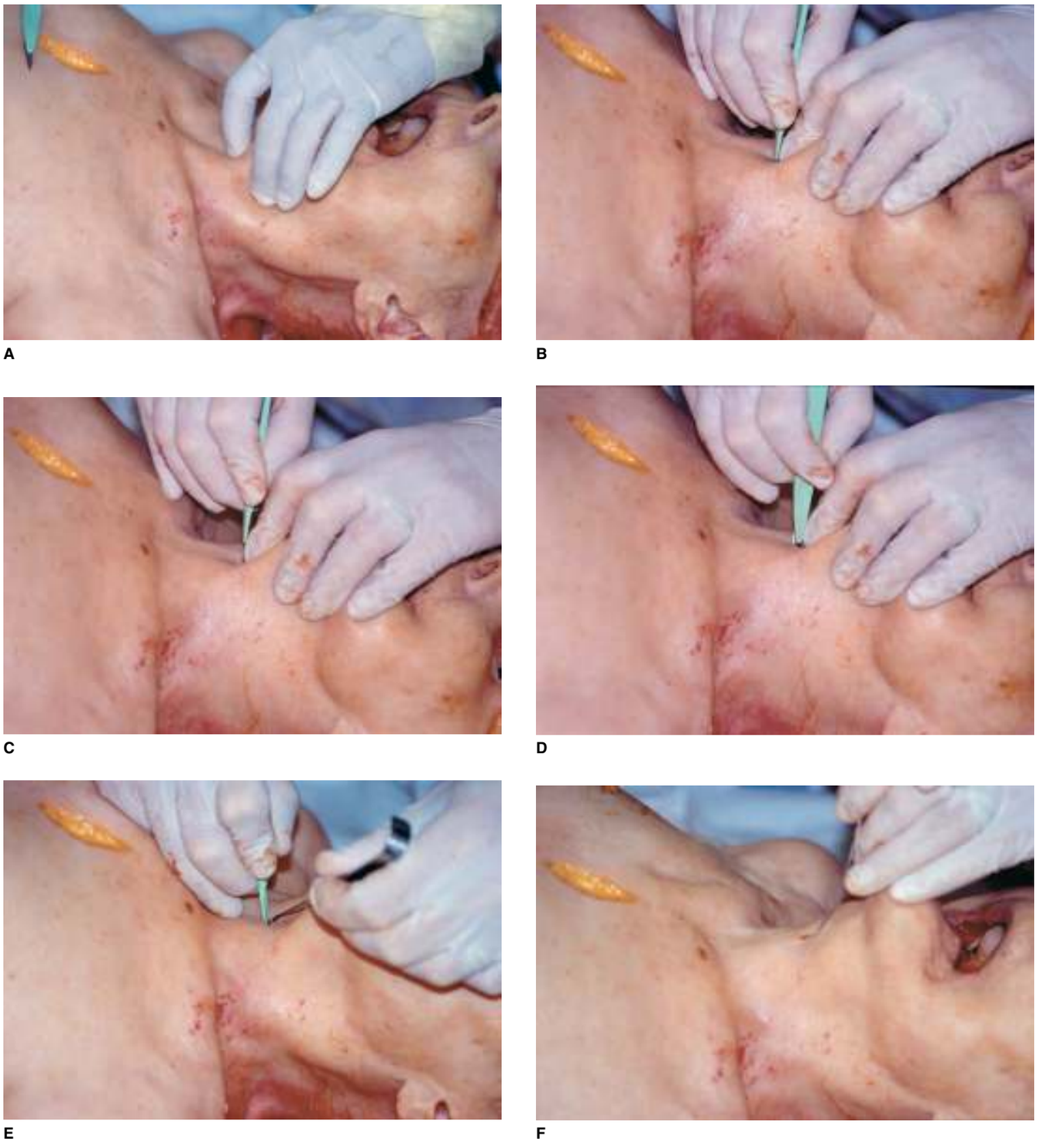


FIGURE 32-9. The alternative method performed on a cadaver. ***A.*** The larynx is stabilized and the Emergency Physician's index finger overlies the cricothyroid membrane. ***B.*** A stab incision is made into the cricothyroid membrane using the finger as a guide. ***C.*** The incision is extended toward the Emergency Physician. ***D.*** The scalpel is rotated 180° and the incision is extended away from the Emergency Physician. ***E.*** A tracheal hook is inserted over the scalpel blade to grasp the inferior border of the thyroid cartilage. ***F.*** The tracheal hook is lifted upward and superiorly to control the airway. The scalpel has then been removed. ***G.*** The Trousseau dilator is inserted into the cricothyroid membrane. ***H.*** The jaws of the dilator are opened in the sagittal plane to widen the incision. ***I.*** The dilator is rotated 90°. ***J.*** The jaws of the dilator are opened to open the incision transversely. ***K.*** An endotracheal tube is inserted through the incision and into the trachea.

G

H

I

J

K

FIGURE 32-9. *(Continued)*

the airway (**Figure 32-10*D***). **Do not release the hold of the tracheal hook on the hyoid bone.** Remove the scalpel from the incision.

Make an incision inferiorly and in the midline starting at the site where the tracheal hook exits the skin. **Extend the incision directly inferiorly without regard to the anatomy of the neck. Do not release the tension on the tracheal hook.** Identify the cricothyroid membrane. Make a transverse incision through the cricothyroid membrane. Dilate the opening and insert a tracheostomy tube or endotracheal tube into the trachea as described previously.

SELDINGER TECHNIQUE

A percutaneous cricothyroidotomy kit is available from several manufacturers. One of the more commonly used kits is the Melker Percutaneous Cricothyrotomy Set (Cook Inc., Bloomington, IN). It is a self-contained kit that may be used in the prehospital setting, Emergency Department, or Operating Room. It contains percutaneous needles, a catheter-over-the-needle, a syringe, a #15 scalpel blade, percutaneous airway catheters, dilators that fit inside the

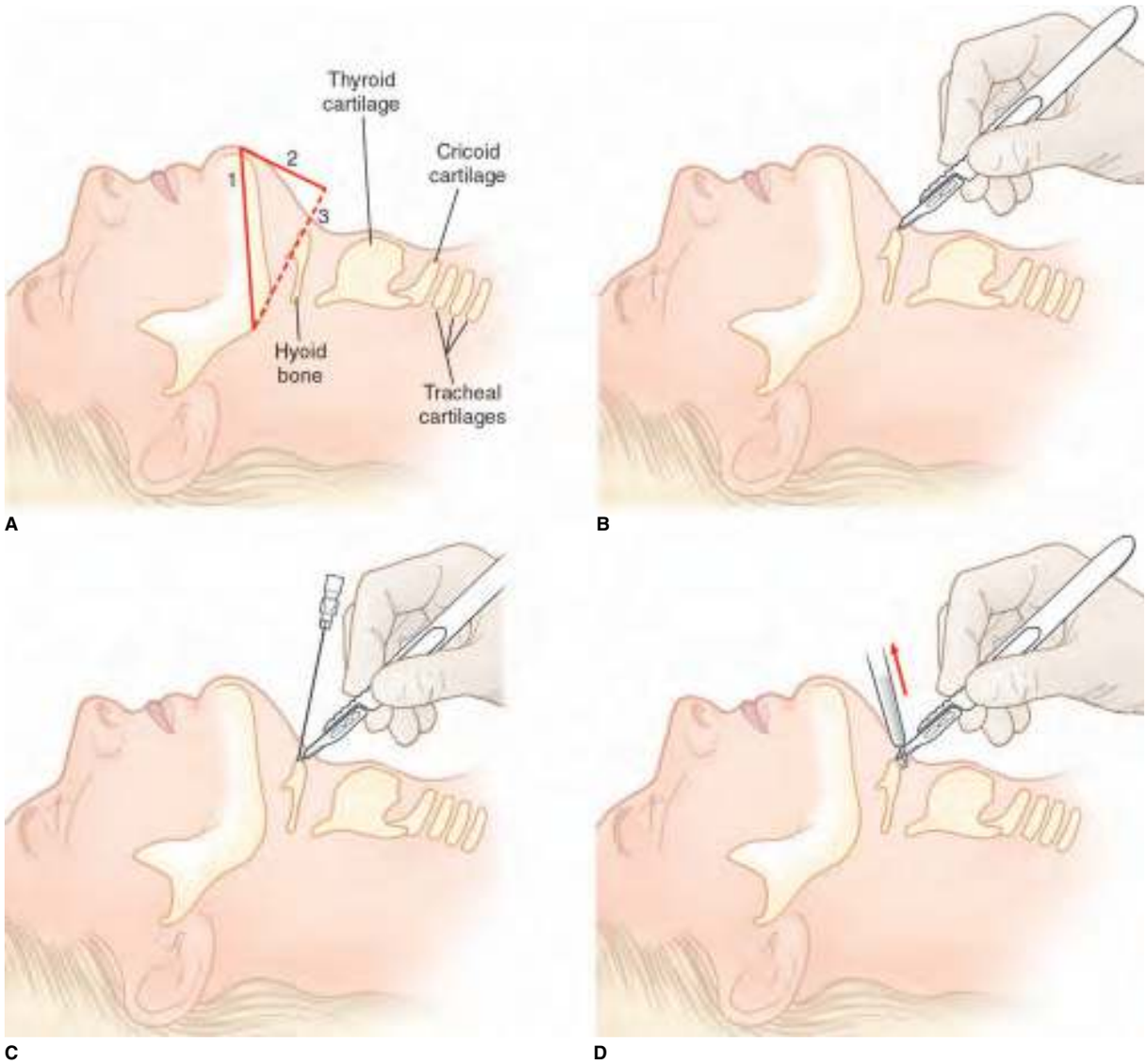


FIGURE 32-10. Cricothyroidotomy in a patient with neck swelling. ***A.*** Locate the hyoid bone. Line 1 is from the angle of the mandible to the tip of the chin. Line 2 is half the length of line 1 and perpendicular to it. Line 3 is from the end of line 2 to the angle of the patient's mandible. ***B.*** A #11 scalpel blade is inserted in the midline and aimed along line 3 until it contacts the hyoid bone. ***C.*** An alternative method. A spinal needle is used to locate the hyoid bone. A #11 scalpel blade is inserted along the tract of the spinal needle until the hyoid bone is contacted. ***D.*** A tracheal hook is inserted along the scalpel blade and used to grasp the hyoid bone. The tracheal hook is lifted (*arrow*) anteriorly and superiorly to elevate and control the airway.

airway catheters, a 30 cm flexible guidewire, and a tracheal tie (**Figure 32-11**). The dilator was developed to fit inside the airway catheter (**Figure 32-12**). The dilator and airway catheter are inserted as a unit during the procedure.

The percutaneous cricothyroidotomy kit can be used to establish an airway using a modification of the Seldinger technique.[14,37,38] This technique can be used to establish an airway in approximately the time it takes to create a surgical cricothyroidotomy.[37,38] A percutaneous cricothyroidotomy is a simpler and quicker technique for those with little surgical experience with which to establish an airway other than traditional surgical methods. The technique is similar to inserting a central venous line (Chapter 63).[39] This technique is familiar and preferred by Emergency Physicians over a surgical cricothyroidotomy.[40]

Clean, prep, drape, and anesthetize the patient's neck. Lubricate the dilator liberally and insert it through the airway catheter (**Figure 32-12**). Lubricate the airway catheter and dilator after it has been assembled into a unit. Stabilize the trachea with the nondominant hand and identify the landmarks as previously described. Leave the nondominant index finger over the center of the cricothyroid membrane.

Make a stab incision just through the skin over the center of the cricothyroid membrane with the #11 scalpel blade (**Figure 32-13*A***). Insert the catheter-over-the-needle attached to a 5 mL syringe containing saline through the skin incision aiming inferiorly (**Figure 32-13*B***). Insert and advance the catheter-over-the-needle at a 30° to 45° angle to the skin (**Figure 32-13*B***). Advance the catheter-over-the-needle while simultaneously aspirating with the syringe. Stop advancing the catheter-over-the-needle when the airway has been entered. This will be signified by a loss of resistance and air bubbles in the syringe (**Figure 32-13*B***).

Hold the syringe securely and advance the catheter over the needle until the hub is at the skin of the neck. Hold the catheter hub securely against the skin of the neck and remove the needle and syringe. Insert and advance the guidewire through the catheter and into the trachea (**Figure 32-13*C***). Grasp the guidewire securely

FIGURE 32-11. The contents of a percutaneous cricothyroidotomy kit. (Photo courtesy of Cook Medical, Bloomington, IN.)

FIGURE 32-12. The dilator is placed inside the airway catheter to form a unit.

and remove the catheter over the guidewire (**Figure 32-13*D***). **Do not release the hold on the guidewire to prevent it from completely entering the patient's airway.**

Insert the dilator/airway catheter unit over the guidewire and into the trachea in a semicircular motion (**Figure 32-13*E***). The tip of the dilator is rigid. **Insert it gently to prevent injury to or perforation of the posterior tracheal wall by holding it 1 to 2 cm from the skin.** Continue to advance the unit until the flange is against the skin of the neck. Hold the airway catheter securely. Remove the guidewire and dilator as a unit, leaving the airway catheter in place (**Figure 32-13*F***). Begin ventilation of the patient and secure the airway catheter.

NEEDLE CRICOTHYROIDOTOMY

Perform a needle cricothyroidotomy instead of a surgical cricothyroidotomy in children less than 8 to 10 years of age. A surgical cricothyroidotomy is technically more difficult. The child has a laryngeal prominence that is difficult to palpate as it is not well developed. The cricothyroid membrane is small and often will not allow the passage of an airway tube. The larynx is anatomically positioned relatively higher than in an adult and is more difficult to access. A commercially available kit (ENK Oxygen Flow Modulator Set, Cook Medical, Bloomington, IN) (**Figure 32-14**), a reinforced large-bore catheter (Emergency Transtracheal Airway Catheter, Cook Medical, Bloomington, IN) (**Figure 32-15**), or commonly available equipment in the Emergency Department may be used to perform this procedure.

Stand at the side of the bed and adjacent to the patient's head and neck.[14] **Reidentify the anatomic landmarks. This is crucial to performing this procedure.** Use the nondominant hand and place the

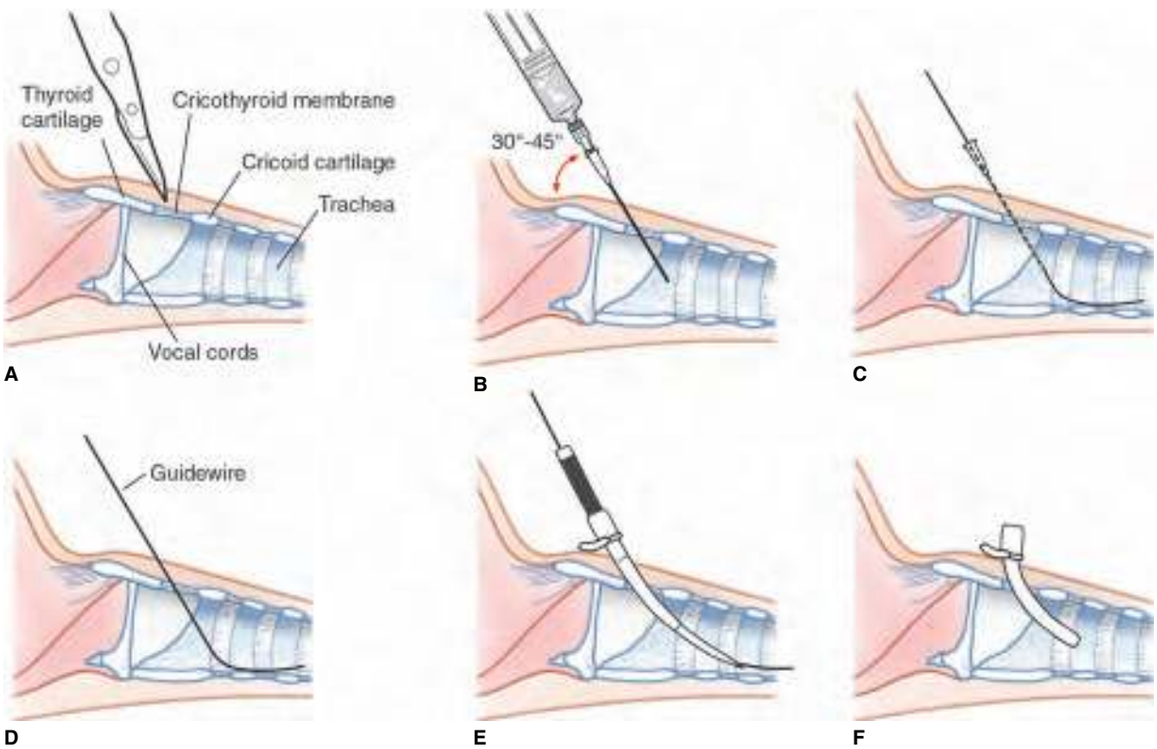


FIGURE 32-13. The percutaneous cricothyroidotomy. ***A.*** A stab incision is made in the midline over the cricothyroid membrane. ***B.*** A catheter-over-the-needle is inserted at a 30° to 45° angle to the skin and advanced inferiorly. Negative pressure is applied to a saline-containing syringe during catheter insertion. Air bubbles in the saline confirm intratracheal placement of the catheter. ***C.*** The catheter has been advanced until the hub is against the skin. The needle and syringe have been removed. A guidewire is inserted through the catheter. ***D.*** The catheter has been removed, leaving the guidewire in place. ***E.*** The dilator/airway catheter unit is advanced over the guidewire and into the trachea. ***F.*** The guidewire and dilator have been removed, leaving the airway catheter in place.

FIGURE 32-14. The ENK Oxygen Flow Modulator. (Photo courtesy of Cook Medical, Bloomington, IN.)

thumb on one side of the thyroid cartilage and the middle finger on the other side. Use these fingers to stabilize the larynx.[11,27] Use the index finger to identify the anatomic landmarks.[7,8,14] Start at the laryngeal prominence (i.e., Adam's apple) and move inferiorly. The soft membranous defect inferior to the laryngeal prominence is the cricothyroid membrane. Below this is the cartilaginous ring of the cricoid cartilage.

Attach a 12 to 16 gauge catheter-over-the-needle (i.e., angiocatheter) onto a 5 or 10 mL syringe containing 5 mL of sterile saline. Insert the catheter-over-the-needle through the skin, subcutaneous tissue, and inferior aspect of the cricothyroid membrane. The lower left quadrant of the cricothyroid membrane is the preferred site, as this region is least likely to contain cricothyroid arteries and veins.[41] Direct the catheter-over-the-needle inferiorly and at a 30° to 45° angle (**Figure 32-16*A***). Aspirate with the syringe as it is advanced (**Figure 32-16*B***). Continue to advance the catheter-over-the-needle while maintaining negative pressure until air bubbles are visible in the syringe and a loss of resistance is felt. These both signify that the catheter-over-the-needle is within the trachea.

The catheter-over-the-needle is within the trachea. Securely hold the needle and advance the catheter until the hub is against the skin (**Figure 32-16*C***). Remove the needle and syringe (**Figure 32-16*C***). Reattach the syringe without the needle to the catheter. Aspirate once again to reconfirm placement of the catheter within the trachea. The 2 to 3 cm catheter is long enough to pass into the trachea without sitting against the posterior wall. **There is the risk of forcing air submucosally if the catheter tip directly touches or faces the posterior tracheal wall.** Grasp and hold the catheter hub firmly at the skin of the neck. Remove the syringe. Attach the oxygen tubing to the catheter (**Figure 32-16*D***). Begin ventilation and continue until a more permanent and secure airway is established.

FIGURE 32-15. The Emergency Transtracheal Airway Catheter. (Photo courtesy of Cook Medical, Bloomington, IN.)

The patient may be oxygenated and ventilated by several methods. The first involves inserting the adapter piece from a #3.0 endotracheal tube to the catheter hub and then connecting it directly to the bag-valve device or a ventilator. This method allows for the confirmation of breath sounds and provides better ventilation of the patient. The second method involves direct connection of the high-flow oxygen tubing to the hub of the catheter. This method requires cyclic ventilation for 1 to 2 seconds followed by exhalation for 4 to 5 seconds.[25] This method provides adequate oxygenation, less adequate ventilation, and is more labor-intensive.

The Ventrain (Dolphys Medical, Eindhoven, The Netherlands) was developed to attach to the Luer lock of its proprietary catheter or other catheter (**Figure 32-17**). The other end attaches to a high-flow oxygen source. The single-use device provides a form of jet ventilation. The minute ventilation depends on the flow rate of the oxygen, the properties of the lungs, and the airways being ventilated. It was designed for difficult or obstructed airways. The Ventrain reduces the risk of intrapulmonary pressure building up and air trapping using expiratory ventilation assistance (EVA). It uses suction during expiration to actively remove gas from the lungs. The device can also be attached to an airway exchange catheter or a bronchoscope. An additional side connector connects to an end-tidal CO_2 monitor.

Confirm the presence of breath sounds with ventilations. Secure the catheter to the skin. This may be done with nylon sutures or strips of adhesive tape. **The patient should undergo orotracheal intubation or a formal tracheostomy as soon as possible because of the risk of dislodging the catheter and the suboptimal ventilation associated with this technique.**

FOUR-STEP TECHNIQUE

A rapid four-step technique (RFST) is preferred by some Emergency Physicians. The steps include the identification of landmarks, making the horizontal incision, placing the tracheal hook, and inserting the tube.[42-45] There is a theoretical risk of increased complications using this technique.[42-45] This has not been the case in humans.[4] The advantages are the short time to perform the procedure, the requirement of only one person, the elevation of the cricoid cartilage with an easier route to insert the tube, and the performance from the head of the bed. It does not work well in obese patients and patients with swelling.

ALTERNATIVE TECHNIQUES

An endotracheal tube can be used if a tracheostomy tube is not available. The endotracheal tube is much longer than is needed. Remove the 15 mm connector from the proximal end of the endotracheal tube. Cut the endotracheal tube with scissors just above where the cuff port tubing enters the endotracheal tube. Place the 15 mm connector on the shortened endotracheal tube. This eliminates some dead space and makes the endotracheal tube easier to manage. The procedure to insert an endotracheal tube is the same as that for a tracheostomy tube.

A cricothyroidotomy is a seldom performed procedure. A tracheal hook may not be immediately available or found when one is required (**Figure 32-18*A***). A simple alternative is to make one using a 16 or 18 gauge needle, a 10 mL syringe, and a hemostat or needle driver (**Figure 32-18*B***). Securely apply the needle onto the syringe. Use the hemostat or needle driver to place two 90° bends in the distal needle (**Figure 32-18*B***). The syringe acts as a handle and the bent needle as a tracheal hook.

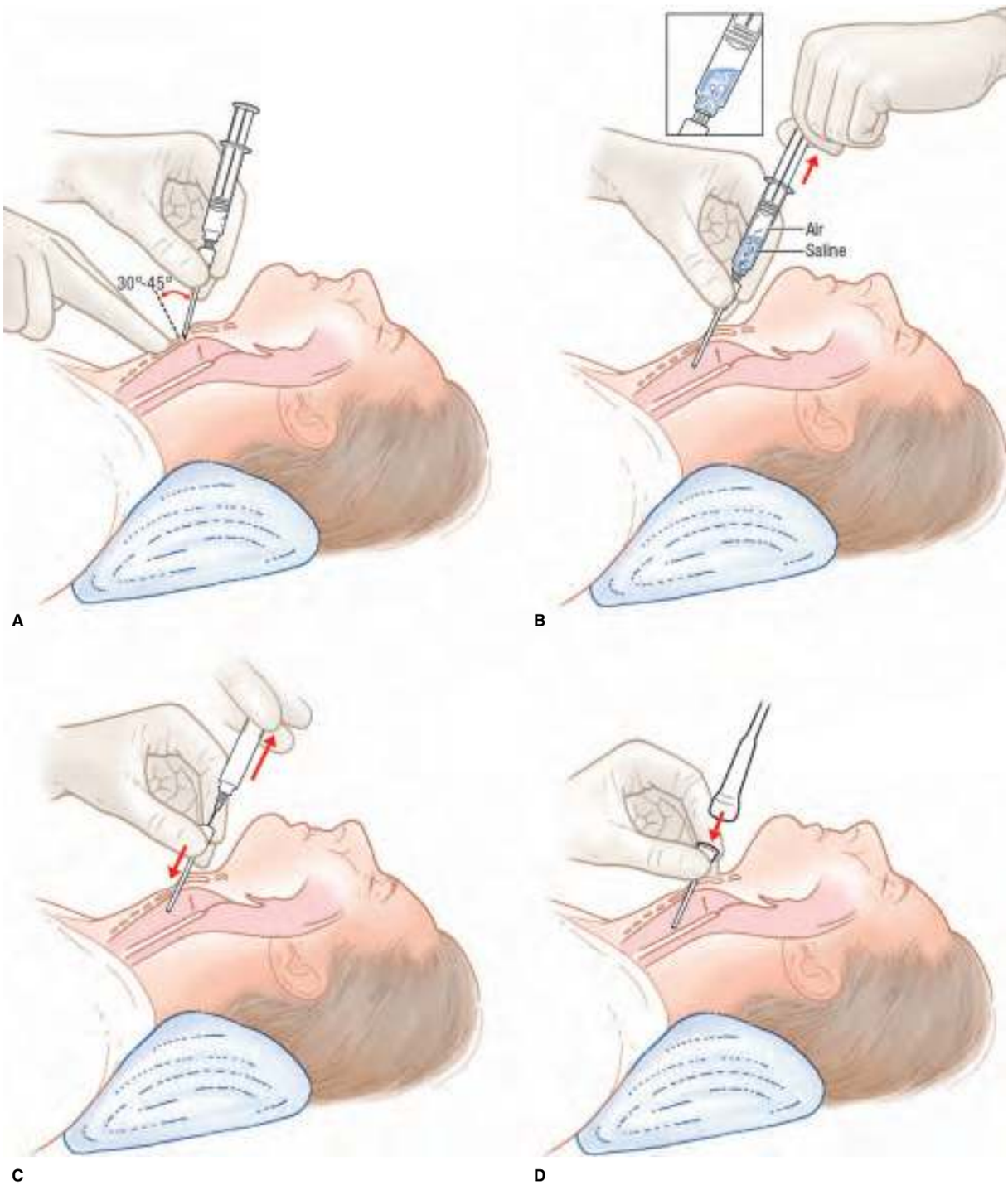


FIGURE 32-16. Insertion of the transtracheal catheter. ***A.*** The catheter-over-the-needle is inserted 30° to 45° to the perpendicular (*dotted line*) and aimed inferiorly. ***B.*** Application of negative pressure (*arrow*) to a saline-containing syringe during catheter-over-the-needle insertion. Air bubbles in the saline confirm intratracheal placement of the catheter. ***C.*** The catheter is advanced until the hub is against the skin. The needle and syringe are then removed. ***D.*** High-pressure oxygen tubing is attached to the catheter, and ventilation is begun.

Numerous other cricothyroidotomy kits are available (**Table 32-1**). The QuickTrach (Rusch Inc. of Teleflex Medical, Research Triangle Park, NC) is a preassembled, one-piece device (**Figure 32-19**). The SurgiCric Set (VBM Medizintechnik GmbH, Germany) is preassembled but requires the use of an obturator (**Figure 32-20**). The Control-Cric (Pulmodyne, Indianapolis, IN) is a surgical device (**Figure 32-21**).[46] It consists of the dual-sided Cric-Knife and the Cric-Key with a stylet and cuffed airway. These devices are quick and simple to use and do not require the use of a guidewire or the Seldinger technique. These are more likely to be used in the prehospital setting than in the Emergency Department.

The use of a bougie or endotracheal tube introducer may aid the performance of a surgical cricothyroidotomy (**Figure 32-22**).[47] Begin the procedure as described previously. Once the airway is controlled with the tracheal hook, insert a bougie or endotracheal tube introducer inferiorly with its beveled end through the incision

FIGURE 32-17. The Ventrain. (Photo courtesy of Dolphys Medical, Eindhoven, The Netherlands.)

and into the trachea (**Figure 32-22*A***). The bougie or endotracheal tube introducer will provide tactile feedback as it crosses the tracheal rings. **It is very important to confirm placement into the tracheal lumen as creating a false lumen or passage into subcutaneous tissue can be devastating.** Advance the tracheostomy or endotracheal tube over the bougie or endotracheal tube introducer and into the trachea (**Figure 32-22*B***).

A major advantage of bougies and endotracheal tube introducers is their length. They will not pull out and will ensure that the tract is not lost. It may be more difficult to "railroad" a tracheostomy tube over a bougie because of the tracheostomy tube's fixed 90° angle. This method will most likely be easier with an endotracheal tube.[29]

Several quick assembly and low-cost needle cricothyroidotomy setups can be put together quickly with commonly available equipment in the Emergency Department.[48] Insert a catheter-over-the-needle into the patient's neck as previously described. Securely hold the hub of the catheter against the skin. Remove the needle and leave the angiocatheter through the skin and into the airway. Attach and connect one of the following to the hub of the catheter. The first consists of a 3.5 mm endotracheal tube respiratory adapter and a bag-valve device. The second consists of a 10 mL syringe without the plunger, a 7.0 mm endotracheal tube inserted into the syringe, and a bag-valve device. The third consists of a 3 mL syringe without the plunger, a 7.0 mm endotracheal tube respiratory adapter, and a bag-valve device. The fourth consists of the cut distal 10 cm of intravenous infusion tubing, a 2.5 mm endotracheal tube respiratory adapter, and a bag-valve device.

A

B

FIGURE 32-18. The tracheal hook. ***A.*** Commercial unit. ***B.*** "Homemade" tracheal hook.

FIGURE 32-19. The Rusch QuickTrach. (Photo courtesy of Teleflex Medical, Research Triangle Park, NC.)

ASSESSMENT

Confirm the proper positioning of the tube (Chapter 19). Use auscultation of bilateral breath sounds, chest rise, and end-tidal CO_2 monitoring. Obtain a chest radiograph to confirm the position of the tube and rule out the presence of a pneumothorax.[25]

AFTERCARE

Hold the tracheostomy in place firmly while the patient is ventilated with a bag-valve device or a ventilator.[14] **Do not release the hold on the tracheostomy tube until it is secured.** Obtain hemostasis of the wound edges by grasping any bleeding vessels with a hemostat and placing an absorbable 3–0 suture over the vessel. Pack the wound with iodoform gauze if the skin incision is significantly larger than the tracheostomy tube. Secure the tracheostomy tube by placing

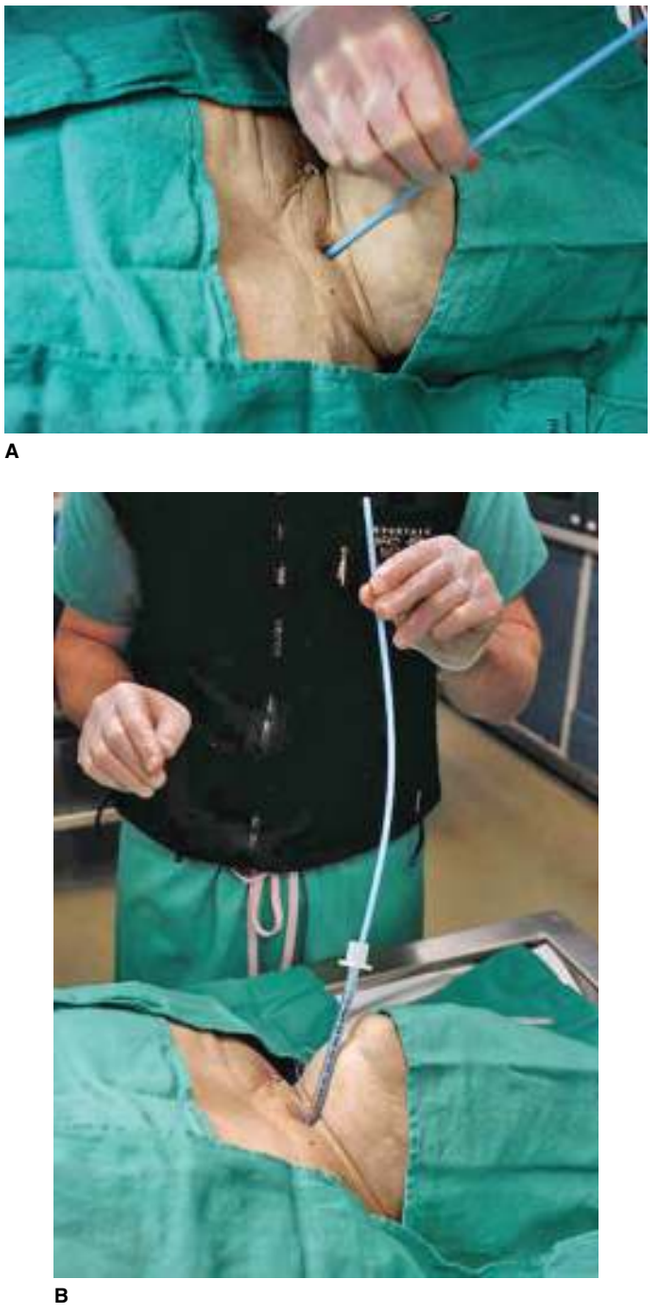
FIGURE 32-20. The SurgiCric. (Photo courtesy of VBM Medizintechnik GmbH, Germany.)

FIGURE 32-21. The Cric-Knife (*left*) and the Cric-Key (*right*). (Photo courtesy of Pulmodyne, Indianapolis, IN.)

twill tape (i.e., tracheostomy tape) through one flange, around the patient's neck, and through the other flange.[14,25] An alternative is to suture the four corners of the tracheostomy flanges to the patient's skin using a 3–0 nylon suture.

Secure an endotracheal tube if it was inserted instead of a tracheostomy tube. Wrap tape around the endotracheal tube as it exits the incision site. Wrap the tape around the patient's neck and back onto the endotracheal tube. Alternatively, a commercially available endotracheal tube holder can be used to secure the tube.

The tracheostomy or endotracheal tube can be removed easily when the indication for airway control no longer exists. This usually occurs after the patient is orotracheally intubated or nasotracheally intubated or has a formal tracheostomy performed. Place an occlusive dressing (e.g., petroleum gauze) over the incision after removal of the tube and until it heals. It is advised that the cricothyroidotomy be converted to a formal tracheostomy to minimize the risk of long-term complications if the patient is predicted to require ventilatory management for more than 7 days.



A

B

FIGURE 32-22. Using a bougie for a cricothyroidotomy. ***A.*** The bougie is inserted into the trachea. ***B.*** An endotracheal tube has been placed over the bougie and advanced into the trachea. (Photo used with permission from reference 47.)

COMPLICATIONS

Complications following a cricothyroidotomy can be classified as early or late based on when they occur. Early complications will be recognized either immediately after the performance or within a few hours. Late complications may not be apparent for weeks to months following the procedure. There are numerous complications associated with performing a cricothyroidotomy (**Table 32-2**).[5,7,9,11,12,14,26,27,49,50] These are often due to performing a cricothyroidotomy under stressful conditions.

The most serious early complication is malposition of the tracheostomy tube within the soft tissues of the neck. The patient cannot be ventilated or oxygenated if the tube is not within the trachea. This is easily recognized by auscultating the chest. It should be noticed immediately by resistance on manual ventilation with a bag-valve-mask device. It can be remedied by removing the tracheostomy tube and replacing it in its proper location. The tube may be misplaced above or below the cricothyroid membrane. Placement above the cricothyroid membrane often results from inadequate palpation of landmarks and is associated with an incision into the larynx. Convert the airway as soon as possible to a tracheostomy once this malposition is recognized.[9] Placement below the cricoid cartilage has been estimated to occur in 10% of cricothyroidotomies.[5] There is no specific treatment required for this other than the recognition that the patient has a high tracheostomy.

TABLE 32-2 Complications Associated with Cricothyroidotomy and Preventive Measures

Complication	Preventive measures
Aspiration	Appropriate patient positioning Insert nasogastric tube to decompress stomach Suctioning
Bleeding	Avoid thyroid isthmus Make incision in midline Make incision over cricothyroid membrane
Cartilage (cricoid or thyroid) fracture or laceration	Confirm landmarks and positioning before making incision Know neck anatomy Use small tubes
Creation of a false passage	Assess airflow through tube Confirm landmarks and positioning before making incision Know neck anatomy Monitor oxygenation and ventilation
Dysphonia or hoarseness	Avoid vocal cords Do not force tube inside airway Use small tubes Use tracheal hook
Endotracheal tube in bronchus	Avoid insertion of endotracheal tube too far Use cut endotracheal tube
Esophageal perforation	Do not insert scalpel too deeply
Laryngeal injury	Avoid excessive traction with the tracheal hook Use small tube
Recurrent laryngeal nerve injury	Avoid posterior tracheal wall Make incision in midline
Subglottic stenosis	Avoid long-term use Use small tube
Thyrohyoid membrane incision	Confirm landmarks and positioning before making incision Know neck anatomy
Tracheal stenosis	Remove as soon as possible Use low-pressure cuff
Tube dislodgement	Cut endotracheal tube Maintain control of tube until secured Monitor insertion depth
Tube occlusion	Monitor ease of ventilation Monitor pulse oxygenation

Laryngeal injury may occur if the tracheostomy tube that is inserted is larger than the cricothyroid membrane. The cricothyroid membrane is only 9 to 10 mm in height in the average adult.[11,14] Placement of a tracheostomy tube with a larger outer diameter may cause a fracture of the thyroid cartilage. It is important that the tracheostomy tube placed is no larger than a #6.0 or a #7.0.[11,14] Placement of a larger tube is associated with an increased incidence of subglottic granulation and stenosis.[5,7]

Incisional bleeding occurs in 4% to 8% of patients following a cricothyroidotomy.[5,7,9,11,49] This may result from transection of the anterior jugular veins if the incision extends too far laterally. Bleeding is usually easily treated by suture ligation of the bleeding vessels and packing around the tracheostomy tube with iodoform gauze. A small amount of bleeding or oozing from the cricothyroid arteries can be tamponaded by packing iodoform gauze around the tracheostomy tube. Blood can occlude the tube.[51]

An emergent cricothyroidotomy is usually performed in less than sterile circumstances. There is a risk of developing a wound infection.[49] This is usually the result of skin flora. It can often be treated with wet-to-dry saline dressing changes. In some cases, the infection will not resolve until the cricothyroidotomy tube has been removed. This is done by decannulation or conversion to a formal tracheostomy. Antibiotics are usually not required.

Late complications take two general forms: progressive airway obstruction and chronic voice changes. Patients with progressive airway obstruction present with slowly increasing stridor and dyspnea weeks to months after the procedure. This usually results from subglottic stenosis and granulation tissue formation at the site of the stoma.[5,9,26] It is unclear whether this complication results from the cricothyroidotomy or the tracheostomy tube. The majority of these patients have had prolonged endotracheal intubations before the cricothyroidotomy or prolonged tracheostomy placement after the cricothyroidotomy.[5,26,27,50] In one series, patients with a cricothyroidotomy for a prolonged period (average 72 days) had a 52% incidence of chronic obstruction compared to no obstruction in patients with cricothyroidotomy for a shorter period (average 27 days).[50] Another study recommended that the tracheostomy tube be removed by the fourth day to minimize these long-term complications.[12]

A final late complication is that of voice changes. This has been described in up to 25% of patients who received a cricothyroidotomy.[9,26,27,49] Many of these patients had other methods of airway management either before or after the cricothyroidotomy.

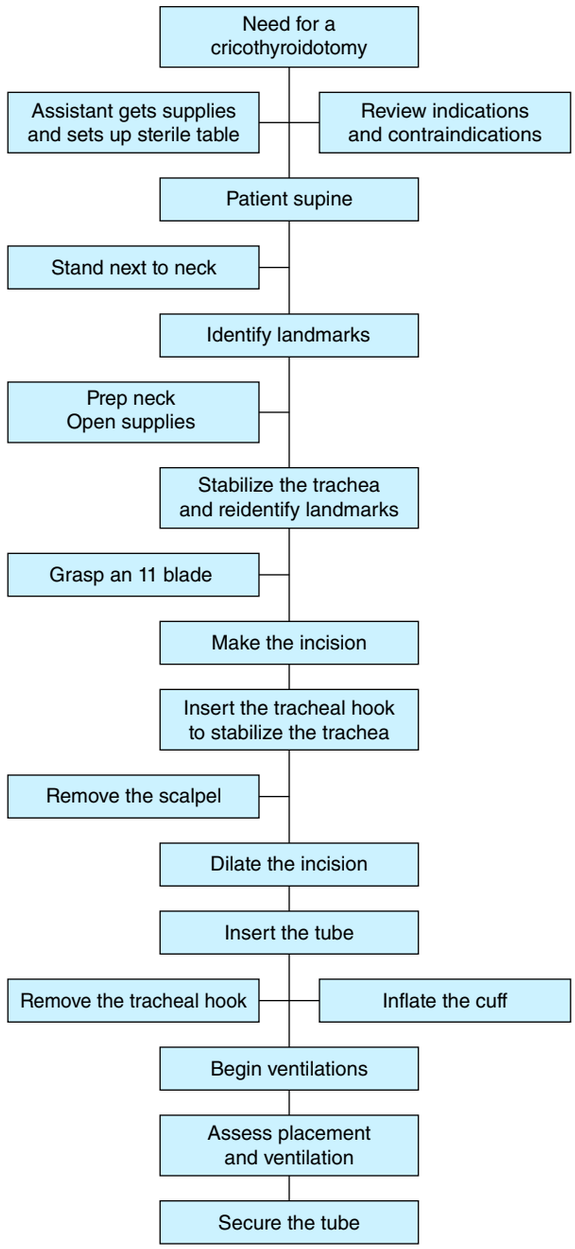


FIGURE 32-23. An algorithm for performing a cricothyroidotomy.

Voice changes tend to be somewhat nonspecific. Patients will complain of decreased volume, fatigue, and hoarseness. These changes may be due to small amounts of granulation tissue at the stoma site and generally resolve over time.[7]

SUMMARY

A cricothyroidotomy is a potentially lifesaving airway management technique. It is an important procedure for the Emergency Physician to be skilled in, as it may represent the only access to the patient's airway. It can be used to provide oxygenation and ventilation to a patient when other less invasive airway control methods have failed or are contraindicated. It is a reliable, relatively safe, and a simple procedure that can be performed within a few minutes (**Figure 32-23**). Self-contained percutaneous and surgical cricothyroidotomy kits are commercially available and simple to use. Knowledge of the anatomy of the anterior neck is essential in order to minimize complications.

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33 Tracheostomy

Eric S. Levy and Christopher J. Haines

INTRODUCTION

Control of the airway is the first priority in the resuscitation of a critically ill patient and must be accomplished before any other intervention can proceed. Emergency Physicians are equipped with multiple nonsurgical techniques and devices to secure an airway including orotracheal intubation, nasotracheal intubation, and laryngeal mask airways. Unfortunately, there are cases in which these methods become impossible or are contraindicated. In these instances, a surgical airway must be obtained and can be accomplished by performing a cricothyroidotomy or tracheostomy. Cricothyroidotomy is described in Chapter 32. This chapter will focus on the indications, technique, and complications for a tracheostomy.

A tracheotomy is the surgical creation of an opening into the trachea, while a tracheostomy refers to the more permanent procedure of bringing the tracheal mucosa into contact with the skin of the neck.[1-3] The most traditional role for a tracheostomy is as an elective procedure done in patients with the need for a prolonged artificial airway. The role of a tracheostomy for emergent airway access has diminished as newer, safer, and equally effective techniques have evolved.

Familiarity with the methods to perform a tracheostomy is still valuable. Knowledge of proper techniques, possible indications, limitations, and likely complications will guide one's judgment in critical moments when it most counts. Understanding the procedure for a tracheostomy will allow Emergency Physicians to properly care for a problem or complication when a patient with a tracheostomy tube presents to the Emergency Department.

ANATOMY AND PATHOPHYSIOLOGY

A surgical approach to the airway relies upon a sound knowledge of the anatomy of the neck and a safe approach to the trachea. **A careful review of this anatomy illustrates how critical it is to remain in the midline in order to avoid morbidity and mortality**. External landmarks are useful in identifying the significant structures of the airway (**Figure 33-1**).[4,5] The laryngeal prominence is a useful guide to the thyroid cartilage. The cricoid cartilage can be identified as a ring just inferior to the thyroid cartilage. In the absence of edema or a hematoma, a finger marched down the midline from the cricoid cartilage can palpate and identify the cartilaginous rings of the trachea. In an emergent situation, these external landmarks may be all a physician has to guide the establishment of a surgical airway.

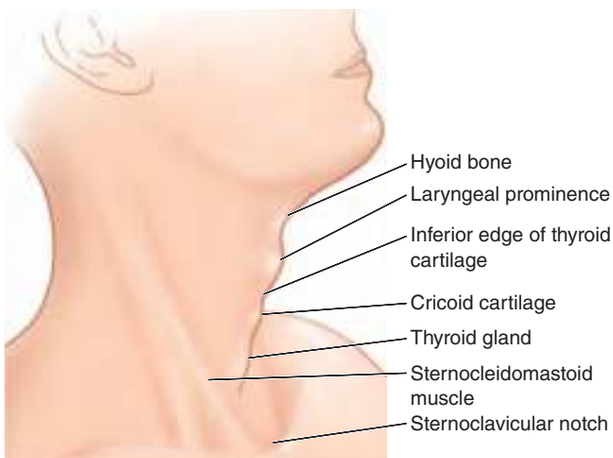

FIGURE 33-1. Lateral view of the topographic anatomy of the neck.

The neck is a complex three-dimensional structure with numerous vital structures coursing through a small space (**Figures 33-2 through 33-6**). The cervical portion of the airway is anterior, superficial, and midline. It is covered by skin, subcutaneous tissue, and numerous muscles (**Figure 33-2**). The basic cartilaginous framework of the airway begins superiorly at the hyoid bone and continues inferiorly with the larynx and trachea (**Figure 33-3**). The external skeleton of the larynx comprises the hyoid bone, thyroid cartilage, and cricoid cartilage.[6] The hyoid bone is a U-shaped structure attached to the mandible, tongue, and base of the skull by muscles. It is the most stable portion of the airway. Even in the presence of pathology, the hyoid bone is remarkably constant in position and can be considered a stable landmark.[7-9]

The larynx is easy to identify externally. The prominent thyroid cartilage forms the laryngeal prominence (i.e., the Adam's apple) at its inferior pole (**Figure 33-3**). It is a freely mobile structure that is anchored by muscles and moves with deglutition. Airway manipulation done on an awake patient will need to fix the larynx to avoid involuntary movement of the larynx from reflex swallowing. The thyroid cartilage is attached to the cricoid cartilage via the cricothyroid membrane (**Figure 33-3**). This is the site for a cricothyroidotomy and can be identified by palpating a slight indentation inferior to the laryngeal prominence. The cricoid is signet ring-shaped and is the only complete cartilaginous ring in the airway. **Procedures should avoid damage to the cricoid cartilage, fearing a loss of stability in the airway.** The cricotracheal ligament attaches the cricoid cartilage to the trachea.

The trachea is a cartilaginous and membranous tube that is approximately 10 to 11 cm long and 2 to 2.5 cm wide in the average adult. In the pediatric patient, the length and width of the trachea will vary depending on the size and age of the child.[10] It extends from the neck into the thorax, where it ends at the carina by dividing into the right and left mainstem bronchi (**Figure 33-4**). It is made up of 16 to 20 incomplete U-shaped cartilaginous rings anteriorly and laterally. The posterior portion is comprised of a fibromuscular membrane. This membrane attaches the cartilaginous rings to one another and imparts great elasticity in the trachea.[2] Full extension of the neck adds significant length to the supraclavicular trachea. This feature should be taken advantage of when performing a tracheostomy.[11]

The trachea is bordered anteriorly by skin, subcutaneous tissue, platysma muscle, pretracheal fascia, and the thyroid gland (**Figure 33-5**). The pretracheal fascia is the anterior portion of the deep cervical fascia. It descends from the thyroid and cricoid cartilages and splits to enclose the thyroid gland, trachea, and esophagus. The pretracheal fascia continues downward into the thorax and mediastinum. Anterior to the trachea, it is very thin and inconsequential. Laterally, it is extremely thick and blends with the carotid sheath.

The thyroid gland lies anterior to the second through fourth tracheal rings and is inevitably encountered during a tracheostomy. It is a richly vascular structure (**Figure 33-6**) that receives its blood supply from the superior thyroid artery (a branch of the external carotid artery) and the inferior thyroid artery (a branch of the thyrocervical trunk). These vessels anastomose into a rich plexus on the anterior surface of the thyroid gland. These major arteries do not usually cross the midline.

Unfortunately, the midline is not always free of blood vessels. An unpaired thyroid ima artery will occasionally be found in the

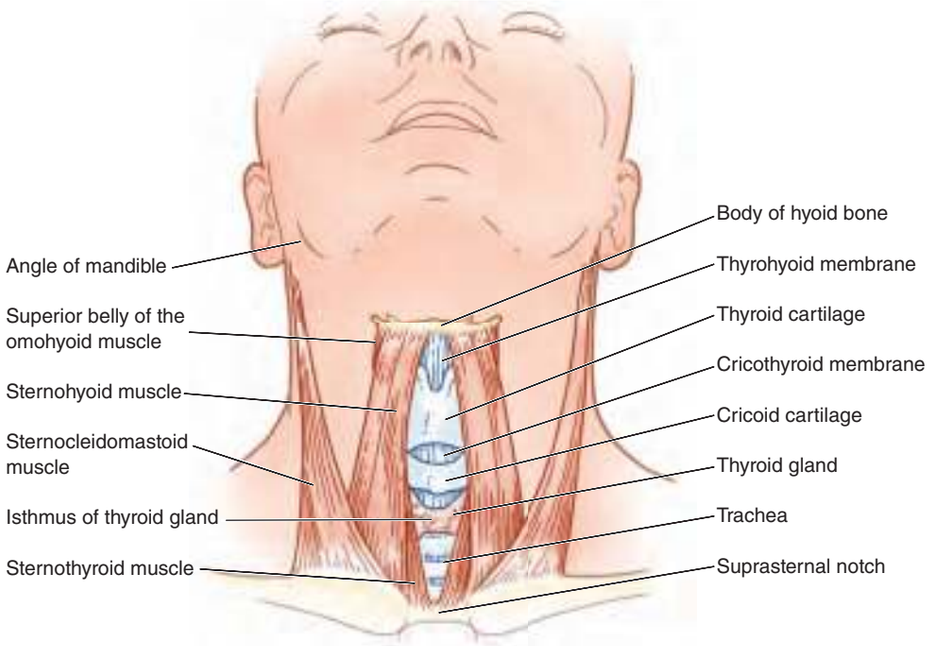

FIGURE 33-2. The superficial muscles and airway structures in the neck.

midline to supply the isthmus of the thyroid gland. The anterior thyroid veins often form a vascular arch across the midline and just inferior to the thyroid gland. A hastily performed tracheostomy or one carried out under difficult conditions may encounter significant hemorrhage from transected vessels.

A number of vital structures surround the trachea and are at risk for injury during a tracheostomy. Their positions relative to one another are best appreciated in a cross-sectional view of the neck (**Figure 33-5**). The deep strap muscles, the sternohyoid and sternothyroid, run adjacent to the trachea and may have to be reflected away for adequate visualization (**Figure 33-2**). The right and left lobes of the thyroid gland encase the upper trachea (**Figures 33-5 and 33-6**) with the thyroid isthmus crossing anteriorly from the second to the third tracheal rings.[2] Close to the thyroid lobes, the recurrent laryngeal nerves run vertically on both sides of the trachea. **These nerves are particularly vulnerable when dissection is extended laterally.**[12] The common carotid artery, internal jugular vein, and vagus nerve are contained by the carotid sheath and lie adjacent to the thyroid gland. The carotid sheath lies extremely close to the trachea in infants and has been mistaken for the trachea in emergent situations.[12] The innominate or brachiocephalic artery crosses left to right immediately anterior to the trachea, while the

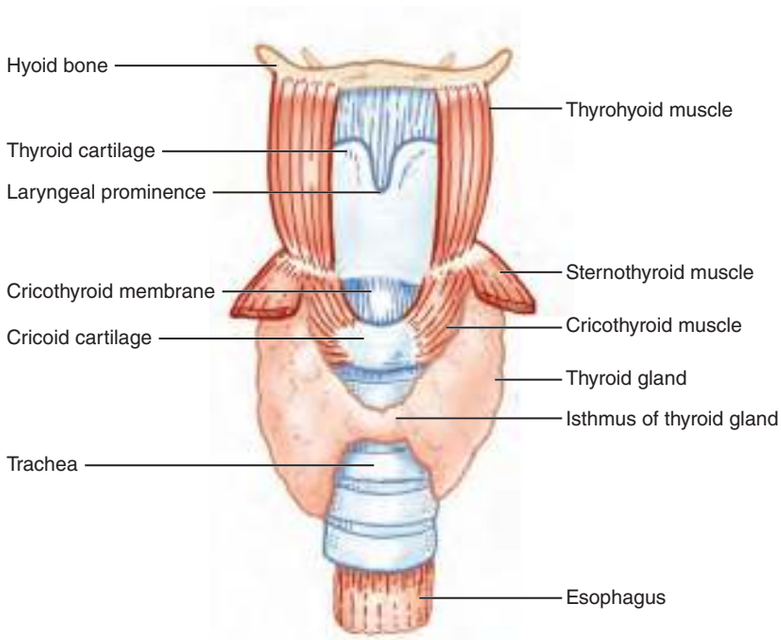

FIGURE 33-3. The framework of the airway in the neck.

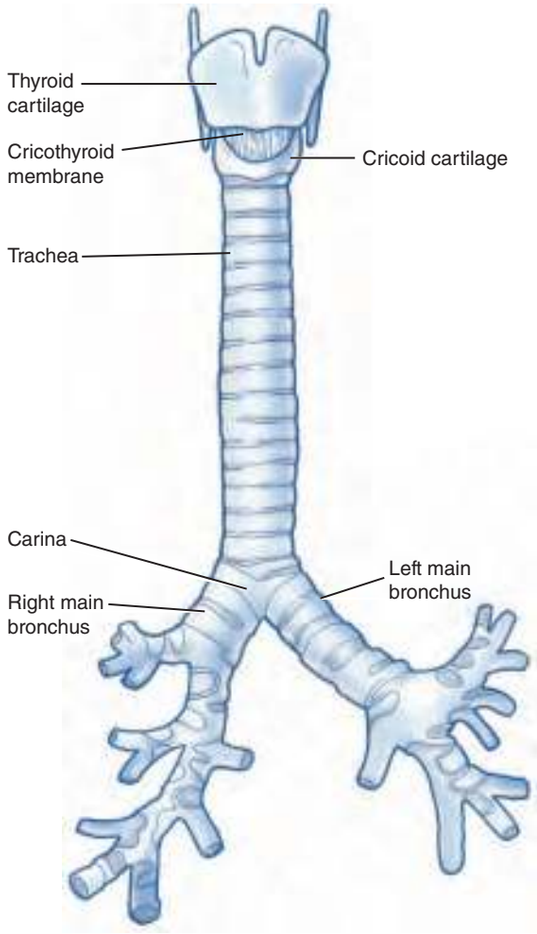

FIGURE 33-4. The cartilaginous framework of the airway.

esophagus lies immediately posterior to the trachea. Erosion of the anterior tracheal wall can occur with prolonged tracheostomy tube placement, leading to a tracheoinnominate artery fistula. The parietal pleura can be found on either side of the trachea at the sternal notch and is also at risk of injury during a tracheostomy.

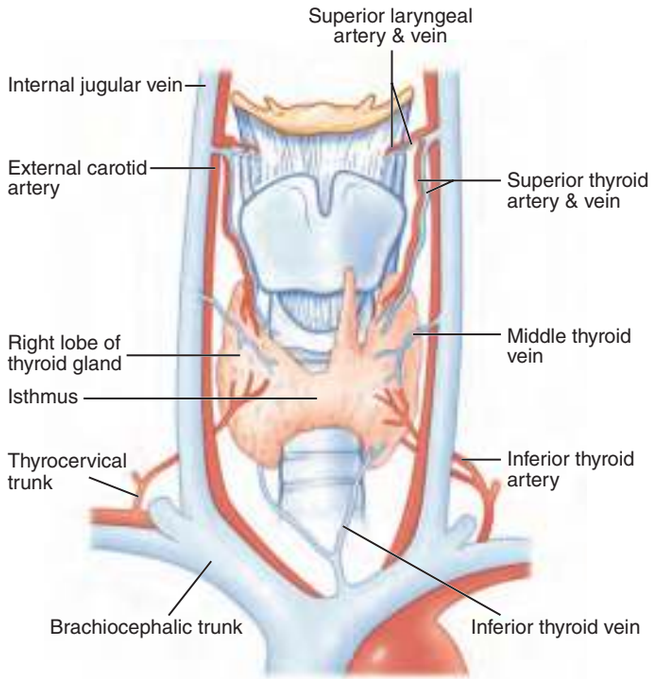

FIGURE 33-6. Vascular structures supplying and surrounding the thyroid gland.

INDICATIONS

The tracheostomy is an ancient and time-honored technique for securing and maintaining an artificial airway. The American Academy of Otolaryngology–Head and Neck Surgery has proposed specific clinical indicators for the use of a tracheostomy (**Table 33-1**).[13] A number of indications for tracheostomy are widely accepted (**Table 33-2**).[7,11-20] As mentioned previously, a tracheostomy is generally considered an elective procedure done under nonemergent conditions after the airway has been secured by other techniques.[21-25] Its use as an emergency procedure is controversial. Used by battlefield surgeons during wartime, its reputation as a procedure of last resort is not without reason. **It is fraught with danger when performed under emergency circumstances.**

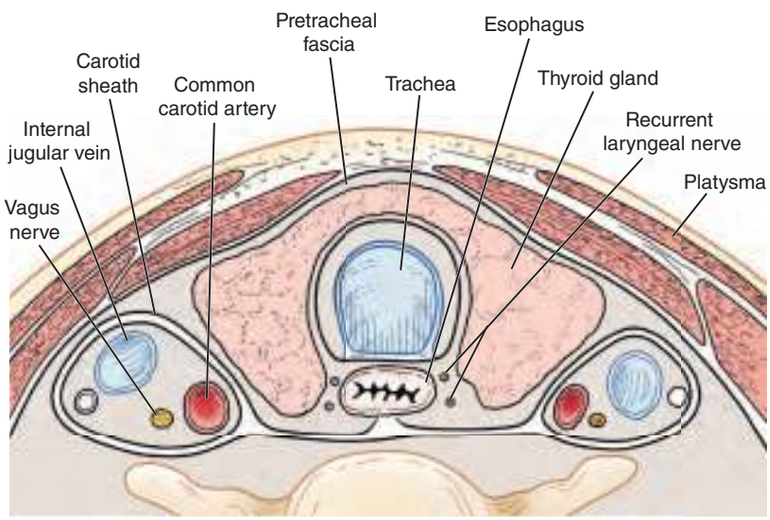

FIGURE 33-5. Cross-sectional anatomy of the neck at the level of the isthmus of the thyroid gland.

TABLE 33-1 Clinical Indications for Performing a Tracheostomy, as Proposed by the American Academy of Otolaryngology–Head and Neck Surgery

Adjunct to manage head and neck surgery
Adjunct to manage head and neck trauma
Bilateral vocal cord paralysis
Difficult intubation
Facilitation of ventilation support
Inability of patient to manage secretions
Need for prolonged mechanical ventilation
Obstructive sleep apnea
Upper airway obstruction

Source: Adapted from reference 13.

A tracheostomy is frequently considered in the treatment of upper airway obstructions including angioedema, airway foreign bodies, complex facial fractures, deep space neck infections, epiglottis, and multiple lacerations to the floor of the mouth. Although these conditions can create serious and immediate airway compromise, the airway can be managed in most cases with orotracheal intubation. **An emergency tracheostomy is not the treatment of choice but rather the choice of last resort**.

There are a few clinical settings in which an emergency tracheostomy should be considered. These include laryngotracheal injury with airway disruption, severe maxillofacial trauma, complete subglottic obstruction, and the need for an airway when all other methods have failed.[12,26,27] **With the exception of these limited applications, an emergency tracheostomy is discouraged.**[12,15,28,29]

LARYNGOTRACHEAL TRAUMA

Most head and neck trauma patients can be managed with nonsurgical airway techniques. There are a number of options available including orotracheal intubation, nasotracheal intubation, intubation guided by a lighted stylet or "lightwand," retrograde guidewire intubation, fiberoptic-assisted intubation, and video laryngoscopy. Nasotracheal intubation should be avoided in patients with a potential head injury. **If a surgical airway is necessary, a cricothyroidotomy is usually the procedure of choice.**

TABLE 33-2 Commonly Cited Indications for Performing a Tracheostomy

Airway obstruction
Congenital airway anomalies
Edema
Foreign body
Hemorrhage
Infection
Trauma
Tumor
Inability to open mouth
Angioedema
Impaired clearance of secretions
Inability to intubate by other measures
Laryngeal injury
Laryngospasm
Maxillomandibular fixation
Maxillomandibular trismus
Need for aggressive pulmonary toilet
Need for prolonged ventilatory support
Surgical airway in an infant
Unstable cervical spine
Trauma to face and neck with airway compromise

Source: Adapted from references 7, 11-20.

Laryngotracheal injuries are rare but potentially life threatening. When suspected, immediate efforts should focus on getting the patient to the Operating Room. These injuries can result from either blunt or penetrating injury to the neck. They are often accompanied by edema, hemorrhage, subcutaneous emphysema, and fracture of either the thyroid or cricoid cartilages. A tracheostomy should be considered when rapid access to the cricothyroid membrane may be limited, making it difficult to perform a cricothyroidotomy.[12,30-32] Blunt injury has been described when the anterior neck forcefully strikes a fixed object such as a rope or cable. A classic example of this type of injury is forcefully striking the anterior neck against the steering wheel during high-speed motor vehicle collisions.[12,31,32] Penetrating injuries to the airway are usually apparent; however, blunt injuries to the trachea require a high index of suspicion.[32,33] In a review of 51 patients with blunt injury to the trachea, the most common presenting signs and symptoms included dysphonia, hemoptysis, hoarseness, respiratory distress, and subcutaneous emphysema.[32] In this same review, a high rate of endotracheal intubation failure was reported with the conclusion that emergent tracheostomy was the best means of airway control.[32] In cases of penetrating trauma to the larynx, Emergency Physicians may consider obtaining emergent airway control directly through the wound as a temporary measure prior to operative management.[10]

Laryngotracheal disruption, occurring when the larynx and trachea become separated, is an unusual injury. This is the one injury in which a tracheostomy is the undisputed method for establishing and securing the airway. Patients with laryngotracheal disruption may exhibit varying degrees of aphonia, blood-streaked sputum, bruising over the anterior neck, hoarseness, respiratory distress, and subcutaneous emphysema. A defect may be palpable in the patient's neck. Soft tissue radiographs of the neck may reveal an interrupted air column. **If the airway is disrupted, a "low tracheostomy" between the fourth and fifth tracheal rings should be performed.** The severed airway will tend to retract into the thorax, and a low tracheostomy will offer the best chance of securing the dismembered segment. Misguided attempts to visualize the airway, intubate orally, or perform a cricothyroidotomy may further damage the already precarious airway. This is fortunately a rare injury with only a few case reports in the literature.[26,27] A tracheostomy performed in this setting is more treacherous than usual. **Although a tracheostomy may be the only way to secure the airway, this airway intervention has a better chance of success when performed in the Operating Room.**

SURGICAL AIRWAYS IN THE PEDIATRIC PATIENT

Surgical approaches to the airway are more difficult and complicated in pediatric patients, especially newborns and infants.[3,34,35] The larynx is positioned more cephalad than in the adult, allowing relative protection by the mandibular arch.[10] It is more anteriorly located and significantly smaller than in adults, as well as more floppy and mobile. The infant's cricothyroid membrane, unlike that of the adult, is extremely narrow and cannot easily be used for access to the airway. For this reason, a surgical cricothyroidotomy should be avoided until a child is older than age 12 years.[10,12] **Attempts at creating emergency surgical airways in children are typically acts of desperation. A calm and reasoned approach to the pediatric airway and common respiratory problems is essential.** Efforts should first be made to suction the airway clear of secretions followed by ventilation with a bag-valve-mask device. **Direct visualization by laryngoscopy and orotracheal intubation should be attempted before resorting to a surgical airway. If an unstable patient cannot be ventilated, a needle cricothyroidotomy can serve as a**

temporizing measure until a surgical team can be assembled and better control achieved.[10,12]

In the pediatric population, laryngotracheal injury is rare.[12] Children typically sustain blunt anterior neck injury as the result of high-speed collisions involving bicycles, falls, and motor vehicles. This injury type may be the result of sports-related activity. Clinical signs and symptoms of laryngeal or tracheal trauma mirror that of adults. Airway management in these patients can be achieved with a tracheostomy. However, this may be quite difficult and is only advocated by some authors.[10] **Attempts to obtain surgical assistance should be sought immediately.**

CONTRAINDICATIONS

An emergency tracheostomy is contraindicated when other methods can be used to secure the airway. There are a few instances when an immediate surgical airway is necessary. **Cricothyroidotomy is the procedure of choice when a surgical approach to the airway is required**. A cricothyroidotomy is faster, more direct, relies predominantly on external landmarks, requires only a single operator, can be done with ambient lighting, and requires a limited amount of equipment.[13,36] **In contrast, a tracheostomy is a procedure requiring multiple steps.** The tracheostomy involves direct visualization to dissect through vascular structures and requires better light than is commonly present at the bedside. A tracheostomy is easier and therefore faster to execute if one has an assistant, proper suctioning equipment, and electrocautery. Without these advantages, the technique is difficult and likely to be complicated. A "timely trach" can be performed within 5 to 10 minutes.[13] However, reported times to definitive airway during elective open tracheostomies have ranged from 13.5 to 105 minutes.[36-39] Although this time frame may be adequate for urgent situations, it is too slow for the true emergency in a patient who lacks an airway.

There are numerous relative contraindications and no absolute contraindications.[40] These are relative as the alternative is death. A known high-riding innominate artery may be injured in the procedure. A coagulopathy or thrombocytopenia can result in uncontrollable bleeding. Fracture of the larynx may result in airway collapse and uncontrolled bleeding. The issue with young children has already been discussed. Infection surrounding the procedure site can compromise the airway or result in spread of the infection. High levels of positive end-expiratory pressure are difficult to maintain. A laryngotracheal disruption may turn into a worse airway nightmare as the distal trachea moves further into the chest. The procedure is more difficult in the morbidly obese patient. Finally, inexperience with the tracheostomy procedure makes it more difficult to perform.

EQUIPMENT

A tracheostomy requires an extensive amount of equipment. Appropriate supplies should be sterilized and assembled in a prepackaged tray. A list of required equipment is given in **Table 33-3**. If such a tray is not available, supplies that are immediately on hand have to suffice. A thoracotomy or major procedure tray will contain most of the required equipment.

PATIENT PREPARATION

Patient preparation will depend largely on the circumstances dictating the procedure. Rapid control will have to be established with a sedative if the patient is uncooperative, hypoxic, or thrashing about. The ideal agent is one that sedates with minimal hemodynamic consequences, preserves spontaneous respirations, and leaves an intact gag reflex (i.e., ketamine). **Procedural sedation and analgesia (Chapter 159) can cause the patient to stop breathing and worsen the situation.** Calm reassurance may allow the procedure to be performed under local anesthesia if the patient is awake and cooperative.[41] A local anesthetic is sufficient if the patient is unconscious.

TABLE 33-3 The Supplies Required to Perform a Tracheostomy

Patient preparation	Setup
Cardiac monitor	Povidone iodine or chlorhexidine solution
Pulse oximeter	Surgical drapes to enclose the field
Intravenous line with saline	Sterile gown, gloves, and mask
Oxygen	Hat
Ambu bag for ventilating patient	
Procedure	
Local anesthesia	Two pairs of scissors, one straight and one curved
10 mL syringe	Two tissue forceps without teeth
1% lidocaine	Two Allis forceps, to grasp the trachea
18 gauge needle	Two small rakes, for exposure
25 gauge needle	Mastoid retractor
#10 scalpel blade and handle	Trousseau dilator
#11 scalpel blade and handle	Two tracheal hooks
Two skin forceps	10 mL syringe
Eight small curved hemostats	Umbilical tape
Sterile 4×4 gauze squares, two dozen	Needle holder
Two Kocher forceps (if needed, to clamp the thyroid)	
Frazier suction catheter with suction tubing	
Suture ligatures (3–0 chromic, 3–0 silk, 3–0 nylon)	
Tracheostomy tube, appropriate size for patient	
Water-soluble lubricant or anesthetic jelly	
Suction source and tubing	

Place a rolled towel under the patient's shoulders and neck if no contraindications exist (Figure 33-7). This detail cannot be emphasized enough. Full neck extension brings the airway anterior and increases the length of the supraclavicular trachea by as much as 2.6 cm.[10,42] It enlarges the surgical field for improved access. Neck extension tends to fix the airway in position and pull it taut. **When the neck is in a neutral or flexed position, the trachea lies more posteriorly and is more "floppy," it is easier to stray off midline, and the surgical field may be reduced to a dark hole with poor visibility. A certain degree of extension is helpful in the pediatric patient, but full extension may occlude the airway.**

FIGURE 33-7. Optimal patient positioning for a tracheostomy with the neck extended.

FIGURE 33-8. Hand positioning to palpate the anatomic landmarks.

Check the equipment, briefly and quickly, prior to beginning the incision. A tracheostomy tube that is appropriate for the patient's size should be selected. An average male will accommodate a size 7 or 8 Shiley tracheal tube. An average-size female will accommodate a size 6 or 7 Shiley tracheal tube. The cuff of the tracheal tube should be tested prior to use. An endotracheal tube can be used as an alternative to a tracheostomy tube.

Identify the anatomic landmarks required to perform this procedure. Using the nondominant hand, place the thumb on one side and the middle finger on the other side of the patient's trachea (**Figure 33-8**). Identify the laryngeal prominence (i.e., the Adam's apple) with the index finger. Slide the index finger caudally to identify the cricothyroid membrane, the cricoid cartilage, and the tracheal rings.

Clean the neck of any dirt and debris. Even during the resuscitation of an unstable patient, there should be adequate time for the use of aseptic solution as well as sterile technique. Prepare yourself by applying a hat, mask, eye protection, sterile gown, and sterile gloves. Apply a sterile drape to isolate a surgical field. Infiltrate local anesthetic solution along the planned incision line. It was once thought that injection of local anesthetic into the trachea blunts the cough reflex and provides a sense of depth of the airway. A recent review of 97 patients who underwent an awake tracheostomy noted that this could result in cough, agitation, and less beneficial outcomes.[43]

Once the patient is positioned, prepped, and anesthetized, establishment of the airway should be just 2 to 3 minutes away. **The last step in preparation should be the mental decision that no other technique will suffice and the commitment to proceed with confidence**.

TECHNIQUE

In the most critical situation, the simplest technique is likely to be the most successful.[44] The right-handed operator should stand to the patient's right and fix the airway with their nondominant left hand (**Figure 33-8**). Left-handed operators should adjust their technique based on their preference for handedness. Apply the thumb and third digit of the nondominant hand on the thyroid cartilage while the index finger palpates the cricoid and tracheal rings (**Figure 33-8**). An awake or lightly anesthetized patient may swallow or gulp during the procedure, thereby moving the landmarks. Fixing the left hand firmly on the upper airway will minimize this distraction.

A vertical incision is preferred in the emergent setting because it allows greater exposure of the cricothyroid membrane and the trachea and avoids traversing lateral structures.[12] Make a 3 or 4 cm vertical midline incision through the skin and subcutaneous tissues beginning just below the cricoid cartilage and extending inferiorly to the supraclavicular notch (**Figure 33-9**). A larger skin incision causes no harm as long as it remains superficial to avoid damaging the cricoid cartilage. **Extreme care should be taken to ensure that the incision and further dissection remain in the midline.**[12]

Once the skin and subcutaneous tissue have been incised, attention should be directed toward clearing the pretracheal space and defining the tracheal rings. Divide the superficial and deep strap

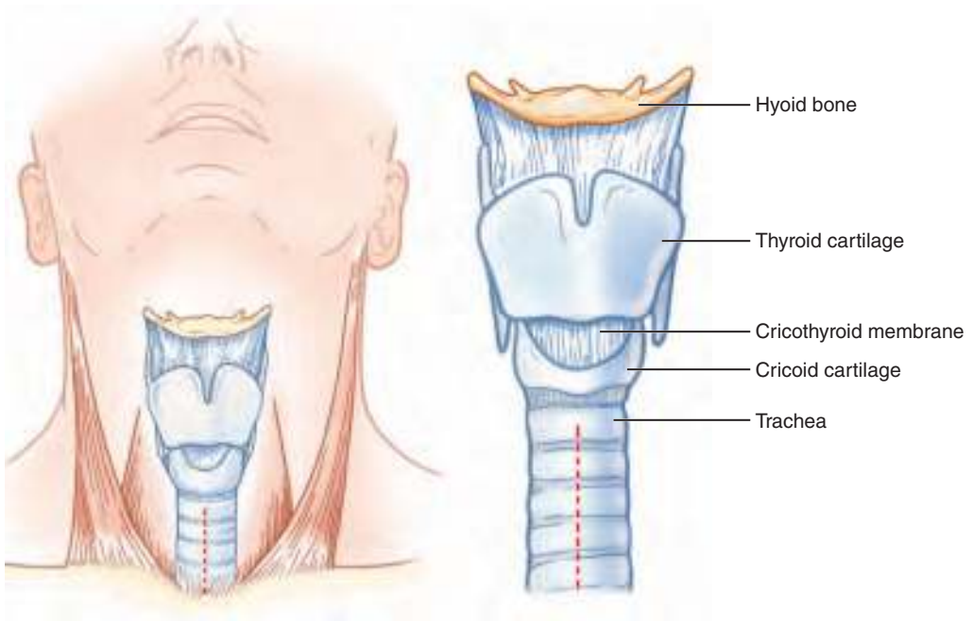

FIGURE 33-9. The skin incision is made in the midline, beginning below the cricoid cartilage and extending down toward the supraclavicular notch. An incision made with these landmarks will lie over the second through fourth tracheal rings.

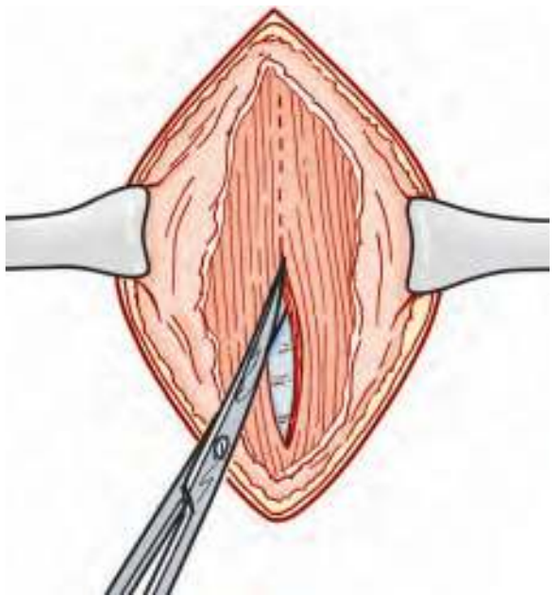

FIGURE 33-10. The skin and subcutaneous tissues have been retracted. The strap muscles are divided in the midline to expose the pretracheal space.

muscles in the midline and retract them away from the trachea (**Figure 33-10**). Bluntly dissect free and reflect away any blood vessels in front of the trachea. If they impede progress, they can be clamped with hemostats and divided. The thyroid gland lies above the trachea. Bluntly dissect between it and the trachea with a hemostat to mobilize the thyroid gland (**Figure 33-11**). An alternative option to consider in the emergent setting is to use the nondominant index finger to dissect the pretracheal space bluntly and reflect the thyroid isthmus either superiorly or inferiorly. This minimalist blunt approach lacks finesse but is effective and timely.[15]

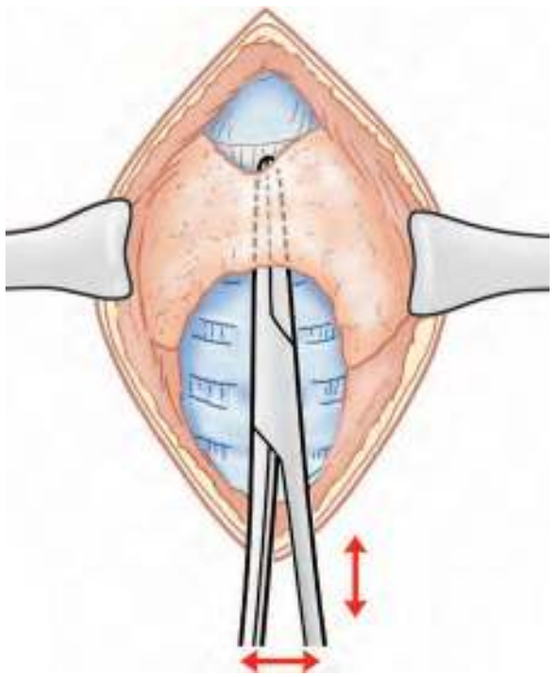

FIGURE 33-11. The thyroid gland is bluntly dissected from the trachea. Arrows represent movement of the hemostat.

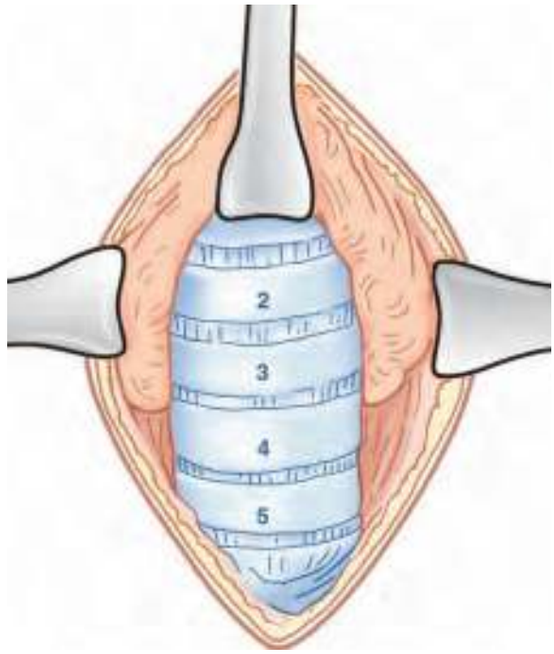

FIGURE 33-12. The thyroid gland is retracted upward and out of the surgical field.

Retract the thyroid gland upward (**Figure 33-12**). If this becomes difficult, it may be faster to divide the isthmus between two hemostats (**Figure 33-13**). The hemostats will limit the bleeding from obstructing the surgical field. The divided edges can be oversewn after the tracheostomy tube is in place. Once the thyroid has been divided, the operating field will be cluttered with hemostats and may be partially obstructed. **Do not apply traction to the instruments as this can avulse tissue and lead to bleeding and further obstruction of the surgical field.**

When ready to make the tracheal incision, it may be helpful to have an assistant place a tracheal hook under the first tracheal ring

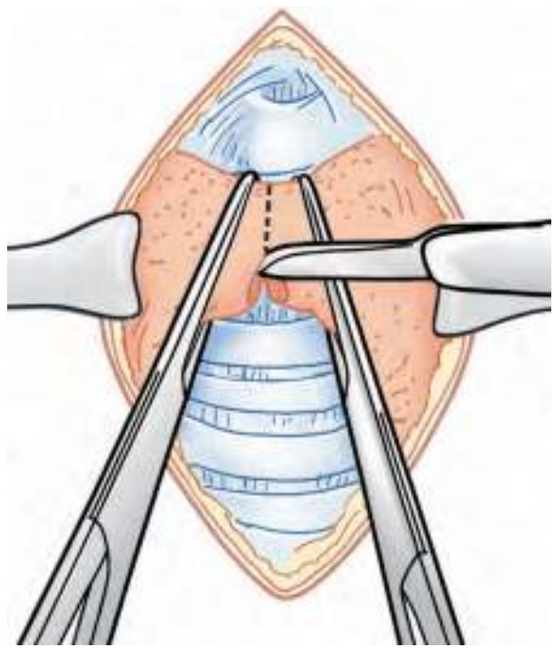

FIGURE 33-13. An alternative method of clearing the thyroid gland from the surgical field. The isthmus is clamped and transected.

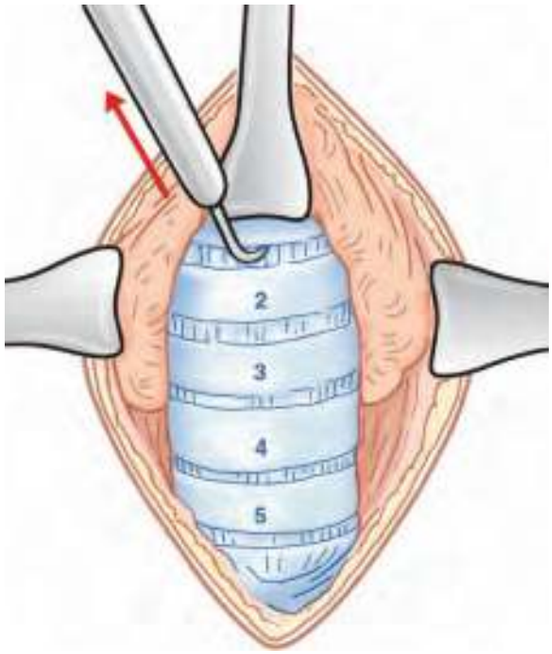

FIGURE 33-14. A tracheal hook is placed below the first tracheal ring to elevate and immobilize the trachea.

and apply traction superiorly and anteriorly (**Figure 33-14**). Have the assistant hold the tracheal hook in position. This will elevate and immobilize the trachea. The assistant should direct their hands superior to the wound to keep the field unobstructed. If the neck is properly extended, this may be unnecessary. **If at any time identification of the trachea becomes difficult, needle aspiration may be used to confirm the presence of an air-filled tube.**

Make an incision in the trachea. The preferred tracheotomy is a midline vertical incision extending from the second through the fourth tracheal rings (**Figure 33-15*A***). There are other options, but they are discouraged in the emergent setting because they stray from the midline (**Figures 33-15*B* to *D***). Insert a Trousseau dilator

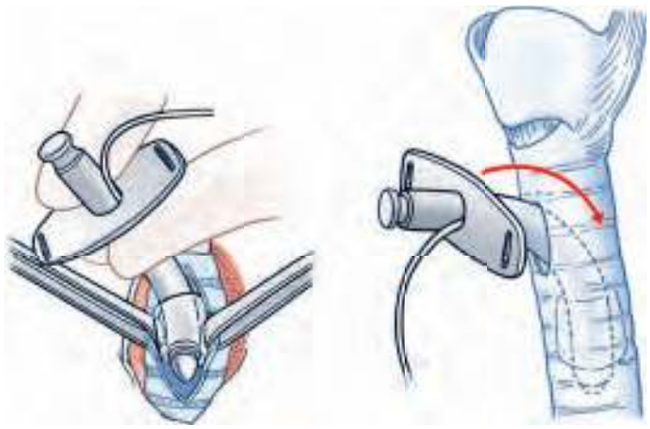

FIGURE 33-16. Insertion of the tracheal tube. ***A.*** The incision is held open with a Trousseau dilator, hemostat, or Allis forceps (shown here) as the tracheostomy tube is inserted. ***B.*** The tracheostomy tube is advanced and the cuff inflated.

or hemostat into the incision. Use the dilator to open and widen the incision. Alternatively, the sides of the tracheal incision can be grasped and held open with Allis forceps (**Figure 33-16*A***).

Liberally lubricate the appropriate-size tracheostomy tube. Insert the tracheostomy tube, with its obturator, through the tracheotomy while the incision is held open (**Figure 33-16*A***). Advance the tracheostomy tube and inflate the cuff (**Figure 33-16*B***). Hold the tracheostomy tube in place securely. Remove the obturator and insert the inner cannula. Attach a bag-valve device and begin ventilating the patient.

USE OF ULTRASONOGRAPHY

There has been a recent surge of bedside ultrasound in the Emergency Department. A tracheostomy is no exception.[45-50] The use of ultrasound improves percutaneous tracheostomy. It allows identification of the landmarks, aids in choosing an appropriate site for the procedure, and is helpful in estimating the distance from the skin to the airway. Ultrasound allows precise localization of the needles and

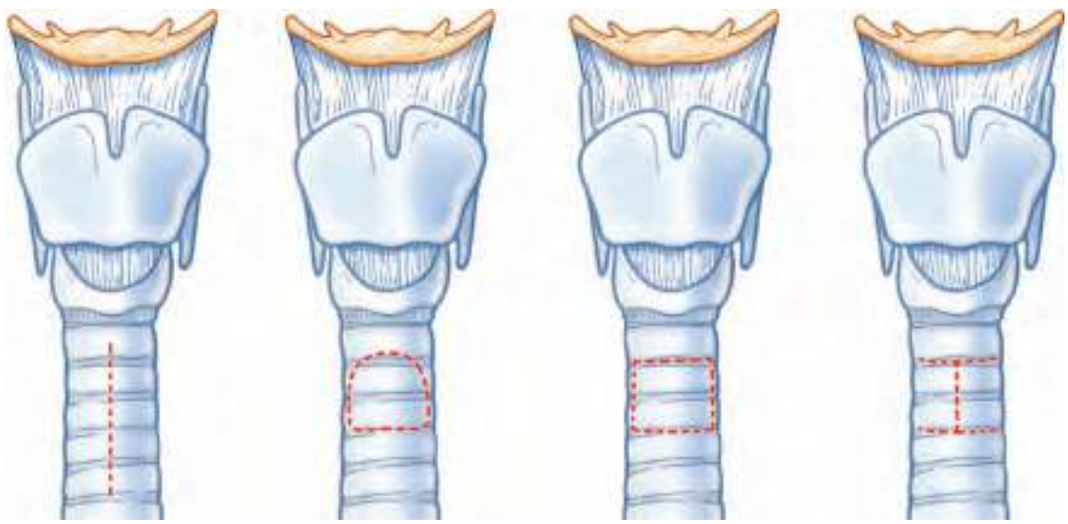

FIGURE 33-15. Types of tracheal incisions. ***A.*** A midline vertical incision through the second, third, and fourth tracheal rings. This is the preferred technique in an emergent procedure. ***B.*** A U-shaped inferiorly based window. ***C.*** A window has been created by excising the second and third tracheal rings. ***D.*** The T-flap.

A B

FIGURE 33-17. Commercially available percutaneous tracheostomy kits. ***A.*** The Ciaglia Blue Rhino kit. ***B.*** The Dolphin BT Ciaglia Balloon Assisted Trach Kit. (Photos courtesy of Cook Medical Inc.)

increases first-pass puncture. It reduces complications and can be used to detect postprocedure complications (e.g., a pneumothorax).

ALTERNATIVE TECHNIQUES

Since 1969, a number of authors have described a third invasive option for airway control, the percutaneous dilatational tracheostomy (**Figure 33-17**).[51-60] Although techniques vary somewhat, they all rely on an initial puncture of the airway followed by progressive dilatation of a tract. Once a sufficiently large tract is formed, an airway tube is placed. There is now enough evidence to argue that percutaneous tracheostomies are competitive with, and perhaps preferable to, formal open tracheostomies done under elective conditions.[37,38,61-73] Several large series of percutaneous tracheostomies are now complete, but significant results are available only for elective tracheostomies done in patients who have already been intubated.[62,64,65,70] There are small case studies that have been published regarding the successful use of percutaneous dilatational tracheostomy in an emergency setting. These authors conclude that percutaneous dilatational tracheostomy is an effective airway, providing an alternative to endotracheal intubation. The resultant airway is stable and does not require conversion in the immediate postresuscitation period.[74] Furthermore, a recent case series of four patients undergoing percutaneous dilatational tracheostomy in the intensive care setting illustrated the possible benefits of performing the procedure in combination with ultrasound guidance.[75] The authors concluded that the use of ultrasound could identify patients unsuitable for the procedure, prevent puncture of aberrant vessels, estimate the distance from the surface of the skin to the trachea, and ensure accurate placement of the needle into the trachea.[75] While these studies are promising, larger studies are needed to determine the ultimate role of percutaneous dilatational tracheostomy in the emergency setting.[74] **The cricothyroidotomy remains the procedure of choice for emergency surgical access to the airway.**

ASSESSMENT

Once the airway is established, tracheostomy tube positioning should be confirmed by auscultation, ease of ventilation, and pulse oximetry. Obtain a chest and neck radiograph to confirm tracheostomy tube position and exclude a pneumothorax.

AFTERCARE

Once the airway has been established, inspect the wound to ensure hemostasis. Any clamped vessels or thyroid tissue should be tied. The skin edges do not need to be closed unless the skin incision was overly zealous. If the skin is reapproximated, close it loosely to avoid the development of subcutaneous emphysema. Secure the tracheostomy tube with umbilical tape wrapped around the neck. Suture the flange of the tracheostomy tube to the skin using 3–0 silk as an additional safeguard (**Figure 33-18**). If the wound is oozing, place gauze between the skin and the tracheostomy tube.

Special care should be taken to protect the artificial airway. Suction the lumen frequently and as necessary to prevent obstruction from blood or secretions. Administer humidified oxygen through the tube to prevent dried and inspissated secretions from occluding the tracheostomy tube.

COMPLICATIONS

An elective tracheostomy is a relatively basic and common surgical procedure. Despite this, it has an unusually high complication rate. Authors report widely different morbidity rates, perhaps determined in part by the clinical settings if not their own biases. Reported morbidity ranges from 6% to 58%, with procedures done emergently having the highest rates of complications.[22,23,25,36] However discomforting these numbers may be, the risk is certainly acceptable in the face of an unstable airway in a dying patient.

Complications from a tracheostomy can be divided into immediate operative complications, postoperative complications, and delayed complications. A summary of reported complications are listed in **Table 33-4**. **It cannot be stressed enough that the best way**

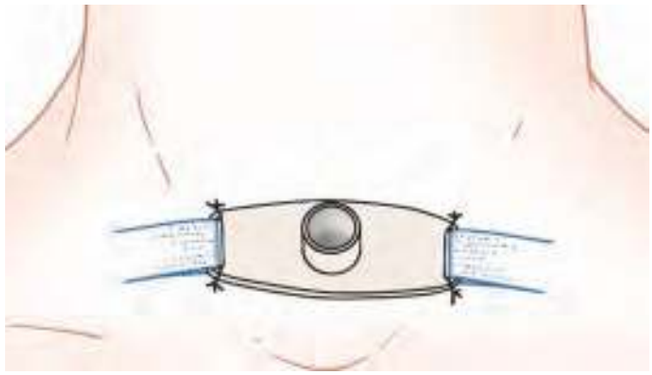

FIGURE 33-18. The tracheostomy tube is secured in place with umbilical tape ("tracheal tie") and then sutured.

TABLE 33-4 Complications of Tracheostomies

Immediate	Delayed	Postoperative
Air embolism	Accidental decannulation	Accidental decannulation
Aortic arch rupture	Delayed wound problems	Aspiration
Apnea	Excess granulation tissue	Delayed hemorrhage
Cardiac arrest	Persistent stoma	Delayed wound problems
Cardiac dysrhythmias	Fused vocal cords	Excess granulation tissue
Cartilage injury	Hemorrhage	Persistent stoma
Cricoid	Infection	Disrupted tract
Trachea	Mediastinitis	Displaced tube
Tracheal rings	Pneumonia	Infection
Cuff rupture	Sternoclavicular joint	Mediastinitis
Damage to surrounding structures	Stoma	Pneumonia
Anterior jugular vein	Tracheitis	Sternoclavicular joint
Common carotid artery	Wound	Stoma
Esophagus	Subglottic stenosis	Tracheitis
Internal jugular vein	Tracheal stenosis	Wound
Pleura	Tracheal tube obstruction	Mediastinal emphysema
Pneumothorax	Tracheal tube cuff rupture	Obstructed tube by mucus plug
Pneumomediastinum	Tracheoesophageal fistula	Subcutaneous emphysema
Posterior tracheal perforation	Tracheoinnominate artery fistula	Tracheal tube cuff rupture
Recurrent laryngeal nerve	Tracheomalacia	Voice changes
Subcutaneous emphysema	Voice changes	
Voice changes		

Source: Adapted from references 13, 15 18, 21-23, 28, 29, 35, 39, 40, and 76-86.

to avoid complications is to avert the need for a tracheostomy. Under emergent circumstances, a variety of things can and do go wrong. It is best if one has thought about these possibilities, considered alternative strategies for airway management, and sought assistance from surgical colleagues. A more detailed discussion regarding chronic complications is found in Chapter 34.

The most common problem with a hastily performed tracheostomy is hemorrhage. Most bleeding can be controlled with the application of direct pressure. An assistant may be invaluable in providing sufficient control until the airway is established. **The search for a bleeding source during the procedure will waste valuable time and can usually wait until the airway is established.** The best way to avoid this difficulty is strict hemostasis during the procedure and being careful to avoid the transection of any blood vessels. The next most likely problem is inadvertent injury to adjacent structures. **Injury to adjacent structures is avoided by remaining strictly in the midline, positioning the patient properly, and using a tracheal hook.** Additional problems that can be encountered include the creation of a false passage. This should not occur if the operator has visualized the trachea and remained in the midline. In pediatric patients, pneumomediastinum is one of the most frequent early complications of a tracheostomy.[87]

The Emergency Physician should be cautious not to adopt methods for tracheostomies learned from elective procedures performed in the Operating Room under controlled circumstances. **The emergent tracheostomy must be carried out with the simplest, fastest, and most straightforward technique possible**. Tracheostomies done under elective conditions may use rescue stay sutures and more elaborate tracheal incisions. The added benefits of these features do not offset the additional time required to perform them.

Any physician who cares for patients under emergency circumstances should think through the clinical scenarios in which an emergent tracheostomy may be necessary. Expertise in surgical airway techniques should first be obtained in a laboratory setting. Unless one is poised to respond with a plan of action for the emergent airway and prepared with the necessary surgical skills, such situations create chaos and all too often end in disaster.

SUMMARY

A well-trained Emergency Physician must be prepared for any kind of airway emergency and should be skilled in a variety of approaches. Optimal airway management begins with optimal medical management of the patient, including the early identification of possible airway compromise and aggressive preventive treatment. Many airway problems can be averted with anticipatory action. In the armamentarium of airway procedures, a tracheostomy will be and should be a rare solution. The Emergency Physician who is knowledgeable about and comfortable with alternative airway techniques, including surgical access, will be prepared to act decisively yet appropriately upon encountering a challenging airway crisis. While a cricothyroidotomy is the surgical airway procedure of choice, a tracheostomy should be considered for laryngeal injuries with airway disruption or when all other methods have failed.

In the future, an automatic device may make emergent tracheotomies faster and safer.[88] Researchers in Madrid have designed a device and patented it. The device identifies the trachea and makes a clean incision. It does this by locating the correct location, adjusting the needle preload, making the incision, dilating the incision, and maintaining the airway.

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34 Tracheostomy Care

H. Gene Hern Jr and Julie K. Oliva

INTRODUCTION

Tracheostomy care and management of tracheostomy complications are tremendously important to the Emergency Physician. Rapid assessment and understanding of tracheostomies and their potential complications can be lifesaving in the critically ill and tracheostomy-dependent patient.

Tracheostomies have been performed since ancient times but have been perfected in the last few centuries. A Greek physician named Asclepiades of Bismuth was the first credited in 100 BC with performing a successful tracheostomy.[1] Two of the four physicians summoned to President George Washington's deathbed were said to have argued for tracheostomy as his only means of survival. Trousseau reported successful tracheostomies in more than 2000 cases of upper airway obstruction secondary to diphtheria in the 1800s.[2] Chevalier Jackson perfected the tracheostomy technique and reduced the operative mortality from 25% to less than 1% in the 20th century.[3] This is roughly what it remains today.

The important aspects of tracheostomy care include the assessment of respiratory distress in the tracheostomy patient, proper suctioning techniques, and assessment and evaluation of possible complications arising from the tracheostomy or its placement. Tracheostomy care will be divided into routine care and emergent care.

ANATOMY AND PATHOPHYSIOLOGY

The trachea is a fibromuscular tube with approximately 16 to 20 cartilaginous arches extending from the cricoid cartilage to the division at the carina into right and left mainstem bronchi (**Figure 33-4**). The surface of the tracheal mucosa is covered in respiratory epithelium. This epithelium is responsible for tracheal secretion, mucociliary "elevator" movement of secretions and debris, and humidification. The remaining part of the upper respiratory tract bypassed by the tracheostomy plays a major role in warming and humidifying inspired air.

The terms *tracheostomy* and *tracheotomy* are widely interchanged in current parlance. Tracheotomy refers to the actual incision through the skin to the trachea, which is then kept open by a tracheotomy tube. A tracheostomy refers to the procedure in which the tracheal opening is sutured to the skin incision. This creates a more permanent orifice. The term *tracheostomy* will be used for the remaining sections of this chapter.

A tracheostomy is created by an incision at the level of the second or third tracheal rings. An incision is made into the trachea after the subcutaneous tissue is dissected and anatomic structures identified. A hook is inserted into the incision and used to stabilize the trachea while a tube is placed into the trachea. The trachea is secured to the overlying skin and the tube is secured in place. Further details can be found in Chapter 33.

TRACHEOSTOMY TUBES

Tracheostomy tubes vary in their composition, angles, and types and the presence or absence of a cuff (**Tables 34-1, 34-2, and 34-3**). The basic tube consists of an outer cannula and an inner cannula (**Figure 34-1**). The size of the tracheostomy tube is usually defined by its inner diameter. The outer cannula is the more permanent fixture in the tracheostomy. The inner cannula is a low-profile tube that inserts into the outer cannula. It can easily be removed and replaced. The inner and outer cannulas contain a locking mechanism by which the inner cannula is secured into the outer cannula. The proximal end of the inner cannula contains a standard 15 mm connector that allows direct connections to a ventilator or bag-valve device. The tracheostomy tube is available in fenestrated and nonfenestrated versions (**Figure 34-2**).

Pediatric tracheostomy tubes have a much smaller inner diameter and do not accommodate inner cannulas (**Figure 34-3**). They require more frequent suctioning and changing since pediatric tracheostomy tubes are not made with an inner cannula.

TABLE 34-1 Some of the Commonly Used Tracheostomy Tubes

	Bivona Mid-Range Aire-Cuf		Portex Flex Disposable Inner Cannula (DIC)		Shiley Single-Cannula Tube (SCT)	
ID (mm)	OD (mm)	Length (mm)	OD (mm)	Length (mm)	OD (mm)	Length (mm)
6.0	8.8	67	8.2	64	8.3	67
7.0	10.0	80	9.6	70	9.6	80
8.0	11.0	89	10.9	74	10.9	89
9.0	12.3	99	12.3	80	12.1	99
9.5	13.3	105	N/A	N/A	N/A	N/A
10.0	N/A	N/A	13.7	80	13.3	105

ID, inner diameter; N/A, not applicable; OD, outer diameter.

TABLE 34-2 Some of the Commonly Used Shiley XLT Extra-Length Tracheostomy Tubes

	Distal extension tubes		Proximal extension tubes	
ID (mm)	OD (mm)	Length (mm)	OD (mm)	Length (mm)
5.0	9.6	90 (48 distal, 37 radial, 5 proximal)	9.6	90 (33 distal, 37 radial, 20 proximal)
6.0	11.0	95 (49 distal, 38 radial, 8 proximal)	11.0	95 (34 distal, 38 radial, 23 proximal)
7.0	12.3	100 (49 distal, 39 radial, 12 proximal)	12.3	100 (34 distal, 39 radial, 27 proximal)
8.0	13.3	105 (50 distal, 40 radial, 15 proximal)	13.3	105 (35 distal, 40 radial, 30 proximal)

ID, inner diameter; OD, outer diameter.

Tracheostomy tubes are manufactured with and without cuffs (**Figure 34-4**). These high-volume, low-pressure cuffs can be used for extended periods with minimal mucosal injury. **Cuffed tubes are used in patients who are dependent on mechanical ventilation or have a history of aspiration.**

Obturators are solid devices that aid in the smooth insertion of the tracheostomy tube (**Figures 34-3 and 34-5**). An obturator will totally occlude the outer cannula and extend a few millimeters beyond the distal end when placed inside the outer cannula. The smooth tip of the obturator allows the outer cannula to be inserted with minimal effort and prevents the edges of the cannula from getting caught and damaging tissue. The obturator is removed and replaced with a low-profile inner cannula once the outer cannula has been inserted.

ROUTINE CARE

The routine care of the patient with a tracheostomy includes cleaning of the tube, humidification of incoming air, and suctioning of the tracheostomy. Inspired air that bypasses the upper respiratory tract in patients with tracheostomies is not as warm or humidified as air inspired through the nose or mouth. The mucociliary "elevator" becomes impaired when cold dry air is inspired into the trachea and results in thicker secretions. It is important to warm and humidify the inspired air for the patient with a tracheostomy who is in the immediate postoperative period, who is dependent on mechanical ventilation, or who has a history of thick secretions.[4,5]

Care of the tracheostomy must include ensuring adequate cleanliness of the tube. Cleanse the skin site with diluted hydrogen peroxide at a 50% concentration.[5] This can be applied to cotton-tipped swabs or other similar absorbent devices and then to the tube.[5] Keep the skin surrounding the tracheostomy dry between cleanings with tracheal bandages or gauze sponges. **Note the underlying skin condition.** Erythematous or macerated skin can become eroded or infected. Use and teach the patient or their caregiver proper skin-care techniques to ensure skin viability. **Ensure that the tracheal bandages or tape used to secure the tracheostomy in place is not so tight that skin perfusion is compromised.** A good rule of thumb is to have two finger breadths of laxity between the skin and the securing ties.[6]

TABLE 34-3 Some of the Commonly Used Adjustable Flange Tracheostomy Tubes

	Bivona Mid-Range Aire-Cuf Adjustable Neck Flange		Rusch Ultra TracheoFlex Adjustable Flange	
ID (mm)	OD (mm)	Length (mm)	OD (mm)	Length (mm)
6.0	9.2	110	N/A	N/A
7.0	10.6	120	10.4	63
8.0	11.7	130	11.4	88
9.0	12.9	140	12.4	117
10.0	N/A	N/A	13.4	117
11.0	N/A	N/A	14.4	116

ID, inner diameter; N/A, not applicable; OD, outer diameter.

FIGURE 34-1. The tracheostomy tube consists of an outer cannula (*left*) and an inner cannula (*middle*). The inner cannula inserts and locks into the outer cannula (*right*).

SUCTIONING

Suction the tracheostomy when there are thick and tenacious secretions at the tracheostomy lumen or when the patient is having difficulty clearing secretions.[7] Suctioning through the tracheostomy will eliminate debris and infectious agents, improve oxygenation, and prevent atelectasis. Other indications for suctioning include suspicion of airway obstruction, diminished or coarse breath sounds, unexplained decreases in oxygen saturation levels, increased airway pressures, or before and after the tracheostomy tube is changed.[8] Do not suction as part of "routine care" when there are few secretions or if the patient is adequately able to generate enough force to clear the secretions.[9] The suctioning of tracheostomies is often essential to proper pulmonary toilet.[7] **Suctioning can be hazardous.** Known complications from tracheal suctioning include arrhythmias, atelectasis, cardiac arrest, hypotension, hypoxia, infections, tracheal mucosal damage, and vagus stimulation.[8,10]

Suctioning can be very frightening to the patient and must be done with some expediency and professionalism.[7] These patients' anxiety levels are quite high as their ability to breathe may be compromised by secretions. The patient may be hypoxic and may have a decreased ability to communicate freely at baseline.[5]

EQUIPMENT

- Protective clothing (i.e., disposable gowns, gloves, face shields, goggles, shoe covers)
- Bag-valve device
- Flexible multi-eyed suction catheter, less than half the inner cannula diameter
- Saline bullets
- Continuous wall suction
- Pressure regulator to maintain suction pressure

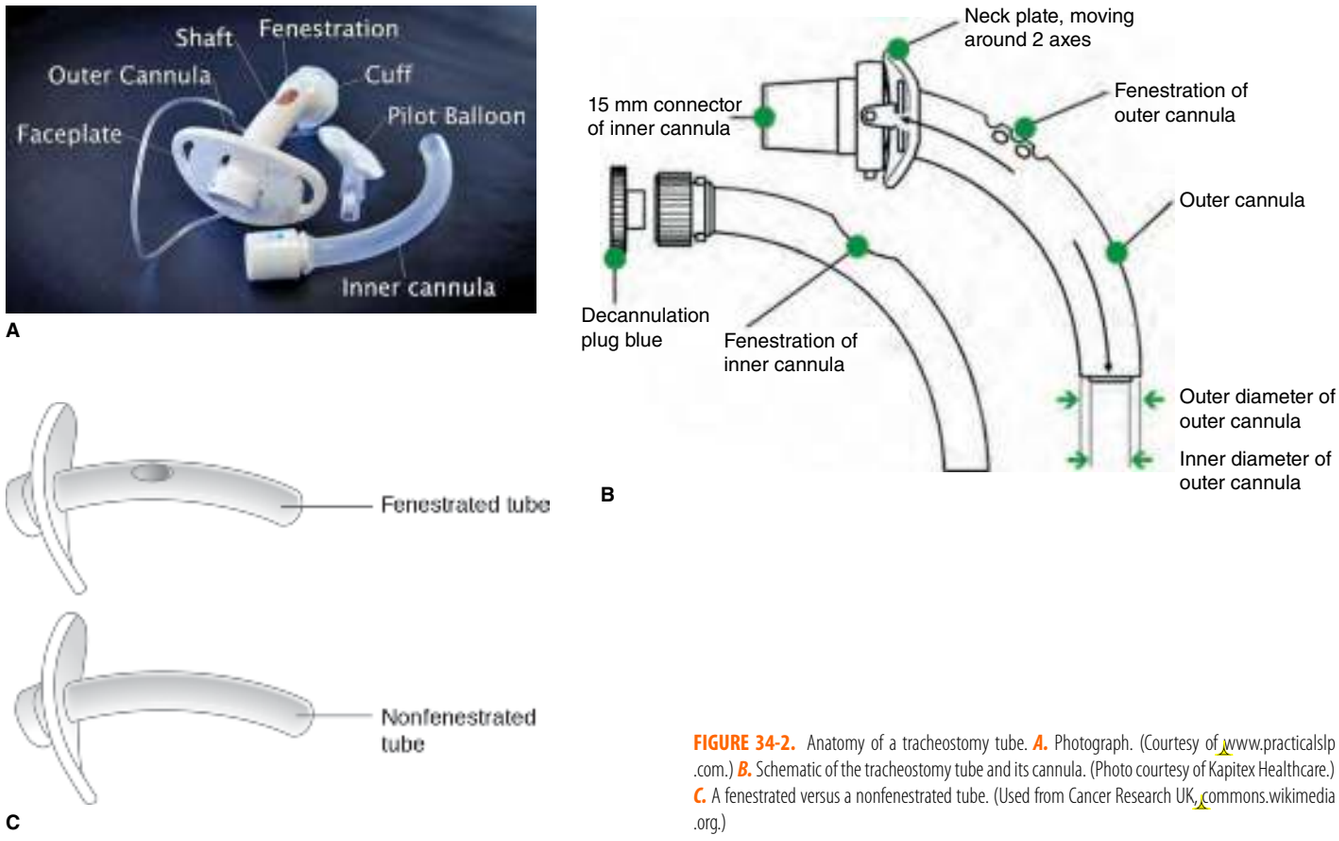


FIGURE 34-2. Anatomy of a tracheostomy tube. ***A.*** Photograph. (Courtesy of www.practicalslp.com.) ***B.*** Schematic of the tracheostomy tube and its cannula. (Photo courtesy of Kapitex Healthcare.) ***C.*** A fenestrated versus a nonfenestrated tube. (Used from Cancer Research UK, commons.wikimedia.org.)

PATIENT PREPARATION

Place the patient in an upright or semirecumbent position. Hyperextend the patient's neck if possible. Preoxygenate the patient with 100% oxygen prior to suctioning. **Hypoxia is a common complication of suctioning and can be virtually eliminated with proper preoxygenation.** Proper preoxygenation often prevents cardiac arrhythmias from occurring during suctioning.[10] Pretreatment with atropine in neonates and children has been suggested to minimize bradycardic episodes.[11]

It has been debated which technique of preoxygenation is best. Options include hand ventilation with a bag-valve device for five to eight breaths, hyperventilation with 100% O_2 via a nonrebreathing mask, or hyperventilation with 100% O_2 via a ventilator. The advantage of hand ventilation is that there is faster delivery of oxygen to the lungs rather than waiting for the higher percentage of oxygen to bleed down the ventilator tubing into the lungs. **Maintaining tidal volumes and positive end-expiratory pressure may be more important.** The bag-valve device may result in decreased cardiac output and hypotension secondary to increased intrathoracic pressures.[8] The method of preoxygenation is up to the Emergency Physician's discretion.

FIGURE 34-3. The pediatric tracheostomy tube and its obturator.

FIGURE 34-4. Tracheostomy tubes may be uncuffed (*left*) or cuffed (*middle*). The inflated cuff is a high-volume, low-pressure system (*right*).

FIGURE 34-5. The obturator is a solid device (*left*) that inserts into the proximal end of the outer cannula and projects from the distal end of the outer cannula (*right*).

TECHNIQUE

Assemble and prepare the equipment. Set the pressure regulator to 60 to 80 mmHg for infants, 80 to 100 mmHg for children, and 100 to 120 mmHg for adolescents and adults. **Higher pressures may cause injury to the tracheal mucosa.** Choose a suction catheter one-half the diameter of the tracheostomy tube. The catheter has an open valve that must be covered to apply suction through the tip of the catheter. Apply cardiac monitoring and pulse oximetry to the patient prior to suctioning if time permits.

Perform the procedure using a clean technique. Wash your hands and apply sterile gloves. The use of a face mask, eye protection, and a gown is highly recommended. The insertion of the suction catheter often induces the patient's cough reflex. Proper protective clothing will prevent the Emergency Physician from being exposed to respiratory secretions.

Gently insert the suction catheter into the trachea (**Figure 34-6**). Advance it approximately 8 to 10 cm until the tip is at the level of the carina. **Do not ever apply suction pressure during insertion of the suction catheter.** Withdraw the catheter approximately 2 to 3 cm and apply suction by placing a finger over the catheter's open valve.[12] **Continue to apply suction as the catheter is simultaneously rotated and withdrawn.** This technique will limit the amount of mucosal damage from the suction catheter. The mucosal surface will invaginate into the holes in the suction catheter tip. The resulting trauma may cause bleeding or erosion of the tracheal mucosal surface if the catheter is not being withdrawn when suction is applied.[9]

Suction the airway for no more than 10 to 15 seconds.[9] This will ensure that the patient experiences a minimal amount of hypoxia. Preoxygenation with 100% oxygen must precede each suctioning episode if the suctioning must be repeated.

The standard use of saline to loosen secretions is somewhat controversial. Some authors have suggested that saline is used to break up thick and tenacious sputum and mucus.[11] There is little support for this assertion. It has been shown that saline instillation increases the cough reflex and stimulates a cough response, which may increase mucus clearance. Others have noted that little saline is recovered with suctioning and that saline itself may cause a gradual decrease in oxygen saturation.[8] The instillation of saline into the tracheostomy cannot be recommended.

FIGURE 34-6. Insertion of a suction catheter. The vent is uncovered during insertion to prevent tracheal mucosal injury.

EMERGENT CARE

Immediate attention must be given when a patient with a tracheostomy presents to the Emergency Department complaining of shortness of breath or respiratory distress. Obstruction and hypoxia are frequent causes of morbidity in this patient population. What follows is a discussion of the algorithm for airway obstruction and respiratory distress in the patient with a tracheostomy.[10]

EQUIPMENT

- Protective clothing (i.e., disposable gowns, gloves, face shields, goggles, shoe covers)
- Bag-valve-mask device
- Flexible multi-eyed suction catheter less than half the inner cannula diameter
- Saline bullets
- Continuous wall suction
- Suction pressure regulator
- Continuous electrocardiogram monitor
- Pulse oximetry
- Tracheal tubes of various sizes, at least the current size of the tube and one smaller
- Water-soluble lubricant
- Tracheal airway kit (e.g., dilator, forceps, and hook)
- Endotracheal tubes
- Laryngoscope handle and blades
- Access to advanced airway equipment, including a fiberoptic scope

PATIENT PREPARATION

The evaluation of any patient with a tracheostomy who is in respiratory distress begins with placing the patient in a room capable of advanced airway management. Place the patient on 100% oxygen and obtain intravenous access, cardiac monitoring, and continuous pulse oximetry. Equipment should be readily available and accessible. This includes endotracheal and tracheostomy tubes of various sizes as well as a laryngoscope and laryngoscope blades.

The Emergency Physician must evaluate the type of tracheostomy tube present. Recognition of the type of tube will aid in the evaluation of possible complications and the management of the respiratory distress. The entire tracheostomy tube may have to be removed for further cleaning after suctioning is performed if the tube has no inner cannula (e.g., pediatric tubes). The patient's respiratory distress may be due to aspiration of secretions or gastric contents if the tracheostomy tube has no cuff.

TECHNIQUE

Inspect the tracheostomy tube for obvious signs of obstruction. The degree of obstruction will increase exponentially as the cross-sectional diameter of the tracheostomy tube decreases. The amount of force required to create airflow through the tube increases dramatically as aspirated material, blood, or dried secretions gather in the inner cannula. Secretions may act as a ball-valve mechanism and allow air to move inward but not outward.

Remove an obvious obstruction or foreign body if it is visible at the tracheostomy tube opening. The Emergency Physician must then suction the patient through the inner cannula using the technique described above. **Keep in mind the importance of preoxygenation.** Remove the inner cannula if this does not adequately relieve the patient's respiratory distress. Inspect it for dried secretions and clean it later if necessary. Suction the patient through the outer cannula. The outer cannula may need to be removed if no diminution of symptoms is noted.

Before removing the outer cannula, it is important to have all the necessary equipment to replace it at the bedside. If the outer cannula has a cuff, deflate it. Remove the outer cannula with a smooth circular motion. Inspect it for a foreign body and dried secretions. The outer cannula may be cleaned and replaced or may be replaced with an entirely new tracheostomy tube. The Emergency Physician may elect to use a fiberoptic scope or red rubber catheter to aid in tube placement. Each of these devices allows the tracheostomy tube to be placed over it and guided into the tracheal lumen.

Remove a relatively new tracheostomy tube (i.e., less than 4 weeks old) over a red rubber catheter (**Figure 34-7**). This will ensure that the tracheostomy tube is inserted into the trachea and not a false passage (**Figure 34-8**). Lubricate the red rubber catheter. Insert the catheter through the outer cannula and into the trachea (**Figure 34-7*A***). Advance the catheter 8 or 9 cm. While holding the catheter securely, remove the outer cannula over the catheter (**Figure 34-7*B***). Lubricate a new outer cannula. Insert the outer cannula over the catheter and gently advance it into the trachea (**Figure 34-7*C***). Remove the catheter and insert the inner cannula into the outer cannula.

A suction catheter can be used as an alternative to a red rubber catheter. **Attach the suction catheter to an oxygen source.** Lubricate the end of the suction tubing. Advance an outer cannula over the distal end of the suction tubing. Insert the suction catheter into the tracheostomy to a depth of 8 or 9 cm. Place a finger over the open valve of the suction catheter to provide oxygen to the patient through the catheter. **This will prevent the patient from becoming hypoxemic during the procedure.** Advance the outer cannula over the catheter and into the trachea. Remove the suction catheter and insert the inner cannula into the outer cannula.

The tracheostomy tube can be replaced manually if no assist device is used (**Figure 34-9**). Lubricate the obturator and insert it into the outer cannula. Inflate the cuff and check its integrity. Deflate the cuff. Lubricate the outer cannula liberally. Place the tip of the

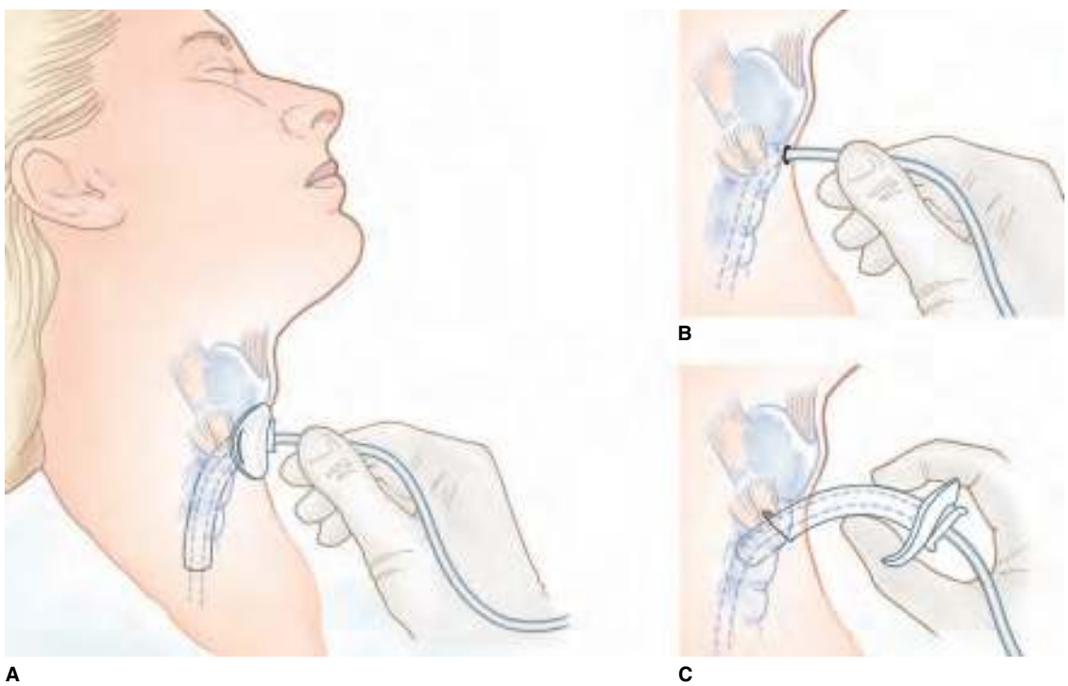


FIGURE 34-7. Removal of the outer cannula over a catheter. ***A.*** The catheter is inserted through the outer cannula to a depth of 8 to 9 cm. ***B.*** The outer cannula has been removed over the catheter. ***C.*** The new outer cannula is inserted over the catheter and into the trachea.

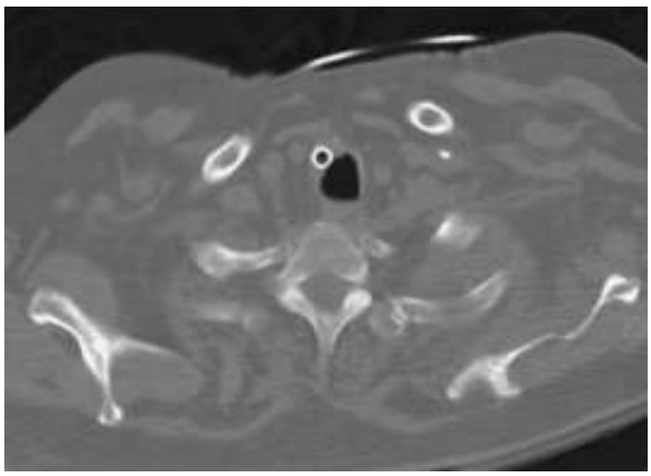


FIGURE 34-8. CT scan image through the torso demonstrating the tracheostomy tube in a false passage outside of the trachea.

obturator perpendicular to the patient's neck and insert it with a semicircular motion (**Figure 34-9*A***). Continue advancing the outer cannula with a semicircular motion as it curves into the trachea (**Figure 34-9*B***). Remove the obturator and insert the inner cannula into the outer cannula. Begin ventilation of the patient if necessary.

Repeat the procedure with a tracheostomy tube one size smaller if the new tracheostomy tube will not advance into the trachea. **Do not force the tube as it can create a false passage** (**Figure 34-8**). Attempt to insert an uncuffed tube if it still will not advance. Alternatively, insert the tracheostomy tube over a catheter (**Figure 34-10**). Lubricate a red rubber catheter (or oxygen catheter) and insert it 8 or 9 cm through the tracheostomy (**Figure 34-10*A***). Insert a lubricated outer cannula over the catheter and into the trachea (**Figure 34-10*B***). Remove the catheter and insert the inner cannula. As a last resort, a tracheostomy hook and Trousseau dilator can be used to lift and open the tracheostomy site to allow the insertion of a tracheostomy tube. Sometimes a tracheostomy tube cannot be passed into the trachea and the patient cannot be oxygenated or ventilated. There is the fear the airway will be lost. Attempt endotracheal intubation.[4] The management is summarized in **Figure 34-11**.

DISLODGEMENT OF THE TRACHEOSTOMY

The tracheostomy tube can accidentally be dislodged.[13] This can result in respiratory distress. Predisposing factors to tracheostomy tube displacement include agitation, coughing, edema of the area, loose ties, and placement problems. Management depends on the timing of the dislodgement relative to the surgery to make the tracheotomy (**Figures 34-12, 34-13, and 34-14**). It is divided into fresh sites (i.e., < 7 days), relatively fresh sites (i.e., 7 to 30 days), and "old" sites (i.e., > 30 days) from the time of surgery. Much of the management has already been discussed above.

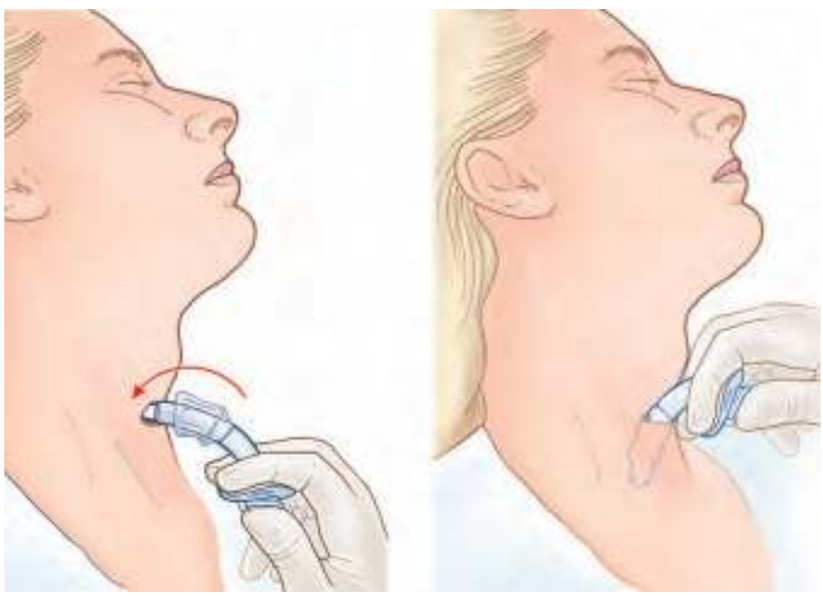
ASSESSMENT

The adequacy of airway maneuvers in the patient with a tracheostomy resides in the patient's response to the interventions. Secure the tracheostomy tube if the patient's pulse oximetry and heart rate return to baseline and the patient appears more comfortable. Take care to ensure adequate skin care beneath the tracheostomy tube site.

Further causes of respiratory distress must be evaluated if a patient remains in respiratory distress despite all appropriate actions. Obtain a chest radiograph. Consult a Pulmonologist for fiberoptic bronchoscopy to evaluate the patient for mucus plugging or foreign-body aspiration. Do not forget to consider other causes of respiratory distress in the patient with a tracheostomy (e.g., heart failure, myocardial infarction, pneumonia, pneumothorax, and pulmonary embolus.)

AFTERCARE

Observe the patient to ensure the stability of the airway. Further suctioning can be performed as required during this time. Educate the patient, their family, and their caregiver about preventive measures regarding tracheostomy care.



A **B**

FIGURE 34-9. Manual insertion of a tracheostomy tube. ***A.*** It is positioned 90° to the tracheostomy site and advanced with a semicircular motion (*arrow*). ***B.*** The system continues to be advanced, following the curve of the tube, until the flange is against the skin.

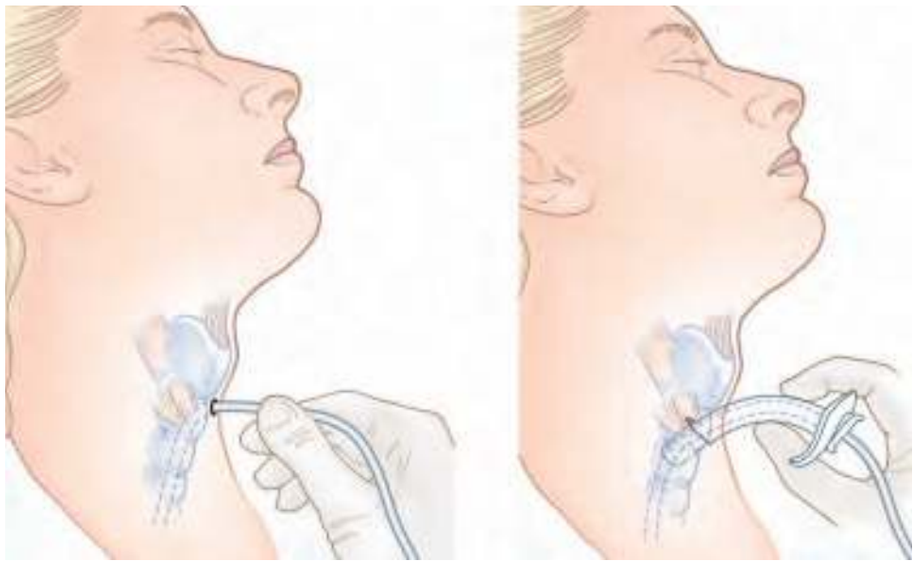


FIGURE 34-10. Insertion of the tracheostomy tube over a catheter. ***A.*** Insert the catheter through the tracheostomy to a depth of 8 to 9 cm. ***B.*** Advance the outer cannula over the catheter.

Evaluate the patient for further conditions that may preclude them from being discharged once the patient's respiratory distress has been addressed. Were there just some dried secretions in the inner cannula? Does the patient have a new source of secretions (e.g., bacterial pneumonia) that could not be managed at home? Are the caregivers at home knowledgeable about the tracheostomy and trained to deal with complications? Admit the patient if there is any question about the patient's ability to deal with further episodes of

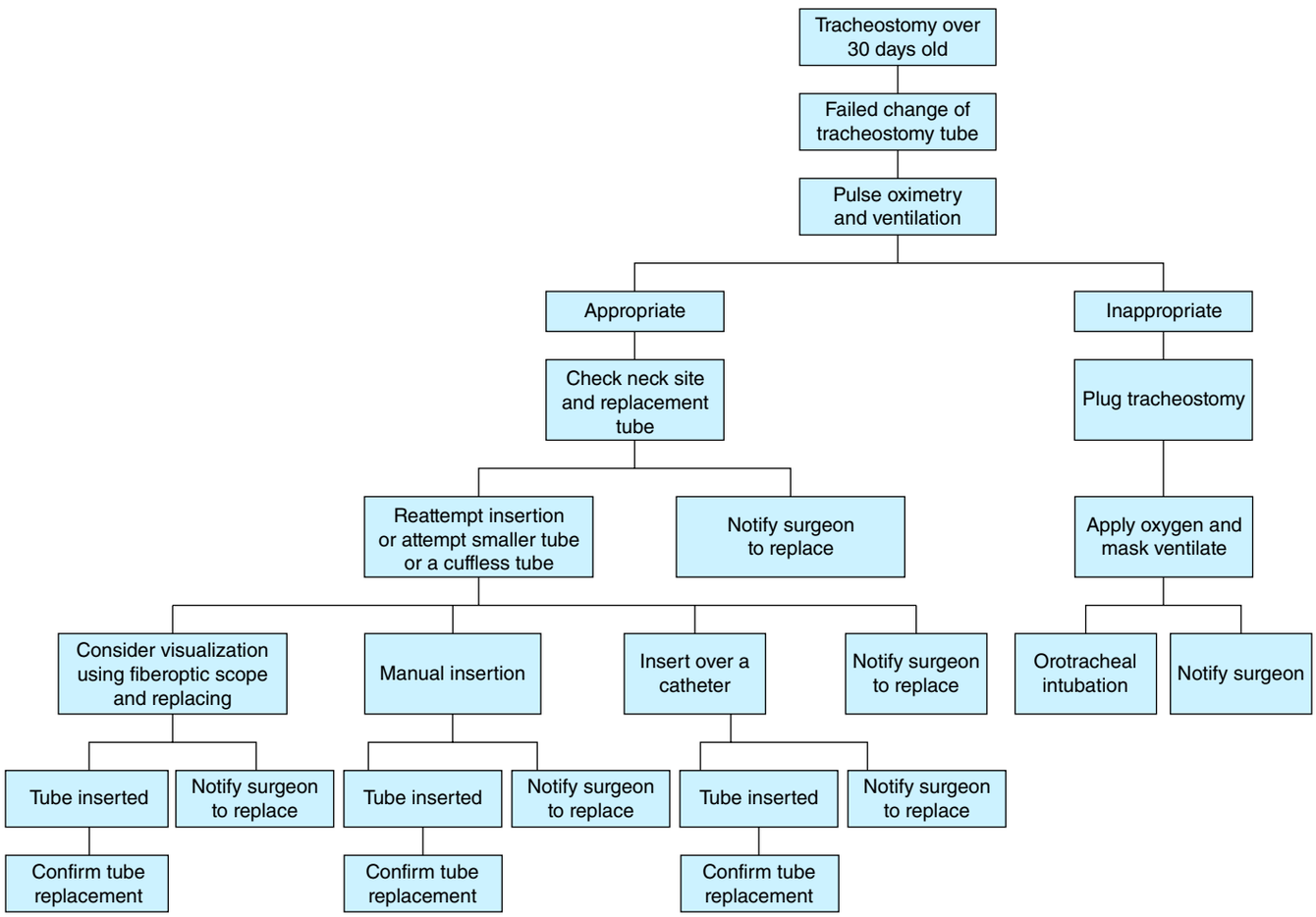


FIGURE 34-11. Algorithm for the steps with a failed tracheal tube replacement.

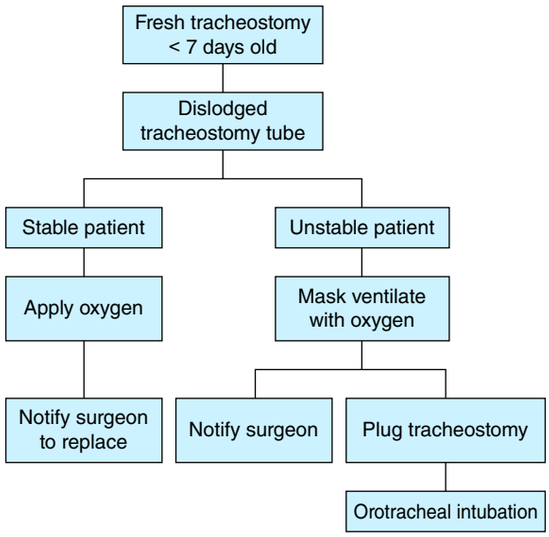


FIGURE 34-12. Algorithm for a dislodged tracheostomy tube < 7 days old.

respiratory compromise. The patient may require skilled home care or a skilled nursing facility to fully care for the tracheostomy.

COMPLICATIONS OF THE TRACHEOSTOMY

Respiratory compromise from plugging of the tracheostomy with secretions may bring the patient to the Emergency Department. Other complications of the tracheostomy may cause the patient to present to the Emergency Department.[14] One retrospective review over a 7-year period showed that 33% of patients presented with dislodged tracheal tubes, 30% presented with infection (one-quarter of these had cellulitis around the tracheostomy; the rest had bronchitis or pneumonia), 18% had plugged tracheal tubes, 11% had bleeding, 5% had tracheal or stomal stenosis, and 3% had a pneumothorax.[2]

BLEEDING

Bleeding is a significant concern in the patient with a tracheostomy.[13] Bleeding at the site of a recent tracheostomy may be a frequent complication. It can be extremely serious. Bleeding can arise from granulation tissue, venous sources, or arterial sources including the great vessels. Tracheoinnominate fistulas are quite rare. They occur in less than 2% of cases, but they carry a mortality rate of up to 50%.[11] They may present as the classic "exsanguinating bleed" but often present with a less impressive sentinel bleed. **Any bleeding of more than a few milliliters of blood should raise concern for a possible fistula of the innominate artery. Prompt critical resuscitation measures and emergent consultation with a Vascular Surgeon and Otolaryngologist are required. Definitive management is surgical.** Techniques for temporarily controlling bleeding from the innominate artery include local digital pressure, hyperinflation of the tracheostomy tube cuff, and traction on the tracheostomy tube. An alternative method is to deflate the tracheostomy tube cuff, reposition the cuff at the bleeding site, and then reinflate or hyperinflate the cuff. **Do not remove the tracheostomy tube until the airway is secured by another means from above (e.g., orally or nasally) when bleeding occurs.**

FREE AIR

Pneumothoraces, pneumomediastinum, and subcutaneous air occur in a small number of patients.[11] Pediatric patients are at a higher risk as the dome of the pleura in a child is closer to the site of the operation. As patients "fight" a ventilator or attempt to inspire against an obstructed airway, they can generate tremendous negative inspiratory pressures. This can result in the dissection of air between the tissue planes and into the thoracic cavity. Small pneumothoraces and pneumomediastinum can be observed as they will most likely resorb with no further complications. A large pneumothorax will require drainage. **The possibility of a tension pneumothorax must always be considered in patients with tracheostomies and respiratory distress or hypotension.**

GRANULOMAS

Tracheal wall granulomas can develop at the tracheostomy site or near the tip of the tracheostomy tube. They are formed in response to mechanical trauma to the mucosa. Granulomas are sometimes a source of bleeding. Direct pressure or cautery may be required for hemostasis. Small granulomas are often observed. Large or symptomatic granulomas require excision.

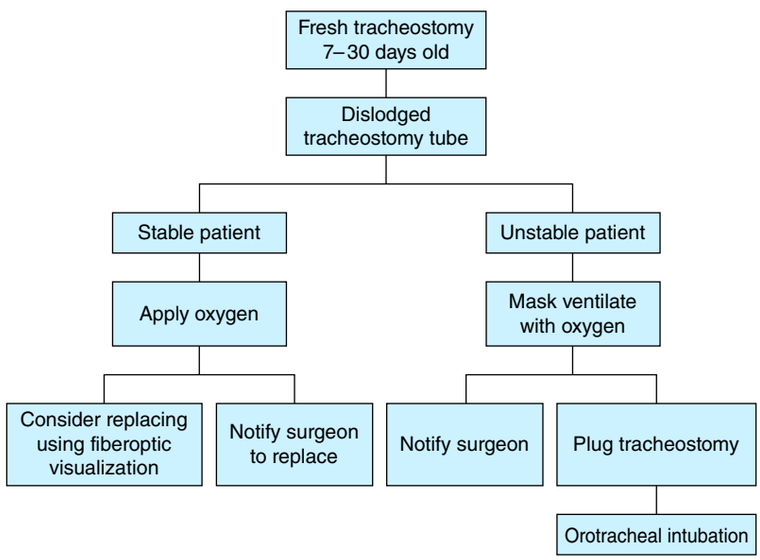


FIGURE 34-13. Algorithm for a dislodged tracheostomy tube 7 to 30 days old.

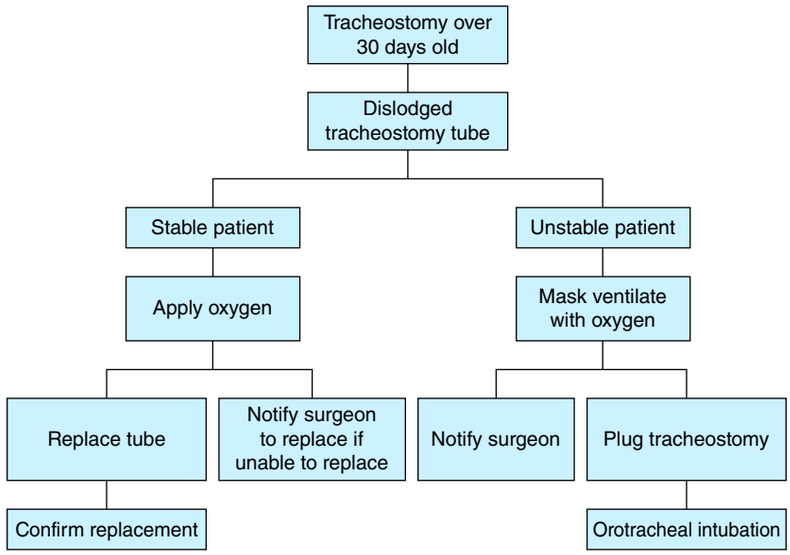


FIGURE 34-14. Algorithm for a dislodged tracheostomy tube > 30 days old.

INFECTIONS

Tracheostomy-related infections can present at any time. Stoma infections are considered local skin infections. Base antibiotic treatment on potential pathogens and local antibiotic resistance patterns. Tracheitis may be bacterial or viral in origin. Common bacterial etiologies include *Staphylococcus aureus*, group A *Streptococcus*, and *Haemophilus influenzae*. Base the determination of a bacterial infection versus colonization on clinical findings and culture results. Management of tracheitis includes broad-spectrum antibiotics for common pathogens, maintenance of a patent airway through suctioning, and possible bronchoscopy.

LARYNGOTRACHEAL STENOSIS

Laryngotracheal stenosis is a complication of long-term endotracheal intubation or direct tissue trauma from the tracheostomy. The tracheal tissues become irritated from the tracheostomy tube. This results in tissue edema that leads to erosions into the mucosa, ulcerations, and eventually scar tissue. Treatment involves surgery to remove the scar tissue and to create an artificial airway to bypass the stenosis.

DEPRESSED SCAR FORMATION

The tracheostomy wound is left open to granulate and heal by secondary intention. This can result in skin atrophy, soft tissue atrophy, and tissue adhesions to the anterior tracheal wall. Scar contracture and a depressed scar can result as the tissue heals. This may result in patient discomfort with head movement or swallowing. Treatment requires surgical scar excision and revision.

SUPRASTOMAL COLLAPSE

Pressure placed on the tracheal cartilages by the tracheostomy tube results in cartilage inflammation, chondritis, and necrosis of the cartilaginous rings. Mild suprastomal collapse usually requires no treatment. Moderate and severe collapse may require a tracheal stent, suturing the anterior trachea to the skin and subcutaneous tissues, or pulling the anterior trachea anteriorly and securing it in place.

TRACHEOESOPHAGEAL FISTULA

A tracheoesophageal fistula is a rare complication of a tracheostomy.[13] The tracheostomy tube can cause pressure necrosis on the posterior tracheal wall that continues to erode into the esophagus. The result is an open tract between the trachea and the esophagus. Patients often have difficulty eating, aspiration pneumonitis, or respiratory difficulty associated with eating and drinking. Treatment requires surgical management.

TRACHEOMALACIA

Tracheomalacia is the lack of support for the trachea by its cartilaginous rings. It can result from degradation of the cartilaginous tracheal rings from the tracheostomy tube or from a tracheoesophageal fistula. Bronchoscopy is required to assess the severity of the tracheal collapse prior to possible intratracheal stenting.

SUMMARY

Emergency Physicians should be familiar with tracheostomy equipment (e.g., the outer and inner cannulae, obturators, and cuff management). Patients presenting to an Emergency Department may require immediate and critical intervention to resuscitate them. Familiarity with various techniques to evaluate the patient with a tracheostomy should include tracheal suctioning, removal of the inner and outer cannulae, replacement of a tracheostomy tube, and evaluation for other emergent conditions relating to tracheostomies. This includes bleeding, infection, and pneumothorax at the very minimum. The Emergency Physician should be mindful of other conditions of the esophagus, trachea, or soft tissues that might complicate the care of the patient with a tracheostomy.

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35 Transtracheal Aspiration

Joseph A. Salomone III

INTRODUCTION

Transtracheal aspiration is a technique for the collection of lower respiratory tract and bronchial secretions for laboratory evaluation and culture.[1,2] This technique is useful when standard sputum collection has not provided adequate material or determination of the infective agent(s). Specimens collected by this technique are free of contamination from nasal, oral, and pharyngeal secretions. This technique was first described in 1959.[3] Several modifications to the original technique have been made.[4-7] This technique may be more properly named transcricothyroid membrane aspiration. It is not used as much as in the past since the development of bronchoscopy. Despite this, it is safe, takes less preparation, takes less personnel, takes less equipment, and is much less expensive when compared to bronchoscopy.

ANATOMY AND PATHOPHYSIOLOGY

The most superficial portion of the cervical airway begins at the inferior thyroid cartilage and extends inferiorly to the thyroid isthmus (**Figure 35-1**). The inferior border of the thyroid cartilage is attached to the cricoid cartilage by the cricothyroid ligament. This is formed by a thicker central conus elasticus and laterally by thinner ligaments that are covered by the cricothyroid muscles (**Figure 35-1*B***). The internal surface is covered by the mucous membrane of the larynx. Collectively, this is often referred to as the cricothyroid membrane or cricovocal membrane. The paired cricothyroid arteries cross from lateral to medial to form an arch that anteriorly crosses the upper one-third of the cricothyroid membrane. The pyramidal lobe of the thyroid occasionally extends superiorly to this level.

INDICATIONS

Transtracheal aspiration is indicated for the collection of tracheobronchial secretions for laboratory evaluation. Often, previous attempts to collect standard coughed and expectorated sputum samples have failed to yield adequate samples or reveal the etiology of a pulmonary infection. Patients who do not appear to be responding to the appropriate antibiotic regimen that was indicated by evaluation and culture of sputum samples may benefit from this technique to better determine the pathogen(s). This is particularly true in cases of atypical or mixed flora, as in suspected aspiration pneumonias, where this technique may yield superior culture results when compared to sputum samples.[8]

CONTRAINDICATIONS

Patients who are unable to cooperate with or tolerate the required positioning should not be selected for this technique.[9] Agitated patients requiring sedation that may affect respiratory effort should be avoided. Traumatically injured patients should have the cervical spine cleared for possible injury prior to performing the procedure. Patients with known or suspected blood dyscrasias (e.g., abnormal platelet counts, elevated prothrombin or partial thromboplastin times) should not be subjected to this technique due to the increased risk of tracheal hemorrhage. The Emergency Physician must be able to easily identify the patient's anatomic landmarks, including the thyroid and cricoid cartilages and the intervening cricothyroid membrane. Patients with abnormal or distorted anatomy should be excluded. Patients who are endotracheally intubated or have a tracheostomy do not require this procedure.

EQUIPMENT

- Sterile gown, gloves, and mask
- Pillow or padding for shoulders

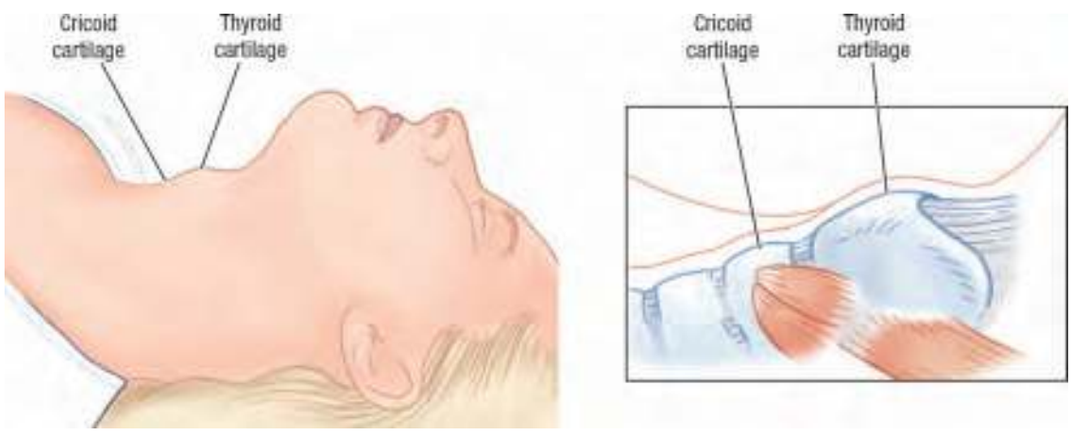

FIGURE 35-1. Anatomy of the airway structures of the neck. ***A.*** Topographic anatomy. ***B.*** The cartilaginous structures.

- Povidone iodine solution or chlorhexidine solution
- Sterile gauze squares
- Normal saline solution, sterile and preservative-free
- Local anesthetic solution, 3 mL (1% lidocaine HCl)
- 3 mL syringes
- 30 mL syringe
- 25 to 27 gauge needles, ½ inch long
- 18 to 22 gauge catheter-through-the-needle, 3 inches long
- 18 to 22 gauge catheter-over-the-needle, 3 inches long
- 18 to 19 gauge needles, 1½ inches long
- Pulse oximeter
- Cardiac monitor
- Sterile specimen container
- Bandage, 1 inch wide
- Sterile drapes or towels
- Resuscitation equipment including emergent airway management supplies

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative. Obtain an informed consent. Place the patient on cardiac monitoring and continuous pulse oximetry. Administer supplemental oxygen and establish intravenous access.

Sedation is not generally required. A small dose of midazolam (1 to 5 mg IV) may be used, if appropriate, for light sedation. Deep sedation should be avoided, as it may compromise respiratory effort and increase the risk of aspiration of gastric contents.

Place the patient supine in bed. Place a pillow or appropriate padding under the patient's shoulders and upper back to allow for comfortable hyperextension of the neck. Identify by palpation the thyroid cartilage, laryngeal prominence (Adam's apple), cricoid cartilage, and cricothyroid membrane. These are the anatomic landmarks that will be used to identify the proper site for performing the procedure.

TECHNIQUE

CATHETER-THROUGH-THE-NEEDLE TECHNIQUE

The operator should follow universal precautions with the use of a mask, eye protection, a sterile gown, and sterile gloves. Using sterile technique, prepare the equipment. Draw 3 to 5 mL of sterile and preservative-free normal saline solution into a 30 mL syringe with a sterile needle. Attach an appropriately sized needle from a catheter-through-the-needle set (18 to 20 gauge for an adult, 20 to 22 gauge for a child) to a 3 mL syringe. An alternative technique is to draw up 1 to 2 mL of sterile saline into the syringe before attaching the catheter-through-the-needle. Draw up 1 to 3 mL of local anesthetic solution into a 3 mL syringe armed with a 25 to 27 gauge needle.

Position the patient as noted above. Using sterile technique, prepare the neck. Clean the anterior neck of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. Place sterile towels or a sterile drape to isolate the anterior neck. Palpate the anterior neck and reidentify the thyroid cartilage, laryngeal prominence, cricoid cartilage, and cricothyroid membrane. Leave the nondominant index finger over the cricothyroid membrane for reference.

Apply a small subcutaneous wheal of local anesthetic solution below the skin at the anterior midpoint of the cricothyroid membrane (**Figure 35-2**). Inject 0.5 to 1.0 mL of local anesthetic solution into the subcutaneous tissues down to the level of the cricothyroid membrane, taking care not to distort the anatomy. Reidentify the cricothyroid membrane by palpation. Insert the needle on the syringe, directed caudally and at a 30° to 45° angle to the skin (**Figure 35-3**). Continue to advance the needle while applying negative pressure to the syringe (**Figure 35-3*A***). Stop advancing the needle when air is aspirated into the syringe. This signifies that the needle is inside the trachea. If using a saline-filled syringe, air bubbles will be clearly visible within the saline. Hold the needle securely and remove the syringe. Insert the catheter through the needle (**Figure 35-3*B***). While holding the catheter securely, withdraw the needle until the tip has exited the skin of the neck (**Figure 35-3*C***). **Place the needle guard over the needle.** This will prevent shearing off of the catheter. Apply the 30 mL syringe containing saline to the catheter (**Figure 35-3*C***).

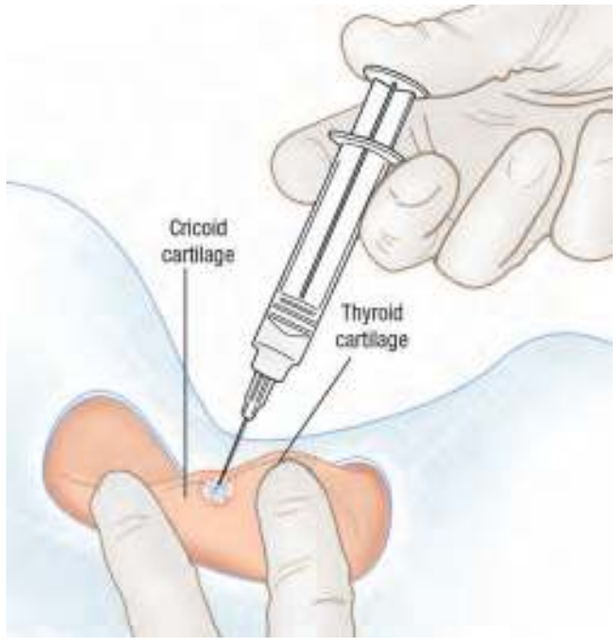

FIGURE 35-2. A subcutaneous wheal of local anesthetic solution is placed over the middle of the cricothyroid membrane.

Ask the patient to cough if they are not already doing so. Aspirate with the 30 mL syringe as the patient coughs. If no specimen is obtained, instill the sterile saline. Once again, ask the patient to cough if not stimulated by the saline. Aspirate until a specimen is acquired. An alternative to using a large syringe for aspiration is the use of low wall suction and a Lukens tube or similar trap device to collect the specimen.

Remove the catheter, needle, and syringe as one unit. Hold direct pressure on the puncture site for 3 to 5 minutes. Apply a bandage or sterile dressing to the puncture site. Place the specimen in a sterile container and have it transported to the laboratory.

Many Emergency Physicians are reluctant to use the catheter-through-the-needle system because there is the possibility of shearing off the catheter within the trachea. **This can be prevented by applying the needle guard over the needle immediately after it is withdrawn from the skin.**

CATHETER-OVER-THE-NEEDLE TECHNIQUE

The more commonly used technique is to use a catheter-over-the-needle (angiocatheter) system (**Figure 35-4**). The operator should

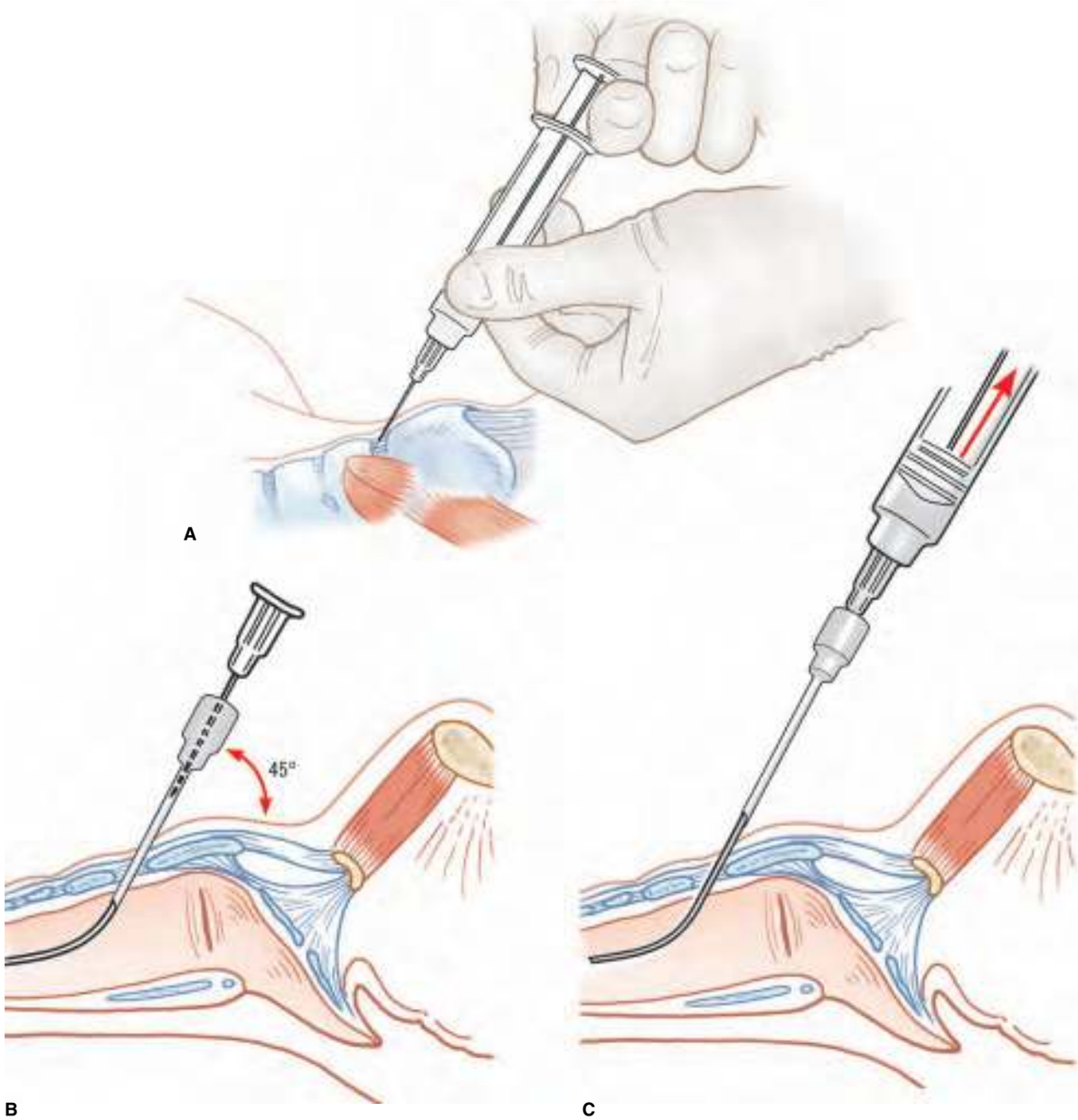

FIGURE 35-3. The transtracheal aspiration technique with a catheter-through-the-needle system. ***A.*** The needle is inserted through the cricothyroid membrane while negative pressure is applied to the syringe. ***B.*** The syringe has been removed and the catheter advanced through the needle. ***C.*** The needle has been withdrawn until its tip exits the skin. A syringe containing saline is attached to the hub of the catheter.

follow universal precautions. Cleanse, prepare, and anesthetize the patient as above.

Using sterile technique, prepare the equipment. Draw 3 to 5 mL of sterile and preservative-free normal saline solution into a 30 mL syringe with a sterile needle. Attach an appropriately sized catheter-over-the-needle (18 to 20 gauge for an adult, 20 to 22 gauge for a child) to a 3 mL syringe. An alternative technique is to draw up 1 to 2 mL of sterile saline into the syringe before attaching the catheter-over-the-needle. Draw up 1 to 3 mL of local anesthetic solution into a 3 mL syringe armed with a 25 to 27 gauge needle.

Reidentify the cricothyroid membrane by palpation. Insert the catheter-over-the needle on the syringe, directed caudally and at a 30° to 45° angle to the skin (**Figure 35-4*A***). Continue to advance the catheter-over-the-needle while applying negative pressure to the syringe. Stop advancing the catheter-over-the-needle as soon as air is aspirated into the syringe. This signifies that the needle is inside the trachea. If using a saline-filled syringe, air bubbles will be clearly visible within the saline (**Figure 35-4*B***). Hold the syringe securely and advance the catheter until its hub is against the skin (**Figure 35-4*C***). Remove the needle and syringe. Apply the 30 mL syringe containing saline to the catheter (**Figure 35-4*D***).

Ask the patient to cough if they are not already doing so. Aspirate with the 30 mL syringe as the patient coughs. If no specimen is obtained, instill the sterile saline. Once again, ask the patient to cough if not stimulated by the saline. Aspirate until a specimen is acquired. An alternative to using a large syringe for aspiration is the use of low wall suction and a Lukens tube or similar trap device to collect the specimen.

Remove the catheter. Hold direct pressure on the puncture site for 3 to 5 minutes. Apply a bandage or sterile dressing to the puncture site. Place the specimen in a sterile container and have it transported to the laboratory.

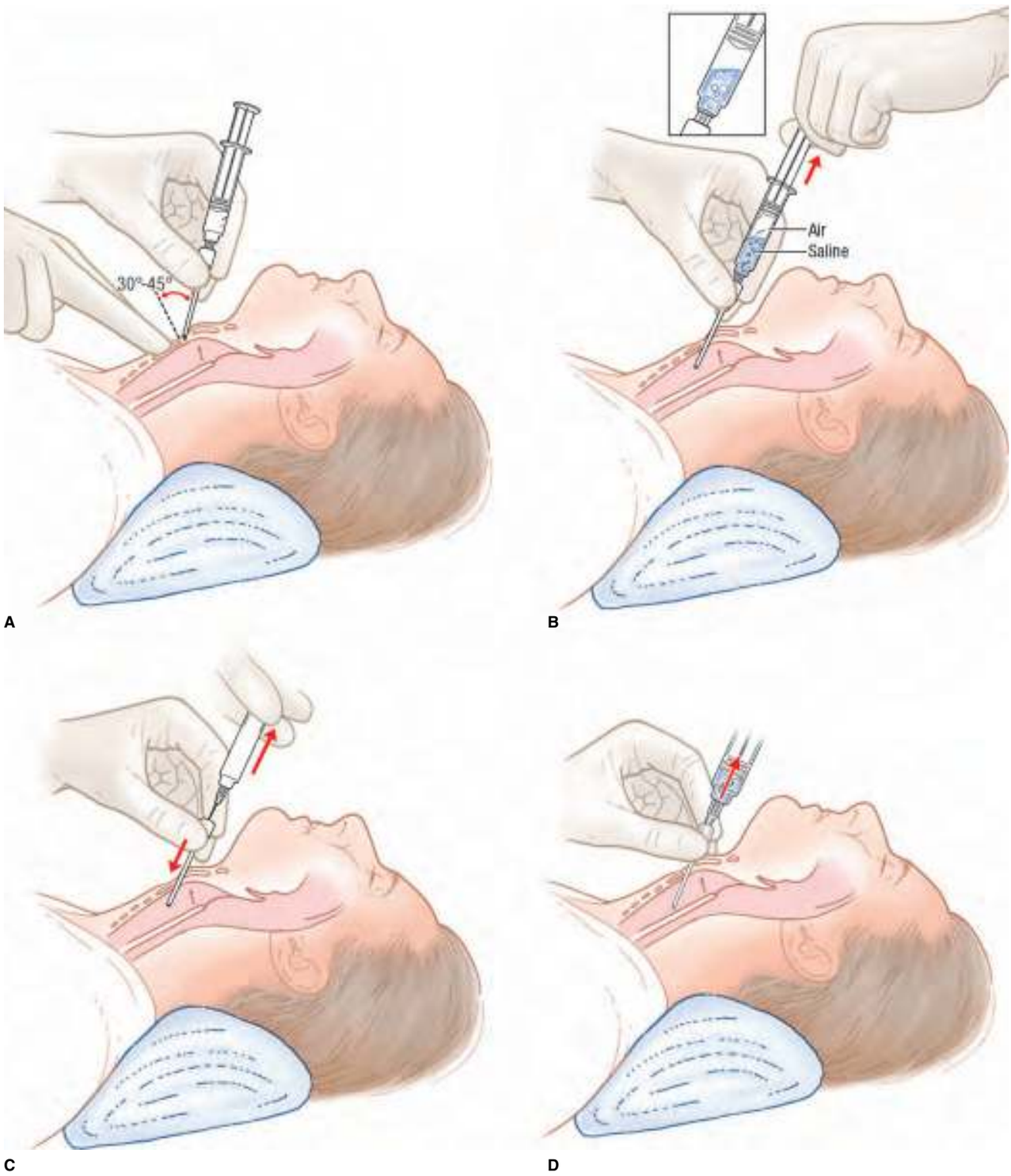

FIGURE 35-4. The transtracheal aspiration technique with a catheter-over-the-needle system. ***A.*** The catheter-over-the-needle is inserted through the cricothyroid membrane. ***B.*** Negative pressure is applied to the syringe. ***C.*** The catheter is advanced until its hub is against the skin. ***D.*** The needle and syringe have been withdrawn. A syringe containing saline is attached to the hub of the catheter.

ASSESSMENT

The patient should be observed for bleeding at the puncture site, the development of subcutaneous emphysema, any changes in sputum production, or hemoptysis. Obtain a chest radiograph immediately after the procedure to look for subcutaneous air and/or a pneumothorax.

AFTERCARE

The patient should remain on continuous pulse oximetry to monitor possible deterioration in respiratory status. Obtain a chest radiograph in 24 hours to look for subcutaneous air and/or a pneumothorax. Any procedure that might stimulate coughing should be avoided for at least 24 hours.

COMPLICATIONS

The complications range from minimal hemoptysis and localized subcutaneous emphysema to massive pulmonary hemorrhage and death.[10-15] There are no major complications if done properly.[1,2] Minimal hemoptysis was seen in 15% of pediatric patients in one study and commonly in several adult studies. Localized subcutaneous emphysema in the anterior neck occurred in 5% to 18% of patients. There are rare reports of fatal endotracheal hemorrhage, profound coughing with development of massive subcutaneous and mediastinal emphysema, vomiting and aspiration of gastric contents, cardiac dysrhythmias, and sudden cardiac death. There is at least one reported case of fatal gastrointestinal hemorrhage from ruptured esophageal varices and Mallory-Weiss tears following "unrestrainable" coughing.

SUMMARY

Transtracheal aspiration is a useful technique for obtaining uncontaminated specimens for analysis and culture. The procedure is best used in patients who have complicated courses or are failing to respond to appropriate treatment or when there is a high index of suspicion for aspiration pneumonia and more atypical infectious agents.

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36 Ventilator Management

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INTRODUCTION

Ventilator management or mechanical ventilation management includes the indication of mechanical ventilation, choosing the ventilator mode, initiation of mechanical ventilation, prevention of complications, the assessment of adequacy of therapy, and readiness for liberation.

Normal inspiration is performed by the expansion of the thorax or chest cavity when the muscles of inspiration contract. Contraction of the diaphragm results in it descending and enlarging the vertical size of the thoracic cavity. The external intercostal muscles contract and raise the ribs slightly to increase the circumference of the thorax. The contraction of these muscles is the "work" required to inspire. The intrapleural pressure becomes more negative during inspiration in relation to atmospheric pressure. This negative intrapleural pressure goes from –5 cmH_2O at the end of expiration to –10 cmH_2O at the end of inspiration. The negative intrapleural pressure is transmitted to the alveolar space. The transpulmonary pressure or the gradient across the lung widens. As a result, the alveoli have a negative pressure during spontaneous inspiration. Pressure at the mouth is still atmospheric. Air flows from the mouth into the alveoli, and the alveoli expand. The volume of gas builds up in the alveoli, and the pressure returns to zero. The airflow stops. This is the end of inspiration, and no more gas moves into the lungs.

Normal exhalation is passive and does not require any work. The muscles relax, the diaphragm moves upward to its resting position, and the ribs return to their normal position. The volume of thoracic cavity decreases and air is forced out of the alveoli. The thoracic volume decreases to resting, and the intrapleural pressure returns to –5 cmH_2O. The pressure inside the alveolus increases during exhalation and becomes slightly positive (i.e., +5 cmH_2O). Pressure is lower at the mouth than inside the alveoli. The transairway pressure gradient causes air to move out of the lungs. Exhalation ends when the pressure in the alveoli and the mouth is equal.

This chapter covers the basics of ventilator management. Many Emergency Departments have ventilators ready to use (**Figures 36-1 and 36-2**). They are applied to the patients who require endotracheal

FIGURE 36-1. Example of a ventilator.

A

B

FIGURE 36-2. Examples of a ventilator monitor screen.

intubation before they are admitted. Refer to a text for more detailed information regarding managing a ventilator.

ANATOMY AND PATHOPHYSIOLOGY

COMPLIANCE

Monitoring changes in compliance is a valuable means of assessing changes in the patient's condition during mechanical ventilation.[1] Compliance usually is measured under conditions of no gas flow. It is referred to as a static compliance or static effective compliance.

RESISTANCE

Resistance, or frictional forces, associated with ventilation is the result of the anatomic structure of the conductive airways and the tissue viscous resistance of the lungs and adjacent tissues.[1] The movement and displacement of structures create resistance to breathing. Tissue resistance remains constant under most circumstances. A patient with lung fibrosis or obesity has increased tissue resistance, but it usually does not change with mechanical ventilation. Tissue resistance increases if a patient develops ascites.

Resistance of the airways is often evaluated during mechanical ventilation. The ability of air to flow through conductive airways depends on the gas density, the length and diameter of the endotracheal tube, the flow rate of the gas through the endotracheal tube, and the gas viscosity. Density, endotracheal tube length, and viscosity remain constant. The diameter of the airway lumen and the flow of gas into the lungs can decrease (e.g., from bronchospasm, increased secretions, mucosal edema, or kinks in the endotracheal tube). The rate at which gas flows into the lungs can be controlled with mechanical ventilation.

More of the pressure for breathing with higher resistance goes to the airways and not the alveoli. This results in a smaller volume of gas available for gas exchange. Another disadvantage of high resistance is that more force must be exerted to get the gas to flow through the obstructed airways. Spontaneously breathing patients use the accessory muscles to generate this increased force. This generates more negative intrapleural pressure and a greater pressure gradient between the upper airway and the pleural space to achieve gas flow. The same occurs during mechanical ventilation. More pressure is exerted by the ventilator to try to move the gas into the patient's lungs through obstructed airways or through a small endotracheal tube.

TYPES OF VENTILATORS

There are two classes of ventilators.[1] There are those that produce breathing patterns that mimic normally respiration. These are called conventional, and their maximum rate is 150 breaths/min. Others produce respiratory patterns at frequencies much higher than we produce for breathing and are called high-frequency ventilators. These ventilators can produce rates up to 900 breaths/min.

CONVENTIONAL VENTILATORS

Conventional ventilators use respiratory patterns that approximate those produced by normal spontaneous breathing. Tidal volumes clear the anatomic dead space during inspiration, and respiratory rates are in the range of normal rates. Gas transport is by convective flow, and mixing in the alveoli occurs by molecular diffusion.

NEGATIVE-PRESSURE VENTILATION

Negative-pressure ventilation mimics the actual function of the respiratory muscles to allow breathing through normal physiologic mechanisms.[1] The best example is the iron lungs. Negative pressure generated around the thoracic area is transmitted across the chest wall, into the intrapleural space, and into the intraalveolar space. As the intrapleural pressure becomes negative, the space inside the alveoli becomes increasingly negative in relation to the pressure at the mouth. This pressure gradient results in movement of air into the lungs. Expiration occurs when the negative pressure around the chest wall is eliminated. The normal elastic recoil of the lungs and chest wall allows air to flow out of the lungs passively.

Negative-pressure ventilators have several advantages. The upper airway can be maintained without the use of an endotracheal tube. The patient can talk and eat. Negative-pressure ventilation has fewer physiologic disadvantages than positive-pressure ventilation. The normal cardiovascular response is not always present in hypovolemic patients to compensate for the negative pressure on the abdomen. This results in the patient having significant pooling of blood in the abdomen and reduced venous return to the heart. Difficulty accessing the patient complicates care activities.

POSITIVE-PRESSURE VENTILATION

Positive-pressure ventilation occurs when a mechanical ventilator moves air into the lungs using an endotracheal tube or mask.[1] The inflating pressure at the upper airway during inspiration equals the sum of the pressures required to overcome the compliance of the lung and chest wall and the resistance of the airways. The pressure in the alveoli during inspiration progressively builds and becomes more positive. Positive alveolar pressure is transmitted across the visceral pleura. This results in the intrapleural space becoming positive at the end of inspiration, and the ventilator stops delivering positive pressure. Mouth pressure returns to ambient pressure while the alveolar pressure is still positive. This gradient between the alveolus and the mouth causes air to flow out.

DEFINITION OF PRESSURES IN POSITIVE-PRESSURE VENTILATION

BASELINE PRESSURE

Pressures are read from a baseline value.[1] The baseline pressure is normally zero or atmospheric. This indicates that no additional pressure is applied at the airway opening during expiration and before inspiration. The baseline pressure is higher than zero when the ventilator pressure is higher during exhalation. This is called positive end-expiratory pressure (PEEP). PEEP prevents the patient from exhaling to zero or atmospheric pressure. PEEP increases the volume of gas in the lungs at the end of exhalation or increases the functional residual capacity. PEEP applied by the ventilator is called extrinsic PEEP. It prevents de-recruitment or collapse of alveoli. The PEEP to prevent de-recruitment is less than the PEEP initially needed to recruit alveoli and keep them open.

Auto-PEEP or intrinsic PEEP is a complication of positive-pressure ventilation. Air is accidentally trapped in the lungs. This occurs when a patient does not have enough time to exhale before the ventilator delivers another breath.

PEAK PRESSURE

The pressure rises progressively to a peak pressure during positive-pressure ventilation.[1] This is the highest pressure recorded at the end of inspiration. Peak pressure is also called peak inspiratory pressure (PIP) or peak airway pressure. The pressures measured during inspiration are the sum of the pressure required to force gas through the resistance of the airways and the pressure of the gas volume as it fills the alveoli.

PLATEAU PRESSURE

The plateau pressure is measured after a breath has been delivered to the patient and before exhalation.[1] Exhalation is prevented by the ventilator for 0.5 to 1.5 seconds. On the ventilator, select inflation hold or inspiratory pause to get the plateau pressure. Plateau pressure measurement is similar to breath-holding at the end of inspiration when the pressure inside the alveoli and mouth is equal with no flow. The relaxation of the respiratory muscles and the elastic recoil of the lung tissues exert a force on the inflated lungs, creating a positive pressure. The reading is stable during a breath-hold and "plateaus." The plateau pressure reflects the effect of the elastic recoil on the gas volume inside the alveoli and any pressure exerted by the volume in the ventilator circuit that is acted upon by the recoil of the circuit.

END EXHALATION PRESSURE

Air can be trapped in the lungs during mechanical ventilation if not enough time is allowed for exhalation. The most effective way to prevent this is to monitor the pressure in the ventilator at the end of exhalation. Air trapping or auto-PEEP is present if no extrinsic PEEP is added and the baseline pressure is greater than the normal baseline.

HOW VENTILATORS WORK

INTERNAL FUNCTION

A ventilator is plugged into an electrical outlet, plugged into a high-pressure gas source, or uses a rechargeable battery. The ventilator operator sets the controls on a control panel (i.e., user interface) to establish the pressure and pattern of gas flow from the ventilator. A control system inside interprets the settings. The control system produces and regulates the desired output.

VENTILATOR COMPONENTS

There are only a few components of a ventilator.[1] A power source or input power (i.e., electrical or gas source) is from electrically powered ventilators, pneumatically powered ventilators, or combined power ventilators. There is a positive- or negative-pressure generator. The control systems and circuits consist of open and closed loop systems to control ventilator function, the control panel or user interface, and the pneumatic circuit. The power transmission and conversion system results in volume displacement using flow control valves. The output from the ventilator controls flow waveforms, pressure, and volume.

POWER SOURCE OR INPUT POWER

The ventilator's power source enables the machine to ventilate the patient. The power source may be electrical power, pneumatic or gas power, or a combination of the two. Electrically powered ventilators rely on electricity. The electrical source may be a battery or a wall outlet. Battery power usually is used for a short time (e.g., transporting a patient). The battery can also be used as a backup power source. Some ventilators use a compressed gas for power. These machines use 50 psi gas sources and have built-in internal reducing valves to allow the operating pressure to be lower than the source pressure

POSITIVE- AND NEGATIVE-PRESSURE GENERATORS

Ventilator gas flow into the lungs is based on different methods of changing the transrespiratory pressure or the pressure at the airway opening minus pressure at the body surface. A ventilator can control pressure at the mouth or around the body. A negative-pressure ventilator generates negative pressure at the body surface that is transmitted to the pleural space and the alveoli. A pressure gradient develops between the airway opening and the alveoli. This results in air flow into the lungs. The volume delivered depends on the pressure difference between the alveolus and the pleural space as well as the lung and chest wall compliance.

Gas flows into the lung with positive-pressure ventilation because the ventilator establishes a pressure gradient generating a positive pressure at the airway opening.

CONTROL SYSTEMS AND CIRCUITS

The control system or control circuit or decision-making system regulates ventilator function. The computer or microprocessor control is the loop system. Most ventilators that are not microprocessor controlled are called open loop or "unintelligent" systems.

The control is set (e.g., tidal volume), and the ventilator delivers that volume to the patient circuit. This is called an unintelligent system because the ventilator cannot be programmed to respond to changing conditions. An open loop ventilator cannot adjust its function to correct for the leakage. It outputs a set volume and does not measure or change it. Closed loop systems are often described as "intelligent" systems because they compare the set control to the measured control. Differences between the two alter the volume delivery. An example of a closed loop system is mandatory minute ventilation (MMV). A minimum minute ventilation that is lower than the patient's spontaneous minute ventilation is selected. The ventilator monitors the patient's spontaneous minute ventilation. If the patient spontaneous minute ventilation falls below a set value, the ventilator increases its output to meet the minimum set minute ventilation.

CONTROL PANEL OR USER INTERFACE

The control panel or user interface is located on the surface of the ventilator. It is set and monitored. The internal control system uses the settings to control the function of the drive mechanism. The control panel has ways for setting alarms, FIO_2, inspiratory time, respiratory rate, and tidal volume. These controls regulate the variables of flow, pressure, time, and volume. The value for each of these can vary within a wide range. Alarms can be set to respond to changes in a variety of monitored variables.

PNEUMATIC CIRCUIT

A pneumatic circuit or pathway is a series of tubes that allow gas to flow inside the ventilator and to the patient. The pressure gradient created by the ventilator generates the flow of gas. The gas flow passes through the pneumatic circuit to the patient. The gas is directed from the generating source inside the ventilator, through the internal pneumatic circuit to the ventilator's outside surface, and through an external circuit to the patient. The gasses are exhaled through an expiratory limb and exhalation valve.

The basic elements of a circuit are simple. The main inspiratory line connects the ventilator output to the patient's airway adapter or connector. The airway adapter is also called a patient adapter or Y connector because of its shape. The expiratory line carries expired gas from the patient to the exhalation valve, which releases exhaled gas into the room.

ADJUNCTS TO THE CIRCUIT

The above essential parts are aided by other components added to the circuit to optimize gas delivery and ventilator function.[1] A device to warm and humidify inspired air is often used. This requires a thermometer to measure the temperature of inspired air. An apnea or low-pressure alarm indicates leaks or if the patient is not ventilating adequately. A nebulizer to power a micronebulizer delivers aerosolized medications. A volume-measuring device determines the patient's exhaled volume. Bacterial filters clear the gas administered to the patient and exhaled by the patient. A pressure gauge measures pressures in the upper airway.

Normal spontaneous breathing provides 100% relative humidity at 37°C and contains 44 mg/L of water (i.e., absolute humidity). Conditioning of inhaled gasses typically occurs down to the fourth or fifth generation of subsegmental bronchi and is called the isothermic saturation boundary. The conditioning of inspired air is normally provided by the nose and upper airway. These structures are bypassed by the endotracheal tube during mechanical ventilation. A humidifier must be added to the ventilator circuit to make up for this loss. The humidification system for the ventilator should provide at least 30 mgH_2O/L of absolute humidity at a range of 31°C to 35°C for all available flows. Some prefer a delivered temperature range of 35°C to 37°C.

Humidity is commonly provided by heated humidification systems. Devices in this category include active heat and moisture exchanger (AHME), passover, vapor phase, and wick humidifiers. Heated humidifiers typically include a servo-controlled heater, a temperature readout, and a temperature alarm. The temperature probe is placed close to the patient airway. The high-temperature alarm is set at 37°C to 38°C so that inspired gas does not exceed 37°C. A minimum alarm setting of 30°C is appropriate. Condensate will accumulate in the circuit when the temperature in the patient circuit is less than the temperature of the gas leaving the humidifier. A cool room environment contributes to the condensate accumulation. Using heated wire circuits on the inspiratory and expiratory lines can significantly reduce this condensation.

The relative humidity in the circuit decreases if the temperature of the gas in the patient circuit is higher than the humidifier. This can happen in heated wire circuits. Drying of secretions can occur if a deficit exists between the amount of humidity provided and the amount needed by the patient. Thick secretions that are hard to suction and the presence of bronchial casts and mucous plugs are signs of drying of the airways.

ALARMS

Alarms warn of possible dangers related to the ventilator system. Low-pressure alarms are usually set at 5 to 10 cmH_2O below peak inspiratory pressure. They detect disconnections and leaks in the system. High-pressure alarms are set at 10 cmH_2O above peak inspiratory pressure and usually end inspiration when activated. They indicate when the patient coughs, if secretions increase, if compliance drops, or if there are kinks in the endotracheal tube or circuit tubing. Low PEEP/continuous positive airway pressure (CPAP) alarms are usually set at 2 to 5 cmH_2O below the PEEP level and indicate when the PEEP or CPAP level has dropped. Apnea alarms are used to monitor mandatory and/or spontaneous breaths. An apnea period of 20 seconds is the maximum accepted. Apnea alarms can be set so the patient will not miss two consecutive breaths. Apnea settings provide full ventilator support for the patient if apnea occurs.

Most ventilators have a ratio alarm or indicator that warns when the inspiratory time is more than half the set cycle time. Some ventilators will automatically shut off at the end inspiration if the expiration time gets so short that the patient does not have time to exhale. Low gas source alarms alert that the available high-pressure gas source is no longer functional. These alarms can be critical for newer microprocessor ventilators that rely on high-pressure gas to function. The alarm cannot be silenced if gas is critical to ventilator operation.

Other ventilator alarms include low tidal volume, low and high expiratory volume, low and high breath rates, and low and high oxygen percentage. There are no predetermined levels for setting these parameters. Use the appropriate judgment when setting alarms to indicate possible changes in patient condition.

FLOW CONTROL VALVES

Modern ventilators use flow control valves to control or direct gas flow by opening and closing completely or in small increments. These valves are motor-based mechanisms. They have a rapid response time and great flexibility in flow control. Flow control valves include proportional solenoid valves, stepper motors with valves, and digital valves with on/off configurations.

GRAPHIC DISPLAYS

Modern ventilators provide graphic displays in loops and waveforms. Loop displays show one variable plotted against another. Waveform displays show flow, pressure, and volume on the vertical axis with time on the horizontal axis.

INDICATIONS FOR MECHANICAL VENTILATION

Respiratory support must start with noninvasive support using the noninvasive ventilation, unless there are clear contraindications for the use of noninvasive methods (e.g., patients with pending respiratory failure). The concept of escalating respiratory support is essential because the invasive respiratory support of extracorporeal membrane oxygenation (ECMO) and mechanical ventilation are associated with major hazards. The indications for intubation and mechanical ventilation initiation are discussed in Chapters 16 and 18.

The main indications for mechanical ventilation include apnea, respiratory distress, hypoxemia, and hypercapnia. It is intuitive to restore ventilation in apneic patients or patients who lost their airway. Patients exhibiting respiratory distress, increased work of breathing, or increased respiratory rate (e.g., the earliest sign of respiratory distress, accessory muscle use, tachycardia, and diaphoresis) face respiratory fatigue and failure. Mechanical ventilation helps to unload the increased work of breathing and stop the vicious circle of acidosis associated respiratory depression. Hypoxemia is defined as the partial pressure of oxygen in arterial blood (PaO_2) < 60 mmHg with PaO_2 values adjusted to patient age. Mechanical ventilation is usually initiated at a fraction of inspired oxygen (FIO_2) of 1.0 or 100%. This high FIO_2 is diagnostic and therapeutic. Patient failure to respond to high FIO_2 points to shunt pathology as the contributor to hypoxemia. **FIO_2 must be weaned as soon as the patient becomes stable.**

Severe hypercapnia is a high carbon dioxide (CO_2) and acidosis that suppresses respiratory muscle contractility. Hypercapnia is defined as a partial pressure of CO_2 (PCO_2) > 50 mmHg. This definition may not be applicable in certain situations as the baseline in partial pressure of CO_2 in arterial blood ($PaCO_2$) in patients with chronic CO_2 retention may exceed 50 mmHg. Permissive hypercapnia may be deliberately instituted to allow for more liberal management in $PaCO_2$ targets. Hypercapnic failure in chronic obstructive pulmonary disease (COPD) must take into consideration the patient's clinical status, the trend in CO_2 level, and the pH decline. $PaCO_2$ levels > 50 mmHg indicates a significant rise in patients with acute hypercapnic failure secondary to muscle disorders (e.g., Guillain-Barré syndrome, myasthenia gravis, and spinal cord injury).[2,3]

CONTRAINDICATIONS TO MECHANICAL VENTILATION

All contraindications are relative. These include a tension pneumothorax and hypovolemic shock. Mechanical ventilation with high tidal volumes and PEEP will hinder the respiratory rate and worsen the hemodynamics. Endotracheal intubation followed by spontaneous breathing is a more appropriate option in hypovolemic patients. Mechanical ventilation will prevent healing of bronchopleural fistulas. Lung isolation of bronchopleural fistulas is recommended when mechanical ventilation is indicated. High PEEP contributes to high intracranial pressure (ICP).[4,5] Right ventricular failure may become worse using mechanical ventilation.[6,7] These relative contraindications may be the reason(s) the patient needs to be intubated and mechanically ventilated (e.g., to manage high ICP, to offload the work of breathing, or to offload the failing heart).

TABLE 36-1 Summary of Techniques

Mode of ventilation	Set variables	Mandatory breath	Spontaneous breath
ACV	Flow Flow shape (constants or decelerating) Respiratory rate Tidal volume	Fixed volume Respiratory rate	The spontaneous breath is similar to preset tidal volume
SIMV	Flow Pressure support Respiratory rate Tidal volume	Fixed volume Respiratory rate	Any respiratory rate with tidal volume varies according to pressure support
PCV	Pressure Respiratory rate Tidal volume	Set pressure Variable tidal volume	Depends on the mode PCV-ACV: same as tidal volume as the mandatory PCV-SIMV: tidal volume varies with the preset pressure support
PSV	Pressure	None	Patient efforts determine the respiratory rate and tidal volume assisted by the preset pressure

TECHNIQUES OF MECHANICAL VENTILATION

Understanding the different techniques of mechanical ventilation requires knowledge of control variables and phase variables (**Tables 36-1 and 36-2**).[8,9]

CONTROL VARIABLES

There are three control variables. These are volume, pressure, and flow. Only one control variable is set and is called the independent variable. The other two variables will be the dependent variables. If the volume is constant (i.e., volume controlled), the pressure varies inversely with lung compliance. If the pressure is set or constant (i.e., pressure controlled), the volume varies in the same direction as lung compliance.

PHASE VARIABLE TRIGGERS

Triggers are what initiates the breath. The inspiratory cycle starts when the triggers are met. There are four main basic triggers. The time trigger is the fixed number of breaths per time unit and is mandatory. Synchronized intermediate ventilation (SIMV) and assist controlled ventilation (ACV or AC) are time-triggered modes. Flow trigger breathing starts when the patient breaths to a predetermined amount of flow. Pressure support ventilation (PSV) is an example of flow-triggered breathing. Pressure trigger breathing starts when

TABLE 36-2 Summary of Ventilator Modes

	Cycled	Initiated	Limitation
Continuous positive airway pressure (CPAP)	Flow	Flow or pressure	Pressure
Pressure control (PC)	Time	Time	Pressure
Pressure-regulated volume control (PRVC)	Volume	Time	Volume
Pressure support (PS)	Flow	Flow or pressure	Pressure
Synchronized intermittent ventilation (SIMV)	Time or volume	Flow, pressure, or time	Volume
Volume control (VC)	Time or volume	Time	Volume
Volume support (VS)	Flow	Flow or pressure	Volume

the patient creates negative pressure in the circuit. Volume trigger breathing starts when the patient breathes in a small volume.

PHASE VARIABLE CYCLES

The cycle is what switches from inhalation to exhalation and vice versa. Volume cycled is determined when the predetermined volume is achieved. The breathing effort stops and exhalation starts. Flow cycled results in stopping of the breathing once flow drops to a predetermined value. Pressure cycled stops the inhalation once the predetermined peak pressure is achieved and exhalation begins. Time cycled is timed inspiration regardless of the flow, pressure, or volume.

PHASE VARIABLE LIMITS

The limit is what stops the breathing. The set value of the limit cannot be exceeded during inspiration. There are limits to flow, minute ventilation, pressure, and volume. An example is during volume-controlled ventilation. If peak airway pressure reaches the pressure limit, the ventilator will stop inspiration and start the expiration phase.

THE DIFFERENT TECHNIQUES OF MECHANICAL VENTILATION

There are four conventional commonly used modes for mechanical ventilation (**Tables 36-1 and 36-2**).[10] These are assisted control ventilation (AC or ACV), synchronized intermittent mandatory ventilation (SIMV) derived from intermittent mandatory ventilation (IMV), pressure control ventilation (PCV), and pressure support ventilation (PSV). Other modes or techniques are not as commonly used but are growing. These include airway pressure release ventilation (APRV), pressure-regulated volume control (PRVC), volume support ventilation (VSV), and proportional assist ventilation (PAV). All are made possible by advances in ventilator software. High-frequency oscillatory ventilation is widely used but not classified as a conventional mode of mechanical ventilation.

ASSISTED CONTROLLED VENTILATION

The machine maintains a constant number of breaths and a constant tidal volume. The patient can trigger the ventilation, and this is variable. The tidal volume remains the same as the ventilator-delivered volume. There is constant flow and constant volume. The variables of airway and plateau pressure vary according to lung compliance. The advantages of ACV mode include the decreased work of breathing. ACV will rest patients with increased work of breathing. ACV provides adequate minute ventilation.

There are many disadvantages to ACV. There is a higher risk of barotrauma. ACV causes patient discomfort because of constant flow throughout the inspiratory cycle compared with PCV, which has a descending flow pattern. **The ventilated patient needs mismatch.** Set the tidal volume below the patient's requirement. Tidal volume hunger results in the patient triggering the ventilator more often and increases the work of breathing. Respiratory alkalosis secondary to mandatory big tidal volumes occurs if the set tidal volume is higher than patient requirements. There is a risk of auto-PEEP secondary to mandatory high tidal volumes, especially in patients with obstructive disorders. Prolonged use of ACV may lead to respiratory muscle weakness and atrophy.

SYNCHRONIZED INTERMITTENT VENTILATION

The ventilator delivers the preset respiratory rate and tidal volume. The patient can trigger the ventilation, and it is variable. The tidal volume varies according to patient effort and the pressure support in each added breath. The triggers are time and flow when the patient initiates the breath. The cycle is timed with volume control. There are limits on flow, pressure, and volume.

There are numerous advantages to SIMV. It is more comfortable in awake patients or those with minimal sedation. The patient determines the respiratory rate and tidal volume. **SIMV allows a guaranteed minute ventilation if the patient fails to trigger a breath.** The patient-determined tidal volume helps the ventilator-needed mismatch and lowers the risk of respiratory alkalosis compared to ACV. Patients use their respiratory muscles, which lowers the risk of muscle atrophy.

Increased work of breathing occurs if excessive effort is needed during the spontaneous breath and inadequate pressure support is available. There is a risk of hypoventilation if the patient fatigues and the spontaneous breath does not meet the patient's needs combined with the low set mandatory tidal volume.

PRESSURE-CONTROLLED VENTILATION

PCV is the mode that controls pressure with little control of tidal volume and minute ventilation. The higher the set pressure, the bigger is the tidal volume achieved in patients with normal lung compliance. **PCV is mainly used in low-compliance conditions to decrease the risk of barotrauma.** PCV, continuous mandatory ventilation (CMV), IMV, SIMV, and ACV can be pressure controlled. The trigger to PCV is flow or time. The cycle is time controlled. There are limits on flow and pressure. PCV results in better oxygenation with a higher PaO_2 in the patient because of the maintained lung distention during inspiration.[3]

PCV results in a variable tidal volume and fluctuating minute ventilation. Decreased lung compliance results in decreases in the tidal volume with PCV. Increased airway resistance decreases the tidal volume. Increase in tidal volume is seen in fixed inspiration-to-expiration (I:E) ratios.

PRESSURE SUPPORT VENTILATION

PSV is a form of augmented spontaneous breathing. The patient decides their respiratory rate and tidal volume. The tidal volume varies according to the set pressure support. Exhalation is a passive process. **PSV can only be used in patients who can initiate breathing.** Flow triggers the ventilator breathing and cycle. The decrease in flow to 25% of the predetermined value ends inspiration and starts passive exhalation. Time cycling can be used as backup mode. There are limits on flow and volume. **PSV is considered the most comfortable form of mechanical ventilation**, with patients report the most comfort with this method. The patient decides the expiratory flow, inspiratory flow, and respiratory rate.

PSV decreases the work of breathing with adequate pressure support. This manifests as improved tidal volume and a drop in the respiratory rate. PSV is used in preparation for weaning from mechanical ventilation. Patients with adequate tidal volume on minimal pressure support (i.e., <7 mmHg) to overcome the endotracheal tube resistance are potential candidates for mechanical ventilation discontinuation.

PSV risks hypoventilation if the tidal volume drops with poor lung mechanics. PSV is poorly tolerated if the patient has an obstructive lung disorder. It is associated with low tidal volumes and hypoventilation. PSV may lead to patient–ventilator asynchrony. This asynchrony happens when the COPD patient needs a longer time to fill their lungs. The pressure continues to rise in this prolonged inspiratory phase when the patient decides to start expiration.

INITIAL VENTILATOR SETTINGS

Place the patient in a semi recumbent position if no contraindications exist.[11] Provide analgesia, paralysis, and sedation.[12-19] Select the

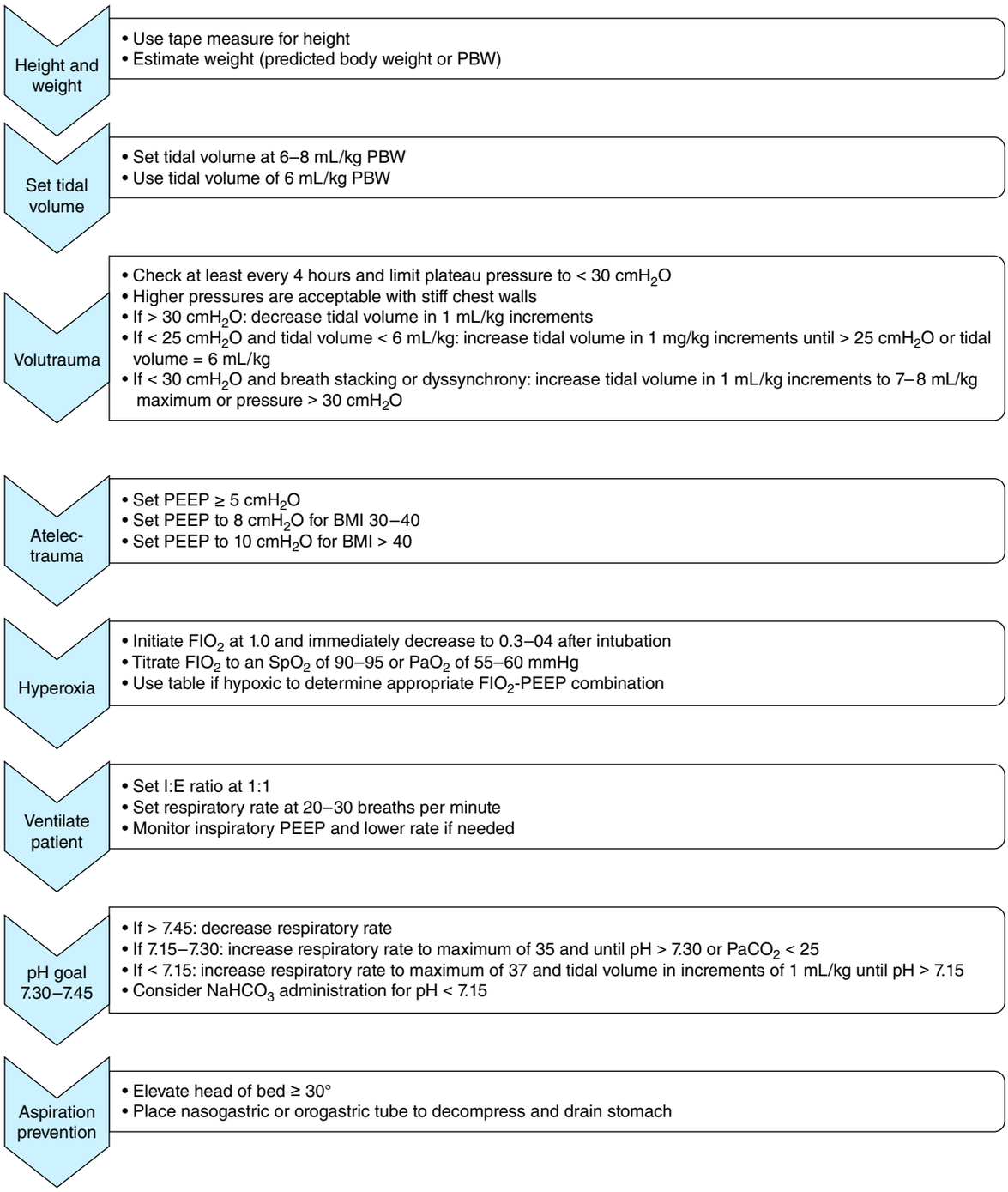


FIGURE 36-3. Example of a protocol for ventilator settings. (Modified from references 20, 21, 23, and 48.)

ventilation mode, FIO_2, flow rate, I:E ratio, PEEP, respiratory rate, and tidal volume (**Figure 36-3** and **Tables 36-3 to 36-5**).[9,20-22] It is a universal recommendation to start at a low tidal volume of 6 to 8 mL/kg.[23] Set the tidal volume lower in acute respiratory distress syndrome to prevent volume-induced lung injury. Healthy adults can be started at 10 to 12 breaths/min. Patients with restrictive lung disorders can be started at rates of 18 to 20 breaths/min to compensate for the low tidal volume ventilation.

Set the flow between 40 and 100 L/min. Lower flow rates will lengthen the inspiratory time and improve alveoli recruitment. Higher flow rates will lengthen the expiratory time and improve lung emptying. The healthy patient spends double the time in expiration than in inspiration. The I:E ratio is usually set at 1:2. Use inverse ratios of 1:1 and 2:1 for alveoli recruitment. The FIO_2 begins at 1.0 or 100% for diagnostic and therapeutic purposes. Titrate the oxygen to prevent hyperoxia and decrease any mortality associated with hyperoxia.[24,25]

PEEP is applied to open the collapsed alveoli, keep alveoli open, increase the functional residual capacity (FRC), and improve oxygenation. PEEP decreases atelectasis and decreases the shear forces by keeping the alveoli open. **PEEP must be titrated to patient physiology.** Add PEEP gradually and assess the effect on compliance and

TABLE 36-3 Initial Ventilator Settings for Common Conditions

Condition	Ventilator mode	FIO_2	I:E ratio	PEEP (cmH_2O)	Respiratory rate/minute	Tidal volume (mL/kg)	Comments
Normal	SIMV-VC	1.0*	1:2	4–5	10–14	8	Do not cause harm. Use lung protection settings. Adjust settings to keep O_2 and CO_2 normal.
Acute respiratory distress syndrome	SIMV-VC	1.0*	2:1	10–15	10–14	6	Watch pressures. Accept higher CO_2. Titrate FIO_2 and PEEP to provide oxygenation. Lower tidal volume as necessary. Recruit alveoli. Avoid atelectasis. Reduce shunting.
Asthma or COPD	SIMV-VC	1.0*	1:4 or 1:5	0–5	8–10	6	Maintain pH > 7.15. Watch for auto-PEEP, barotrauma, breath stacking, and volutrauma. Allow adequate exhalation. May have to accept higher peak pressures. Consider permissive hypercapnia.
Head injury	SIMV-VC	1.0*	1:2	5	14–16	6–8	Avoid high intrathoracic pressure, which reduces venous return. Avoid high PEEP. PCO_2 levels of 35–40.
Hypovolemia	SIMV-VC	1.0*	1:2	0–4	10–12	6	Avoid high intrathoracic pressure, which reduces venous return. Avoid high PEEP.
Metabolic acidosis	SIMV-VC	1.0*	1:1 or 1:2	5	20–30	8–10	Compensate for acidosis with rate. Titrate rate and tidal volume by arterial blood gasses.
Obesity	SIMV-VC	1.0*	1:1 or 2:1	10–15	12–14	8–10	Minimize disconnections and suctioning to prevent atelectasis. Avoid atelectasis and shunting.

*Start at 1.0 and titrate down to 0.4 or a pulse oximetry of 95% or PO_2 > 70; avoid hyperoxia.

TABLE 36-4 The Tidal Volume (mL/kg) for Women Based on Their Height in Inches and Calculated Predicted Body Weight (PBW)

Height (inches)	PBW (kg)	4 mL/kg	5 mL/kg	6 mL/kg	7 mL/kg	8 mL/kg
4′0″ (48)	17.9	72	90	107	125	143
4′1″ (49)	20.2	81	101	121	141	162
4′2″ (50)	22.5	90	113	135	158	180
4′3″ (51)	24.8	99	124	149	174	198
4′4″ (52)	27.1	108	136	163	190	217
4′5″ (53)	29.4	118	147	176	206	235
4′6″ (54)	31.7	127	159	190	222	254
4′7″ (55)	34	136	170	204	238	272
4′8″ (56)	36.3	145	182	218	254	290
4′9″ (57)	38.6	154	193	232	270	309
4′10″ (58)	40.9	164	205	245	286	327
4′11″ (59)	43.2	173	216	259	302	346
5′0″ (60)	45.5	182	228	273	319	364
5′1″ (61)	47.8	191	239	287	335	382
5′2″ (62)	50.1	200	251	301	351	401
5′3″ (63)	52.4	210	262	314	367	419
5′4″ (64)	54.7	219	274	328	383	438
5′5″ (65)	57	228	285	342	399	456
5′6″ (66)	59.3	237	297	356	415	474
5′7″ (67)	61.6	246	308	370	431	493
5′8″ (68)	63.9	256	320	383	447	511
5′9″ (69)	66.2	265	331	397	463	530
5′10″ (70)	68.5	274	343	411	480	548
5′11″ (71)	70.8	283	354	425	496	566
6′0″ (72)	73.1	292	366	439	512	585
6′1″ (73)	75.4	302	377	452	528	603
6′2″ (74)	77.7	311	389	466	544	622
6′3″ (75)	80	320	400	480	560	640
6′4″ (76)	82.3	329	412	494	576	658
6′5″ (77)	84.6	338	423	508	592	677
6′6″ (78)	86.9	348	435	521	608	695
6′7″ (79)	89.2	357	446	535	624	714
6′8″ (80)	91.5	366	458	549	641	732
6′9″ (81)	93.8	375	469	563	657	750
6′10″ (82)	96.1	384	481	577	673	769
6′11″ (83)	98.4	394	492	590	689	787
7′0″ (84)	100.7	403	504	604	705	806

Calculated predicted body weight (kg) = 45.5 + 2.3 (height [in] – 60).
Source: Modified from reference 23.

TABLE 36-5 The Tidal Volume (mL/kg) for Men Based on Their Height in Inches and Calculated Predicted Body Weight (PBW)

Height (inches)	PBW (kg)	4 mL/kg	5 mL/kg	6 mL/kg	7 mL/kg	8 mL/kg
4′0″ (48)	22.4	90	112	134	157	179
4′1″ (49)	24.7	99	124	148	173	198
4′2″ (50)	27	108	135	162	189	216
4′3″ (51)	29.3	117	147	176	205	234
4′4″ (52)	31.6	126	158	190	221	253
4′5″ (53)	33.9	136	170	203	237	271
4′6″ (54)	36.2	145	181	217	253	290
4′7″ (55)	38.5	154	193	231	270	308
4′8″ (56)	40.8	163	204	245	286	326
4′9″ (57)	43.1	172	216	259	302	345
4′0″ (58)	45.4	182	227	272	318	363
4′1″ (59)	47.7	191	239	286	334	382
5′0″ (60)	50	200	250	300	350	400
5′1″ (61)	52.3	209	262	314	366	418
5′2″ (62)	54.6	218	273	328	382	437
5′3″ (63)	56.9	228	285	341	398	455
5′4″ (64)	59.2	237	296	355	414	474
5′5″ (65)	61.5	246	308	369	431	492
5′6″ (66)	63.8	255	319	383	447	510
5′7″ (67)	66.1	264	331	397	463	529
5′8″ (68)	68.4	274	342	410	479	547
5′9″ (69)	70.7	283	354	424	495	566
5′0″ (70)	73	292	365	438	511	584
5′11″ (71)	75.3	301	377	452	527	602
6′0″ (72)	77.6	310	388	466	543	621
6′1″ (73)	79.9	320	400	479	559	639
6′2″ (74)	82.2	329	411	493	575	658
6′3″ (75)	84.5	338	423	507	592	676
6′4″ (76)	86.8	347	434	521	608	694
6′5″ (77)	89.1	356	446	535	624	713
6′6″ (78)	91.4	366	457	548	640	731
6′7″ (79)	93.7	375	469	562	656	750
6′8″ (80)	96	384	480	576	672	768
6′9″ (81)	98.3	393	492	590	688	786
6′10″ (82)	100.6	402	503	604	704	805
6′11″ (83)	102.9	412	515	617	720	823
7′0″ (84)	105.2	421	526	631	736	842

Calculated predicted body weight (kg) = 50 + 2.3 (height [in] – 60).
Source: Modified from reference 23.

TABLE 36-6 The Effect of Changes to the Ventilator Settings

Ventilator changes	Effect on $PaCO_2$	Effect on PaO_2
Increase I:E ratio	None	Increase
Increase FIO_2	None	Increase
Increase peak expiratory pressure	Increase	Increase
Increase peak inspiratory pressure	Decrease	Increase
Increase respiratory rate	Decrease	Increase
Increase tidal volume	Decrease	Increase

oxygenation. Excessive PEEP will lead to lung overdistention with detrimental effects on the lung and hemodynamics.

Adjust the ventilator settings to maintain the $PaCO_2$ and PaO_2 (Table 36-6). The allowable $PaCO_2$ and PaO_2 will vary and depend on the patient's condition. Attempt the following after intubation to decrease mortality: arterial blood gas, capnography, chest radiography, early sedation, gastric emptying, and tidal volumes appropriate for the condition.[26]

COMPLICATIONS OF MECHANICAL VENTILATION

There are many complications associated with mechanical ventilation.[27,28] High tidal volumes can lead to shear stress on the alveoli. Limit the tidal volume to 6 to 8 mL/kg.[29,30] The ventilator can induce diaphragm dysfunction. Select mechanical ventilation modes that encourage patient effort. Attempt early liberation from mechanical ventilation. Ventilator-associated pneumonia is common.[31] Prevent aspiration using head elevation, oral hygiene, and placement of tubes subglottically.[32] Barotrauma can injure the alveoli.[33] Limit the plateau pressure to <35 mmHg and the transpulmonary pressure. Low tidal volumes lead to atelectasis (i.e., atelectrauma). Apply adequate PEEP. Hyperoxia can cause increased mortality.[24] Use the lowest FIO_2 possible to maintain the saturation > 90.

Ventilator alarms may ring, and the patient may worsen. The alarms may be set too low, or the patient may have a problem (**Table 36-7**). First determine if it is a true alarm due to high or low airway pressure. Look for the cause, and correct this if found. The patient on a ventilator may worsen. Use the mnemonics **DOPES** and **DOTTS** to assess for the common reasons for worsening (**Table 36-8**). Look for the cause of patient's worsening status and correct this if found.

The endotracheal tube cuff can be a source of problems. Underinflated cuffs can result in air leaks and aspiration pneumonia. Overinflated cuffs can result in fistulas from the trachea, tracheal mucosal necrosis, and tracheal perforation. The IntelliCuff (Hamilton Medical AG, Switzerland) was developed to provide and maintain appropriate cuff pressure (**Figure 36-4**). This is integral to their ventilators and avoids manual monitoring and adjustment to the cuff. It continuously monitors endotracheal cuff pressure, automatically adjusts the cuff, and decreases complications.[34-38] The IntelliCuff attaches to the inflation port of a standard endotracheal tube and sounds an alarm if the cuff leaks or the endotracheal tube is disconnected.

TABLE 36-7 Troubleshooting Ventilator Alarms

High airway pressure	Low airway pressure
Air trapping	Air escaping chest (drains, tubes, or wounds)
Bronchospasm	Endotracheal tube cuff deflated
Endotracheal tube blocked (biting, cough, gagging, mucous)	Esophageal intubation
Endotracheal tube in main bronchus	Tidal volume set too low
Maximum pressure set too low	Ventilator circuit disconnected
Patient–ventilator dyssynchrony	
PEEP set too high	
Pneumothorax	

TABLE 36-8 Mnemonics for the Crashing Ventilator Patient

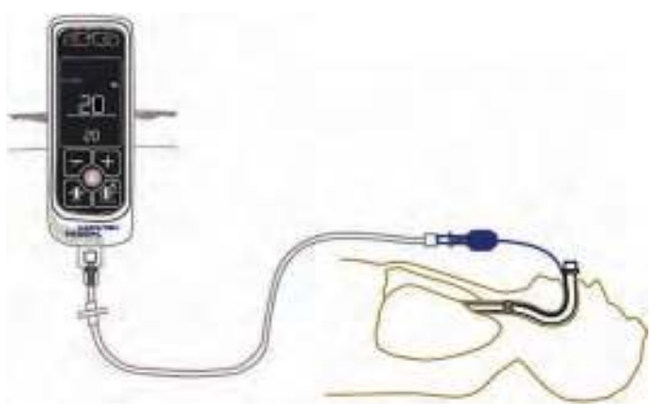
DOPES mnemonic	DOTTS mnemonic
Displaced endotracheal tube or cuff not inflated	**D**isconnect ventilator and bag patient
Obstruction of endotracheal tube	**O**xygenate with bag-valve-mask and feel for resistance
Pneumothorax	**T**ube function and position (check for kinking, migration, or plugging)
Equipment malfunctioning	**T**weak the ventilator settings
Stacking breaths or auto-PEEP	**S**onography (look for mainstem intubation or pneumothorax)

The ventilated patient may become anxious or show signs of respiratory distress (**Table 36-9**).[39] The biggest problems are hypoxia and pain, with hypoxemia being life threatening. Look for the signs of these events and make any corrections needed.

AFTERCARE OF MECHANICAL VENTILATION

Provide sedation to allow synchrony with the ventilator. Sedation must be interrupted for patient neurologic assessment and

A



B

FIGURE 36-4. The IntelliCuff. ***A.*** Control unit on a ventilator. ***B.*** Schematic of the control unit attached to the cuff inflation port. (Photos courtesy of Hamilton Medical AG.)

TABLE 36-9 Causes of Patient Anxiety and Respiratory Distress

Adverse drug effects	Evaluate medications
	Explain interventions
	Limit staffing changes
	Orient to surroundings
	Provide calming and reassurance
Air leak volume loss	Correct air leaks in endotracheal (ET) cuff, ET tube, or ventilator
	Ensure correct tidal volume is delivered
Airway irritation	Bronchospasm, cough, or secretions
	Drainage of tube condensation into the patient's airway
	Suction only as needed
	Thin respiratory secretions
Attempting to speak	Implement communication system (e.g., patient writing)
Biting ET tube	Decreased tidal volume and peak airway pressure alarms
	Insert bite block
	Paralyze and sedate patient
	Try to reason with patient
ET tube displacement or moving	Secure ET tube to patient with holder
	Support ET tube on ventilator arm
	Use caution with movement or turning
ET tube or holder causing discomfort or pressure erosion	Monitor skin breakdown
	Reposition ET tube and holder
	Secure ET tube with holder
Inadequate inspiratory flow rate	Adjust flow rate setting on ventilator to eliminate air hunger
	Manually ventilate patient synchronous with their breaths
Low tidal volume alarm	Decreased lung compliance
	Increased airway resistance
	Pressure limit alarm
Peak airway pressure alarm	Decreased lung compliance
	Increased airway resistance
	Pressure limit alarm
Sleep deprivation	Coordinate interruptions
	Evaluate medications
	Explain interventions
	Limit staffing changes
	Orient to surroundings
	Provide calming and reassurance
	Provide noise cancellation earplugs or headsets
	Relaxation technique implementation

Source: Modified from reference 39.

to assess ventilator liberation. Assess the patient for mechanical ventilation-associated complications. **The goal is to work toward liberation of mechanical ventilation as early as possible.** Some patients may not be ready for ventilator liberation and extubation (**Table 36-10**).[40]

LIBERATION FROM MECHANICAL VENTILATION

Patients are considered for weaning from mechanical ventilation when they pass the three-step approach.[23,41,42] One to two times a day, check if the patient can be liberated from the ventilator. Step 1 is the resolution of the cause of intubation and mechanical ventilation. An example is the patient with upper airway edema. They must have an adequate cuff-leak test before the weaning trial. Patients with an altered mental status or coma must regain their baseline level of consciousness. Patients intubated after hemodynamic instability must exhibit adequate perfusion and somewhat normal vital signs.

Step 2 is stable physiologic conditions. The patient must be hemodynamically stabile (i.e., systolic blood pressure ≥ 90 mmHg) with minimal vasopressor and inotropic support. There should be minimal ventilation support, an FIO_2 of ≤ 0.4 or 40%, pressure support ≤ 5 mmHg, PEEP ≤ 5 mmHg, adequate oxygenation, and a normal

TABLE 36-10 Predictors of Failed Extubation

Airway obstruction	Airway edema
	Airway trauma
	Anasarca
	COPD
	Multiple attempts to secure the airway
	Pneumonia
	Pulmonary edema
	Prolonged intubation
	Reactive airway disease
	Recent upper respiratory infection
	Reduced lung compliance
	Sleep apnea
Failure to oxygenate	Hypoventilation
	Pneumonia
	Pulmonary contusion(s)
	Pulmonary edema
	Ventilation-perfusion mismatch
Failure to ventilate	Abdominal distension
	Malnutrition
	Muscle weakness or wasting
	Residual effects of paralysis and sedation
	Residual muscle weakness
	Splinting from torso pain
Inability to clear respirator secretions	Altered mental status
	Excessive secretions
	Neuromuscular weakness

Source: Modified from reference 40.

work of breathing. The patient should be afebrile. Laboratory analysis should show normal acid-base balance, phosphorus levels, and potassium levels. The patient should be conscious with minimal sedation, have an adequate cough reflex, and have an adequate gag reflex. There should be total reversal of paralysis in the patient who received paralytics.

Step 3 is normal lung mechanics. The patient must have acceptable spontaneous breathing. The patient's respiratory shallow breathing index (RSBI) should be <105 (i.e., respiratory rate ÷ tidal volume). A vital capacity of 10 mL/kg of ideal body weight (IBW) is required. The maximum negative inspiratory pressure should be < −30 mmHg. The thoracic compliance should be > 25 mL/1 cmH_2O. The measured work of breathing should be <0.8 J/L.

Following the three-step approach is essential for success of mechanical ventilation liberation. Reintubation is associated with worse outcomes. A spontaneous breathing trial (SBT) can be initiated for the patient who meets the criteria for extubation. SBT can be performed using T-piece, PSV ≤ 5, or automatic tube compensation. The patient can be extubated in 30 minutes after SBT initiation if the following criteria are met: no abnormal breathing, no diaphoresis, no marked accessory muscle use, no signs of increased work of breathing, no tachycardia, PaO_2 ≥ 60 mmHg, pH of 7.30, respiratory rate <25, RSBI <105, SpO_2 > 90, and spontaneous tidal volume > 4 mL/kg calculated predicted body weight. High-risk airways can be extubated over an exchange catheter. High-risk patients can be extubated to noninvasive ventilation or a high-flow nasal cannula to decrease the risk of reintubation (Chapter 11).[43-47]

SUMMARY

Mechanical ventilation is a lifesaving measure. Prolonged mechanical ventilation is associated with complications and morbidities. A thorough understanding of the indications and different modes of mechanical ventilation is essential for successful application. The common terms used with mechanical ventilation should be known

TABLE 36-11 Definitions of Patient and Ventilator Parameters

- I:E ratio: The relative amount of time of inspiration to expiration. It is usually set between 1:2 and 1:3.
- Fraction of inspired oxygen (FIO_2): The inspired oxygen concentration from 0.21 (i.e., 21% or room air) to 1.0 (i.e., 100%).
- Functional residual capacity: The volume of gas in the lungs at the end of expiration.
- Mandatory breath: The timing or volume of the breath is controlled by the ventilator that triggers the breath.
- Minute ventilation: The volume of gas entering or exiting the lungs per minute. It is normally between 5 and 10 L/min.
- Peak airway pressure: The total (highest) pressure measured in cmH_2O required to deliver the tidal volume.
- Peak flow or peak inspiratory flow: The highest flow or speed set to deliver the tidal volume during inspiration. Higher flow rates deliver the gas faster at shorter inspiratory times.
- PEEP: The positive pressure maintained at the end of expiration to keep the alveoli open.
- Plateau pressure: The pressure, measured in cmH_2O, to distend the lung.
- Respiratory rate: The number of breaths per minute. It is usually set between 10 and 20 breaths/min.
- Spontaneous breath: The patient controls the timing and volume of the breath.
- Tidal volume: The volume of gas during inhalation or exhalation. It is usually set between 6 and 12 mL/kg.
- Trigger sensitivity: The effort required by the patient to trigger a ventilator breath.

Source: Modified from reference 39.

by all Emergency Physicians (**Table 36-11**). Efforts should be focused on mechanical ventilation liberation as soon as the patient meets criteria for safe extubation.

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SECTION 3

Cardiothoracic Procedures

37 Cardiac Ultrasound

Basem F. Khishfe

INTRODUCTION

Cardiac ultrasound (US) or basic echocardiography has been proven to be an invaluable tool for identifying critical pathology and directing decision making in the Emergency Department (ED).[1] Cardiac US is a key element in the evaluation of the trauma patient, the cardiac arrest patient, and the patient with undifferentiated hypotension.[2-6] Even though cardiac ultrasonography can be challenging, an Emergency Physician (EP) can become competent in basic cardiac ultrasonography with practice. The EP must have a keen grasp of the spatial anatomy of the heart to properly perform and interpret the US examination. Cardiac US can help EPs to risk-stratify patients and guide resuscitative efforts. Pericardial tamponade is a difficult diagnosis to make without cardiac US. A quick bedside US can assess a patient's cardiac activity, global cardiac function, presence or absence of an effusion, and volume status. This chapter will cover basic cardiac anatomy, the indications for emergency echocardiography, techniques, and image interpretation.

ANATOMY AND PATHOPHYSIOLOGY

GENERAL ANATOMY AND PHYSIOLOGY

It is essential to know the anatomy of the heart before performing any US examination (**Figure 37-1**). The heart is a three-dimensional organ that lies obliquely in the middle of the chest. It consists of four chambers: the left atrium, the right atrium, the left ventricle, and the right ventricle. The atria are thin-walled muscular structures. The ventricles are more voluminous and muscular, with the left ventricle having the thickest wall. The base of the heart is the superior portion. It is formed by the left atrium and, to a lesser extent, the right atrium. The apex of the heart consists of the inferolateral portion of the left ventricle. The anterior surface of the heart abuts the chest wall and is mainly formed by the right ventricle. The left ventricle forms the majority of the inferior surface, with the inferior portion of the right ventricle making a minor contribution. The heart has two axes, and both are used extensively in ultrasonography.

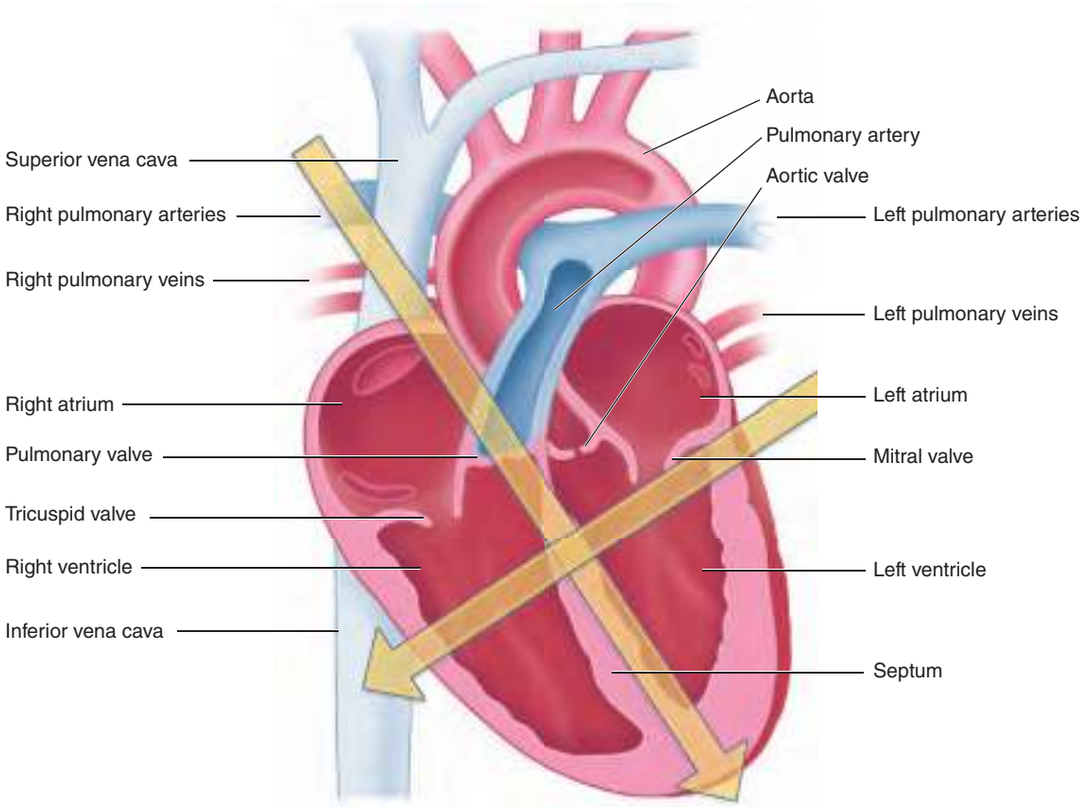

FIGURE 37-1. The cardiac anatomy. The long axis of the heart (*long arrow*) extends from the base to the apex. The short axis (*short arrow*) is a transverse slice perpendicular to the long axis.

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The long axis extends from the base to the apex, roughly along a line from the right shoulder to the left hip. The short axis slices the heart transversely and perpendicular to the long axis, roughly along a line from the left shoulder to the right hip.

The right heart delivers blood to the lungs to be oxygenated, and the left heart distributes it to the rest of the body. The right atrium receives deoxygenated blood from the body via the superior and inferior vena cava (IVC). Blood flows from the right atrium through the tricuspid valve and into the right ventricle during diastole. Blood is pumped from the right ventricle through the pulmonary valve and into the pulmonary artery during systole. The blood gets oxygenated in the lungs and then flows into the left atrium via the pulmonary veins. Blood flows across the mitral valve into the left ventricle during diastole. It is then pumped by the left ventricle for distribution to the body via the aorta during systole.

The heart is contained within the two-layered pericardial sac. The visceral pericardium is a single layer of cells in direct approximation with the epicardium. The tougher outer fibrous parietal pericardium surrounds the visceral pericardium. The two layers form a potential space that contains a small volume of fluid (approximately 20 to 50 mL).[7] This pericardial fluid allows the heart to freely move within the fibrous pericardium. Larger volumes of fluid can collect in this potential space in pathologic states.

ULTRASOUND FOR SUSPECTED PERICARDIAL FLUID

A pericardial effusion is a collection of fluid in the space between the pericardium and epicardium. A variety of conditions can lead to this space being filled by fluid, pus, or blood. It can be due to trauma, uremia, infection, neoplasm, connective tissue disorders, iatrogenic complications, idiopathic conditions, chyle, and numerous other rare causes.[8,9]

The diagnosis of a pericardial effusion should be considered in any patient presenting with shortness of breath, chest pain, and decreased exercise tolerance. Hypotension, distended neck veins, pulsus paradoxus, a pericardial rub on physical examination, and low voltage or electrical alternans on electrocardiogram (ECG) are all consistent with a pericardial effusion. Unfortunately, most of these findings are neither sensitive nor specific for a pericardial effusion or cardiac tamponade.[10] Hence, cardiac US is critical in making the diagnosis. Mandavia and colleagues investigated the ability of EPs to diagnose a pericardial effusion with US.[1] Of the 515 patients enrolled, 103 had pericardial effusion identified by the EPs, for a sensitivity of 96% and a specificity of 98% according to the comparative standard. Cardiac tamponade occurs when the pericardial fluid impedes the ability of the ventricles to fill and leads to decreased cardiac output and cardiovascular collapse.[8,11] Several sonographic signs suggestive of tamponade physiology have been described, although appreciation of it may be subtle. **The most important finding is a circumferential pericardial effusion with a hyperdynamic heart that demonstrates diastolic collapse of the right ventricle or right atrium.**

The amount of fluid necessary to cause tamponade typically depends on how rapidly it accumulates. Fluid that collects more slowly will allow the parietal pericardium to expand accordingly and is generally better tolerated (**Figure 37-2**).[11] Pericardial fluid appears as an anechoic collection (**Figure 37-3**). Small effusions may layer posteriorly or appear as thin black stripes under the pericardium. As effusions increase in volume, they may be seen anteriorly and circumferentially. Pericardial effusions are usually characterized as small, moderate, or large. This is relatively subjective, and grading systems differ. An effusion is considered small if the posterior space measures less than 1 cm, moderate if 1 to 2 cm, and large if more than 2 cm.[12,13] Loculated effusions are usually spherical or lenticular

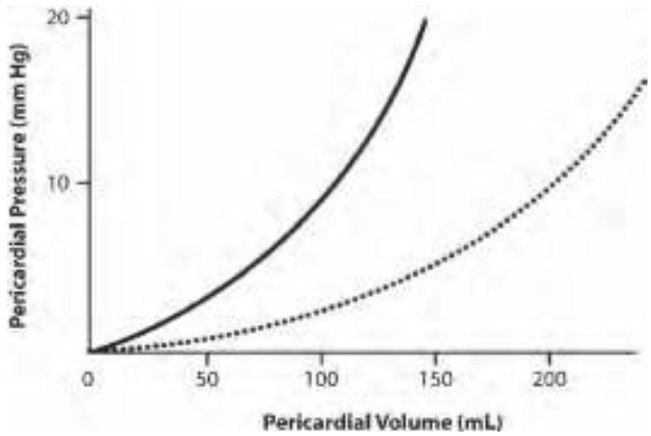


FIGURE 37-2. Pressure-volume relationship of the pericardial space. An acute effusion is represented by the solid line. A chronic effusion is represented by the dotted line. (Used with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

collections with echogenic borders and septae. An epicardial fat pad may be confused for pericardial fluid. It can usually be differentiated because it appears as an isolated thin hypoechoic layer. In comparison, the pericardial fluid generally appears anechoic (**Figure 37-4**).[14]

Cardiac US as part of the FAST examination (Chapter 8) has been used to diagnose hemopericardium in the setting of trauma, particularly penetrating injuries.[2,15] Rozycki et al evaluated the use of US performed by Surgeons, Cardiologists, and Technologists for diagnosing a hemopericardium due to penetrating torso trauma.[2] This study revealed a sensitivity of 100%, specificity of 96.9%, and accuracy of 97.3%. Plummer et al demonstrated that a rapid sonographic evaluation of the heart performed by EPs expedites lifesaving management for victims of penetrating trauma.[15] Both length of time to operative intervention and mortality were significantly improved by ED cardiac US usage.

ULTRASOUND IN CARDIAC ARREST

Cardiac arrest is the end result of numerous different pathologic states. Sonographic asystole is the absence of ventricular contraction.[16] Rare contractions of the atria and/or mitral valve may

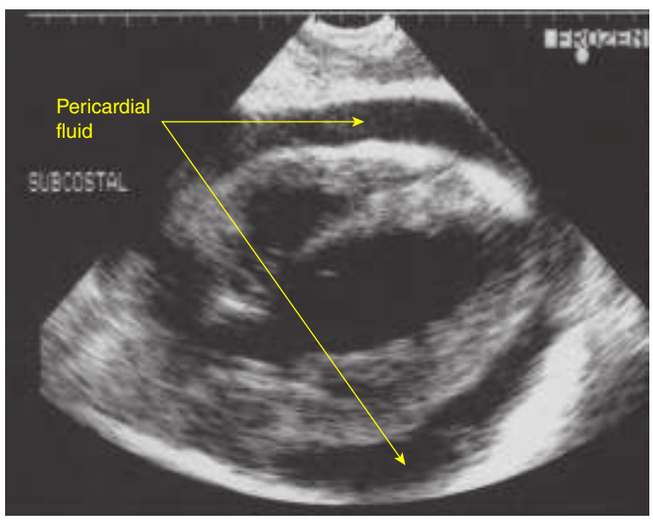


FIGURE 37-3. Free pericardial fluid is visualized as an anechoic stripe surrounding the heart. (Modified with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

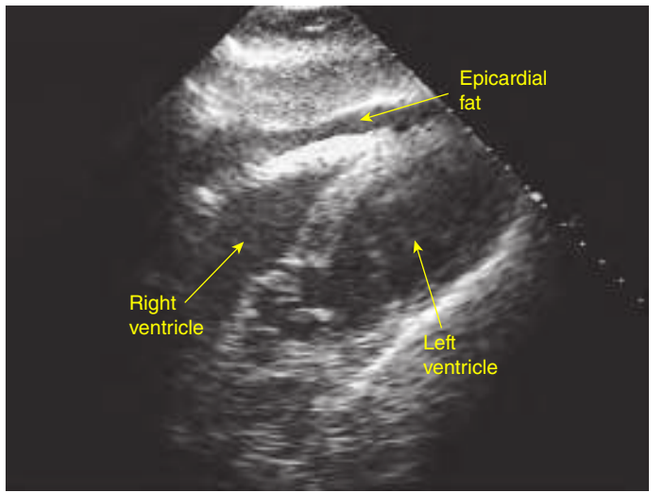


FIGURE 37-4. Subcostal long axis view of the heart demonstrating the epicardial fat pad anterior to the right ventricle. The epicardial fat pad is hypoechoic and not anechoic as usually seen with fluid. (Modified with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

continue despite a terminal event. **It is important to base prognosis on ventricular contractions. Ensure that artificial respirations and compressions are held during the US.** Respiratory effort can occasionally appear as ventricular movement. Pulseless electrical activity (PEA) describes the state in which no pulses are palpable but cardiac electrical activity is visible on ECG monitoring. Cardiac activity may or may not be present upon US of a heart in PEA. Cardiac arrest can occur from nonperfusing tachyarrhythmias and bradycardias. Cardiac motion is present in the case of ventricular fibrillation but is unorganized, preventing adequate cardiac filling and ejection of blood. Severe bradycardia causes the heart to beat at a rate too slow to provide adequate perfusion of the heart and other tissues.

US is useful for guiding the management of patients in cardiac arrest. Confirmation of asystole helps the EP in deciding to terminate resuscitation efforts. Blaivas and Fox, in their study of US in cardiac arrest, suggest that patients who arrive to the ED with cardiac standstill on US have little to no chance of survival.[4]

PEA refers to a spectrum of etiologies that can be visualized with US, from subpalpable hypotension with contracting ventricles to terminal erratic cardiac twitches. In an observational study performed by Tayal and Kline, PEA patients without sonographic cardiac activity all died.[3] Several patients with sonographic motion of the myocardium had reversible causes such as a pericardial effusion. US has value in diagnosing several other etiologies of PEA, including cardiac tamponade,[1] myocardial infarction,[17] cardiogenic shock,[5,6] pulmonary embolism (PE),[18] aortic rupture,[19] hypovolemia, and hemorrhagic shock states.[20]

Circumstance may arise when ventricular fibrillation is not appreciated on the ECG monitor but is diagnosed by US. Fine ventricular fibrillation can look like asystole on the ECG monitor. Ultrasonography of fine ventricular fibrillation appears as rapid trembling of the ventricular myocardium. It appears as a shivering myocardium versus the lack of any motion for asystole.

ULTRASOUND FOR CONGESTIVE HEART FAILURE AND LEFT VENTRICULAR FUNCTION

Congestive heart failure (CHF) is an increasingly common condition that distorts normal cardiac anatomy.[21] Dilation of the left ventricle, decreased ejection fraction, and decreased cardiac output are seen in systolic heart failure.[22] With a loss of healthy contractility and enlargement of the left ventricle, dilation of the other cardiac chambers generally ensues. Diastolic failure is due to prolonged exposure to high cardiac afterload, as seen with aortic stenosis and uncontrolled hypertension.[23] This results in hypertrophy of the left ventricular myocardium. The thickened myocardium cannot relax appropriately to allow ventricular filling during diastole. Diastolic and systolic heart failure both result in a heart that appears globally enlarged. The point of maximal impulse (PMI) is lateralized due to enlargement of the left ventricle from either dilatation or hypertrophy.

Assessment of left ventricular (LV) function is a fundamental application of the cardiac US. It is particularly useful in the presentation of patients with new-onset CHF. The ability to visualize the LV function allows the EP to better manage the patient presenting with chest pain, dyspnea, or unexplained hypotension. It aids in distinguishing CHF from other causes of dyspnea such as chronic obstructive pulmonary disease, pneumonia, pericardial effusion, and pulmonary embolism.[24,25]

Moore et al investigated the use of bedside US for estimations of LV function in hypotensive patients.[26] It was concluded that EPs with focused cardiac US training can make accurate determinations in comparison to primary Cardiologists for patients with normal and severely depressed ejection fraction. Randazzo et al conducted a study in which EPs performed bedside cardiac US examinations on 115 patients.[27] Overall agreement between EP categorization of LV ejection fraction and formal Cardiologist echocardiogram was 86.1%.

Quantitative measurements and complex statistical calculations exist in echocardiography for determining the function of the left ventricle. However, the visual estimate of the global LV function has been shown to be quite accurate.[28] Classification of LV function can be simplified into hyperdynamic, normal, mild to moderately depressed, and severely depressed. The more cardiac USs performed, the more comfortable the EP becomes categorizing a patient's LV function. A hyperdynamic heart may suggest hypovolemia or vasodilation and should prompt the EP to volume resuscitate the patient. However, it is imperative to differentiate a hyperdynamic heart from one that is tachycardic with a normal ejection fraction. Hypokinetic contractions appear stiff. Patients with impaired LV function generally have dilated cardiac chambers, limited contractility, and limited inward movement of the ventricular walls during systole (**Figure 37-5**).

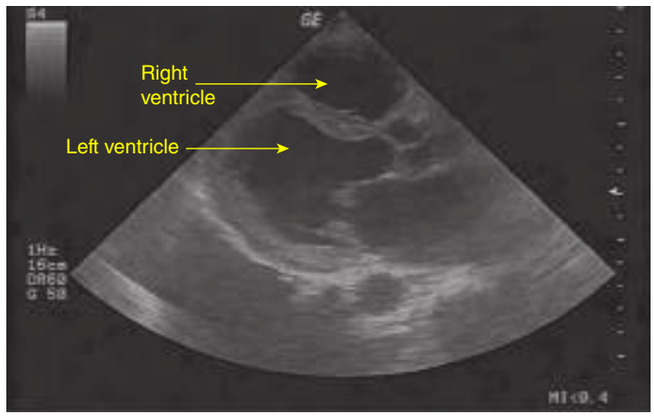


FIGURE 37-5. Parasternal long axis view of the heart in a patient with CHF. The enlarged left ventricle has limited contractility and a poor ejection fraction.

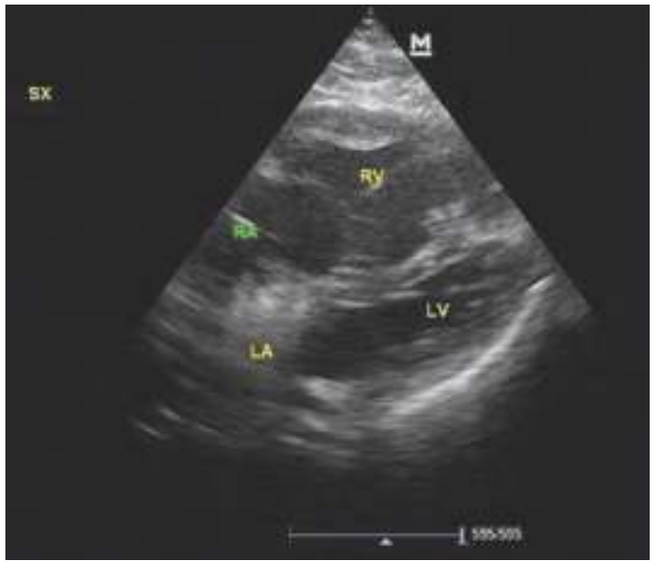


FIGURE 37-6. Concern for a PE is raised by the dilatation of the right ventricle. The diameter of the right ventricle is essentially bigger than the diameter of the left ventricle. LA, left atrium; RA, right atrium.

ULTRASOUND FOR PULMONARY EMBOLISM

Bedside US is not sufficiently accurate for the diagnosis of pulmonary embolism (PE) by itself. There are sonographic clues that may help expedite intervention. US is especially helpful in patients with large PEs and hemodynamic compromise that are often too unstable for conventional diagnostic imaging such as computed tomography and ventilation-perfusion scans.

The thin-walled right ventricle (RV) is extremely sensitive to load, and as such, small changes in pressure could lead to large changes in volume. The RV is smaller than the LV. Signs of RV strain include when the RV is noted to be equal or larger than the LV (**Figure 37-6**) or when the RV bows into the LV septum. In the appropriate clinical setting and when combined with other variables, dilation of the RV or RV straining suggests outflow tract obstruction due to PE.[29,30] Paradoxical bulging of the septum toward the left ventricle during diastolic filling is a sign of right heart strain (**Figure 37-7**).[30] **Always keep in mind that numerous other conditions such as emphysema, pulmonary hypertension, and right ventricular infarction could cause right heart strain**.[24,25] RV strain is poorly sensitive for PE but is reasonably specific.[31] RV strain provides prognostic information in patients with proven PE. The mortality of patients with proven PE and RV strain on US is 10 times the mortality of patients without RV strain.[30,32] Konstantinides et al showed that hemodynamically stable patients with a proven PE and right heart strain on US findings of right heart dysfunction benefited from thrombolytic therapy.[33] The administration of thrombolytic therapy reduced the need for escalation of treatment but did not affect mortality.[33]

ULTRASOUND TO ASSESS UNDIFFERENTIATED SHOCK

The assessment of critical patients with undifferentiated shock is increasingly incorporating information from a number of US applications, including cardiac, inferior vena cava (IVC), thoracic, and FAST images. Cardiac US is one of the most useful studies providing a fast and noninvasive direct view into the cardiovascular problem. A visual picture of the heart and the IVC may quickly establish whether poor perfusion is due to a pump problem (i.e., the heart) or a pipe problem (i.e., the volume). Shock due to heart failure, cardiac tamponade, and PE were described previously. Hypovolemic and distributive shock generally presents with a tachycardic hyperdynamic heart. A variety of algorithms have been proposed for the use of US in the care of the hypotensive patient.[5,34] There is evidence that the algorithms alter patient management in 78% of the cases.[34,35] They result in a more focused differential diagnosis and a more accurate ultimate diagnosis for patients in shock.[6]

US evaluation of the IVC provides a surrogate marker of central venous pressures (CVPs) and predicts fluid responsiveness.[36] The combined results of the cardiac US and the IVC US can help classify the type of shock and help optimize interventions. Although IVC size does not correlate perfectly with CVP monitor readings, a small and collapsed central vein predicts the need of intravascular volume repletion.[37] An IVC diameter of 1.5 to 2 cm is considered normal (**Figure 37-8**).[36] A larger IVC indicates elevated CVP. The diameter of the IVC varies with respiration. Negative intrathoracic pressure produced by inspiration causes the IVC to collapse (**Figure 37-9**). A collapse of 50% is considered normal, with greater collapse indicative of low CVP and negligible collapse indicative of high CVP.[20,36] Simply stated, "fat" great veins are consistent with an elevated CVP and "flat" great veins are consistent with a low CVP. Brief serial examinations during fluid resuscitation can help guide the need for additional fluid.

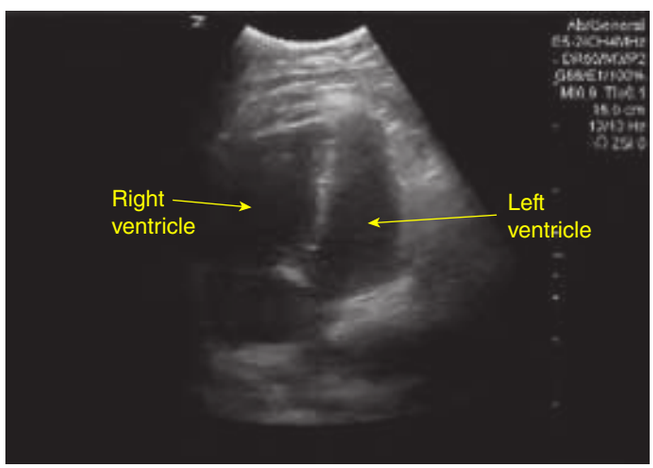


FIGURE 37-7. Right ventricular dilatation and bulging of the septum into the left ventricle is seen in the presence of a large PE.

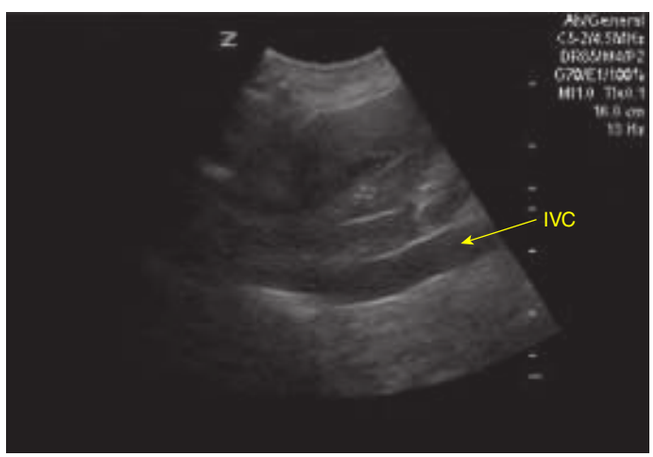
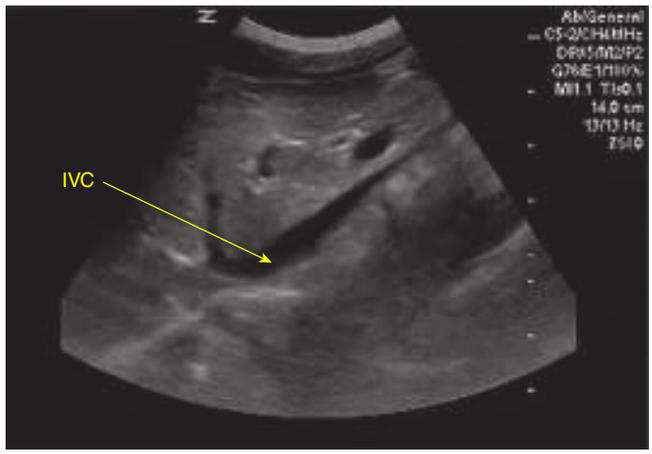


FIGURE 37-8. The normal caliber of the IVC as it passes beneath the liver in caudal-cephalad direction.



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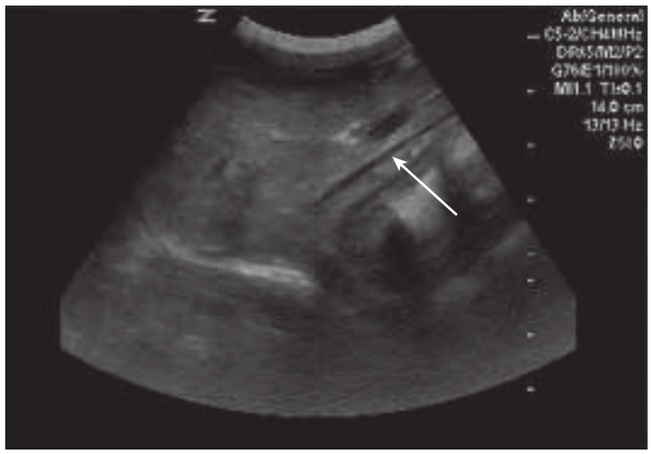

B

FIGURE 37-9. Longitudinal view of the IVC. ***A.*** The diameter of the IVC is narrow in this septic patient. ***B.*** Upon inspiration, the IVC collapses nearly completely (*white arrow*).

INDICATIONS

Cardiac US has been shown to be beneficial in providing diagnostic and prognostic information in the ED (**Table 37-1**). The first described applications for cardiac US were identifying pericardial fluid and evaluation of the cardiac activity.[38] These should be the foundation on which further cardiac assessment is built.[34] As comfort with those applications improves, more advanced applications can be performed. Those advanced applications include, but are not limited to, the assessment of chest pain, global and left ventricular contractility (i.e., hypercontractile, normal, or decreased), myocardial wall motion abnormalities, RV straining, evidence for PE, assessment of hypotension, and valvular abnormalities (e.g., stenosis, regurgitation, thrombus, or vegetations) (**Figure 37-10**); procedural guidance (e.g., pericardiocentesis and transvenous pacemaker placement); and estimation of CVP by great vein measurements.[17,39-50] Evaluations for cardiac myxomas, cardiac tumors, septal defects, dynamic function, and pediatric echocardiography are other cardiac US considerations.

TABLE 37-1 Indications for Emergency Cardiac Ultrasonography

Primary indications	Advanced indications
Cardiac arrest	Cardiac chamber dilation
Pericardial effusion	Cardiac arrest
Traumatic hemopericardium	Cardiac index
	Cardiac tumors
	Chest pain
	Central venous pressure estimates
	Intracardiac thrombus identification
	Hypotension
	Left ventricular function
	Myocardial wall motion defects
	Pediatrics
	Procedural guidance
	Pulmonary embolus
	Septal wall defects
	Shock
	Thoracic aortic dissection
	Valvular dysfunction
	Valvular vegetations
	Ventricular aneurysms
	Wall motion abnormalities

CONTRAINDICATIONS

US is a noninvasive diagnostic modality. It is contraindicated only if it would delay and negatively impact a clinically obvious need for emergent operative intervention. The cardiac US exam should not be performed unsupervised by providers who have not been adequately trained.

EQUIPMENT

- US machine
- US gel
- Phased array low-frequency US probe
- US probe cover or glove

A complete discussion of US equipment is beyond the scope of this chapter. See Chapter 6 for a more complete discussion. Decisions regarding machines and probes depend on the user, cost, and intended applications. The cardiac US exam can be performed with a good-quality, low-frequency probe. Probes with smaller footprints allow for easier viewing between the ribs. The preferred probe for transthoracic imaging is a small-footprint, low-frequency, phased

FIGURE 37-10. An infectious thrombus (*white arrow*) is located on the anterior leaflet of the mitral valve in this parasternal long axis view.

array or microconvex probe (**Table 6-2**). Curved sequential probes can be used and may produce superior images. The large footprint of these probes can be cumbersome when trying to image between ribs or in a small subxiphoid space.

PATIENT PREPARATION

Little to no preparation is required to perform the cardiac US exam. Wipe any debris and liquids from the patient's skin in the areas to be scanned. Place the US probe in a probe cover or glove if the patient's skin is contaminated with blood, vomit, other body fluids, or other substances that may contaminate or damage the probe.

TECHNIQUES

Echocardiography is a complex field. US is a dynamic tool that is well suited to imaging an organ in motion such as the heart. Much information about cardiac function and flow can be gained from complicated statistical calculation packages, advanced Doppler, and M-mode measurements. This section focuses on the primary views most relevant to the EP with the hope of providing a foundation for more in-depth study of advanced cardiac US techniques in the future.

ORIENTATION INDICATOR

The probe orientation indicator in echocardiography is conventionally set to the right side of the screen. In Radiology, the probe indicator is set to the left side of the screen. Some controversy exists in EM as to which side of the screen to have the probe indicator for cardiac US. Most US machines will set the indicator to the right side of the screen when you choose the cardiac mode, which will flip the screen image 180°. Place the probe with the marker pointing to the patient's left shoulder. However, if you decide to keep the screen marker on the left, as it is in abdominal imaging, then rotate the probe marker 180° to the patient's right hip. **Either way will generate the same image when looking at the screen.** The approach preferred by this author is to set the orientation indicator to the left side of the screen and rotate the probe marker 180°. This orientation will be described here. However, if the other option is preferred, just invert the probe positions described here by 180°.

SUBXIPHOID VIEW

The subxiphoid view is probably the view most commonly used by EPs. It provides visualization of the four heart chambers and allows for a superior evaluation for pericardial fluid (**Figure 37-11**). It can be performed without interruption of cardiopulmonary resuscitation or the insertion of chest tubes and subclavian central venous lines. The subxiphoid view is often the easiest view to incorporate into the FAST exam. This view can be limited in patients with a protuberant abdomen, abdominal pain, abdominal injuries, free air below the diaphragm, and/or nausea.

Place the probe below the sternum at the coastal margin with the probe's marker to the patient's right. The plane of the US probe should be angled to the left chest with the probe pressed nearly flat on the abdomen. Apply a firm amount of pressure to get the US beam below the rib cage (**Figure 37-11*A***). Although the heart lies more to the patient's left, so does the stomach, which contains air that scatters the US beam. It is helpful to move the probe to the right side of the patient and use the liver as an acoustic window into the left chest. Increase the US depth until the posterior pericardial space is completely visualized. Ask the patient to take a deep breath to bring their heart inferiorly and into the scanning plane to improve the image.

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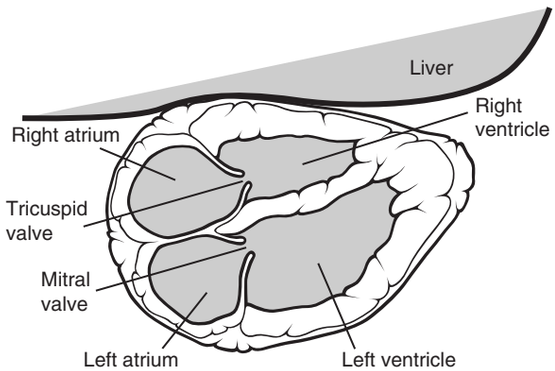

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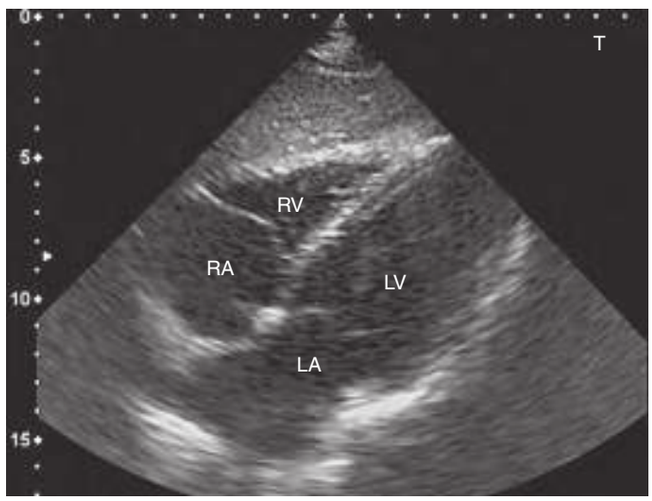

C

FIGURE 37-11. The subxiphoid view. ***A.*** Patient and probe positioning. ***B.*** Diagram of the US image. ***C.*** The US image. LA, left atrium; RA, right atrium. (Used with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

To obtain a complete view of the heart, the US beam depth must be increased beyond that typically used for most other cardiac and abdominal imaging. To grasp the spatial orientation of this view, bear in mind that the probe is aimed from the inferior aspect of the heart. The US beam first traverses the left lobe of

the liver, and hence, liver tissue is seen at the top of the image (**Figures 37-11*B* and *C***). The plane of the beam then slices through the right-sided chambers, the septum, and then the left-sided chambers (**Figures 37-11*B* and *C***). The echogenic pericardium is seen surrounding the myocardium.

PARASTERNAL LONG AXIS VIEW

The parasternal long axis (PSLA) view is usually easier to obtain, provides clearer images, and is better tolerated by the patient. Patients with a heart that is high in the chest or with abdominal distension typically have an excellent PSLA view and a poor subxiphoid view. This is in contrast to patients with hyperinflated lungs (e.g., with chronic obstructive pulmonary disease or on a ventilator) who have a better subxiphoid view than PSLA view.

PSLA view is usually obtained by placing the probe lateral to the sternum in the third or fourth intercostal space (**Figure 37-12*A***). The probe marker will be roughly aimed toward the patient's left hip. The PSLA view usually includes the RV, left atrium, mitral valve, LV, and outflow tract (**Figures 37-12*B* and *C***). The RV sits underneath the probe and is visualized in the near field of the screen (**Figures 37-12*B* and *C***). The LV lies beneath the RV. The interventricular septum is well visualized and extends to the apex of the heart on the left side of the screen. The base of the heart can be visualized on the right side of the screen (**Figure 37-12*C***). The LV empties into the aortic outflow tract, with the aortic valve and root usually visible. The left atrium lies deep, and the mitral valve can be seen opening into the LV (**Figures 37-12*B* and *C***).

The descending aorta may be visualized in a transverse slice along the underside of the heart and is an important landmark in distinguishing pericardial from pleural fluid. Pleural effusions will taper to the descending aorta, whereas pericardial effusions will cross anterior to the descending aorta. This is due to the fact that the pleura inserts where the descending aorta travels through the thoracic cavity. The pericardium is a self-contained space that crosses the midline (**Figure 37-13**). Pericardial fluid collects posteriorly and will appear as a black stripe separating the myocardium from the pericardium and descending aorta (**Figure 37-13**). Pleural fluid, on the other hand, will reside outside of the bright pericardium and tapers to a stop at the descending aorta.

Adjustments on the tilt of the probe may optimize the view. Place the patient in the left lateral decubitus position, if possible, to bring the heart closer to the chest wall and improve the image. Slide the probe laterally, to a more cephalad intercostal space, or open its rotation with a counterclockwise turn to better optimize the view. Move the probe to a more caudal intercostal space, close the angle of the probe with a clockwise rotation, or use the subxiphoid view to better optimize the view for patients with emphysema and an inferiorly displaced heart.

PARASTERNAL SHORT AXIS VIEW

The parasternal short axis (PSSA) view slices through the heart transversely (**Figure 37-14**). The PSSA view provides an excellent circumferential view of the LV and is often used for the assessment of contractility and regional wall motion abnormalities. Place the probe similar to the PSLA view, but with the probe rotated 90° to rest along the heart's short axis (**Figure 37-14**). Aim the marker toward the patient's right hip. The LV appears as a prominent circle in the center of the screen, with the RV resting atop it as a flatter or crescent-shaped chamber (**Figures 37-14*B* and 37-15**). Tilting or sliding the probe along the heart's long axis toward the patient's left hip allows visualization of the apex of the heart. Tilting or sliding the probe toward the patient's right shoulder allows visualization of

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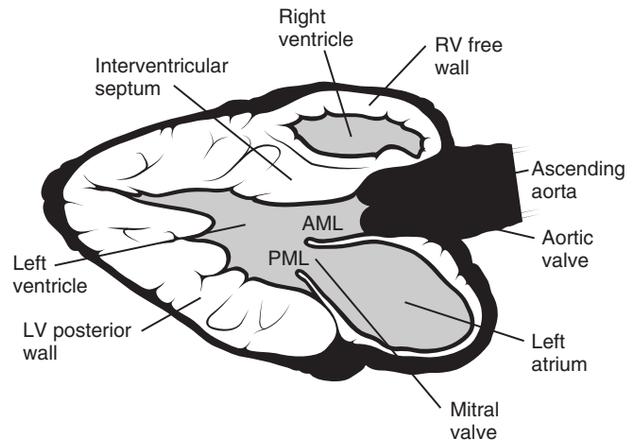

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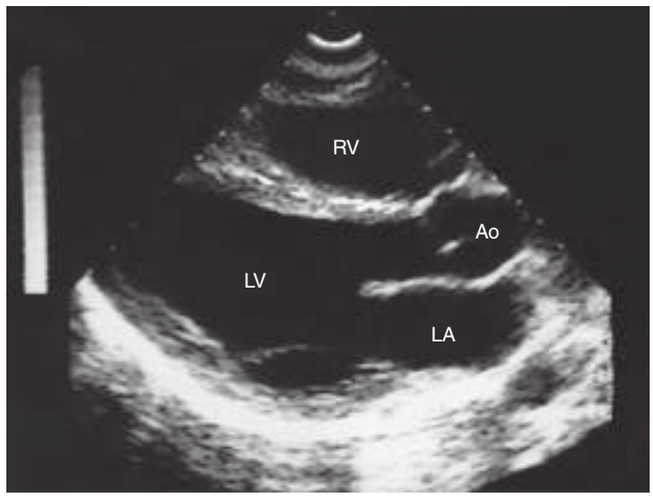

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FIGURE 37-12. The parasternal long axis view. ***A.*** Patient and probe positioning. ***B.*** Diagram of the US image. ***C.*** The US image. Ao, aorta; LA, left atrium. (Used with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

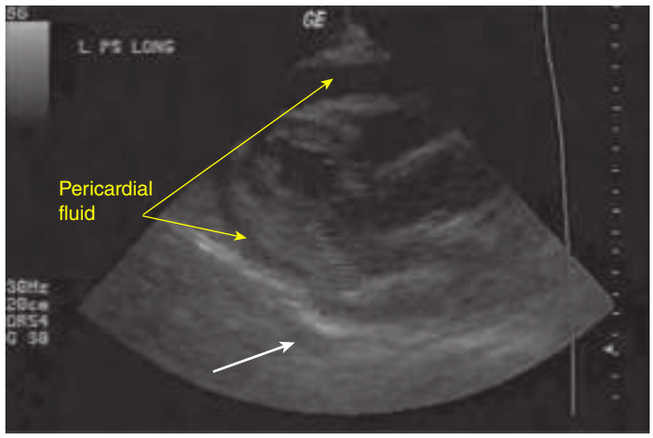

FIGURE 37-13. The parasternal long axis view of a pericardial effusion. Pericardial fluid separates the myocardium from the pericardium and the poorly visualized descending aorta (*white arrow*).

the base of the heart. The ventricles should cone down to a tip at the apex. When tilting back up from the apex through the heart, the papillary muscles (**Figure 37-15**) and mitral valve with its "fish-mouth" appearance (**Figure 37-16**) will come into view. Continue tilting the probe upward toward the base of the heart. The three leaflets of the aortic valve will be seen centrally (**Figure 37-17**).

APICAL FOUR-CHAMBER VIEW

The apical four-chamber view is typically the most difficult view to obtain. Placing the patient in the left lateral decubitus position will greatly improve the ability to obtain this view by allowing the apex of the heart to be pressed against the left chest. Place the probe at the PMI, just inferior to the nipple and angling up through the heart (**Figure 37-18*A***). Aim the probe marker to the patient's right. Alternatively, once you get the PSSA view, slide the probe down to the apex of the heart and tilt it upward toward the base of the heart to obtain the apical four-chamber view (**Figure 37-18*A***). The apical view will show the ventricles side by side in the near field of the screen with the atria in the far field (**Figures 37-18*B* and *C***). The interventricular septum should run vertically down the screen.

The LV appears larger and has thicker walls than the RV. The mitral valve sits slightly lower than the tricuspid valve. Ventricular function, flow across the valves, and septal defects can be assessed with this view. This view allows for comparing chamber size and evaluating for RV dilatation if there is concern for an obstructive PE.

SUBCOSTAL INFERIOR VENA CAVA VIEW

Sonographic evaluation of the IVC can provide valuable hemodynamic information to the EP.[20] This view is also referred to as the subxiphoid long axis view. Place the probe in the subxiphoid space, perpendicular to the patient's abdominal wall and with the marker aimed toward the patient's head (**Figure 37-19*A***). The longitudinal IVC will appear posteriorly, beneath the liver and the bowel, as a long black cylinder (**Figures 37-19*B*, *C*, and *D***). **It is important to distinguish the IVC from the aorta.** The aorta lies on the patient's left side, is more "pipe-like" in appearance, is noncompressible when pressure is applied by the US probe, and has a recognizable pulsatility. The IVC is compressible when pressure is applied by the US probe and varies in diameter with respiration (**Figures 37-19*C* and *D***). It may be beneficial to start in the transverse plane in which both the IVC and the aorta are visualized (**Figure 37-20**). Starting with the transverse view, rotate the US probe 90°, maintaining the IVC in the center of the screen to obtain the longitudinal view (**Figure 37-19**). Tilt the probe cephalad to visualize the IVC entering the right atrium (**Figures 37-19*C* and *D***).

Measurements of the IVC proximal to its entrance into the right atrium allow for a noninvasive estimate of CVP. The negative pressure generated in the chest by inspiration draws blood cephalad and decreases the diameter of the IVC (**Figures 37-19*C* and *D***). Normal dimensions for the IVC include a diameter of 1.5 to 2.0 cm and an inspiratory collapse of 50%.[20] A smaller diameter and greater inspiratory collapse are indicative of a low CVP.[20,36] A larger diameter

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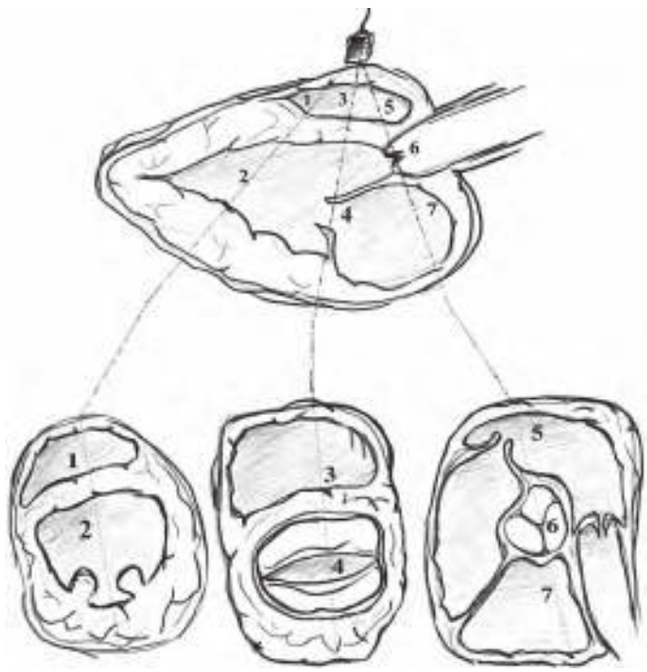

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FIGURE 37-14. The parasternal short axis view. ***A.*** Patient and probe positioning. ***B.*** Diagram of the three US views depending on probe angulation. (Used with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

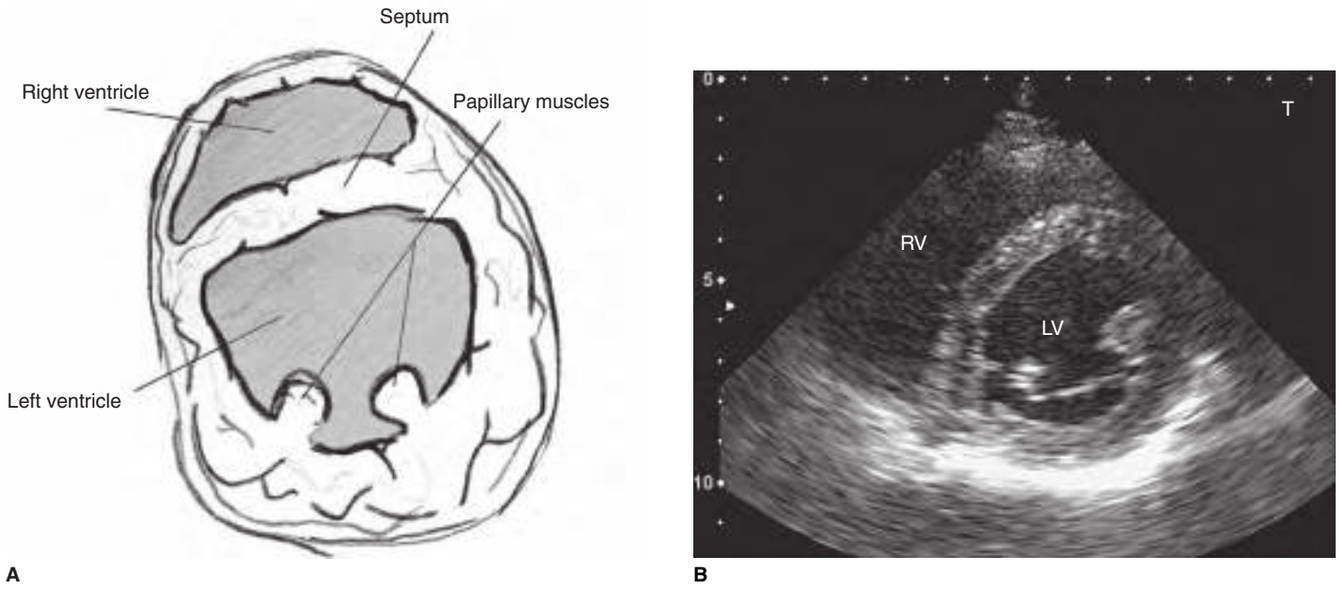


FIGURE 37-15. The parasternal short axis view at the level of the papillary muscles. ***A.*** Diagram of the US image. ***B.*** The US image. (Used with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

and lesser inspiratory collapse reflect a high CVP.[20,36] Obtain estimates by having the patient sniff deeply and freeze the image after inspiration. Use the cine-rewind feature on the US machine to identify images or frames that allow for the measurement of the maximal and minimal IVC diameters through the respiratory cycle (**Figure 37-19**). M-mode tracing of the respiratory cycle allows for precise measures of the inspiratory and expiratory IVC diameters (**Figure 37-21**).

ULTRASOUND GUIDANCE FOR PERICARDIOCENTESIS

US-guided pericardiocentesis has proven to be safe and is the method of choice for most institutions. A brief description of the procedure is provided in this section. Please refer to Chapter 48 for the complete details regarding pericardiocentesis.

Emergent pericardiocentesis can be guided by US using either a static or a dynamic approach. For the static approach, visualize the effusion by US and determine the best approach for needle placement. Remove the probe from the patient and proceed with the pericardiocentesis procedure. For the dynamic approach, the heart is visualized throughout the procedure to guide needle placement. Sterile technique is required for the US probe and cord. The availability of a second ultrasonographer or an assistant for dynamic guidance is helpful, particularly if an agitated-saline injection is attempted.

For the static approach, visualize the path of needle penetration with the corresponding US image. Measure the distance from the top of the image to the pericardial space to determine the depth of needle insertion. It is important to note that the liver is often visualized in the anticipated needle trajectory with the subxiphoid US view.

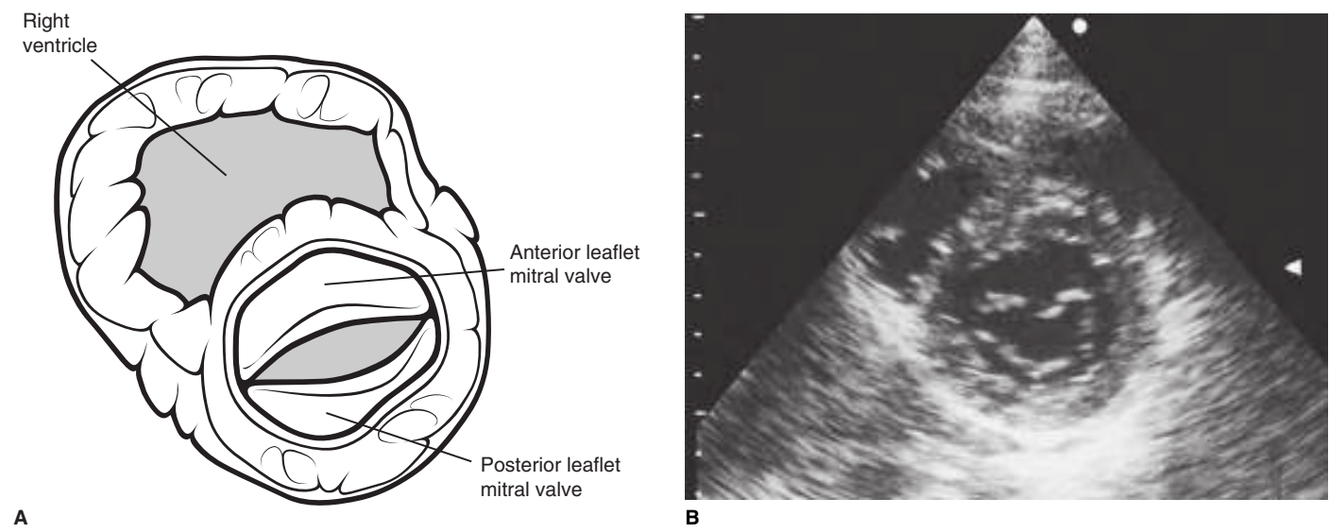
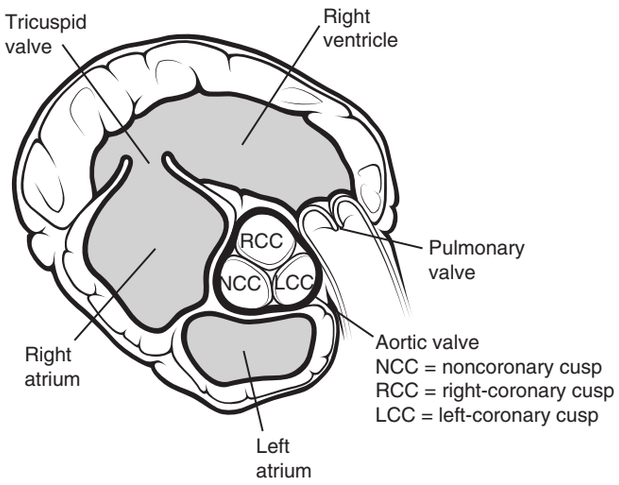


FIGURE 37-16. The parasternal short axis view at the level of the mitral valve. ***A.*** Diagram of the US image. ***B.*** The US image. (Used with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)



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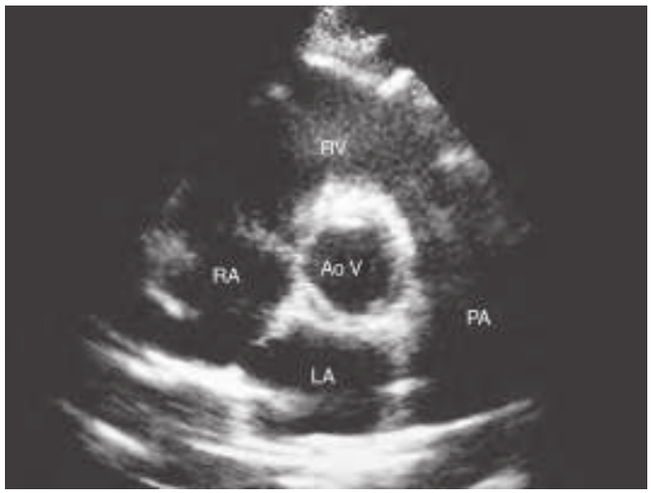
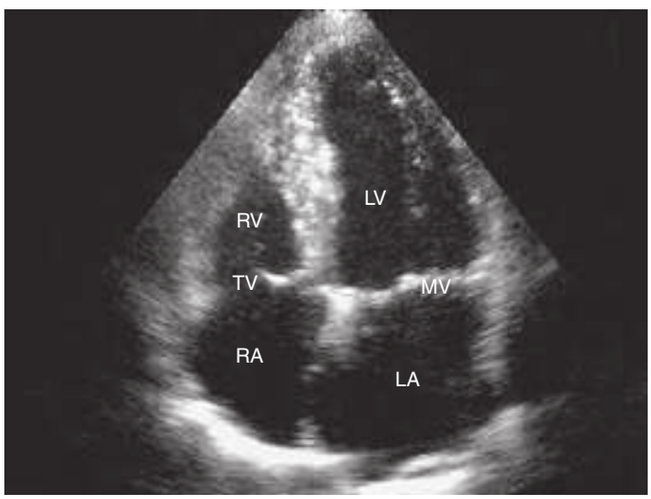


B

FIGURE 37-17. The parasternal short axis view at the level of the base of the heart. ***A.*** Diagram of the US image. ***B.*** The US image. AoV, aortic valve; LA, left atrium; PA, pulmonary artery; RA, right atrium. (Used with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

A



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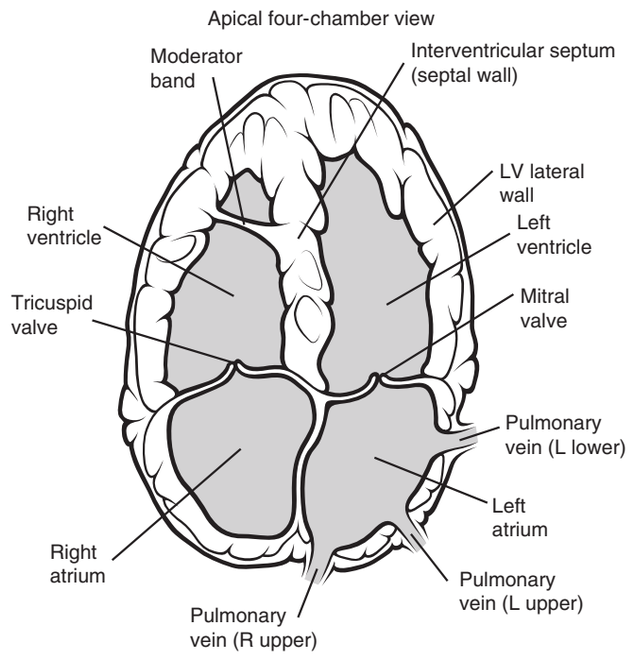
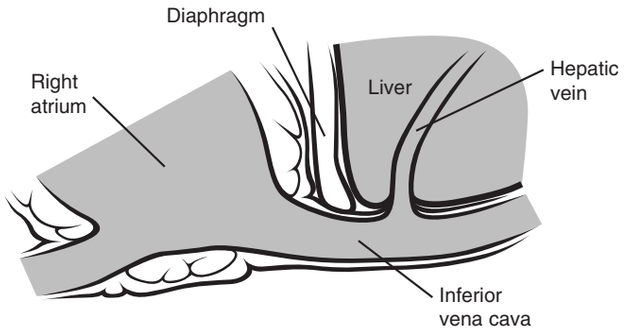


B

FIGURE 37-18. The apical four-chamber view of the heart. ***A.*** Patient and probe positioning. ***B.*** Diagram of the US image. ***C.*** The US image. LA, left atrium; MV, mitral valve; RA, right atrium; TV, tricuspid valve. (Used with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

A



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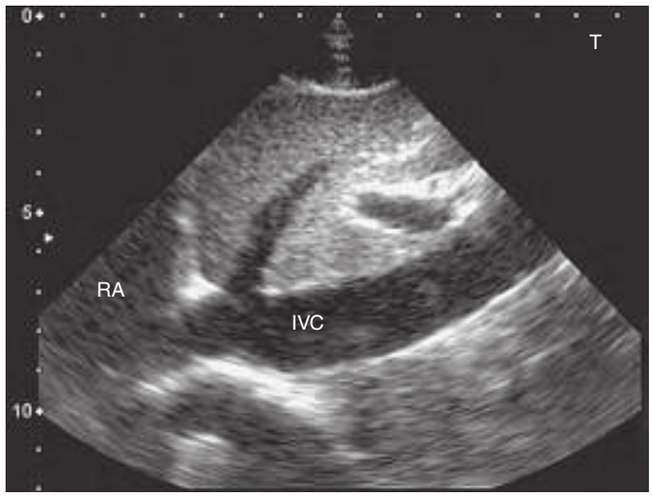

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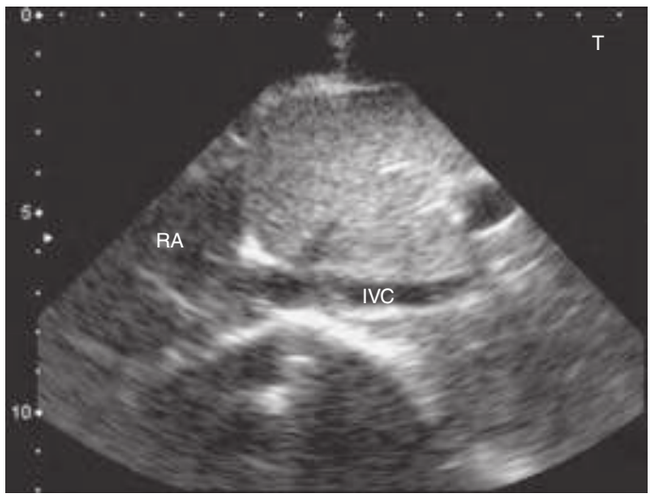

D

FIGURE 37-19. The subcostal IVC view. ***A.*** Patient and probe positioning. **B.** Diagram of the US image. ***C.**** US of the IVC during expiration. ***D.*** Diagram of the IVC during inspiration. RA, right atrium. (Used with permission from Ma OJ, Mateer JM, Blaivas M: *Emergency Ultrasound*, 2nd ed., McGraw-Hill, 2008.)

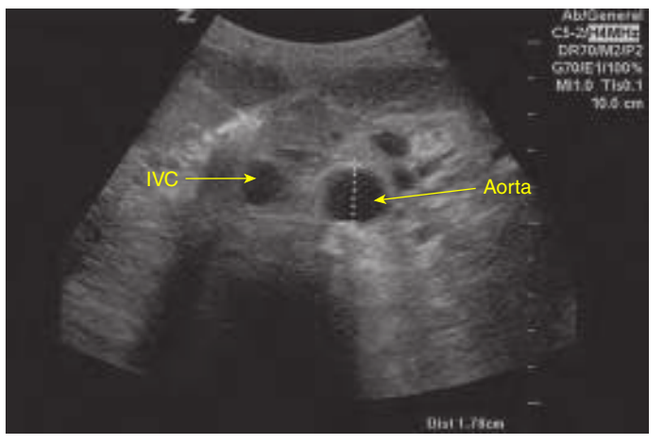

FIGURE 37-20. The transverse IVC view. The IVC is located to the right of the patient's aorta.

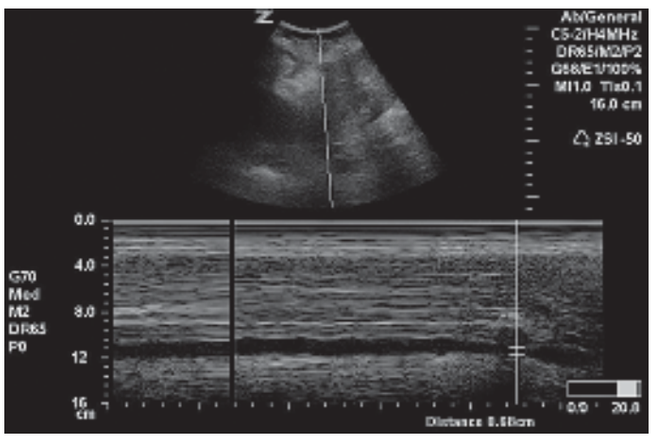

FIGURE 37-21. M-mode tracing of the IVC. Respiratory variation is seen. The dark stripe represents the diameter of the IVC over time. Measurements are taken at the point of inspiratory collapse (*white lines*).

FIGURE 37-22. US probe and pericardiocentesis needle positioning.

Use the PSLA view because the pericardium is more superficial in this view. Proceed with the pericardiocentesis procedure.

For the dynamic approach, insert the needle directly adjacent to the transducer. Angle the probe to interface with the plane of needle insertion (**Figure 37-22**). Insert the needle over the superior edge of the rib and along the plane of the US beam. It is optional to use color Doppler in the near field to ensure the intercostal and internal mammary arteries are not punctured. Advance the needle while aspirating with the syringe as the needle is advanced. The pericardium may "tent" as the echogenic needle presses upon it (**Figure 37-23*A***) and then enters the pericardial space (**Figure 37-23*B***).

Injection of agitated saline may be attempted to confirm needle placement in the pericardial space. Agitate the saline by rapidly injecting saline back and forth from one syringe into another through two ports of a three-way stopcock, with the third port connected by sterile tubing to the pericardiocentesis needle. Once microbubbles have formed, inject the agitated saline into the pericardial space. The fluid will appear on US as a bright white scattering within the pericardial sac.

A

B

FIGURE 37-23. Pericardiocentesis. ***A.*** The needle is visualized tenting (*white arrow*) the anterior pericardium. A hazy pericardial effusion is present. ***B.*** The needle is visualized within the pericardial fluid as a bright white point (*white arrow*). (Image courtesy of Jason Gookhul, MD.)

ULTRASOUND GUIDANCE FOR CARDIAC PACING

US can be used to confirm the placement of intracardiac leads or the capture of cardiac pacing (Chapters 41 to 44). It is difficult to appreciate mechanical capture by simply looking for electrical changes on the ECG monitor. Transcutaneous cardiac pacing discharges often cause simultaneous jerking of the patient that masks a palpable pulse. Cardiac US evaluation during cardiac pacing allows visualization of mechanical contractions of the heart. During placement of the transvenous cardiac pacing wire, it can be visualized passing through the right atrium and tricuspid valve into the RV. A subcostal view of the IVC can confirm errant passage of the wire down the IVC.

SUMMARY

Limited ED cardiac ultrasonography provides real-time information to answer specific questions. It is not intended to replace formal cardiac echocardiography. There is a body of evidence that shows that non-Cardiologists can accurately and safely perform limited cardiac US examinations. The use of cardiac US in the ED has risen from a few basic examinations into a sophisticated series of examinations in a very short time. This chapter provided an introduction to ED cardiac ultrasonography. The EP can become very comfortable with cardiac US as a supplement to the evaluation and management of patients. ED cardiac ultrasonography is a valuable tool that can easily be incorporated into the daily clinical practice of EM.

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38 Chemical Cardioversion

Paul Casey and Thomas Alcorn

INTRODUCTION

Cardioversion is a procedure performed to restore a fast or irregular heartbeat back to normal rhythm.[1] There are two general types of cardioversion. The first is synchronized cardioversion with electricity, which is the most effective treatment.[2-7] The second is pharmacologic or chemical cardioversion. The decision on which type of cardioversion to use is governed by the patient's clinical condition. For unstable patients with hypotension, altered mental status, signs of shock, chest pain, or signs of heart failure, synchronized cardioversion is indicated.[2-4] For patients who are hemodynamically stable without any of the above signs or symptoms, chemical cardioversion may be considered.[8] The use of chemical cardioversion is less expensive than electrical cardioversion, but patients have more complications.[9]

The specific dysrhythmias for which chemical cardioversion is an option include supraventricular tachycardias (e.g., atrial fibrillation

and atrial flutter) and ventricular tachycardia. This chapter will focus on chemical cardioversion for these tachyarrhythmias.

ANATOMY AND PATHOPHYSIOLOGY

The orchestrated contraction of the atria and ventricles of the heart is the result of an organized electrical impulse passing through the myocytes. This electrical impulse originates in the pacemaker myocytes in the sinoatrial (SA) node in the right atrium and spreads through the atria, resulting in contraction. The impulse arrives at the atrioventricular (AV) node and is subsequently passed down the His bundle and Purkinje fibers to the ventricles, resulting in ventricular contraction. This electromechanical coupling results in approximately 75 mL of oxygenated blood being propelled from the left ventricle into the aorta around 60 to 100 times per minute. A disruption in this sequence may result in cardiac dysrhythmias, which interrupt the delivery of oxygenated blood to vital organs and tissues.

SUPRAVENTRICULAR TACHYCARDIA (SVT)

SVT is an umbrella term that encompasses tachyarrhythmias originating in the atria. These can be further classified into those originating in the atria and those originating from the AV node (**Table 38-1**).[10] Sinus tachycardia is the most common cause of tachycardia. It is usually an appropriate cardiac physiologic response to an underlying condition (e.g., hypovolemia, anemia, pain, fever, coronary ischemia, pulmonary embolism) and falls outside the scope of this discussion. The most common tachydysrhythmias are AV nodal reentrant tachycardia, AV reentrant tachycardia, atrial fibrillation, and atrial flutter.

AV NODAL REENTRANT TACHYCARDIA (AVNRT) AND AV REENTRANT TACHYCARDIA (AVRT)

AVNRT and AVRT result from the presence of abnormal electrical circuits. In most individuals, the AV node has a single pathway that conducts the impulse in anterograde fashion, resulting in the depolarization of the bundle of His. In some cases, the AV nodal tissue may have two pathways, one that conducts rapidly and one that conducts more slowly. AVNRT is triggered when a premature atrial impulse reaches the AV node while the faster circuit is still refractory from the prior impulse but the slow pathway conducts. That electrical impulse is conducted in a retrograde fashion through the fast circuit, resulting in a recurrent, fast, and simultaneous impulse conduction to the atria and ventricles.

The etiology of the tachydysrhythmia in AVRT is the bypass tract in the cardiac musculature adjacent to the AV node. These bypass tracts may conduct impulses in an anterograde or retrograde fashion and, in some instances, in both directions. When an impulse is conducted down the accessory pathway in an anterograde manner, the result is ventricular preexcitation, which may be clinically evident by the presence of a delta wave on electrocardiogram (ECG). The impulse may then travel in a retrograde manner through the AV node and result in antidromic (from the Greek term meaning "against running") AVRT. The impulse travels normally down the AV node and conducts in a retrograde manner through the bypass tract, resulting in orthodromic (from the term meaning "right" or "correct running" in Greek) AVRT. These patterns may be clinically distinguished. The QRS complex of an orthodromic AVRT results in a narrow complex tachycardia, whereas antidromic AVRT results in a wide complex tachycardia.

TABLE 38-1 The Common Supraventricular Tachycardias[10]

Atrial tachyarrhythmias	AV tachyarrhythmias
Atrial fibrillation	AV nodal reentrant tachycardia
Atrial flutter	AV reentrant tachycardia
Multifocal atrial tachycardia	Junctional ectopic tachycardia
Sinus nodal reentrant tachycardia	
Sinus tachycardia	

ATRIAL FLUTTER AND ATRIAL FIBRILLATION

Atrial fibrillation, the most common tachyarrhythmia, is the result of multiple electrical impulses being generated in the atria simultaneously.[11] Erratic impulses are conducted through the AV node to the ventricles and result in a rapid ventricular response that manifests on an ECG as an irregularly irregular narrow complex tachycardia. The risk factors for developing atrial fibrillation include older age, male sex, and underlying cardiovascular disease, including hypertension.

Atrial flutter, on the other hand, is the result of a reentrant circuit adjacent to the tricuspid valve in the right atrium. Atrial flutter causes regular impulses from the reentrant circuit in the atrium that manifest on an ECG as an organized regular rhythm. Most commonly, every other atrial impulse is conducted to the ventricles (i.e., 2:1 conduction), resulting in a ventricular rate of 140 to 150 beats per minute. The characteristic flutter waves on the ECG are caused by the regular generation of impulses from the reentrant circuit.

VENTRICULAR TACHYCARDIA

Ventricular tachycardia is most commonly the result of significant underlying heart disease (e.g., hypertrophic cardiomyopathy, dilated cardiomyopathy, coronary artery disease with prior myocardial infarction), although idiopathic ventricular tachycardia is a recognized clinical entity.[2] The regular coordinated ventricular contractions generated by impulses conducted through the His-Purkinje system are replaced by impulses from a nidus in the ventricle or distal conduction system. The result is ineffective rapid regular or irregular ventricular contractions. The impulses are generated below the AV node, and ventricular tachycardia manifests as a wide complex tachycardia (i.e., QRS interval > 120 milliseconds) on the ECG that may be regular or irregular.

INDICATIONS

Chemical cardioversion is indicated when a restoration of normal sinus rhythm is needed. Chemical cardioversion is only indicated in the stable patient. A stable patient will have a pulse and no evidence of hemodynamic compromise (e.g., hypotension, altered mental status, chest pain, or heart failure) other than a rapid heart rate. For the stable patient in atrial fibrillation or atrial flutter, the decision to chemically cardiovert to sinus rhythm versus rate control depends on the duration of symptoms, symptom severity, age, preexisting heart disease, and underlying medical conditions.[6,12] These decisions are often outside the scope of Emergency Medicine and best made in consultation with a Cardiologist. One may consider chemical cardioversion in a stable patient with both SVTs and ventricular tachycardias. The patient may prefer chemical cardioversion to electrical cardioversion. The advantages of chemical cardioversion are that it does not require the patient to be fasted, it is painless, and it does not require the administration of anesthesia.

CONTRAINDICATIONS

Chemical cardioversion is not indicated in unstable patients with signs or symptoms of hypoperfusion or patients without a pulse (e.g., pulseless ventricular tachycardia or ventricular fibrillation).[2]

Electrical cardioversion remains the treatment of choice.[2-7] Chemical cardioversion of atrial fibrillation or atrial flutter should only be considered when the duration of the atrial dysrhythmia is known and is less than 48 hours or if the patient has been anticoagulated for the appropriate duration prior to attempting chemical cardioversion.[13] This decision is commonly made in consultation with a Cardiologist.

EQUIPMENT

- Noninvasive blood pressure monitor
- Heart rate monitor
- Pulse oximeter
- Cardiac monitor
- Intravenous access supplies (Chapters 59 and 61)
- Supplemental oxygen
- Bag-valve mask
- Suction device
- Intubation equipment (Chapter 18)
- Medications for chemical cardioversion
- Code cart with advanced cardiac life support (ACLS) medications
- ECG machine for continuous 12-lead ECG

ADENOSINE

Adenosine is an endogenous nucleoside occurring in all cells of the body.[11,14] Each milliliter of adenosine for injection contains 3 mg of adenosine and 9 mg of sodium chloride in water. The pH of the solution is between 4.5 and 7.5. The intravenous bolus dose usually has no systemic hemodynamic effects.

Adenosine slows the conduction time through the AV node, can interrupt the reentry pathways through the AV node, and can restore normal sinus rhythm in patients with paroxysmal supraventricular tachycardia (PSVT) and PSVT associated with Wolff-Parkinson-White syndrome.[10] Adenosine is antagonized competitively by methylxanthines (e.g., caffeine and theophylline). Larger doses of adenosine may be required, or adenosine may not be effective. The effect of adenosine is potentiated by blockers of nucleoside transport (e.g., dipyridamole). Smaller doses of adenosine may be effective. Adenosine is not effective in converting rhythms other than PSVT (e.g., atrial flutter, atrial fibrillation, or ventricular tachycardia) to normal sinus rhythm.

Intravenously administered adenosine is rapidly cleared from the circulation via cellular uptake. Intracellular adenosine is rapidly metabolized via phosphorylation to adenosine monophosphate by adenosine kinase or via deamination to inosine by adenosine deaminase in the cytosol. Inosine formed by deamination of adenosine can leave the cell intact or can be degraded to hypoxanthine, xanthine, and ultimately uric acid. Adenosine monophosphate formed by phosphorylation is incorporated into the high-energy phosphate pool. Adenosine requires no hepatic or renal function for its activation or inactivation.

Intravenous adenosine injection is contraindicated in patients with second- or third-degree AV block, sinus node disease (e.g., sick sinus syndrome or symptomatic bradycardia), or a known hypersensitivity to adenosine.

At the time of conversion to normal sinus rhythm, a variety of rhythms may appear on the ECG. They usually last only a few seconds without intervention. These include premature ventricular contractions, atrial premature contractions, atrial fibrillation, sinus bradycardia, sinus tachycardia, skipped beats, and varying degrees of AV nodal block.

Adenosine exerts its effect by decreasing conduction through the AV node and may produce a short-lasting first-, second-, or third-degree heart block. The very short half-life of adenosine allows these effects to be self-limiting. **Administer treatment for these rhythms if they are prolonged. Do not administer additional doses if a high-level block occurs with a dose of adenosine.** Higher degrees of heart block may be produced in the presence of carbamazepine. Transient or prolonged episodes of asystole have been reported with fatal outcomes. Use adenosine with caution in patients receiving digoxin or the combination of digoxin and verapamil to prevent complete heart block and ventricular fibrillation.

No controlled studies have been conducted in pediatric patients to establish the safety and efficacy of adenosine for the conversion of PSVT. However, intravenous adenosine has been safely used for the treatment of PSVT in neonates, infants, children, and adolescents.

Adenosine injection should be given as a rapid bolus by peripheral intravenous (IV) injection into a vein or an IV line as close to the heart as possible, followed by a rapid saline flush.

AMIODARONE

Amiodarone is a class III antiarrhythmic drug.[11,14] Each milliliter of amiodarone for injection contains 50 mg of amiodarone hydrochloride, 20.2 mg of benzyl alcohol, 100 mg of polysorbate 80, and water.

Amiodarone is considered a class III antiarrhythmic drug but possesses the electrophysiologic characteristics of all four antiarrhythmic classes. It blocks sodium channels at rapid pacing frequencies, exerts a noncompetitive antisympathetic action, lengthens the cardiac action potential, and has negative chronotropic effects in nodal tissues. Amiodarone also blocks myocardial potassium channels, which contributes to a slowing of conduction and prolongation of the refractory period. The antisympathetic action, block of calcium channels, and block of potassium channels are responsible for the negative dromotropic effects on the sinus node and for the slowing of conduction and prolongation of the refractory period of the AV node. Its vasodilatory action can decrease cardiac workload and myocardial oxygen consumption.

Administration of amiodarone prolongs intranodal conduction and the refractory period of the AV node. It has little or no effect on sinus cycle length, refractory period of the right atrium and right ventricle, repolarization (i.e., QTc), intraventricular conduction (i.e., QRS interval), and infranodal conduction. Administer amiodarone for acute treatment until the ventricular arrhythmias are stabilized.[3] Most patients will require therapy for 48 to 96 hours.

Amiodarone is metabolized to desethylamiodarone (DEA) by the cytochrome P450 (CYP450) enzyme group, specifically CYP3A4 and CYP2C8. The CYP3A4 isoenzyme is present in both the liver and intestines. The highly variable systemic availability of oral amiodarone may be attributed to the large interindividual variability in CYP3A4 activity. Amiodarone is an inhibitor of CYP3A4 and P-glycoprotein. It has the potential for interactions with drugs or substances that may be substrates, inhibitors, or inducers of CYP3A4 and substrates of P-glycoprotein. Examples include protease inhibitors, histamine H_1 and H_2 antagonists, antidepressants, immunosuppressives, HMG-CoA drugs, cardiac glycosides, other antiarrhythmics, anticoagulants, and others. **It is suggested to look up the interactions with other medications before using amiodarone.**

Amiodarone is primarily eliminated by hepatic metabolism and biliary excretion. There is negligible excretion of amiodarone or DEA in urine. Neither amiodarone nor DEA is dialyzable. Amiodarone and DEA cross the placenta, and both appear in breast milk.

No data are available on the activity of DEA in man. It has a significant electrophysiologic and antiarrhythmic effects in animals

similar to amiodarone. DEA's role and contribution to the antiarrhythmic activity of oral amiodarone are not certain. There is evidence of amiodarone activity before significant concentrations of DEA are attained.

Amiodarone is contraindicated in patients with known hypersensitivity to any of the components, an allergy to iodine, cardiogenic shock, marked sinus bradycardia, and second- or third-degree AV block unless a functioning pacemaker is available.

Hypotension is the most common adverse effect of amiodarone. **Clinically significant hypotension is related to the rate of infusion and not the dose.** Treat hypotension by slowing the infusion. Additional therapy may include IV fluids, positive inotropes, and vasopressors. Other side effects include bradycardia, AV block, elevated liver enzymes, worsening of existing arrhythmias, precipitation of a new arrhythmia (e.g., torsades de pointes), acute eye problems, thyrotoxicosis, hyperthyroidism, and hypothyroidism. The combination of amiodarone with other antiarrhythmic agents that prolong the QTc interval should be reserved for patients with life-threatening ventricular arrhythmias incompletely responsive to a single agent. Do not use amiodarone with other drugs that prolong the QTc interval (e.g., fluoroquinolones) unless life-threatening arrhythmias occur.

PROCAINAMIDE

Procainamide hydrochloride is an old antiarrhythmic agent.[11,14] Each milliliter of procainamide for injection contains 500 mg of procainamide, 1 mg of methylparaben, 1.8 mg of sodium metabisulfite, and water. It has a pH of 4.0 to 6.0. Procainamide hydrochloride is a class IA cardiac antiarrhythmic drug.

Procainamide increases the effective refractory period of the atria and, to a lesser extent, the His-Purkinje system and ventricles of the heart. It reduces impulse conduction velocity in the atria, His-Purkinje fibers, and ventricular muscle. It has variable effects on the AV node with a direct slowing action and a weaker vagolytic effect. The myocardial excitability is reduced in the atria, Purkinje fibers, papillary muscles, and ventricles by an increase in the threshold for excitation combined with inhibition of ectopic pacemaker activity by slowing the slow phase of diastolic depolarization. This decreases automaticity in ectopic sites. Contractility of the undamaged heart is usually not affected.

Therapeutic levels of procainamide may exert vagolytic effects and produce a slight acceleration of the heart rate. High or toxic concentrations may prolong AV conduction time, cause an AV block, or cause abnormal automaticity. The ECG may show a slight sinus tachycardia due to the anticholinergic action, widened QRS complexes, prolonged QT and PR intervals due to longer systole and slower conduction, and a decrease in QRS and T-wave amplitude.

Administration of procainamide can produce therapeutic levels within minutes after the infusion is started. Approximately 20% is reversibly bound to plasma proteins. It binds more slowly and reversibly to the heart, liver, lungs, and kidney. A significant fraction of the circulating procainamide may be metabolized in hepatocytes to *N*-acetylprocainamide (NAPA). NAPA has significant antiarrhythmic activity and slower renal clearance than procainamide. Hepatic acetylation rate capability, renal function, and age have significant effects on the effective biologic therapeutic action.

Procainamide is indicated for the treatment of ventricular arrhythmias (e.g., sustained ventricular tachycardia).[2,15] Its use with lesser arrhythmias is not recommended due to its proarrhythmic effect. **Do not treat patients with premature ventricular contractions.**

Side effects are common. **Procainamide has the potential to produce serious hematologic disorders. This includes fatal leukopenia or agranulocytosis.** Do not administer procainamide to patients with complete heart block because it can suppress nodal pacemakers, suppress ventricular pacemakers, and cause asystole. It may be difficult to recognize complete heart block in patients with ventricular tachycardia. Significant ventricular slowing without evidence of AV conduction appearing during procainamide administration is a reason to stop the infusion. **Do not administer procainamide to patients with second-degree AV block or hemiblocks due to the possibility of increased block severity unless the patient has an electrical pacemaker.** Avoid procainamide if the patient is sensitive to procaine or other ester-type local anesthetics. **Procainamide is contraindicated in patients with torsades de pointes.** Procainamide may aggravate this condition instead of suppressing it.

The National Heart, Lung, and Blood Institute's Cardiac Arrhythmia Suppression Trial (CAST) was a long-term, multicenter, randomized, double-blind study in patients with asymptomatic non–life-threatening ventricular arrhythmias who had a myocardial infarction more than 6 days but less than 2 years previously. Excessive mortality or nonfatal cardiac arrest was seen in patients treated with encainide or flecainide. These results and the lack of evidence of improved survival for any antiarrhythmic drug in patients without life-threatening arrhythmias resulted in a warning to not use procainamide in patients with non–life-threatening ventricular arrhythmias.

Monitor the patient's blood pressure during administration of procainamide. It can cause significant hypotension.[2] Use caution to avoid rapid administration of procainamide. Stop the infusion temporarily if the blood pressure decreases by 15 mmHg or more. ECG monitoring is recommended for observation of the response to treatment and detection of QRS complex widening, prolongation of the PR interval, or any signs of heart block.

SOTALOL

Sotalol hydrochloride is an antiarrhythmic drug with class II (β-adrenoceptor blocking) and class III (cardiac action potential duration prolongation) properties.[11,14] It can cause life-threatening ventricular tachycardia associated with QT interval prolongation.

Sotalol is used for the treatment of life-threatening ventricular arrhythmias and the maintenance of normal sinus rhythm in patients with atrial fibrillation or atrial flutter. It should not be used in patients with premature ventricular contractions and those with minimally symptomatic or easily reversible atrial fibrillation or atrial flutter. Other contraindications to the use of sotalol include sinus bradycardia, second-degree AV block, third-degree AV block, sick sinus syndrome, congenital or acquired long QT syndrome, serum potassium < 4 meq/L, cardiogenic shock, decompensated heart failure, asthma or other bronchospastic conditions, prolonged QT interval > 450 milliseconds, and a creatine clearance < 40 mL/min.

Sotalol can cause QT prolongation, bradycardia, AV block, hypotension, and worsening heart failure. Do not abruptly reduce the dose or discontinue the sotalol due to acute exacerbation of coronary artery disease upon cessation of therapy. The sotalol may mask symptoms of hypoglycemia or worsen hyperglycemia in diabetic patients. Avoid its use with other antiarrhythmics or drugs that prolong the QT interval. Avoid using sotalol with digoxin and calcium channel blockers due to the increased risk of bradycardia, hypotension, and heart failure.

The use of sotalol in any age group with decreased renal function should be at lower doses or increased intervals between doses. The normal starting dose is 80 mg twice a day.[3] Closely monitor heart rate and QTc interval. Dose escalations in renal impairment should be done after administration of multiple doses at appropriate intervals. Sotalol is partly removed by dialysis.

IBUTILIDE

Ibutilide fumarate is an antiarrhythmic drug with predominantly class III cardiac action potential prolongation properties.[14,16] Each milliliter of ibutilide for injection contains 0.1 mg of ibutilide fumarate (which is equivalent to 0.087 mg of ibutilide free base), 0.189 mg of sodium acetate trihydrate, 8.90 mg of sodium chloride, hydrochloric acid to adjust the pH to approximately 4.6, and water.

Ibutilide prolongs the action potential duration in isolated adult cardiac myocytes and increases the atrial and ventricular refractory periods. Ibutilide delays repolarization by the activation of a slow inward current of predominantly sodium. Most class III antiarrhythmics block outward potassium currents. These effects lead to prolongation of atrial and ventricular action potential duration and the refractory period. Ibutilide produces mild slowing of the sinus rate and AV conduction. There is no clinically significant effect on QRS duration at IV doses up to 0.03 mg/kg administered over 10 minutes. There is no established relationship between plasma concentration and antiarrhythmic effect. Ibutilide produces a dose-related prolongation of the QT interval. It has no clinically significant effects on cardiac output, mean pulmonary arterial pressure, or pulmonary capillary wedge pressure at doses up to 0.03 mg/kg.

Ibutilide has a high systemic plasma clearance that approximates liver blood flow (about 29 mL/min/kg), a large steady-state volume of distribution (about 11 L/kg), and minimal (about 40%) protein binding. It is cleared rapidly and highly distributed in patients being treated for atrial flutter or atrial fibrillation. The pharmacokinetics in patients with atrial flutter or atrial fibrillation are similar regardless of the type of arrhythmia, patient age, sex, or the concomitant use of digoxin, calcium channel blockers, or beta-blockers. Most is excreted in the urine, with about 7% of the dose excreted as unchanged ibutilide and the remainder (about 19%) excreted in the feces.

Ibutilide is indicated for the rapid conversion of atrial fibrillation or atrial flutter of recent onset to sinus rhythm.[3,16] Patients with atrial arrhythmias of longer duration are less likely to respond to ibutilide. Ibutilide is indicated for rapid IV therapy of ≤ 30 minutes. It is dosed to the termination of the arrhythmia or to a maximum of two doses of 0.01 mg/kg up to a maximum of 1 mg per dose over 10 minutes. Its use can cause potentially fatal arrhythmias (e.g., sustained polymorphic ventricular tachycardia and torsades de pointes). **It is essential that ibutilide be administered in a setting of continuous ECG monitoring and by personnel trained in identification and treatment of acute ventricular arrhythmias. Patients with atrial fibrillation of more than 2 to 3 days in duration must be adequately anticoagulated before conversion with ibutilide so they do not throw a clot and have a stroke.**

Ibutilide is contraindicated in patients who have previously demonstrated hypersensitivity to ibutilide or any of the other components. Ibutilide can induce or worsen ventricular arrhythmias with fatal consequences. Torsades de pointes may occur because of ibutilide's effect on cardiac repolarization, or it can cause ventricular tachycardia in the absence of excessive prolongation of the QT interval. Antiarrhythmics should not be given concomitantly with ibutilide or within 4 hours after infusion because of their potential to prolong the refractory period. Ibutilide can cause heart block. Use caution in patients whose plasma digoxin levels are above or suspected to be above the therapeutic range. Ibutilide should not be administered to pregnant women and those who are breastfeeding.

MISCELLANEOUS AGENTS

The patient is sometimes given dofetilide, dronedarone, mexiletine, or propafenone to convert atrial fibrillation or atrial flutter.[11,14,17] These agents are all administered orally and titrated to effect or side effects. These medications are usually not administered in the Emergency Department and not covered in this chapter.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure, and place this in the medical record.

Ensure the patient is stable with a pulse. They should have no hypotension, altered mental status, signs of shock, ischemic chest discomfort, or acute heart failure. Place the crash cart in the room. Apply pads to the patient if a stable rhythm deteriorates into an unstable rhythm. Fully monitor the patient. Apply a noninvasive blood pressure cuff, cardiac monitor, and pulse oximetry. Obtain venous access. Apply a nasal cannula attached to wall oxygen.

Obtain a 12-lead ECG and identify the rhythm. The first important distinction when interpreting the ECG is whether the QRS interval is wide (i.e., > 0.12 seconds) versus narrow (i.e., < 0.12 seconds). **The cause of a wide complex tachycardia is ventricular tachycardia until proven otherwise.**[8] The differences between SVT with a wide QRS complex due to aberrancy or preexcitation versus ventricular tachycardia are nuances and represent a diagnostic challenge.[8] The 12-lead ECG may help distinguish the different rhythms. Discern if the rhythm is regular or irregular and if it is monomorphic or polymorphic. Determining the rhythm will dictate which medication to use for the chemical cardioversion.

TECHNIQUES

NARROW COMPLEX TACHYCARDIA

This SVT is not due to sinus tachycardia. Consider vagal maneuvers (e.g., unilateral carotid massage) first to see if the patient converts to a sinus rhythm. This may or may not work. Administer adenosine next by rapid IV push over 1 to 2 seconds through a peripheral IV as proximal to the heart as possible. This is due to the very short half-life of adenosine. The IV should be ideally placed in the upper arm. The recommended approach to ensure rapid administration is with a T-connector or stopcock with the normal saline flush and adenosine connected.

The initial dose is 0.1 mg/kg to a maximum dose of 6 mg. Immediately follow the adenosine with a rapid 10 mL flush of normal saline. Observe the monitor for a pause followed by the conversion to a normal sinus rhythm. The adenosine may be repeated at 0.2 mg/kg to a maximum of 12 mg and 0.3 mg/kg to a maximum of 18 mg. Slowing the rate for a brief time with adenosine allows the underlying rhythm to be identified. This may lead to the treatment for atrial fibrillation or atrial flutter.

MONOMORPHIC VENTRICULAR TACHYCARDIA

Evidence suggests IV adenosine is relatively safe for the diagnosis and treatment of ventricular tachycardia that is monomorphic and regular. Administer adenosine as described above. Emergency Physicians usually and immediately administer amiodarone given the familiarity with it in the unstable patient with shock-resistant ventricular tachycardia. However, multiple drug options are available. Amiodarone was previously the American Heart Association's (AHA) antiarrhythmic of choice for patients in ventricular tachycardia. This was based on expert opinion rather than evidence. **The 2015 AHA guidelines list amiodarone, procainamide, and sotalol as options.** Avoid procainamide and sotalol in patients with a prolonged QT interval and heart failure. A second agent should not be given after procainamide or sotalol without an expert consultation

(e.g., a Cardiologist or Electrophysiologist) per the 2015 AHA guidelines. Recent studies have suggested procainamide has increased efficacy and decreased side effects when compared to other antiarrhythmics. More research needs to be done on this subject. Lidocaine is no longer included in the AHA guidelines for management of wide complex tachycardias.

The doses of the medications are quite different. Administer IV procainamide at 20 to 50 mg/min over 10 to 20 minutes or 100 mg every 5 minutes until resolution of ventricular tachycardia, occurrence of side effects, or a maximum of 17 mg/kg.[2,4] Administer 150 mg of amiodarone IV over 10 minutes, followed by 1 mg/min for 6 hours, and then 0.5 mg/min for at least 18 hours. Administer sotalol at 1.5 mg/kg IV over 5 minutes.

POLYMORPHIC VENTRICULAR TACHYCARDIA

Administer IV magnesium if the patient is known to have prolonged QT syndrome or a prolonged QT interval is observed on ECG review when the patient was in sinus rhythm. Use the dose of 1 to 2 gm of magnesium over 5 to 60 minutes. The most common etiology of polymorphic ventricular tachycardia is myocardial ischemia in the absence of a prolonged QT interval. Administer amiodarone and beta-blockers because magnesium is likely to be ineffective. Consult a Cardiologist for further recommendations and management.

ATRIAL FIBRILLATION OR FLUTTER

Administer amiodarone, ibutilide, or sotalol for atrial fibrillation or atrial flutter.[18] Consider consulting a Cardiologist first. Structural heart disease often prevents the chemical cardioversion or causes the reversion of normal sinus rhythm to the preceding rhythm.[5,17] The Cardiologist may use a combination of medications to control the rate or rhythm before later attempting electrical cardioversion.[3,19,20] This combination therapy is not performed in the Emergency Department.

AFTERCARE

Obtain a 12-lead ECG and a repeat set of vital signs.[4] Consult an Interventional Cardiologist if there is any evidence of cardiac ischemia. Correct any underlying causes (e.g., electrolyte abnormalities). Consult a Cardiologist or Electrophysiologist for further evaluation. Disposition varies depending on the etiology and type of arrhythmia.[1,5,21-23] Most patients will be admitted for cardiac monitoring, evaluation of the arrhythmia, and treatment of any underlying causes.[5]

COMPLICATIONS

The most worrisome complication during chemical cardioversion is the conversion into an unstable arrhythmia. This can be due to the medications given or a progression of the preexisting dysrhythmia. **It is important to be prepared and ready to initiate electrical cardioversion or cardiopulmonary resuscitation.**

Medication complications are specific to the medications administered to the patient. Many of these medications may precipitate hypotension. A review of all the medication-related complications is beyond the scope of this chapter. Some of the common medication-related complications were noted previously. Amiodarone can cause bradycardia, hypotension, or pulmonary toxicity, or worsen the dysrhythmia. Procainamide can cause bradycardia and hypotension, increase the QRS duration over 50%, and worsen the dysrhythmia. Sotalol can cause dose-related bradycardia, chest pain, and fatigue.

SUMMARY

The Emergency Physician must rapidly identify and treat patients who are hemodynamically unstable or symptomatic due to an arrhythmia. These patients are not candidates for chemical cardioversion and should receive electrical cardioversion (Chapter 40). The goal of chemical cardioversion is the restoration of a normal sinus rhythm in stable patients with a pulse. The pharmacologic management of chemical cardioversion varies depending on the underlying rhythm.

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39 Automated External Defibrillation

James Montoya and Molly Garcia

INTRODUCTION

Cardiovascular disease is the most common cause of death in the developed world. It is responsible for 350,000 deaths per year in the United States.[1] Approximately 40% of those with out-of-hospital cardiac arrest (OOHCA) have an initial presenting rhythm of ventricular fibrillation.[2] Early defibrillation increases the chances of survival in these situations.

The American Heart Association (AHA) undertook a program in 1991 dedicated to increasing the rate of survival from OOHCA with education regarding the concept of a "chain of survival."[3] The chain is a series of actions that give the highest chances of survival. The links of the chain include calling 911, performing early cardiopulmonary resuscitation (CPR), providing rapid defibrillation, and survival. Inherent are the concepts of early defibrillation and "time is muscle."

The AHA had further advocated in 1995 the use of automated external defibrillators (AEDs) by laypersons as a key link in the chain (**Figure 39-1**).[2] This allowed all but early advanced life support by laypersons. The AED automatically analyzes the heart rhythm and delivers an electrical shock to restore the heart to a normal rhythm.[4]

Most cardiac arrests occur outside the hospital. Ventricular fibrillation is the most common rhythm in OOHCA. There has been a concerted effort in the United States to increase the availability of AEDs in public spaces (e.g., airports, casinos, stadiums) and to responding police officers, resulting in a much higher survival in ventricular fibrillation arrest.[5-11] The approximate 10% survival rate after OOHCA and in-hospital sudden cardiac arrest remains dismally low.[12] The rates versus historical norms in bystander-witnessed ventricular fibrillation arrest have been much improved to as high as 31% in some studies by this effort.[13,14]

Health care professionals often use traditional manual defibrillators (Chapter 40). These are not widely available outside of the hospital. The Emergency Physician must interpret the heart rhythm, know when cardioversion or defibrillation is appropriate, and manually operate the defibrillator. A thorough understanding of how to properly operate AEDs is important. Some AEDs switch modes to become manual defibrillators.

ANATOMY AND PATHOPHYSIOLOGY

The heart is composed of two systems, a physical pump driven by the rhythmic discharge of the electrical system. The electrical system is a conduit composed of the sinus node, the atrioventricular node, the bundle of His, and the left and right bundle branches (**Figure 39-2**). There is an intrinsic regular electrical discharge originating in the sinus node propagating toward the left ventricle in normal sinus rhythm. This electrical discharge has an effective vector from the sinus node toward the left ventricle. Disruptions in this system or dysfunction of the sinus node leads to abnormal electrical activity or an arrhythmia.

Cardioversion and defibrillation are different. Cardioversion is the process of using synchronized (i.e., during the R-wave or QRS complex) electrical shock to interrupt and terminate the abnormal electrical activity, allowing the sinus node to resume its normal activity. Defibrillation uses unsynchronized (i.e., any time during the cardiac cycle) electrical shock. Several dysrhythmias have been demonstrated to be amenable to cardioversion or defibrillation (e.g., atrial fibrillation with rapid ventricular rate, supraventricular tachycardia, and stable ventricular tachycardia). **Ventricular tachycardia and ventricular fibrillation are the only two cardiac rhythms in which shock is signaled as "indicated" and defibrillation is recommended by an AED.**

INDICATIONS

The use of an AED is indicated in patients who have suffered collapse, are unresponsive, or are in cardiac arrest.[15-20] Patients are often positioned in places of high public traffic and areas of congregation (e.g., airports, concert arenas, and stadiums). First responders (e.g., firefighters and police officers) carry AEDs as standard equipment in some areas. **The application of the AED is indicated in anyone who has collapsed.** The AED will recommend "shock" in patients if ventricular fibrillation or ventricular tachycardia is detected.[21]

CONTRAINDICATIONS

Only apply AEDs to patients who are in cardiac arrest and/or unconscious. Do not apply the AED to patients who are breathing, are conscious, or have palpable pulses. AEDs will identify ventricular tachycardia to be a shockable rhythm even if the patient is alert and the rhythm may be amenable to chemical cardioversion.[14] Other rhythms seen in cardiac arrest are pulseless electrical activity (PEA) and asystole. Administration of a shock when the patient is in PEA is not indicated since the electrical activity of the heart is already organized. Shocking a patient in asystole will have no effect since there is no electrical activity of the heart to reset. An AED can be applied to these patients. The AED will determine that a shock is not indicated when these rhythms are detected. Do not use an AED to monitor the heart rhythm if a cardiac monitor is not available.

Medications may sensitize the heart to an electrical stimulus. Some medications may increase the amount of electricity required to defibrillate patients, while others may decrease the energy requirement.[22] Carefully consider defibrillation versus alternate therapies (e.g., Digibind) in patients known to take digitalis or its derivatives.[23]

EQUIPMENT

- AED device (**Figure 39-1**)
- Two self-adhesive electrode pads (**Figure 39-3**)
- Razors, if available but may not be necessary
- Trauma shears or scissors for clothing removal

AED CLASSIFICATION

AEDs are classified as semiautomatic, fully automatic, monophasic, and biphasic.[24] Some AEDs can switch to manual mode. Semiautomatic AEDs are the most common type. The user must push a button on the AED to deliver the shock. Another name for these

FIGURE 39-1. Examples of some of the many AEDs commercially available. ***A.*** Physio-Control Lifepak CR Plus. ***B.*** Zoll Pro Semi-automatic. ***C.*** Cardiac Science Powerheart G3. ***D.*** Samaritan HeartSine 450P.

AEDs are shock advisory defibrillators. Fully automatic AEDs do not require the press of a button to deliver a shock. The AED delivers the shock automatically. Fully automatic AEDs are available in the United States from many different manufacturers, each with somewhat different evaluative algorithms.

Many AEDs are monophasic.[25,26] They are slowly being replaced with biphasic AEDs.[25,26] Monophasic AEDs deliver one shock from the negative electrode to the positive electrode when a shock is indicated. The amount of energy delivered during a monophasic shock is generally between 200 and 360 J.[27] Biphasic AEDs send a shock from the negative electrode to the positive electrode and then in the reverse direction (**Figure 39-4**). Biphasic AEDs can measure the impedance and resistance between the electrodes and adjust how much energy is used. This allows the AED to deliver enough energy needed to reset the heart but not enough to damage the heart. Energy levels typically range between 120 and 200 J.[27] Less battery life is needed with biphasic AEDs since they deliver less energy per shock. Biphasic AEDs provide a more consistent magnitude of current and tend to successfully terminate arrhythmias at lower energies than monophasic AEDS, although neither monophasic nor biphasic AEDs lead to a survival advantage over the other.[28-30] Biphasic AEDs are smaller and easier to carrier or store in areas outside of the hospital.

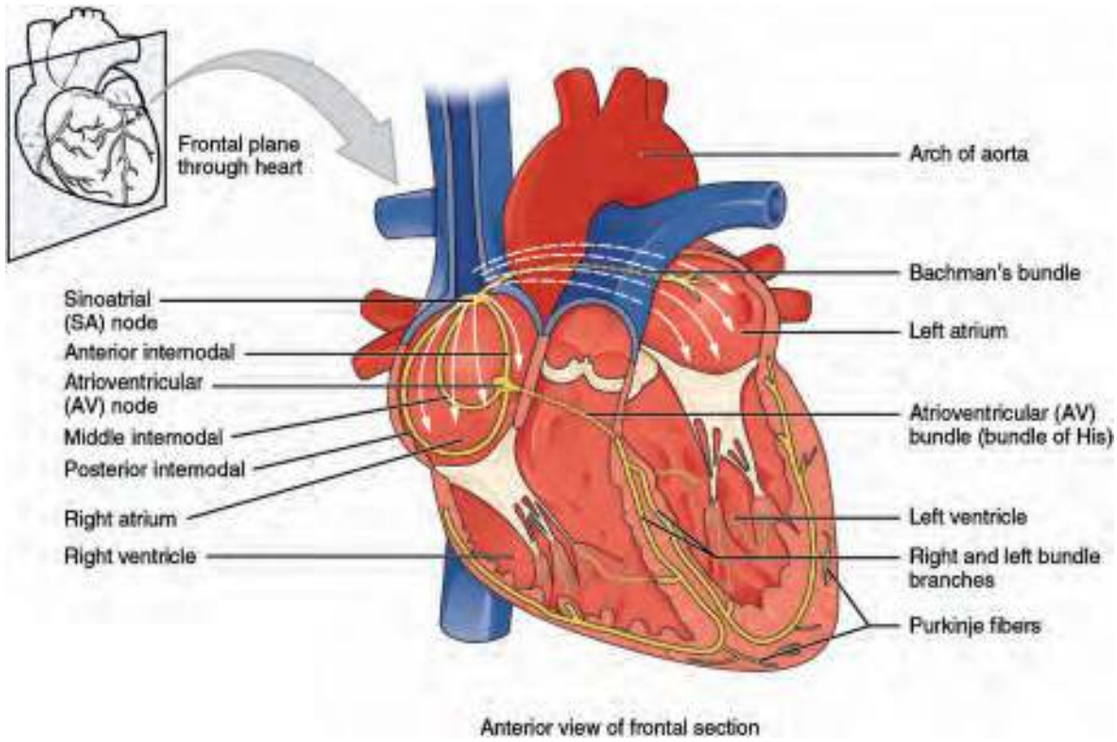

FIGURE 39-2. Conduction system of the heart. (Used from https://commons.wikimedia.org.)

PATIENT PREPARATION

Assess the cardiac, neurologic, and respiratory status of the patient. Start with basic life support care. Place the patient supine on a flat hard surface and initiate CPR. Initiate a search to identify the availability of an AED nearby. Open or cut the patient's shirt to expose skin. Some AEDs have a lid that can be placed under the patient's shoulders to optimize airway opening. Activate 911 and continue CPR if it is determined that cardiac output is insufficient to oxygenate the brain and vital organs.

AEDs are designed for safe and correct operation by the layperson (**Table 39-1**). Directions are generally on the unit in universal

FIGURE 39-3. An example of AED pads. Note each pad has a picture of placement in the adult and child. (Used from https://commons.wikimedia.org.)

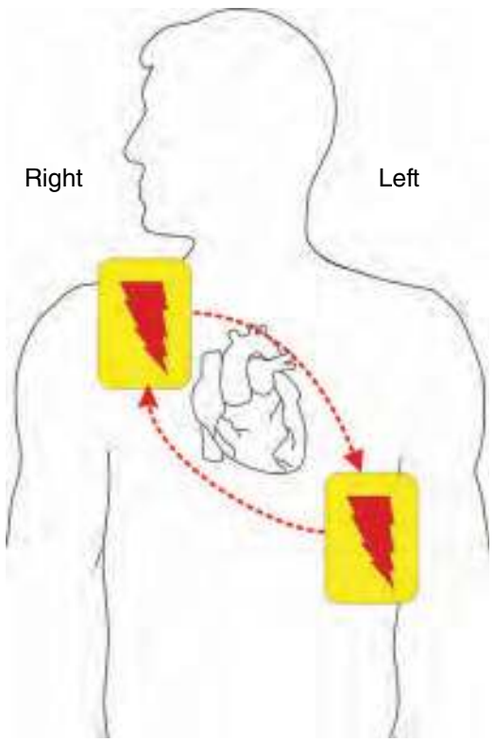

FIGURE 39-4. Biphasic AEDs produce shocks in both directions. Monophasic AEDs only shock in one direction (not shown). (Used from https://commons.wikimedia.org.)

TABLE 39-1 AED Precautions

AEDs can cause harm
Analyze the rhythm without CPR
Change batteries annually
Check the electrode wires for cracks regularly
Do not place AED patches over implanted defibrillators
Do not place AED patches over medication patches
Do not use the AED in seizing patients
Dry off the chest
Have maintenance performed on the AED annually
Liability is waved in most states for bystanders using AEDs
Make sure the AED is programmed for the most current AHA guidelines
Patients must be pulseless
Patients must be unconscious
Patients must be unstable
Periodically check to see if the AED is recalled
Remove the patient from wet environments
Stop transporting when checking rhythms
Use the AED with caution in moving vehicles

pictogram form (**Figure 39-5**). Directions may be delivered by the unit via voice command. The emergency dispatchers at 911 can guide the layperson with little or no previous training with the use of the AED in an emergency.[13]

TECHNIQUE

Turn on the AED. Most AEDs provide audio or visual prompts that will instruct the user of the next steps. Attach the adhesive electrodes of the AED. The adhesive electrodes ("pads") detect the cardiac rhythm. The right pad is placed to the chest wall just below the right clavicle. The left pad is placed lateral to the left breast or apically in position (**Figures 39-3, 39-4, and 39-5**). Additional acceptable placement of pads includes placement in the standard anterior-posterior configuration or in a bi-axillary position (**Figures 40-6 and 40-7**).[31] Individual manufacturers may have guidelines regarding proper pad placement, and this information is usually found in the pictogram or on the packaging (**Figure 39-5**). **Place the pads at least 2.5 cm away from implanted devices and not on top of any transdermal medication patches as they can interfere with the shock delivered and burn the skin.[32] Do not use the AED if the pads touch each other when attached to the patient.[32]**

The chest hair needs to be removed if it does not allow for thorough adhesion of the pads to the chest. Quickly shave the torso hair with a razor if the electrodes do not stick.[32] Alternatively, place an extra pad in the correct position and rapidly pull the pad away from the skin, causing the hair to be removed, if extra AED pads are available. Replace the hair-containing pad with a fresh new pad.

It is necessary to observe if the material surrounding the patient will conduct electricity to the rescuer prior to initiation of rhythm analysis. This includes water or metal. Dry any wetness on the chest or remove the patient from wet environmental conditions. Ensure that no one is touching the metal if the patient is on metal when the shock is delivered.[32]

Begin rhythm analysis by turning the AED on if it is fully automatic or turning it on and pressing the "analyze" button. **Do not touch the patient during rhythm analysis. It can interfere with the computer algorithm used to analyze the heart rhythm.**

The AED is set up with an algorithm to analyze the electrocardiogram (ECG) and advise either shock or do not shock. The user is instructed in semi-automatic units to press another button on the AED to deliver a shock. The fully automatic AED will ask bystanders to "clear for shock" and begin a countdown to initiation of electrical discharge.

The AED will inform rescuers to resume CPR once a shock is delivered. It reanalyzes the rhythm after a predetermined amount of time to determine if additional shocks are necessary. AEDs typically deliver between 120 and 360 J of energy with each shock. The amount of electricity per shock depends on the number of previous shocks administered, the impedance of the chest wall, and whether a monophasic or biphasic waveform is used.[30] Leave the AED attached to the patient for further use. Patients may require further shocks, develop a rhythm not amenable to shock, or have a return of spontaneous circulation (ROSC).

PEDIATRIC CONSIDERATIONS

Ventricular fibrillation occurs in children (e.g., commotio cordis or congenital heart disease). The main issue with AED use in children is that the amount of electricity meant for the adult heart is delivered to the child's heart. There are some AEDs available for use on an infant or pediatric patient.[20] These devices deliver lower energy per shock and use smaller pads (**Figure 39-6**). An adult AED can be used on a pediatric patient if pediatric pads are not available.[33] It is important that the electrodes do not touch when applied to the

FIGURE 39-5. Universal pictogram on an AED.

FIGURE 39-6. An example of pediatric AED pads.

patient. It is more beneficial to use the adult settings than to continue CPR without defibrillation.[34]

Many AEDs can accurately detect ventricular fibrillation in children of all ages and differentiate shockable from nonshockable rhythms with a high degree of sensitivity and specificity.[20,33] Some AEDs are equipped with pediatric attenuator systems (e.g., pad-cable systems or a key) to reduce the delivered energy to a dose suitable for children. Use a pediatric dose-attenuator system for children 1 to 8 years of age. Use a standard AED if providing CPR to a child in cardiac arrest and an AED with a pediatric attenuator system is not available.[33]

Use a manual defibrillator in children less than 1 year of age. Use an AED with pediatric attenuation if a manual defibrillator is not available. A standard AED may be used if neither is available. AEDs with relatively high-energy doses have been successfully used in infants with good neurologic outcomes, minimal skin injury, and minimal myocardial damage.[33,35,36]

AED IN PREGNANCY

The AHA has a scientific statement on arrest in pregnancy[19,37]: "The same currently recommended defibrillation protocol should be used in the pregnant patient as in the non-pregnant patient. There is no modification of the recommended application of electric shock during pregnancy." Remember the need for left and upward compression of the uterus in late pregnancy to alleviate aortocaval compression if additional hands are available. Consider a perimortem cesarean delivery soon after arrest (Chapter 167).

ASSESSMENT

Reinitiate CPR after a shock is delivered. Assess for pulses and neurologic status after 2 minutes as per the advanced cardiac life support protocol. Provide supportive care in the supine or recovery position if the patient has regained consciousness and a pulse, with continuous monitoring of their condition until assistance arrives. The patient has converted into PEA if they return to sinus rhythm via the monitor without pulses. No further shock will be advised by the AED. Resume CPR.

AFTERCARE

Patients can occasionally deteriorate back into a shockable rhythm. Keep the patient connected to the AED until help arrives. Reevaluate the rhythm if the patient's condition changes or deteriorates. Transport the AED with the patient if they have ROSC. Many AEDs store the rhythm(s) detected, number of shocks delivered, and energy delivered, which can be useful after arrival at the Emergency Department.[38,39]

COMPLICATIONS

AED rhythm analysis is automated, and there are very few complications.[21] Device failures are rare.[40] They are categorized as device dependent or operator dependent.[41] Improper storage and neglect of the AED can lead to a depleted battery. A large study reported almost 20% of AED failures were due to battery complications.[41] The recognition sensitivity of AEDs to torsades de pointes and monomorphic ventricular tachycardia below a rate of 150 is poor.[42] This would suggest missed opportunities for defibrillation in these cases. Nearly half of the reported device failures occurred during the attempt to deliver a recommended shock.[41] A significant portion of those failures were due to unanticipated device shut down.[41] The true incidence of these rare but disastrous events is difficult to calculate given the unknown number of uses. Many AEDs are associated with manufacturer defects and are recalled.[4,40]

Most operator-dependent errors are due to set-up. Improper pad placement or connection of the pads to the AED is the most common. Pads must be connected to the AED and placed correctly to provide accurate rhythm analysis. Pads must not be touching or an electrical arc can be generated. Inability to correctly analyze the rhythm may result from improper connections of pads to patient or device, interference from rescuer electrical activity (i.e., contact with the patient), or movement of the patient during rhythm analysis.

Superficial burns and unintended or improper electrical delivery to patient or rescuer may result from the use of an AED. Care must be taken to avoid patient contact during defibrillation as this may result in the rescuer receiving a shock as well. Ensuring that the patient is not lying in a conductive medium (e.g., water) or near a flammable source (e.g., alcohol fumes, nitroglycerin patches) is important to avoid burns and fire that could be potentially generated.

Another common complication is thought to result from the electricity—termination of the ventricular fibrillation with subsequent development of PEA or other arrest rhythm not amenable to shock and without ROSC. Other complications associated with AED use are likely the result of cardiac arrest (e.g., myocardial stunning and pulmonary edema). An undiagnosed history of atrial fibrillation results in a theoretical risk for thromboembolism and stroke due to restoration of normal sinus rhythm and dislodging an atrial hematoma.

SUMMARY

AEDs placed in office buildings, in public access spaces, and with first responders decrease the time to rhythm analysis and defibrillation in those with a shockable rhythm. AEDs can be used in pediatric patients and other special populations. They have low complication rates. AEDs have been developed to allow laypersons to safely use them. They result in an improved survival rate from OOHCA. No difference in these outcomes has been found using monophasic AEDs versus biphasic AEDs. The determination regarding biphasic or monophasic depends on the ability to keep the unit charged, cost, and keeping the AED in working order. Always use the AED in conjunction with the activation of 911 (or local emergency care activation) and after the initiation of CPR.

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40 Cardioversion and Defibrillation

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INTRODUCTION

The application of electricity to the heart induces depolarization of the myocardial cells in a uniform fashion. This may interrupt reentry circuits that are inducing an arrhythmia. Depolarization of the myocardium allows the sinus node to resume its normal pacing function. This is accomplished with the transthoracic application of a direct-current electrical shock.

A large amount of literature exists on the application of electricity for medical applications and dates back to the 17th century. Peter Abildgaard shocked hens in 1775. He found the application of electricity to the body and head renders the animal lifeless and electrical shocks to the chest revived the heart. Claude S. Beck made the first defibrillator that used AC current from a wall socket **(Figure 40-1)**. It was devised for exposed hearts and used in the Operating Room. Naum L. Gurvich developed the world's first commercially available transthoracic DC defibrillator in the USSR in 1952 (**Figure 40-2**).

The techniques of cardioversion and defibrillation are relatively straightforward and practically identical. The main differences are the indications and use of synchronization with cardioversion. **The purpose of cardioversion is to deliver a precisely timed electrical current to the heart to convert an organized rhythm to a more hemodynamically stable rhythm. The purpose of defibrillation is to deliver a randomly timed high-energy electrical current to the heart to restore a normal sinus rhythm.** These techniques are currently performed by emergency medical technicians, nurses, paramedics, physicians, and a variety of other health care workers on a daily basis. This chapter discusses the techniques of manual cardioversion and defibrillation. A discussion of advanced cardiac life support (ACLS), cardiac rhythms, chemical cardioversion (Chapter 38),

FIGURE 40-1. Beck's defibrillator. (Photo used with permission of Dittrick Medical History Center.)

automatic external defibrillation (Chapter 39), and Pediatric Advanced Life Support is beyond the scope of this chapter.

INDICATIONS

CARDIOVERSION

Electrical cardioversion is performed either electively or emergently. The Emergency Department role of electrical cardioversion is usually limited to urgent or emergent situations or when medical therapy has failed.[1,2] This includes symptomatic reentry tachycardias (i.e., supraventricular tachycardia, atrial fibrillation, atrial flutter, and Wolf-Parkinson-White syndrome) and hemodynamically stable ventricular tachycardia associated with acute myocardial infarctions, altered levels of consciousness, chest pain, congestive heart failure, dizziness, dyspnea, hypotension, presyncope, pulmonary edema, shock, or syncope.

FIGURE 40-2. Gurvich's defibrillator. (Photo used with permission from Dittrick Medical History Center.)

Electrical cardioversion is often preferred in the Emergency Department to chemical cardioversion for many reasons. Electrical cardioversion is simple and quick to perform. It is immediately effective in most cases. It may be more successful than chemical cardioversion. The complications are usually minimal. Potential allergic reactions and toxic effects are nonexistent with electrical cardioversion.

DEFIBRILLATION

Defibrillation is indicated when ventricular fibrillation or ventricular tachycardia has not spontaneously converted to an organized rhythm. Ventricular fibrillation and ventricular tachycardia are rarely spontaneously reversible and are not compatible with life. Defibrillation must be performed immediately if the patient is found pulseless, unconscious, apneic, or during the Advanced Cardiac Life Support (ACLS) protocol. "Fine" ventricular fibrillation can be present and may be confused with asystole. It may be secondary to low gain amplitude or improper lead positioning. Rotate the "quick-look" paddles 90° and recheck the monitor. Select a different lead on the monitor and/or increase the gain to determine if the cardiac rhythm is fine ventricular fibrillation or asystole. Ventricular fibrillation or ventricular tachycardia secondary to myocardial ischemia or infarct, electrolyte abnormalities, long QT syndromes, hypothermia, or drug toxicity (e.g., digoxin, tricyclic antidepressants, antiarrhythmics, antihistamine, and macrolide antibiotic combinations) may convert to a more hemodynamically stable rhythm with defibrillation.

CONTRAINDICATIONS

CARDIOVERSION

Cardioversion is contraindicated for several cardiac rhythms or conditions. Do not cardiovert a patient with a rhythm of ectopic atrial tachycardia, junctional tachycardia, multifocal atrial tachycardia, sick sinus syndrome, or sinus tachycardia. Cardioversion is not effective for these rhythms and may result in a worse (i.e., ventricular fibrillation or ventricular tachycardia) postshock rhythm. **Cardioversion of atrial fibrillation should not be attempted unless it is known with certainty that the current episode commenced within the last 48 hours.** Cardioversion of chronic atrial fibrillation, or atrial fibrillation having lasted longer than 48 hours, may dislodge atrial thrombi and result in thrombus embolization and end-organ injury (e.g., stroke). There is some controversy in the literature regarding the cardioversion of atrial flutter greater than 48 hours old without anticoagulation.[3,4] Elective cardioversion of atrial flutter should not be performed if the rhythm has lasted for longer than 48 hours. Atrial fibrillation and flutter can coexist. **Do not cardiovert a patient with a known thrombus in the atria, atrial appendage, or ventricle without first consulting a Cardiologist. Cardioversion in patients with digoxin toxicity should be avoided.** Cardioversion in digoxin toxicity is usually ineffective and has been associated with postshock ventricular tachycardia and ventricular fibrillation.[5] Cardioversion is contraindicated when the patient is without a pulse or has an underlying cardiac rhythm of asystole.

Alterations in the chemical or metabolic milieu of the myocardium may cause subsidiary pacemakers to become more dominant and overtake the sinus mode. This is referred to as enhanced automaticity and can be due to drugs (e.g., digoxin), hypoxia, or electrolyte abnormalities (e.g., hypokalemia or hypomagnesemia). Uniform depolarization with electricity does not terminate this abnormality, as uniform depolarization already exists. The rhythms

that may occur are sinus tachycardia, ectopic atrial tachycardia, multifocal atrial tachycardia, and the digoxin toxic rhythms. Treatment of the underlying etiology is the treatment of choice.

DEFIBRILLATION

There are few contraindications to defibrillation. The main contraindication is in a patient who has made it clear that he or she does not wish to be resuscitated. Defibrillation should not be used for arrhythmias other than ventricular tachycardia or ventricular fibrillation.

EQUIPMENT

- Cardioverter-defibrillator unit
- Conductive jelly or pads
- Suction source, tubing, and catheter
- Airway management supplies
- Advanced Cardiac Life Support (ACLS) medications
- Intravenous sedative agents
- Cardiac monitor
- Noninvasive blood pressure monitor
- Pulse oximeter
- Oxygen source and tubing
- Nasal cannula or face mask to deliver oxygen

THE CARDIOVERTER-DEFIBRILLATOR UNIT

The typical cardioverter-defibrillator unit performs cardioversion, defibrillation, and pacing (**Figures 40-3, 40-4, and 40-5**). A list of available features on a typical unit is listed in **Table 40-1**. Newer models feature lower power outputs to accommodate their use in children, pediatric and adult paddles, biphasic waveforms, and cardiac pacing capabilities. Each Emergency Physician should be familiar with the specific unit at their facility. The general features of the unit are discussed below.

The unit is self-contained. It plugs into a standard electrical outlet. The unit contains rechargeable batteries, which allows it to be portable. An oscilloscope provides real-time monitoring of the patient's cardiac rhythm. A continuous electrocardiographic (ECG) rhythm strip providing documentation on paper is standard with each unit. It produces a hard copy for the medical record. Numerous dials or electronic touchpads with digital displays allow the operator to set the working mode, energy level, pacemaker settings, and oscilloscope input (i.e., ECG leads or "quick-look" paddles). The depolarizer within the machine provides direct electric current for cardioversion and defibrillation.

The synchronizer permits the discharge of electric current based on the patient's ECG waveform. It searches for the R and S waves of the ECG tracing to determine the proper time to discharge the current. It avoids delivering the current during the repolarization phase of the myocardial action potential, when the heart may convert to ventricular fibrillation or ventricular tachycardia. A brief delay is noted while the synchronizer searches for an appropriate time to discharge the current when the operator pushes the button to discharge the unit.

FIGURE 40-4. An example of a cardioverter-defibrillator with paddles.

MONOPHASIC VERSUS BIPHASIC UNITS

All cardioverter-defibrillator units in the past generated monophasic waveform current to deliver the shock. Biphasic waveform current generating units were developed in the late 1990s. **The biphasic units deliver more current at a lower energy level to cardiovert or**

FIGURE 40-3. Two examples of cardioverter-defibrillator units.

FIGURE 40-5. An example of a cardioverter-defibrillator with patches.

TABLE 40-1 Characteristics of a Typical Cardioverter-Defibrillator Unit

Adult and pediatric paddles
Cardioversion capability
Continuous ECG rhythm-strip documentation on paper
Defibrillator capability
Depolarizer
On/off switch
Oscilloscope to monitor cardiac rhythms
Pacing capabilities
Portability
"Quick-look" paddles or pads
Safety mechanism to prevent accidental electrical discharge
Standard ECG leads (three) to attach to the shoulders and lower extremity
Synchronizer
Wide range of energy selection

defibrillate (Tables 40-2 and 40-3). Biphasic units are more effective in cardioverting and defibrillating patients. Biphasic units are significantly more expensive than monophasic units. Determine which type of unit is available at your facility to determine the proper energy levels to administer.

TYPES OF ELECTRODES

The electrodes are referred to as either paddles (**Figure 40-4**) or patches (**Figure 40-5**) depending on their configuration. They must be firmly applied to the patient's torso. They allow a "quick look" and transmit the patient's cardiac rhythm to the oscilloscope and let the Emergency Physician make decisions. Each paddle has a button on which a thumb is to be placed (**Figure 40-4**). This serves as a safety mechanism. Both buttons must be depressed simultaneously to discharge the current. This prevents accidental and premature discharge of current, which may cause injury to the patient, the operator, or bystanders. Most newer units use self-adhesive, single patient use, disposable patches as an alternative to paddles (**Figure 40-5**). The patches apply to the torso similar to ECG leads. They connect by cables to the cardioverter-defibrillator unit.

Paddles or patches come in various shapes and sizes. Adult paddles or patches are round, oval, or rectangular in shape. They measure 8 to 10 cm in greatest diameter. They can be used on children weighing more than 10 kg or over 1 year of age, adolescents, and adults. Pediatric paddles or patches also come in a variety of shapes and measure 4 to 6 cm in greatest diameter. The pediatric paddles are to be used in children weighing less than 10 kg or less than 1 year of age.[6] Some units contain both adult and pediatric paddles or the ability to use both adult and pediatric patches. The adult electrode slides off the paddle handle to reveal the pediatric-size electrode.

TABLE 40-2 Recommended Initial and Subsequent Monophasic Energy Levels for Cardioversion or Defibrillation

Cardiac rhythm	Initial energy setting	Subsequent energy settings*
Adults		
Atrial fibrillation	100 J	200, 300, 360 J
Atrial flutter	50 J	100, 200, 300, 360 J
Supraventricular tachycardia	50 J	100, 200, 300, 360 J
Ventricular tachycardia	200 J	300, 360 J
Ventricular fibrillation	200 J	300, 360 J
Children		
Supraventricular tachycardia	0.5 J/kg	1.0 J/kg
Ventricular tachycardia	2.0 J/kg	4.0 J/kg
Ventricular fibrillation	2.0 J/kg	4.0 J/kg

*To be performed sequentially in this order.

TABLE 40-3 Recommended Initial and Subsequent Biphasic Energy Levels for Cardioversion or Defibrillation

Situation	Initial energy setting	Subsequent energy settings*
Adults		
Defibrillation	120 J	150, 200, 200 J
Synchronized cardioversion	70 J†	120, 150, 150 J
	75 J‡	
Children		
Defibrillation	2.0 J/kg	2.0, 2.0, 2.0 J/kg

*To be performed sequentially in this order.
†E Series.
‡M Series, CCT, R Series.

The cutoff of 10 kg and 1 year of age is relative in choosing the proper paddle or patch for a small child. **Choose the largest paddle or patch that will achieve complete and full contact with the child's chest wall.** Larger paddles and patches will allow a greater amount of myocardium to be depolarized while decreasing the current density applied and minimize myocardial injury. **The paddles or patches must be at least 2 to 3 cm apart to prevent electrical bridging and burn injury to the child. Using paddles or patches that are too large will deliver the electric current over too great an area and decrease its effectiveness.** The opposite is true in adults. Using paddles or patches that are too small will deliver the electric current over a small area, making it too intense, and increases the potential damage to the myocardium.

ELECTRODE POSITIONING

The paddles or patches may be positioned in several different patterns.[7-9] The most commonly used positions are anterolateral for paddles (**Figure 40-6**) and either anterolateral or anteroposterior (**Figure 40-7**) for patches. Anterolateral paddles or patches are positioned with the anterior paddle at the right upper sternal border over the second and third intercostal spaces. The lateral paddle or patch is placed in the left midaxillary line centered over the fourth and fifth intercostal spaces. Anteroposterior placement is often used

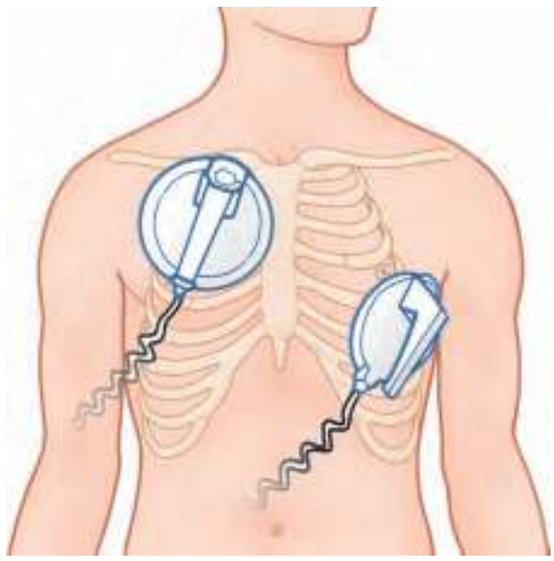

FIGURE 40-6. Anterolateral pad and paddle positioning.

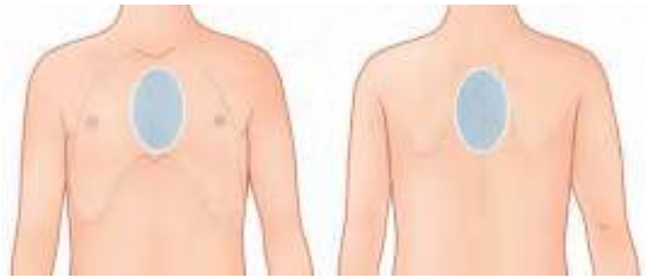

FIGURE 40-7. Anteroposterior pad and paddle positioning.

with disposable patches rather than paddles (**Figure 40-7**). The anterior patch is centered over the sternum, and the posterior patch is placed between the scapulae. A randomized prospective trial evaluated the anteroposterior versus anterolateral patch position in converting atrial fibrillation.[9] The anteroposterior patch position was more effective in converting the rhythm. Another study showed no difference in the patch position.[10] A third study of atrial fibrillation showed the anterolateral position worked best.[11] Adult patches or paddles can be substituted if pediatric patches or paddles are required but not available.[7] Roll the child onto their right side and place the patches in the anteroposterior position. Other positions include the latero-lateral (i.e., axilla-to-axilla) position and the parasternal-infraclavicular (i.e., oblique, left anterior chest to right posterior infrascapular) position.[8]

CONDUCTIVE CONTACT MEDIUM

Electrically conductive contact medium should always be applied between the electrode and the patient's chest wall. Conductive gel pads are commercially available and primarily used. The contact material helps to maximize current flow, minimize resistance, reduce transthoracic impedance, and prevent thermal or electrical burns to the chest wall. The self-adhesive disposable patches are prelubricated with contact medium and need no additional contact medium.

Contact medium is required with the older units that use paddles. Self-adhesive, disposable conductive gel pads can be used. An alternative is the gel or paste form of contact medium. The contact medium should be applied to the paddles generously. **It should not connect the paddles because it would divert the electric current along the chest wall, away from the heart, and cause burns to the chest wall. S**aline-soaked gauze squares can be used in an emergency if contact medium is not available. The saline must be squeezed out of the gauze squares to prevent the accumulation of liquid on the chest wall and bridge the two paddles.

PATIENT PREPARATION

Place the patient supine on a bed. Attach the cardiac monitor, noninvasive blood pressure monitor, pulse oximetry, and oxygen to the patient. Obtain intravenous access. **Suction and resuscitation equipment should be readily available in case it is needed.** Cardioversion is scary and extremely uncomfortable for patients. Explain the risks, benefits, and alternative procedures to the patient and/or their representative if cardioverting. Obtain an informed consent for the procedure and place this in the medical record. Premedicate the patient prior to cardioversion if no contraindications exist, the patient is hemodynamically stable, and they can tolerate a delay to cardioversion.[12] The choice of the appropriate sedative agent (Chapters 12 and 159) is physician-dependent. Commonly used agents include etomidate, ketamine, midazolam, methohexital, propofol, and thiopental. Diazepam and lorazepam are not often used because of the long delay to onset of their action.

TECHNIQUES

Stand at the patient's left side. Turn on the cardioverter-defibrillator unit. Set the display to "quick-look." Instruct the nurses to apply the ECG leads to the patient. Grasp the left paddle labeled *sternum* with the left hand and the right paddle labeled *apex* with the right hand (**Figure 40-4**). This is the anterolateral paddle position. Apply the paddles and observe the patient's cardiac rhythm. Set the mode as asynchronous (i.e., defibrillation) or synchronous (i.e., cardioversion) based on the patient's cardiac rhythm. Set the energy level (**Tables 40-2 and 40-3**). **Older defibrillator units deliver monophasic waveform energy. Newer units deliver biphasic energy, which is more effective and delivers more current at lower energy settings.**

Apply conductive pads to the patient's torso in the anterolateral position. Alternatively, apply conductive jelly to the paddles liberally and rub them together to coat the electrode surface completely. Apply the paddles firmly to the torso in the anterolateral position. **The paddles should be separated from each other by at least 2 to 3 cm to prevent arcing of the current and injury to the patient.**

Prepare to deliver the electric current to the patient. Charge the paddles. This must be done on the unit or the paddles before the initial and each subsequent discharge. It takes approximately 2 to 5 seconds to charge the paddles following activation of the charge button. Cardiopulmonary resuscitation (CPR) can be performed while the unit is charging. **Ensure that nurses and other assistants are not touching the patient or the stretcher by saying "clear."** The assistant ventilating through a bag-valve device attached to an endotracheal tube does not need to drop the bag, as plastic is nonconductive. Consider turning off the oxygen during shock delivery. **The person who will deliver the charge to the patient should ensure that their body is not in direct contact with the patient or the stretcher.**

The American Heart Association (AHA) recommends limiting the disruptions to CPR yet also recommends stopping CPR during defibrillation.[13,14] This has led to interest in hands-on defibrillation. It has been shown in limited studies to be safe and effective, and it does not require the cessation of CPR.[15,16] The best advice for now is to use hands-off defibrillation until more information on the safety of this procedure is obtained.

Reevaluate the patient's cardiac rhythm. Deliver the charge by simultaneously pressing the discharge buttons on each paddle. Observe the monitor and reevaluate the patient's cardiac rhythm. The unit can be recharged to deliver another electric charge to the patient if indicated.

The technique using self-adhesive disposable patches on newer units is similar with a few exceptions. Apply the patches in the anteroposterior position (**Figure 40-7**). No supplemental contact medium is required. Select the desired energy level (**Tables 40-2 and 40-3**). Charge the cardioverter-defibrillator unit by pressing the charge button on the unit. Press the discharge button on the cardioverter-defibrillator unit to deliver the charge to the patient. Reevaluate the need for further therapy.

The cardioverter-defibrillator unit may not deliver a shock. Check that the unit is plugged in, that no touching or bridging of the pads or gel has occurred on the patient's chest, and that the paddles are charged. The cardioverter-defibrillator unit may not be able to properly sense the R wave in synchronization mode with a rapid supraventricular rhythm. Reattempt to deliver the shock by holding down the discharge button for 15 seconds. This will give the cardioverter-defibrillator unit more time to determine and find a proper time to deliver the shock. **Switching to asynchronous mode will allow the delivery of the shock, but risks shocking at the inappropriate time and converting the rhythm to ventricular tachycardia or ventricular fibrillation.** Consider intravenous medication to slow the heart rate or chemically convert the rhythm rather than applying an asynchronous shock.

ALTERNATIVE TECHNIQUE

The use of double defibrillation has caught the attention of researchers. It uses two machines with their own patches (**Table 40-4**) and the sequential defibrillation of the patient.[17-26] The patient receives between 400 and 600 J, depending on the setting of each defibrillator. It has been used by Electrophysiologists in the catheterization laboratory and for refractory ventricular fibrillation. The defibrillation may work because of the higher energy applied across the myocardium, the different defibrillation vectors, or another not yet discovered mechanism. The results are mixed in terms of survival to hospital discharge. More research is being done to answer these questions. For the time being, it does not hurt to try if standard defibrillation does not work.

AFTERCARE

No specific aftercare is required related to the procedure of cardioversion or defibrillation. If using a cardioverter-defibrillator unit with paddles and conductive gel or paste, it is possible to cause a thermal burn to the skin. This should be treated as any other skin burn. Continuously monitor the patient after they are cardioverted or defibrillated. Almost all patients receiving cardioversion or defibrillation will be admitted to the hospital. A successfully cardioverted patient may occasionally be discharged home. The decision to discharge the patient should be made in consultation with the patients Primary Care Physician and/or a Cardiologist.

COMPLICATIONS

Complications of cardioversion can range from none to death. Thermal and electrical burns are potential injuries. Skin burns may result, the severity of which increases depending on the energy level used and the number of shocks delivered. Care must be taken to avoid contact between the ECG monitor leads and the paddles or contact of the paddles with each other, as sparks or fire may result. Burns can be minimized by using electrically conductive contact media and firmly applying the paddles to the patient. Remove any fluid materials on the chest wall (e.g., conductive jelly, saline, sweat, urine, or water) as they can form a bridge between the paddles or pads and result in arcing and thermal burns to the thorax. Remove any nitroglycerin patches or ointments from the patient's torso. Ensure that there are no open oxygen sources that could ignite when the unit is discharged. Repeated shocks will produce only a mild erythema to the chest wall if performed correctly. Systemic emboli may occur from clots in the left atrium becoming dislodged if the underlying rhythm prior to the cardioversion or defibrillation is atrial fibrillation.

Occasionally, hypertension, other arrhythmias, or heart block may develop. Ventricular fibrillation may result from a synchronized or nonsynchronized shock delivered on the T wave.[27-29] This usually occurs immediately and can be corrected with a nonsynchronized countershock. **Ensure that the unit is in synchronous mode, not asynchronous mode, when cardioverting an organized cardiac rhythm.** Always observe the monitor before delivering a countershock to ensure that it is required. Change the monitor lead so that the T wave is smaller than the R wave and the unit will not cardiovert during a vulnerable period if the T wave is large. Ventricular fibrillation that occurs within 30 to 60 seconds after the delivery of a synchronous shock is often due to digoxin toxicity and is difficult or impossible to correct. Transient ST-segment elevation may occur.[30]

Creatine kinase enzyme elevations may occur, with most being skeletal muscle in origin.[28] Cardiac enzymes can become elevated. The higher the energy level used and the more countershocks given, the greater is the muscle damage. Usually no significant permanent myocardial damage occurs.

Do not apply the paddles or patches directly over an implanted defibrillator or pacemaker. The electric discharge can permanently damage these devices. Adjust the paddle or patch position so they are not directly over these devices.

Avoid injury to yourself or others by ensuring that no one is in contact with the bed or the patient when the shock is administered. Such injuries can range from mild shocks and burns to cardiac dysrhythmias. An improperly functioning unit can cause injury despite being used properly. Periodic maintenance and calibration of the unit are necessary.

TABLE 40-4 Comparison of Methods for Dual Sequential Defibrillation[19-23]

Defibrillation pad location set 1	Defibrillation pad location set 2
Right parasternal and cardiac apex	Next to set 1
Below right clavicle and cardiac apex	Anteroposterior
Left chest next to sternum and left lateral chest	Left anterior and posterior chest
Right parasternal and cardiac apex	Next to set 1
Below right clavicle and cardiac apex	Anteroposterior

SUMMARY

Cardioversion and defibrillation are the processes of applying electric current to a patient's chest to terminate a dysrhythmia. Cardioversion is a safe and effective method of converting reentry arrhythmias. A trial of medical therapy is warranted if the patient is stable. Cardioversion should be initiated as soon as possible if the patient is unstable. Consider administering parenteral sedation as cardioversion is anxiety provoking and painful for the patient. Cardioversion should be performed in the synchronized mode. Always be prepared for ventricular fibrillation or ventricular tachycardia as a result of cardioversion of an organized rhythm. ACLS medications and airway support must be readily available. Defibrillation is essentially cardioversion of unstable ventricular tachycardia or ventricular fibrillation. It is performed like cardioversion except that synchronization and sedation are not required.

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41 Transcutaneous Cardiac Pacing

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INTRODUCTION

Transcutaneous cardiac pacing (TCP) was first documented as a technique in 1872.[1] It was successfully demonstrated in two patients with underlying cardiac disease and symptomatic bradycardia.[1] The clinical difficulty of open-chest pacing, transvenous pacing, and open-chest cardiac massage for the treatment of "ventricular standstill" in an emergent setting has been recognized.[1,2] Animal experiments were performed using electrodes placed in various positions prior to the use of subcutaneous needle electrodes at points "in a line transversing the ventricles."[1] The use of the procedure during eight surgical cases in which cardiac arrest occurred resulted in five patients successfully surviving to discharge.[2] Cardiac monitoring during surgery was not routinely performed and the underlying rhythm being paced was not specifically determined in all reported cases.

Further work related to TCP seems to have lapsed until the 1980s. Several groups studied the technique for treatment of symptomatic bradycardias, asystolic cardiac arrest, and bradyasystolic cardiac arrest in the hospital and in the Emergency Department.[3] They concluded that TCP is as successful as transvenous cardiac pacing in obtaining electrical and mechanical capture in bradyasystolic arrests. TCP was easier to initiate than transvenous cardiac pacing. However, TCP did not improve the overall survival rates for these patients.[3]

TCP was recommended for cardiac emergencies by the International Liaison Committee on Resuscitation (ILCOR) guidelines beginning in 1980 and is currently recommended for treatment of symptomatic bradycardias, especially when the conduction block is at or below the His-Purkinje level.[4] TCP is no longer indicated for the treatment of asystolic cardiac arrest since there are no improvements in rate of hospital admission or survival to hospital discharge.[5]

With current technology and equipment, TCP offers several advantages when compared to the placement of a transthoracic cardiac pacemaker (Chapter 42) or transvenous cardiac pacemaker (Chapter 43) in the Emergency Department. The procedure requires minimal training and can be performed quickly. TCP is a noninvasive procedure and is not associated with the major complications of placing more invasive pacemakers. This includes inadvertent arterial puncture, hemorrhage, pneumothorax, or cardiac tamponade from cardiac rupture. **TCP is an ideal early and temporary intervention for patients requiring stabilizing cardiac pacing support until more invasive procedures can be arranged in the proper clinical setting.**

ANATOMY AND PHYSIOLOGY

Electrical impulses originating in the sinoatrial (SA) node create an action potential that is conducted along the intrinsic cardiac nerve pathways to the atrioventricular (AV) node and disseminated through the His-Purkinje system. This action potential stimulates electrolyte flux, myocardial muscle depolarization, and subsequent cardiac muscle contraction. Electrical propagation and myocardial contraction occur separately in the atria and ventricles. The atria contract slightly ahead of the ventricles during ventricular diastole. This timing delay assists in filling the ventricles prior to their next ventricular systolic phase. The intrinsic heart rate is controlled by a balance of input from the sympathetic nervous system acting directly on the cardiac muscle and the parasympathetic nervous system acting most prominently at the SA and AV nodes.

The intrinsic heart rate can be disrupted by a wide variety of disease processes. The blood supply for the SA node is from an early branch of the right coronary artery. Coronary artery disease can disrupt blood flow and oxygen supply to the myocardium and create areas of cardiac ischemia that affect the conduction system, the impulse production, or conduction at the SA and AV nodes. Cardiac ischemia can result in action potential conduction delays and heart blocks, with resultant bradycardia and hypotension. Electrolyte disturbances (e.g., hyperkalemia), structural heart disease, drug toxicity (e.g., calcium channel blockers and beta-blockers), systemic

toxins (e.g., cyanide), and systemic hypothermia are other causes of intrinsic conduction delays and heart blocks.

TCP delivers an extrinsic electrical impulse that overrides the intrinsic cardiac action potential. Initial pulse durations used were short at 1 to 2 milliseconds. They have now been extended to 20 to 40 milliseconds to decrease the threshold current (i.e., the current needed for cardiac muscle stimulation). The longer pulse duration decreases patient comfort as the degree of skeletal muscle contraction is generated by the TCP pulse.[6] Most TCP devices can generate up to 200 milliamps (mA) of current. The mean pacing current needed to obtain capture ranges from 40 to 100 mA.[6-9]

INDICATIONS

TCP is technically the fastest and easiest method of emergency cardiac pacing. It is a temporary intervention prior to implementation of transvenous cardiac pacing, placement of a permanent cardiac pacemaker for primary cardiac dysfunction, or until the underlying etiology of the bradycardia can be reversed (e.g., myocarditis).[10-15] It is indicated for patients with symptomatic bradycardia from any etiology in whom pharmacologic interventions (e.g., atropine) have been unsuccessful.[16] It can be effectively applied in the prehospital setting by Emergency Medical Services (EMS) personnel, in a clinic, in the Emergency Department, or in the hospital. **TCP is quick, simple, and not associated with the morbidity or mortality of transvenous cardiac pacing in patients with bradyarrhythmias expected to be transient (e.g., digoxin toxicity) or AV block in the setting of inferior wall myocardial infarction.**

It is indicated in patients with AV conduction blocks and sinus node dysfunction.[17] It should be performed in symptomatic patients (e.g., syncope, presyncope, dizziness, fatigue) with complete or third-degree AV block, asystolic pauses exceeding 3 seconds, or an escape pacemaker rate less than 40 beats per minute.[18] Patients with type I or type II second-degree AV block who are symptomatic should be transcutaneously paced. Symptomatic bifascicular block is an indication for temporary TCP. Patients with sinus node dysfunction are candidates for pacing. Sinus node dysfunction includes sinus pause with symptoms of cerebral hypoperfusion (e.g., syncope, presyncope, dizziness), chronic sinus node dysfunction with or without symptoms but with escape rates of less than 40 beats per minute, or symptomatic sinus bradycardia.

TCP electrodes should be placed on patients in anticipation of potential clinical deterioration and bradyarrhythmias in the appropriate setting.[8] For example, a patient with a beta-blocker overdose may present with a slow or normal heart rate but otherwise be asymptomatic. Hemodynamic deterioration is possible. Preparation for TCP (i.e., electrodes applied and connected to generator unit, unit in standby mode, etc.) can be easily accomplished while further efforts related to the diagnosis and treatment are undertaken. The transcutaneous pacer can be immediately activated without the delays associated with obtaining the equipment and setting up the system if the patient develops a bradyarrhythmia.

TCP can be used for overdrive cardiac pacing in event of a supraventricular or ventricular tachydysrhythmia (e.g., ventricular tachycardia, torsade de pointes, or paroxysmal supraventricular tachycardia) in the patient who is clinically stable.[19-24] A major limitation to overdrive cardiac pacing would be the maximum rate that the pulse generating unit can achieve. The technique requires pacing the patient at a rate of 20 to 60 beats per minute faster than the tachydysrhythmia.[23] Patients do not usually tolerate transcutaneous overdrive pacing due to the accompanying chest wall contractions and discomfort. The transvenous or transthoracic route is preferred for overdrive pacing of the myocardium.

CONTRAINDICATIONS

There are no absolute contraindications to TCP.[14,15] Hypothermia has been considered a relative contraindication to TCP. The associated bradycardia seen during hypothermia is thought to be a result of direct myocardial depression and decreased metabolic rate.[25,26] Concerns related to ventricular irritability, dysrhythmias, and resistance to defibrillation have led to the idea that electrical manipulation of the hypothermic myocardium should be avoided.[27] There are case reports of successful TCP use during rewarming for severe hypothermia.[28] TCP is relatively contraindicated for prolonged bradyasystolic cardiac arrest due to the overall poor resuscitation rates and outcomes of these patients.[8,29,30] TCP is no longer indicated for the treatment of asystolic cardiac arrest since there are no improvements in the rates of hospital admission or survival to hospital discharge in this setting.[8]

EQUIPMENT

- Pulse generator and monitor unit
- Pacing cable attached to the monitor
- Pacemaker patches or electrodes
  - 6×7 cm for infants and young children
  - 13×15 cm for older children and adult
- Electrocardiogram (ECG) patches or electrodes (if the pacemaker patches and unit do not monitor the patient)
- ECG cable attached to cardiac monitor (if the pacemaker patches and unit do not monitor the patient)
- Intravenous access supplies
- Sedative and analgesic medications
- Povidone iodine or chlorhexidine solution
- Skin razor

Most commercially available cardiac defibrillators used in the Emergency Department are a combination cardioverter, defibrillator, and cardiac pacer with an associated ECG monitor (**Figure 41-1**). The unit is usually available on all code carts in the Emergency Department and throughout the hospital. The Emergency Physician should become familiar with their specific institutional equipment prior to an emergent situation requiring its use.

The pacing patches are either round or rectangular and come packaged as pairs with illustrations to demonstrate proper placement

FIGURE 41-1. The cardiac monitor/defibrillator with TCP capabilities and attached surface patches.

FIGURE 41-2. TCP electrodes with diagrams demonstrating placement of the electrode.

of the electrode (**Figure 41-2**). The negative electrode will be labeled "front" or "apex." The positive electrode may be labeled "back" or "posterior." Most modern TCP electrodes are disposable, single patient use, and multipurpose. The one electrode can be used to perform cardioversion, defibrillation, ECG monitoring, and TCP. Some older units may not have this flexibility and require separate ECG leads.

PATIENT PREPARATION

If time allows, discuss the procedure with the patient and/or their representative. This should include the reason for performing the procedure, the risks of the procedure including pain-related issues and how pain will be addressed, and the benefits of the procedure including expected symptom improvement. There is a small risk of developing a ventricular tachydysrhythmia during TCP.[31] A written and signed consent documenting this conversation is always preferred. However, it is not required in an emergent situation. Documentation of the conversation in the medical record should be adequate in an emergent situation. Preparation for transvenous cardiac pacing (Chapter 43) can be simultaneously undertaken if TCP is unsuccessful.

The patient's skin should be relatively clean and free of debris. Warm, soapy water can be used as a cleansing agent. Avoid potentially flammable cleansing solvents (e.g., alcohol-based solutions). Dry the skin. Trim the back and chest hair if the patient is hirsute and the pacing patches are poorly adherent. Trimming is preferred to shaving to avoid skin disruption or abrasions that may cause increased pain, skin irritation, and bacteremia during the procedure. If shaving is required and pacing is not emergently required, apply povidone iodine or chlorhexidine to the area and allow it to dry. This will decontaminate the skin prior to shaving.

Administer sedative and analgesic medications (Chapter 159) to manage the discomfort related to the chest wall skeletal muscle contractions associated with TCP.[31] Pain levels associated with TCP were found to be moderate to moderately severe (mean of 3.2 on a 5-point scale) in 30 healthy volunteers.[9]

TECHNIQUE

ELECTRODE PAD PLACEMENT

Two pacing electrodes must be applied to the thorax (**Figure 41-2**). Place the front, anterior, or negative labeled electrode on the anterior chest wall, and centered over the apex of the heart (**Figure 41-3*A***) or over the V_3 lead position (**Figure 41-3*B***). The anterior electrode should be positioned in females by lifting the breast and placing it under the fold of the breast and against the chest wall. Place the back, posterior, or positive labeled electrode directly behind the anterior electrode and to the left of the thoracic spine, between the spine and the scapula (**Figure 41-3*C***). Avoid trapping air or debris under the pad during placement. The positive electrode may be alternatively placed on the right upper chest and the negative electrode over the apex of the heart (**Figure 41-4**).

TCP

Connect the cable from the electrodes to the pacer/monitor unit. Turn the output current (mA) dial as low as possible (**Figure 41-5**). Set the pacing rate between 80 and 90 beats per minute. Select the pacer function of the pacer/monitor unit. Turn the unit on. Gradually increase the current output by 5 to 10 mA at a time until capture is achieved (**Figure 41-6**). **Note the mA output value that is required to initiate TCP. This is known as the threshold current.** Increase the output current approximately 10%, or 5 to 10 mA, above threshold current. Maintain the output current at this level.

Start with the output current set at maximum in the near-arrest or unconscious patient. Turn the unit on. Dial the output current down until capture is lost. This is the threshold current. Raise the output to restore capture. Increase the output current approximately 10%, or 5 to 10 mA, above threshold current. Maintain the output current at this level.

Some pacer/monitor units can operate in a "standby mode." This mode can be used for patients who are at risk of developing symptomatic bradycardia but are currently clinically stable. Set the unit to "pacing mode." Attach the pacer electrodes to the patient and the unit. Initiate a brief period of TCP at a rate slightly faster than the patient's intrinsic rate to determine the threshold current and if

FIGURE 41-3. Placement of TCP electrodes. ***A.*** Anterior (negative) electrode placed over the cardiac apex. ***B.*** Anterior (negative) electrode position centered over the V_3 lead position. ***C.*** Posterior (positive) electrode position.

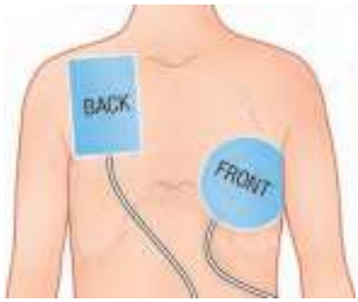

FIGURE 41-4. Alternative TCP electrode positions. The negative electrode is positioned over the cardiac apex. The positive electrode is positioned on the right anterior chest wall.

capture is possible. Turn the unit off. Set a reduced rate on the pacer dial. This rate should be below the patient's current heart rate but also be the minimal heart rate acceptable to the Emergency Physician for the patient. Switch the unit to "standby mode" and turn it on. The pacer function will automatically engage if the patient's heart rate falls below the value set on the unit.

OVERDRIVE PACING

Full cardiac resuscitation equipment should be available and ready at the bedside as rhythm acceleration and subsequent hemodynamic instability are possible.[20] The pacer electrodes and pacing unit set-up remain the same to perform overdrive TCP for a tachydysrhythmia. Administer sedative and analgesic medications as required. Set the current output at 120 mA, as this current generally will exceed the threshold value for most patients. Turn on the unit and initiate asynchronous cardiac pacing at a rate of 20 to 60 beats per minute higher than the intrinsic rate of the tachydysrhythmia.[20,23] Once capture is achieved, decrease the current output to just above the threshold current. Slowly lower the pacing rate to decrease the patient's heart rate to the desired level. TCP for overdrive pacing may not be possible due to the upper heart rate limit of the available pacing unit.

FIGURE 41-5. Monitor dials demonstrating separate defibrillation, pacing, electrical current (mA), and heart rate to set for TCP.

PACING PEDIATRIC PATIENTS

There are limited data available on the use of TCP in the pediatric population. The indications for the procedure are the same as those in adults. The technique used to accomplish TCP is the same as in an adult. TCP is often more effective in children because their smaller chest wall results in a lower transthoracic resistance to the pacing current.

There are special considerations when performing TCP in the pediatric population. TCP has been undertaken in newborns with complete AV node block.[32] This small case series of two patients, as well as others, documented significant thermal injury to the underlying skin.[32,33] This was most likely related to the newborn's thin and fragile skin. Increased monitoring of the skin surrounding the electrodes is mandatory in children, especially in cases of prolonged TCP. Pediatric-sized transcutaneous pacing electrodes are available and should be used to limit surface contact. A study comparing the use of adult versus two specially sized pediatric electrodes for TCP demonstrated a lower mean current output with the smaller pads (63 mA versus 51 to 53 mA).[34] Appropriately sized electrodes are available for children under 15 kg (33 pounds) and should be used.

Only one study has documented the outcomes of TCP in pediatric out-of-hospital cardiac arrest.[35] In six drowning victims and three sudden infant death syndrome patients who received TCP by EMS providers, only two patients achieved electrical and mechanical capture. Both patients had an initial rhythm of asystole. In total, seven patients presented with asystole and two patients presented with ventricular fibrillation. Only one patient with an initial rhythm of asystole survived to hospital discharge but was severely neurologically impaired and died 6 months later. Current guidelines do not recommend TCP for asystolic arrests in children.[5]

ASSESSMENT OF SUCCESSFUL PACING

The Emergency Physician must assess the patient for both electrical capture and associated mechanical capture. **Successful capture is characterized by a wide QRS complex, since it is ventricular in origin, and a broad T wave.** It is easy to mistake the wide, slurred afterpotential following an external pacing spike for electrical capture. **Electrical capture is best judged by the presence of a consistent ST segment and T wave after each generated pacer spike (Figure 41-6*C*).** Paced beats below the patient's intrinsic cardiac rate may not produce an associated QRS complex (**Figure 41-6*B***), and electrical capture is not achieved. The pacing rate must be increased to achieve capture. **Table 41-1** lists the common causes of failure to capture and suggested solutions.

Mechanical capture must be ensured once electrical capture is achieved. This is done with a palpable pulse rate or arterial catheter blood pressure monitoring.[11] The patient will have a pulse rate that is exactly equal to that of the paced rhythm on the cardiac monitor if mechanical capture is achieved. Assess the pulse using palpation of the carotid or femoral artery to avoid confusion with skeletal muscle contractions generated by the pacing current. Mechanical capture has not been achieved if the palpated pulse rate is less than that of the paced rate. Increase the threshold current. Mechanical capture is also achieved when the invasive arterial blood pressure line

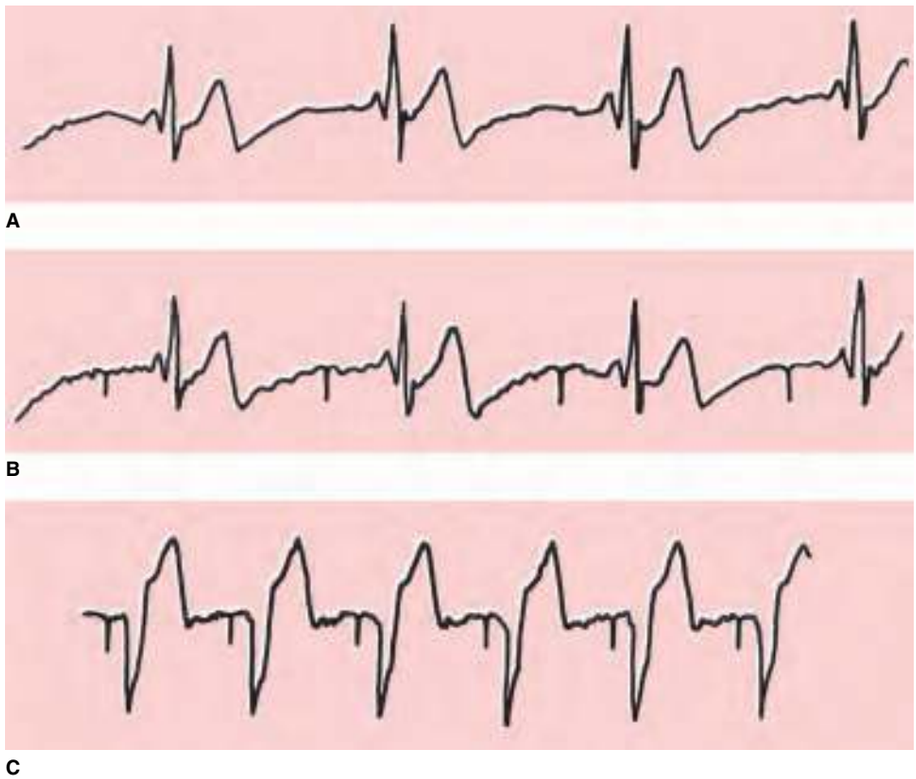

FIGURE 41-6. Assessing electrocardiographic capture with TCP. ***A.*** Intrinsic patient bradycardic rhythm. ***B.*** No electrical capture as TCP is below the threshold level. ***C.*** TCP with electrical capture.

demonstrates a pulse rate that is exactly equal to that of the paced rhythm on the cardiac monitor.

Threshold current values will vary depending on the clinical situation. Transcutaneous pacing thresholds tend to be lowest in healthy individuals or in patients with minimal hemodynamic compromise. The threshold is usually in the range of 40 to 80 mA.[21,36] Most patients are paced using a current in the range of 20 to 140 mA.[8] No clear correlation has been established between the pacing threshold and patient age, weight, body size, chest diameter, or etiology of heart disease.[8,36,37] Thresholds are usually elevated following thoracic surgery; in patients with chronic obstructive pulmonary disease, a pneumothorax, a pericardial effusion, or heavy thoracic chest wall musculature; and after positive-pressure ventilation.[8]

TABLE 41-1 Common Causes of Failure to Capture and Suggested Solutions

Etiology	Solution
Suboptimal electrode placement	Reposition electrodes to avoid the spine, scapula, and sternum
Negative electrode placed posteriorly	Place negative electrode anteriorly over the cardiac apex or the V_3 lead position
Poor skin–electrode contact	Clean skin of sweat and debris; dry skin thoroughly; trim hair
Faulty electrical contact	Check electrical connections
Generator battery depletion	Change battery; plug generator into an electric outlet
Increased intrathoracic air	Reduce positive-pressure ventilation; relieve pneumothorax
Pericardial effusion	Pericardiocentesis; pericardial window
Myocardial ischemia/metabolic derangement	Cardiopulmonary resuscitation; ventilation; correct acidosis; correct hypoxia; correct electrolyte abnormalities
High threshold	Use stimuli of longer pulse width; shave hair for improved pad attachment; apply pressure to pads; apply pads with fresh gel

Source: Modified from Ellenbogen KA, Wood MA: *Cardiac Pacing and ICDs*, 5th ed. Oxford: Blackwell, 2008.

Success rates in achieving ventricular capture vary widely depending on the setting where TCP is being used. Success rates appear to be highest (over 90%) when TCP is used prophylactically or early (e.g., within 5 minutes of bradycardic arrest).[8] A 78% success rate was reported in diverse clinical situations.[8] **The time to the initiation of TCP largely determines the success rate. There can be changes in the pacing threshold leading to capture failure with prolonged pacing.** Failure to capture may be encountered due to a variety of reasons.

Ultrasound has been used as a method to confirm electrical and mechanical capture.[38-41] It can be used to determine the ventricular contraction rate. The patient will have a heart rate exactly equal to that of the set paced rate if mechanical capture is achieved. This eliminates the artifact seen on the ECG monitor from chest wall muscle contractions.

AFTERCARE

The most important assessment in the aftercare period is to ensure continuous electrical and mechanical capture. This should be accomplished by frequent checks of the cardiac monitor, palpation of a pulse, and measurement of a blood pressure. This is more easily established using an arterial line. **Threshold current values can change with prolonged TCP.** The system can be somewhat tenuous in practical use, and frequent checks are advisable. Insert a transvenous cardiac pacer in the Emergency Department or arrange with a Cardiologist to place one in the catheterization lab if the patient requires prolonged TCP.

Patient comfort must be continuously reassessed. Pain can be due to chest wall muscle contractions or other causes (**Table 41-2**). **Do**

TABLE 41-2 Causes of Painful Transcutaneous Pacing and Suggested Solutions

Etiology	Solution
Conductive foreign body beneath electrode	Remove foreign body
Electrode over skin abrasions (shaved)	Reposition electrodes; avoid shaving beneath electrodes
Apprehensive patient or low pain tolerance	Administer parenteral narcotics and/or benzodiazepines
Sweat or salt deposits, saline, conductive jelly, blood, or vomitus on skin	Cleanse and dry skin
High threshold to pacing	Use longer pulse-width stimuli
Thoracic wall muscle contractions	Intravenous sedation and/or analgesics

Source: Modified from Ellenbogen KA, Wood MA: *Cardiac Pacing and ICDs*, 5th ed. Oxford: Blackwell, 2008.

not assume that pain is from chest wall muscle contractions until other etiologies are ruled out. Repeated doses of a sedative and/or analgesics may be required to ease the discomfort of chest wall muscle contractions. **Periodically assess the skin under the electrodes for burns or damage.** Reposition the electrodes if skin erythema or any signs of a burn are present. **This is especially important in patients who are young children, unconscious, or have altered mental status as they cannot complain of pain.**

COMPLICATIONS

There are few major complications related to this procedure.[42] One of the most important complications to consider is the failure to recognize underlying rhythm changes in the patient or loss of capture (i.e., electrical or mechanical) once the procedure is completed. A rhythm change to ventricular fibrillation is easy to miss and can be ascribed to pacing artifacts and chest wall muscle contraction. These complications are mitigated by close patient and monitor surveillance during TCP. Tachydysrhythmias related to TCP have been documented, but the overall risk for this complication is considered small.[20,23] Myocardial damage from TCP has been examined in animals and humans without significant findings.[2,43,44] Thermal injury to the skin from the electrodes is a known complication, especially in situations of prolonged TCP, and can be minimized with careful observation and electrode adjustments.[45] Pain is the most commonly reported complication (**Table 41-2**) and needs to be continually addressed. The most common etiology of the pain is from chest wall muscle contractions.

SUMMARY

TCP is a temporary method of cardiac pacing in patients with severe symptomatic bradyarrhythmias due to high-grade AV blocks, sinus node dysfunction, or bradyasystolic cardiac arrest, or rarely for overdrive pacing to suppress ventricular and supraventricular tachyarrhythmias. It is comparatively easy to perform and requires minimal training. Once capture is achieved, the current output should be set at a level slightly higher than the pacing threshold. Successful TCP can be established in 80% to 90% of patients. Discomfort and pain due to muscle contraction are the most common side effects. Most patients will require sedation and/or analgesics to tolerate TCP for a significant length of time.

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42 Transthoracic Cardiac Pacing

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INTRODUCTION

Transthoracic cardiac pacing is a historic technique of pacing the heart with an electrode introduced percutaneously into the ventricular cavity using a needle trocar introducer. There is sparse literature on transthoracic cardiac pacing, its benefits, and complications. Transthoracic pacing was a faster alternative to transvenous pacing in the patient with an acutely unstable dysrhythmia. The advent of effective and efficient transcutaneous pacing has made the indications for transthoracic pacing extremely rare. The technique of transthoracic pacing is included in this text because it is occasionally performed in situations where transcutaneous pacing is unavailable or ineffective.[1-3]

The history of electrical stimulation of the heart dates to 1862 when Walsh discussed the possibility of causing the heart to contract through stimulation of the sympathetic nervous trunk by an induced current.[4] It was largely understood by 1910 that the neuromuscular mechanism of the heart was electrically dependent. A needle electrode was used in 1928 to carry stimulating current directly to heart muscle.[5] The first successful clinical application of external cardiac pacing was in 1952 with the resuscitation of two patients in asystole following bradycardia from a high-degree atrioventricular (AV) block.[6] It was concluded that external cardiac pacing was a safe and effective means of resuscitating ventricular standstill. The devices caused significant chest pain, skeletal muscle spasm, and superficial skin burns and disrupted electrocardiogram (ECG) monitoring.[6] The quest for alternative pacing modalities continued with the refinement of the transesophageal technique in 1957.[7] The emergency use of a lumbar puncture needle introduced 5 mm into the myocardium, through which a conducting wire was introduced, was used to produce transthoracic cardiac pacing.[8] A transvenous wire catheter was passed in 1959 that successfully applied an electrical current to the endocardial surface of the right atrium.[9] Transvenous pacing subsequently became the most widely accepted method of emergency cardiac pacing until the reemergence of a modified external pacing system in 1981.[10]

ANATOMY AND PATHOPHYSIOLOGY

The heart is the only muscle of the body that generates its own electrical impulses. The initial cardiac impulse starts in the right atrium at the sinoatrial (SA) node. The sympathetic and parasympathetic nervous systems control the rate of impulse generation at the SA node. The electrical stimulus is conducted along the internal conduction pathways of the heart to the muscular atrial and ventricular walls. A delicate balance between electrolyte flux to create action potentials, myocardial integrity to allow impulses to become contractions, and an intact conduction system must be maintained. The blood supply to the conduction system of the heart originates from the right coronary artery. Arrhythmias and conduction delays are often the result of inadequate blood flow to the heart due to ventricular infarction and coronary artery occlusion.

INDICATIONS

Transthoracic cardiac pacing is a simple procedure and can be accomplished rapidly. The indications for transthoracic pacing appear extremely limited. There is only a 40% success rate in achieving pacing by the transthoracic route.[11] Transcutaneous pacing success rates of greater than 80% have been routinely demonstrated.[12] **Transthoracic pacing should be reserved to clinical situations where there is no transcutaneous pacing available, when transcutaneous pacing in the perimorbid patient has been unsuccessful, or when the placement of a transvenous pacer is thought to be too time consuming.[13,14]**

Unstable bradydysrhythmia is the obvious indication for emergency cardiac pacing. Several case reports document successful transthoracic pacing in patients with asystolic arrest.[1] Other studies document successful pacing out of asystole in patients with in situ pacemakers.[15] A study of approximately 300 patients with witnessed or early asystole demonstrated no improved outcome with early transcutaneous pacing intervention by first response emergency medical personnel.[16] It was concluded that emergency transthoracic cardiac pacing has a very low success rate in asystole but may be lifesaving in the rare case.

Transthoracic cardiac pacing would be most effective following cardiac arrest from primary cardiac disease.[13] Transthoracic cardiac pacing may not be effective in cardiac arrest secondary to hypovolemia (e.g., trauma), severe electrolyte abnormalities, acid-base abnormalities, sepsis, or drug intoxication. Transthoracic cardiac pacing may be lifesaving when bradycardia or asystole secondary to prolonged ischemia during hypovolemic shock persists despite correction of the underlying pathology.

The use of transthoracic cardiac pacing in unstable patients is more controversial. Transthoracic cardiac pacing may be considered in patients with unstable sinus bradycardia, junctional bradycardia, atrial fibrillation with high-degree AV block, and AV dissociation with inadequate ventricular response.[13]

CONTRAINDICATIONS

Transthoracic cardiac pacing should not be performed in hemodynamically stable awake patients. It is contraindicated if the patient has a dysrhythmia that could be quickly and easily corrected by medication, cardioversion, or electrical defibrillation.

Transthoracic cardiac pacing may be ineffective in electromechanical dissociation and ventricular fibrillation. The heart becomes insensitive to pacemaker activity in ventricular fibrillation.[17-20] Any mode of pacing is ineffective in pulseless electrical activity.[17-20] A pacemaker is ineffective in patients with prolonged cardiac arrest.[16] Patients with cardiac arrest for more than 5 to 10 minutes could not be resuscitated with the use of cardiac pacing, but patients who had pacemakers placed within 2 to 4 minutes after cardiac standstill were successfully resuscitated.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Transthoracic cardiac pacing wires/kit
- Pacemaker generator
- 10 mL syringe
- Water-soluble lubricant
- Sterile gauze 4×4 squares
- Sterile drapes
- Sterile gloves and gown
- Face mask
- Cap
- Ultrasound machine (optional)
- Ultrasound transducer (optional)
- Sterile ultrasound gel (optional)
- Sterile ultrasound transducer cover (optional)

The instrumentation for transthoracic cardiac pacing comes in a sterile, one-time-use, prepackaged kit. The equipment consists of a 37 cm bipolar J-shaped pacing wire, a 6 inch 18 gauge blunt-end steel cannula with a pointed inner trocar, and a plastic electrical connector that accepts the pacing wire and can be attached to a battery-powered external pacemaker generator (**Figure 42-1**). The cannula and trocar function as a catheter-over-the-needle.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient if they are awake and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. A signed consent is not required, and time is often lacking to obtain a signature. Document in the medical record that the patient was informed of the risks and benefits of the procedure. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

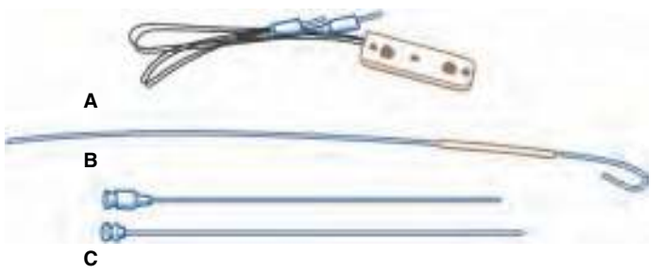

FIGURE 42-1. Equipment required for transthoracic cardiac pacing. ***A.*** Electrical connector. ***B.*** Bipolar pacing wire with a sleeve. ***C.*** Blunt steel cannula with pointed trocar.

Place the patient supine. Clean the chest and subxiphoid area of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the chest and subxiphoid area. Allow it to dry if time permits. Apply sterile drapes to delineate a sterile field.

Cardiopulmonary resuscitation (CPR) can be continued during most of the procedure but should be stopped while the intracardiac needle is being inserted to avoid possible damage to the lung or myocardium. Full ventilation of the lungs is recommended during subxiphoid cannula placement to depress the diaphragm and minimize the risk of injury to the liver and stomach. Insert a nasogastric tube to decompress the stomach prior to performing the procedure. Fully monitor the patient with a noninvasive blood pressure cuff or arterial line, pulse oximetry, and cardiac monitor. Obtain IV access (e.g., intraosseously, intravenously, or central venously).

The Emergency Physician should prepare themselves and the equipment. Don a cap, mask, sterile gloves, and sterile gown. Open the transthoracic cardiac pacing kit onto a sterile field. Lubricate the trocar liberally and insert it securely into the steel cannula. Prepare the ultrasound transducer if using ultrasound with the procedure. Have an assistant apply ultrasound gel over the footprint of the transducer. Instruct the assistant to place the gel-coated transducer in the sterile cover while you hold it. Have the assistant place sterile ultrasound gel on the patient's upper abdomen and lower chest.

TECHNIQUE

Quickly identify the anatomic landmarks necessary to perform this procedure and determine the approach. The cannula-over-the-trocar can be inserted through the left fifth intercostal space parasternally, 4 cm lateral to the midsternal line, or 6 cm lateral to the midsternal line (**Figure 42-2*A***). Aim the tip of the cannula-over-the-trocar toward the second costal cartilage. Alternatively, insert the cannula-over-the-trocar through the left xiphocostal junction and aimed toward either the right shoulder, left shoulder, or sternal notch (**Figure 42-2*B***).

If bedside ultrasound is available, identifying the right ventricle via the parasternal approach through the fifth intercostal space may be the technique of choice. Without ultrasound, the simplest and quickest approach is to insert the trocar at the left xiphocostal junction and aimed toward the sternal notch. This technique is described below.

Briefly stop CPR. Insert the cannula-over-the-trocar from the left xiphocostal region at a 30° to 40° angle to the skin and directed toward the sternal notch (**Figure 42-2*B***). Advance the cannula-over-the-trocar approximately three-fourths of its length. Hold and stabilize the cannula. Withdraw the trocar. Attach a 10 mL syringe to the cannula. Apply negative pressure to the syringe. The aspiration of blood into the syringe confirms proper positioning of the cannula within the ventricle. If blood is not aspirated, withdraw the cannula and restart the procedure. Placement is ideally confirmed with bedside ultrasound.

Insert the transcutaneous pacing wire. Advance the plastic sheath over the pacing wire until it straightens out and covers the J-shaped end of the pacing wire. Insert the plastic sheath into the cannula hub. Advance the pacing wire through the cannula and into the ventricle. Stop advancing the pacing wire when 4 to 5 cm remains outside the cannula. **No resistance should be felt while the pacing wire is being advanced. The cannula is not within the ventricle if resistance is felt. Remove the cannula and pacing wire as a unit and restart the procedure.** The pacing

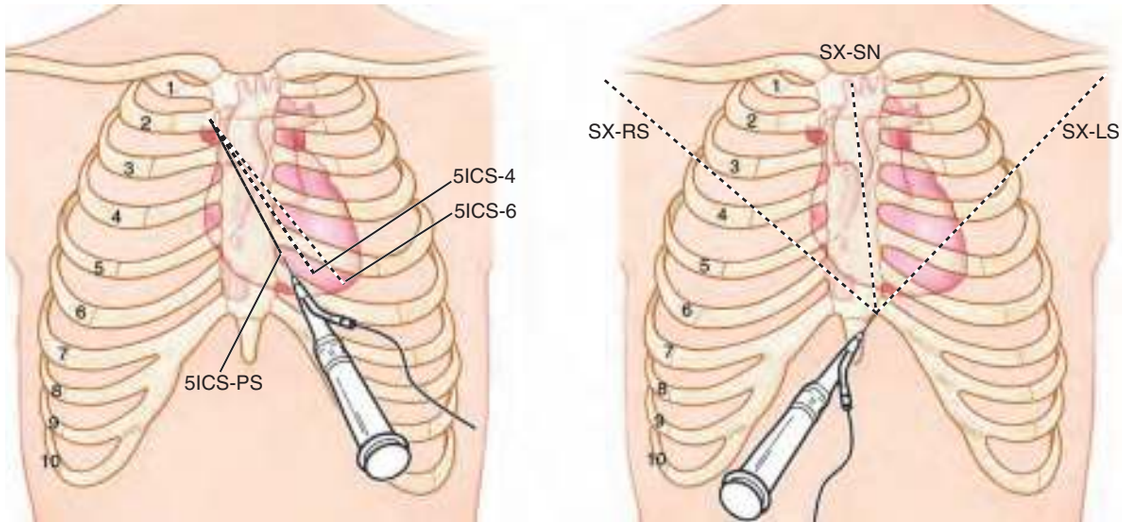

FIGURE 42-2. Placement of a percutaneous transthoracic cardiac pacemaker. ***A***. Parasternal approaches. 5ICS-PS, fifth intercostal space immediately to the left of the sternum; 5ICS-4, fifth intercostal space, 4 cm from the midsternal line; 5ICS-6, fifth intercostal space, 6 cm from the midsternal line. ***B.*** Subxiphoid approaches. SX-RS, subxiphoid–right shoulder; SX-SN, subxiphoid–sternal notch; SX-LS, subxiphoid–left shoulder.

wire will reform its J-shape when it is inside the ventricle. Hold the pacing wire securely. Withdraw the cannula over the pacing wire. **Do not release the hold on the proximal end of the pacing wire outside the patient's thorax to prevent it from slipping inside the thorax**.

Insert the proximal end of the pacing wire into the plastic connector. Secure it with the screws in the body of the connector. Connect the positive and negative terminals of the plastic connector to the pacemaker generator. Turn on the pacemaker generator. Set the pacing rate at 70 to 90 beats per minute in an asynchronous mode. Set the current output to the maximum milliampere rate on the pacemaker generator. Demonstrate myocardial capture of the electrical stimulus (i.e., each pacer spike followed by a QRS complex). Lower the current output until 1:1 pacing is lost. Gradually increase the current output to attain stimulation threshold when 1:1 capture is regained. The optimal current output is two to three times the stimulation threshold. Change the mode of the pacemaker to a demand pacemaker with a backup rate of 60 to 70 beats per minute. A complete description of the functioning of the pacemaker generator is reviewed in Chapter 41.

Pacer spikes should be visualized on the ECG tracing on the cardiac monitor. Check the contact between the pacer wire and electrical connector if pacer spikes are not visualized. Check the batteries in the pacemaker energy source. Pacer spikes not followed by myocardial capture usually indicate inadequate positioning of the pacing electrode. Gently manipulate the transthoracic pacemaker wire to change its position.

ASSESSMENT

The positioning of the pacing wire should be verified by chest radiograph or bedside ultrasound.[21] Ideal placement of the pacing wire should be at the mid-myocardial septum to preserve right ventricle performance while preventing dyssynchrony of the ventricles. This is important to consider as failing to do so can result in a lower ejection fraction from the left ventricle and impaired blood flow to the coronary arteries.[22,23] Obtain a 12-lead ECG to document capture and to verify the positioning of the pacing wire based on the QRS configuration in the ECG. **A left bundle branch block configuration will be demonstrated on the ECG if the pacing wire is in the right ventricle.** A pacing wire in the right atrium or left ventricle will still pace the myocardium, but the ECG will have a different QRS configuration. **An atrially positioned pacing wire will be ineffective if the patient is in AV block.**

AFTERCARE

Secure the pacing wire to the skin with 3–0 nylon suture. A transvenous or permanent pacemaker should be inserted as soon as possible. Consult a Cardiologist immediately and admit the patient to an Intensive Care Unit.

COMPLICATIONS

Analysis of the complications of transthoracic cardiac pacing is greatly limited by the paucity of short-term survivors and the absence of radiographic or pathologic evaluation of nonsurvivors. Complications include laceration of the right atrium, ventricles, coronary arteries, great vessels, vena cava, stomach, liver, and lung. Hemopericardium is a ubiquitous finding in some autopsy studies, and cardiac tamponade has been reported.[24,25] Pneumothorax has been reported and is a particular concern in persons receiving positive-pressure ventilation.[26]

SUMMARY

The technique of transthoracic cardiac pacing has been clinically feasible for more than 50 years, but there is extremely limited literature supporting its regular use. The technique is simple and can be performed in less than a minute. However, it carries a high risk for multiple significant complications. There is no clear understanding of the effect of transthoracic cardiac pacing on the outcome of cardiac arrest. Most of the available information comes from animal studies, retrospective analysis, and anecdotal data.

Transthoracic cardiac pacing may be useful in the setting of cardiac arrest with asystole or a pulseless idioventricular rhythm. It may be performed if transcutaneous cardiac pacing is not available or effective. Transthoracic cardiac pacing can be initiated promptly

in the hemodynamically unstable patient with drug-resistant bradycardia producing cardiovascular collapse or lethal escape rhythms whose clinical condition does not warrant a delay to insert a transvenous pacing catheter. Bedside ultrasound may be useful in guiding and confirming the appropriate placement of the transthoracic pacing device.

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43 Transvenous Cardiac Pacing

Myles C. McClelland, Thuy Nguyen, and Alfred ~~Coates~~ III

INTRODUCTION

Emergency cardiac pacing can be accomplished by several methods. These include epicardial, esophageal, transcutaneous, transthoracic, and transvenous pacing. Emergency cardiac pacing can be a temporizing and lifesaving technique that should be familiar to Emergency Physicians.[1-3] It will allow the patient to maintain a cardiac rhythm while providing oxygen and nutrients to the vital organs.

The earliest use of electricity to stimulate the heart can be found in an essay written in the late 1700s.[4] It discusses the use of electric current and artificial ventilation to revive victims of drowning. Transvenous pacing was first attempted on dogs in 1905. The transvenous approach in humans was developed in 1959 using a stiff pacing wire. Semiflexible pacing wires were developed in 1964 and were placed using fluoroscopic guidance. The demand pacemaker was developed in 1966. Catheter technology improved with the semi-floating catheter in 1969 and the balloon tip catheter in 1973. The technology and technique have since been developed to allow successful transvenous cardiac pacing in humans. It involves the placement of a pacing wire through the central venous circulation and into direct contact with the myocardium of the right ventricle.

ANATOMY AND PATHOPHYSIOLOGY

The heart is the only muscle of the body that generates its own electric impulses (**Figure 43-1**). Its automaticity and subsequent rhythmic contractions propel blood to the tissues of the body. The initial cardiac impulse starts in the right atrium of the heart at the sinoatrial (SA) node. The sympathetic and parasympathetic nervous systems control the rate of impulse generation at the SA node. The electric stimulus is conducted along the internal conduction pathways of the heart to the muscular atrial and ventricular walls. A delicate balance between electrolyte flux to create action potentials, myocardial integrity to allow impulses to become contractions, and an intact conduction system must be maintained. **Conduction system problems are often the result of inadequate blood flow to the heart due to ventricular infarction and coronary artery occlusion.** The blood supply to the conduction system of the heart

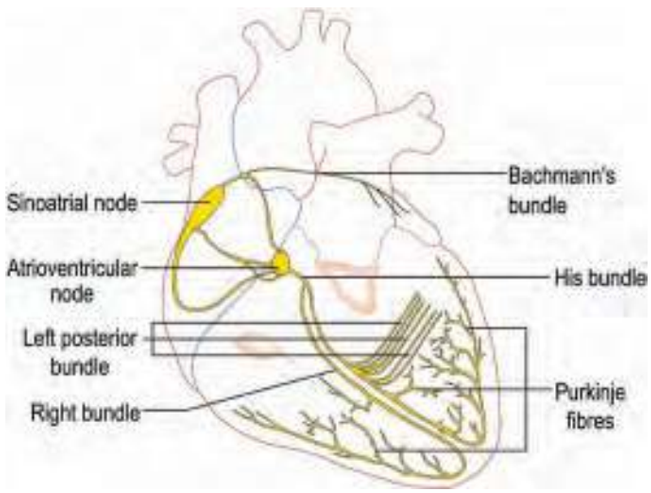


FIGURE 43-1. Conduction system of the heart. (Used from Madhero88, https://commons.wikimedia.org.)

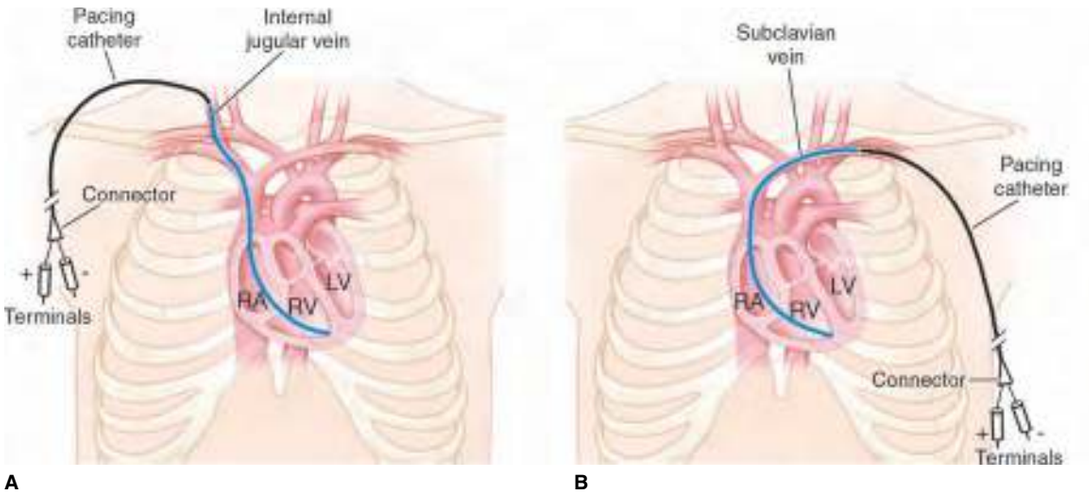


FIGURE 43-2. Common sites for introducing a transvenous pacing catheter. ***A.*** The right internal jugular vein. ***B.*** The left subclavian vein.

usually originates from the right coronary artery. **Occlusion of the right coronary artery can result in arrhythmias and conduction delays.**

A transvenous pacing catheter may be introduced through the femoral, internal jugular, or subclavian veins.[5] **The right internal jugular vein and left subclavian vein are the recommended sites (Figure 43-2). These routes allow a more direct and easy access for the pacing catheter to enter the right ventricle**. The right internal jugular vein is preferred. It allows a relatively straight line of access through the superior vena cava and right atrium into the right ventricle.[6] The left subclavian vein is a good second choice if access to the right internal jugular vein is not accessible. The left subclavian vein is the site of choice for a permanent pacemaker if required. Thus, many will not use this site. The other routes are technically more difficult to use and often require fluoroscopy for proper placement of the pacing catheter. The right subclavian vein and left internal jugular vein require several turns for the pacing catheter to navigate. This increases the difficulty of entering the right ventricle.

The femoral vein is often used in infants and younger children to insert a transvenous cardiac pacing catheter (**Figure 43-3**). Insertion of the pacing catheter via the femoral vein often requires fluoroscopy. The disadvantages of using the femoral vein for vascular access include the potential for deep venous thromboses, infection, restricted mobility, and thrombophlebitis. The infant and young child's relatively large head and short neck make access to the internal jugular vein difficult. The subclavian vein in an infant and child is situated more posterior to the clavicle than in an adult. This makes it more difficult to access the subclavian vein while increasing the chance of causing a pneumothorax.

Pacing the left ventricle through a femoral artery approach has been suggested in emergent situations.[7] These instances usually involve the inability to obtain venous access due to scarring, previous procedures, or venous thrombosis. This nonstandard approach has been used successfully in situations when transvenous cardiac pacing was not feasible.[7,8] This technique cannot currently be recommended for routine use but is a potential alternative technique for use in dire circumstances.

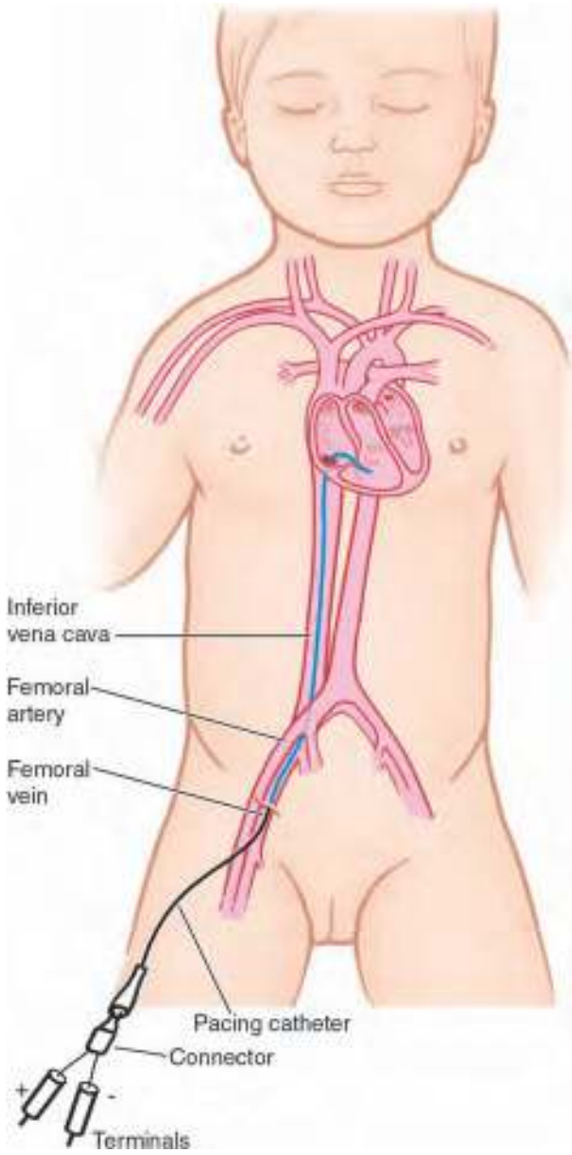


FIGURE 43-3. The femoral vein is used in children to access the central venous circulation and introduce a transvenous pacing catheter.

INDICATIONS

The indications for transvenous pacing are the same as for other methods of cardiac pacing.[1,9-11] Patients with atrial or ventricular arrhythmias that require overdrive pacing, atrioventricular

dissociation, bradycardia unresponsive to drug therapy, and conduction delays that may degenerate into complete heart block can benefit from transvenous pacing.[4,6,12-14] Specific bradycardic conditions that may benefit from transvenous cardiac pacing include atrial fibrillation with a slow ventricle response rate, second-degree heart block, sick sinus syndrome, sinus arrest, sinus bradycardia, sinus node dysfunction, symptomatic bradycardia, and third-degree heart block. Patients with myocardial infarctions or new heart blocks may require cardiac pacing. Tachyarrhythmic conditions that may benefit from overdrive cardiac pacing include atrial fibrillation, atrial flutter, supraventricular tachycardias not responsive to drugs or cardioversion, torsades de pointes, ventricular tachyarrhythmias due to drug toxicity (e.g., quinidine or digoxin), and Wolfe-Parkinson-White tachyarrhythmias. Transvenous pacing may be used in patients who do not tolerate transcutaneous pacing or whose heart does not capture with transcutaneous pacing. A transvenous pacing catheter may be temporarily inserted to pace the myocardium if a permanent pacemaker is not functioning. A more complete discussion on the indications for cardiac pacing is presented in Chapter 41 on transcutaneous cardiac pacing.

Transcutaneous pacing is a first option that is easy to initiate. It may be replaced with transvenous pacing. Transcutaneous pacing pads will not stick very well to hairy or diaphoretic patients. Avoid transcutaneous pacing over an implantable defibrillator. Transcutaneous pacing requires a lot of energy to the chest wall. This may be uncomfortable to the patient. Transvenous pacing avoids giving the patient narcotics, which are needed for transcutaneous pacing pain, resulting in possible blood pressure drops. Pulses are easier to palpate using transvenous pacing. Chest wall artifacts with transcutaneous pacing may interfere with arrhythmia detection.

CONTRAINDICATIONS

Do not insert a transvenous pacing catheter into the heart in patients who are hypothermic. These patients have increased irritability of the myocardium and are prone to life-threatening ventricular fibrillation if the pacing wire contacts the heart muscle.[4,6] Relative contraindications to the placement of a transvenous pacing catheter include digoxin toxicity and other drug ingestions that may increase the irritability of the myocardium. Patients who are asystolic for extended periods of time have a low likelihood of successful resuscitation.[6] These patients are not candidates for a transvenous pacer. Do not insert a pacing catheter through infected skin or areas with any skin lesions (e.g., burns, cellulitis, or dermatides) to prevent possible infections (e.g., endocarditis, myocarditis, or sepsis). Avoid areas that contain subcutaneous devices (e.g., permanent pacer, AICD, and Port-A-Cath) and choose an alternative site. Do not prophylactically place a transvenous pacing catheter in a patient with a myocardial infarction unless they have a new heart block or symptomatic bradycardia. Patients taking anticoagulants, with bleeding diatheses, or on concurrent thrombolytic therapy should not have a transvenous pacing catheter placed except in emergent situations due to the risk of hemorrhage and bleeding complications. Other relative contraindications include the presence of a prosthetic tricuspid valve, coagulopathy, distortion of local and anatomic landmarks, and known abnormal cardiac anatomy.

EQUIPMENT

- Flexible, flow-directed, transvenous cardiac pacing catheter (**Figure 43-4**)
  - 3 or 4 French for infants and children
  - 5 or 6 French for adolescents and adults
- Pacemaker generator (**Figure 43-5**)

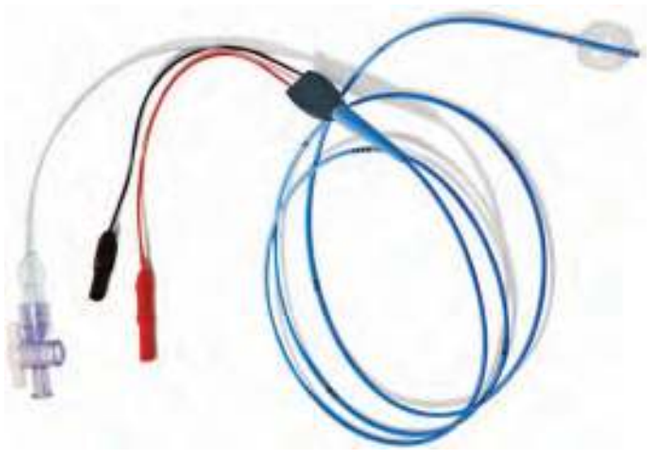


FIGURE 43-4. An example of a flexible, flow-directed transvenous pacing catheter.

- Spare battery for pacemaker
- Sterile drapes
- Sterile gloves and gown
- Face mask with face shield or goggles
- Hat
- Povidone iodine or chlorhexidine solution
- Cordis or Swan introducer catheter kit, one size larger than the pacing catheter
- Cardiac monitor
- Local anesthetic solution
- 3–0 nylon suture
- Gauze squares
- Skin tape
- Alligator clips and connecting wire
- Towels for shoulder rolls
- Defibrillator
- Airway management equipment
- Resuscitative drugs
- Ultrasound (US) machine
- US transducer, 3.5 MHz curvilinear or cardiac array
- Sterile US gel
- Sterile US transducer covers

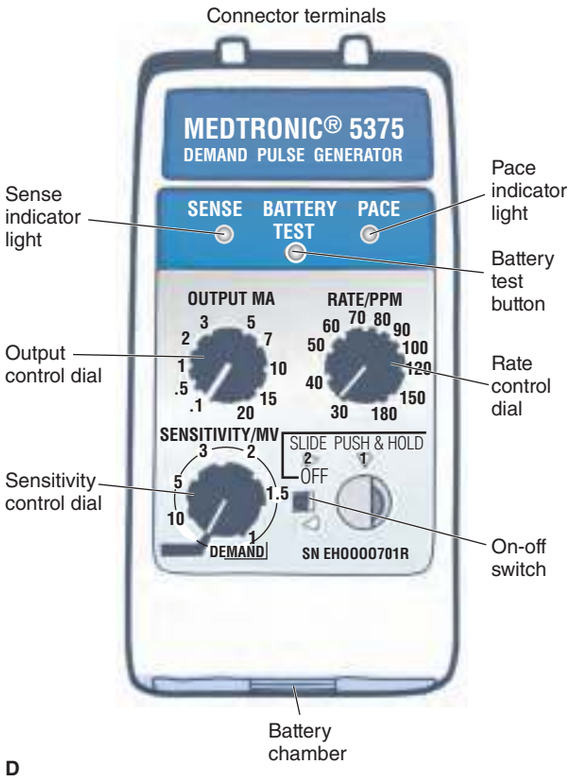
Transvenous cardiac pacing kits are commercially available. They contain the central venous introducer catheter, transvenous pacing wire, and all the required supplies except the pacemaker generator. These single-use and disposable kits are convenient and worth the expense so that time is not expended gathering multiple kits and supplies in an emergent situation when time may be of the essence.

The pacemaker generator is a simple device. Examples are seen in **Figure 43-5**. Newer models of pacemaker generators have digital displays and other more sophisticated pacing options but function essentially the same as older models. **The Emergency Physician must be familiar with the pacemaker generator and its use prior to needing it in an emergent situation**. The on/off switch is used to turn the unit on. In the "on" position, a spring-loaded safety prevents the unit from accidentally being turned off. The rate-control dial allows adjustments to the number of pacing stimuli per minute. The upper rate limit is often inadequate if overdrive pacing is required. A pacemaker generator with higher rates for overdrive

A

B

C



D

FIGURE 43-5. Examples of pacemaker generators.

pacing is available. These are rarely kept in the Emergency Department but are usually available from the hospital catheterization lab. The pace indicator light is illuminated whenever a pacing stimulus is generated. The sense indicator light is illuminated whenever a cardiac impulse is sensed. The battery test button is used to determine if the battery has sufficient voltage to operate the pacemaker generator. Depress the battery test button to check the battery voltage. The battery has sufficient voltage if both the pacing and sensing indicator lights illuminate simultaneously. The output control dial is used to adjust the amplitude of the stimulus current. The sensitivity control dial is used to suppress the pulse generator. The bottom of the pacemaker has an access panel under which the battery is located. The top of the pacemaker has positive and negative terminals where the electrodes of the pacemaker catheter insert. Numerous pacemaker

generators are available, each with its own idiosyncrasies. The above instructions and functions are similar to most models.

The pacemaker wires are enclosed in a catheter and come in a variety of lengths and sizes (**Figure 43-4**). They are typically 100 cm in length with markings at intervals of every 10 cm. They come in both flexible and rigid styles. **The rigid catheters are not often used due to the possibility of venous and myocardial perforation.** The flexible catheters have a balloon at the tip. It allows the catheter to flow with the blood into the chambers of the heart.

The pacemaker catheter should ideally be inserted under electrocardiographic (ECG) guidance. An insulated wire with an alligator clip at each end is required. One end is attached to the pacemaker wire and the other to the ECG lead. This allows the Emergency Physician to observe the ECG waveforms as they change while the catheter is advanced through the heart.[6,12,15] This technique is known as ECG positioning and requires the patient to have intrinsic cardiac activity.

PATIENT PREPARATION

Explain the risks, benefits, and possible complications to the patient and/or their representatives. An appropriate representative may accept for the patient if the patient is unable to consent. The patient may also give verbal consent if they are unable to sign but fully understand the risks and benefits of the procedure. Don full personal protective equipment to protect from contact with the patient's blood and body fluids. This will also partially protect the patient from an iatrogenic infection. **While time is of the essence and this is an emergent procedure, aseptic technique should be followed.**

Place the patient supine and, if possible, in the Trendelenburg position. Place the patient on continuous pulse oximetry, cardiac monitoring, and supplemental oxygen. Establish peripheral intravenous access. Clean and prep the skin in a sterile fashion with povidone iodine or chlorhexidine at the site chosen to access the central venous system. Place rolled towels under the patient's shoulders if the internal jugular vein or the subclavian vein is being used as the site of vascular access. Turn the patient's head to the opposite side of venous access. Apply sterile drapes to fully cover the patient except the site used for vascular access.

TECHNIQUE

Access the central venous circulation by placing a Swan or Cordis introducer catheter sheath. Some authors recommend using the supraclavicular approach for central venous access.[16] This approach may decrease complications due to well-defined surface landmarks, high success rates of vascular access, minimal interference with other procedures, location away from the pleural dome, avoidance of scarring and thrombosis from previous central venous access attempts, minimization of catheter movement, and having a straight path into the right ventricle. Refer to Chapter 63 for the complete details on inserting central venous catheters.

Prepare the pacing catheter (**Figure 43-6**). Attach the precordial lead V_1 to the pacemaker catheter negative terminal. This is accomplished using an insulated wire with an alligator clip at each end. Attach one alligator clip to the negative pacemaker wire. Attach the other alligator clip to the V_1 lead of the ECG monitor. Inflate the pacing catheter balloon with 1.5 mL of air in a container of sterile saline to assess the integrity of the balloon. The presence of bubbles in the saline indicates a balloon leak. Turn on the ECG monitor and set it to lead V_1. Touch the tip of the pacing catheter and observe the monitor to confirm that the monitor is recording. A large pressure wave should be seen that soon returns to baseline.

FIGURE 43-6. The negative pacemaker terminal is connected to an insulated wire. The insulated wire will be connected to the ECG lead V_1.

Insert the pacemaker catheter through the rubber diaphragm of the central venous introducer sheath. Advance the catheter 10 cm through the sheath. This ensures that the pacing catheter balloon is past the introducer catheter and within the vascular system. Inflate the balloon with 1.5 mL of sterile saline. **Slowly advance the catheter while always observing the ECG monitor (Figure 43-7).** The P wave and the QRS complex are both small in amplitude and inverted in the subclavian or internal jugular vein (**Figure 43-7*A***). The P wave increases in amplitude but is still inverted while the QRS complex is unchanged in the superior vena cava (**Figure 43-7*B***). A large P wave with a negative polarity and a small QRS complex will be observed when the pacing catheter reaches the right atrium (**Figure 43-7*C***). Continue to advance the catheter. The P waves become upright and the QRS complex increases in amplitude as the lower atrium is entered (**Figure 43-7*D***). Continue to advance the catheter into the right ventricle. **The QRS complex should appear normal on the V_1 lead.** The P waves are upright with a large-amplitude QRS complex when the catheter is floating freely in the right ventricle (**Figure 43-7*E***). Stop advancing the catheter once the right ventricle is entered and deflate the balloon. Slowly advance the catheter until ST-segment elevation is observed (**Figure 43-7*F***). This indicates that the catheter is abutting the right ventricular wall.

The transvenous pacing catheter may occasionally not enter the right ventricle, or it may advance past the right ventricle (**Figure 43-8**).[17] The amplitude of the P wave and QRS complex will decrease if the catheter exits the right atrium and enters the inferior vena cava (**Figure 43-7*G***). Withdraw the catheter several centimeters until the atrial waveforms are again seen (**Figures 43-7*C* and *D***) and then readvance the catheter. The P wave will become negative and the QRS amplitude will decrease if the catheter exits the right ventricle and enters the pulmonary artery (**Figure 43-7*H***). Withdraw the catheter several centimeters until the right ventricle waveforms are again seen (**Figures 43-7E**) and then readvance the catheter.

Connect the pacemaker generator to the catheter (**Figure 43-9**). Disconnect the negative terminal of the pacemaker catheter from the ECG lead. Connect the pacemaker catheter terminals on the proximal end of the catheter to the negative and positive terminals of the pacemaker generator. **The positive (+) lead connects to the proximal port on the pacemaker generator. The negative (–) lead connects to the distal port on the pacemaker generator.** Set the pacemaker generator on demand mode with a rate of 70 to 80 beats per minute.

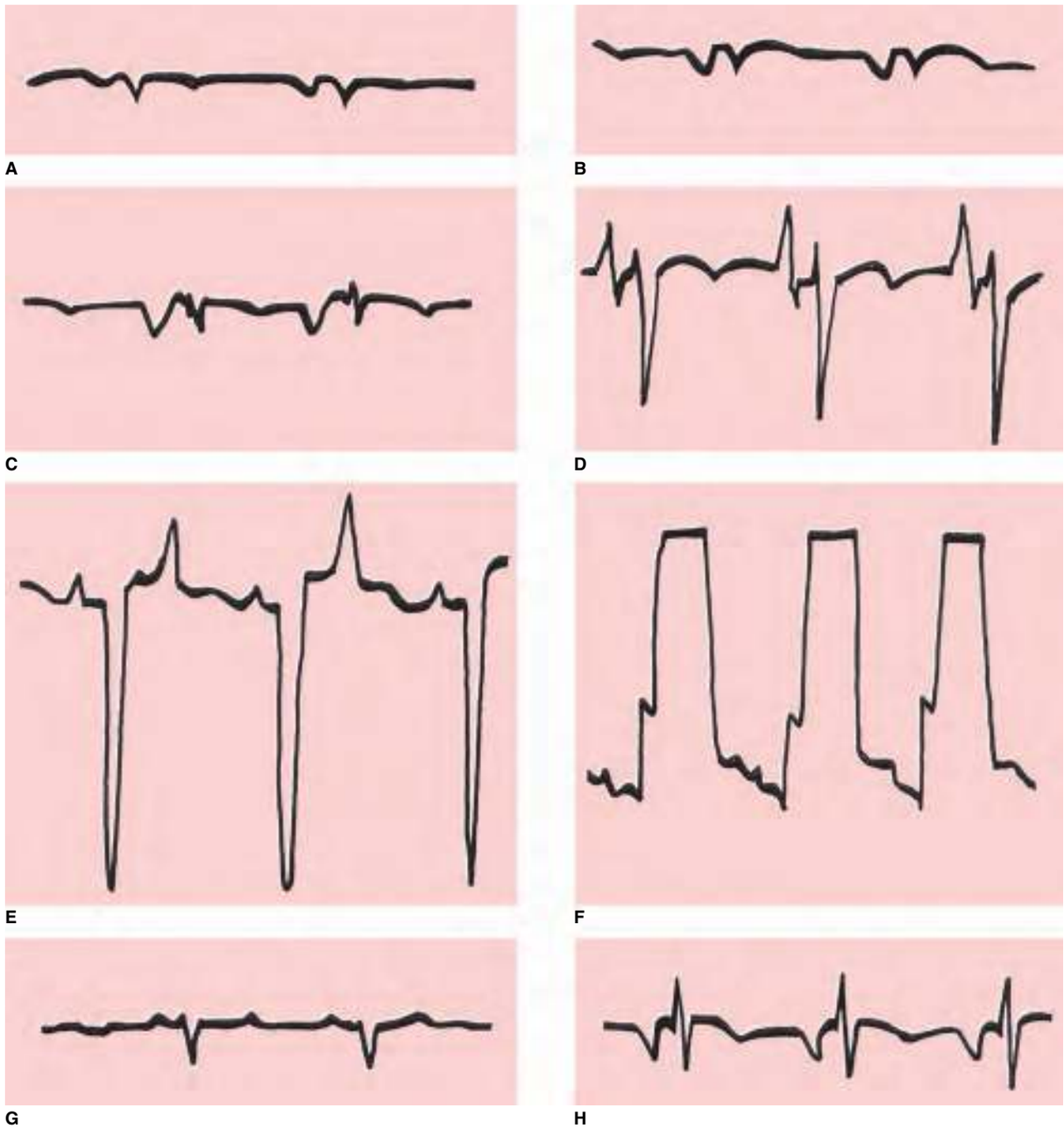


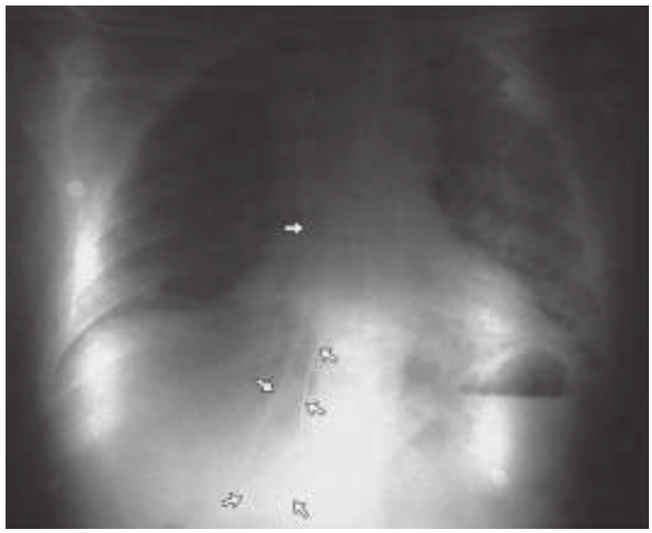
FIGURE 43-7. Typical ECG tracings seen with the transvenous pacing catheter within the different anatomic sites. ***A.*** The subclavian or internal jugular vein. ***B.*** The superior vena cava. ***C.*** The high right atrium. ***D.*** The low right atrium. ***E.*** Free floating in the right ventricle. ***F.*** Abutting the right ventricular wall. ***G.*** The inferior vena cava. ***H.*** The pulmonary artery.

Start with 5 mA of energy on the output dial. Turn on the pacemaker. Increase the energy until capture is seen on the monitor. This is signaled by pacing spikes and a wide QRS complex in lead V_1 in a left bundle branch pattern. **Decrease the pacemaker generator output to just below where pacing stops once capture is attained. This is known as the threshold point. Resume pacing at 2 mA above the threshold point.**[6]

ULTRASOUND-GUIDED TECHNIQUE

It is often difficult to successfully place the tip of the pacing catheter into the apex of the right ventricle. Lead placement can be guided and confirmed with the assistance of ultrasonography.[18,19] The pacing electrode is easily visible on US, and its correct position can be confirmed (**Figure 43-10**). Aguilera and colleagues reported success in eight of nine patients in whom US-guided transvenous pacing was attempted in the Emergency Department.[19] Initial misplacement of the electrode was visualized in three patients and allowed correct repositioning. They concluded that US has promising potential use as an adjunct in emergency cardiac pacing. US can delineate the right ventricle as well as provide quick and noninvasive confirmation of lead placement. It is useful as an aid to detecting and repositioning misplaced pacer leads. Consider using US in conjunction with ECG guidance given the time-sensitive nature of transvenous pacing.

The subxiphoid US view is preferred because it allows visualization of the right atrium and right ventricle. It is a view easily obtained with the patient in the supine position. The subxiphoid view is likely



A

B

FIGURE 43-8. The transvenous pacing catheter loops in the inferior vena cava and then enters the heart. ***A.*** Radiograph. ***B.*** US. (Used with permission from reference 17.)

FIGURE 43-9. The transvenous pacing catheter connected to the pacemaker generator.

FIGURE 43-10. Subxiphoid cardiac US image. The transvenous pacing electrode (*large arrow*) is a linear hyperechoic structure passing through the right ventricle to its apex. The interventricular septum is denoted by the *small arrows*.

to be the cardiac window with which Emergency Physicians are most familiar and comfortable. The transvenous pacing electrode is a strong reflector of ultrasound waves and will appear as a linear hyperechoic structure (**Figure 43-10**).[19] The electrode can be seen passing through the right ventricle to the apex (**Figure 43-10**). The apical cardiac view is an option. Obtaining an apical view is more technically difficult and is preferentially performed with the patient in the left lateral decubitus position, which may not be practical. **Care must be taken not to mistake the ventricular septum or wall for the electrode.**

There are limitations in the use of US. Visualization may be difficult in the obese patient or those with chronic obstructive pulmonary disease and expanded lung volumes. Mechanical heart valves may cause significant artifacts that obscure the images. The pacing catheter often lies in multiple echo planes, making visualization more difficult. The lack of catheter visualization may require multiple repositionings of the catheter. Visualization of the catheter may require tilting of the transducer, looking at several different angles with the transducer, and looking at additional views of the heart. All these maneuvers can result in delays before being able to pace the heart.

Visualize the patient's heart using a subxiphoid US view with a 3.5 MHz curvilinear array transducer. Instruct an assistant to hold the transducer in position while the Emergency Physician performs the procedure. Insert the pacing catheter as described above using the ECG-guided technique. Visualize the pacing catheter using US after it enters the right atrium and as it enters the right ventricle, as also noted by the changing ECG waveforms. Continue to advance the pacing catheter under US guidance until its tip is lodged in the apex of the right ventricle (**Figure 43-10**). This may require manipulation of the pacing catheter and/or US transducer to maintain visualization. Continue the remainder of the procedure as noted in the previous section. Ventricular capture is noted as cardiac contractions on the US monitor that are at the same rate as the pacemaker generator setting.

ALTERNATIVE TECHNIQUE

Blind transvenous catheter placement is an alternative. The pacing catheter is inserted "blindly" and without ECG guidance. This technique is often used when alligator clips are not available to connect

the pacing catheter terminals to the ECG monitor. **Always use a flexible catheter. Never place a rigid catheter blindly due to the risk of myocardial perforation**.

Prepare the catheter. Test the balloon as described previously. Make sure the pacemaker generator is off. Connect the pacemaker catheter terminals to the pacemaker generator. Insert the pacemaker catheter through the rubber diaphragm of the central venous introducer sheath. Advance the catheter 10 cm through the sheath. Inflate the balloon with 1.5 mL of sterile saline.

Turn on the pacemaker. **Set the pacemaker on demand mode with a rate twice the patient's native heart rate**. This usually ranges from 80 to 120 beats per minute. Set the output dial to 1.5 to 2.0 mA.

Advance the catheter while observing the sensing indicator light. The sensing indicator will illuminate with every other native heartbeat when the catheter enters the right ventricle. Stop advancing the catheter. Deflate the balloon. Increase the output dial to 10 mA. Slowly advance the catheter until ventricular capture occurs on the cardiac monitor. **Electrical capture is indicated when the ECG monitor attached to the patient's skin by electrodes shows pacer spikes and the development of wide QRS complexes. Do not advance the catheter more than 10 cm past the point where the sensing indicator began to illuminate**. Withdraw the catheter and rotate it 90° if ventricular capture is not successful within 10 cm. Readvance the catheter up to 10 cm. The pacemaker generator output may be set at 20 mA and the pacing catheter readvanced. Continue to repeat the process until ventricular capture is successful. Slowly decrease the ventricular rate to 70 beats per minute once capture occurs. Decrease the pacemaker generator output to 2 mA above the threshold point.[6]

This blind procedure can be modified to be used with US. Visualize the pacing catheter after it enters the right ventricle. Use US to help guide the tip of the pacing catheter into the apex of the right ventricle or to confirm proper positioning of the pacing catheter (**Figure 43-10**).

ASSESSMENT OF SUCCESSFUL PACING

The Emergency Physician must assess the patient for electrical capture and mechanical capture when considering if the procedure was successful.[2] Successful capture is characterized by a wide QRS complex, since it is ventricular in origin, and a broad T wave. It is easy to mistake the wide, slurred afterpotential following an external pacing spike for electrical capture. **Electrical capture is best judged by the presence of a consistent ST segment and T wave after each generated pacer spike (Figure 43-7*C*).** Paced beats below the patient's intrinsic cardiac rate may not produce an associated QRS complex (**Figure 43-7*B***) and electrical capture is not achieved. The pacing rate must be increased to achieve capture in this situation.

Mechanical capture must be ensured once electrical capture is achieved by a palpable pulse rate or arterial catheter blood pressure monitoring. The patient will have a pulse rate that is exactly equal to that of the paced rhythm on the cardiac monitor if mechanical capture is achieved. Assess the pulse using palpation of the carotid or femoral artery to avoid confusion with skeletal muscle contractions generated by the pacing current. **Mechanical capture has not been achieved if the palpated pulse rate is less than that of the paced rate.** Increase the threshold current. **Mechanical capture is achieved when the invasive arterial blood pressure line demonstrates a pulse rate that is exactly equal to that of the paced rhythm on the cardiac monitor.**

US can confirm electrical and mechanical capture.[20,21] Use US to determine the ventricular contraction rate. The patient will have a heart rate that is exactly equal to that of the set paced rate if mechanical capture is achieved. Refer to Chapter 37 for the complete details of cardiac US.

AFTERCARE

Secure the pacing catheter by suturing it to the chest wall. Infiltrate subcutaneously with 2 mL of local anesthetic solution 1 cm from where the catheter exits the central venous sheath. Secure the catheter to the skin using 3–0 nylon. Apply antibacterial ointment to the site where the pacing catheter exits the central venous sheath. Apply an adhesive dressing (e.g., Tegaderm) over the sheath and catheter. Obtain an ECG. It will show the characteristic left bundle branch block pattern. Obtain a postprocedural chest radiograph to assess the catheter position and to rule out an iatrogenic pneumothorax or hemothorax. Admit the patient to an Intensive Care Unit. Consult a Cardiologist for possible permanent pacemaker placement.

The most important assessment in the aftercare period is to ensure continuous electrical and mechanical capture. This should be accomplished by frequent checks of the cardiac monitor, palpation of a pulse, and measurement of a blood pressure. This is more easily established using an arterial line (Chapter 72).

COMPLICATIONS

Perforation of the ventricular septum, the atria, or the free wall of the ventricle may occur during catheter placement (**Figure 43-11**).[22,23] This is more commonly seen with the rigid catheters and in elderly patients with kyphoscoliosis in whom right heart catheterization can be more difficult.[24] Septal perforation should be suspected if the pattern on the ECG changes from a left to a right bundle branch block.[6,25] Septal perforation should be suspected if there is an increase in the pacing threshold. Ventricular perforation can present as a failure to capture or as cardiac tamponade. A friction rub may be audible on cardiac auscultation if a perforation is present. Perforation of the inferior wall could stimulate and pace the diaphragm.[6,25] The treatment for these complications is to withdraw and reposition the pacing catheter. **The patient must then be evaluated and observed for the possibility of cardiac tamponade.**

Multiple attempts to place the tip of the pacing catheter in the apex of the right ventricle can result in complications. Movement of the catheter forward and backward can form a loop within the

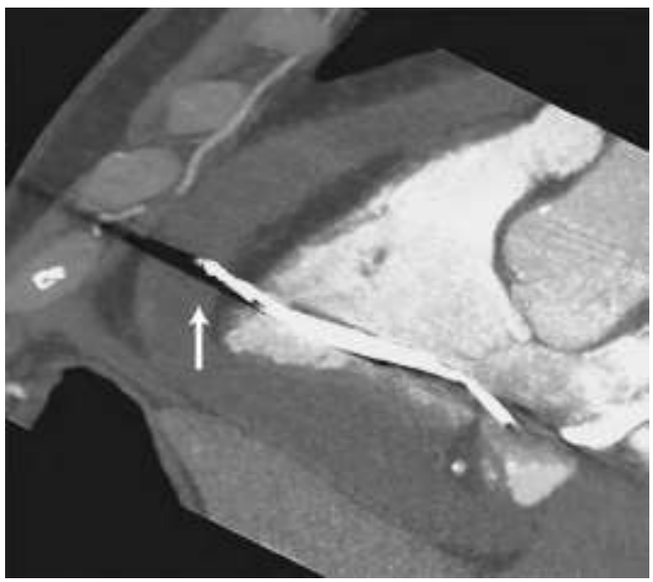
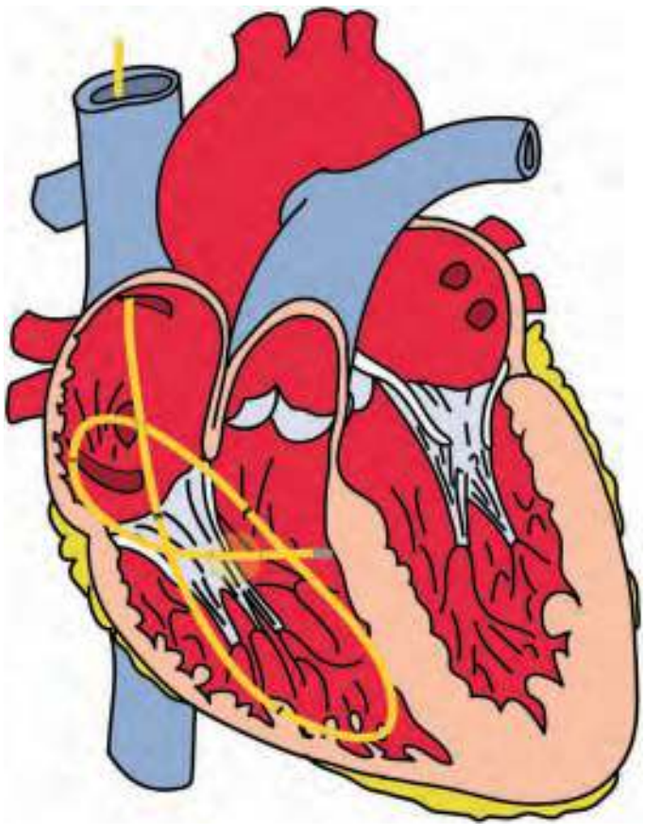


FIGURE 43-11. A pacing lead perforated the right ventricle and caused a pericardial effusion. (Used with permission from reference 23.)



A

B

FIGURE 43-12. The pacing catheter can form a loop within the right ventricle. ***A.*** Schematic. ***B.*** Radiograph. (Used with permission Charles Bruen, MD, and www.ressuscreview.com.)

cardiac chambers (**Figure 43-12**). The catheter tip can advance through the loop and result in a knot being formed in the pacing catheter. This requires an Interventional Cardiologist to unknot and remove the catheter in the cardiac catheterization laboratory under fluoroscopic guidance.

Advancement of the pacing catheter through a patent foramen ovale will result in left ventricular pacing.[26,27] This is recognized as a right bundle branch pattern on the ECG. US can be used to visualize the pacing catheter in the left ventricle or the lack of it in the right ventricle. Radiographic or angiography studies will be needed to determine if the tip of the catheter passed through the foramen or perforated the interatrial or interventricular septum. Blood clots can form anywhere along the transvenous catheter. These clots can break loose and flow through a patent foramen ovale to cause a transient ischemic attack or stroke.

The transvenous catheter can be inserted too far or past the heart. The catheter can be in the inferior vena cava or loop in the inferior vena cava (**Figure 43-8**).[17] The heart waveform may be lost once the tip of the catheter enters the inferior vena cava.

Cardiac arrhythmias may occur during insertion of the pacing catheter. Ventricular arrhythmias can occur during the procedure and even after the procedure is completed. Immediately withdraw the catheter a few centimeters and observe the rhythm. If it resolves, readvance the catheter. A defibrillator and cardiac resuscitation drugs must be available to facilitate immediate treatment of any rhythm disturbance.[6,25]

Infection can be a delayed complication. **Always use strict aseptic techniques when inserting the central venous introducer sheath and the transvenous pacing catheter.** There is currently no clinical evidence to suggest that the use of prophylactic antibiotics decreases infections or infectious complications. The use of prophylactic antibiotics cannot be currently recommended. The most common organisms are skin flora. The types of infection can range from cellulitis at the puncture site to myocarditis and florid sepsis.[6,12,25] Infection requires parenteral antibiotic therapy, the catheter to be removed, and the catheter tip to be cultured. If the pacing catheter is absolutely required, select a new puncture site and insert a new pacing catheter.

The pacing catheter balloon can be a source of complications.[28,29] Air embolism has been reported. This can be prevented by assessing the balloon for leaks prior to inserting the catheter. Do not overinflate the balloon. An air embolism or a piece of ruptured balloon may obstruct part of the pulmonary circulation. Do not inflate the balloon after it is inserted more than 10 to 15 cm into the ventricle. The vessel may rupture if the balloon is inflated when the catheter tip is in a branch of the pulmonary artery. Forward and backward movement of the pacing catheter with the balloon inflated may result in rupture of the chordae tendineae with subsequent tricuspid regurgitation.

The pacing circuit (i.e., catheter, wires, and pacemaker) can also be a source of complications. The catheter may become dislodged or fractured. The pacemaker may fail due to battery drainage, generator failure, or electrical interference.

Anatomic variations may be present and not known to the patient or the Emergency Physician.[30] These include persistent left-sided superior vena cava, congenital lack of a vein, or congenital duplication of a vein.[30] Anatomic variations can increase the technical difficulty of the procedure. It might be necessary to use a femoral approach if the internal jugular or subclavian vein approaches are unsuccessful, regardless of the reason.[30]

Complications related to obtaining central venous access are discussed in Chapter 63. They include air embolism, infection, sepsis, cellulitis, pneumothorax, improper placement, arterial puncture, venous thrombosis, venous thrombophlebitis, and guidewire complications. Complications may be decreased by using the supraclavicular approach rather than the subclavian or internal jugular approaches.[16]

EVOLVING TECHNOLOGY

Temporary dual-chamber pacemakers have been made commercially available. An absolute application for this technology has not been validated in an emergency. As our understanding of cardiac disease evolves, emergent applications may be revealed. Dual-chamber

pacemakers allow for AV synchronization. They have demonstrated benefit in pathologies (e.g., carotid sinus syndrome, high-degree AV block, sinus bradycardia, and sinus node dysfunction) and in symptomatic patients (e.g., heart failure, seizure, or syncope). This might provide benefit when AV synchronization is critical or in the rare instance when cardiac outflow is dependent on atrial kick.

The technique for placing a dual-chamber pacemaker is similar to placing a single-lead transvenous pacemaker. The second lead is in the atrium instead of the ventricle. It is important to be familiar with the North American and British pacing societies (NBG) coding system (Chapter 45).[31] The NBG is a five-letter system for identifying the modes of pacing. The first letter designates which chamber is being paced. The second letter designates which chamber is being sensed. The third letter designates the mode responding to sensing. The fourth and fifth letters are designated for internal pacing modes.

SUMMARY

Placement of a transvenous cardiac pacing catheter can be a lifesaving procedure. It is a safe method to electrically stimulate the heart. It is indicated when an unstable rhythm of the heart is refractory to medications and transcutaneous pacing. Proper placement of the pacing catheter is cardinal to its functioning. Recognition of the ECG changes that occur in the different anatomic areas helps to guide its placement. Be aware of the potential complications and their management. This procedure should be mastered by all Emergency Physicians caring for critically ill and/or injured patients.

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44 Transesophageal Cardiac Pacing

John Bass and Chad Gorbatkin

INTRODUCTION

Cardiac pacing is an essential skill for Emergency Physicians and can be accomplished through a variety of techniques. Transesophageal cardiac pacing is a time-proven and potentially lifesaving procedure. The esophagus has long been considered a noninvasive and useful window to the heart.

The ability to capture and record an electrocardiogram (ECG) transesophageally was first demonstrated by Max Cremer in 1906.[1] This was based on the close anatomic proximity of the cardiac atria to the esophagus. The proximity allows an electrical impulse in the esophagus to be transmitted to the left atrium. Electrode technology and pacing mechanisms have advanced. The ability to identify, stabilize, and terminate atrial dysrhythmias through this minimally invasive procedure persists.[2] Transesophageal pacing is well-tolerated by patients and effective for temporarily treating patients with atrial rhythm disturbances. This technique provides an additional tool for Emergency Physicians.

ANATOMY AND PATHOPHYSIOLOGY

The heart is the only muscle that produces its own electrical impulse. The electrical impulse originates from the sinoatrial node of the right atrium and is conducted through the heart's intrinsic electrical conducting system to the atria and ventricles to produce coordinated and sequential mechanical contractions (**Figure 39-2**).[3] The rate of impulse generation is governed by the sympathetic and parasympathetic nervous systems. The conversion of an electrical impulse to a mechanical action is a delicate balance between chemical signaling, electrolyte shifts, an intact cardiac conducting system, and healthy myocardial tissue. Abnormalities in conduction and resultant arrhythmias may be related to abnormalities in any of these factors. Aberrant conduction system development, impaired myocardium due to decreased blood supply, or impaired myocardium due to infarct often underlies arrhythmias.

The heart is in the anterior thoracic cavity. It is surrounded by the sternum and chest wall anteriorly, the lungs laterally, and in proximity to the esophagus posteriorly. The left atrium is typically the closest point of contact to the esophagus (**Figure 44-1**).[4] Cadaveric models and imaging analysis show an atrioesophageal distance of typically less than 1 cm, and often only a few millimeters.[5] This is impacted by individual variability and disease states that may alter cardiac muscle size.

INDICATIONS

Transesophageal cardiac pacing can be a simple and easily initiated procedure. Weigh its use against clinical circumstances. Transesophageal atrial pacing is most frequently used for management of symptomatic bradycardia, termination of atrial fibrillation, and termination of supraventricular tachycardia.[1,6-11] Sinus node dysfunction or delays in electrical impulse conduction may manifest as bradycardia. End-organ hypoperfusion can occur with decreased cardiac output and result in a variety of symptoms (e.g., altered mental status or fatigue). Use transesophageal pacing to temporarily restore a heart rate that is compatible with end-organ perfusion and in preparation for definitive treatment of the underlying condition.

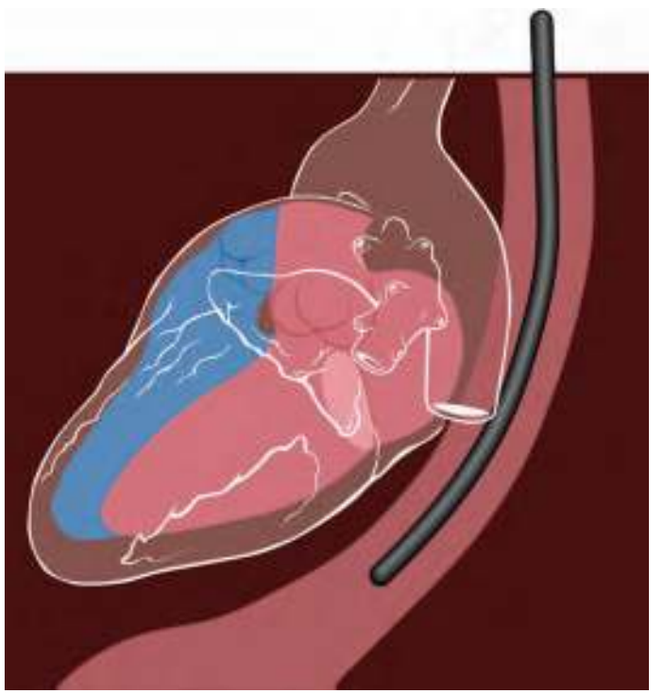


FIGURE 44-1. The relationship of the esophageal catheter to the heart. (Used from www.commonswikimedia.org.)

An accelerated heart rate can result in similar symptoms. Such tachyarrhythmias, especially in pediatric patients, are often supraventricular in origin. Transesophageal pacing is a well-tolerated and effective means of suppressing or abolishing inappropriate tachyarrhythmias given the possible complications associated with transvenous access and pacing in pediatric patients (**Figure 44-2**).[12,13]

Several studies favor the use of transesophageal pacing. A small prospective study showed no difference in conversion rates or procedure duration when comparing scheduled transcutaneous to transesophageal electrical cardioversion for adults with atrial fibrillation.[14] Transesophageal electrical cardioversion in adults has a 97% restoration rate of sinus rhythm.[14] Studies in pediatric patients have shown an 81% successful conversion rate of primarily atrial reentrant tachycardia without significant complications.[15] Indications for transesophageal pacing include decompensated sinus bradycardia, contraindications to or ineffective pharmacologic resuscitation, transcutaneous pacing failing to capture, and unavailable or delayed transvenous pacing.[16,17]

Transesophageal pacing is used more frequently in Cardiology suites than the Emergency Department. It is used for diagnosis of complex supraventricular arrhythmias with amplified P waves, echocardiography, and nonexercise stress testing. It is used for overdrive pacing of atrial flutter and other supraventricular tachycardias. Cardiologists induce atrial arrhythmias to determine effectiveness of therapy. They temporarily pace bradycardia, hypotension, and sick sinus syndrome. Transesophageal pacing is used temporarily in pediatrics. It is also used to determine abnormal left atrial activity during ablations.

CONTRAINDICATIONS

No absolute contraindication to transesophageal atrial pacing exists in the unstable or symptomatic patient with an intact esophagus. **It is not a first-line means of resuscitation in the hemodynamically unstable or decompensating patient. This procedure is not intended as a life-sustaining practice and should not be used for patients with total heart block.** These individuals require timely and dependable ventricular pacing, which cannot be guaranteed with this technique. Transesophageal ventricular pacing is limited by the distance between the esophagus and heart. It requires much greater energy to achieve capture. These factors increase the risk of local tissue injury and inconsistent cardiac pacing during an emergent or resuscitative event.

Assess the patient for historical or physical features that could make the passage of a transesophageal probe difficult or dangerous.[18] The ability to follow directions is important to safe completion of this procedure. Known anatomic variants (e.g., diverticular disease or esophageal strictures), surgical interventions, or trauma may limit the success of transesophageal pacing and necessitate an alternative means of patient stabilization or diagnosis.

EQUIPMENT

- Lidocaine (e.g., aerosolized, gel, or paste)
- 5 or 10 French bipolar or quadripolar pacing catheter or pill electrode (**Figure 44-3**)
- Cardiac monitor or ECG machine
- ECG lead wire with alligator clip
- Pulse generator or cardiac stimulator (**Figure 44-4**)
- Ultrasound (US) machine
- US gel

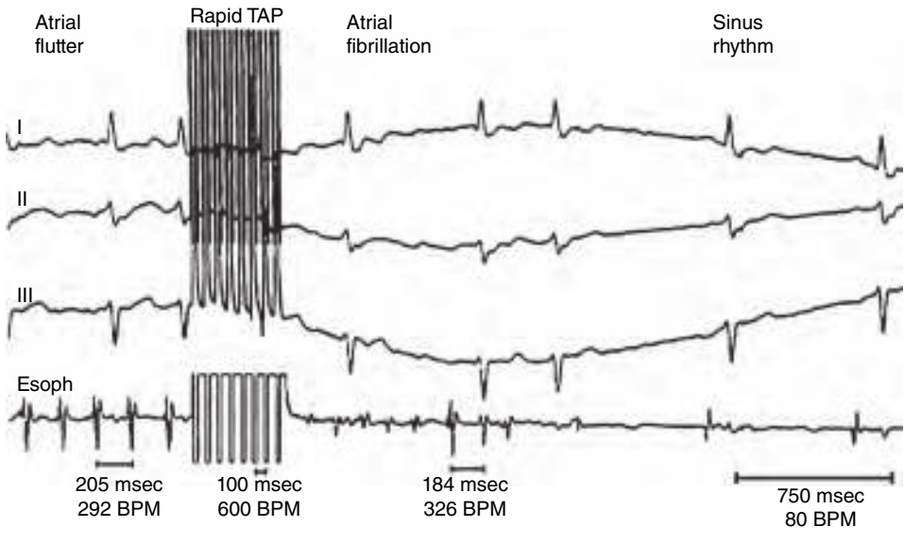
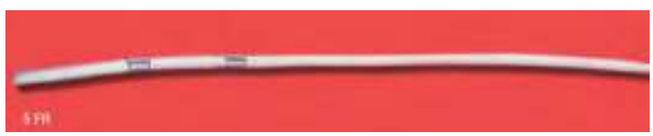


FIGURE 44-2. Cardioversion of atrial flutter to normal sinus rhythm. Note the transient atrial fibrillation. (Courtesy of CardioCommand Inc., Tampa, FL.)

- US transducer, 6 to 12 MHz, high-frequency
- US transducer, curvilinear or microconvex in obese patients

Transesophageal atrial pacing is often accomplished through one of two pacing devices. First is a pill electrode secured to a flexible wire and swallowed by the patient.[19] This is rarely available in the Emergency Department. Second is a flexible catheter that may be inserted via the nares or mouth into the esophagus (**Figure 44-3**).[19] Use bipolar or quadripolar devices as they are readily available and successful at safely obtaining atrial capture. A pulse generator or commercially available cardiac stimulator as typically used in electrophysiology is used for atrial pacing (**Figure 44-4**).[18,20] Certain unique aspects of transesophageal pacing make the use of external



A

B

FIGURE 44-3. The tips of the esophageal pacing electrode. ***A.*** Tapcath 205 (CardioCommand Inc., Tampa, FL). ***B.*** Ecosoft S. (Courtesy of FIAB, Firenze, Italy.)

A

B

FIGURE 44-4. Esophageal cardiac pacemakers. ***A.*** The Model 7A Stimulator and the Model 3 Preamplifier. (Courtesy of CardioCommand Inc., Tampa, FL.) ***B.*** The Asynchronous Esophageal Cardiac Pacer 2007. (Courtesy of FIAB, Firenze, Italy.)

pacing generators ineffective and necessitate a dedicated transesophageal pacer.

Many hospitals use the CardioCommand devices (CardioCommand Inc., Tampa, FL) (**Figure 44-4*A***). The Model 7A stimulator was designed for transesophageal pacing. It has dedicated controls and sets up quickly. It can regulate capture frequency, heart rate, pacing intensity, and pulse width. The Model 3 Amplifier links the esophageal catheter and the ECG monitor. It eliminates artifacts (e.g., due to breathing, esophageal peristalsis, and swallowing). It allows simultaneous recording of esophageal and surface ECGs. It also transmits the paging impulses from the stimulator to the esophageal catheter.

Equipment for successful transesophageal atrial pacing is available in most hospitals. Commercially available prepackaged kits are useful in facilities completing frequent transesophageal pacing or serving a population that might benefit from emergent access to this procedure.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Discuss the patient expectations.[18] Obtain an informed consent for the procedure, and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Screen patients for prior medical and surgical history that might complicate transesophageal pacing. Apply external hemodynamic monitoring (i.e., blood pressure cuff, ECG monitoring, and pulse oximetry). Provide supplemental oxygen. The use of pharmacologic adjuncts depends on route of administration and patient tolerance. Consider topical anesthesia with aerosolized lidocaine spray to desensitize the nares or oropharynx. Consider the use of anxiolysis with weight-based intravenous midazolam to promote patient tolerance of the procedure on a case-by-case basis.[15] Consider procedural sedation, especially in children (Chapter 159). Consider the administration of an antiemetic. **Have airway management, a cardioverter-defibrillator unit, emergent resuscitation, and suction available and nearby**. Patient positioning is dependent on patient preference and provider comfort when the patient is awake. Patients are often seated upright to facilitate esophageal catheter passage. Frequently assess the patient for cardiac instability, discomfort, or pulmonary distress.[18]

TECHNIQUE

Place the transesophageal atrial electrode in accordance with patient participation and comfort. Use a swallowed tethered pill electrode for older patients or those capable of cooperating. An alternative method and the method most used in the Emergency Department is an electrode catheter inserted through the nares. Depth of insertion (DOI) guidelines are available on commercial transesophageal pacing kits based on patient height. Atrial capture is often successful at an oral DOI calculated as follows: DOI (cm) = (patient height [cm] ÷ 5); or DOI (cm) = (height [inches] ÷ 2 ± 3 cm for variability).[20,21] Add an additional 3 to 4 cm to the calculated DOI for nasal insertion.[20,21] Atrial capture is achieved within 36 to 37 cm as the DOI.[22,23] An example is a 6 foot tall individual measuring 183 cm tall. The insertion depth is 36.6 cm ± 3 cm for oropharyngeal insertion or 40 cm ± 3 cm for nasal placement.

Insert the esophageal pacing catheter like a nasogastric tube (Chapter 75). Attach the alligator clip to the negative terminal of the esophageal pacing catheter and the other side to lead V_1 of the ECG monitor. Turn on the ECG monitor set to lead V_1. Insert the pacing catheter to an initial DOI of [(height {cm} ÷ 5) + 5] orally or

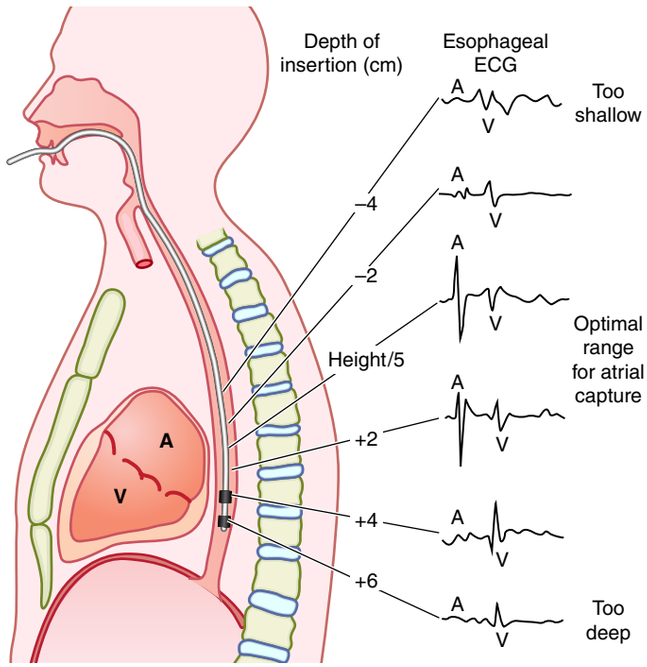


FIGURE 44-5. The optimal positioning of the esophageal electrode and its impact on atrial depolarization amplitude. (Courtesy of CardioCommand Inc., Tampa, FL.)

[(height {cm} ÷ 5) + 9] nasally. Withdraw the pacing catheter while watching the ECG until the point of maximal esophagoatrial ECG deflection, which closely approximates the point of minimum atrial pacing threshold (**Figure 44-5**).[24]

Identifying the minimal atrial threshold will help improve patient comfort and limit the potentially harmful use of excessive amperage during this procedure.[1,21] Lowest stimulatory thresholds are often captured with atrial pulse widths of approximately 10 milliseconds.[22,25] Adjust the electrical current to produce consistent and reliable atrial pacing. This often occurs between 5 and 25 milliamperes.[26] A dedicated pacing device is required to safely complete this procedure rather than those typically used for endocardial or transthoracic pacing (**Figure 44-4**).[27] This is due to the unique strength duration curve associated with esophageal pacing.

ALTERNATIVE TECHNIQUES

The technique of transesophageal pacer placement and initiation has limited variability. Confirmation of placement may be aided by US. The utility of US in confirming the placement of nasogastric tubes is well known and could theoretically be applied to transesophageal pacing.[28] Evaluate the anterior neck with US. Assess the location, orientation, and contour of the esophagus for any variation.

Placement of a transesophageal pacing catheter may be confirmed in real time during the advancement of the device or afterward. Prepare the US transducer. Have an assistant apply US gel over the footprint of the transducer. Place the transducer on the anterior neck with the marker facing the patient's right. A hyperechoic structure in the esophagus corresponds with the catheter.[29] Advance the device to the predetermined depth. The catheter can be removed and advancement reattempted depending on clinical circumstances and patient tolerance if concerned about incorrect placement.

Little is known about the use of US for confirming placement of pacing pill electrodes. The likelihood of incorrect positioning is presumed to be low given the necessity of patient participation in swallowing the device unless underlying structural variation or

dysphagia is present. The tethering wire or electrode may be seen in the esophagus depending on echogenicity or depth. Further investigation on this topic may be warranted.

PEDIATRIC CONSIDERATIONS

Transesophageal pacing is most frequently cited in pediatric cases of atrial or supraventricular dysrhythmias. No specific changes or alterations to this technique are required for children.[13,30] Use appropriately sized catheters. Use a 6 French pacing catheter for neonates or children up to 15 kg and a 7 French pacing catheter for larger children.[31] Adjust medication doses according to patient weight and medical comorbidities.

ASSESSMENT

Assess the patient for electrical and mechanical capture after placement of the transesophageal pacing catheter. Successful electrical capture is demonstrated by a prominent P-wave deflection on the ECG with restoration of a regular rhythm at the paced rate. Address failure to capture by ensuring optimal catheter placement and adjusting pacer depth.[3]

Confirm mechanical capture through physical examination and hemodynamic monitoring. A palpable pulse matching the paced rate will correspond to the cardiac monitor. Intraarterial blood pressure monitoring is a suitable alternative to pulse palpation. Absence of a pulse at the paced rate represents a failure of mechanical capture. Increase the pacing current until capture is confirmed.[3,21]

AFTERCARE

Secure the pacing catheter following successful transesophageal pacing and confirmation of the point of maximal ECG deflection. Tape the catheter to the patient's nose or cheek, similar to a nasogastric or orogastric tube (Chapter 75). Apply additional catheter securing devices or restrain the patient if they are considered a high risk for accidental catheter removal.[18]

Regularly assess the patient for electrical and mechanical capture. Ensure continuous ECG monitoring, frequent pulse palpation, and frequent blood pressure checks for patient safety and to rapidly identify any changes in transesophageal pacing. **This technique is intended for short-term or temporizing use, and invasive hemodynamic monitoring is not warranted unless indicated for another reason.**

Make patient comfort a priority throughout this procedure. Provide deescalation and verbal reassurance for the management of agitation or anxiety. Redosing of anxiolysis or pain control medications may be needed throughout the procedure to decrease discomfort.

Remove the pacing catheter or pill electrode in patients no longer requiring transesophageal pacing. Assess the patient for possible trauma sustained during placement or maintenance of the device. Complete a challenge of oral intake when appropriate.[18] Local irritation and discomfort are possible. Oral intolerance or progressive pain following intake should prompt further imaging or investigation for esophageal injury.[32]

COMPLICATIONS

Complications related to transesophageal atrial pacing are rare. The complications associated with nasogastric insertion are discussed in detail in Chapter 75. This minimally invasive procedure is well tolerated by patients. There has been no reported mortality or long-term morbidity throughout its worldwide use as a diagnostic and therapeutic tool. Nonsustained ventricular fibrillation and ventricular tachycardia have been rarely reported in infants with Wolf-Parkinson-White syndrome.[1] The means to emergently stabilize and cardiovert/defibrillate patients should be readily available.

The transesophageal pacer is used for a limited time. Esophageal irritation is a possible complication of prolonged transesophageal pacing, as evidenced by a mild esophageal ulceration discovered during autopsy of an adult undergoing 60 hours of continuous pacing.[1] There are no documented cases of esophageal perforation or long-term dysfunction associated with this procedure. Accidental placement in the trachea could result in patient injury or respiratory distress.

Consider patient comfort, participatory ability, and aspiration risks. These are minimized by adequate anesthesia and careful explanation of the procedure. Patient discomfort may limit the initiation or completion of transesophageal pacing. **Airway protection is vital throughout this procedure.** Assess the patient for aspiration risk. Include the time of last oral intake, known medical comorbidities, and prior adverse responses to procedures. **Tools necessary for emergent airway management should be available during transesophageal pacing.**

SUMMARY

Transesophageal pacing has been clinically implemented since the late 1950s for the diagnosis and management of supraventricular dysrhythmias and symptomatic bradycardia. Extremely limited literature exists to support its regular use. This procedure may be initiated within minutes following very little patient preparation. Transesophageal pacing has demonstrated great success for temporary stabilization and conversion of certain abnormal cardiac rhythms, especially in pediatric populations. Transesophageal pacing may be initiated if transcutaneous or transvenous pacing is not readily available or clinical circumstances do not support these techniques. This technique is not intended for use during asystole and has not been validated for the management of drug-induced bradycardia.

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45 Pacemaker Assessment

Elizabeth Kwan

INTRODUCTION

Cardiac rhythm management devices (CRMDs) are becoming increasingly common as our population ages and indications for their use broaden.[1] These devices are becoming increasingly complex. Single-chamber devices with only a pacing function have given way to dual-chamber devices with the ability to sense and programmable features (e.g., automatic mode switching or rate responsiveness). Implantable cardioverter-defibrillators (ICD) have been developed as primary and secondary prevention for sudden cardiac death and have a backup pacemaker function. **Biventricular pacemakers are known as cardiac resynchronization therapy (CRT). They were developed to coordinate the action of the right and left ventricles for patients with reduced systolic function and delayed conduction defined by a wide QRS.** Most CRT devices have an associated defibrillator (CRT-D) since indications for CRT are associated with a high risk for ventricular tachycardia (VT) and ventricular fibrillation (VF).

Have a very low threshold for formal device interrogation and Cardiologist consultation when a malfunction is suspected given the complexity of the current generation of pacemakers. **The Emergency Physician must be careful not to prematurely attribute a patient's presentation to a pacemaker malfunction.** Consider life-threatening and other conditions while awaiting pacemaker interrogation. Any critical illness causing severe metabolic derangements (e.g., acidosis, hypoxemia, or hyperkalemia) can interfere with device function by altering the tissue interface with the myocardium.

Pacemakers have become more complex. They are more reliable with better hermetic sealing, better batteries and circuitry, the use of bipolar instead of unipolar leads, and special programming to prevent rhythm-related complications. **Pseudo-malfunctions occur when a device is functioning as programmed but there are unexpected or unusual electrocardiogram (ECG) findings. The pseudo-malfunction is more common than a true malfunction.**

It may not be obvious when a patient presents to the Emergency Department if their symptoms are due to a pacemaker malfunction. The patient may not even provide a history of having pacemaker. The goal of this chapter is to prepare the Emergency Physician to manage patients presenting with symptoms that may be related to these devices and to understand how the presence of a pacemaker may affect patient care. The common term *pacemaker spike* is used instead of *pacemaker artifact* throughout this chapter.

INDICATIONS FOR PACEMAKER PLACEMENT

Consensus guidelines for pacemaker placement have been established by the American College of Cardiology (ACC), the American Heart Association (AHA), and the Heart Rhythm Society (HRS).[2] Pacemakers are placed most often for symptomatic bradycardia from sinus node dysfunction or atrioventricular block (**Figures 45-1 and 45-2**). CRT pacemakers are placed for advanced heart failure with conduction delays as defined by a wide QRS interval. CRT improves cardiac function, quality of life, and survival.[3] Placement of ICDs is for primary and secondary prevention of VT or VF. ICD patients often overlap with patients needing CRT, with most CRT devices having a defibrillator, known as CRT-D.[4]

PACEMAKER PARTS AND FUNCTION

The pacemaker has the functions of creating electrical impulses to pace the myocardium, sensing the heart's intrinsic electrical activity, and responding by pacing or inhibiting pacing. It stores diagnostic information. **The pacemaker can inhibit itself but cannot inhibit the heart.** It may incidentally suppress intrinsic activity because paced depolarizations will make the myocardium refractory to native impulses. All references to pacemaker inhibition refer to inhibition of its own pacing activity. **A function of newer pacemakers is rate responsiveness. This allows for increased physical activity by increasing the paced rate in response to increased metabolic demands.**

Pacemakers consist of a pulse generator connected to a lead or leads. The generator has a battery and circuitry that interprets

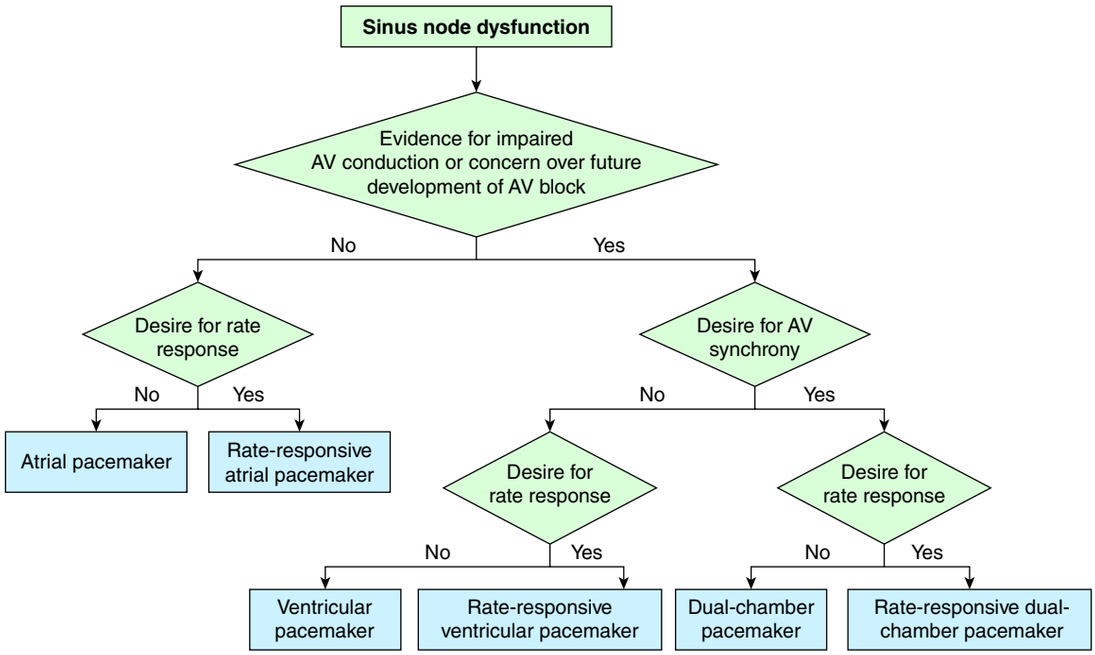

FIGURE 45-1. Selection of pacemaker systems for patients with sinus node dysfunction. Decisions are illustrated by diamonds. Shaded boxes indicate type of pacemaker. AV, atrioventricular. (Used with permission from reference 2.)

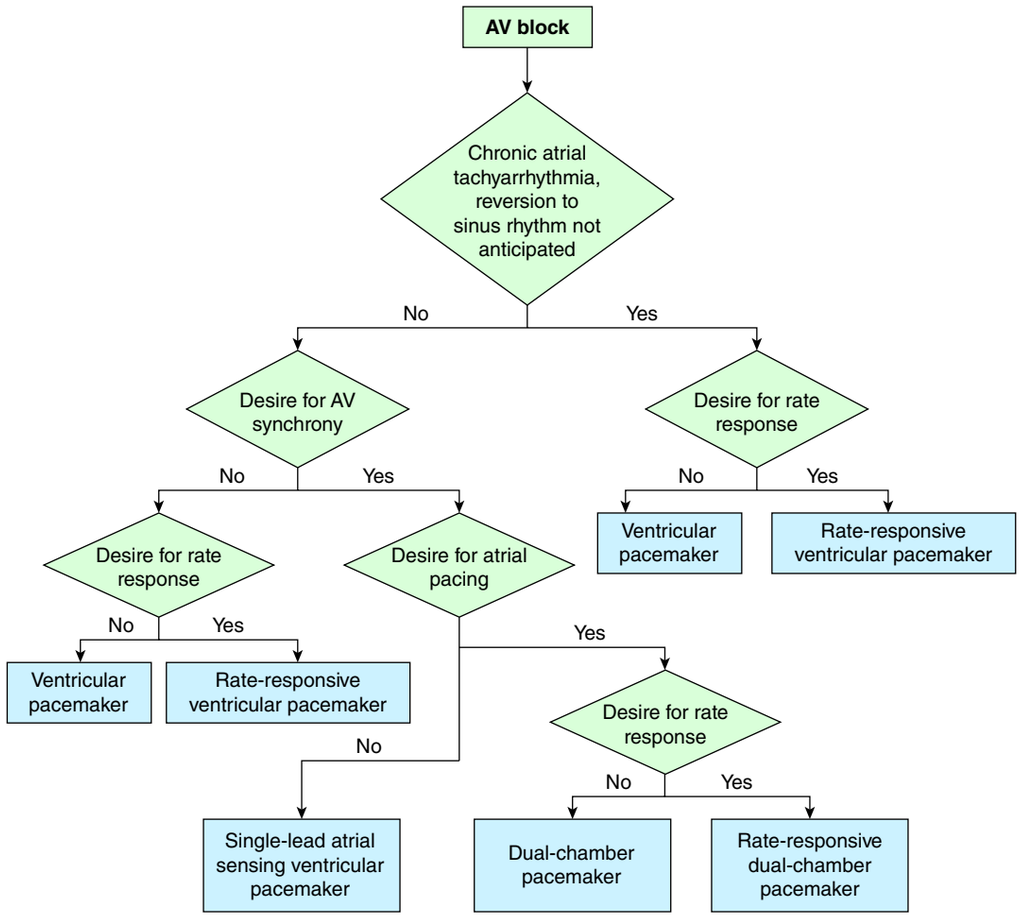

FIGURE 45-2. Selection of pacemaker systems for patients with atrioventricular (AV) block. Decisions are illustrated by diamonds. Shaded boxes indicate type of pacemaker. (Used with permission from reference 1.)

sensor information and creates pacing stimuli. The generator is usually placed in a subcutaneous pocket in the upper anterior chest. The leads are wires connected to the pacing generator and deliver the electrical impulses from the generator, detect the intrinsic electrical activity of the native heart, and deliver those signals back to the generator.

Unipolar leads leave a bigger spike on the ECG and are less common than bipolar leads. The negative electrode of unipolar leads is at the end of the wire, and the metal generator casing acts as the positive electrode. The current to pace or to sense must travel through all the tissue between them to complete the circuit.

Bipolar leads contain two layers of insulated wires. The negative electrode is at the tip of the lead. The positive electrode forms a ring just proximal to the tip of the lead. **The current flows in a much smaller area in the bipolar lead because the electrodes are close together.** This may result in bipolar pacing spikes not visible on the ECG. Bipolar leads are much less likely to stimulate other body tissues or to oversense and mistake electrical activity outside the heart as cardiac impulses.

Leads are usually placed into the subclavian vein and fluoroscopically guided into position in the heart. They may be fixed passively into the trabeculae of the myocardial wall or fixed using screws. Leads are right sided in dual-chamber pacemakers and endocardial (i.e., placed on the inner surface of the heart). The atrial lead is in the right atrial appendage. The right ventricular (RV) lead can be at the apex, RV outflow tract, or the interventricular septum. CRT is biventricular pacing with a third lead to the left ventricle (LV) to allow the simultaneous pacing of the RV and LV. The LV lead passes through the coronary sinus, passes through the myocardium via veins to the epicardium (i.e., outer surface of the heart), and passes along the posterior or lateral free wall of the LV.

Pacemakers work by forming a closed circuit that includes the pacemaker and the myocardial tissue to carry out its pacing and sensing functions. The battery stores the voltage, which is the potential to move electrons. Leads deliver electrons as current. The current in this closed circuit moves from the battery, through the leads, from the negative electrode across the myocardial tissue, and then back to the positive electrode. **The circuit must be a closed loop for any movement of current.** Additional circuitry within the generator carries out programming.

PACEMAKER MODES

The North American Society for Pacing and Electrophysiology and the British Pacing and Electrophysiology Group use a universal five-letter pacemaker code (**Table 45-1**). The combination of these letters describes the pacemaker mode or function. The code does not describe the characteristics, specific functions, or unique functions that are specific to each pacemaker or its manufacturer. The character position is labeled in Roman numerals I through V. The first three letters are most commonly used.

The first letter designates the chamber(s) in which pacing occurs. It can be designated as atrial (A), ventricular (V), both atrial and ventricular (D or dual), or none (O). The second letter designates the chamber(s) used by the pacemaker to sense intrinsic electrical cardiac activity. It can be designated as atrial (A), ventricular (V), both atrial and ventricular (D or dual), or none (O). The third letter designates how the pacemaker responds to sensed intrinsic electrical activity. A sensed event may inhibit the pacemaker (I), trigger the pacemaker (T), both inhibit and trigger the pacemaker (D), or cause no response (O) from the pacemaker generator.

How would an emergent transvenous pacemaker with a single lead in the RV be described? When first being placed in asynchronous mode, it would be VOO: paces the ventricle (V– –), with no sensing (VO–) and no response to sensing (VOO). It changes to VVI once set for sensing. It paces in the ventricle (V– –), is sensed in the ventricle (VV–), and is inhibited when intrinsic electrical activity is sensed (VVI).

DDD pacemakers represented 82% of new implantations in 2009, compared to single-chamber VVI or AAI pacemakers, which accounted for 14% and 1%, respectively.[5] Patients with sinus dysfunction but intact atrioventricular (AV) conduction may only need AAI pacing. They often get a dual-chamber pacemaker because there is the risk of developing an AV conduction problem and replacing the device and leads is not without risk. Biventricular CRT devices represented 4% of new implantations in 2009, but represented 10% of cardiac rhythm management devices placed from 2007 to 2013 in another study.[4] CRT devices may continue to grow as a proportion of CRMD as more studies support expanding indications for CRT and deleterious effects on LV function from long-term pacing of a RV lead, particularly in patients with preexisting LV dysfunction.

The fourth and fifth letters are rarely used, as these functions are not often used. The fourth letter refers to whether the pacemaker is rate responsive (R) or if this feature is absent or turned off (O). Rate-responsive pacemakers estimate metabolic demands by using an accelerometer to detect motion and using changes in chest wall impedance as a measure of minute ventilation. It interprets an increase in motion or minute ventilation as increased activity and increases the pacemaker rate to accommodate exercise. This is an important feature for active patients. Rate responsiveness is turned off when a magnet is placed over the device and the pacemaker is made asynchronous.[6,7] The fifth letter designates antitachyarrhythmic function.

PACEMAKER TIMING INTERVALS

Pacemakers have multiple programmable intervals to determine when to deliver a pacing output. It is important to understand basic pacemaker timing intervals to understand pacemaker function and safety features. The pacemaker timing cycle starts DDD pacemakers with either an intrinsic or paced atrial depolarization (**Figure 45-3**). This starts the atrial ventricular interval (AVI), the time from atrial

TABLE 45-1 The Generic and Standard Pacemaker Codes

Position	I	II	III	IV	V
Interpretation	Chamber(s) paced	Chamber(s) sensed	Response to sensing	Programmability rate modulation	Antitachyarrhythmia function(s)
Variable	0	0	0	0	0
	A	A	T	P^1	P^2
	V	V	I	M	S
	D (A + V)	D (A + V)	D (T + I)	C	D (P + S)
				R	

A, atria; C, communicating; D, dual; I, inhibited; M, multiprogrammable; 0, none; P^1, simple programmable; P^2, pacing; R, rate modulation; S, shock; T, triggered; V, ventricle.

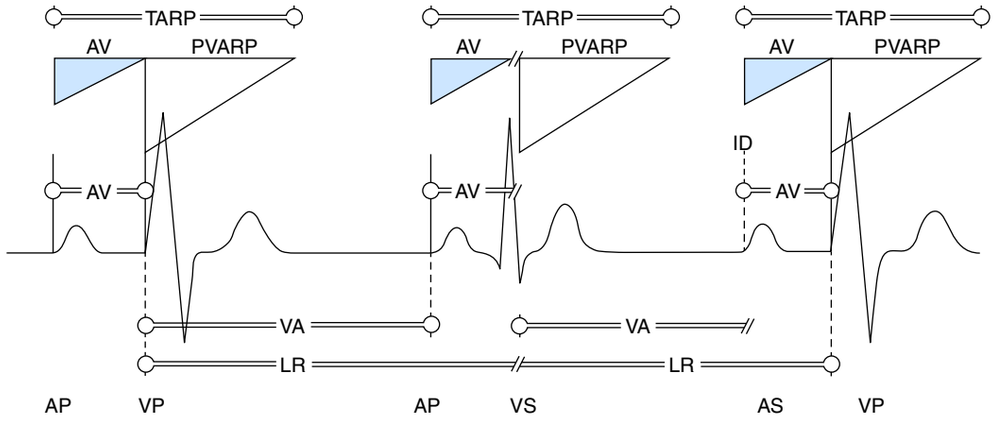

FIGURE 45-3. Dual-chamber pacemaker timing cycles. The timing cycle in DDD consists of a lower rate (LR) limit, an atrioventricular (AV) interval, a ventricular refractory period, a postventricular atrial refractory period (PVARP), and an upper rate limit. If intrinsic atrial and ventricular activity occur before the LR limit times out, both channels are inhibited and no pacing occurs. In the absence of intrinsic atrial and ventricular activity, AV sequential pacing occurs (first cycle). If no atrial activity is sensed before the ventriculoatrial (VA) interval is completed, an atrial pacing artifact is delivered, which initiates the AV interval. If intrinsic ventricular activity occurs before the termination of the AV interval, the ventricular output from the pacemaker is inhibited (i.e., atrial pacing with intrinsic conduction [second cycle]). If a P wave is sensed before the VA interval is completed, output from the atrial channel is inhibited. The AV interval is initiated, and if no ventricular activity is sensed before the AV interval terminates, a ventricular pacing artifact is delivered (i.e., P-synchronous ventricular pacing [third cycle]). AP, atrial pace; AS, atrial-sensed event; ID, intrinsic defiection; TARP, total atrial refractory period; VP, ventricular pace; VS, ventricular event. (Used with permission from reference 7.)

to ventricular depolarization, which is analogous to the PR interval. A paced QRS is triggered if there is no intrinsic QRS by the end of a programmed AV delay. The AV delay is analogous to the longest PR interval the pacemaker will tolerate. If an intrinsic QRS occurs within the programmed AV delay, there will be no ventricular pacing artifact on the surface ECG and the AVI will be shorter than the programmed AV delay. The QRS starts the ventricular-atrial interval, the time from ventricular to atrial depolarization. This is also known as the atrial escape interval (AEI), or the programmed amount of time the pacemaker will wait for an intrinsic P wave to "escape" before pacing the atrium.[8]

Immediately after ventricular depolarization the postventricular atrial refractory period (PVARP) begins. The AVI and PVARP make up the total atrial refractory period (TARP). The atrial lead is refractory during the TARP and will not deliver a pacing impulse. The TARP determines the pacemaker's upper rate limit or the minimum amount of time before the next atrial impulse can be delivered to start the next AVI and trigger a paced ventricular depolarization. The lower rate limit is defined by the programmed intervals that define the maximum time in the cycle that the pacemaker will wait for intrinsic depolarization (i.e., the AV delay plus the AEI). The pacemaker will trigger an atrial paced beat to restart the AVI if no intrinsic P wave occurs by the end of the AEI. The pacemaker will sense and record the impulse in the refractory period but will not alter the timing cycle.[8]

There is a brief blanking period in all chambers in addition to the refractory period immediately after each pacing stimulus during which the pacer will not sense. This is meant to prevent "cross-talk," when a pacing stimulus in one chamber is interpreted as a depolarization in the other. If an atrial pacing stimulus starts being interpreted as an intrinsic ventricular signal, ventricular pacing will be inappropriately inhibited indefinitely and lead to asystole.[8]

COMMON SAFETY FEATURES

Ventricular safety pacing is a protection against cross-talk and precipitating ventricular tachyarrhythmias by pacing early before the vulnerable part of the T wave. There is a cross-talk sensing window after the brief blanking period. The pacemaker delivers a ventricular pacer spike early if any ventricular activity is detected during this period. Ventricular safety pacing prevents asystole if the ventricular signal detected was cross-talk (i.e., a misinterpreted atrial pacing event). The early ventricular pacer spike prevents pacing during the vulnerable part of the T wave that could precipitate ventricular arrhythmias if the ventricular signal in the cross-talk window was a premature ventricular contraction (PVC). The myocardium would be refractory from the PVC, and there is no additional QRS. Suspect ventricular safety pacing on the ECG when a ventricular pacer spike occurs soon after an atrial pacer spike or a PVC that lands when an atrial pacer spike is expected.[9]

MINIMIZING RIGHT VENTRICULAR PACING

RV pacing contracts the myocardium starting in RV and then spreads to the septum and then the LV. It simulates a left bundle branch block (LBBB) on the ECG. This altered conduction causes adverse remodeling, altered LV perfusion, and dyssynchrony.[10,11] Pacing that maintains AV synchrony (e.g., DDD with intact sinus node function or AAI with intact AV conduction) has additional advantages. Atrial or dual-chamber pacing reduces the incidence of atrial fibrillation from 22% to 17% and decreases the risk of stroke.[12] Keeping AV synchrony results in a better ejection fraction than ventricular pacing by preserving "atrial kick." It reduces the risk of the pacemaker syndrome ranging from discomfort to overt heart failure resulting from AV asynchrony.

The degree of adverse effects of RV pacing may be related to its frequency and duration.[12] Advances in pacemaker technology allow minimizing RV pacing when possible through automatic mode switching and allows slow native rates. These measures preserve AV synchrony, minimizing the long-term harm to LV function, and minimizing battery usage.

AUTOMATIC MODE SWITCH

Automatic mode switching allows a pacemaker to function as AAI with a backup DDD function if absent AV conduction is detected. The pacemaker will periodically revert to AAI pacing to assess intrinsic AV conduction.[13] **Mode switching protects against fast atrial rates.** The pacemaker can switch from a DDD mode if an atrial tachycardia is detected to DDI, DVI, or VVI to stop the triggering of ventricular pacing by atrial impulses. The pacemaker

will go back to DDD mode when the intrinsic atrial rate falls below a programmed threshold.[12]

HYSTERESIS, REST, AND SLEEP MODES

The heart rate naturally slows during sleep to meet the lower physiologic demands. Pacemakers have modes to mimic this phenomenon and allow rates slower than the preprogrammed lower rate limit. The heart benefits from the decreased heart rate during sleep. Experimental data on patients with DDD pacemakers show that systolic and diastolic function decline without this rest.[14]

Manufacturers use different approaches. St. Jude offers a "rest mode" of 10 to 20 beats a minute less than the lower rate limit if patient activity decreases below a preset threshold for 15 to 20 minutes. This is measured with the same accelerometer as the rate response feature. Medtronic "sleep mode" decreases the paced rate during specific hours of the day. Rest and sleep modes do not rely on sensing the intrinsic rhythm and will not be turned off by magnet application.[15] Boston Scientific pacemakers have a hysteresis function, derived from the Greek word for "late." The pacemaker may have a programmed lower rate limit of 60 beats per minute when pacing and a hysteresis rate of 50 beats per minute. The pacemaker will allow the intrinsic rate to fall as low as 50 beats per minute before it starts pacing at a rate of 60 beats per minute. This feature relies on sensing the intrinsic rate. Turning off sensing with the application of a magnet turns off hysteresis.[15]

DIFFERENTIAL DIAGNOSIS OF POTENTIAL PACEMAKER MALFUNCTION BASED ON ECG FINDINGS

CATEGORIZING PACEMAKER MALFUNCTION

The conventional categories of pacemaker malfunction are based on electrophysiologic mechanisms. These categories are more useful to someone programming a pacemaker than the Emergency Physician managing a patient with limited information. **Failure to pace is noted by a lack of the pacemaker spike on the ECG and the failure to deliver a stimulus to the myocardium when there is a pause in the intrinsic cardiac electrical activity. Failure to sense is a result of the inability of the pacemaker to sense the native cardiac activity. It is recognized by noting pacemaker spikes on the ECG despite the patient's intrinsic cardiac rate being higher than the pacemaker's programmed rate. Failure to capture is detected by the lack of a QRS complex (i.e., myocardial depolarization) or a lack of a QRS complex after an appropriately timed and placed pacemaker spike on the ECG (Figure 35-3).** Failure to pace, sense, and capture are precipitated by changes in the circuit formed by the pacemaker and the myocardium.

Problems can arise at any part of the circuit. The battery can be depleted. Circuitry within the generator can be damaged. Leads can fracture, get disconnected from the generator, or dislodge from the myocardium. Lead insulation can break. The myocardium completes the circuit, and changes (e.g., fibrosis, inflammation, ischemia, or metabolic changes) can affect pacemaker function. **Problems at the myocardial interface are numerous, common, and time sensitive. Hyperkalemia, ischemia, hypoxia, hypercarbia, or many other metabolic derangements can alter the pacing threshold or change the intrinsic electrical activity sensed (Table 45-2).** Malfunctions can arise from the damage or failure of the circuitry within the generator that carries out the programming.

Given the complexity of pacemaker programming and safety features, an approach that considers both malfunctions and pseudomalfunctions may be more useful to the Emergency Physician.

TABLE 45-2 Factors That Can Change Pacemaker Capture Thresholds[18]

Metabolic factors that increase the capture threshold
Acidosis
Alkalosis
Hypercarbia
Hyperkalemia
Hypoxemia
Myxedema
Severe hyperglycemia
Medications that increase the pacing threshold
Dofetilide (class III antiarrhythmic)
Flecainide (class IC antiarrhythmic)
Mineralocorticoids
Propafenone (class IC antiarrhythmic)
Sotalol (class III antiarrhythmic)
Medications that possibly increase the pacing threshold
Beta-adrenergic blockers (class II antiarrhythmic)
Ibutilide (class III antiarrhythmic)
Lidocaine (class IB antiarrhythmic)
Procainamide (class IA antiarrhythmic)
Quinidine (class IA antiarrhythmic)
Medications that decrease the pacing threshold
Atropine
Epinephrine
Glucocorticoids
Isoproterenol

Figure 45-4 summarizes the differential diagnosis of the potential pacemaker malfunctions based on the ECG appearance.

FAILURE TO CAPTURE

Failure to capture is detected by the lack of a QRS complex or lack of a QRS complex after an appropriately timed and placed pacemaker spike on the ECG (Figure 35-5). It occurs when the generated pacing impulse is incapable of effectively depolarizing the myocardium. This can be due to inadequate output delivered to the myocardium or to an increased threshold required due to conditions at the tissue interface. Lack of capture or intermittent capture could be a result of the inadequate energy generation by the pacemaker (i.e., battery failure), increased resistance at the electrode-myocardium interface (i.e., lead fracture or displacement), poor electrode positioning, prolongation of the refractory state of the myocardium (e.g., myocardial infarction, electrolyte abnormalities, supratherapeutic levels of antidysrhythmic drugs), or perforation of the myocardium by the electrode (**Table 45-2**).[16] **Hyperkalemia and ischemia are common and immediately life-threatening causes of increased capture threshold.** Isoproterenol has been shown to be an effective therapy for patients with failure to capture from high antidysrhythmic drug levels.[16,17] Fibrosis and inflammation at the tissue interface in the weeks following implantation or from cardiomyopathies can increase the capture threshold. Any critically ill patient with a pacemaker may develop failure to capture.

Failure to capture during the postimplantation period could result from an elevated voltage threshold for pacing due to tissue changes at the electrode-myocardium interface.[16,17] The occurrence of postimplantation failure to capture typically occurs in the first few weeks after implantation. Its incidence has decreased because of recent advances in steroid-eluting leads.[16,17]

A poor threshold may be present from the time of implantation. A chronic rise in threshold can be related to fibrosis around the tip of the lead causing lack of capture or intermittent capture. Severe metabolic abnormalities and drugs can increase the pacing

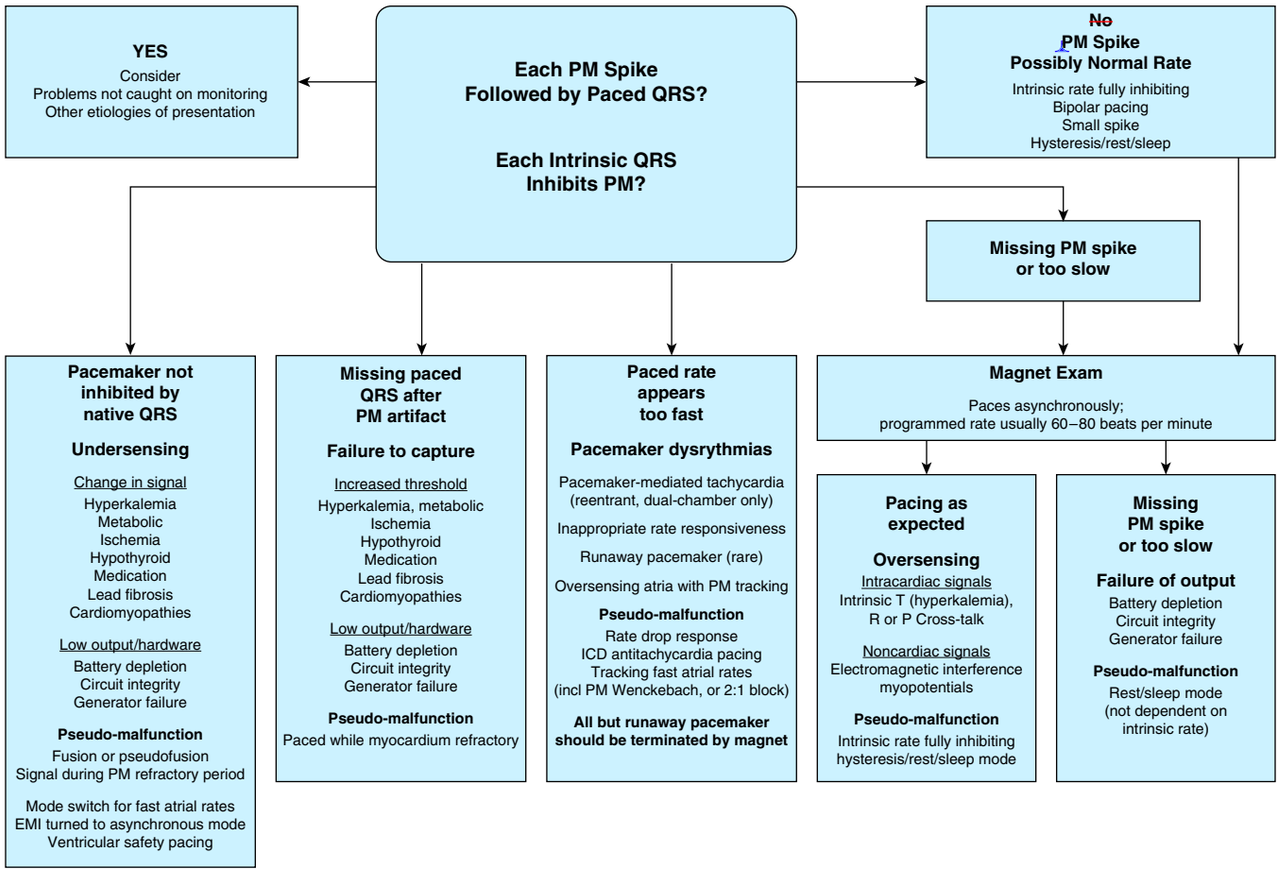

FIGURE 45-4. Differential diagnosis of pacemaker malfunction (PM). EMI, electromagnetic interference.

threshold. A myocardial infarction involving the myocardium at the tip of the pacer leads will cause a rise in the pacing threshold. Lead fracture and poor connections between the electrode and generator can present as lack of capture or intermittent capture. The pacemaker generator battery may fail and present with too low a voltage to capture the heart but enough voltage to generate a pacemaker spike. Insulation breaks in the pacemaker lead allow parallel electrical circuits to occur in the system and may cause various pacemaker abnormalities.

FAILURE TO PACE

Failure to pace is a result of either pacemaker output failure (lack of a pacer spike) or failure to capture (lack of a myocardium stimulation after a pacer spike). Failure to pace is noted by a lack of the pacemaker spike on the ECG and the failure to deliver a stimulus to the myocardium when there is a pause in the intrinsic cardiac electrical activity. **The pacer-dependent patient may complain of chest pain, dizziness, lightheadedness, weakness, near-syncope, syncope, or other signs of hypoperfusion.**

Failure to pace can be intermittent or continuous, cause long pauses on telemetry, or show a rate slower than the programmed minimum rate. This can be life threatening for a pacemaker-dependent patient. Be prepared to use transcutaneous pacing (Chapter 41) or to place a transvenous pacemaker (Chapter 43) for potentially unstable patients.

FAILURE TO PACE DUE TO OVERSENSING

Oversensing is the inappropriate inhibition of the pacemaker due to its sensing of signals that it should otherwise ignore. The most common reason for failure to pace is oversensing. The pacemaker is inappropriately inhibited. Oversensing may be due to intracardiac signals (e.g., P waves, R waves, or T waves) or noncardiac signals (e.g., myopotentials from the chest wall). T waves may be sensed inappropriately (e.g., hyperkalemia, LV hypertrophy, and severe hyperglycemia).[9] The R wave may be sensed as an intrinsic atrial signal. This is reflected on the ECG as a slow rate or a prolongation of the pacing spike interval (**Figure 45-6**). Ventricular oversensing is associated with slow heart rates since the pacemaker is

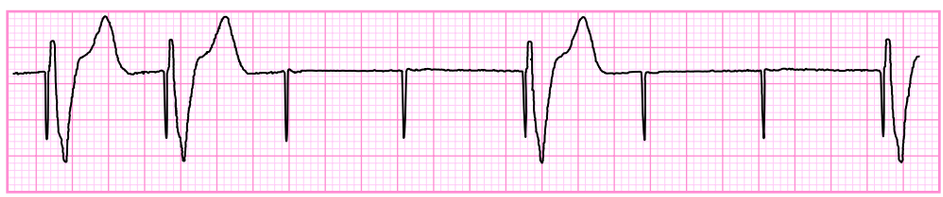

FIGURE 45-5. ECG monitor strip of an AV sequential pacemaker demonstrating lack of capture or intermittent capture.

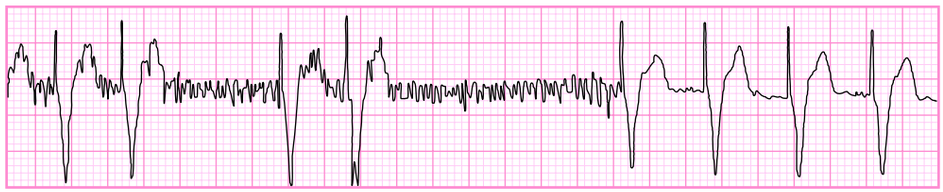

FIGURE 45-6. ECG monitor strip demonstrating oversensing.

inhibited by intrinsic signals. Atrial oversensing may cause fast rates if the pacemaker is tracking misinterpreted signals that are fast. The patient may develop symptoms from loss of AV synchrony if the pacemaker stops tracking these signals by automatic mode switch from DDD to VVI.

Cross-talk when a paced event from one chamber is mistakenly interpreted as activity in the other chamber is a form of intracardiac oversensing. Brief blanking periods immediately after pacing during which no sensing occurs are meant to prevent cross-talk. Ventricular safety pacing within a programmed cross-talk window following the blanking period is additional protection against asystole when there is cross-talk. Noncardiac signals may be sensed (e.g., electromagnetic interference [EMI] and myopotentials from the chest wall or diaphragm).

EMI can cause intermittent oversensing. Pacemakers may be programmed to turn to VVI or VOO as safety features when there is ongoing EMI. This mode switch prevents inappropriate inhibition, resulting in long pauses or asystole. EMI is unlikely to affect CRMD outside of the medical environment.[18,19] Nonmedical sources of EMI include airport metal detectors, cellular phones, cosmetic lasers, and retail antitheft sensors. It is recommended to avoid keeping a cellular phone over the CRMD pocket and to avoid lingering near antitheft sensors in stores. Hospital sources of EMI include cardioversion, defibrillation, dental composite curing, electrocautery, ionizing radiation, magnetic resonance imaging (MRI) machines, and radiofrequency ablation.[20] The battery-powered single-use cautery devices used in the Emergency Department are unlikely to cause CRMD malfunction.[20]

Oversensing has decreased in prevalence due to the use of bipolar pacing devices.[21] Oversensing can be detected by placing a magnet over the pacemaker. The pacemaker will fire at the programmed rate if it is working properly. This indicates that the failure to pace the myocardium in a patient with bradycardia is due to oversensing. The unit may be sensing a large T wave as a QRS complex. Alternatively, it may be sensing a normal T wave as a QRS complex if the QRS complexes are small in amplitude. Tape the magnet in place over the pacemaker generator if a patient's bradycardia is corrected. The magnet may not be directly over the pacemaker generator if the generator is pacing intermittently. Reposition the magnet and observe the results. A component of the system (i.e., generator, battery, or leads) has failed if no pacemaker spikes are seen on the ECG. The battery may be depleted or the set rate has been changed if the pacemaker spikes occur at less than the programmed rate.

FAILURE TO PACE DUE TO FAILURE OF OUTPUT

Failure of output can originate from problems in any component of the pacemaker hardware or discontinuity of the circuit (**Figure 45-7**). The closed circuit must be intact for current to be delivered. Failure to pace can result from the lead being disconnected from the generator or dislodged from the myocardium, fractured leads, or insulation failure. Batteries commonly used in pacemakers can last 5 to 10 years depending on proportion of time paced, impedance from leads and the tissue interface, and the programmed output. Pacemakers are programmed to show elective replacement indicators of battery depletion, which vary by manufacturer. Many indicators begin notifications when they have 3 months of remaining life. The indicator may be an incremental slowing of the rate or a lower paced rate on the magnet examination, which puts the pacemaker in asynchronous mode. The pacemaker behavior and its behavior in magnet mode can be unpredictable at the end of battery life.[18] Battery depletion can cause failure to pace, failure to sense, or failure to capture.

The incorrect diagnosis of a failure to pace and a lack of pacemaker output can be made if the patient's pacemaker spike is very small. The evaluation of multiple leads of the ECG tracing usually prevents this misdiagnosis.

A magnet exam distinguishes oversensing from failure of output in the setting of a missing pacemaker spike or an inappropriately slow rate. The magnet flips a reed switch that turns off sensing and initiates pacing at a programmed rate, usually at a rate of

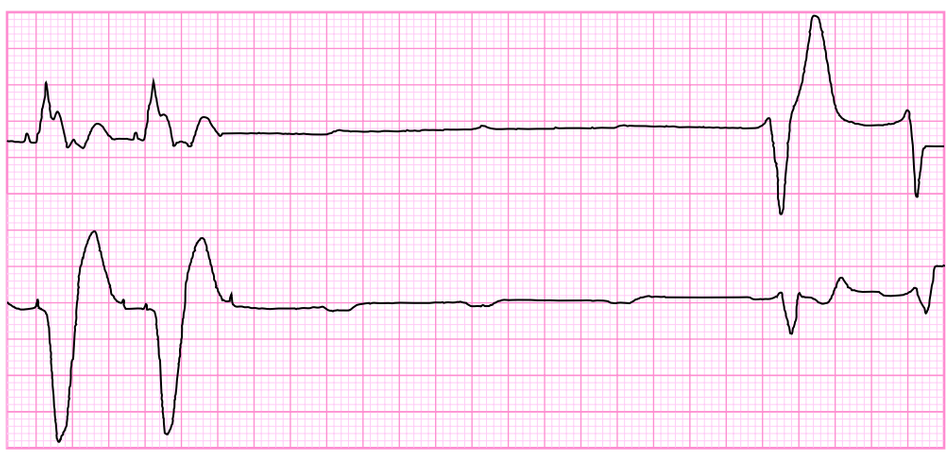

FIGURE 45-7. ECG monitor strip demonstrating failure to pace from output failure.

60 to 80 beats per minute in DOO or VOO mode. The magnet will restore pacing when failure to pace is from oversensing.

The magnet can be taped in place over the pacemaker generator until the pacemaker can be reprogrammed. Make sure the magnet is directly over the pacemaker generator if pacing is intermittent. Two magnets may be needed if the patient is obese or the generator is behind the pectoral muscle. A component of the system (i.e., battery, generator, or leads) has failed if no pacemaker spikes are seen on the ECG. The battery may be depleted or the set rate has been changed if the pacemaker spikes occur at less than the programmed rate.

FAILURE TO SENSE OR UNDERSENSING

Failure to sense is a result of the inability of the pacemaker to sense the native cardiac activity. It is recognized by noting pacemaker spikes on the ECG despite the patient's intrinsic cardiac rate being higher than the pacemaker's programmed rate (**Figure 45-8**). The pacemaker does not sense the preceding QRS complex appropriately and fires. Patients might present with weakness, lightheadedness, and syncope due to alterations in rhythm from competition with the native cardiac rhythm. Causes of undersensing include conditions that alter the nature of cardiac signals (e.g., new bundle branch blocks, myocardial ischemia, PVCs, or premature atrial contractions). Other etiologies of failure to sense include battery failure, breaks in the lead insulation, inappropriate sensitivity programming of the pulse generator, lead dislodgement, poor electrode position, and reed switch malfunction. Many of these etiologies can result in failure to capture. Low-amplitude QRS complexes or broad QRS complexes with a low slow rate (e.g., a bundle branch block) may be responsible for pacemaker sensing problems.[18]

Multiple pseudo-malfunctions can appear to be undersensing. Fusion beats and pseudo-fusion beats reflect the normal delay required for sensing and inhibition (**Figure 45-9**). Pacing may occur immediately after an intrinsic beat that occurs within the blanking and refractory period. No sensing occurs, and intrinsic beats will be ignored during the blanking period immediately after pacing. Intrinsic signals are sensed and recorded but do not alter the timing cycle during the refractory period and do not inhibit the pacemaker. Ventricular safety pacing appears as a pacemaker spike closely following a PVC that occurs within the cross-talk window. Automatic mode switch for fast atrial rates and electromagnetic interference can cause a pacemaker to change to asynchronous pacing.

PACED RATE APPEARS FAST

Most pacemaker dysrhythmias can cause a fast rate, but most will not exceed its programmed upper rate limit or maximum tracking rate (MTR). True malfunctions include pacemaker-mediated tachycardia, inappropriate rate response, and oversensing the atria.

PACEMAKER-MEDIATED TACHYCARDIA

Pacemaker-mediated tachycardia (PMT) is a reentrant loop tachycardia in which the pacemaker fires at a high rate and is only seen with dual-chamber pacemakers that sense the atria (**Figures 45-10** and **46-4**). Retrograde conduction of a PVC or a paced ventricular depolarization through the AV node causes the pacemaker to oversense the atrium, trigger another pacing stimulus in the ventricle, and form a reentrant loop. PMT will not exceed the MTR. Treatment of PMT requires interrupting the reentrant loop (**Figure 46-4**).

A magnet examination will terminate PMT since the reentrant loop requires sensing. This may also be accomplished by cutting the leads after they exit the generator, isometric pectoral exercises, medication that blocks the AV node (e.g., adenosine, beta-blockers, or calcium channel blockers), reprogramming, transcutaneous pacing, or a Valsalva maneuver.[22] Consult a Cardiologist prior to performing any of these maneuvers. Modern pacemakers have algorithms to prevent and terminate PMT. The blanking period after a pacing event during which no sensing occurs and the cross-talk window that triggers ventricular safety pacing is meant to prevent PMT.

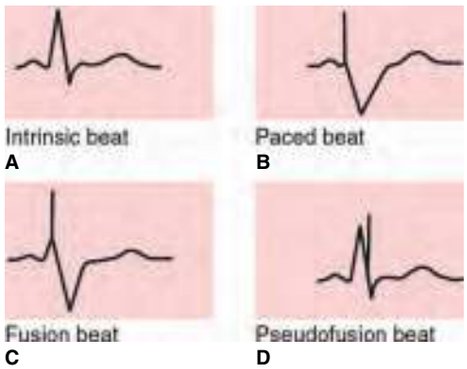

FIGURE 45-9. Schematic of typical ECG beats.

TRACKING ATRIAL TACHYCARDIA

The patient may present with heart rates up to the upper rate limit due exercising or tracking a supraventricular tachycardia (e.g., atrial fibrillation or oversensing atrial signals) if the pacemaker does not have an automatic mode switch to stop tracking fast atrial rates. The pacemaker will prolong the AV delay progressively to prevent pacing faster than the upper rate limit as the atrial rate exceeds the upper rate limit. The ECG will show a pacemaker Wenckebach with progressively longer PR intervals and then a 2:1 block if the rate continues to increase.[8]

INAPPROPRIATE RATE RESPONSIVENESS (SENSOR-DRIVEN TACHYCARDIA)

Rate-responsive pacemakers use an accelerometer to measure movement and chest wall electrical impedance to measure minute ventilation as an estimate of metabolic demands from exercise. The accelerometer can misinterpret movement of the generator by arm, shoulder, or muscle tremor. Interference from monitoring

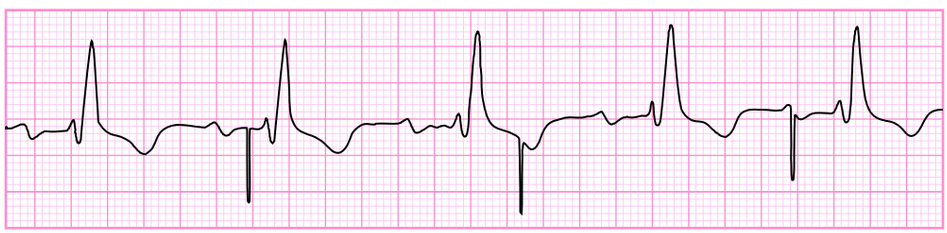

FIGURE 45-8. Schematic of an ECG monitor strip demonstrating lack of appropriate sensing or failure to sense.

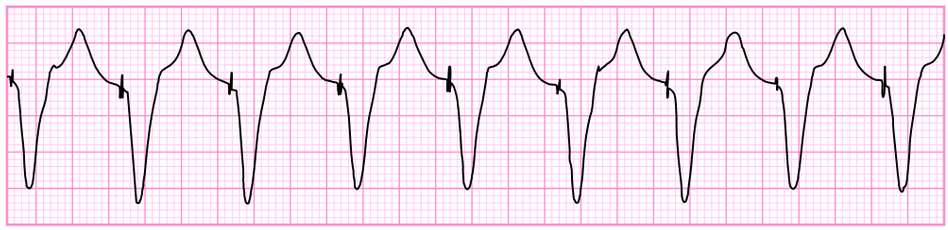

FIGURE 45-10. Schematic of an ECG monitor strip demonstrating pacemaker-mediated tachycardia. (Used from https://commons.wikimedia.com.)

equipment or hyperventilating can trigger an inappropriate rate response. Chest wall impedance is used in common cardiac monitoring equipment to measure respiratory rate and detect when the ECG leads get disconnected. The resulting tachycardia is a wide complex that is regular and may be mistaken for VT.[23,24] Taking the patient off the monitor will restore normal pacing if the tachycardia is due to inappropriate rate response to the monitoring equipment. Rate responsiveness is turned off when a magnet is placed over the pacemaker generator. Asynchronous pacing of an intrinsic T wave can precipitate VF if the patient is in VT.[23,24]

RATE DROP RESPONSE

Patients with recurrent vasovagal syncope may have pacemakers programmed to have a rate drop response that can be mistaken for malfunction. The pacemaker detects a sudden drop in the intrinsic rate and responds with 1 to 2 minutes of pacing at 100 to 120 beats per minute or 70% to 80% of MTR. This has been shown to reduce episodes of syncope from 1.2 to 0.3 per month.[25] The battery may be depleted or the set rate may have been changed if the pacemaker spikes occur at less than the programmed rate.

RUNAWAY PACEMAKER

Runaway pacemaker is an exceedingly rare pulse generator failure. Its incidence has dropped dramatically with improved batteries, improved circuitry, and improvements in hermetic sealing of the generator. Runaway pacemaker may cause dramatically rapid rates, unlike PMT, which does not exceed the programmed upper rate limit.[16,17,26] Rates higher than 2000 beats per minute have been reported. The actual heart rate is unpredictable because pacemaker impulses from a failing generator may not all meet capture threshold to trigger ventricular depolarization. **The frequent pacing stimuli increase the risk of an R-on-T phenomenon inducing VT or VF.** Magnet application has no effect on runaway pacemaker since turning off sensing would not fix the generator's mechanical failure.[18,27] Treatment requires emergent pacemaker reprogramming, emergent replacement, or disconnecting (i.e., cutting) the leads in the pacemaker pocket.[9,18,26]

ANTITACHYCARDIA PACING

A pseudo-malfunction that may exceed the MTR is antitachycardia pacing (ATP) in a patient with an ICD. The ICD uses a programmed algorithm to terminate the identified dysrhythmias of VF and VT. The algorithm includes one or more trials of ATP with 6 to 10 paced impulses at a rate close to the VT rate. Using ATP will terminate VT quickly and painlessly in up to 89% of cases with VT rates of 188 to 250 beats per minute and prevents painful shocks.[28,29]

PACEMAKER COMPLICATIONS

There are many complications associated with pacemakers and their insertion. Common complications include lead fractures, pneumothorax, pocket infections, thrombosis, and tricuspid regurgitation (**Figure 45-11**).[30] They can be divided into short-term (**Figure 45-12**), intermediate-term (**Figure 45-13**), and long-term (**Figure 45-14**) complications.

ACUTE COMPLICATIONS FROM PACEMAKER INSERTION (IMPLANTATION)

Complications may occur from the implantation procedure. Discomfort and ecchymosis at the incision site or the pacemaker pocket are common in the first few days. Dehiscence of the incision can occur, especially if a large hematoma in the pocket puts excessive stress or pressure on the incision. Nonsteroidal anti-inflammatory drugs, excluding aspirin, are adequate and appropriate to alleviate the discomfort. Assure the patient that the discomfort and ecchymosis will resolve spontaneously. The patient should not be taking aspirin in the immediate postimplantation period unless authorized and/or prescribed by the Cardiologist. The pacemaker can migrate, cause pressure on the overlying skin, and result in skin erosions that require pacemaker relocation and wound debridement.

Complications from the procedure include all the complications of central venous catheterization (Chapter 63), complications from lead placement within the heart, and placement of the generator in a subcutaneous pocket.[31] Central venous line complications include air embolism, arterial puncture, arteriovenous fistula, bleeding, brachial plexus injury, hemothorax, infection, and pneumothorax. Incision and pocket complications include ecchymosis (**Figure 45-12*B***), generator migration, hematoma formation (**Figure 45-12*B***), infection, and wound dehiscence. The transvenous leads are placed under fluoroscopy and affixed by screws or passively lodged into the trabecular endocardial surface. Placement may cause injury to vessels, leading to bleeding or thrombosis. Complications within the heart

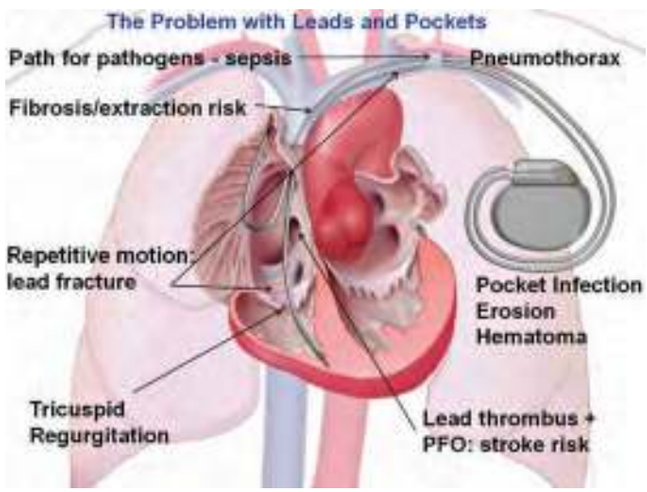

FIGURE 45-11. Some of the more common complications associated with pacemakers. PFO, patent foramen ovale. (Used with permission from reference 30.)

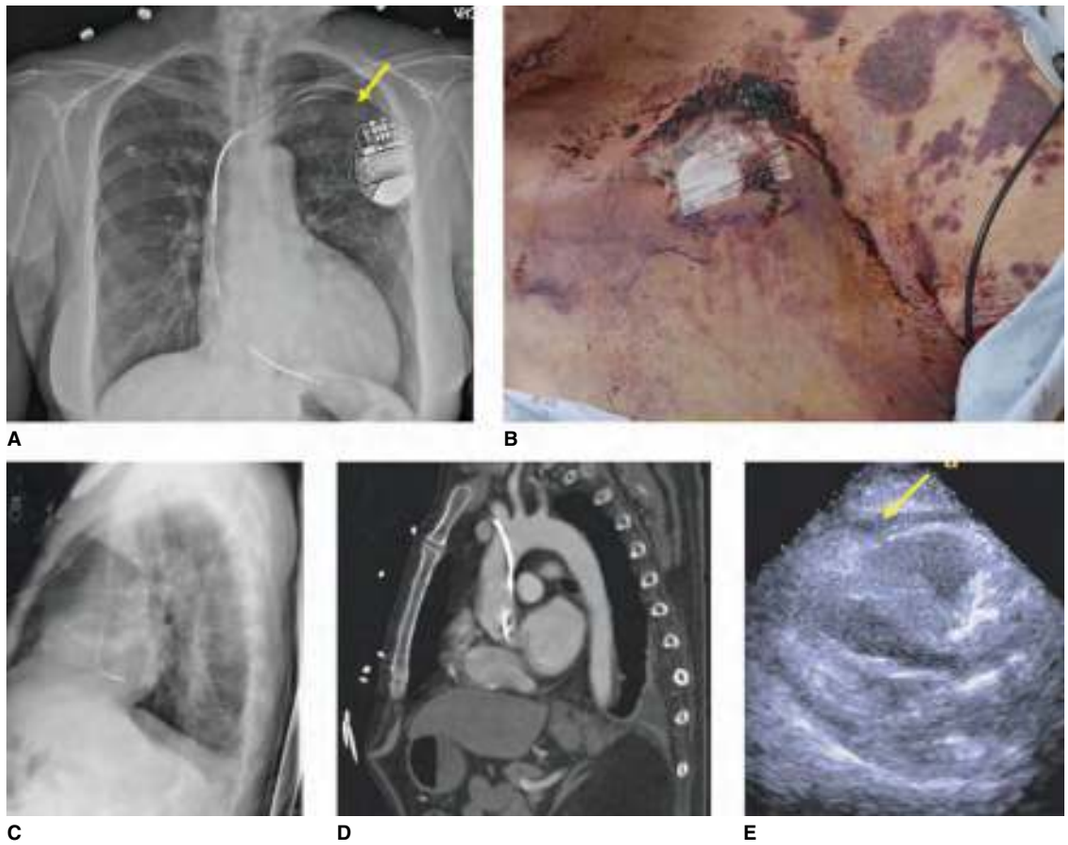

FIGURE 45-12. Short-term complications associated with pacemaker implantation. ***A.*** Pneumothorax. The *arrow* highlights the border of the collapsed lung. ***B.*** Hematoma and ecchymosis. ***C.*** Chest radiograph of arterial placement of pacing lead. The lead is pointed posteriorly while traveling through the ascending aorta. ***D.*** CT image of figure C. ***E.*** Acute pericardial effusion (*arrow*) after right ventricular lead implantation. (Used with permission from reference 7.)

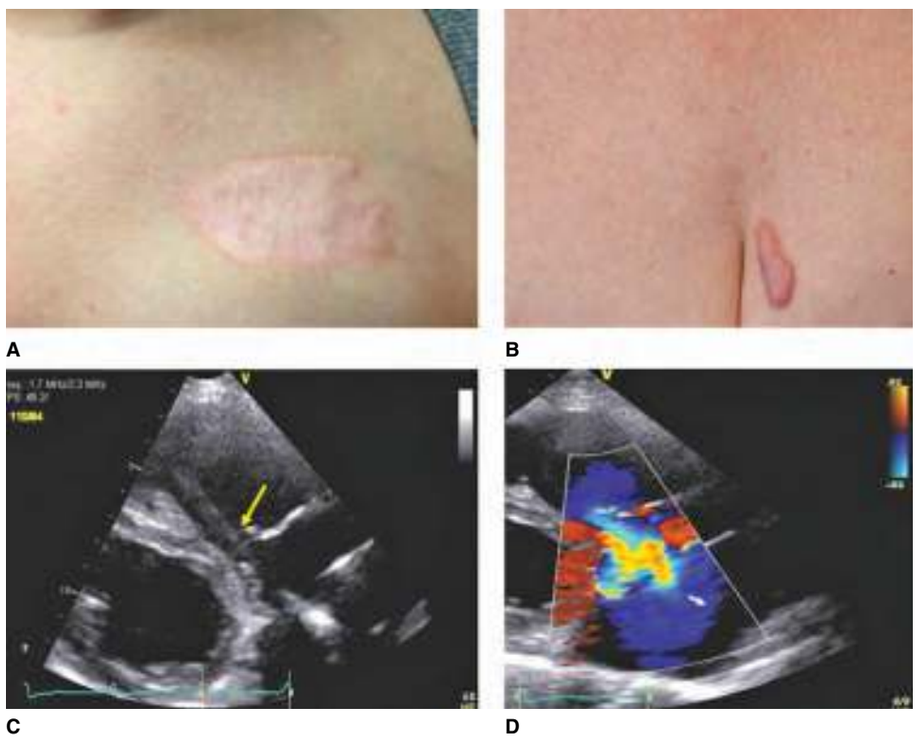

FIGURE 45-13. Intermediate-term complications associated with pacemaker implantation. ***A.*** Hypertrophic scar. ***B.*** Keloid formation. ***C.*** M-mode echocardiographic image of the RV lead through the tricuspid valve (*arrow*). ***D.*** Doppler image of figure C demonstrating severe tricuspid regurgitation. (Used with permission from reference 7.)

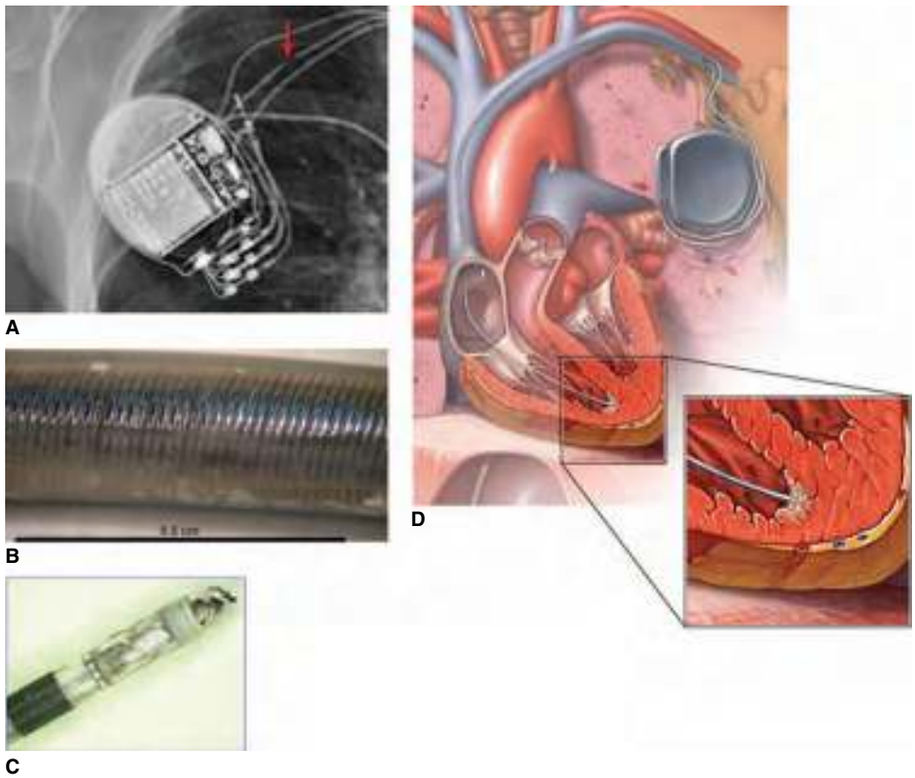

FIGURE 45-14. Long-term complications associated with pacemaker implantation. ***A.*** Chest radiograph demonstrating a lead fracture (*arrow*). ***B.*** Lead insulation break. ***C.*** Crystal deposition in a lead leading to high thresholds and impedances. ***D.*** Fibrosis at the lead-myocardial interface can result in high thresholds, impaired sensing, and exit blocks. (Used with permission from reference 7.)

include perforation and valve damage (**Figures 45-12*E*, 45-13*C*, and 45-13*D***). Placement of the LV lead for biventricular pacing is technically challenging and has been associated with coronary sinus dissection.[32] The leads can be misplaced (**Figures 45-12*C* and *D***).

PACEMAKER SYNDROME

The pacemaker syndrome is caused by the adverse hemodynamic effects of AV asynchrony causing the patient to be symptomatic or limiting their ability to be fully functional. The pacemaker is usually functioning as programmed. Patients may complain of anxiety, apprehension, dizziness, dyspnea, fatigue, or neck pulsations. The Mode Selection Trial proposed the following diagnostic criteria: (1) new or worsened dyspnea, edema, elevated jugular venous pressure, orthopnea, or rales with ventriculoatrial conduction during ventricular pacing; or (2) dizziness, presyncope, syncope, or weakness with a > 20 mmHg reduction in systolic blood pressure when the patient is in VVIR pacing compared with atrial pacing or normal sinus rhythm.[33]

AV asynchrony leads to decreased cardiac output and decreased LV filling. Elevated atrial pressures may initiate a vagal reflex, causing near-syncope or syncope. High wedge pressure results in shortness of breath. Patients with retrograde 1:1 ventricular to atrial conduction are more symptomatic.[33,34] Pacemaker syndrome can occur in the absence of retrograde conduction.[33,34] This diagnosis will not be clear from the emergency evaluation since symptoms are nonspecific and comprehensive echocardiography is not usually available around the clock. Other life-threatening causes of symptoms (e.g., ischemia or sepsis) must be evaluated. Pacemaker syndrome may resolve with a change from ventricular pacing (e.g., VVI to a mode that restores AV synchrony such as DDD). Provide supportive care until the pacemaker can be upgraded to one that restores AV synchrony (e.g., changing a single-chamber pacemaker to a dual-chamber pacemaker).

HEMATOMAS

A hematoma may form at the site of the subcutaneous pacemaker generator (**Figure 45-12*B***). This can be due to anticoagulation therapy, aspirin therapy, or an injury to a subcutaneous artery or vein. A hematoma can be managed with the application of dry, warm compresses to the area and oral analgesics. Hematomas of the pocket are usually self-limited but should be followed closely. History of a postoperative hematoma is an independent risk factor for infection.[35,36] The Cardiologist may choose to evacuate the hematoma if it compromises the incision site. Patients taking aspirin and anticoagulants may have held the medications before the procedure, but bleeding complications may still occur. Risks and benefits of continuing these medications should be discussed between the patient and their Cardiologist. **Do not aspirate to evacuate the hematoma because it can cause further trauma, may introduce infection, or may damage the device.**[22] Aspiration often does not work and does not address the etiology of the bleeding.

LEAD COMPLICATIONS

Acute presentations of pacemaker malfunction may be due to mechanical complications of leads (e.g., disconnection, dislodgment, fracture, perforation, or extracardiac pacing) (**Figure 45-14**).[31,36] Leads can become disconnected from the generator soon after implantation. Dislodgement of leads from the myocardium often occurs within 3 months of implantation.[37]

Perforation may occur during the implantation or occur over time when a lead erodes through the myocardium. The incidence

of late lead perforation is thought to be < 1% based on manufacturer registry data.[38] Patient presentations ranged from asymptomatic, dizziness, and fatigue to symptoms of extracardiac pacing (e.g., chest wall pacing, hiccups, and intercostal muscle contractions), pericarditis, pericardial effusion, or tamponade (**Figure 45-12*E***). A change from a left bundle branch block (LBBB) pattern to a right bundle branch block (RBBB) pattern on the ECG can represent a septal perforation. Chest radiographs or echocardiography may make the diagnosis but are not sensitive enough to exclude perforation. Computed tomography (CT) is widely available at all hours but provides only a static image and lacks specificity. Exact localization of the lead tip is limited by artifact. In a study of 100 patients with pacemakers who received unrelated chest CT scans, 15 were thought to have a cardiac perforation. This far exceeds all other estimates of lead perforation incidence. Diagnosis can be made by fluoroscopy. Management is by surgical or transvenous removal of the leads, both of which carry significant risks.[36,39]

Extracardiac pacing is unusual with bipolar pacing leads as the current travels a very small distance between electrodes. Hiccups or chest wall complaints from diaphragmatic or intercostal pacing can occur with or without perforation of the RV wall. An insulation break, defect in the pacing wire, or other area of current leakage can stimulate the pectoral or other chest skeletal muscle. Changes in programming may minimize the stimulation until the defective component can be replaced.[37] Decreasing the pulse width and/or voltage output can minimize the stimulation until the defective component can be replaced.

Pectoral muscle stimulation is less common with the currently available bipolar pacemakers. An insulation break or defect in the pacing wire before it enters the subclavian vein will allow the current to flow into the pacemaker generator and cause skeletal muscle stimulation. This can also be seen with current leakage from the connector of the pacing wires or sealing plugs. In rare instances, erosion of the protective coating of the pacemaker generator can cause this phenomenon. Decreasing the pulse width and/or voltage output can minimize the stimulation until the defective component can be replaced.

THROMBOSIS

Thrombosis and stenosis of the veins (e.g., brachiocephalic or subclavian) or pacemaker leads are common (**Figure 45-15**).[31,40] The incidence of thrombosis is unclear as many patients are asymptomatic. A prospective study of 145 patients with newly implanted leads found 23% developed venous thrombosis on Doppler ultrasound during follow-up at 3, 6, and 12 months. Only 3 of the 34 patients diagnosed with thrombosis had any signs or symptoms. An increased risk of thrombosis was seen with each additional pacemaker lead, hormone therapy, and history of thrombosis.[41] Patients with thrombosis may present with ipsilateral arm swelling and discomfort. Patients may develop superior vena cava syndrome if the thrombosis propagates to the superior vena cava. Upper extremity deep venous thrombosis (DVT) requires anticoagulation and not removal of the leads.[42] A thrombus can form in the right atrium.[43] Patients may present with symptoms of a pulmonary embolism.

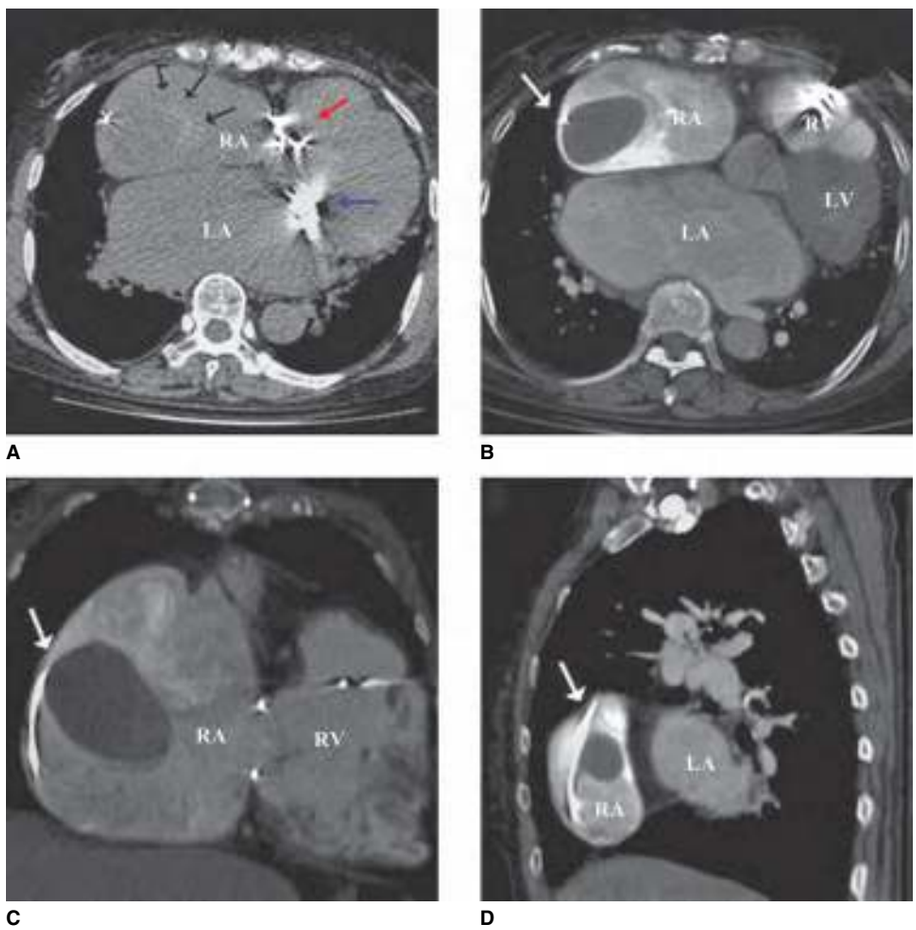

FIGURE 45-15. Chest CT angiography demonstrating a large mass attached to the lead (*white arrows*) in the right atrium. LA, left atrium; RA, right atrium. (Used with permission from reference 40.)

INFECTION

CRMD poses a risk of infection. Infections can occur as a complication of implantation or in a long-implanted device.[31] Incisional and pocket infections may present with erythema, fluctuance, tenderness, or dehiscence of the wound (**Figure 45-16**). *Staphylococcus* species are the most common organisms.[1,44] *Staphylococcus* species adhere to polymers and create biofilms, which makes CRMD especially vulnerable to colonization. **Do not aspirate the pocket for cultures as this can cause damage the device or introduce infectious agents.** No clinical signs or symptoms will rule out spread of the infection from the pocket to the intravascular leads or endocardium. Draw blood cultures and begin empiric broad coverage for skin flora.

Infection of the leads or device-related endocarditis is much more serious. **Consider endocarditis in any patient with a pocket infection or a fever without a clear alternative source.** Patients may have an indolent course or present acutely ill with heart failure, sepsis, or septic emboli. The incidence of CRMD infection is 1% to 1.3%, but it carries a 3.7% to 11.3% mortality.[35]

The diagnosis of a CRMD infection may be hard to make in the ED. There is no uniform gold standard. Diagnostic criteria include positive blood cultures, echocardiogram evidence of vegetations (e.g., on the leads, valves, or endocardium), absence of other causes for fever, and resolution of symptoms after device removal. Pocket infections can spread to intravascular leads and the endocardium without obvious local signs of inflammation. Bacteremia can seed an infection of a CRMD. *Staphylococcus* bacteremia from a documented non-CRMD source has been shown in one study to seed the CRMD in 36% of cases, with ICDs most vulnerable.[45]

CRMD infections are especially hard to treat, with a recurrence rate of 50% without full lead extraction. The most significant risk factors for CRMD infection include postoperative hematoma, history of prior CRMD infection, end-stage renal disease, steroid use, diabetes mellitus, and an abdominal generator pocket.[35] Transthoracic echocardiogram may detect vegetations in only 23% of cases, compared to the transesophageal approach, which detects vegetations in 94% of cases.[46] Standard treatment has been at least 6 weeks of intravenous antibiotic therapy and device removal.[1,47] Lead extraction, however, is technically challenging and carries significant risk of bleeding, perforation, or thromboembolic complications. CRMD infection has a recurrence rate of 50% with incomplete device removal.[35]

ALLERGIC REACTIONS

Allergic reactions to the metal components of the pacemaker have been noted in the past. Current pacemaker generators and leads are coated with a substance to prevent the body from being exposed to the metal. Allergic reactions to the pacemaker covering are very rare but have been reported.

TWIDDLER SYNDROME

Twiddler Syndrome can be seen with any implantable subcutaneous device. Patients manipulate the device intentionally or unintentionally. This moves the pacemaker within the subcutaneous pocket, curling the leads (**Figure 47-17**).[48-50] Higher risk is seen in the elderly from dementia, the obese from looser pockets, and in those with obsessive-compulsive disorders. Newer pacemakers are smaller and easier to turn. View the chest radiograph for any changes. The leads can be displaced from the pacemaker, displaced from the heart, or fractured.

This can result in syncope or pacing of structures if the device is providing impulses to other structures (e.g., abdominal muscle wall spasm, arm spasm, chest wall, diaphragm spasm, and hiccups). The patient may remain asymptomatic until they receive an inappropriate discharge or die from the device not working. The device can show problems associated with lead displacement or fracture. The device needs to be fixed by the specialist who inserted it. They will

FIGURE 45-16. Signs of a pocket infection. ***A.*** Skin changes 2 weeks after implantation. ***B.*** Pocket purulence. ***C.*** Pocket erosion. (Used with permission from reference 7.)

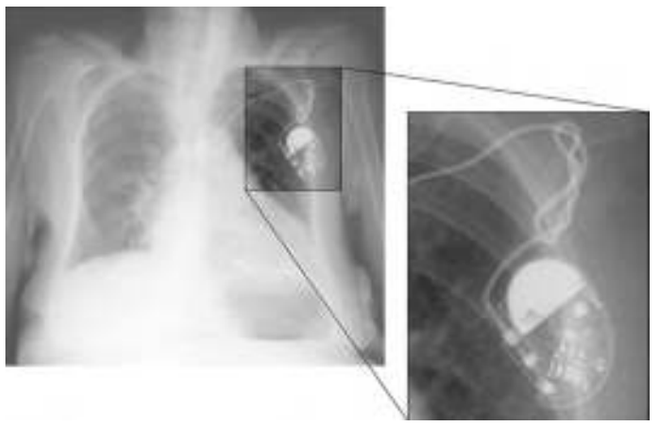

FIGURE 45-17. Chest radiograph of a patient with Twiddler syndrome. Note the twisted lead. (Used with permission from reference 7.)

replace the lead(s), reposition the lead(s), and secure the pacemaker in the pocket. This syndrome is not limited to pacemakers (e.g., ICDs, nerve stimulators, or ports). It is seen less commonly with defibrillators, which are bigger devices and harder to turn.

EMERGENCY EVALUATION OF PACEMAKER FUNCTION

Patients who present to the ED with a complaint that may be associated with their pacemaker require a thorough evaluation. True device malfunction is not as common as pseudo-malfunction. **Do not assume the pacemaker is the sole cause of a patient's symptoms.** This may delay care for other serious conditions. The myocardial interface is part of the closed circuit that allows a pacemaker to function. Any severe metabolic abnormality, conditions (e.g., ischemia or inflammation affecting the myocardium), or medications may interfere with pacemaker function (**Table 45-2**). Interrogate a pacemaker with a potential malfunction. **Do not defer other portions of the workup while awaiting interrogation.**

EMERGENCY MANAGEMENT

Assess patients with potential pacemaker malfunction for stability with the conventional "ABC" approach to the airway, breathing, and circulation. Place patient on a pulse oximeter and cardiac monitor. Obtain a full set of vital signs and an ECG. Have a low threshold to apply defibrillator pads in case the patient requires transcutaneous pacing or defibrillation, even if the patient appears hemodynamically stable. Obtain intravenous access. Identify and treat any immediate life-threatening conditions (e.g., hyperkalemia or acute ischemia).

WHAT DEVICE DOES MY PATIENT HAVE?

The simplest way to find out the kind and type of device is to ask the patient for their wallet ID card from the manufacturer. The patient may know their history, or the medical record may contain the type of device and the manufacturer. It is still very easy to identify the device by making a few calls.[51] There are only five main manufacturers of CRMD, each with a 24-hour phone number to assist clinicians (**Table 45-3**). The manufacturer representative will have a database of their patients and can arrange interrogations.[51] The appearance of the device on a chest radiograph can help identify the type of device and its manufacturer (**Figure 46-3**). An algorithm to identify devices based on the generator appearance on a chest radiograph has been made into a free mobile phone application called CRMD Finder.[51]

TABLE 45-3 Cardiac Rhythm Management Device Manufacturer Phone Numbers[51]

Manufacturer	Phone number
Biotronik	1-800-547-0394
Boston Scientific (Guidant)	1-800-CARDIAC
	1-800-227-3422
Medtronic	1-800-MEDTRONIC
	1-800-633-8766
Sorin GP (ELA Medical)	1-800-352-6466
St. Jude	1-800-PACEICD
	1-800-722-3774

CHEST RADIOGRAPHY

Obtain overpenetrated posteroanterior and lateral chest radiographs. This is helpful in locating the generator and lead positions. Look for a loose connection where the lead connects to the pacemaker generator. Manipulation of the pulse generator within the pocket may relieve or reproduce the patient's problem. A pneumothorax and/or hemothorax may be detected in patients whose pacemakers have been recently implanted (**Figure 45-12*A***).

The pacemaker lead may have become dislodged from its implantation site. This is extremely uncommon with current systems, as they have safety mechanisms to prevent lead dislodgement. Ensure that the distal end of the pacing wire is within the cardiac silhouette and against the myocardium. It may be free-floating within the ventricle or may have perforated the ventricular wall. Obtain previous chest radiographs and compare them to the current radiographs to help determine if the leads have been displaced.

Lead fractures can occur anywhere along the length of the pacing wire (**Figure 45-14*A***). They most often occur at stress points adjacent to the pacemaker or just under the clavicle as the pacing wire enters the subclavian vein. The lead has a J-shaped retention wire to help maintain its shape. The tip of the retention wire may occasionally protrude from the plastic-coated lead. This protruding wire has the potential to puncture the right atrium or superior vena cava and cause a hemorrhagic pericardial effusion that may result in cardiac tamponade (**Figure 45-12*E***).

HISTORY

Carefully obtain a history to avoid premature limiting of the differential diagnosis. Gather information from the family members, medical records, records from nursing facilities, or outside records as needed. Dizziness, near-syncope, palpitations, syncope, or any symptom that may resemble those prior to pacemaker implantation may reflect a potential pacemaker malfunction. Pertinent device history includes the type of device, when it was placed, and why it was placed. **A pacemaker-dependent patient may be in immediate danger if the device is not functioning.** Other patients may only need the device as a backup. Inquire about the most recent device check, battery change, and any recent reprogramming. Trauma or other manipulation may dislodge the leads from the myocardium, dislodge the leads from the generator, or damage the leads. Direct trauma over the pacemaker generator can render it inoperable. Inquire about infection risks (e.g., immunocompromised, injection drug use, or sources of potential bacteremia).

PHYSICAL EXAMINATION

Perform a thorough examination of the patient. Observe the vital signs for bradycardia, fever, hypertension, hypotension, or tachycardia. Evaluate the veins of the head and neck for venous engorgement

TABLE 45-4 Summary of the ECG Evaluation

1. Does the ECG suggest a condition (e.g., bradycardia, hyperkalemia, or ischemia) that needs immediate action?
2. Is the heart rate inappropriately fast, inappropriately slow, or have long pauses?
3. Atrial pacing and sensing: Are intrinsic P waves visible? Is the pacemaker sensing them, and is the pacemaker properly inhibited?
4. Atrial capture: Is each atrial pacer spike followed by a paced P wave?
5. Ventricular pacing and sensing: Are there intrinsic QRS complexes? Is the pacemaker sensing them and properly inhibited?
6. Ventricular capture: Is each ventricular pacer spike followed by a paced QRS complex?

suggesting a central venous thrombosis or a superior vena cava syndrome. Edema of the ipsilateral upper extremity indicates thrombosis and possible occlusion of the subclavian vein. Identify the location of the pacemaker pocket and implantation scar. Note if the pacemaker generator has moved from its original position. This can result in a partial or complete disconnection of the leads from the generator. Inspect the pacemaker pocket for signs of infection (i.e., a discharge, edema, skin erosion, erythema, redness, tenderness, and/or warmth).

ECG INTERPRETATION

A systematic approach to the paced ECG can help narrow the differential diagnosis when pacemaker malfunction is suspected (Table 45-4). Identify any signs of bradycardia, ischemia, or hyperkalemia that need emergent action and characterize pacemaker function. Consider obtaining a long rhythm strip for more data. Bipolar leads have very small pacer spikes that may not be visible in every lead. The lead is usually implanted in the RV with a typical paced QRS complex having an LBBB (**Figures 45-9, 45-18, 45-19, and 45-20**). An RBBB pattern may be normal in 17% of patients with RV pacing.[52] Patients with biventricular pacing may have an RBBB pattern. A new change from LBBB to RBBB suggests migration of the lead into the LV, potentially from perforation. Compare the axis of the pacer spike and QRS complex to an old ECG. Any change in axis suggests lead migration.

Determine whether the patient is presently paced and in what chamber. The QRS morphology of an intrinsic beat will be different and may have a different axis from a paced beat (**Figures 45-9*A* and *B***). A paced beat occurs immediately after a pacer spike (**Figure 45-9*B***). **Fusion or pseudo-fusion beats occur when the pacemaker fires on an intrinsically occurring P wave or QRS complex and are not signs of pacemaker malfunction (Figures 45-9*C* and *D*). A fusion beat is a QRS complex that has been formed by depolarization of the myocardium that was initiated by both the pacemaker spike and the patient's intrinsic electrical activity (Figure 45-9*C*).** The QRS morphology of a fusion beat is a hybrid of the intrinsic and paced QRS morphology. **A pseudofusion beat is a QRS complex formed exclusively by the patient's intrinsic electrical activity and resembles the intrinsic QRS complex (Figure 45-9*D*).** A pacemaker stimulus is fired just before the intracardiac voltage to activate the sensing circuit and inhibit the pacemaker so the pacemaker spike appears toward the end of a pseudo-fusion QRS complex.

A properly functioning pacemaker will sense intrinsic cardiac electrical activity. A pacemaker spike will be seen followed by a QRS complex in a single-chamber or ventricular pacemaker if the intrinsic cardiac activity is below the programmed rate (**Figure 45-18**). A pacemaker spike will be followed by a P wave and a second pacemaker spike will be seen followed by a QRS complex if the patient has a dual-chamber pacemaker (**Figures 45-19 and 45-20**). No pacemaker spike will be seen on the ECG if the intrinsic cardiac electric activity is above the programmed rate.

Patients with CRT are usually paced continuously. A biventricular pacing ECG will usually have an atrial and two closely spaced RV and LV pacing stimuli (**Figure 45-21**). The coordinated RV and LV depolarizations result in a hybrid QRS that is usually narrower

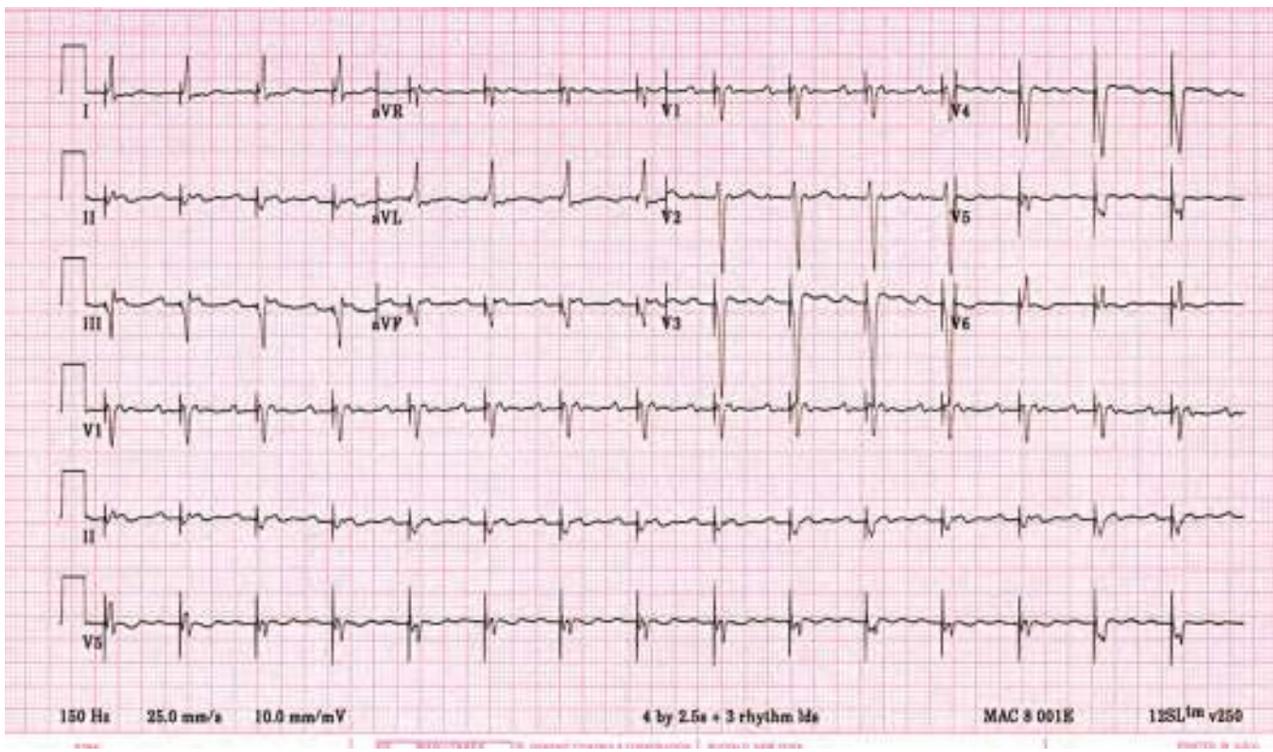

FIGURE 45-18. A 12-lead ECG of a single-chamber or ventricular pacemaker.

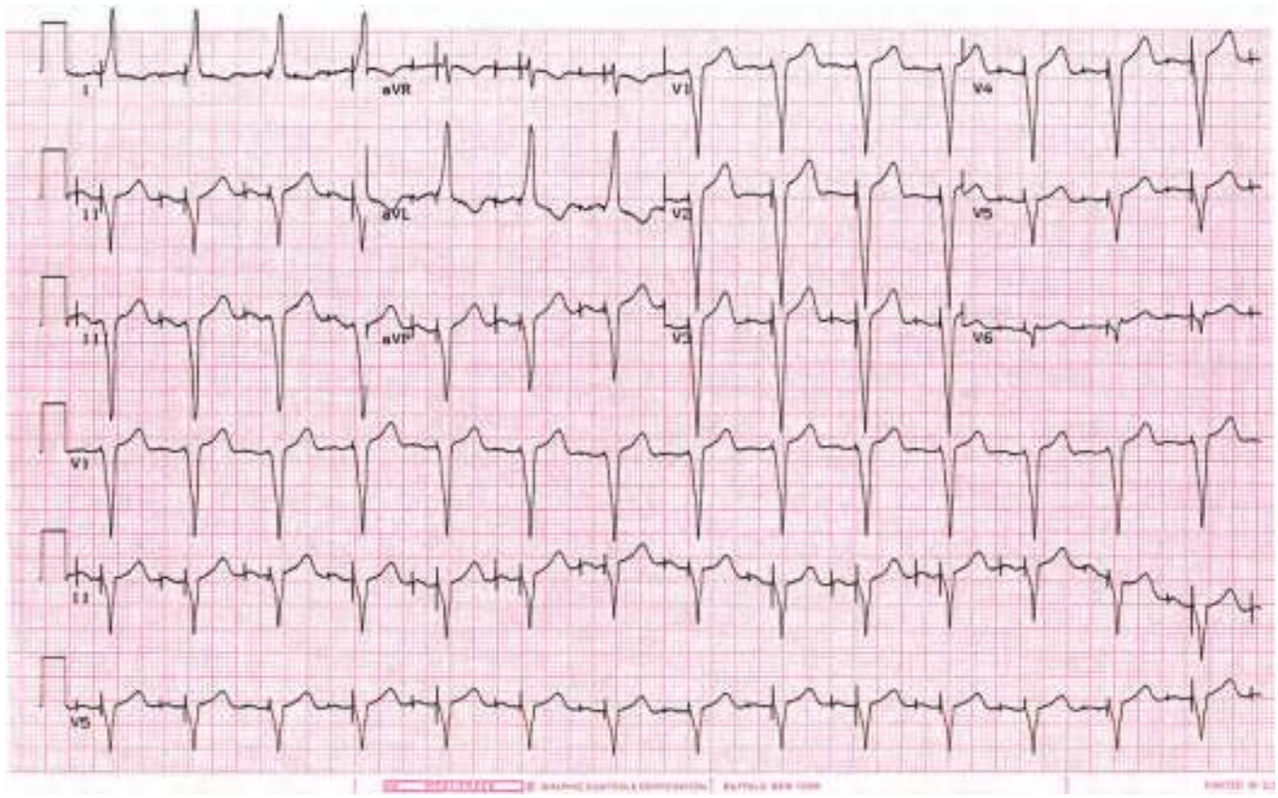

FIGURE 45-19. A 12-lead ECG of a dual-chamber or atrioventricular sequential pacemaker.

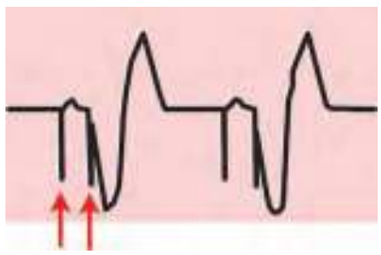

FIGURE 45-20. Schematic of an ECG monitor strip of a dual-chamber pacemaker. Atrial (*first arrow*) and ventricular (*second arrow*) pacing spikes are clearly visible.

than that of RV pacing. Lead positions are variable in CRT, resulting in variable ECG morphology.

IMAGING

Obtain a posteroanterior and lateral chest radiograph to best visualize the device, lead dislodgement, insulation breaks, or fracture. Obtain a single portable radiograph if the patient is unstable. Order an additional overpenetrated view of the device if using the radiograph to help identify the device, and be explicit in your request to the technician.

The chest radiograph can reveal the number and appearance of leads, the appearance of the pulse generator, and the manufacturer-specific radiopaque alphanumeric codes (ANC). Identification of

FIGURE 45-21. ECG monitor strip demonstrating biventricular pacing.

the ANC was less than 20% accurate on chest radiographs using standard resolution of a hospital electronic medical records system.[51] View the radiographs on a radiology monitor if possible. The number of leads and their position can help distinguish a single- or dual-chamber pacemaker with leads to only the RV. A CRT device has a third lead to the LV. Pacemaker leads are uniformly thin. ICDs and CRT-Ds will have leads that have segments of thicker shock coils. Devices with ICDs have a significantly bigger pulse generator than a pacemaker alone because they have both a battery and a similarly sized capacitor to store the higher voltage needed for defibrillation. A device with an ICD will have two radiopaque shadows within the generator. An algorithm to identify devices based on radiograph appearance of the generator was published in 2011 and has been made into a free mobile phone application (i.e., CRMD Finder).[51]

Look for complications in recently placed devices from the procedure (e.g., a pneumothorax). Confirm the leads are in the correct position. Old radiographs allow comparison for changes. A right atrial (RA) lead enters the RA and curves superiorly to reach the RA appendage. This forms a J shape on the lateral chest radiograph with the tip running medially and anteriorly. The RV lead will be L-shaped, run anteriorly and medially, and the tip near the apex. The LV lead of biventricular devices goes through the coronary sinus to the lateral or posterior wall of the LV through the cardiac veins. The LV lead on a posteroanterior radiograph may have a serpentine course running laterally or may be hard to distinguish from an RV lead. It will run posteriorly on the lateral radiograph.[53]

Other imaging may be obtained. An echocardiogram is the test of choice when suspecting device infection, lead dislodgement, lead perforation, pacemaker syndrome, or tricuspid regurgitation from lead placement. CT can also evaluate lead dislodgement or perforation. The CT scan is limited by its static nature and artifacts from the metal in the generator and leads. The advantages of CT are its wide availability and the potential to diagnose other etiologies of the patient's symptoms. Fluoroscopy provides a definitive diagnosis when there is a suspicion for lead dislodgment or lead perforation or other imaging modalities are not diagnostic.[39] Ultrasound can detect thrombosis in an upper extremity. Superior vena cava syndrome can be a clinical diagnosis confirmed and differentiated from pulmonary embolism by contrast CT. Contrast venography is the gold standard for diagnosis of superior vena cava syndrome and may be helpful for surgical planning.[54]

ADDITIONAL STUDIES

Obtain laboratory studies tailored to the clinical situation. Always obtain medication levels for cardiotoxic drugs, electrolytes, and glucose levels. Consider blood cultures for endocarditis or bacteremia, troponin for ischemia, and a lactate for infection. Order a formal device interrogation and consult the patient's Cardiologist.

MAGNET EXAMINATION

The magnet examination turns off the pacemaker's sensing function. A doughnut-shaped magnet is made for this procedure (**Figure 45-22**). A generic magnet may be used in the absence of a pacer magnet. Two magnets may be required in the obese patient or those whose generator is under the pectoral muscles. The magnet examination is usually safe, and its effects do not last once the magnet is removed. The magnet examination is performed routinely at clinic evaluations and remotely from home when a patient is established and compliant.

The magnet can distinguish oversensing from component failure as the cause of failure to pace. The magnet can terminate pacemaker dysrhythmias that depend on sensing. The magnet examination

FIGURE 45-22. An example of a pacemaker magnet.

also supplies information on battery depletion. Pacemakers at the end of their battery life are unpredictable. **Be prepared with staff and equipment in case of an emergency to start transcutaneous or transvenous pacing. Be prepared to defibrillate in the unlikely event that asynchronous pacing on an intrinsic T wave initiates VT or VF. Have Advanced Cardiac Life Support (ACLS) medications readily available.**

Place the magnet over the pacemaker generator while simultaneously obtaining a 12-lead ECG and rhythm strip. The magnetic field causes the reed switch inside the pacemaker to close. This bypasses the sensing amplifier and converts the pacemaker into the asynchronous (i.e., VOO or DOO) mode (**Figure 45-23**). The pacemaker fires at the manufacturer-specific asynchronous rate, which is usually between 60 and 80 beats per minute. The magnet rate may be slower if the battery is nearing the end of its life. The magnet examination results are unpredictable if the battery is at the very end of its life. Pacemaker spikes occurring during the refractory period of an intrinsic QRS complex will not be captured (**Figure 45-23*B***).

The application of the magnet over a generator pacing inappropriately slowly or with unexpected pauses can show a variety of results. The pacemaker will pace asynchronously at a programmed rate if it is working properly. This indicates that the failure to pace the myocardium in a patient with bradycardia is due to oversensing. The unit may be sensing a large T wave as a QRS complex. Alternatively, it may be sensing a normal T wave as a QRS complex if the QRS complexes are small in amplitude. Tape the magnet over the pacemaker generator if the bradycardia is corrected. A pacemaker continuing to pace slowly or having inappropriate pauses

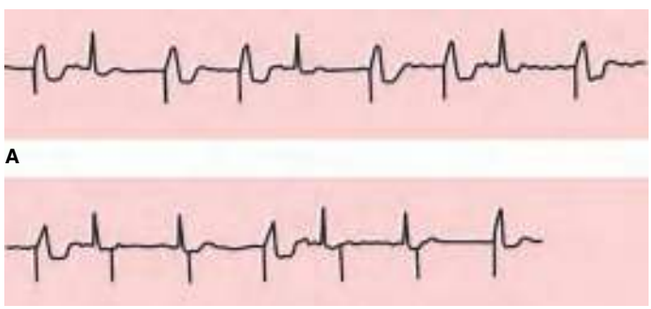

FIGURE 45-23. Schematic of a ECG monitor strip. ***A.*** Pacemaker activity without a magnet applied. ***B.*** Pacemaker activity with a magnet applied.

suggests a failure of a component in the system (i.e., battery, generator, or leads). The battery may be depleted or the set rate has been changed if the pacemaker spikes occur at less than the programmed rate. Rarely is the pacemaker in a "sleep mode" that is based on time of day.[15]

The magnet will work on a combination of pacemakers and defibrillators. The magnet application does not affect the pacemaker. The magnet switches the reed switch to turn the defibrillator off. This lasts until the magnet is removed.

PACEMAKER INTERROGATION

Pacemakers store a record of each sensed and paced event, which allows a determination of undersensing and oversensing. It also provides other information (e.g., arrhythmia logs, battery life, heart rate range, ICD discharges, lead integrity, and percentage of time pacing).[21] **The device interrogation by the manufacturer representative or cardiology technician is a critical part of the emergency evaluation of a patient presenting with symptoms that might be attributed to the pacemaker.** Each manufacturer can arrange interrogation through their 24-hour phone line (**Table 45-3**). The interrogator may be able to identify problems and reprogram the device to avoid an admission in consultation with the patient's Cardiologist.[16]

OTHER MANAGEMENT CONSIDERATIONS

DIAGNOSING ACUTE CORONARY SYNDROME

Early diagnosis and reperfusion of acute coronary occlusion are critical to reducing mortality.[55] Patients with a paced rhythm and myocardial infarction are less likely to have emergent reperfusion and have a higher mortality at 30 days and 1 year.[56] Ventricular pacing makes the ECG diagnosis of acute ischemia more challenging, but ECG can still be diagnostic. Repolarization causes baseline discordant ST-segment and T-wave abnormalities in the opposite direction of the majority of the QRS deflection like the ECG in nonpaced LBBBs. Sgarbossa criteria were first used in LBBB to diagnose acute myocardial infarction and then applied in pacemaker patients. These criteria were highly specific but had only a 20% sensitivity.[57] Modified Sgarbossa criteria performed better to diagnose acute coronary occlusion requiring reperfusion (**Table 45-5**).[58] A validation study showed 80% sensitivity and 98% specificity in LBBB patients.[58] Case reports show that modified Sgarbossa criteria have been successfully applied to patients with paced ventricular rhythms.[59,60] The presence of any one of the three criteria would fulfill modified Sgarbossa criteria and support the diagnosis of an acute coronary occlusion or "ST-segment elevation myocardial infarction (STEMI) equivalent" in a patient with an LBBB or paced ventricular rhythm.

Even less evidence is available for the ECG diagnosis of acute coronary occlusion in biventricular pacemaker patients. The baseline ECG morphology is variable because the QRS is a combination of RV and LV depolarization and lead locations are variable. Case reports show that biventricular pacemaker patients who have acute coronary occlusion may meet conventional STEMI criteria, Sgarbossa criteria, or modified Sgarbossa criteria.[61-63]

TABLE 45-5 Modified Sgarbossa Criteria to Predict Acute Coronary Occlusion[58]

1. ST elevation ≥ 1 mm in same direction (concordant) as QRS complex in any lead
2. ST depression ≥ 1 mm in any lead from V_1 to V_3
3. ST elevation ÷ S-wave amplitude exceeding 0.25 in any lead

ACLS CONSIDERATIONS

ACLS can be performed in CRMD patients as usual with a few additional considerations. Be prepared to apply external defibrillation as necessary. Tape a magnet in place to avoid unintentional ICD shocks during the resuscitation. Most ICDs are programmed to do no more than five successive shocks to preserve battery life.[28] Changes in pacing and defibrillation thresholds are seen with metabolic derangements of critical illness, a pneumothorax from cardiopulmonary resuscitation (CPR), and resuscitation medications (e.g., amiodarone and lidocaine) (**Table 45-2**). This may lead to oversensing or undersensing.[34]

Apply cardioversion and defibrillation pads at least 8 cm from the device, with the current path perpendicular to the plane of the pacing system. Placement of the pads directly anterior and posterior to the LV is recommended when possible. Do not delay emergent defibrillation to reposition the pads. ICD shocks are not dangerous to rescuers doing CPR or monitoring equipment. The device requires interrogation after cardioversion or defibrillation to assure return to appropriate settings.[28,34]

Central venous access using the femoral vein is preferred when a CRMD is in place because chronic thrombosis associated with leads may be present in the subclavian vein. Guidewire contact during placement may trigger a response from the device.[34]

FUTURE CRMD TECHNOLOGY

LEADLESS PACEMAKERS

Many of the complications of ICDs and pacemakers are related to transvenous leads and subcutaneous pockets. Leadless ICDs and pacemakers have been developed (**Figures 45-24 and 45-25**).[64-66] Leadless pacemakers are an emerging technology that includes all components of a pacemaker system in a device the size of a large pill placed directly into the RV apex and attached to the myocardium. Complication rates compare favorably to conventional pacemakers, but this technology is currently limited to single-chamber pacing.[67] Subcutaneous ICDs (i.e., S-ICD) are tunneled within the chest wall and have the comparable ability to detect and terminate ventricular tachyarrhythmias but lack backup pacing abilities and antitachycardia pacing.[68]

REMOTE MONITORING

Remote monitoring (RM) of CRMDs is already available from every major manufacturer. Patients may be alerted to a malfunction and present to the ED as this technology becomes more widely adopted. RM CRMDs have a built-in micro-antenna that continuously and wirelessly transmits information (e.g., arrhythmia information, battery status, heart rate ranges, lead impedance, and programming).

FIGURE 45-24. Examples of a single-component leadless pacemaker. The Micra (Medtronic, Minneapolis, MN) and the Nanostim (St. Jude Medical, St. Paul, MN) are next to each other for size comparison. (Used with permission from reference 30.)

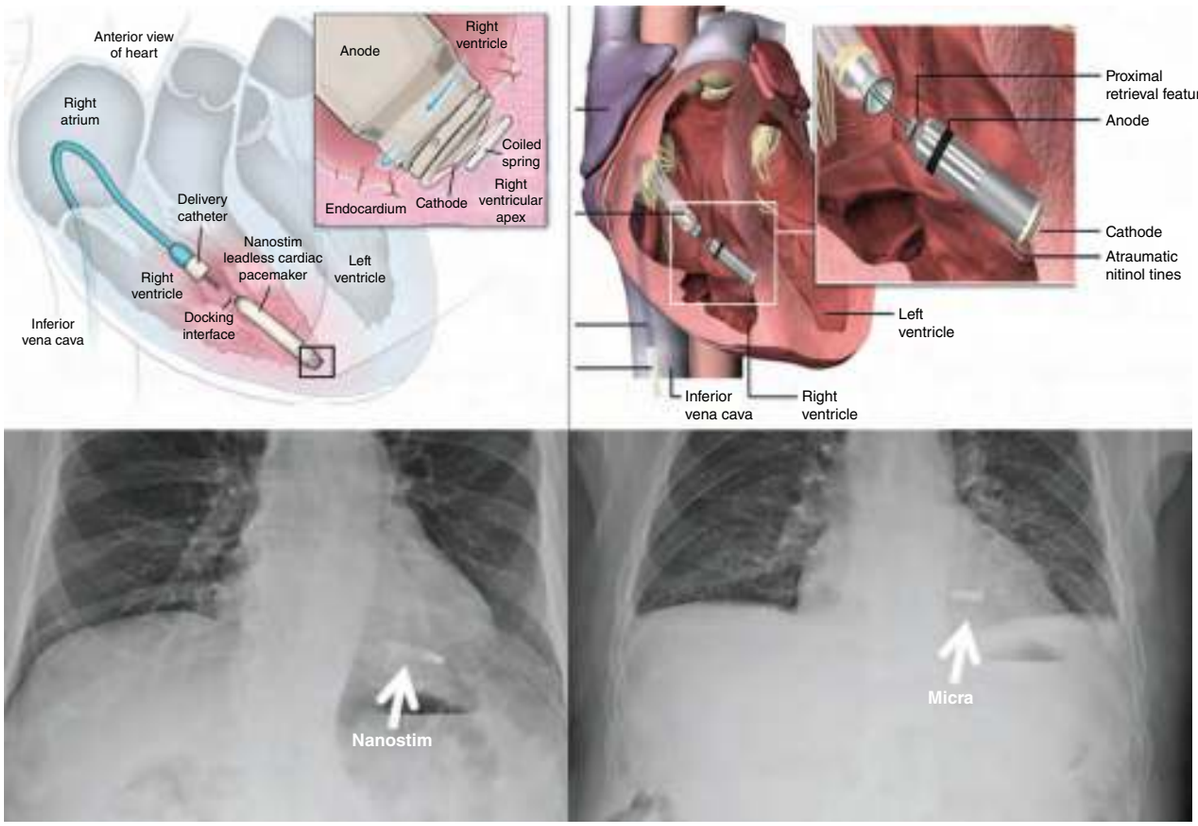

FIGURE 45-25. Leadless pacemaker fixation mechanism and radiographic appearance. Nanostim active fixation leadless pacemaker (St. Jude Medical, St. Paul, MN) is on the left and the Micra passive fixation transcatheter pacing system (Medtronic, Minneapolis, MN) is on the right. (Used with permission from reference 30.)

They allow the patient to use their cell phone or tablet to securely transmit the data. The pacemaker alerts the patient and Cardiologist of any detected change in clinical condition or malfunction. **RM can identify problems (e.g., impending battery or lead failure) before they become clinically apparent.** These transmissions are not currently in real-time but reviewed during office hours.[69]

Multiple large, prospective studies have shown RM to be cost efficient, reliable, and safe. Health care utilization decreased by 45% in one study, mainly as a result of reduced office visits. Most RM use is currently for ICD and CRT-D devices, which require follow-up every 3 to 6 months and have a shorter, less predictable battery life than pacemakers.[69] A prospective study of a quarter of a million consecutively implanted RM-enabled devices from 2008 to 2011 demonstrated that 53% of patients never used the RM feature.[70]

OTHER DEVELOPMENTS

Other developments have occurred in the area of pacemakers and defibrillators. Mesh antibacterial envelopes can be used to cover the implanted devices. They release antibiotics after implantation as they dissolve. Antibacterial envelopes may decrease the pocket infection rates. They also stabilize the device, preventing migration.

Solar-powered pacemakers have been developed.[71,72] They solve the problem of the current battery lasting 5 to 12 years. They use sunlight as an alternative power source. A small amount of light can penetrate the skin. It has been suggested that the solar cell be implanted in the neck. It only takes a few minutes of sunlight to charge the device for 24 hours.

MRI-compatible devices have been developed. A sensor automatically puts the device into the safe mode when the patient enters the MRI. This pauses the device only when the patient enters the MRI machine. It requires the Cardiologist to turn on the sensor.

SUMMARY

CRMDs have become more common and complex. The Emergency Physician needs a working knowledge of this technology and a systematic approach to evaluate these patients. Safety features have greatly reduced pacemaker dysrhythmias and malfunctions. Pseudo-malfunctions are more common than true malfunctions. Consider the integrity of the entire pacemaker circuit, including the myocardium, and nonpacemaker etiologies of a patient's presentation. Disruption to the myocardial-pacemaker interface is common. Immediately life-threatening conditions (e.g., hyperkalemia and ischemia) may affect pacemaker function. Device interrogation and Cardiologist consultation are critical steps but should not defer the workup of other emergent conditions.

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46 Automatic Implantable Cardioverter-Defibrillator Assessment

Carlos J. Roldan

INTRODUCTION

The introduction of implantable cardioverter-defibrillator (ICD) technology has revolutionized the fields of cardiology and electrophysiology. More than 100,000 such devices are implanted annually in the United States alone. ICDs allow life-threatening ventricular tachycardia and ventricular fibrillation to be safely controlled and benefit patients at risk for sudden cardiac death. Multiple studies have examined the prophylactic indication for ICD therapy in high-risk groups.[1-7] The ICD has become a more common therapeutic option for Brugada syndrome, prolonged QT syndrome, and cardiomyopathies.[8]

An ICD is often placed prophylactically to prevent sudden cardiac death from ventricular arrhythmias. An ICD follows an algorithm and applies a full-energy shock, a low-energy shock, or it can overdrive pace the heart. All ICDs have a pacemaker function that can be set. Some ICDs can transmit to remote monitors.

The Emergency Department is often the initial contact point for these patients. Emergency Physicians must be familiar with the problems that can be encountered by a patient with an ICD.[9-13] This chapter describes technical aspects, basic interrogation of the device, and a general approach to a patient who presents to the Emergency Department with an ICD.

TECHNICAL CONSIDERATIONS

The ICD has four main functions. It recognizes and records local atrial and ventricular electrogram signals. It then classifies the sensed signals to programmable heart rate zones. The ICD provides a shock to terminate ventricular tachycardia or ventricular fibrillation. It has a pacing capability for bradycardia and/or cardiac resynchronization therapy. Therapy to terminate the arrhythmia is initiated with a high-energy shock of up to 40 J when detection criteria are satisfied.

ICD technology has progressed exponentially since its introduction in the early 1980s.[14] The ICD system is comprised of a pulse generator, a battery, and a lead system. The lead system is required for sensing, pacing, and the delivery of therapy. The earliest systems required that the pulse generators be placed abdominally due to their large size (**Figure 46-1**). Defibrillation was delivered via two epicardial patches positioned anteriorly and posteriorly. A transvenous spring electrode in the superior vena cava was occasionally used with an epicardial patch. Sensing was achieved through separate epicardial screw-in electrodes. Initial lead placement required a sternotomy, lateral thoracotomy, or a subxiphoid approach, which made early implants quite cumbersome.[15]

The smaller size devices used now allow for superficial implantation of the pulse generator in the anterior chest wall (**Figure 46-2**). The current ICD systems are comprised of three main parts (**Figure 46-3**). The pulse generator is programmable and capable of analyzing and recording the patient's heart and rhythm. The ICD generator houses the batteries, high-voltage capacitors, and microprocessors necessary to process sensed intrinsic cardiac electrical activity.

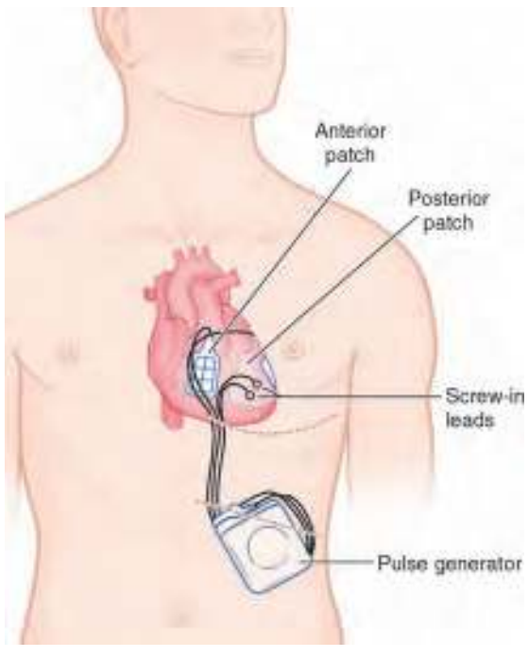


FIGURE 46-1. Abdominal placement of the ICD generator. Initial implants required a thoracotomy to position the epicardial patches needed for defibrillation and the screw-in sensing leads. The leads were tunneled abdominally to the ICD generator.

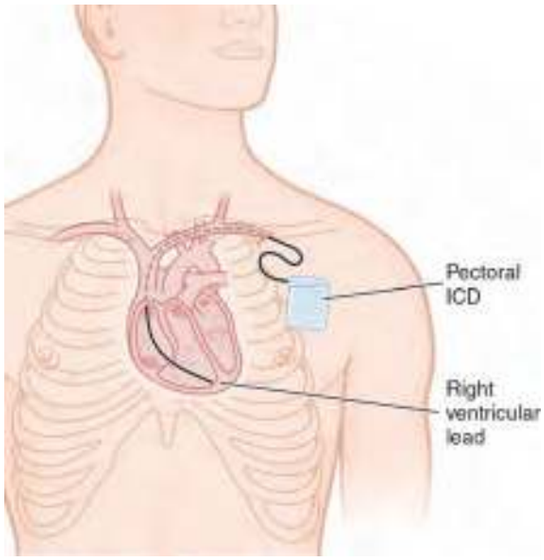


FIGURE 46-2. The current nonthoracotomy system. The development of smaller generators and biphasic waveforms for defibrillation allowed for transvenous positioning of the ICD leads and a pectorally located generator.

The generator is a minicomputer within a hermetically sealed titanium can (e.g., case) capable of generating shocks. Typical ICDs contain lithium silver vanadium oxide cells that store between 2 and 7 volts.[16] The high voltages necessary for defibrillation are generated with the aid of high-voltage capacitors that generate 700 to 800 volts of defibrillation energy in under 20 seconds. ICD leads can be inserted in different combinations such as a single wire in the right ventricle (e.g., single-chamber ICD), two leads (e.g., in the right atrium and right ventricle, or a dual-chamber ICD), or three leads (i.e., biventricular ICD with one in the right atrium, one in the right ventricle, and one on the outer wall of the left ventricle; **Figure 46-3**). The leads are required for sensing, pacing, and the delivery of therapy. The final component is the battery to power the ICD system.

ICD implantation has evolved quite rapidly due to advancements in lead technology, generator technology, and the development of biphasic defibrillation waveforms, which lowered the energy requirements necessary for successful defibrillation.[17] The creation of a lead combining pacing and sensing capabilities with a high-voltage electrode coil allowed for nonthoracotomy system implants with reduced surgical morbidity and mortality.[18] The leads are positioned transvenously via the subclavian vein and fixed to the inside of the right ventricle. The leads were once tunneled subcutaneously to the abdomen due to the large generator size of early defibrillators. Technology has advanced to the development of more compact generators. The smallest commercially available devices today are under 40 cm^3 and weigh well under 100 gm. Smaller generators allow for subcutaneous pectoral implantation and simplification of the implantation process (**Figure 46-2**).[16]

Current ICDs allow extensive programmability for tiered antitachycardia pacing (ATP), tiered high-voltage therapies, single- or dual-chamber bradycardia pacing, supraventricular tachycardia discrimination algorithms, and detailed diagnostics of tachycardic and bradycardic episodes. Implantable loop recorders and home monitoring functions extend the technical capabilities for automatic detection of arrhythmias that may not be symptomatic. It allows for fully automatic and wireless data transmission, including episode counters.[15,17] This allows alterations in device programming or medication dose modifications in the outpatient setting and avoids hospitalization.[19,20]

ATP is also known as overdrive pacing. It may be enabled to manage ventricular tachycardia (**Figure 46-4**).[21-23] ATP commonly consists of a burst of pacing (i.e., 6 to 10 beats) at a rate faster than the ventricular tachycardia rate. ATP may be felt by the patient. It is painless and often terminates the ventricular tachycardia before the

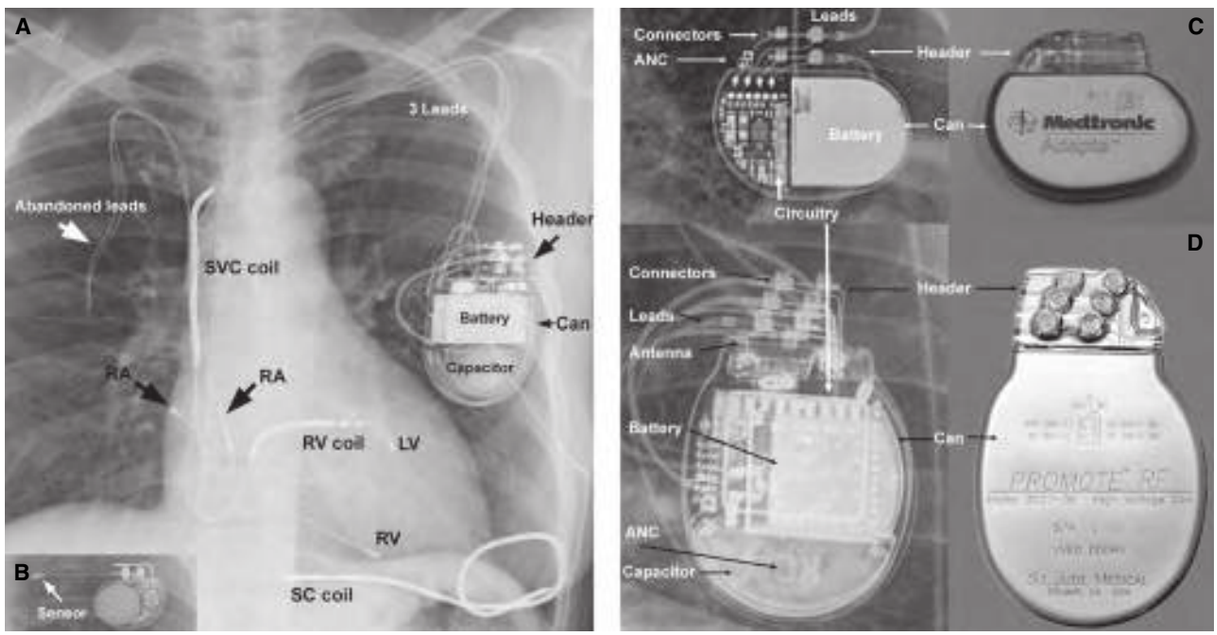


FIGURE 46-3. Assessing the ICD. ***A.*** Chest radiograph of a patient with a biventricular ICD. Note the right atrium (RA), right ventricle (RV), and left ventricle (LV) leads connected to the header. The RV lead has a combination of RV pace/sense lead, RV coil, and superior vena cava (SVC) coil. The subcutaneous (SC) coil is connected to the SVC port. Another set of RA and RV abandoned leads can be seen from a previous device implantation. ***B.*** The Transoma Medical implantable loop recorder. Note the flexible sensor (antenna) with no transvenous leads. ***C.*** The Medtronic Adapta pacemaker. ***D.*** The St. Jude Medical Promote ICD. (Photos used with permission from reference 39.)

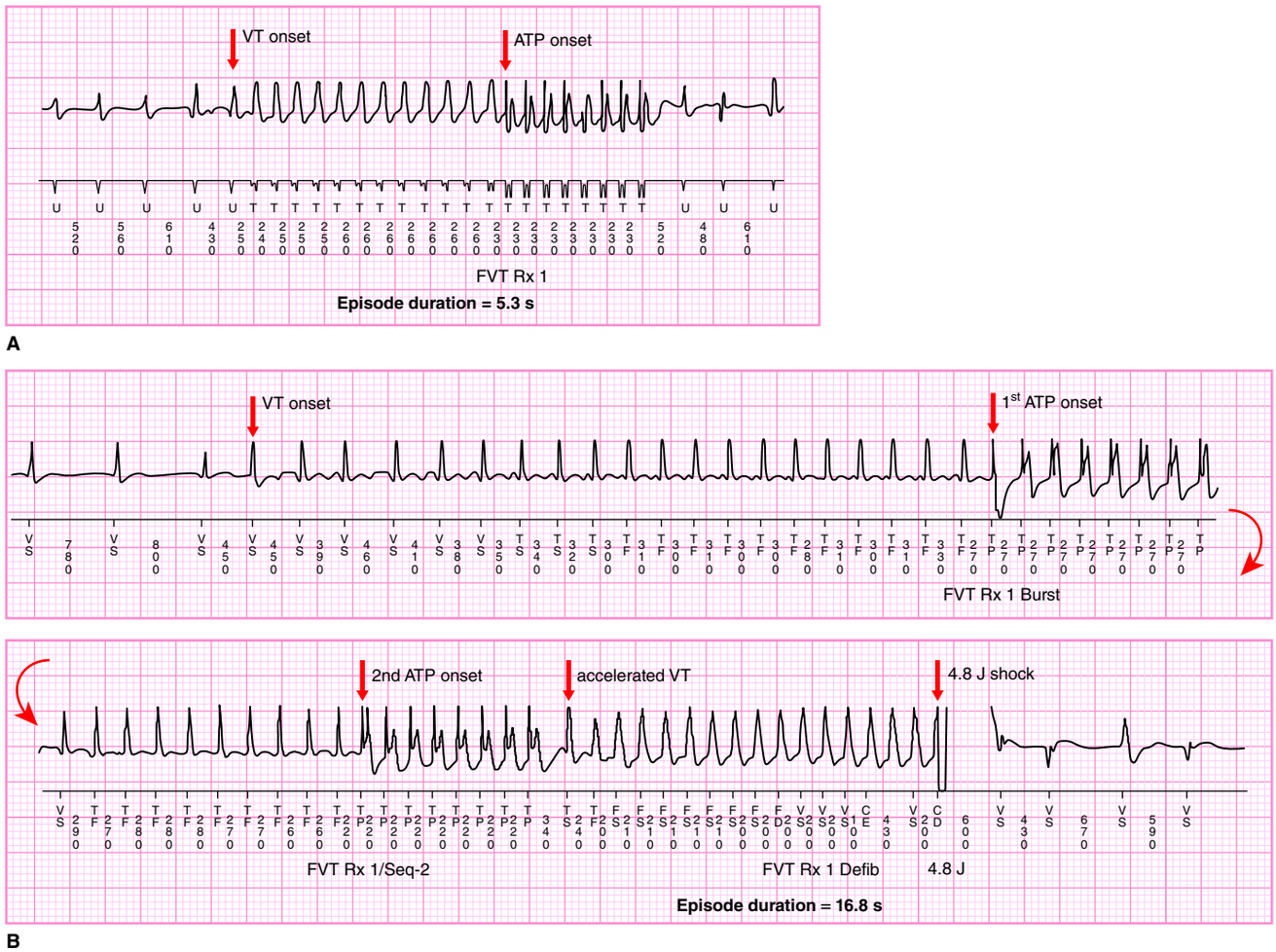


FIGURE 46-4. ATP electrocardiogram tracing. ***A.*** Successful termination of ventricular tachycardia (VT). ***B.*** Failed ATP for ventricular fibrillation followed by shock termination. (Photos used with permission from reference 23.)

patient becomes symptomatic. ATP is the initial preferred treatment, even for fast ventricular tachycardia, with shocks programmed as a backup if ATP fails.[24] Current ICDs have an antibradycardia function that works like a pacemaker. The ICD will provide a pacing function with significantly less forceful shocks if the heart rate falls below the programmed rate. The ATP results in more patient comfort and preserves the battery life of the ICD compared to a shock.

Perform exercise testing with the realization that increasing the heart rate above the programmed ventricular tachycardia and ventricular fibrillation detection rate is likely to elicit therapy (i.e., a shock) from the ICD. **Patients are instructed to consult their Electrophysiologist before any exercise or stress testing to determine the programmed ICD arrhythmia detection rate. Stop the exercise or stress test to avoid the delivery of an inappropriate shock if the programmed rate is approached.**

ROUTINE ICD FOLLOW-UP

A systematic follow-up procedure to assess the integrity of the ICD system is recommended every 1 to 6 months depending on the device, battery status, and arrhythmia frequency (**Table 46-1**). Interrogate the device with the appropriate system analyzer to assure battery voltage, lead and electrode integrity, and capacitor charge times. System integrity test results can reveal malfunctions that may compromise the device's ability to treat an arrhythmia effectively.[25,26]

Each recorded arrhythmic episode should be analyzed to determine the appropriateness and effectiveness of therapies once the system's integrity has been verified. This is achieved by examining the stored diagnostic information contained within the ICD. This may include arrhythmic interval values, charge time, classification markers for

TABLE 46-1 The ICD Follow-Up Checklist

- Detailed history of shocks, palpitations, light-headedness, syncope, chest pain, and dyspnea, or symptoms of congestive heart failure
- Note any changes in medications (i.e., antiarrhythmic therapy)
- Physical examination (examine incision site for any indication of infection)
- Interrogate device with appropriate system analyzer
- Check lead parameters including pacing thresholds and lead impedances (sensing is established by examining intracardiac R waves)
- Determine capacitor reformation times
- Check battery voltage (longevity is typically 5 to 10 years, depending on use)
- Measure impedance of the high-voltage coil (this may require the administration of a subthreshold shock in older ICDs)
- Analyze arrhythmia counters (examine corresponding intracardiac electrograms to determine appropriateness of therapy)
- Make the necessary program changes (e.g., adjusting tachycardia zones, changing detection cutoff rates, turning on special features to aid tachycardia discrimination [stability, sudden-onset criteria, electrogram width], and adjusting the sensitivities)
- Confirm changes and reinterrogate device

each interval, device classifications of therapy success, energy, episode plots, impedance values for shocks, intracardiac electrograms, and textual episode descriptions. This diagnostic information can be extremely valuable to determine whether programming changes should be made.[27]

BATTERY AND CAPACITORS

Battery longevity is significantly different among manufacturers. Battery voltages and capacitor charge times must be noted. Capacitors with smaller or larger capacities and changes in the initial shock polarity have not significantly improved defibrillation efficacy.[28] A variation in the number and position of electrodes can significantly influence the defibrillation threshold.[28] Several factors are associated with service life of ICD devices. Differences in longevity emerged among systems and manufacturers.

Generator replacement is usually recommended when voltages fall below the elective replacement indicator (ERI), which is approximately 2.6 volts. The battery has reached its end of life (EOL) when the battery voltage falls below 2.2 volts. This signifies a more urgent need for battery replacement. The need to replace a generator is also dependent on capacitor charge times. The occurrence of two consecutive charge times greater than 16 seconds is considered prolonged and may warrant urgent replacement regardless of the battery voltage. Automatic capacitor reformation is usually set to every 6 months to replenish their charge. This function can be programmed at preset time intervals. More frequent reformation is required and performed as the device reaches the battery EOL and based on manufacturers' recommendations.

LEAD INTEGRITY

There was concern in the past about the long-term reliability of chronically implanted leads because an insulation defect or conductor disruption could lead to ICD lead failure. Lead failure can affect the high-voltage lead or the pace-sense circuit of the lead. Most ICDs now provide lead-impedance monitoring for early detection of lead failure. An abnormal impedance indicates conductor fracture or insulation defect. An audible signal or vibration notifies the patient, who should then immediately contact their Cardiologist or present to the Emergency Department. Retrospective data from single centers suggest a potential clinical benefit of patient notification for ICD lead failure detection.[29]

Various parameters are checked to ensure lead integrity (e.g., pacing thresholds, lead impedance, and the size of intracardiac R waves). A pacing threshold of ≤ 1.0 volt at a pulse width of 0.5 milliseconds is desired at the time of implantation. An acute rise in threshold may be seen initially and generally falls with time. Chronic thresholds range between 0.5 and 2.0 volts. Epicardial leads generally exhibit higher thresholds. Any change in thresholds must be compared with prior trends to assess its significance. Pacing thresholds may also change with the administration of antiarrhythmic drug therapy. Potential complications of ICD lead failure include oversensing of electrical noise, undersensing of ventricular tachyarrhythmias, inappropriate therapy, and lethal antiarrhythmic therapy.[30]

Modern ICDs perform serial impedance measurements automatically. It is unlikely that such discrete measurements will reveal abnormal impedance if lead failure causes sporadic dysfunction. Multiple impedance measurements per day may be necessary to enhance the sensitivity of the patient alert.[31] Lead impedances will vary based on the type of lead. The high-voltage impedance measurement differs between devices.[32] Normal impedances range from 300 to 1200 ohms. A sudden change in impedance may signal a problem with lead integrity. A high impedance reading suggests the possibility of lead fracture (**Figure 46-5**).[33] A low impedance reading indicates a problem with the insulation. The impedance of the high-voltage coil must also be evaluated.

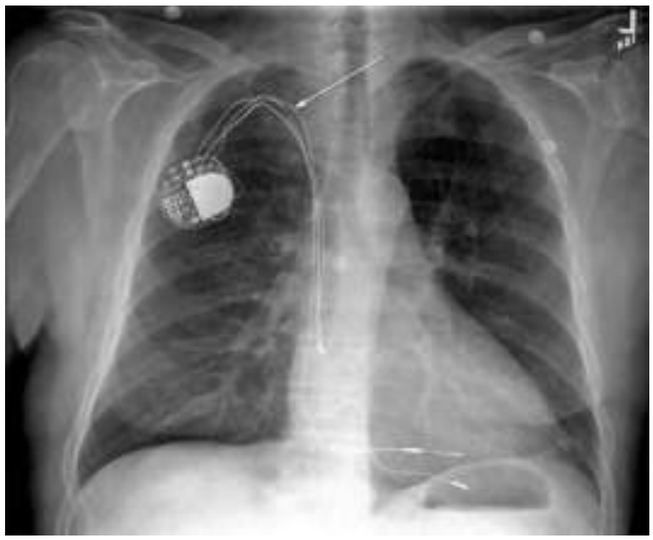


FIGURE 46-5. A lead fracture. (Photo used with permission from reference 33.)

Epicardial and active can systems demonstrate lower impedance values compared to endocardial systems due to their larger surface area. Normal values generally range between 20 and 80 ohms. High impedance values usually indicate a fracture in the defibrillation system. Any stark changes in impedance levels may suggest a patch problem (e.g., crinkling, migration, or seroma formation). Any impedance change in an endocardial system may indicate lead dislodgement.

R-wave amplitude is a direct measure of intracardiac electrogram activity and determines the device's ability to sense. An R wave of ≤ 5 millivolts is desired at implantation to ensure adequate detection of ventricular fibrillation. The most common explanations for a decrease in R-wave amplitude after an implant are lead dislodgment or local factors (e.g., edema or fibrosis). This change may be associated with an increase in lead impedance and pacing thresholds.

ANALYZING APPROPRIATENESS OF THERAPY

Current ICDs have simultaneous marker channels with real-time intracardiac electrograms (**Figure 46-6**). It is important to document intracardiac activity during sinus rhythm. These electrograms can be used as a basis of comparison with tachycardia events. Examine the signals for evidence of noise, which may be an indicator of sensing problems, a connector issue, or a faulty adaptor. Electrograms must be examined during various maneuvers (e.g., arm maneuvers, bending, and deep breathing).[11] Examine intracardiac electrograms for T-wave oversensing or detection of pacemaker spikes as ventricular signals.[11,34] Rule out oversensing by the atrial lead in dual-chamber devices.[11] Wide variations in electrogram size may suggest that the lead is not stable or well fixed.

THE ICD IN THE EMERGENCY DEPARTMENT

A patient with an ICD may present to the Emergency Department for various reasons. Manufacturers offer 24-hour technical support. They maintain a registry of devices implanted and evaluations performed. **A staff of field engineers is often available for help with the assessment, interrogation, and management of ICD function.** The ICD may be interrogated by the Emergency Physician.[35,36]

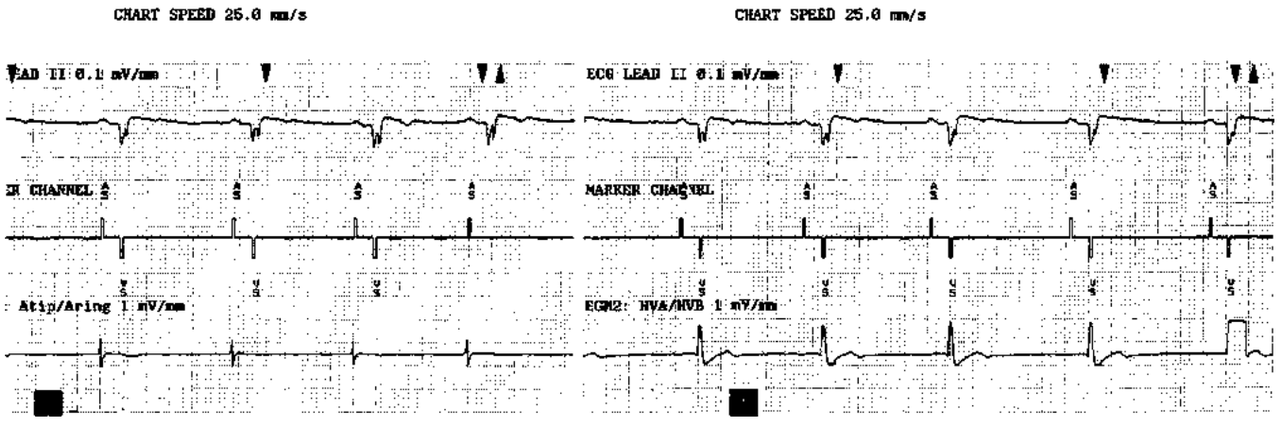


FIGURE 46-6. The intracardiac electrogram as recorded by the ICD. Printouts were obtained from a Medtronic Gem II DR dual-chamber ICD. The first line shows lead II of an ECG rhythm strip. The marker channel directly under the rhythm strip indicates behavior (i.e., sensing and pacing) in each chamber. The third line represents the intracardiac electrogram from the atrium (*left*) and ventricle (*right*). Measured in millivolts per millimeter, larger signals imply better sensing.

It can result in a faster ICD interrogation versus waiting for a field engineer to come to the Emergency Department. **The U.S. Food and Drug Administration (FDA) is responsible for assessing the premarket safety, postmarket safety, and effectiveness of medical devices marketed in the United States. They require manufacturers to submit annual reports detailing the number of device implants and malfunctions that have occurred.**[37]

Individuals with recently implanted devices may be quite anxious and may seek medical attention after a single ICD discharge. Patients may present after multiple ICD discharges or in full cardiac arrest. The warmth, local redness, and pain associated with a potential infection of the ICD pocket may prompt the individual to seek medical attention (**Figure 46-7**).

Patients with ICDs must be placed on continuous telemetry. Perform a detailed and complete history and physical examination. Question the patient regarding the number of shocks received, symptomatic palpitations, presyncope, symptoms of congestive heart failure, or symptoms consistent with angina. Examine the pocket site for evidence of local infection, including warmth, tenderness, and discharge. Upper extremity or neck swelling ipsilateral to the inserted endocardial lead suggests the possibility of a subclavian vein or superior vena cava thrombosis.[38] Bilateral head edema and neck vein swelling suggest the possibility of a superior vena cava thrombosis or superior vena cava syndrome. Document any changes in medications or whether antiarrhythmic therapy has recently commenced. Obtain routine blood work (e.g., complete blood count, serum electrolytes, magnesium level, renal function indices, cardiac enzymes, troponin, and quantitative levels of measurable medications).

FIGURE 46-7. A pocket infection with scabbing. (Photo used with permission from reference 61.)

Identify the ICD model. Patients are generally given identification cards that list the manufacturer, lead system, generator model, and a 24-hour emergency contact number. This information is often not available from the patient in the Emergency Department. An overpenetrated chest radiograph showing the generator in an emergency can demonstrate the radiopaque identifier of the manufacturer (**Figure 46-3**).

The chest radiograph may be of diagnostic importance. It is not only important for device identification but can also give useful information about lead integrity (**Figures 46-3 and 46-5**).[39] A chest radiograph is routinely performed to assure lead positioning and slack in the lead and to rule out other thoracic pathology.[40] The chest radiograph is an excellent tool in patients with the older epicardial patch system to demonstrate patch crinkling, fracture, or migration. The leads can be assessed for fractures or discontinuities with endocardial systems (**Figures 46-3 and 46-5**). Fractures can occur anywhere along the lead. They are most commonly seen near the junction of the first rib and the clavicle, a condition referred to as "subclavian crush."

Make attempts to obtain a 12-lead electrocardiogram (ECG) before any interventions are made to terminate the arrhythmia if the patient presents with a ventricular arrhythmia and is hemodynamically stable. This may be difficult due to concomitant discharges from the device, an anxious staff, and an anxious patient. Antiarrhythmic medications (e.g., amiodarone, beta-blockers, and procainamide) may have to be considered in the event of recurrent ventricular tachycardia.

EMERGENCY DEACTIVATION (MAGNET BEHAVIOR)

The ability of an ICD to identify and treat tachyarrhythmias can be temporarily disabled with the use of a magnet (Figure 46-8).[41] This situation may arise in the setting of multiple ICD discharges where the shocks are not tolerated or prior to a surgical procedure where electrocautery is necessary. Emergency deactivation with a magnet can result in serious complications (e.g., causing a battery indicator to switch to "end of life" or the loss of some

FIGURE 46-8. An example of a magnet to disable the ICD.

antitachycardia therapies).[42] **The application of a donut-shaped magnet (Figure 46-8) overlying the ICD pulse generator forms a magnetic field that trips a reed switch in the ICD generator circuit.**[11] This results in a suspension of tachycardia detection and therapy delivery. A single magnet will suffice in most cases. Two or more magnets may be required to achieve deactivation in obese patients or in the presence of pockets with significant edema.[43] **The patient must be fully monitored, a cardioverter-defibrillator unit must be readily available, and Advanced Cardiac Life Support (ACLS) medications must be readily available if an ICD is to be deactivated.**

The magnet response of an ICD varies subtly from manufacturer to manufacturer. The application of a magnet to Medtronic devices temporarily disables tachycardia detection and therapy with no effect on bradycardia pacing. Removal of the magnet will resume arrhythmia detection. Newer Medtronic ICDs will elicit a continuous beep lasting for 15 seconds if a magnet is placed directly over the ICD. A magnet applied over Guidant ICDs also inhibits tachycardia therapy with no effect on bradycardic pacing. These devices will generate beeping tones, which change to a continuous tone. The constant tone indicates that the device is off and will not deliver tachycardia therapy. The device can be turned back on by reapplying the magnet over the ICD for 30 seconds. Tones will now change from continuous to beeping synchronous with R waves, signifying that the device is on again. Newer generation Guidant ICDs have a built-in electrocautery feature that can be activated by use of the Guidant programmer. This will suspend tachyarrhythmia therapies and pace in the DOO mode. Regular functioning of the ICD is restored by turning this feature off.

ICD DISCHARGES

Patients who experience a shock but feel unwell after the event or who receive more than one symptomatic ICD therapy within a short time (i.e., minutes to hours) require emergent evaluation.[43] **Most ICDs have a limit to the number of shocks they can generate before they stop producing shocks. This number is usually five to seven shocks.** The ICD can reset and start shocking the patient again to the set maximum number of shocks if the cardiac rhythm reverts to normal sinus rhythm. Multiple ICD discharges or shocks in a short time can cause severe battery depletion. **Possible etiologies that warrant medical attention include ongoing arrhythmias not adequately treated by the device, myocardial infarction, electrolytes imbalances, and ICD malfunction.**

Emergency Department patients presenting with multiple ICD discharges require immediate attention. Multiple discharges are usually not well tolerated and can be emotionally devastating to the patient from a psychological perspective.[38] Myocardial injury and transient reduction in left ventricular function can occur from multiple shocks. This has been associated with a poorer long-term prognosis. Multiple discharges can lead to premature depletion of the battery life.

ESTABLISHING THE ETIOLOGY

It is important to establish the etiology of the shocks to administer proper and prompt management (Table 46-2). Determine whether the shocks are appropriate for ventricular tachycardia or fibrillation, inappropriate therapy, or phantom shocks.[44] More than one-third of patients with a history of ventricular tachycardia or ventricular fibrillation receive a shock within 2 years of the ICD implantation.[45] Recurrent ventricular tachyarrhythmia is a common cause of repeated ICD firing. Ineffective termination of a tachyarrhythmia can be the result of an increase in defibrillation thresholds secondary to concomitant antiarrhythmic drug therapy and lead migration or lead dislodgement. Inefficient termination can occur if inappropriately low amounts of energy are programmed for the initially administered shock. Shocks may be the result of inappropriate detection of supraventricular tachycardias (SVTs), most frequently sinus tachycardia and atrial fibrillation.[46] The administration of a low-energy shock may convert a benign SVT into an unstable ventricular arrhythmia, resulting in an ICD proarrhythmia. The introduction of an atrial lead in dual-chamber devices has aided in the discrimination process between SVTs and ventricular tachyarrhythmias. Rapid SVTs are particularly a problem in children and athletic individuals in whom exercise or reductions in medications that slow heart rate (e.g., beta-blockers) are commonly encountered causes of inadequate shocks from sinus or supraventricular tachycardia. Inappropriate ICD firing can occur because of the erroneous detection of noise or interference that can be the result of insulation breakdown or a loose set screw (**Figure 46-9**). Oversensing of T waves, pacing artifacts, R waves, and electromagnetic interference may lead to inappropriate detection and discharge. Patients who have received painful shocks occasionally suffer from phantom shocks (i.e., the perception of a shock in the absence of any arrhythmia or therapy from the ICD).[44] Consider random component failure if all other causes have been ruled out.

TABLE 46-2 Causes of Frequent ICD Discharges

Appropriate
Nonsustained ventricular tachyarrhythmias
Sustained ventricular tachyarrhythmias
One shock needed to terminate each episode of sustained ventricular tachyarrhythmia
Recurring episodes, each one terminated by a shock
Inappropriate
Increase in defibrillation thresholds (antiarrhythmic drug therapy)
Oversensing of signals
Double and triple counting of pacing artifacts
Electromagnetic interference
Environmental electrical noise
P-wave oversensing
Sensing lead failure (migration or dislodgement)
T-wave oversensing
Phantom (absence of both arrhythmia and ICD discharge)
Random component failure
Supraventricular tachyarrhythmia that satisfies detection criteria
Atrial fibrillation
Paroxysmal supraventricular tachycardia
Sinus tachycardia

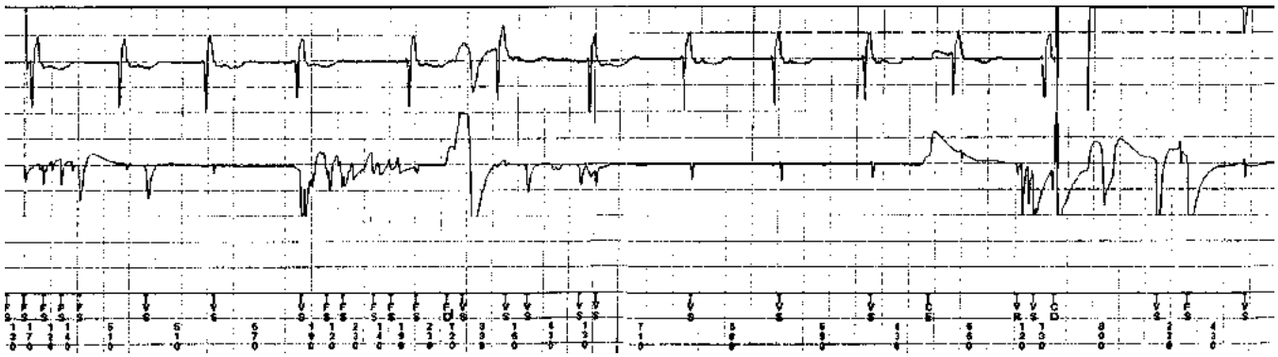


FIGURE 46-9. Inappropriate ICD discharge. Intracardiac electrogram of a patient with a Ventak Mini III presenting with repetitive ICD discharges. Examination of the intracardiac electrograms (line 2) demonstrates noise sensed as ventricular fibrillation (FS) resulting in an inappropriate shock (CD). Noise was traced to an insulation break in the ICD lead. Lead replacement corrected the problem.

APPROACH TO THE PATIENT WITH MULTIPLE ICD DISCHARGES

These patients must be under constant ECG monitoring. Apply defibrillator pads in anticipation of the development of an unstable cardiac arrhythmia. This may be the only means of establishing a shock-rhythm correlation in devices with limited stored diagnostic capabilities. Sedation is reasonable in extremely anxious patients. Obtain an Electrophysiology consultation for assistance in interrogating the ICD. The device should be interrogated and stored electrograms obtained for analysis.[47] It is often useful to inquire about the pattern of ICD discharge. Consecutive shocks occurring within a few seconds suggest an inappropriate discharge for SVT, oversensing, or device failure. Isolated shocks occurring every few minutes may be indicative of recurrent ventricular tachycardia. Progressive dyspnea on exertion, shortness of breath, orthopnea, or paroxysmal nocturnal dyspnea suggests new-onset or worsening heart failure, which can precipitate ventricular arrhythmias. Potential reversible causes (e.g., electrolyte abnormalities) need to be identified and promptly corrected.

Careful examination of the 12-lead ECG is crucial. ST-segment changes may imply an acute coronary syndrome and determine the need for primary intervention or thrombolytic therapy. An ECG obtained during an actual shock may establish whether the culprit arrhythmia is a supraventricular or ventricular tachycardia.

Emergently deactivate the ICD if the discharges are inappropriate. Causes of inappropriate shocks include SVT and misinterpretation of complexes (e.g., premature ventricular contractions and T waves) as dysrhythmias. Manage supraventricular tachyarrhythmias with intravenous drug therapy (e.g., adenosine, diltiazem, or verapamil). Make attempts to control the patient's ventricular response in the situation where discharges are secondary to rapid atrial fibrillation with atrioventricular (AV) nodal blocking agents (e.g., beta-blockers, digoxin, diltiazem, verapamil). Attempt chemical cardioversion or electrical cardioversion in the event of hemodynamic instability. Shocks secondary to prolonged episodes of nonsustained ventricular tachycardia can be prevented by adjusting initial detection parameters coupled with the addition of antiarrhythmic drug therapy.

Patients with an ICD can develop "electrical storm." **Patients in electrical storm require immediate attention.** This condition involves recurrent, hemodynamically unstable ventricular tachycardia or fibrillation occurring two or more times in a 24-hour period.[48] Potential triggers can be found in approximately 66% of patients and include new or worsened heart failure, changes in antiarrhythmic medication, psychological stress, and hypokalemia. Electrical storm consists of monomorphic ventricular tachycardia in most patients and indicates the presence of a reentry mechanism. Ventricular fibrillation is rare and may be indicative of acute ischemia. The key intervention in electrical storm is reduction of the elevated sympathetic tone by intravenous beta-blockers, benzodiazepines, and amiodarone.[49]

Consult an Electrophysiologist promptly and stabilize the patient prior to transfer to the Intensive Care Unit (ICU). Apply defibrillator pads in anticipation of the development of unstable cardiac arrhythmias. **Potential reversible causes (e.g., electrolyte abnormalities) need to be identified and promptly corrected.** The treatment of choice is magnesium and/or temporary cardiac pacing if torsades de pointes is diagnosed. Thrombolysis or urgent catheterization with intervention may be needed in the setting of an acute myocardial infarction. Deactivate the ICD if analysis of stored electrograms demonstrates ineffective discharges. Attempts to terminate the arrhythmia in the hemodynamically stable patient via ATP is a useful option. Intravenous antiarrhythmic drugs are a necessary adjunct in these situations. The administration of amiodarone in combination with beta-blocker therapy has been shown to be successful in the management of electrical storm.[50] A combination of antiarrhythmic drugs will often be required.

ICDs AND CARDIAC RESUSCITATION

Manage patients with an ongoing arrhythmia by ACLS guidelines regardless of the presence of an ICD. Although in the most instances ICD function will be found to be appropriate, this cannot be assumed.[27] Follow ACLS guidelines and consider the device inactive.[51,52] Deactivate the device if the clinical situation permits and an ICD programmer is readily available. This will prevent the reinduction of ventricular fibrillation or tachycardia due to concomitant ICD discharges that may occur during cardiopulmonary resuscitation (CPR)**. There is often some hesitation to initiate resuscitative measures in patients with an ICD for fear of getting shocked. This fear is understandable but unwarranted. A mild electric shock might be perceived. These discharges do not pose a risk to persons administering CPR nor do they damage external monitoring devices.**

External defibrillation is permissible. The paddles or pads should be positioned away from the device. Individuals with epicardial patches may require higher energies for defibrillation. Current can be shunted from the myocardium through the patches. The insulated portion of the patch serves as a shield from the administered shock. An anteroposterior paddle configuration has been suggested in patients with epicardial patches for changing the defibrillation vector.[53]

The ICD may be reset after external defibrillation is delivered, especially if the paddles or pads are in proximity to the generator. It is important that devices be reinterrogated after the successful completion of a resuscitation to ensure that programmed parameters have not been altered.

INFECTION OF AN ICD

Technologic advances have made a gradual reduction in the size of the pulse generator possible. This small size permits superficial implantation of the pulse generator in the anterior chest wall.[54] The ICD implantation technique is similar to pacemaker implantation. ICD infections can involve the generator pocket, the leads, or both. Infection is more likely after a recent generator replacement.[55,56] **An infected ICD system represents a serious medical situation that should be dealt with urgently (Figure 46-7).** The diagnosis of an ICD infection is based on finding infection in the pocket site (e.g., drainage, pain, redness, swelling, or ulceration).

In the past, the incidence of infection ranged from 2% to 11% in systems that were implanted via thoracotomy or sternotomy.[57] The infection rates for nonthoracotomy implants approach those of pacemakers and range from 0.8% to 1.5%.[55,57-60] Infections generally present clinically within 6 months of the implant, but more typically within the first 3 months. Suspect an infection when local and systemic signs and symptoms of inflammation or a skin erosion are apparent (**Figure 46-7**).[61] Prompt referral to a specialist is warranted. Removal of the device and leads is required in almost all cases. Endocarditis prophylaxis with antibiotics is not generally warranted.

Systemic symptoms are seen in up to 50% of patients, especially in those with infections caused by *Staphylococcus aureus*.[62] The pocket and/or incision site is often visibly erythematous, warm, and tender. A fever may be present. Blood work often reveals a leukocytosis. Frank suppuration or device erosion may be seen. Pericarditis may be evident if epicardial patches are infected. Blood cultures may aid in documenting the culprit organism. Infections occurring late are generally indolent and rarely present with fever or leukocytosis, and blood cultures are generally negative.

The most common microorganisms in 50% of infections include *S. aureus* and coagulase-negative staphylococci.[56] Other common organisms include *Escherichia coli, Pseudomonas, Serratia, Corynebacterium, Propionibacterium acnes, Candida* species, streptococci, and atypical mycobacteria.[63] Infection is generally the result of skin contamination during implantation of the ICD. It can also occur due to hematogenous seeding from distant intravenous sites, indwelling catheters, or concomitant infections (e.g., respiratory or urinary tract). A hematoma in the pocket increases the infection risk.[64,65]

The TYRX Absorbable Antibiotic Envelope (Medtronics, Minneapolis, MN) was approved for use by the FDA in 2014. It is an envelope that covers the ICD before it is implanted. It releases rifampin and minocycline to prevent infection at the implantation site. The envelope starts to dissolve in 1 month and is dissolved between 2 and 3 months of implantation. It may be impossible to tell an infection from an allergic reaction to the contents of the envelope.

APPROACH TO AN INFECTED ICD

The goals of therapy include identifying the culprit organisms, establishing the extent of the infection, and containment of the infection.[56] **Consider endocarditis in the differential of all patients with an ICD and a fever.** Obtain routine laboratory tests, including a complete blood count and differential. Draw blood for cultures. It must be kept in mind that blood cultures are often negative. Wound cultures and Gram stains may be helpful in differentiating an infection from a pocket hematoma, sterile subcutaneous fluid accumulation, or an inflammatory reaction to pacemaker components. **Attempts to aspirate the pocket should be performed only in consultation with an Electrophysiologist and/or Surgeon. A sterile pocket or hematoma can often become infected after an aspiration.**

There is no single diagnostic modality that can determine the extent of the infection. ICD infections should never be assumed to be localized, as organisms can migrate from the leads into the heart. A chest radiograph may reveal patch deformities or wrinkling, suggesting distal migration of the infection. A computed tomography (CT) scan can detect localized fluid accumulation and patch wrinkling. **Transesophageal echocardiography (TEE) has been the method of choice to confirm the presence of vegetations on the leads or coil.** Intracardiac echocardiography (ICE) has shown accuracy as a diagnostic tool.

A few reports suggest that infection of a defibrillator system may be controlled. The treatment of choice continues to be removing the entire system followed by the administration of parenteral antibiotic agents.[66] Some localized infections restricted to the ICD generator have been managed with the removal of the generator, debridement, and systemic antibiotic therapy. Vancomycin is frequently used as an empiric agent when cultures are still pending given its good coverage against coagulase-negative staphylococci and methicillin-resistant *S. aureus* (MRSA). Empiric gram-negative and fungal coverage may be necessary in the immunocompromised patient.

ELECTROMAGNETIC INTERFERENCE AND ICDs

The ability of ICDs to function is dependent on their ability to sense intrinsic cardiac electrical activity. Hermetic shielding, filtering, interference rejection circuits, and bipolar sensing have safeguarded ICDs against the effects of common electromagnetic sources. Exposure to EMI may still result in oversensing, asynchronous pacing, ventricular inhibition, and spurious ICD discharges. EMI may lead to loss of output, increased pacing thresholds, and decreased R-wave amplitude. Common sources of EMI include cellular phones, electronic article surveillance (i.e., antitheft) devices, and metal detectors. Issues only arise with prolonged exposure. Patients are advised to use the opposite ear when using a cell phone and not stay near antitheft devices or metal detectors long. Occupational sources of EMI include high-voltage power lines, electrical transformers, arc welding, and electric motors. EMI can be encountered through medical equipment and procedures (e.g., magnetic resonance imaging, electrocautery, spinal cord stimulators, transcutaneous electric nerve stimulator units, radiofrequency catheter ablation, therapeutic diathermy, and lithotripsy).[67,68] It appears that some spinal cord stimulators may not cause EMI.[69]

Instruct patients with ICDs and pacemakers to avoid environments with large magnetic fields. Extremely low-frequency electromagnetic fields in daily life do not interfere with ICD sensing.[70,71] Strong electromagnetic fields of 50 Hz or greater in some occupational environments can cause inappropriate sensing and may lead to false detection of arrhythmias and a shock to the patient. The use of cellular phones is permissible. The FDA recommends avoiding direct contact of the phone with the ICD. Use the contralateral ear while using the phone. Inappropriate shocks have been documented through electronic article surveillance systems. Studies have deemed it safe for patients to walk through these systems if they avoid lingering around these devices. New concerns have been raised regarding household appliances. Patients can be reassured that EMI is unlikely to affect their ICDs if induction ovens are used in their kitchens.[72]

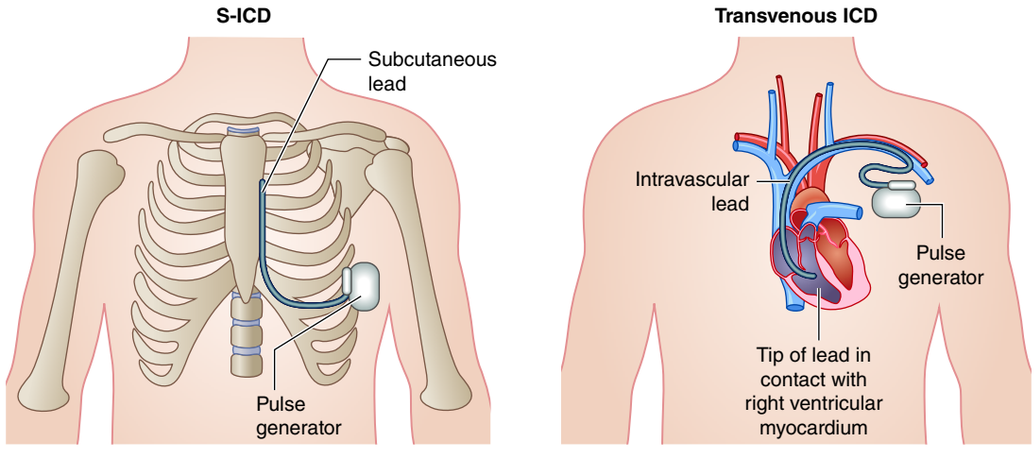


FIGURE 46-10. Comparison of the subcutaneous ICD with the transvenous ICD. (Photo used with permission from reference 74.)

The strong magnetic fields of magnetic resonance imaging (MRI) machines can interfere with ICD functioning. They can induce electrical current flow in the ICD lead that can initiate an arrhythmia or be sensed as an arrhythmia and precipitate spurious therapies. Patients with an ICD should not be placed in an MRI field without special cardiac monitoring. Newer technology has led to development of some MRI-compatible ICDs (e.g., Biotronic, Boston Scientific, and Medtronic). There is no such problem with fluoroscopy, CT scanning, or nuclear-based imaging.

SUBCUTANEOUS ICD TECHNOLOGY

There are constant improvements being made in ICD technology. Minimally invasive and entirely subcutaneous ICDs (S-ICD) have been developed (**Figures 46-10 and 46-11**).[73-75] The advantages include that it has a simplified implantation procedure, it does not require vascular access or intracardiac leads, no imaging or fluoroscopy equipment is required, and the device may be as effective as current ICD systems.[76] Potential complications (e.g., deep venous thrombosis, intracardiac thromboses, tricuspid valve disease, or complications related to central venous access) can be eliminated.

FIGURE 46-11. Chest radiograph of a subcutaneous defibrillator. (Photo used with permission from reference 73.)

The electrode is implanted parasternally, and the ICD generator is implanted on the anterolateral chest wall (**Figures 46-10 and 46-11**). This system can effectively treat ventricular fibrillation.[76] The Emergency Department evaluation of the S-ICD is the same as the transvenously placed ICD.

WEARABLE ICD TECHNOLOGY

A patient may present to the Emergency Department with a wearable ICD (Zoll Medical Corp., Pittsburgh, PA). These are composed of the defibrillator vest, the wearable computer/monitor/power source, the battery charger, and the blue tooth transmitting unit that transmits to the manufacturer for monitoring the rhythms (**Figure 46-12**). The unit talks, vibrates, and warns the patient of an impending shock. The monitor has a display screen. A rechargeable battery snaps into the monitor. Removal of the battery inactivates the device. The wearable ICD is approximately $4000 per month, a

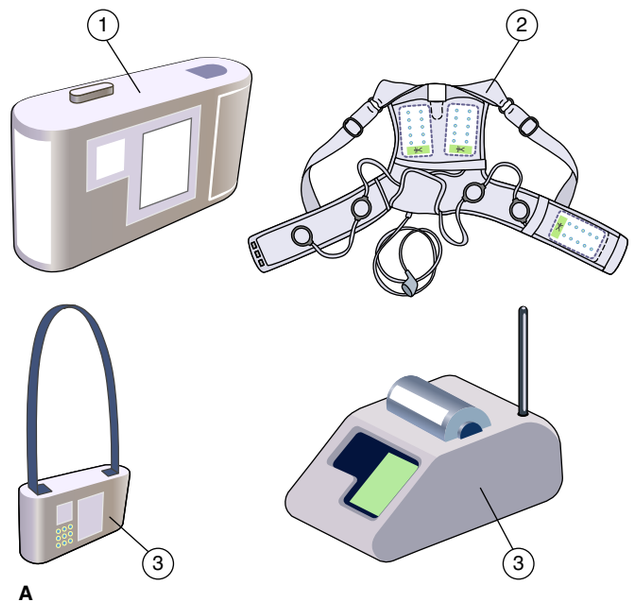
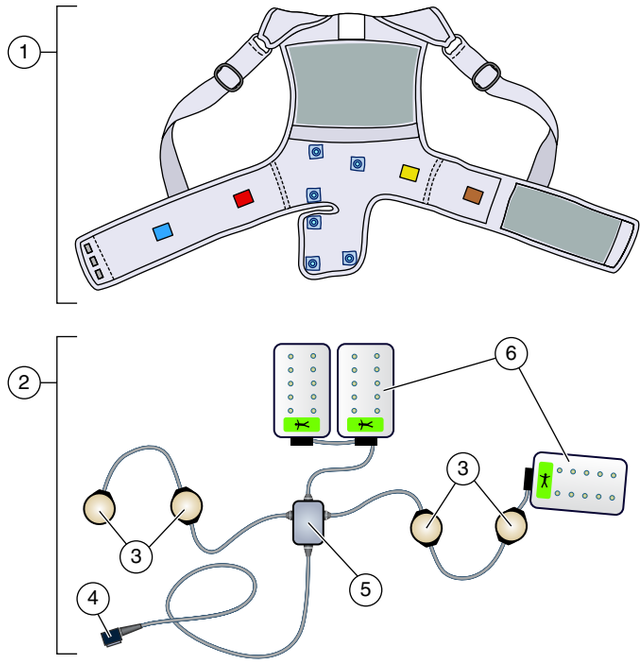


FIGURE 46-12. The wearable defibrillator. ***A.*** The components include the portable computer/monitor/power source (1), the wearable vest (2), the charger and Bluetooth transmitter (3), and the wearable case for the around the neck or waist (4). ***B.*** The vest (1), electrode belt or defibrillator (2), heart sensors (3), monitor connection cable (4), vibration box (5), and defibrillator therapy pads (6). ***C.*** The loaded electrode belt. (Photos courtesy of Zoll Medical.)



B

C

FIGURE 46-12. (*Continued*)

cost significantly lower than that of an ICD. It can be used to bridge the time between diagnosis and ICD placement.

The patient may experience a shock and come to the Emergency Department. Remove the battery pack to inactivate the device. Remove the vest with the defibrillator unit from the patient. Apply defibrillator pads/patches to the patient and attach these to the machine. Call a field engineer to interrogate the device or the manufacturer.

TWIDDLER SYNDROME

Twiddler Syndrome can be seen with any implantable subcutaneous device. Patients manipulate the device intentionally or unintentionally. This moves the ICD within the subcutaneous pocket, curling the leads (**Figure 46-13**).[77] The leads can be displaced from the ICD, displaced from the heart, or fracture. This can result in syncope or pacing of structures if the device is providing impulses to other structures (e.g., abdominal muscle wall spasm, arm spasm, chest wall, diaphragm spasm, and hiccups). The device can demonstrate problems associated with lead displacement or fracture. The device needs to be fixed by the specialist who inserted it. They will replace the lead(s), reposition the lead(s), and secure the ICD in the pocket.

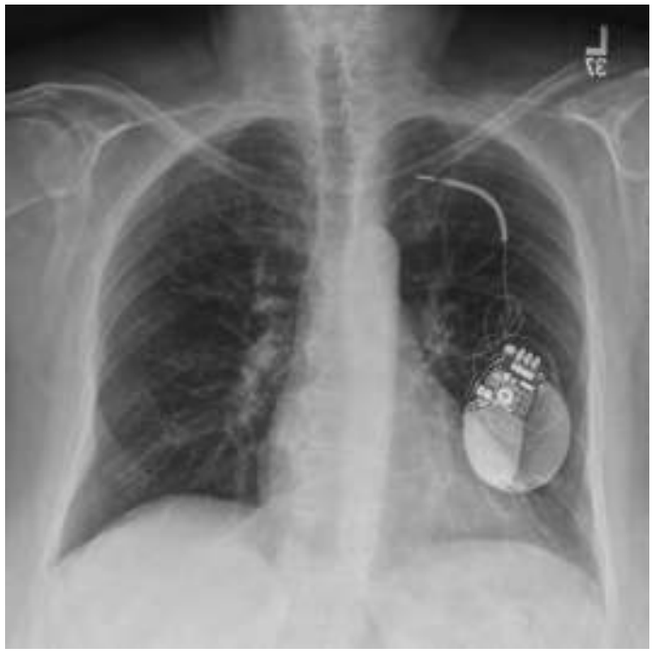


FIGURE 46-13. Chest radiograph of a patient with Twiddler syndrome. (Photo used with permission from reference 77.)

SUMMARY

Expanding clinical indications, advanced new technology, and the increasing number of annual implants require Emergency Physicians to become familiar with problems typically encountered by the patient with an ICD. Complications associated with ICDs are not uncommon. Troubleshooting and programming should be performed in conjunction with consulting an Electrophysiologist. Patients presenting with cardiopulmonary arrest may require the device to be deactivated and external defibrillation performed. Do not apply external defibrillation paddles directly over the ICD.

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47 Left Ventricular Assist Device (LVAD) Assessment

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INTRODUCTION

Heart failure affects more than 5 million Americans, of whom approximately 6% have advanced heart failure.[1,2] Advanced heart failure is characterized by severe progressive symptoms despite optimal medical therapy, recurrent hospital admissions, and very poor prognoses. Therapeutic options include cardiac transplantation, mechanical circulatory support, and palliative care. Cardiac transplantation is limited by donor availability and recipient eligibility.[3,4]

Left ventricular assist devices (LVADs) provide mechanical circulatory support. They are pumps that move blood from the left ventricle to the aorta. LVADs were first approved in the mid-1990s to support patients as a bridge to transplant (BTT) until a suitable donor heart became available. They have advanced in recent years as a superior alternative to medical therapy in patients who are not eligible for transplantation (i.e., destination therapy [DT]).[5] Strict criteria have been established and must be met for a patient to be considered a candidate for a LVAD as DT (**Table 47-1**).[6]

The introduction of smaller rotatory, continuous-flow, durable intracorporeal pumps has led to an exponential increase in the number of implants. More than 10,000 continuous-flow LVADs (CF-LVADs) have been implanted in the United States.[7,8] The 1-year survival after CF-LVAD implantation for DT has been reported to be approximately 80%.[9] The complication rates are high. Within 1 year of implantation, approximately 80% of patients are readmitted for LVAD-associated causes (e.g., device infection, device malfunction, or management of nontherapeutic anticoagulation) and non–LVAD-associated causes (e.g., infections, progressive right ventricular failure, psychosocial issues).[10,11] This significant morbidity, along with the increasing number of patients supported by LVADs, requires that Emergency Physicians understand the function and complications of LVADs.[12-14]

TABLE 47-1 Indications for an LVAD

Balloon pump dependent for 7 days
Failure to respond to optimal medical management for at least 45 of the last 60 days
IV inotrope dependent for 14 days
Left ventricular ejection fraction < 25%
New York Heart Association functional class IV
Not a heart transplant candidate
Severe functional limitation with a peak $VO_2 \leq 14$ mL/kg/min

LVAD DEVICES

PUMP FUNDAMENTALS

The LVAD can be conceptualized as a pump that uses energy to move fluid from one site to another (**Figures 47-1 and 47-2**).[15] LVADs have an inflow cannula (i.e., suction pipe) in the left ventricle, a pump, an outflow cannula (i.e., discharge pipe) in the ascending aorta, and a driveline that connects the pump with the system controller. The system controller is connected to a power source (i.e., batteries or direct power). Continuous-flow pumps can be divided into axial and centrifugal. An axial pump uses a propeller in a pipe and transmits rotational energy without a change in the direction of blood. A centrifugal pump uses an impeller and pushes the blood and directs it tangentially. These mechanisms have a fundamentally different hydrodynamic performance.[16]

The pressure difference across the inlet and outlet of the pump is known as the head pressure. The head pressure of an LVAD varies during the cardiac cycle, increasing during diastole and decreasing during systole. The head pump curves describe the relationship of head pressure on the y-axis and pump flow on the x-axis at the defined operating speed (**Figure 47-3**).[17] There is an inverse relationship between head pressure and flow. The pump speed is fixed. When the head pressure increases, most of the rotational energy transmitted by the pump is "wasted" generating pressure. When the head pressure decreases, the rotational energy is used to generate flow.

Centrifugal flow devices have "flat" head pump curves (**Figure 47-3**). This means that small variations in head pump pressures are associated with large variations in flow. Axial flow pumps have a steep head pump curve (**Figure 47-3**). They show smaller variation of flow with similar reductions in head pressure. The clinical significance of this hydrodynamic behavior is increased flow pulsatility and afterload sensitivity in patients with centrifugal pumps.[17] Axial pumps show high pressure differentials and power consumption at lower flow rates. The pump pulls harder at lower flow rates. In the context of relative or absolute hypovolemia, the patient is more likely to suffer suction phenomena or the contact of the inflow cannula and the left ventricular endocardium.

This chapter will focus on two specific devices (**Figure 47-1**). The Heart Mate II (Thoratec Corp., Pleasanton, CA) was approved for BTT and DT. It is a second-generation preperitoneal axial pump

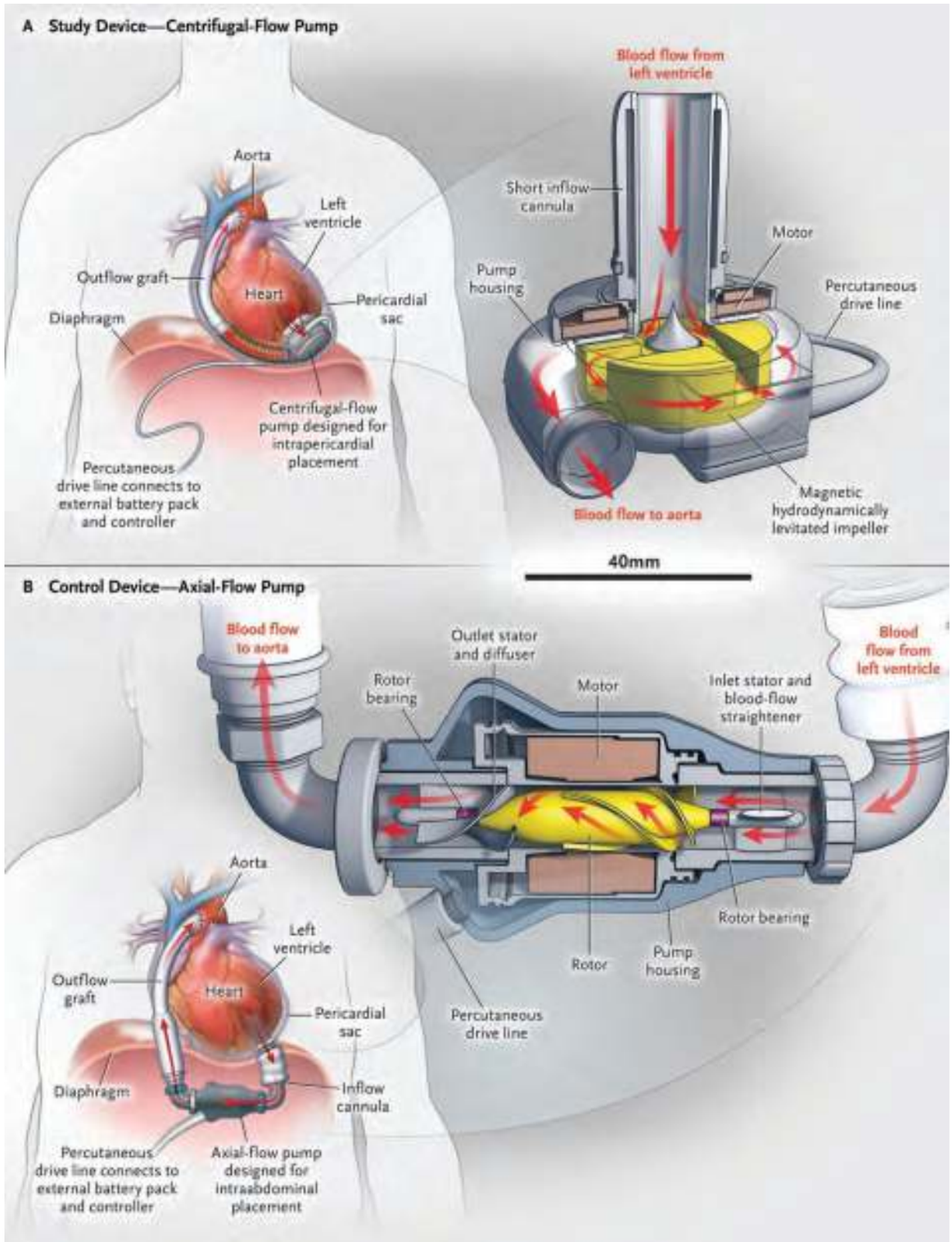


FIGURE 47-1. Two types of LVADs. ***A.*** A compact centrifugal-flow device with an integrated inflow cannula designed for intrapericardial placement. The device incorporates a bearing-less design with magnetic and hydrodynamic levitation of the internal impeller. ***B.*** An axial-flow device that requires bearing support of the internal impeller and is implanted outside the pericardium in a preperitoneal (pump) pocket. (Used with permission from reference 15.)

with a titanium propeller supported by mechanical bearings that can rotate between 6000 and 15,000 revolutions per minute (rpm). The typical speed is 8800 to 9200 rpm. The HeartWare HVAD (HeartWare, Framingham, MA) is only approved for BTT (**Figures 47-1 and 47-4**). It is a third-generation small intrapericardial centrifugal pump. The impeller is supported by hydrodynamic and magnetic mechanisms that are friction free. It can rotate at a speed range between 1800 and 4000 rpm. The typical speed is 2600 to 2900 rpm.

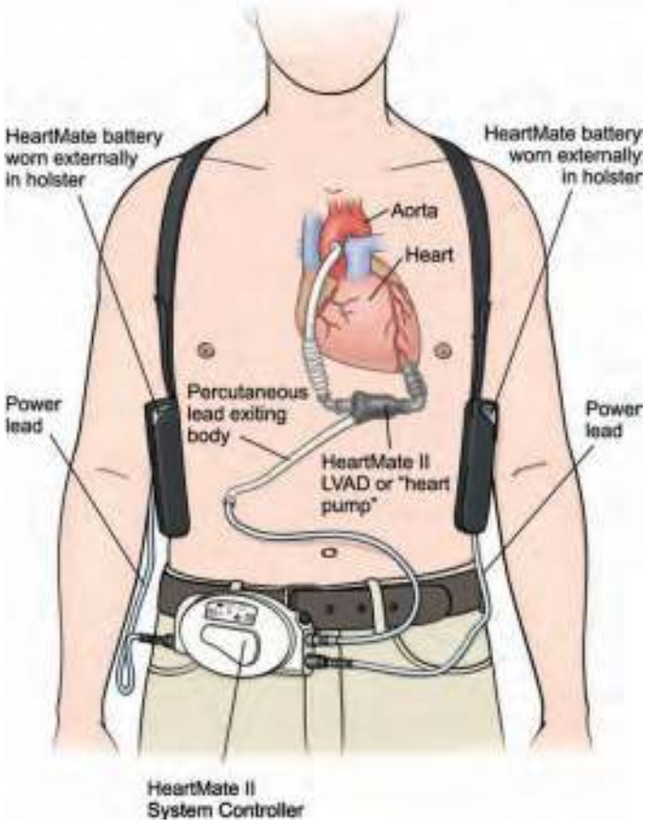


FIGURE 47-2. An example of a patient with the components of an LVAD. (Courtesy of Thoratec Corp.)

SYSTEM CONTROLLER

The system controller is a small computer that regulates LVAD function (**Figure 47-4C**). It serves as a user interface by displaying advisory alarms, battery status, flow, hazard alarms, power, pulsatility parameters, and speed. Pump speed is measured in rpm and is the only variable that can be programmed by the operator. Pump power is a direct measure of the energy consumption by the pump. Flow is not directly measured in these pumps using a flow meter. LVAD flow is estimated using power in the Heart Mate. LVAD flow is estimated by using hematocrit, pump speed, and power in the HeartWare. Trends in flow are clinically useful, but absolute values do not accurately represent the cardiac output. The increase in intracavitary pressure during ventricular systole causes a decrease in total head pressure with a consequent increase in flow.

This variation in flow during the cardiac cycle represents the pulsatility. Pulsatility depends on the function of the left ventricle as a pump and preload. Pump flow pulsatility is the difference between the minimum (i.e., trough) and maximum flows. The Heart Mate pulsatility index (PI) represents the delta flow/average flow over 15 seconds. Values range between 1 and 10. The higher the PI, the less LVAD support. PI event occurs when there is a 45% change in flow from the previous 15-second running average. The pulsatility of the HeartWare is displayed in the pump flow waveform.

ANTICOAGULATION

Current LVADs have demonstrated acceptable hemocompatibility. Warfarin anticoagulation with international normalized ratio (INR) values of 2 to 3 and antiaggregation with 81 to 325 mg of aspirin daily remain the standard of care.

OTHER CARDIAC FACTORS INFLUENCING LVAD FUNCTIONS

LVADs provide left ventricular unloading. Several patient conditions may influence LVAD efficiency. The degree of left ventricular dysfunction, preexisting right ventricular dysfunction, and progressive right ventricular dysfunction may contribute to the appearance of a low-flow state and congestion. Aortic insufficiency may lead to ineffective cardiac output and heart failure symptomatology. The patient is subject to right to left shunting in the context of low left-sided filling pressures with an untreated patent foramen ovale.

EVALUATION OF THE LVAD PATIENT

Patients with LVADs are subject to the same clinic conditions as any member of the population (**Figure 47-5**).[18] **Do not anchor on the LVAD as the cause of the complaint. Treat the appropriate pathology while being sensitive to the alterations in the patient's physiology and increased risks secondary to the LVAD.** There are some common clinical scenarios leading to Emergency Department evaluation of LVAD patients. The patient may have no symptoms, but the device is alarming. The patient may be symptomatic (e.g., chills, fatigue, fever, gastrointestinal bleeding, neurologic deficits, and shortness of breath) with or without the LVAD alarming. The patient may present unconscious with or without signs of circulatory collapse and with or without the LVAD alarming.

LVAD implantation occurs after a careful multidisciplinary team evaluation of cardiac (e.g., arrhythmias, ischemic heart disease, right ventricular function, and valvular heart disease) and noncardiac (e.g., hepatic, neurologic, and pulmonary function, psychosocial factors, renal factors) considerations. All certified ventricular assist device centers have LVAD coordinators that play a central role in the longitudinal follow-up of these patients. **Obtain contact information from the implanting center and the patient's LVAD coordinator as early as possible to help facilitate care.** A list of high-yield questions is provided in **Table 47-2**.

PHYSICAL EXAMINATION OF THE LVAD PATIENT

Approximately 60% of patients with current CF-LVADs do not have a palpable pulse. A manual cuff can be used to measure blood pressure (BP) when a radial pulse is readily palpable. Record the mean BP if the systolic blood pressure (SBP) and diastolic blood pressure (DBP) are attainable. The mean BP can be defined as [DBP + (SBP – DBP) ÷ 3]. Consider the use of a Terumo BP cuff. The Doppler technique can be used in case of failure to obtain BP recordings. The opening Doppler BP is used as the SBP.[19] Place an arterial line (Chapter 72) in critically ill patients in whom manual or Doppler BP measurement is technically difficult or unreliable or continuous pressure measurement is needed to help guide care.

The use of Doppler is indicated when the radial pulse is not palpable. Place the manual BP cuff following standard recommendations of size and location. Place the Doppler probe over the brachial artery and auscultate the brachial pulse. Inflate the cuff until the pulse is no longer audible by Doppler. Slowly deflate the cuff in 2 to 3 mmHg/sec increments, allowing the reestablishment of flow. Record the opening pressure when the pulse becomes audible. The opening pressure will closely estimate the mean BP. The mean BP goal in patients supported by CF-LVADs should be approximately 70 to 90 mmHg.

Examine the patient and the LVAD. **Pulse oximetry may not be reliable in nonpulsatile flow states. Perform arterial blood gasses if there is a question of adequate oxygenation.** Direct the chest auscultation to detect the continuous hum characteristic of

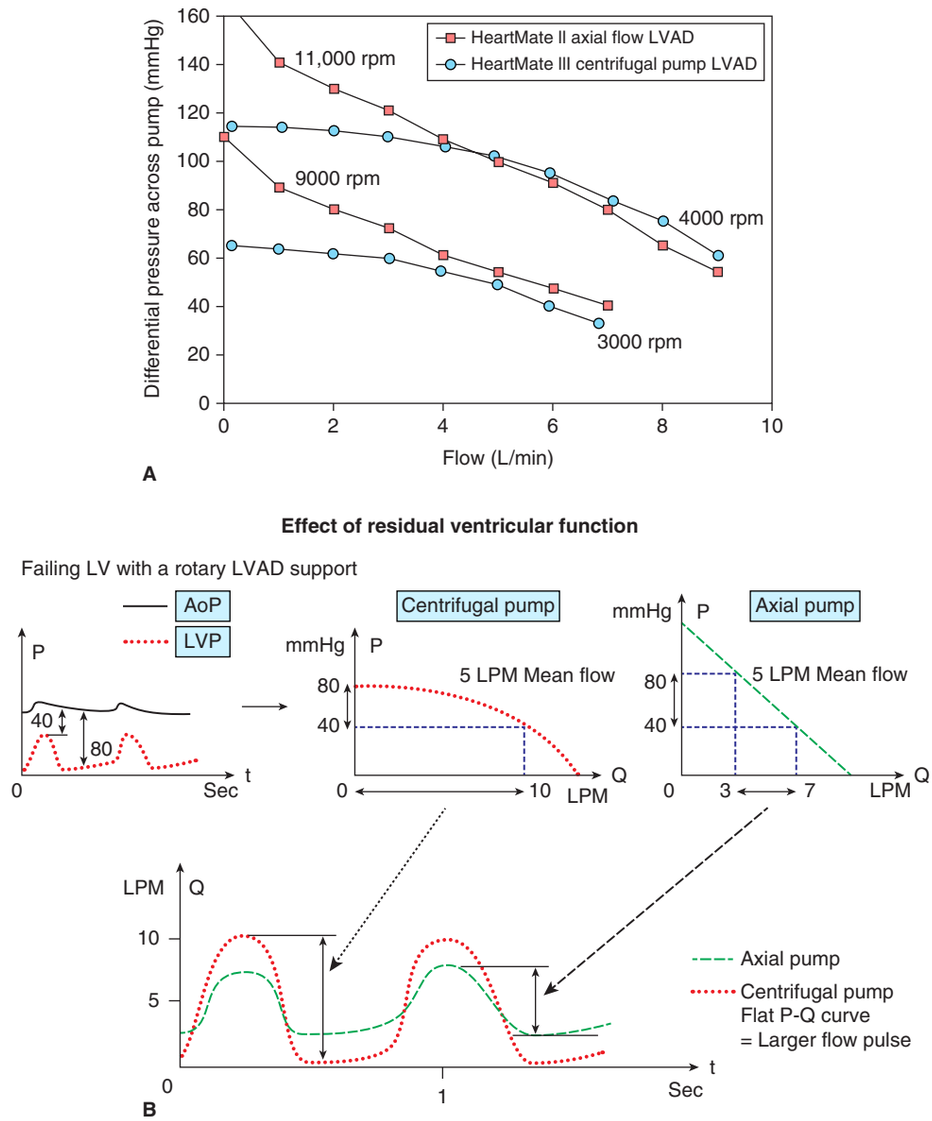


FIGURE 47-3. Differences between axial and centrifugal pumps. ***A.*** Typical pressure–flow relationship. ***B.*** Demonstration of the greater sensitivity of centrifugal pumps. AoP, aortic pressure; LV, left ventricle; LVP, left ventricular pressure. (Used with permission from reference 17.)

a working CF-LVAD. Focus the physical examination on detecting congestion, hypoperfusion, and neurologic deficits. Carefully evaluate the driveline integrity.[20] **Do not pull on the driveline.** Evaluate the driveline exit site to assess for signs of inflammation or infection (e.g., drainage, induration, pain, or redness). Evaluate the system controller for signs of burns, deterioration, immersion, or other signs of wearing. Evaluate any LVAD alarms as described below.

ANCILLARY TESTS

Chest radiographs can be useful (**Figure 47-6**).[18] They can detect causes of dyspnea or fever (e.g., cardiomegaly, infiltrates, pericardial effusions, or pleural effusions). Gross malalignment of the inflow cannula can be observed in chest radiographs. Consider a Panorex radiograph to rule out a dental infection. Electrocardiogram evaluation may disclose atrial fibrillation, which has been associated with an increased risk of ischemic stroke and ventricular tachycardia in patients who present with nonspecific symptoms.

Focused laboratory evaluation may provide useful information. A complete blood count (CBC) can be used to determine acute anemia from bleeding or hemolysis. Thrombocytopenia can be related to platelet consumption. Leukocytosis is associated with an infection. Evaluate the driveline and look for signs of septic embolism. Lactate dehydrogenase (LDH) is increased in hemolysis that may be related to pump thrombosis. Low-grade elevation of LDH is frequently seen in LVAD patients. Therapeutic anticoagulation evaluation looks at the INR for patients using warfarin. Consider pump thrombosis with a low INR, high LDH, and power spikes. Renal function increases in renal failure from hypoperfusion and/or renal congestion due to right ventricular failure. Liver function test alterations may be a clue to right ventricular failure. Brain natriuretic peptide (BNP) may be elevated in incomplete unloading pump thrombosis, left ventricular failure, or right ventricular failure.

Echocardiography provides essential information for the assessment of the LVAD patient. The parasternal long axis is useful to evaluate for aortic valve opening, left ventricular filling, left ventricular function, pericardial effusion, and right ventricle function. M-mode is used to characterize the timing of atrioventricular valve opening. Color Doppler may be useful to assess for the presence of aortic insufficiency. Additional views will complement the evaluation of

FIGURE 47-4. The HeartWare LVAD. ***A.*** The device. ***B.*** The open device showing the impeller. ***C.*** The controller. (Courtesy of Medtronic.)

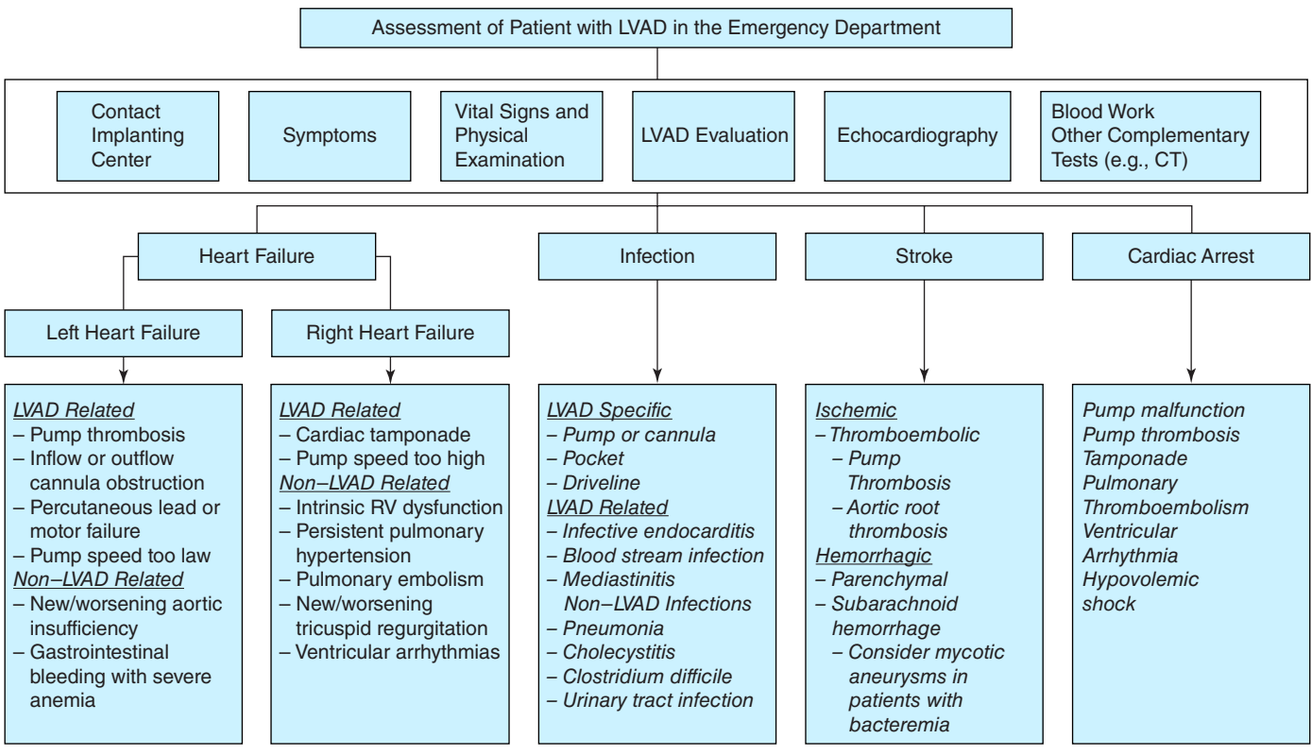


FIGURE 47-5. Assessment of the patient with an LVAD. CT, computed tomography; RV, right ventricle.

TABLE 47-2 High-Yield Questions

Question	Possible etiology
Are you experiencing shortness of breath and/or leg swelling?	Left-sided heart failure Right-sided heart failure
Have you noticed the development of dark urine?	Acquired hemolysis Rotor pump thrombosis
Are you experiencing fatigue?	Acute blood loss Gastrointestinal bleeding Heart failure Hemolytic anemia
Have you experienced darker and/or bloody stools?	Acquired arteriovenous malformations Gastrointestinal bleeding
Have you noticed drainage and/or pain around your driveline exit site?	LVAD-related infection
Have you experienced fever and/or chills?	LVAD-related infection
Have you experienced any alarms? If so, what kind (e.g., advisory or hazard)?	Driveline malfunction Hypovolemia Pump thrombosis
Have you had any incidence of trauma to driveline?	Driveline fracture

ventricular function and provide information on the inflow cannula position. The inflow cannula position is considered normal when aligned with the left ventricle inflow tract. Algorithms for the evaluation of left ventricle filling pressures and detailed guidelines for echocardiographic evaluation have previously been published and are beyond the scope of the Emergency Physician. A ramp test (i.e., changes in left ventricle diameter and atrioventricular valve opening are measured with progressive LVAD speed changes) should only be performed in experienced centers. Suspect incomplete unloading if the left ventricle is dilated and the atrioventricular valve is opening.

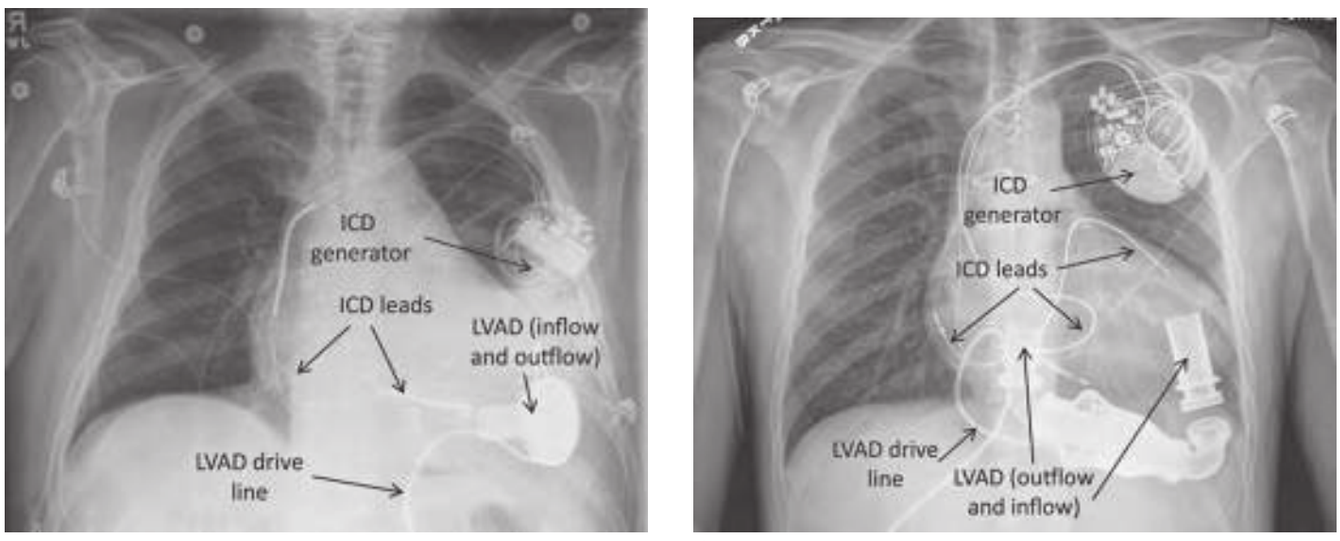
LVAD INTERROGATION

Evaluate the system controller and patient for evidence of device function.[20] Identify any alarms (**Table 47-3**). Verify all connections and that the batteries are charged. Analyze alarms in the clinical context of the patient (**Table 47-3**). Suspect a short to shield phenomenon when low-flow alarms or pump stoppage occurs when the device is connected to the power base unit and resolves when it is

TABLE 47-3 Some of the More Common Alarms on an LVAD Controller

Alarm	Cause	Actions
LVAD stopped; read heart flashing	Battery depletion Controller malfunction Driveline disconnection	Connect batteries Connect driveline Replace batteries Replace controller
High flow	Increased atrioventricular opening Moderate to severe mitral regurgitation Sepsis Vasodilation	Hold vasodilators Treat sepsis
Low flow	Arrhythmias Bleeding Decreased atrioventricular opening Hypovolemia Kinking or obstruction of tubing Pericardial effusion/tamponade Right ventricular dysfunction Right ventricular enlargement	Bolus fluids Relieve kinking of tubing Relieve obstruction of tubing Relieve pericardial effusion Relieve pericardial tamponade Transfuse and stop bleeding Treat arrhythmia Treat increased systemic resistance
High power	Pump thrombosis	Contact implanting center IV anticoagulation if INR is < 2
High pulsatility index; increased pulsatility waveform	Recovery of left ventricle function Percutaneous lead damage	Look for evidence of recovery Assess LVAD components
Low pulsatility index	Excessive pump speed Hypovolemia Poor native ventricular function	Add inotropic support Adjust pump speed Fluid bolus
Suction event*	Arrhythmias Cannula opening obstruction Excessive ventricular unloading Hypovolemia Kinking or obstruction of tubing	Fluid bolus Lower pump speed Relieve cannula obstruction Relieve kinking of tubing Relieve obstruction of tubing Treat arrhythmias

*May manifest as alarms, low flows, or low pulsatility index. Pulsatility index events occur when there is a 45% +/– change from the previous 15 second running average.



A B

FIGURE 47-6. Chest radiographs of LVADs. ***A.*** HeartWare LVAD. ***B.*** Heart Mate II LVAD. (Used with permission from reference 34.)

connected to batteries. The Heart Mate device driveline consists of three pairs of cables covered by a metallic shield. The energy will be drained from the pump, causing a voltage drop and alarms if there is a loss of integrity to the wires and any wire contacts the metallic shield while the device is connected to a grounded wire on the power base unit.[21]

CLINICAL SCENARIOS AND COMPLICATIONS SEEN IN LVAD PATIENTS

The following section describes some common scenarios and complications for conditions that occur in LVAD patients. Some of the conditions are listed in **Table 47-4**.

ECHOCARDIOGRAPHY

The use of bedside ultrasonography can help determine the etiology of the patient's symptoms. A large right ventricle and a small left ventricle is seen in pulmonary hypertension, right heart strain, and right-sided myocardial infarctions. Correct any acidosis, correct hypoxemia, consider inotropes, and evaluate for a myocardial infarction. A small right ventricle suggests dehydration. Consider a fluid bolus. Enlarged right and left ventricles suggest pump failure or thrombosis. Small right and left ventricles suggest hypovolemia, gastrointestinal bleeds, and sepsis.

HEART FAILURE

The presence of signs and symptoms of heart failure warrants an evaluation. Look for LVAD-related and non–LVAD-related factors (**Figure 47-5**).[22,23]

HYPERTENSION

Consider aggressive BP management (i.e., afterload reduction) with intravenous (IV) agents (e.g., nitroprusside) in the presence of signs or symptoms of heart failure, low LVAD flows, and no evidence of neurologic deficits when the mean arterial pressure is > 110 mmHg. Consider the impact of the negative inotropic effects on the right ventricle before using IV beta-blockers.

HYPOTENSION

Patients with LVADs usually tolerate fluid boluses. Increase the preload with a fluid bolus. Consider the administration of inotropes for right ventricular failure (e.g., dobutamine) or sepsis (e.g., norepinephrine). Temporarily decrease the dosage of diuretics.

PUMP THROMBOSIS

An increase in early pump thrombosis has been reported in patients with CF-LVADs.[24-26] The clinical presentation depends of the degree of pump dysfunction and residual intrinsic left ventricle function. It can range from mild fatigue to cardiogenic shock. Pump thrombosis leads to intravascular hemolysis manifesting in a significant increase in LDH and hemoglobinuria, which may be recognized by the patient as a change in the color of their urine. System controllers show power spikes and high estimated flows. Consider IV heparin anticoagulation and contact the LVAD reference center for further management support.[23] Routine thrombolytic administration is not currently recommended.[27]

OTHER THROMBOTIC COMPLICATIONS

Aortic root thrombosis is caused by stagnant blood flow in the aortic root. Stasis of blood may lead to thrombus formation in the noncoronary cusp and may extend, causing obstruction of coronary arteries with subsequent ischemia. This may present as an acute coronary syndrome or ventricular tachycardia, or may embolize, causing ischemic complications (e.g., stroke). Consider a

TABLE 47-4 Diagnostic Characteristics of Common LVAD Complications

	Right ventricular failure	Pump thrombosis	Sepsis or infection	Hypovolemia
Symptoms	Congestion Shortness of breath	Dark urine Shortness of breath	Driveline site pain Fatigue Fever	Hematemesis Hematochezia Melena
Hemodynamics				
MAP	Decreased	Decreased	Decreased	Decreased
CVP	Increased	Increased	No change	Decreased
PCWP	No change	Increased	No change	Decreased
SvO_2	Decreased	Decreased	Increased	Decreased
Physical examination	Edema Hepatomegaly JVD	Edema Rales	Cellulitis or purulent drainage from the drive-line	Decreased capillary refill Pale mucous membranes
Laboratory	Abnormal LFTs Increased BNP	Hemoglobinuria Increased BNP Increased free plasma hemoglobin Increased LDH May have INR< 2	Leukocytosis	Anemia Guaiac positive INR > 3
Echocardiography	Dilated right atrium or right ventricle	Dilated left atrium or left ventricle Aortic valve opening.	Normal	Underfilled
Device				
Power	Low	Increased	Increased	Decreased
Pulsatility	Low	Decreased	Increased	Decreased
Flow	Low	Increased	Increased	Decreased
Management	Consider RVAD Decreased LVAD speed Inotropes	Medical management: increasing antiplatelets and anticoagulation	IV fluids Stop vasodilators Treat infections	Blood transfusion IV fluids Treat bleeding

BNP, brain natriuretic peptide; CVP, central venous pressure; INR, international normalized ratio; JVD, jugular venous distension; LDH, lactate dehydrogenase; LFTs, liver function tests; LVAD, left ventricular assist device; MAP, mean arterial pressure; PCWP, pulmonary capillary wedge pressure; RVAD, right ventricular assist device.

computed tomography scan with IV contrast if this diagnosis is suspected.[28]

Heart failure is a major risk factor for pulmonary embolism. Consider pulmonary embolism in patients who present with signs of acute right heart failure, especially if their INR is below the therapeutic range. Follow diagnostic and therapeutic guidelines for the treatment of a pulmonary embolism.[29]

RIGHT VENTRICULAR FAILURE

Right ventricular failure is an important cause of morbidity and mortality in LVAD-supported patients.[8] Factors related to the development of right ventricular failure include intrinsic baseline right ventricular function, right ventricular geometrical distortion due to displacement of the interventricular septum to the left, alterations of systolic and diastolic ventricular interdependence, and increased preload to the right ventricle.[30,31] Treatment includes inotropes to augment right ventricular function (e.g., dobutamine, epinephrine, and milrinone), altering the pump speed, IV fluids, and vasopressors to maintain right ventricular perfusion pressure. Intubate the LVAD patient if necessary. Avoid elevated peripheral vascular resistance, high positive end-expiratory pressure, hypercarbia, and hypoxia, which worsen right ventricular function.

The Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) defines right ventricular failure as signs and symptoms of persistent right ventricular dysfunction and central venous pressure > 18 mmHg with a cardiac index < 2.0 L/min/m^2. It is associated with the absence of elevated left atrial/pulmonary capillary wedge pressure > 18 mmHg, tamponade, ventricular arrhythmias, or pneumothorax. Right ventricular failure requires a right ventricle assist device implantation or inhaled nitric oxide or inotropic therapy for more than 1 week at any time after LVAD implantation. Right ventricular failure is severe if it requires a right ventricular assist device. It is moderate if IV inotropes or inhaled pulmonary vasodilators (e.g., inhaled nitrous oxide or prostaglandin E) are used. Right ventricular failure is considered mild if two of the following four criteria are met: central venous pressure > 18 mmHg or mean right atrial pressure > 18 mmHg, cardiac index < 2.3 L/min/m^2 using a pulmonary artery catheter, fluid overload (i.e., ascites or evidence of moderate to worse peripheral edema), or evidence of elevated central venous pressure by echocardiogram (i.e., dilated inferior vena cava without collapse) and in physical examination (i.e., signs of increased jugular venous pressure).

Right ventricular failure can be classified according to the timing of occurrence in relation to LVAD implantation as intraoperative, early (≤ 14 days), and late (> 14 days). Early right ventricular failure usually occurs in the implanting center. Late right ventricular failure may be more difficult to diagnose, and a high index of suspicion is important to avoid underdiagnoses. Right ventricular failure may manifest as progressive dyspnea, ventricular arrhythmias, or frequent hospitalizations.

SUCTION EVENTS

Suction or suck-down events are often due to hypovolemia where the intake cannula is against the wall of the ventricle (**Figure 47-7**). This can cause arrhythmias and syncope. The PI is reduced. Overcome this with IV fluid boluses and decreasing the pump rpms.

AORTIC INSUFFICIENCY

The development of aortic valve insufficiency in patients with a CF-LVAD may lead to incomplete left ventricular unloading and cardiac failure.[25,32] It might be difficult to accurately quantify the degree of aortic valve insufficiency. The presence of heart failure and continuous aortic valve insufficiency in M-mode color Doppler should raise the suspicion of aortic insufficiency.

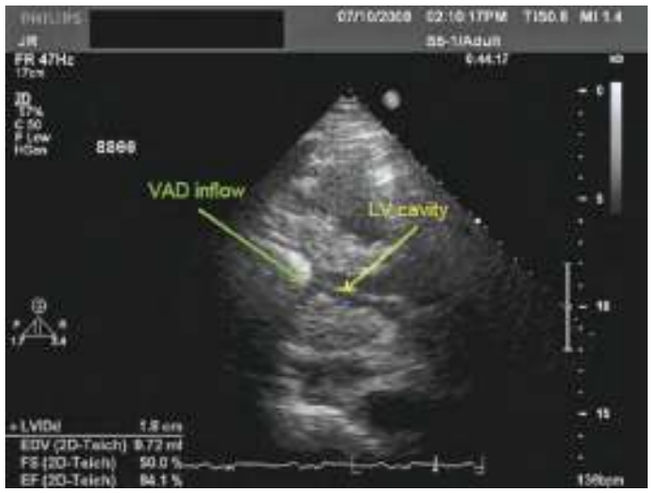


FIGURE 47-7. Ultrasound image of a suction event. LV, left ventricle. (Used with permission from reference 8.)

ARRHYTHMIAS

Approximately 25% of LVAD patients will experience an episode of ventricular arrhythmia.[8,25,33] The incidence of ventricular arrhythmias is higher in the first 30 days of implantation. The most important risk factor for late ventricular arrhythmias (i.e., > 30 days) is a history of preimplantation ventricular arrhythmias. The clinical presentation ranges from nonspecific symptoms to cardiovascular collapse. Factors that may impact the tolerance of ventricular arrhythmias include significant pulmonary hypertension with elevated pulmonary vascular resistance. Some have related the tolerance to ventricular arrhythmias to the capacity of withstanding Fontan circulation. Treatment of ventricular arrhythmias should follow standard advanced cardiac life support (ACLS) protocols, including cardioversion and defibrillation.[34]

GASTROINTESTINAL BLEEDING

Approximately 30% of LVAD patients will develop gastrointestinal bleeding requiring at least 1 unit of red blood cell transfusion within 1 year after device implantation. Factors related to the increased gastrointestinal bleeding include therapeutic anticoagulation, platelet antiaggregation, continuous flow physiology that is related to acquired von Willebrand disease, and arteriovenous malformation in the gastrointestinal tract.[8,25,33] **Arteriovenous malformations and gastritis are the most frequent causes of bleeding.** The patients frequently have recurrent episodes of gastrointestinal bleeding. It is important to inquire about current anticoagulation and antiplatelet regimens. The patient with recurrent gastrointestinal bleeding may have their anticoagulation discontinued.[35]

The diagnostic and therapeutic approach is similar to that of other patients with gastrointestinal bleeding, but some additional considerations are needed. Have a low threshold for admission to the hospital given the presence of concomitant comorbidities. Aggressive anticoagulation reversal is discouraged given the high risk of thrombotic events in the absence of upper gastrointestinal bleeding with hematemesis or hemodynamic instability. **Comanagement with the LVAD reference center is crucial.** Transfuse leukocyte-reduced irradiated blood if possible to avoid allosensitization and cytomegalovirus in patients whom an LVAD was inserted as a BTT.

No studies have assessed the safety of nasogastric tube placement. Nasal arteriovenous malformations are extremely common in this patient population. Epistaxis is not an uncommon complication.

STROKE

It has been estimated that approximately 17% of patients who receive an LVAD will suffer a stroke. Hemorrhagic strokes are just as common or more frequent than ischemic strokes.[8,33,36] **Magnetic resonance imaging is contraindicated in LVAD patients.** The safety of thrombolytic therapy has not been studied. A history of bacteremia and device infection has been associated with increased risk of stroke.[37]

INFECTIONS

Complications related to infection are the second most common cause of death 6 months after device implantation.[8,25,33,38] The International Society of Heart and Lung Transplantation (ISHLT) classification of infections in LVAD patients is shown in **Table 47-5**.[18,39] Pneumonia and sepsis are the most frequently reported complications, followed closely by percutaneous site infections. Percutaneous site infections affect 19% of the patients and are associated with increased mortality. **Careful inspection of the driveline insertion site for any evidence of cellulitis or drainage is mandatory.**[25] Initial investigation for a suspected infection includes a CBC, echocardiography, serial erythrocyte sedimentation rates, and three sets of blood cultures over 24 hours.[18] No standard guidelines for antibiotic therapy exist for this patient population.[40] Cover gram-negative organisms and methicillin-resistant *Staphylococcus aureus* (MRSA). Long-term use of an LVAD can result in reversible

TABLE 47-5 The International Society of Heart and Lung Transplantation Classification of LVAD Patient Infections

Non–LVAD-Related Infections
Appendicitis
Bladder infections
Cholecystitis
Lower respiratory tract infection
Pneumonia
Pyelonephritis
LVAD-Related Infections
Bloodstream infection with central catheters
Esophageal perforation
Infective endocarditis
Mediastinitis
Pocket infection
Sternal wound infection
LVAD-Specific Infections
Cannula infection
Driveline infections
Pocket infection
Pump infection

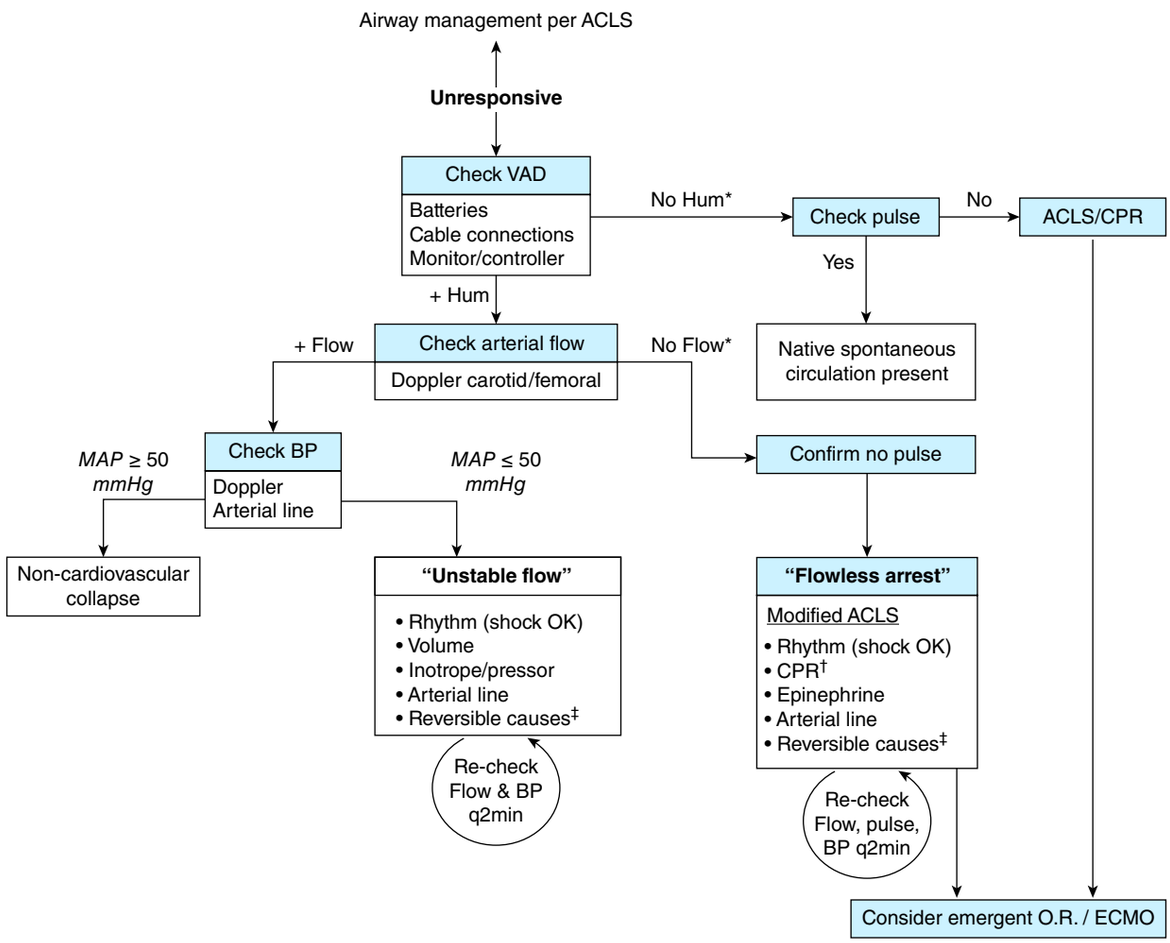


FIGURE 47-8. Management of cardiac arrest in the LVAD patient. ECMO, extracorporeal membrane oxygenation; MAP, mean arterial pressure. (Used with permission from reference 44.)

decreases in lung function.[41] It can also result in decreased cardiac index,[42] increased preload, increased afterload, increased pulmonary capillary wedge pressure, and a decrease in the ratio of right atrial pressure to pulmonary capillary wedge pressure.

CARDIAC ARREST

Patients with LVADs are not included in the special circumstances of resuscitation in the ACLS guidelines.[43] Airway management does not differ from other patients. **The assessment of circulation is significantly different. Most LVAD patients do not have a pulse.** Check the device controller and evaluate rapidly reversible causes of LVAD dysfunction (e.g., connect driveline and change batteries to connect to current). Evaluate the presence of the LVAD "hum" and native pulse.[44] There has been concern that cardiopulmonary resuscitation (CPR) may lead to outflow cannula displacement. This should not represent a contraindication to CPR but rather an additional reason for careful LVAD function evaluation.[45] Only perform CPR if the LVAD pump is not working and the patient has no BP or a low BP (i.e., < 60 mmHg).[46-49] Rapid placement of an arterial line for invasive arterial BP monitoring is extremely useful to guide resuscitative efforts. A comprehensive algorithm is noted in **Figure 47-8**.

SUMMARY

Make a rapid attempt to communicate with the LVAD reference center team if the patient is clinically stable. Knowing how to evaluate LVAD parameters, the common complications surrounding LVADs, and the common device-related and device-associated complications is important to expedite the assessment and care of these patients.

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48 Pericardiocentesis

Marianne Juarez, Jacqueline Nemer

INTRODUCTION

Pericardiocentesis is the removal of fluid from the pericardial space. This is usually performed using a needle and syringe. A pericardial catheter can be placed for ongoing removal of fluid from within the pericardium. Pericardiocentesis may be performed to obtain pericardial fluid for analysis, to relieve the pressure of a pericardial effusion, to improve cardiac output, or as a lifesaving measure to relieve a cardiac tamponade. **The technique is relatively simple to perform yet has a significant rate of complications.**[1]

Penetrating chest injuries and their subsequent outcomes have been described in the romantic and medical literature for centuries. Cardiac tamponade was first described by Riolanus as early as 1649, with pericardiocentesis described in 1827 by Thomas Jowett as an intervention for pericarditis.[2-9] In 1829, Baron Larrey, Napoleon's Surgeon, is reported to have performed the first successful pericardiocentesis.[6] By 1939, Bigger had suggested that some patients with cardiac tamponade could be managed with pericardial tubes alone with prompt operation for recurrence.[8]

ANATOMY AND PATHOPHYSIOLOGY

ANATOMY OF THE HEART AND PERICARDIUM

The pericardium is an inverted cone-shaped sack surrounding the heart that sits atop the diaphragm (**Figure 48-1**).[10] The inner portion, or visceral pericardium, is a single layer of mesothelial cells covering the epicardium. The outer layer is composed of a dense outer fibrous tissue with an inner layer of mesothelial cells known as the parietal pericardium. The fibrous pericardium is attached to the central tendinous portion of the diaphragm inferiorly. The outer fibrous layer blends superiorly with the sheath covering the great vessels. It attaches anteriorly to the posterior surface of the sternum. It is attached posteriorly to the thoracic vertebral column, esophagus, bronchi, and aorta.

The pericardial cavity is a potential space between the visceral and parietal layers of the pericardium. It normally contains up to 50 mL of fluid that acts as a lubricant to the motion of the heart.[11] There are a variety of conditions that result in excess fluid in the pericardial space, which require drainage. The estimated causes and relative frequencies of pericardial effusions are listed in **Table 48-1**.[12-18]

The heart is contained within the pericardial sac (**Figure 48-1**). Numerous portions of the heart are exposed behind the anterior chest wall (**Figure 48-2**) and are vulnerable to injury that may result in a pericardial effusion or cardiac tamponade.[19-21] The surface area that each of these structures contributes to the anterior cardiac silhouette and a general estimate as to the incidence of injury are based on the anatomic structure as listed in **Table 48-2**.[19,22]

PATHOPHYSIOLOGY OF CARDIAC TAMPONADE

The clinical effects of cardiac tamponade occur due to accumulation of fluid under pressure in the pericardial space. This space can become quite large over time in several chronic conditions and contain pericardial effusions up to 2 L without signs of cardiac tamponade.[14,23] The pericardial sac has a very limited ability to stretch in the acute setting if the pericardial fluid volume changes rapidly. This results in an increase in fluid pressure surrounding the heart and possible cardiac tamponade. Cardiac tamponade can be caused acutely by accumulation of pericardial fluid ranging from 60 to 200 mL.[11,24]

The pressure–volume relationship between the size of the pericardial effusion and the pressure imposed on the cardiac chambers becomes exponential in the acute setting. The initial accumulation of fluid produces little or no clinical effect. The initial physiologic strategies of compensation include an increase in the systemic venous pressure, catecholamine release, and tachycardia. Small increases in the fluid amount will generate significant and increasing pressure on the heart chambers once the ability of the pericardial space to distend and accommodate more fluid is overwhelmed. Venous filling of the right heart is drastically impaired as the pericardial pressure rises. The interventricular septum bulges into the left ventricle. Left ventricular filling becomes compromised from the lack of flow from the right ventricle and the bulging inward of the interventricular septum. Cardiac perfusion eventually decreases, the heart contractibility is progressively impaired, and the patient becomes hypotensive.

A progressive decline in cardiac output occurs as pericardial fluid accumulates and intrapericardial pressure increases.[25] The right atrial pressure is initially greater than the intrapericardial pressure as the body compensates by increasing venous return. This is followed by the equilibration of the right atrial and intrapericardial pressures. The heart chambers eventually cannot achieve a pressure lower than the surrounding pericardial fluid pressure. Equilibration of diastolic pressure in each heart chamber occurs and produces the greatest drop in cardiac output. The cardiac chambers collapse as the intrapericardial pressure continues to increase. This results in intractable hypotension and death.

There is a disproportionate effect of the accumulation of small amounts of fluid in this late stage. The patient can proceed from stable and compensated to profoundly unstable quite suddenly. It is dangerous to rely on central venous pressure line monitoring alone to recognize the evolution of cardiac tamponade. It can produce dramatic temporary improvements in the clinical status of the patient if treated by the withdrawal of a small amount of fluid from the pericardial cavity.

Cardiac tamponade is a life-threatening condition that must be diagnosed and treated emergently. It may easily be overlooked

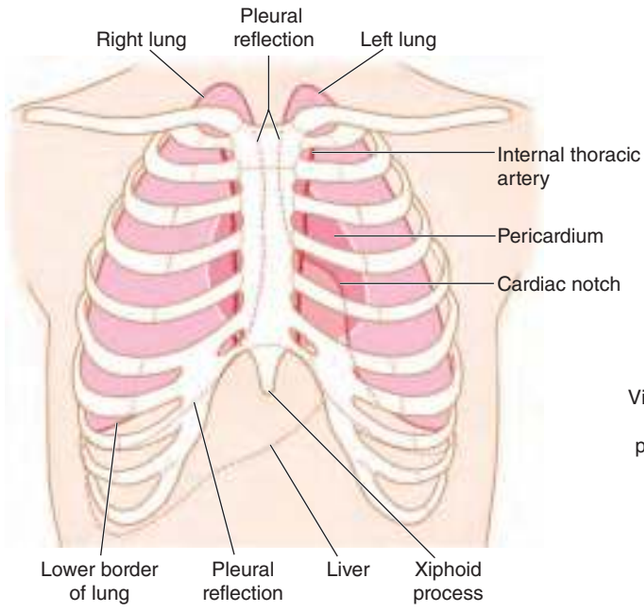


FIGURE 48-1. The pericardium. ***A.*** Relationship of the pericardium to the major thoracic structures. ***B.*** Midsagittal section through the heart and pericardium. ***C.*** Cross-section through the thorax. AIA, anterior interventricular artery; RCA, right coronary artery. ***D.*** Sagittal section through the thorax. (Photos used with permission from reference 10.)

unless a high index of suspicion is maintained in medical and trauma patients. Pericardial effusions and tamponade may occur in several medical conditions (e.g., anticoagulant use, coagulopathies, congestive heart failure, connective tissue disease, drug side effects, endocrine disease, infections, inflammatory bowel disease, malignancies, mediastinitis, postmyocardial infarction syndrome, postpericardiotomy syndrome, radiation therapy, and uremia).[21,26-38] Cardiac tamponade can be a complication of procedures (e.g., cardiac surgery, coronary intervention, electrophysiologic study with intracardiac electrodes, myocardial biopsy, pacemaker lead extraction, radiofrequency ablation, and transvenous pacemaker placement).[21,31,39-42] Cardiac tamponade may be sudden and painful due to hemopericardium from myocardial free wall rupture after myocardial infarction, posttraumatic aortic dissection, rupture of the ascending aorta, or spontaneous aortic dissection.[20,34,43-45]

TABLE 48-1 The Etiologies and Relative Frequencies of Pericardial Effusions

Etiology	Relative frequency (%)
Cancer	15–40
Connective tissue diseases	2–11
Idiopathic	13–14
Infectious (including HIV)	2–14
Postpericardiotomy	2–16
Radiation therapy	4–7
Trauma	7–9
Uremia	5–10

Source: Adapted from references 12–18.

Sauer and Murdock describe a "danger zone" for penetrating torso trauma (**Figure 48-3**).[46] The superior border is bounded by a line through the sternal notch. The lateral borders are bound by the midclavicular lines. The inferior border is identified by a line through the epigastric area. Any penetrating injury involving the "danger zone" has the potential to cause a cardiac injury and cardiac tamponade.

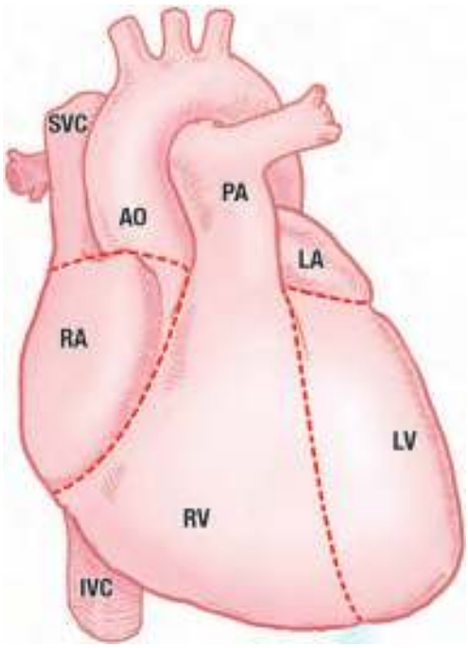


FIGURE 48-2. View of the heart and great vessels that can become injured behind the anterior chest wall. AO, aorta; IVC, inferior vena cava; LA, left atrium; LV, left ventricle; PA, pulmonary artery; RA, right atrium; RV, right ventricle; SVC, superior vena cava.

PATIENT EVALUATION

Several clinical findings are associated with cardiac tamponade. **Beck's triad of muffled heart sounds, hypotension, and jugular venous distention is associated with cardiac tamponade**. **The minority of patients with cardiac tamponade will have all three of the Beck's triad conditions.** Most patients with cardiac tamponade may have at least one of the findings in Beck's triad. Restlessness, fatigue, tachycardia, and tachypnea are often present in cardiac tamponade. **Cardiac tamponade must always be considered as a potential cause of shock in the hypotensive patient. Findings may be subtle or absent if the patient is hypovolemic.** Cardiac tamponade may progress to shock, coma, and death. The differential diagnosis includes acute myocardial infarction, cardiac shock, constrictive pericarditis, hypothermia, pneumothorax, and pulmonary embolism.

The systolic blood pressure (SBP) lowers with inspiration compared to expiration with the difference of less than 10 mmHg in normal physiology. **Pulsus paradoxus is a drop in SBP of ≥ 10 mmHg during inspiration compared to expiration.** Pulsus paradoxus can be present in conditions that limit heart filling (e.g., accumulation of pericardial fluid, constrictive pericarditis, and increased intrathoracic pressures from lung disease). Inflate the blood pressure (BP) cuff until the cuff pressure is greater than the patent's SBP and then slowly release the cuff pressure while auscultating until heart beats are heard only during expiration. Continue deflating the cuff pressure until heart beats are heard continuously in both inspiration and expiration. **Pulsus paradoxus is the difference between these two cuff pressures (Figure 48-4).**[47]

Kussmaul's sign is the paradoxical increase of the jugular venous pressure (JVP) during inspiration instead of the normal decrease in JVP with inspiration. This usually indicates impaired right heart filling. It may be due to myocardial impairment, pericardial fluid, or restriction of the pericardium.

Traumatic cardiac tamponade can be caused by a variety of agents and etiologies. This includes central venous line placement, displaced fractured ribs, intracardiac injection, migrating pins or needles, pacemaker insertion, penetrating thoracic injuries, pericardiocentesis, surgery, and venous bullet embolization. **Cardiac tamponade is the most common presentation of penetrating cardiac**

TABLE 48-2 Structures Vulnerable to Injury Behind the Anterior Chest Wall

Anatomic structure	%*
Right ventricle	55
Left ventricle	20
Right atrium	10
Left atrium	1
Aorta and pulmonary artery	10
Inferior vena cava	4

*The percentages represent the surface area of each structure and the estimates of incidence of injury with cardiac trauma.[19-22]

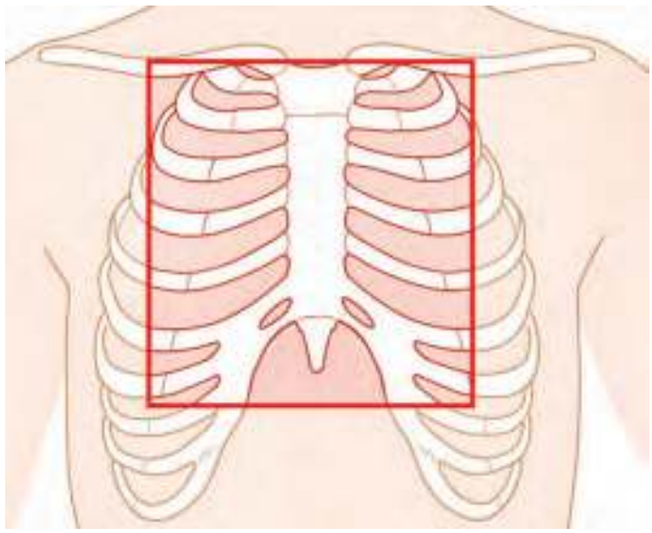


FIGURE 48-3. The "danger zone" for penetrating chest trauma.[46]

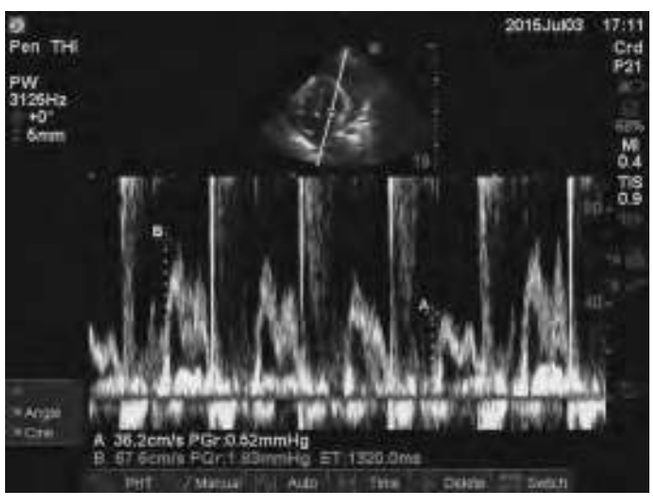


FIGURE 48-4. US apical four-chamber view with the Doppler sample gait placed at the tip of the mitral valve leaflets. Variation of the E wave is noted over the respiratory cycle with the lowest and highest velocities recorded as A and B, respectively. The difference between the two (36.2 cm/s and 67.6 cm/s) is 54%. Greater than 25% difference represents sonographic pulsus paradoxicus. (Photo used with permission from reference 47.)

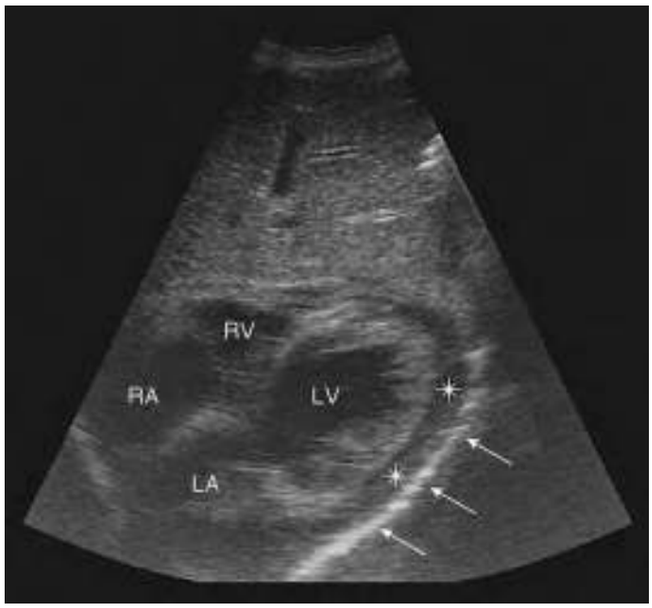


FIGURE 48-5. Subxiphoid ultrasound window demonstrating the four-chamber view (RV, right ventricle; LV, left ventricle; RA, right atrium; LA, left atrium) of a heart with a pericardial effusion (*asterisks*). The liver is noted anterior to the heart. The *arrows* point to the posterior pericardium.

FIGURE 48-6. Pericardial tamponade with right ventricular collapse (*arrowhead*). A large pericardial effusion (*asterisks*) is visible. *Arrows* point to the posterior pericardium. AO, aortic root; LA, left atrium; LV, left ventricle; RV, right ventricle.

injuries overall and must be considered in all trauma patients with penetrating trauma and hypotension.[22]

Bedside cardiac ultrasound (US) has become the diagnostic procedure of choice to identify cardiac tamponade.[47-49] Bedside US can rapidly confirm a suspected pericardial effusion or cardiac tamponade (**Figure 48-5**). US can be used to guide drainage.[48] Blind pericardiocentesis has a high complication rate and a high mortality rate.[15,16,50] US-guided pericardiocentesis can decrease the rate of complications by allowing visualization and avoidance of adjacent anatomic structures. This increased safety factor is supported by multiple studies.[46,50-60]

US features that suggest cardiac tamponade are noted in **Table 48-3**. This includes circumferential pericardial effusion (**Figure 48-6**), dilatation of the inferior vena cava (IVC) without respiratory variation (e.g., IVC that collapses > 50% with inspiration or a forced sniff maneuver), right atrial systolic collapse, and right ventricular diastolic collapse (**Figure 48-6**).

TABLE 48-3: US Findings of Cardiac Tamponade

Ultrasonographic mode	Findings
Doppler	Mitral flow decreases during inspiration
	Mitral flow increases during expiration
	Tricuspid flow increases during inspiration
	Tricuspid flow decreases during expiration
	Peripheral flow decreases in expiration
M-mode color Doppler	Mitral flow decreases during inspiration
	Mitral flow increases during expiration
	Tricuspid flow increases during inspiration
	Tricuspid flow decreases during expiration
M-mode/two-dimensional	Diastolic collapse of the right ventricular free wall
	Inferior vena cava dilation
	Inferior vena cava does not collapse on inspiration
	Increase left ventricular wall thickness in diastole
	Left atrial collapse
	Left ventricular collapse
	Right atrial collapse
	Swinging heart to and fro

Source: Adapted from reference 68.

Evaluate the IVC by placing the US probe in the subxiphoid region, with the probe marker pointing toward the patient's head. The IVC runs parallel with and to the right of the aorta. Use M-mode to measure the diameter of the IVC approximately 2 cm from the right atrium. The normal IVC ranges from 1.5 to 2.5 cm in diameter and displays respiratory variation. **The IVC is dilated, measures > 2.5 cm in diameter, and does not display respiratory variation in cardiac tamponade.** Some authors advocate using transesophageal echocardiography, even in unstable patients, because of its superior imaging when compared to transthoracic echocardiography.[18] Other methods of imaging the pericardium include computed tomography (CT), helical CT, and magnetic resonance imaging. **Use these for stable patients when assessing for a pericardial effusion and not cardiac tamponade.**[61]

Cardiac tamponade may not be identified by electrocardiogram (ECG) or plain radiographic findings. ECG findings are not diagnostic but may be present in patients with a significant pericardial effusion or cardiac tamponade. A large pericardial effusion can result in a "low-voltage" ECG tracing. **Electrical alternans is a change in the morphology or amplitude of the QRS complexes on the ECG as the heart swings back and forth within the pericardial fluid (Figure 48-7).**[62,63] It may be associated with either cardiac tamponade or a large pericardial effusion. Pulseless electrical activity (PEA) in the absence of hypovolemia or a tension pneumothorax is highly suggestive of cardiac tamponade. The finding of an enlarged cardiac silhouette on a chest radiograph may be useful in chronic pericardial effusions but is usually absent or nonspecific in the acute setting.

INDICATIONS

The only indication for emergent pericardiocentesis is the presence of life-threatening physiologic changes of cardiac tamponade, and the diagnosis is consistent with known prior disease, traumatic injury, or an US.[64-66] A pericardiocentesis can be used in penetrating trauma to "buy time" until definitive surgical care.[66,67] Use bedside US if available to demonstrate a pericardial effusion and/or cardiac tamponade. A pericardiocentesis may be performed in a cardiac arrest patient with PEA when other etiologies for PEA have been

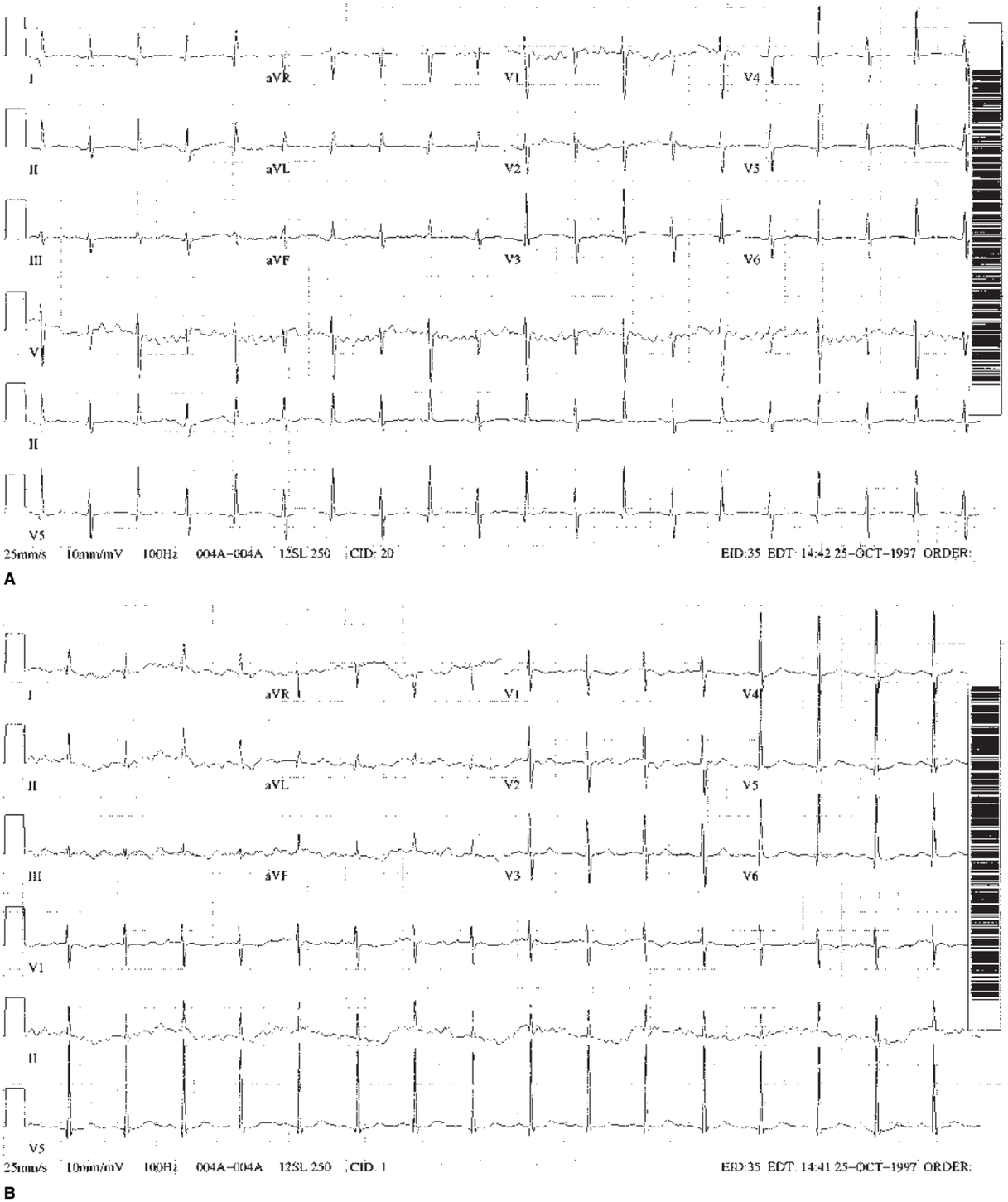


FIGURE 48-7. ECG of electrical alternans. ***A.*** Initial ECG showing electrical alternans. Bedside US revealed a large pericardial effusion with right ventricular diastolic collapse. ***B.*** Resolution of electrical alternans after pericardiocentesis.

ruled out or a pericardial effusion is seen on US. Pericardiocentesis may be performed to obtain pericardial fluid for diagnostic testing.

CONTRAINDICATIONS

There are no absolute contraindications to performing an emergent pericardiocentesis in the unstable patient with signs of cardiac tamponade. Any uncorrected anticoagulation from medications or a bleeding disorder will be an absolute contraindication to performing the procedure in a stable patient. Small, loculated, or posteriorly located effusions in a stable patient are considered contraindications. Pericardiocentesis is contraindicated in cardiac tamponade associated with myocardial free wall rupture after myocardial infarction, spontaneous aortic dissection, posttraumatic aortic dissection, or rupture of the ascending aorta.[68] These are surgical emergencies that require prompt diagnosis and surgery. There is conflicting research

regarding whether these should be treated by needle pericardiocentesis initially. Some reports note the pressure of the intrapericardial blood opposes further bleeding. The removal of pericardial fluid reduces the pressure and may cause further bleeding to occur, often with fatal results.[20,68] Other research reports successful management of type A aortic dissection by pericardial drainage.[58]

Many authors feel there is little to no role for pericardiocentesis in the trauma patient. These authors argue that once the diagnosis is made, the patient should receive a prompt thoracotomy.[69] An Emergency Department thoracotomy (Chapter 54) should be performed if the patient is too unstable for transport to the Operating Room for a formal thoracotomy.

EQUIPMENT

PERICARDIOCENTESIS

- Povidone iodine or chlorhexidine solution
- Sterile gloves and gown
- Face mask with eye shield or goggles
- 1% lidocaine solution
- 25 gauge needle, 5/8 inch long
- 18 gauge needle, 1½ inches long
- Syringes (10, 20, and 60 mL)
- Sterile drapes
- Towel clips
- 16 to 18 gauge spinal needle or catheter-over-the-needle, 7.5 to 12.5 cm long
- 18 to 20 gauge spinal needle or catheter-over-the-needle, 3.75 cm long
- #11 scalpel blade
- 4 × 4 gauze squares
- Alligator clips connected by a wire
- Collection basin
- ECG monitor
- J-tipped guidewire, 0.035 mm in diameter
- Size 6 to 10 French flexible multihole catheter, 5 to 6 inches long, with or without a pigtail
- Three-way stopcock
- Plastic tubing
- US machine
- 3.5 to 5.0 MHz phased-array or curvilinear US transducer
- Sterile US transducer cover (can be a sterile glove)
- Sterile US gel
- Variable-angle needle guide attachment, if available
- Nasogastric tube

SUBXIPHOID PERICARDIAL WINDOW

- Electrocautery set
- Forceps
- Small retractor
- Small rib spreaders
- Sutures, 2–0 Vicryl and 3–0 nylon
- Sterile suction device
- Yankauer suction catheter
- Suction tubing

It is important to become familiar with the supplies available in the Emergency Department. Some Emergency Departments may not have single guidewires and flexible catheters readily available to use for a pericardiocentesis. Commercially produced pericardiocentesis kits are available from numerous manufacturers and contain the required equipment (**Figure 48-8**). A 6 to 10 French single-lumen central venous line access kit may be substituted in an emergency.

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative if the patient's clinical condition permits. Place the signed consent in the medical record. A signed consent is not mandatory if this is an emergent procedure. Document the reason for not obtaining written consent in the medical record. Position the patient semirecumbent at a 30° to 45° angle (**Figure 48-9**) if no contraindications exist. This position brings the heart closer to the anterior chest wall. The supine position is an acceptable alternative.

A

B

FIGURE 48-8. Some of the commercially available pericardiocentesis kits. ***A.*** Cook Medical, Bloomington, IN. ***B.*** Boston Scientific, Marlborough, MA.

FIGURE 48-9. Ideal patient positioning for performing a pericardiocentesis.

Apply cardiac and BP monitoring, pulse oximetry, and supplemental oxygen. The placement of an arterial line may be helpful for additional monitoring (Chapter 72). An arterial line is not necessary and should not delay the pericardiocentesis. **Insert a nasogastric tube to decompress the stomach and decrease the possibility of gastric perforation by the pericardiocentesis needle (Chapter 75).**

Identify the anatomic landmarks necessary to perform this procedure. The needle can be inserted at numerous sites (**Figure 48-10**). These include below the xiphoid process, at the right sternocostal margin, at the left sternocostal margin (i.e., subxiphoid approach), in the left or right fifth intercostal space parasternally (i.e., parasternal approach), or in the left fifth intercostal space at the midclavicular line (i.e., apical approach). The most commonly used site is at the left sternocostal margin (i.e., subxiphoid approach) if bedside US is not available. This is the approach described throughout the "Techniques" section of this chapter.

Surgically prepare the parasternal and subxiphoid areas. Clean any blood, dirt, and debris from the area. Apply povidone iodine or chlorhexidine solution and allow it to dry. Apply sterile drapes to delineate a sterile surgical field. Reidentify the anatomic landmarks and confirm them by bedside US if available. Anesthetize the needle tract with local anesthetic solution if the patient is awake. Place a subcutaneous wheal of local anesthetic solution at the site chosen to insert the needle. Inject local anesthetic solution through the skin wheal and into the subcutaneous and deeper tissues of the thoracic wall.

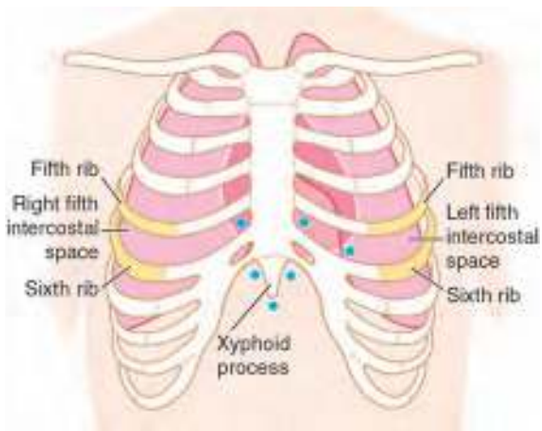


FIGURE 48-10. Potential sites to perform a pericardiocentesis.

Aseptic technique must be followed even though this is an emergent procedure. Don full personal protective equipment (i.e., hat, mask, sterile gown, and sterile gloves). Prepare the equipment. Set up a sterile field on a bedside table. Open all required equipment and place it on the sterile field. Connect the spinal needle to a 20 mL syringe containing 5 mL of sterile saline. Prepare all the supplies prior to prepping the patient. Attach a three-way stopcock between the syringe and spinal needle. The stopcock will facilitate the removal of fluid. Attach intravenous extension tubing to the stopcock.

TECHNIQUES

BLIND INSERTION TECHNIQUE

Puncture the skin with a #11 scalpel blade between the xiphoid process and the left costal margin. Grasp the syringe with the dominant hand. Insert the spinal needle through the skin incision and at a 45° angle to the midsagittal plane (**Figure 48-11*A***) and at a 45° angle to the abdominal wall (**Figure 48-11*B***). Aim the tip of the spinal needle toward the patient's left shoulder (**Figure 48-11*A***). The spinal needle can be alternatively aimed toward the patient's left mid-clavicle, right mid-clavicle, or sternal notch to theoretically lessen the chance of iatrogenic damage to the coronary arteries.

Advance the spinal needle 4 to 5 cm while applying negative pressure to the syringe and observing the cardiac monitor. Inject 0.25 to 0.50 mL of saline occasionally to ensure that the needle remains patent while advancing. Continue advancing the spinal needle while applying negative pressure until there is a return of blood, cardiac pulsations are felt, or there is an abrupt change in the ECG waveform. **Stop advancing the needle and withdraw it in 1 to 2 mm increments until the ECG pattern normalizes if the ECG waveform shows an injury pattern (i.e., ST-segment elevation or ventricular arrhythmia).** ECG changes indicate that the needle is touching or has penetrated the myocardium.

Aspirate with the syringe. A large volume of blood that is quickly and easily withdrawn may indicate that the tip of the spinal needle is within the ventricle. Slowly withdraw the needle while aspirating if there is a suspicion that the needle has entered the ventricle and bedside US is not available. The aspiration of free-flowing blood will stop as the needle passes through the ventricular wall. Aspirate the pericardial fluid when the needle enters the pericardial space.

Techniques to confirm the intraventricular placement of the needle tip have been described. These include observing that the aspirate does not form a clot, comparing the venous hemoglobin to that of the aspirate, injecting fluorescein and looking for a fluorescein flush under the eyelid skin, or injecting 3 mL of dehydrocholic acid (i.e., Decholin) and asking whether the patient experiences a bitter taste.[70] These are time consuming and less reliable than US, if available.

There will be a marked improvement in hemodynamics and clinical status when the pericardial space is entered and fluid is aspirated. The procedure can be terminated once the patient's hemodynamics have improved.

The Emergency Physician may choose to withdraw as much fluid as possible. Stop withdrawing the plunger when the syringe is filled with fluid. Stabilize the spinal needle against the patient's torso and remove the syringe. Attach a new syringe to the spinal needle and continue the procedure. The epicardium will approach the needle tip as the pericardial space is drained. Slowly withdraw the needle 2 to 3 mm if an injury pattern appears on the cardiac monitor. It will return to baseline. Continue to aspirate fluid. Remove the needle when fluid can no longer be aspirated.

Alternatively, attach a three-way stopcock between the spinal needle and the syringe. Attach intravenous extension tubing to the

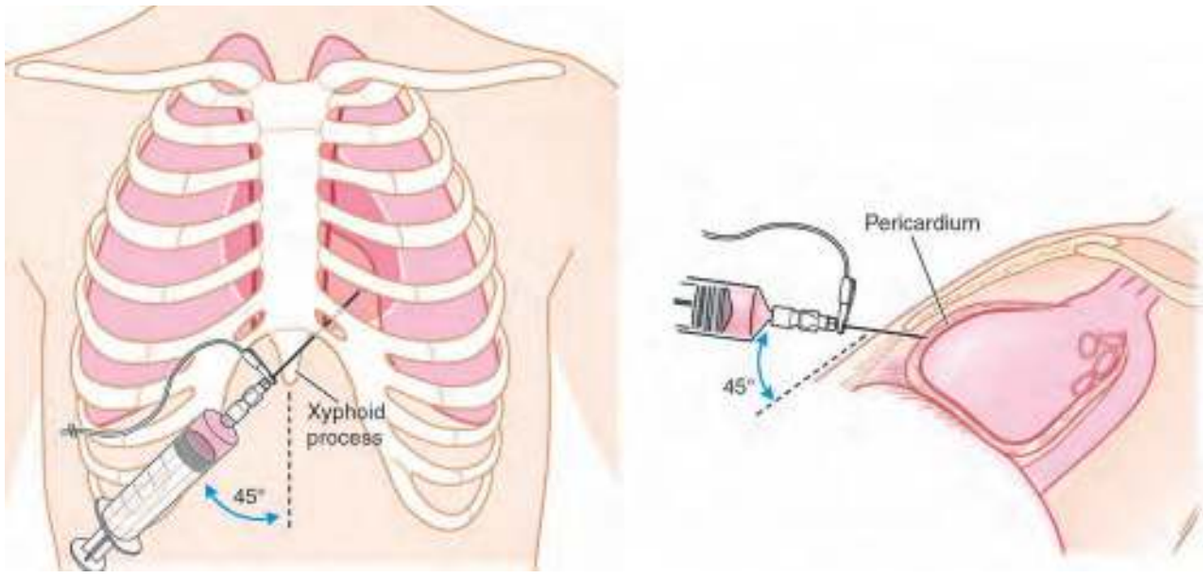


FIGURE 48-11. The subxiphoid approach. The needle is inserted at a 45° angle to the midsagittal plane (***A***) and at a 45° angle to the abdominal wall (***B***). The alligator clip attached to the spinal needle is for the ECG-monitored technique and not the blind technique.

stopcock. An assistant can open and close the stopcock while the Emergency Physician aspirates fluid and ejects it through the extension tubing into a basin. The epicardium will approach the needle tip as the pericardial space is drained. Slowly withdraw the needle 2 to 3 mm if an injury pattern appears on the cardiac monitor. It will return to baseline. Continue to aspirate fluid. Remove the needle when fluid can no longer be aspirated.

ECG-MONITORED TECHNIQUE

The purpose of ECG monitoring is to prevent accidental ventricular puncture with the spinal needle. It is not necessary with the US-guided technique. Attach one alligator clip to the base of the spinal needle and the other to the V_1 lead of the ECG machine or cardiac monitor (**Figure 48-12**). The V_1 lead will serve as an active electrode based at the tip of the spinal needle. An injury pattern noted by ST-segment elevation will be seen if the myocardium is contacted or penetrated by the spinal needle. The presence of a premature ventricular contraction or a ventricular arrhythmia can signify contact with the myocardium.

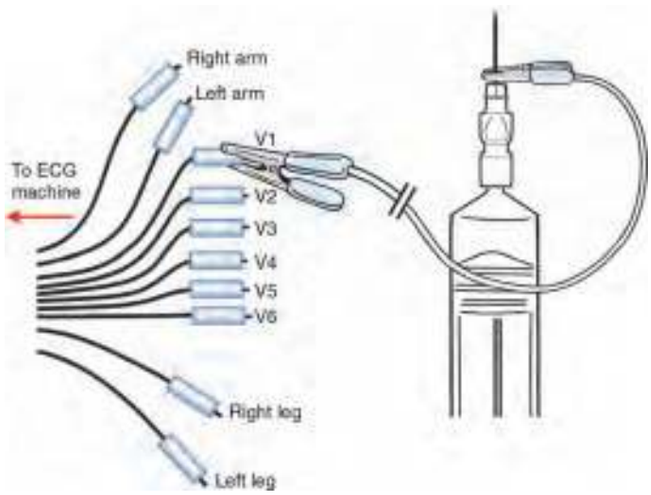


FIGURE 48-12. Equipment preparation for the ECG-monitored technique.

Prepare the patient as previously described. Prepare the equipment (**Figure 48-12**). Turn on the ECG machine or cardiac monitor. Insert and advance the spinal needle, as described previously, while observing the ECG monitor or cardiac monitor. Slowly withdraw the needle in 1 to 2 mm increments if an injury pattern or premature ventricular complexes are seen. Continue to withdraw the needle in 1 to 2 mm increments until the injury pattern resolves. Aspirate the pericardial fluid as described in the preceding section.

SELDINGER TECHNIQUE

An indwelling catheter may be placed in the pericardial cavity to drain the pericardial fluid (**Figure 48-13**). This may be done in cases of medical or traumatic pericardial effusions since the pericardial fluid often reaccumulates. An indwelling catheter allows intermittent drainage of pericardial fluid without the potential complications associated with repeated needle sticks from a pericardiocentesis. This procedure can "buy time" if an Operating Room and/or Surgeon is not immediately available to perform a pericardial window.[66]

The technique is similar to that of placing an indwelling central venous line. Clean and prepare the patient. Insert the spinal needle by US guidance, blindly, or with ECG monitoring as described in the previous sections (**Figure 48-11**). Aspirate to confirm that the tip of the spinal needle is within the pericardial cavity (**Figure 48-13*A***). **It is imperative that the tip of the spinal needle be within the pericardial cavity and not within the cardiac chamber. The position of the needle must be verified by one of the methods described in the section on "Blind Insertion Technique," by fluoroscopy, or by US if intracardiac placement of the needle is suspected.**

Grasp and stabilize the spinal needle with the nondominant hand. Gently remove the syringe from the spinal needle with the dominant hand. Insert the guidewire through the needle and into the pericardial cavity (**Figure 48-13*B***). Advance the guidewire until approximately one-third of its length is within the patient. Stabilize the guidewire with the nondominant hand. Remove the needle over

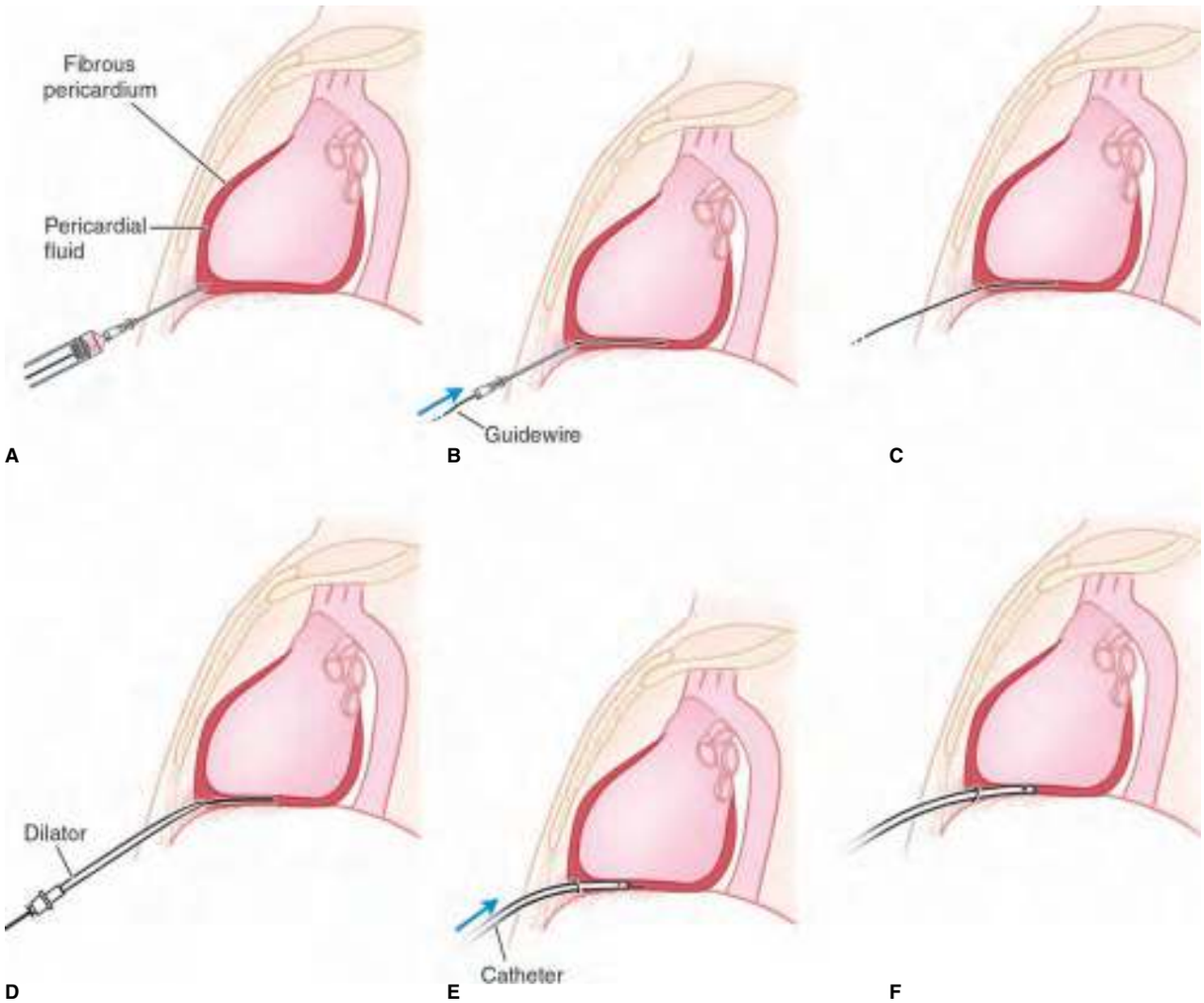


FIGURE 48-13. The Seldinger technique. ***A.*** The spinal needle is inserted into the pericardial space. ***B.*** The guidewire is inserted through the needle. ***C.*** The needle has been removed and the guidewire remains within the pericardial cavity. ***D.*** The dilator is advanced over the guidewire to dilate the needle tract. ***E.*** The catheter is advanced over the guidewire. ***F.*** The guidewire has been removed and the catheter remains within the pericardial cavity.

the guidewire while leaving the guidewire within the pericardial cavity (**Figure 48-13*C***).

Continue to stabilize the guidewire with the nondominant hand. Advance the dilator over the guidewire and into the pericardial cavity (**Figure 48-13*D***). **Dilating a tract through the myocardium can result in cardiac tamponade and/or exsanguination if the guidewire is within the heart. It is imperative to know that the guidewire is within the pericardial cavity and not within the heart**. Remove the dilator while leaving the guidewire within the pericardial cavity. Advance the soft multihole catheter over the guidewire and into the pericardial cavity (**Figure 48-13*E***). Remove the guidewire while leaving the catheter within the pericardial cavity (**Figure 48-13*F***). Secure the catheter at the skin with the nondominant hand. Attach a syringe to the catheter and aspirate pericardial fluid. This should cause a rapid improvement in the patient's clinical status. Detach the syringe and attach a three-way stopcock to the catheter.[23] Secure the catheter to the skin with nylon sutures.

US-GUIDED TECHNIQUE

Many authors feel that US-guided pericardiocentesis is now the standard of care.[14,23,61,71-73] The heart is best scanned with the patient in a semierect or left lateral position if no contraindications exist. Use the 3.5 to 5.0 MHz phased array or curvilinear US transducer. Examine the heart and pericardial space in two-dimensional and Doppler mode to determine the extent of the pericardial effusion and the area with the largest fluid collection.[74,75] This is usually located around the apex of the heart.

View the heart and pericardial space using the three standard cardiac US views. These are the subxiphoid or subcostal view, the parasternal long axis view, and the apical four-chamber view (Chapter 37). Pericardial fluid is anechoic, appears black on US, and will collect in a dependent location if adhesions are not present. Small pericardial effusions will first be seen in the posterior pericardium. Large pericardial effusions appear circumferential and extend around the heart.

The decision to use a parasternal (**Figure 48-14**) or an apical (**Figure 48-15**) window depends on which US views of the pericardial effusion and heart can be obtained. **The ideal site is where the pericardial effusion is most superficial, has a large stripe or thickness, and where no other structures (e.g., lung or liver) are in the path from the skin to the heart.**[60,76] **The subxiphoid window (Figures 48-5 and 48-16) is not recommended for US-guided pericardiocentesis.** This view commonly includes the left lobe of

FIGURE 48-14. Phased-array probe placed in the left parasternal window adjacent to the sternum, between the second and fourth interspace. The probe marker is pointing to the patient's left hip.

FIGURE 48-16. A curvilinear probe placed in the subxiphoid region. The probe is aimed toward the patient's left shoulder. The probe marker is pointing to the patient's right. Note the shallow angle required to visualize the heart.

the liver in the anterior portion of the US image. The pericardiocentesis needle might puncture the liver on the way into the pericardial cavity.

There are established US findings that help to distinguish a pericardial effusion from a cardiac tamponade (**Figure 48-6 and Table 48-3**).[68] These include right ventricular diastolic collapse (**Figure 48-6**), right atrial systolic collapse, large pericardial effusion, and dilatation of the IVC. Place the US transducer in the subxiphoid region with the transducer marker pointing toward the patient's head. The IVC will be seen running parallel to the aorta and to the right of the aorta. Use M-mode to measure the diameter of the IVC. The normal IVC ranges from 1.5 to 2.5 cm in diameter and displays respiratory variation (**Figure 48-17*A***).[77,78] The IVC is dilated, measures > 2.5 cm in diameter, and does not display respiratory variation (**Figure 48-17*B***).[77,78]

Position the patient. Determine the location to perform the pericardiocentesis. Clean, prep, and drape the patient as described

A

B

FIGURE 48-17. IVC variation. ***A.*** Normal IVC collapse during respiration marked with the bars. ***B.*** No IVC collapse during respiration (bars) signifies high pressure transmitted from the heart. (Photos courtesy of Geoffrey E. Hayden, MD, from www.emergencyultrasoundteaching.com.)

FIGURE 48-15. A phased-array probe placed in the apical window at the point of maximal impulse (PMI). The probe marker is pointing to the patient's right.

FIGURE 48-18. Ultrasound probe and the pericardiocentesis needle in the apical window.

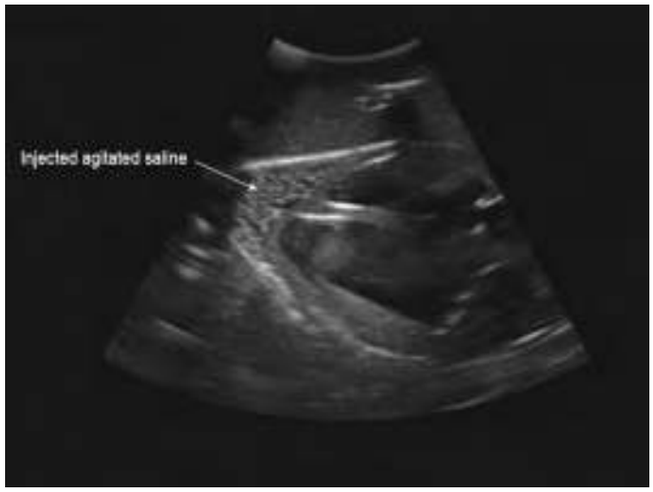


FIGURE 48-20. US image of agitated saline in the pericardial sac.

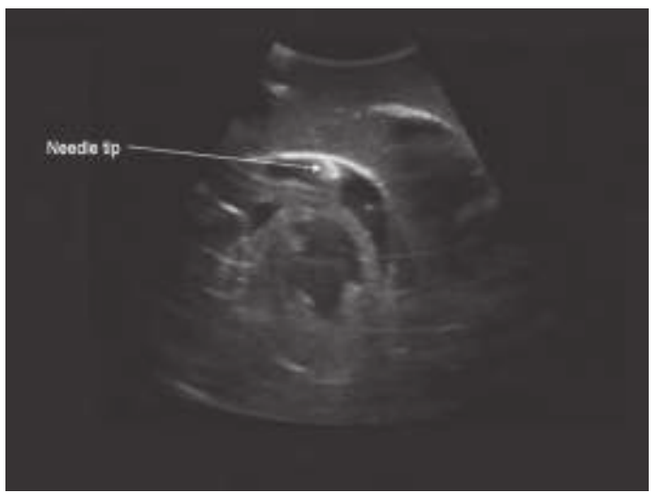
previously. Apply a sterile transducer cover. Apply sterile US gel over the covered transducer. Reidentify and confirm the proper US transducer position.[76] This is usually the point where the pericardial effusion is closest to the chest wall.[74,75] Insert the spinal needle under US guidance into the chest wall (**Figure 48-18**). Insert the needle over the superior border of the rib to avoid the neurovascular intercostal bundle under the inferior edge of the rib. Aspirate as the needle tip is carefully advanced under direct US visualization into the pericardial space (**Figure 48-19**).

Use agitated saline to confirm proper needle placement if there is any question as to whether the needle tip is in the pericardial space or if bloody fluid is aspirated (**Figure 48-20**).[74-76,79,80] This requires two 5 mL syringes, one filled with sterile saline and the other filled with air. Attach the syringes to a three-way stopcock. Rapidly move the sterile saline back and forth between the two syringes and create an aerated saline solution. Rapidly inject the aerated solution through the spinal needle. The identification of hyperechoic "bubbles" on the US image confirms the location of the needle (**Figure 48-20**). An alternative to this technique is to use color Doppler to localize the needle tip.[81,82]

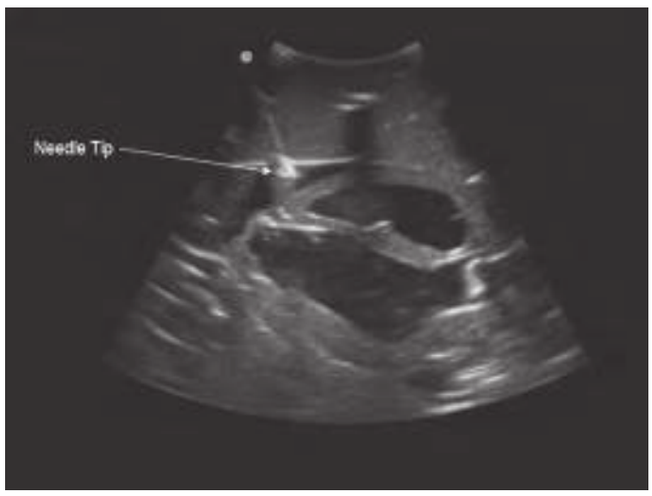
SUBXIPHOID PERICARDIAL WINDOW

The Emergency Physician may decide to place a pericardial window in very rare situations.[83-85] **This procedure should only be performed by an Emergency Physician trained and skilled in this procedure.** A pericardial window will minimize false-negative results seen with cardiac tamponade from myocardial injury by the pericardiocentesis needle, iatrogenic bleeding, and a pericardiocentesis. Prepare the patient as previously described. Inject local anesthetic solution subcutaneously from the xiphoid process across the confluence of the lower ribs and 6 cm down the midline (**Figure 48-21*A***). Inject local anesthetic solution into the muscular layers in the midline over the xiphoid process and continue approximately 8 cm inferiorly.

Make a midline longitudinal incision from the xiphisternal junction to about 8 cm below the tip of the xiphoid process (**Figure 48-21*A***). Incise down to the linea alba. Bluntly dissect the space behind the xiphoid and lower sternum to separate the anterior diaphragm from the sternum. Lift the lower sternum with a retractor (**Figure 48-21*B***). Use an electrocautery unit, if available, for



A



B

FIGURE 48-19. Two ultrasound views of the needle entering the pericardial effusion.

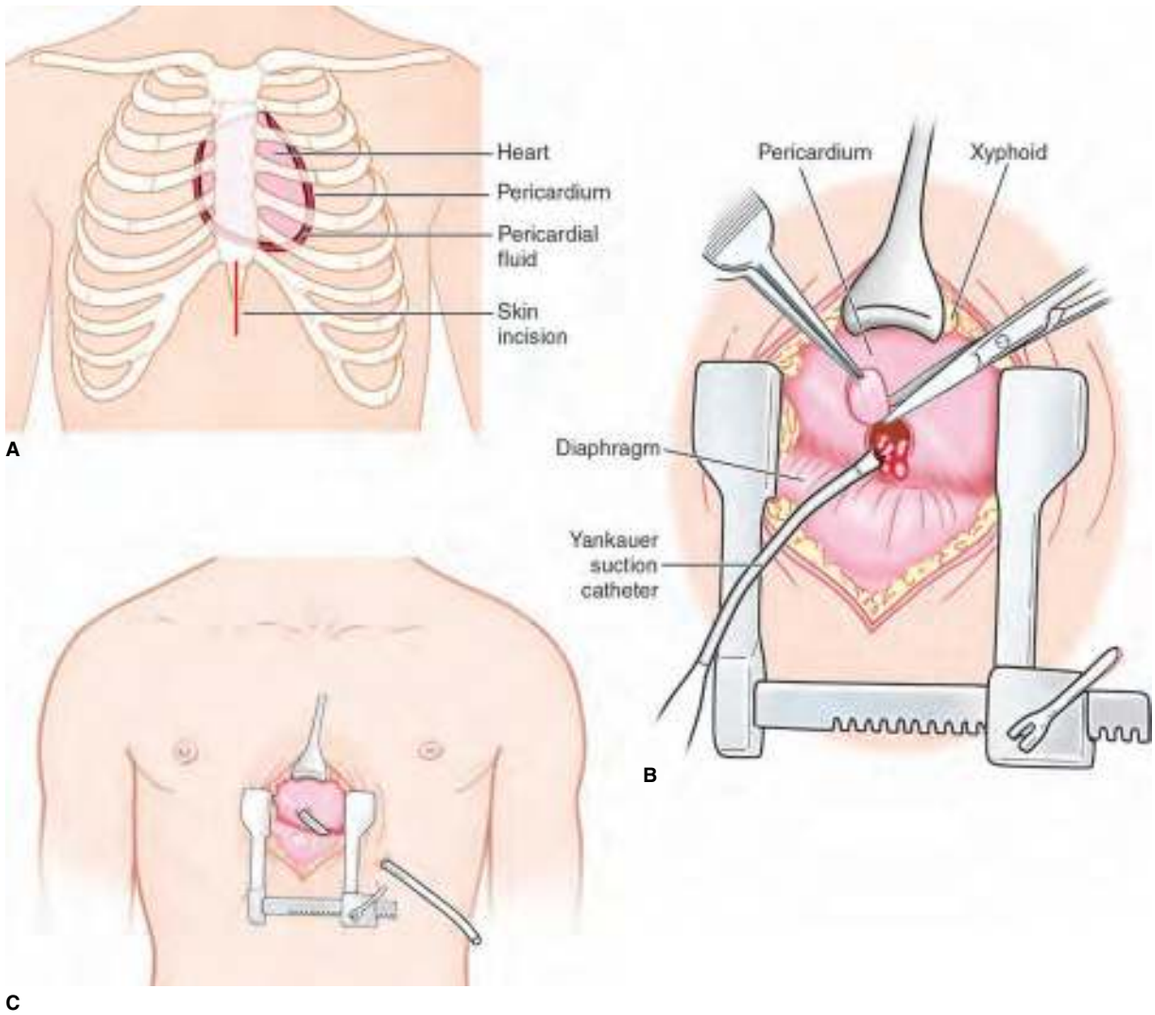


FIGURE 48-21. The pericardial window. ***A.*** The site of the skin incision. ***B.*** The xiphoid and sternum are lifted upward to expose the pericardium. An incision is made and a piece of the pericardium is removed. ***C.*** A chest tube is inserted into the pericardial space to allow continuous drainage of fluid.

hemostasis. Bluntly divide the fatty tissue and retrosternal attachments of the diaphragm to reveal the pericardium beneath the angle of the xiphoid and the left costal margin.[86] It will often appear blue in color due to underlying blood.

Grasp the pericardium with forceps. Incise the pericardium with a scissors or with shallow strokes of a scalpel (**Figure 48-21*B***). Fluid should rush out rapidly. Remove a piece of the pericardium to make sure that it remains open (**Figure 48-21*B***). Gently explore the pericardial space digitally and with the suction catheter to remove any clot and fluid. Allow the skin to stay open and permit the free drainage of the pericardial space.

Alternatively, place a 28 French chest tube in the pericardial space and secure it with a purse-string suture through the fibrous pericardium (**Figure 48-21*C***). The chest tube may exit the skin incision or a separate incision in the skin. Attach the chest tube to a suction source. Close the linea alba with interrupted 2–0 Vicryl sutures. Close the subcutaneous tissue and skin with 3–0 nylon sutures.

ALTERNATIVE TECHNIQUES

CATHETER-OVER-THE-NEEDLE TECHNIQUE

A long catheter-over-the-needle (i.e., angiocatheter) mounted on a syringe may be used instead of a spinal needle mounted on a syringe. Use a 16 to 18 gauge, 7.5 cm or longer catheter-over-the-needle for older children, adolescents, and adults. Use an 18 to 20 gauge, 3.75 cm catheter-over-the-needle for infants and small children. The procedure is the same as using a spinal needle up to the point of the needle entering the pericardial space. Stop advancing the catheter-over-the-needle once the pericardial space is entered. Securely hold the syringe so it does not move inward or pull outward. Advance the catheter over the needle and into the pericardial cavity. Continue to advance the catheter until its hub is against the skin. Securely hold the catheter hub against the skin. Remove the syringe and needle as a unit. Attach a 20 mL syringe or a stopcock to the catheter hub and aspirate. The remainder of the procedure is as described previously.

ALTERNATIVE APPROACHES

The subxiphoid approach is the classic or traditional approach and has been described previously. The use of US is changing the technique to a more apical approach.[59,79] This area tends to be closer to the anterior chest wall and where the pericardial effusion is the largest. The use of US makes the procedure easier to perform.

The blind technique can be performed using the parasternal, intercostal, or periapical approaches. It was previously recommended to insert the needle perpendicular to the skin and 3 to 4 cm lateral to the sternum for the parasternal approach to avoid the

internal mammary artery. Needle insertion just lateral to the sternal border will avoid the internal mammary artery.[87] Do not use the intercostal approach blindly as it increases the risk of lung penetration by the needle and a subsequent pneumothorax. A blind apical approach is not recommended. The lingula of the left lung and the pleural space are close to this site. Penetration of the lingula can result in a pneumothorax.

ASSESSMENT

The lack of blood return does not rule out the diagnosis of cardiac tamponade. False-negative aspirations from a pericardiocentesis are well documented and are described at rates as high as 80%.[88] The occurrence of false-negative aspirations is often due to clotted blood in the pericardial space that cannot be aspirated or from failure to enter the pericardial space.

A dramatic improvement in the patient's clinical status should be observed after the successful drainage of the pericardial space. The patient's blood pressure and cardiac output should increase while intracardiac pressure and intrapericardial pressure decrease. Obtain a chest radiograph after the procedure to rule out a hemothorax and/or pneumothorax. Obtain an ECG to look for changes consistent with coronary artery injury from the needle.

A pericardiocentesis is often performed in the nontraumatic medical patient. Send the pericardial fluid in these cases for chemical, cytologic, and microbial analyses to determine the etiology of the pericardial effusion. Laboratory analysis should include the appearance of the fluid, a cell count and differential, glucose level, lactate dehydrogenase (LDH) level, pH, and total protein level. Cytology may reveal a malignant etiology of the effusion. The microbiology lab should perform an acid-fast stain, aerobic and anaerobic bacterial cultures, fungal cultures, Gram stain, and viral cultures.

AFTERCARE

If a catheter was placed in the pericardial space, secure it with sutures to the skin and check for stability. Consult a Surgeon for definitive care of trauma patients if not already done. Prepare these patients for rapid transport to the Operating Room. Monitor patients for reaccumulation of pericardial fluid and for hemodynamic instability. Repeat the procedure or open the stopcock if placed to reaspirate the pericardial space if fluid reaccumulates. Flush with sterile saline after each aspiration to maintain the patency of the catheter. Consult a Thoracic Surgeon if purulent fluid is aspirated in medical patients. Admit all patients to an Intensive Care Unit for further monitoring, evaluation, and treatment.

COMPLICATIONS

Blind pericardiocentesis complication rates vary from 4% to 40%.[14,52,89] The complication rates of US-guided aspirations have been reported to be less than 5%.[14] Reaccumulation of pericardial fluid may occur in up to 70% of blind aspirations.[11] Continuous drainage with a pericardial catheter can reduce this to 25%.[11] Potential complications include bleeding, decline or death as a result from recurrence or occurrence of tamponade, dysrhythmias, hemoperitoneum, hemothorax, injury (e.g., myocardium, coronary arteries, pericardial vasculature, internal mammary vessels, liver, lung, or gastrointestinal tract), and/or pneumothorax by the spinal needle.[90] **Do not rock the spinal needle or change its direction once it is inserted into the patient to minimize injury.** Penetration of the myocardium can result in asystole, dysrhythmias, ventricular fibrillation, or ventricular tachycardia. False-negative aspirations (i.e., a dry tap) occur if the blood in the pericardial cavity is clotted or if the needle is not within the pericardial cavity. False-positive aspirations may be seen if the needle is within the heart chamber or a vascular structure. Hepatic damage may lead to bile leakage or blood in the abdominal cavity. A vasovagal reaction can occur if the patient is awake.

Air can be introduced into the pericardium.[42,91,92] This is especially true if a catheter is left in the pericardium. This is a rare complication from pericardiocentesis. The etiology of the air is the creation of a communication from the pleura or air leakage around the catheter. This is visible as air in the pericardium on US. Cardiac tamponade from air can occur if enough air enters the pericardial cavity.

Pericardiocentesis can rarely result in paradoxical hemodynamic instability or the pericardial decompression syndrome.[93-95] The patient develops cardiac shock after the pericardiocentesis. The mortality is high for this complication. The etiology is hypothesized but unknown. The drainage of pericardial fluid relieves the pressure on the right ventricle, leads to an increased preload, and combines with increased systemic vascular resistance to cause left ventricular overload. Another theory is the myocardial stunning and hibernation due to the pericardial fluid causing increased pressure and collapse of the coronary arteries. This results in biventricular failure or cardiogenic shock with either etiology.

SUMMARY

Pericardiocentesis is an infrequently performed procedure. It can be lifesaving when a patient has a cardiac tamponade. The procedure is relatively simple yet has a significant rate of complications, morbidity, and mortality. The use of bedside US to assist in making the diagnosis and in guiding the placement of the pericardiocentesis needle dramatically reduces false diagnoses and complications. The exact role of pericardiocentesis in trauma remains controversial. It may be lifesaving in the unstable patient before they are able to receive definitive surgical therapy.

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49 Intracardiac Injection

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INTRODUCTION

The practice of intracardiac injection originated in the 1800s and had been strongly advocated for years before it began to fall out of favor.[1-3] It was commonly performed throughout the 1960s, as it was thought to be the most expeditious route of drug delivery during a cardiac arrest.[4,5] By the mid-1970s, the practice of intracardiac injection declined. Safer and simpler routes of medication administration (i.e., intravenous, endotracheal, and intraosseous) became available. Experimental data suggested that there was no advantage to intracardiac injection over intravenous administration of medications.[6] Cardiopulmonary resuscitation (CPR) must be interrupted to perform an intracardiac injection. The time required for this procedure may be prolonged in difficult patients or in inexperienced hands. Many serious complications may occur from an intracardiac injection.[5] Intracardiac injection should be considered when no other access is readily available.[7]

ANATOMY AND PATHOPHYSIOLOGY

The technique of an intracardiac injection is similar to a pericardiocentesis (Chapter 48). Both techniques use the same anatomic landmarks and approach and involve the transthoracic insertion of a needle through the pericardium. The tip of the needle is inserted into the pericardial space when performing a pericardiocentesis. Intracardiac injection requires the tip of the needle to be inserted directly through the myocardium and into a cardiac chamber.

Echocardiography or bedside ultrasound may be useful in a pericardiocentesis to avoid the lung or myocardium.[8] **However, time is of the essence when performing an intracardiac injection.** The objective is to quickly enter the myocardial cavity. Ultrasonographic guidance is generally not necessary. Certain comorbidities (e.g., chronic obstructive pulmonary disease [COPD], prior lung resection, or dextrocardia) may benefit from ultrasonographic guidance to avoid puncturing the lung.

The technique of intracardiac injection is rapid, simple to perform, and requires no special equipment. It begins with identification of the anatomic landmarks required to perform the procedure (**Figure 49-1**). For the subxiphoid approach, identify and palpate the xiphoid process of the sternum and the left costosternal angle. For the left parasternal approach, identify and palpate the left fourth or fifth intercostal spaces immediately adjacent to the sternum.

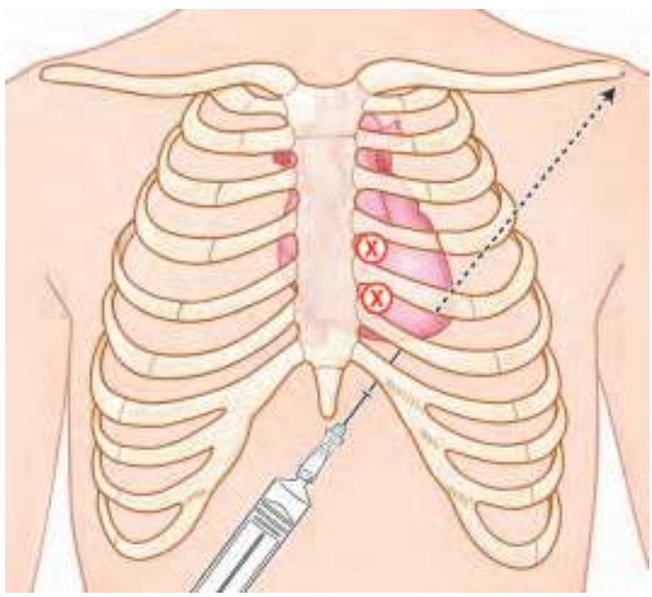

FIGURE 49-1. Intracardiac injection. The needle is inserted 1 cm to the left of the xiphoid process and aimed toward the left shoulder. The needle may also be inserted parasternally in the left fourth or fifth intercostal space (as denoted by the ⊗).

INDICATIONS

Intracardiac injection should be considered when vascular access is not readily available in a patient in cardiac arrest. The goal of the procedure is to administer epinephrine rapidly to improve the likelihood of achieving a return of spontaneous circulation (ROSC).[7] This is typically performed on patients in cardiac arrest with dysrhythmias such as asystole, pulseless electrical activity, or ventricular fibrillation.[9,10] Epinephrine was equally effective when delivered via endotracheal tube, venous access, or intracardiac injection in one animal model study.[6] The American Heart Association (AHA) recommendation and the advent of intraosseous lines have decreased the use of intracardiac injection during open cardiac massage.[9,11] Intracardiac injection has slowly been phased out and should only be attempted if other routes of medication administration have failed.[11]

CONTRAINDICATIONS

There are no absolute or relative contraindications in the moribund patient to performing this procedure. A few clinical conditions may make the procedure more technically difficult. COPD can shift the heart from its normal position and increase the risk of a pneumothorax. Anticoagulation may result in a hemopericardium and cardiac tamponade. Dextrocardia requires an alteration in needle positioning.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- 18 gauge spinal needle or 18 gauge 3½ inch long needle for adults
- 22 gauge spinal needle for children
- Syringes, 5 and 10 mL
- Nasogastric tube
- 1:10,000 epinephrine

Epinephrine is the only resuscitative medication that should be administered by intracardiac injection. Administer 1 mg of epinephrine as the initial and subsequent doses in an adult patient. Administer 0.01 mg/kg (or 0.1 mL/kg) of the 1:10,000 concentration of epinephrine in children.

PATIENT PREPARATION

This procedure is often performed on a patient who is clinically "dead" and as a last effort at resuscitation. An informed consent is not required to perform this procedure. The patient will be supine with CPR in progress. Insert a nasogastric tube to decompress the stomach if time permits. Apply povidone iodine or chlorhexidine solution to the area around the lower sternum, xiphoid process of the sternum, and upper epigastric and left costosternal angles. Identify by palpation the anatomic landmarks required to perform the procedure. Draw up the required dose of epinephrine into a syringe or use prefilled syringes. Attach a spinal needle to the syringe containing the epinephrine.

TECHNIQUES

The two routes for intracardiac injection are the subxiphoid and the left parasternal approaches (**Figure 49-1**). Both are used in a similar fashion. The left parasternal approach offers a more direct and shorter route. However, it is associated with a higher rate of complications. Both approaches require cessation of CPR to perform the procedure. It should take less than 10 seconds to perform this procedure and is within the allowable hands-off time frame of the 2015 AHA guidelines.[7,9] **The procedure of intracardiac injection should be performed as rapidly as possible to avoid prolonged cessation of CPR, but not at the expense of safety to the Emergency Physician, Nurse, and/or ancillary staff.**

SUBXIPHOID APPROACH

The subxiphoid approach is preferred as it has a lower complication rate, including less risk of injuring the left anterior descending artery.[10] Stop performing CPR. Stop ventilating the patient and allow the lungs to passively deflate. Identify the spot 1 cm to the left of the patient's xiphoid process in the costosternal angle (**Figure 49-1**). Insert the needle with the bevel up and at a 30° to 45° angle to the skin of the abdominal wall. Aim the needle toward the patient's left shoulder. Advance the needle while applying negative pressure to the syringe. Stop advancing the needle when blood flows freely into the syringe. This signifies that the tip of the needle is within the cardiac chamber. Quickly inject the epinephrine and withdraw the needle. Resume CPR and ventilation of the patient.

If the attempt at intracardiac injection is unsuccessful, withdraw the needle, flush it, and reattempt intracardiac injection. The needle can become plugged with subcutaneous fat. **CPR and ventilation must be resumed after each attempt at intracardiac injection whether successful or not.** Variations on the direction of the needle can be made for subsequent attempts. The needle may be directed toward the suprasternal notch, left mid-clavicle, or right mid-clavicle.

A spinal needle can be used as an alternative. This may be preferable due to the general population being overweight. Insert the spinal needle through the skin and into the subcutaneous tissue with its obturator in place. Remove the obturator when the tip of the spinal needle is in the subcutaneous tissue. Attach the syringe containing epinephrine to the spinal needle. Gently depress the plunger of the syringe to expel the air within the needle into the subcutaneous tissues. Advance the needle while applying negative pressure. Stop advancing the needle when blood flows freely into the syringe. Quickly inject the epinephrine and withdraw the needle. Resume CPR and ventilation of the patient.

LEFT PARASTERNAL APPROACH

This approach uses the fourth or fifth intercostal space, approximately 1 cm (or 1 finger breadth) lateral to the left sternal border (**Figure 49-1**). Stop performing CPR. Stop ventilating the patient and allow the lungs to passively deflate. Insert the needle perpendicular to the chest wall. Stabilize the needle with one hand and the syringe with the other. **Advance the needle with both hands and without excessive force.[8] It is very easy to plunge into the heart if too much force is applied to the needle.** Apply negative pressure to the syringe as it is advanced. Stop advancing the needle when blood flows freely into the syringe. Quickly inject the epinephrine and then withdraw the needle. Resume CPR and ventilation of the patient.

If the attempt at intracardiac injection is unsuccessful, withdraw the needle, flush it, and reattempt intracardiac injection. The needle can become plugged with subcutaneous fat. **CPR and ventilation must be resumed after each attempt at intracardiac injection whether successful or not.** Variations on the direction of the needle can be made for subsequent attempts. The needle may be directed toward the suprasternal notch, left mid-clavicle, or right mid-clavicle.

PEDIATRIC CONSIDERATIONS

The technique of intracardiac injection for infants and children is essentially the same as that described for the adult patient.[12] While the subxiphoid approach has been adopted as the standard, the left

parasternal approach can also be effectively used. Use a 22-gauge spinal needle for infants and children. **Use caution when inserting and advancing the needle in pediatric patients since the skin and subcutaneous tissue are thin and easily penetrated.** The dose of epinephrine administered is weight-based.

COMPLICATIONS

Complications are rare in pooled data.[5,13-15] A pneumothorax is the most common complication.[16] This is especially true with the parasternal approach or in patients with COPD. Intramyocardial injection has been reported and is associated with intractable ventricular fibrillation.[15] Rarer complications include hemopericardium and perforation of the stomach or liver. Other potential complications include coronary artery lacerations, myocardial lacerations, cardiac tamponade, and pulmonary artery lacerations.[1,4] The use of a small-gauge spinal needle and the subxiphoid approach result in fewer complications as opposed to a large-gauge needle and the left parasternal approach.[17] I**dentification of the appropriate anatomic landmarks for needle insertion and careful adherence to proper technique can minimize complications.**[12,15]

SUMMARY

Intracardiac injection is a possible route of medication delivery to a patient in cardiac arrest. Its popularity has declined in favor of safer and more effective methods of vascular access. Intracardiac injection should be considered as an alternative technique when intravenous access is not readily accessible and/or endotracheal medication administration has not provided the desired effect. It can be performed if it is done safely and does not encourage prolonged cessation of CPR during the procedure.

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50 Needle Thoracostomy

Michael Gottlieb

INTRODUCTION

A tension pneumothorax is a unilateral progressive collection of air in the pleural space (**Figures 50-1 and 50-2**). If not treated, it results in increasing intrapleural pressures, shifting of intrathoracic structures, hypoxemia, and death. It occurs from a one-way air leak into the pleural cavity from the airway conduits, the lung, or the thoracic wall. The air leak causes air to enter the pleural cavity and become trapped, without a method of egress. Rapid decompression of the tension pneumothorax with a catheter-over-the-needle is known as a needle thoracostomy and is lifesaving.

A tension pneumothorax is an immediate life-threatening condition that requires prompt recognition and treatment to prevent the patient's imminent demise. The diagnosis may be suspected based on the patient's prior medical history, the mechanism of injury, physical examination findings, and a patient in extremis. **Importantly, treatment must not be delayed to obtain further diagnostic testing (e.g., chest radiograph).** These patients most often present with acute and dramatic cardiopulmonary compromise, which may be manifest by a combination of the following signs and symptoms: respiratory distress, chest pain, air hunger, hypotension, tachycardia, diaphoresis, unilateral absence of or decrease in breath sounds, hyperresonance to percussion, increased central venous pressure, hypoxemia, cyanosis, deviation of the cardiac point of maximal impulse, and tracheal deviation.

ANATOMY AND PATHOPHYSIOLOGY

The most common cause of a tension pneumothorax is mechanical ventilation with positive pressure in a patient with a visceral pleural injury.[1] A tension pneumothorax is present in 50% of

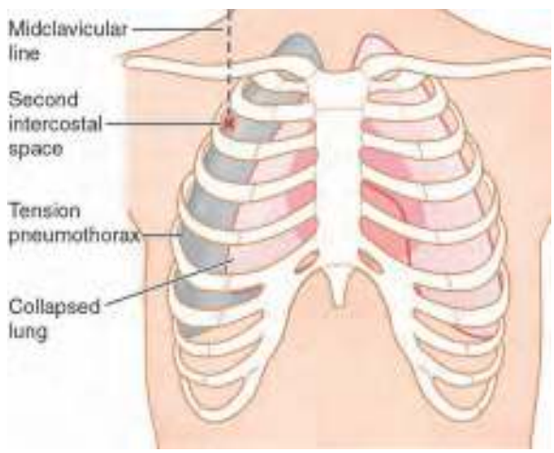

FIGURE 50-1. A right-sided tension pneumothorax. The traditional site for a needle thoracostomy is the second intercostal space in the midclavicular line.

FIGURE 50-2. Radiograph of a left-sided tension pneumothorax with no mediastinal shift. (Used with permission from reference 22.)

ventilator-associated pneumothoraces.[2] When this occurs in Intensive Care Unit (ICU) patients, they often have minimal functional reserve. To further cloud the issue, they are frequently on other supportive interventions (e.g., inotropic agents, complex ventilator settings), making their physical examination challenging and unreliable. They may also have a number of other coexisting factors that are making them unstable. This group of patients has a particularly disastrous course if a tension pneumothorax develops. Rapid diagnosis and treatment are imperative.[3]

The placement of a central venous catheter has been associated with the development of a pneumothorax. The incidence of this is approximately 3% to 6% with use of the subclavian approach, but this has been shown to decrease significantly with the use of ultrasound.[4] A tension pneumothorax may be delayed in approximately 0.4% of attempts to gain central venous access. In one case report, a patient developed a tension pneumothorax while under general anesthesia 10 days after the placement of a subclavian central venous line.[5]

A tension pneumothorax may also occur in the setting of blunt or penetrating trauma of the lung. It may occur uncommonly following a tracheobronchial or esophageal injury. It may complicate a simple pneumothorax if the parenchymal lung leak does not seal spontaneously. In this case, the site of the lung injury acts as a one-way valve, allowing air entry into the pleural space and not allowing it to escape. Occasionally, chest wall defects may result in a tension pneumothorax in the case of a sucking chest wound, wherein the wound itself acts as a ball-valve mechanism. Refer to Chapter 53 for the details of an open chest wound. More rarely, it may occur following markedly displaced fractures of the thoracic spine.

In the past, based on studies using the canine model, the pathophysiology of this disease was considered to be associated primarily with a mechanical pressure–related phenomenon.[6] Air accumulated in the involved pleural space and caused an increasing intrapleural pressure. This pressure caused compression of the ipsilateral lung, displacement of the diaphragm caudally, movement of the mediastinum and heart toward the uninjured side, kinking of the great vessels, and compression of the contralateral lung. This anatomic shift of structures would result in impaired filling of the heart and a disastrous fall in cardiac output.[7] Unfortunately, the canine model is not as similar to the human as was once thought. The mediastinum in the dog is more mobile. It is fenestrated, so that air communicates from one hemithorax to the other. Therefore elevations of intrapleural pressure in dogs would affect central structures and cause cardiovascular compromise more readily than in humans.

The intrathoracic pressure-related mechanism has come into question as the primary event. Experiments have been performed in goats, monkeys, sheep, and swine, all of which have a mediastinum that is more similar to that of the human than the dog.[8-11] These studies support the hypothesis that central hypoxemia is the primary factor in the lethality of a tension pneumothorax and occurs prior to the development of significant hypotension. In this hypothesis, the mechanical pressure–related phenomenon is a late event.

These mechanisms become more confusing and mixed in the ventilator-dependent patient. There is a lack of studies documenting hemodynamic changes in the human subject with a tension pneumothorax.[12-15] In one case report, three ventilated ICU patients demonstrated decreased cardiac output as the first sign of a tension pneumothorax.[13] The authors proposed that the absence of spontaneous breathing did not allow increased variations in negative intrathoracic pressure to act as a compensatory mechanism to prevent hemodynamic compromise. In another similar case, decreased cardiac output and mixed venous oxygen saturation were the dominant signs of a tension pneumothorax.[14] Hemoglobin desaturation via pulse oximetry was shown to be the earliest sign in a ventilator-dependent patient with a tension pneumothorax.[15]

Electrocardiographic (ECG) changes may be seen in association with a tension pneumothorax. In a left-sided tension pneumothorax, the more commonly described ECG changes are a rightward shift of the mean frontal QRS axis, precordial T-wave inversions, reduced R-wave voltage, and decreased or alternating QRS amplitude.[16-18] Other unique changes include PR-segment elevation in the inferior leads and reciprocal PR-segment depression in lead aV_R.[19] One case report cited transient bradycardia, hypotension, and precordial ST-segment elevation, all of which reversed after treatment of a right-sided tension pneumothorax.[20]

Numerous mechanisms have been proposed as the causes of these ECG changes. They include displacement of the heart, rotation of the heart around its anteroposterior or longitudinal axis, transient hypoxia, changes in coronary artery blood flow, changes in pleural pressure, pulmonary resistance, pericardial tension, acute ventricular dilatation, alterations in ventricular repolarization, pressure-induced atrial injury, and insulation of the chest wall from the associated air.[16-21] The vast majority of ECG changes have been noted with left-sided rather than right-sided tension pneumothoraces. **The degree of pneumothorax and the severity of symptoms do not seem to correlate with the magnitude of the ECG abnormalities. In summary, ECG changes are not uncommon in tension pneumothorax and should not distract from the true diagnosis.**

In cases where the diagnosis is in doubt, consider using bedside ultrasound to assess for lung sliding.[22] The transducer may require alternative placement in a tension pneumothorax (**Figure 50-3**). Lack of lung sliding, also referred to as the "barcode sign," should suggest a pneumothorax and prompt needle decompression (**Figure 50-4**). There will be little to no respiratory variation seen upon visualization of the inferior vena cava (**Figure 50-5**). Refer to Chapter 8 for the details of the trauma ultrasound.

INDICATIONS

A tension pneumothorax must be considered in the differential diagnosis of any patient in extremis. If it is a possible etiology for the patient's cardiopulmonary collapse, needle decompression

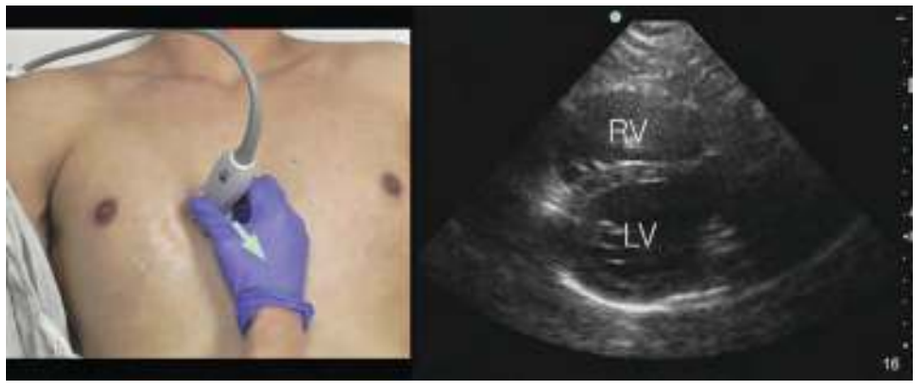

FIGURE 50-3. The use of ultrasound. ***A.*** Positioning of the probe. ***B.*** The location and axis of the right (RV) and left ventricle (LV) are altered secondary to increased left-sided pressure. (Used with permission from reference 22.)

should be performed without delay. In every circumstance, and especially in the emergent setting, one may not be able to be 100% certain of the diagnosis.[23-25] However, needle decompression can be lifesaving. Consider a needle decompression in the correct clinical situation when the patient decompensates with any of the following: pulse oximetry less than 90% on high-flow oxygen, hypotension with no other etiologies, a respiratory rate over 35, a decreased level of consciousness, pneumothorax signs and symptoms, chest wall hyperexpansion and hypomobility, and an open chest wound.[23] A bedside ultrasound can be performed quickly while preparing for the needle decompression.[26,27]

One setting that may be particularly confusing is in the dying patient with left precordial penetrating trauma. The immediate differential would be tension pneumothorax versus pericardial tamponade versus massive hemothorax. Physical examination findings are usually helpful but may also be confusing, mixed, or difficult to elicit in a chaotic and noisy resuscitation. **Needle decompression should be the first maneuver in this situation.** It may be lifesaving and will aid in the diagnosis. It is less invasive, quicker, and easier to perform than a pericardiocentesis or a thoracotomy. A tension pneumothorax is more common than pericardial tamponade in this setting. As for a massive hemothorax, a chest tube setup (Chapter 51) requires some time but should be requested at the time needle decompression is proceeding.

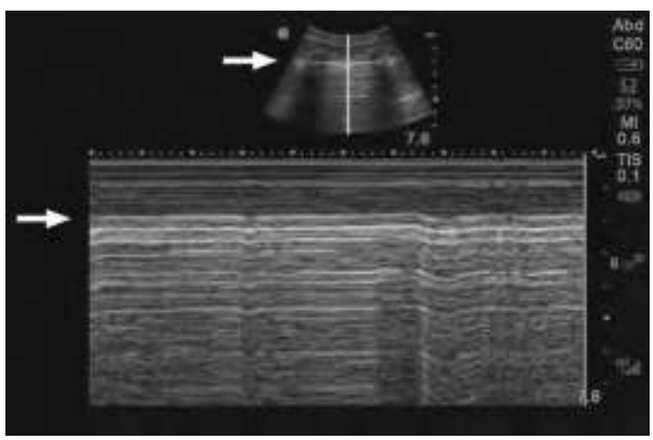

FIGURE 50-4. M-mode image of Figure 50-3. The pleural line (*arrow*) and linear pattern or barcode sign demonstrate no movement of the parietal and visceral pleura. (Used with permission from reference 22.)

CONTRAINDICATIONS

There are no absolute contraindications to performing a needle thoracostomy to decompress a tension pneumothorax. In clinically stable patients, consider a bedside ultrasound to first assess for a pneumothorax.[26-28] It is imperative to identify the anatomic landmarks properly and perform this procedure carefully if the patient has a known or suspected coagulopathy.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- 12 to 16 gauge catheter-over-the-needle, 4.5 cm in length
- Veress needle (optional)[29]
- 5 or 10 mL syringe
- Sterile gloves and gown
- Face mask with an eye shield or goggles
- Ultrasound machine (optional)
- High-frequency (10 to 15 MHz) ultrasound probe (optional)
- Sterile ultrasound probe cover (optional)
- Sterile ultrasound gel (optional)

PATIENT PREPARATION

Briefly describe the procedure to the patient if they are competent, able to understand, and cooperative. Place the patient supine. Some Emergency Physicians place the patient supine with the head of the bed elevated to 30°. This will allow the air to rise to the anterior upper chest. Unfortunately, this is not the most functional position. It is from the supine position that the patient can most easily be accessed and controlled by the greatest number of practitioners. The supine position is optimal to allow for other lifesaving maneuvers (e.g., airway management, cardiopulmonary resuscitation). Clean the skin of any dirt and debris in the area of the procedure. Apply povidone iodine or chlorhexidine to the skin.

Simultaneous with the performance of this procedure, other interventions should be requested including 100% face-mask oxygen (if the patient is not already intubated), pulse oximetry, cardiac monitoring, a chest tube setup, intravenous access, and stat chest radiography. The Emergency Physician should wear full personal protective equipment to protect themselves from contact with the patient's blood and body fluids. Although this is an emergent procedure, aseptic technique should be followed when possible.

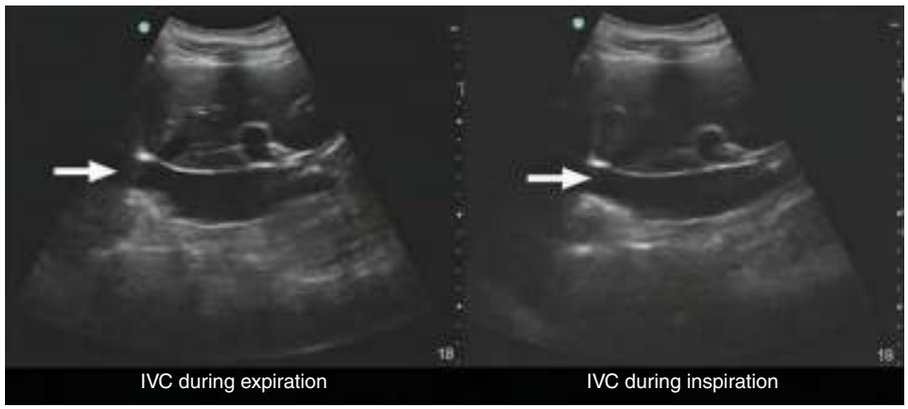

FIGURE 50-5. Sagittal ultrasound images of the inferior vena cave (IVC; *arrow*) during the respiratory cycle. (Used with permission from reference 22.)

TECHNIQUE

The safest, easiest, and most reliable site for a needle thoracostomy to decompress a tension pneumothorax is the second intercostal space in the midclavicular line (Figure 50-1).[1,2,7,30] Attach a 12, 14, or 16 gauge catheter-over-the-needle onto a 5 or 10 mL syringe without the plunger. Identify the second intercostal space in the midclavicular line. Place the nondominant index finger over the needle insertion site. Grasp the syringe with the dominant hand. Insert the catheter-over-the-needle perpendicular to the skin and just above the superior border of the third rib (**Figure 50-6*A***). This will avoid injury to the neurovascular bundle underlying the inferior border of the second rib. Some Emergency Physicians prefer to contact the upper portion of the third rib with the tip of the needle, walk it up the rib until it goes over the edge, and then advance it into the pleural space.

Advance the catheter-over-the-needle until a loss of resistance is felt as the tip of the needle penetrates the pleural space. A rush of air, with or without blood, will be heard escaping from the syringe. Stop advancing the catheter-over-the-needle. Advance the catheter until the hub is against the skin while simultaneously withdrawing the needle (**Figure 50-6*B***).

ALTERNATIVE TECHNIQUES

Other sites have been described for performing a needle thoracostomy. These include the fourth or fifth intercostal space in the anterior or midaxillary line or the second intercostal space in the anterior axillary line.[31-41] There are several problems with these alternative sites. In the fourth or fifth intercostal space, the ribs are close together, with narrower interspaces making needle placement more difficult. There is more rib motion with breathing, and arm movement can make catheter dislodgment or kinking more likely. In the supine patient, air will rise ventrally rather than laterally. Practically speaking, during the resuscitation of the unstable patient, the most important position for the Emergency Physician is at the patient's head. Insertion of a catheter in the second intercostal space in the anterior axillary line is easier from this position than inserting a laterally placed catheter. The fourth or fifth intercostal space in the midaxillary line is the ideal space for a chest tube. Placement of the catheter in these alternative sites would mean having to penetrate more tissue, especially in the obese patient, making reaching the pleural space more difficult and dislodgment of the catheter more likely. The major drawback to using the fourth or fifth intercostal space is the risk of inadvertently inserting the catheter-over-the-needle below the diaphragm and into the liver on the right or the spleen on the left.[42]

If the first attempt at needle thoracostomy fails to decompress the pleural space, a quick bedside ultrasound can be used to measure the chest wall thickness in order to determine the appropriate needle length and to confirm the presence or absence of a pneumothorax. Subcutaneous fat and tissue can be differentiated from air in the pleural space by the presence of the pleura seen deep to the pneumothorax and represented by a bright white line along with the absence of "lung sliding."

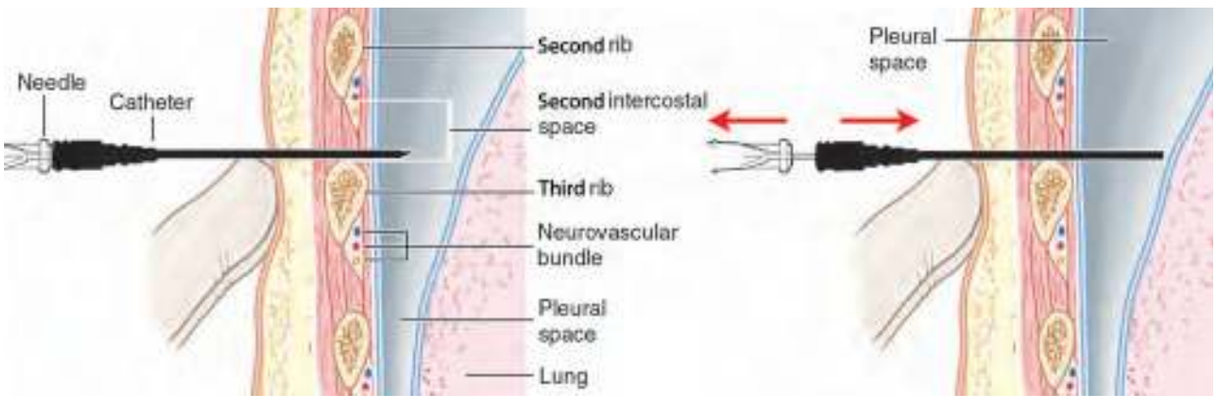

FIGURE 50-6. Decompression of a tension pneumothorax with a catheter-over-the-needle. ***A.*** The catheter-over-the-needle is inserted through the intercostal space and into the pleural cavity. ***B.*** The catheter is advanced and the needle is removed.

If the patient is in extremis, a chest tube should not be the primary therapy for a tension pneumothorax. The setup and performance of a formal tube thoracostomy takes much longer than rapid decompression with a catheter-over-the-needle. However, some experts recommend performing a finger thoracostomy in the fourth or fifth intercostal space (e.g., dissecting down to the rib and entering the pleura with a finger to release the air) followed by chest tube placement.[43] However, this is also more time consuming and should only be considered if needle thoracostomy is unsuccessful.

HANGING DROP TEST

A novel idea has been proposed to use the "hanging drop" test to identify the pleural space and determine if a tension pneumothorax is present.[44] It is quick, easy to perform, and uses supplies readily available in the Emergency Department. Be aware that gathering the supplies and performing this test can take a few minutes, thus delaying the needle decompression.

Using strict aseptic technique, insert a 4.5 cm long spinal needle into the second intercostal space and advance it until the tip is touching the upper border of the third rib. Remove the trocar. Place one to two drops of sterile saline or sterile water into the hub of the spinal needle. The fluid bubble will be elevated above the needle hub. Gently walk the needle up and over the edge of the third rib. Slowly advance the needle through the intercostal tissues while observing the fluid bubble. The negative intrathoracic pressure will suck the fluid bubble into the chest if there is no tension pneumothorax. The fluid bubble will be pushed out of the hub if a tension pneumothorax is present.

SPINAL NEEDLES

Some authors suggests using a spinal-type needle.[44] This cannot be recommended for two reasons. First, if a tension pneumothorax is present, the sharp needle should not be advanced or left in the pleural cavity to relieve a tension pneumothorax. It can puncture an organ or blood vessel. Second, the spinal needle would have to be removed and the procedure repeated with a catheter-over-the-needle to leave a catheter in place while preparing for and inserting a chest tube. Consider using a catheter-over-the-needle to perform this procedure to avoid these issues. If a longer catheter-over-the-needle is required, consider using the catheter-over-the-needle from a central line kit or a percutaneous cricothyroidotomy kit.[45,46]

RUSSELL PNEUMOFIX

The Russell PneumoFix Decompression Needle (Bound Tree Medical, Dublin, OH) is designed for decompression of a tension pneumothorax, a simple pneumothorax, and a pleural effusion. It is a 12 gauge needle with an 11 cm long catheter with graduated markings (**Figure 50-7**). The catheter is quick and simple to insert without the need for a skin incision. The Veress-tipped needle minimizes injury to the lung. It has a low-pressure, one-way valve that allows air and fluid out. It is long enough to reach the pleural cavity in obese patients.

PEDIATRIC CONSIDERATIONS

The basic technique is the same in pediatric patients. Use a smaller length and gauge catheter-over-the-needle in pediatric patients. A 20 or 22 gauge, 1 inch or 2.5 cm catheter-over-the-needle should be used in preterm children and neonates. This same size can also be used in children up to approximately 1 to 2 years of age. Between the approximate ages of 2 and 6 years, consider using an 18 or 20 gauge, 1.5 inch or 3.75 cm long catheter-over-the-needle. Over the age of 6 and into early adolescence, consider using a 16 or 18 gauge, 4.5 cm long catheter-over-the-needle. Use the adult size catheter-over-the-needle for older adolescents or obese children. **It is important to note that these are only recommendations based on average body sizes.** The individual child's size and body habitus need to be taken into consideration when choosing a catheter-over-the-needle size and length.

FIGURE 50-7. The contents of the Russell PneumoFix kit. (Photo courtesy of Bound Tree Medical.)

Some physicians prefer to use a butterfly needle with attached tubing instead of a catheter-over-the-needle in preterm children, neonates, and up to the first year of life.[47] Select a butterfly needle size as described above. Place sterile water or sterile saline in a sterile specimen cup. Insert the distal hub end of the butterfly tubing into the bottom of the specimen container and submerged in the liquid. Tape the tubing onto the rim of the specimen container to secure it and ensure it does not pull out of the liquid in the specimen container. Insert the butterfly needle into the pleural space as described above. Air bubbles will emerge from the tubing and into the sterile liquid as the tension pneumothorax is decompressed. The specimen container also forms a water seal, preventing flow of air into the pleural space, until a tube thoracostomy can be performed. Consider using a small gauge catheter-over-the-needle instead of a butterfly needle to avoid the problems described previously with using a spinal needle.

ASSESSMENT

Once the catheter has been placed into the pleural space, a gush of air should rush out of the syringe. **The procedure will have converted a tension pneumothorax into a simple pneumothorax requiring a tube thoracostomy.** The patient should improve both hemodynamically and symptomatically. Saturations on pulse oximetry should rise after the decompression. If this does not occur, too short of a catheter-over-the-needle may have been used and the procedure should be repeated with a longer one. If a longer catheter-over-the-needle is not available, rapidly perform a tube thoracostomy. Another possibility is that the pleural space was entered appropriately but the diagnosis was incorrect. In this event, alternative causes of the patient's shock state should be sought and consideration should be given to the placement of a "prophylactic" chest

tube. This may prevent later sequelae from the iatrogenic catheter stab wound to the chest and simplify further workup and monitoring of this unstable patient.

AFTERCARE

After insertion of the catheter and improvement in the patient's clinical status, the immediate life threat has been treated. Secure the catheter against the skin with a suture or an assistant holding it in place. **Continue to observe and monitor the patient closely for recurrence of the tension pneumothorax and procedural complications.** Establish intravenous access, cardiac monitoring, and pulse oximetry if not already done. Obtain baseline laboratory studies, an arterial blood gas, and a chest radiograph. Obtain a thorough history and physical examination to search for the etiology of the tension pneumothorax. **A definitive chest tube should be placed using sterile technique to prevent recurrence of the tension pneumothorax and to treat the simple pneumothorax.** Refer to Chapter 51 for the complete details regarding the placement of a chest tube. After a chest tube is inserted, remove the needle thoracostomy catheter and place a simple bandage over the puncture site.

COMPLICATIONS

An incorrect diagnosis of a tension pneumothorax in an unstable patient is always a possibility, even in the best of hands.[48,49] If this is the case, the cause of the patient's shock state must still be aggressively sought and treated. A prophylactic chest tube should be considered for a presumed parenchymal lung injury and potential pneumothorax from the needle thoracostomy.[50-52] This is especially true if the patient is going to be transported out of the resuscitation area, will be given a general anesthetic, or is to be placed on positive-pressure ventilation.

Failure to reach and decompress the pleural space is the major argument against the use of needle thoracostomy. Depending on the patient's body habitus and the catheter length, the pleural space may not have been reached to be decompressed. In a study from the United Kingdom, the chest wall thickness was estimated to range from 1.3 to 5.2 cm by ultrasound in the second intercostal space.[53] A 3.0 cm cannula would fail to penetrate into the pleural cavity in 57% of the patients in this study. A 4.5 cm cannula would fail to penetrate into the pleural cavity in 4% of the patients. In this case, the procedure should be repeated with a longer catheter-over-the-needle.[54] If one is not immediately available, a tube thoracostomy should immediately be performed.

Several studies have evaluated chest wall thickness using ultrasound and computed tomography (CT) scans in order to determine the needle length required to appropriately decompress the pleural space in the general adult population. Initial studies using ultrasound seemed to indicate that using needle lengths of 4.5 cm would be sufficient to reach the pleural space in most patients.[53,55] More recent studies using CT have shown that chest wall thickness varies significantly by gender and age.[55-59] One study found that chest wall thickness was greater than 4.5 cm in 10% of men younger than age 40 and 19% of men older than age 40.[56] The same study found that almost one-third of women less than 40 years of age had a chest wall thickness greater than 4.5 cm, falling to one-fourth for women over 40. The mean chest wall thickness was found to be 5.36 cm in autopsy CT scan studies.[59] The authors recommended use of a 3.25 inch or 8 cm long catheter-over-the-needle. Others also recommend using longer needles.[37,41,54,55,60] It is important to be aware of these variations. However, most authors still advocate using a needle length of 4.5 cm, because longer needle lengths can increase the risk of vascular, pulmonary, or cardiac injury.[61,62]

The second intercostal space in the midclavicular line is the traditional location for a needle thoracostomy. A prospective study of needle thoracostomy sites found that providers often place it more medial than recommended.[63,64] Deviation from this insertion point is more likely to be associated with vascular injury and hemorrhage. This includes injury to the internal mammary artery medially, subclavian vessels superiorly, and the pulmonary trunk and heart inferiorly.

After the initial decompression of a tension pneumothorax, the catheter may become dislodged, clotted, or kinked. **If the tension pneumothorax recurs, immediately repeat the procedure.** Once needle decompression is reaccomplished, perform a tube thoracostomy. If possible, a tube thoracostomy can be initiated by the Emergency Physician while other members of the resuscitation team continue with other interventions. This would help prevent recurrence of the tension pneumothorax while other interventions (e.g., CPR, intubation, venous access) are being performed.

There may be additional complications secondary to the catheter placement.[61-67] A local hematoma or underlying lung laceration may occur. Infectious agents may be introduced into the pleural cavity. If the catheter-over-the-needle is introduced under the inferior border of the rib instead of over the superior border of the rib, the intercostal vessels or nerve may be lacerated. **Proper technique in placing the catheter-over-the-needle should be observed to minimize preventable complications.**

The traditional approach to relieving a tension pneumothorax is the placement of a large-bore needle in the ipsilateral second intercostal space. This may work in the "standard" patient where there are no adhesions or scarring in the pleural space. However, this may not be the proper needle location in the patient with prior pulmonary disease, pleural disease, or pleural adhesions. The classic hospital patient in this category is the patient with adult respiratory distress syndrome on positive end-expiratory pressure (PEEP) with high airway pressures who develops a loculated tension pneumothorax. The needle placed in the standard manner often fails to reach the affected pleural area. Stat chest radiographs are often required to help guide placement of the needles and/or chest tubes in these more complex patients.[7] Bedside ultrasonography is also useful to help guide the procedure.

The needle thoracotomy procedure is not without its complications. This leads some to question the performance of a needle thoracostomy instead of just performing a tube thoracostomy.[47,68] There are no clinical trials comparing needle versus tube thoracostomy to relieve a tension pneumothorax in human subjects. This study was recently undertaken in a swine model.[64] The authors demonstrated that a properly performed needle thoracostomy is as effective over a 4-hour period as a tube thoracostomy. While the authors did not record the time it took to perform the procedures, performing a needle thoracostomy is quicker, easier, and simpler and now just as effective when compared to a tube thoracostomy.

SUMMARY

A tension pneumothorax is a clinical diagnosis that is often made in an agonal patient with respiratory distress, absent or decreased breath sounds over a hemithorax, and severe cardiopulmonary compromise. Needle thoracostomy to decompress the tension pneumothorax should be performed immediately. The traditional site is the second intercostal space in the midclavicular line. The site of needle insertion seems to be changing. This lifesaving procedure is quick, simple to perform, and requires minimal equipment. A needle thoracostomy should be followed as soon as feasible by a tube thoracostomy.

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51 Tube Thoracostomy

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INTRODUCTION

A tube thoracostomy is the placement of a tube through the thoracic wall and into the pleural cavity. It is commonly referred to as a chest tube. It is placed to evacuate air, blood, or other fluid that collects within the pleural space. The etiology of the air or fluid collections can be due to iatrogenic complications, infection, lung disease, malignancy, or trauma.

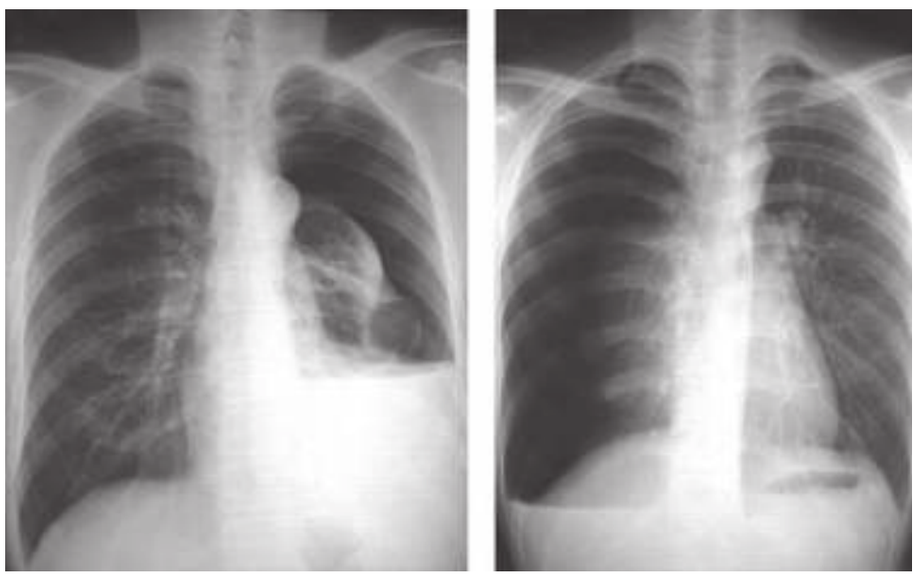
Thoracic trauma accounts for nearly one-quarter of all trauma-related mortality.[1,2] Some injuries require surgical intervention. Most injuries are treated nonoperatively. Injuries to the bronchi, chest wall, esophagus, lung, or trachea may lead to the presence of abnormal air and/or fluid in the pleural space. The use of a tube thoracostomy (i.e., chest tube) in these situations may be diagnostic and therapeutic. Historically, closed-tube drainage of the pleura has been used for various indications for more than a century.[3] This chapter deals primarily with the use of tube thoracostomy following trauma. Much of the information remains the same regardless of whether the patient is a trauma victim or a medical patient.

ANATOMY AND PATHOPHYSIOLOGY

The diaphragm and accessory muscles of respiration contract and generate negative pressure within the pleural space on inspiration. Penetration of the visceral or parietal pleura due to injury disrupts this pressure gradient and allows air to enter the "potential space" between the pleurae and results in a pneumothorax.[1,2] **A simple pneumothorax is the accumulation of air that is not under pressure within the pleural space (Figure 51-1).**[4] It may cause the ipsilateral lung to collapse. The increased pressure in the thoracic cavity may push the mediastinum toward the noninjured side as air continues to accumulate and if there are no adhesions. This can cause angulation of the atriocaval junction, impairment of atrial filling, and a subsequent decrease in cardiac output manifest by hypotension. **The presence of a pneumothorax under pressure accompanied by respiratory and/or circulatory compromise is a tension pneumothorax until proven otherwise and is an immediate life threat (Figure 51-2).**

There are two important points to remember about a tension pneumothorax. It is a clinical diagnosis based on the patient's presenting signs and symptoms. Do not wait for a chest film to establish the diagnosis. The initial treatment of this entity is needle decompression or finger decompression followed by tube thoracostomy. A large-bore needle is inserted in the second intercostal space (ICS) in the midclavicular line at the superior border of the rib. A gush of air will ensue if the patient has a tension pneumothorax and the symptoms will improve. This converts the tension pneumothorax to a simple pneumothorax requiring a chest tube for more definitive management. Refer to Chapter 50 for complete details regarding the needle thoracostomy procedure.

The frequent inadequacy of needle decompression using standard length needles has been noted as obesity rates increase. Recent literature has recommended using 8 cm long needles to assure



A B

FIGURE 51-1. Chest radiographs. ***A.*** Left-sided pneumothorax and hemothorax. ***B.*** Right-sided pneumothorax. (Used with permission from reference 4.)

FIGURE 51-2. A tension pneumothorax.

adequate entry into the pleural space. Some authors advocate using the fourth ICS in the anterior axillary line.[5] There is also support for "simple" or "finger" thoracostomy performed in the fourth ICS.[6,7] Either technique is acceptable as long as the pleura is adequately decompressed.

An open pneumothorax is caused by a traumatic chest wall injury that results in a defect that is greater than or equal to two-thirds the diameter of the patient's trachea (Chapter 53). Air passes via the path of least resistance (i.e., the defect) and leads to equilibration of the intrathoracic and extrathoracic pressures. This compromises oxygenation and ventilation. **An open pneumothorax is an immediate life threat.** Initial treatment may consist of a nearly occlusive "three-sided" dressing creating a one-way valve for egress of air from the pleural cavity. Alternatively, the patient may be placed on positive-pressure ventilation. The chest tube can then be inserted at a site remote from the actual defect.

Injury to the chest may result in laceration of vascular structures (e.g., great vessels, heart, intercostal vessels, internal mammary arteries, or lung parenchymal vessels). The body can absorb small amounts of free blood from the pleural space. The presence of free blood over a prolonged time leads to an increased risk of infection and fibrosis.[3,8] The body cannot effectively clear large quantities of blood or clot from the pleural cavity. Blood in the pleural space (i.e., a hemothorax) can in most cases be treated with the insertion of a chest tube. A tube thoracostomy is usually followed by urgent surgical intervention when a major systemic or pulmonary vessel has been injured and results in a massive hemothorax (i.e., > 1500 mL of blood in the pleural space).

Penetrating wounds or blunt rupture of the thoracic esophagus may result in a hydrothorax, pneumomediastinum, pneumothorax, or some combination. Suspect esophageal injury in any patient with a knife or ice pick wound in a suspicious location, a transmediastinal bullet trajectory, or a severe and sudden compression of the chest or abdomen.[2] A tube thoracostomy is used as part of the treatment if a hydrothorax or pneumothorax is a presenting finding. These patients require urgent surgical attention. Tube thoracostomy may be used in the treatment of traumatic chylothorax resulting from an injury to the thoracic duct.

One special circumstance deserves mention. Certain patients who have sustained significant blunt trauma to the torso may have a diaphragmatic rupture. **Chest radiographs may reveal an air density in the hemithorax that could be mistaken for a pneumothorax.** This may represent the presence of the stomach or colon in the thoracic cavity. Extra care must be taken when entering the pleural cavity so as not to injure a hollow viscus inadvertently when a chest tube is inserted in such a case.

INDICATIONS

The indications for a tube thoracostomy following blunt or penetrating trauma to the chest include the presence of a chylothorax, hemopneumothorax, hemothorax, hydrothorax, simple pneumothorax, or a tension pneumothorax (**Figures 51-1 and 51-2 and Table 51-1**). A chest tube may be placed prophylactically in patients with penetrating injuries to the chest who do not have evidence of a pneumothorax on initial chest radiographs but are expected to undergo endotracheal intubation and general anesthesia. The medical indications for a tube thoracostomy include an empyema, malignant pleural effusions, pneumothorax, pleurodesis, and recurrent pleural effusions. Perform a tube thoracostomy after the needle or finger decompression of a tension pneumothorax to convert it to a simple pneumothorax.

OCCULT PNEUMOTHORAX

Traumatic pneumothoraces and hemothoraces that are not evident on plain chest radiographs are being diagnosed more frequently with computed tomography (CT).[9] The question then arises as to whether these pneumothoraces should be treated. The management of these "occult pneumothoraces and hemothoraces" is somewhat controversial. One study looked at 40 patients who sustained chest trauma and were discovered by CT scan to have pneumothoraces.[10] The study concluded that patients undergoing positive-pressure ventilation should have placement of a chest tube. The study could not confirm that patients with small pneumothoraces who were not going to be ventilated could safely be observed. A review of 2326 CT scans, 80.5% after negative chest radiographs, noted 102 occult pneumothoraces and/or hemothoraces.[11] Only 12 of these patients required tube thoracostomy. Similar results were found for small, isolated occult hemothoraces.[12] A smaller study noted a higher percentage of patients requiring tube thoracostomy, but these patients were undergoing

TABLE 51-1 Indications for Chest Tube Insertion

Bronchopleural fistula
Chest wall trauma patient air-lifted
Chest wall trauma patient mechanically ventilated
Chylothorax
Empyemas
Esophageal rupture and leaking into pleural space
Hemopneumothorax
Hemothorax
Iatrogenic pneumothorax
Malignant pleural effusions
Open pneumothorax
Parapneumonic effusion
Penetrating chest trauma
Pleural effusion
Pleurodesis or sclerosis
Postoperative
Spontaneous pneumothorax
Tension pneumothorax
Traumatic pneumothorax

positive-pressure ventilation.[13] Nonprogressive occult pneumothoraces can usually be managed without a tube thoracostomy.[14] Close clinical observation and/or follow-up plain radiographs are recommended. An initially occult pneumothorax that is increasing in size or is in a patient who develops respiratory distress requires a tube thoracostomy.[14] A patient with an occult pneumothorax who requires endotracheal intubation, general anesthesia, and/or positive-pressure ventilation is an indication for a tube thoracostomy.[15]

CONTRAINDICATIONS

The only absolute contraindication to performing a tube thoracostomy is in the patient who requires an open thoracotomy. Although there are no firm contraindications to performing a tube thoracostomy in a trauma patient, there are some areas of controversy. There is increasing interest in conservative management of smaller traumatic pneumothoraces, particularly in blunt trauma.[16,17] There is less support for conservative management in penetrating trauma.[9] A South African study found that conservative management was safe in cases of small pneumothoraces in patients with stab wounds.[18] There is currently no indication for conservative management of pneumothoraces caused by gunshot wounds. Repeat chest radiographs within 3 to 6 hours to rule out an enlarging pneumothorax or the delayed manifestation of a hemothorax if observation is selected for a patient with a traumatic pneumothorax.[2,10]

Some physicians have begun managing small pneumothoraces with catheter aspiration rather than tube thoracostomy. Much of the evidentiary support for this comes from literature regarding spontaneous pneumothoraces.[19,20] **Tube thoracostomy remains the safest and most complete method of evacuating pneumothoraces and hemothoraces due to traumatic injury.**[21,22]

There are several relative contraindications to performing a tube thoracostomy in the medical patient. These include the presence of a skin infection over the chest tube insertion site, a coagulopathy, large pulmonary blebs or bullae, pulmonary adhesions, loculated pleural effusions, tuberculosis, or previous tube thoracostomies. These patients may require CT or ultrasound guidance to place the chest tube. Correct a coagulopathy before the chest tube is inserted if placement is not required emergently.

There has been some suggestion in the literature that there may be a role for the prehospital placement of chest tubes.[21,23-25] Properly trained aeromedical crews and Physicians in some systems frequently perform tube thoracostomies in the field. This has not gained widespread acceptance.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- 10 to 20 mL syringe
- Local anesthetic solution with epinephrine (1% lidocaine or 0.25% bupivacaine)
- 25 or 27 gauge needle
- #10 scalpel blade on a handle
- Kelly clamps, large and medium
- Chest tubes, sizes 12 to 42 French
- Sterile water
- Chest tube drainage apparatus with a water seal
- Christmas tree connector
- Suction source and tubing
- Needle driver
- Mayo scissors, large curved
- Size 0 or 1–0 suture, silk or nylon
- Petrolatum-impregnated gauze
- 4 × 4 gauze squares
- Adhesive tape, 3 to 4 inches wide
- Sterile drapes
- Sterile gloves and gown
- Surgical cap
- Face mask with a face shield or goggles
- Tincture of benzoin spray or swabs
- 0.25% bupivacaine

Most hospitals and Emergency Departments have prepared their own "chest tube trays" that contain all the equipment required to place a chest tube except the chest tubes, local anesthetic solution, and a collection system. These last three items will vary based on the etiology of the air and/or fluid in the pleural cavity, the age and size of the patient, and physician preference. Commercially produced chest tube kits are also available.

Chest tubes used in the Emergency Department are hollow, clear, straight plastic tubes (**Figure 51-3**). The distal end of the chest tube has numerous fenestrations or holes that allow the passage of air and/or fluid into and through the tube. A radiopaque stripe allows for radiographic localization of the chest tube after it is inserted into the patient. The proximal end of the chest tube is beveled to allow it to fit better on a plastic connector.

Chest tubes are available in numerous sizes and from multiple manufacturers. The lower the number, the smaller is the size of the chest tube. A spontaneous pneumothorax may be drained with an 18 to 26 French tube in adults, a 14 to 16 French tube in children, a 12 to 16 French tube in infants and small children, and a 8 to 12 French tube in neonates. Traumatic pneumothoraces are usually drained with a 28 to 32 French tube in adults and a 16 to 20 French tube in children. Traumatic hemothoraces, traumatic hemopneumothoraces, and empyemas require larger size tubes. A 36 to 42 French tube in adults and a 20 to 24 French tube in children will provide adequate drainage without becoming occluded by blood clots or purulent material.

The procedure requires the use of a large Kelly clamp to bluntly dissect a subcutaneous tract, to puncture the tract, and to dilate a tract through the intercostal muscles. The Kelly clamp requires the

FIGURE 51-3. The chest tube. The proximal end is beveled, while the distal end is fenestrated.

FIGURE 51-4. The Centurion Blunt Dissector. (Photo courtesy of Centurion Medical Products, Howell, MI.)

FIGURE 51-5. A commercially available chest tube drainage system. (Courtesy of Covidean, Minneapolis, MN.)

Emergency Physician to pull the ringed handles apart and in opposite directions to open the jaws of the clamp. Opening the jaws of the Kelly clamp when it is within the subcutaneous tissues or intercostal muscles can be quite difficult. The Centurion Blunt Dissector clamp (**Figure 51-4**) was specifically designed to aid in the insertion of a chest tube (Centurion Medical Products, Howell, MI). The mechanical action of this clamp is opposite that of the Kelly clamp. The jaws are closed in the resting position and the ringed handles are held open with a spring mechanism. The ringed handles are squeezed together to open the jaws of the clamp. Some physicians find this is a more natural, intuitive, and easier motion to perform. This chest tube clamp is disposable and available individually or incorporated into a chest tube insertion tray.

DRAINAGE SYSTEMS

It is important to know how to use the drainage system available at your institution to prevent any complications arising from the use of these devices. The classic glass bottle system with rubber corks is rarely, if ever, used in the United States today in the Emergency Department. Commercially available drainage systems are currently available in most hospitals (**Figure 51-5**). They are made of lightweight plastic, sterile, and intended for single-patient use. They are preassembled, disposable, and may be used for autotransfusions (Chapter 228). They have clear plastic covers to allow easy visualization of the fluid within the unit.

The system is a single unit that consists of three or four chambers, depending on the manufacturer. The first chamber connects to the chest tube with flexible rubber tubing. It collects blood, clots, and/or other fluid expressed through the chest tube. The second chamber is the water seal. It allows one-way flow of air away from the patient and maintains a negative intrathoracic pressure gradient compared to the atmosphere. The third chamber is the suction regulator, which attaches to the wall suction. It draws in atmospheric air when needed to limit the negative pressure of the vacuum. Some manufacturers have included a fourth chamber to assess the patient's intrapleural pressure.

PATIENT PREPARATION

Explain the risks, benefits, complications, and aftercare to the patient and/or their representative if time and the patient's clinical condition permits. Obtain an informed consent for a tube thoracostomy or document in the medical record the verbal discussion whenever possible. A tube thoracostomy is often performed under urgent or emergent conditions following trauma. Lifesaving care should always proceed on the patient's behalf with the appropriate documentation in the medical record after the patient is resuscitated.

There has been considerable discussion in the literature regarding the use of antibiotics in patients requiring a tube thoracostomy for trauma in the hope of preventing an empyema.[26-28] The Eastern Association for the Surgery of Trauma (EAST) practice management guidelines work group reviewed the literature regarding the use of antibiotics in conjunction with chest tube insertion.[29] They found several trials that evaluated infectious complications after a tube thoracotomy using Centers for Disease Control and Prevention (CDC) criteria. The studies each had their weaknesses. The working group recommended that there was sufficient class I and class II evidence to support a recommendation of administering a first-generation cephalosporin intravenously just before making the skin incision and continuing the intravenous antibiotic for 24 hours.[29]

The administration of parenteral analgesics, sedatives, and/or procedural sedation (Chapter 159), if not contraindicated, will be greatly appreciated by the patient as the procedure is quite painful. Employ the appropriate protocols for patient monitoring. Place the patient on the cardiac monitor, noninvasive blood pressure cuff, pulse oximetry, and supplemental oxygen to monitor them during and after the procedure. Frequently check vital signs.

Place the patient supine or semierect with the arm on the involved side raised away from the chest (**Figure 51-6**). Identify the fifth ICS in the midaxillary to anterior axillary line (**Figure 51-6**).[30,31] Consider marking this point on the patient's skin with a pen or marking pen. Consider placing a soft restraint around the wrist to prevent the arm from moving during the procedure (**Figure 51-6**).

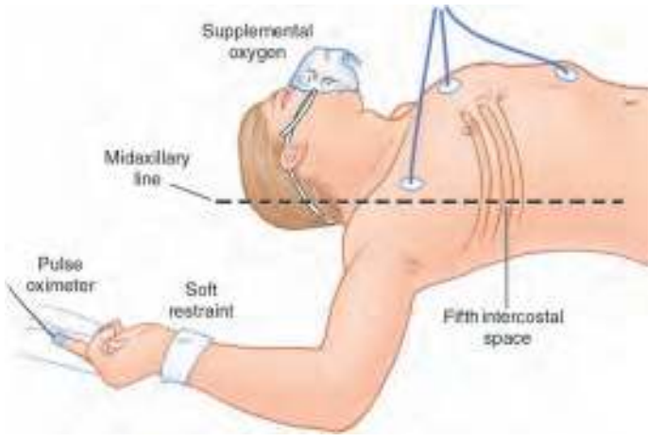


FIGURE 51-6. Patient positioning for a tube thoracostomy. Note the application of supplemental oxygen, pulse oximetry, cardiac monitoring leads, and a soft restraint.

Apply povidone iodine or chlorhexidine solution to the chest wall and allow it to dry. Apply sterile drapes to demarcate a sterile field. **Sterile technique should be observed and followed by all involved personnel. They should don a cap, mask, eye shield, sterile gown, and sterile gloves.**

Reidentify the fifth ICS in the midaxillary to anterior axillary line (Figure 51-6). This is the preferred site for chest tube insertion.[30] The reasons for this are twofold. The diaphragm rises during respiration to the level of the nipple. Chest tube insertion below the fifth ICS unnecessarily risks puncture of the diaphragm or abdominal organs. The area of the midaxillary line is the least muscular area of the chest wall and is thus an easier area from which to gain access to the pleural cavity.[1-3] This area is also described as the "safety space." Its boundaries are the base of the axilla superiorly, the fifth ICS inferiorly, the lateral edge of the pectoris major muscle (i.e., anterior axillary line), and the lateral edge of the latissimus dorsi muscle (i.e., posterior axillary line).

Infiltrate local anesthetic solution into the chest wall and pleural cavity if the patient is awake and aware of their surroundings. This should be performed regardless of whether the patient receives parenteral analgesics, sedatives, and/or procedural sedation. Approximately 10 to 20 mL of local anesthetic solution with epinephrine (e.g., lidocaine or bupivacaine) is required to provide adequate analgesia. Consider using bupivacaine as it provides longer analgesia than lidocaine. **Be aware of the maximum weight-based volume of local anesthetic solution to administer to prevent toxicity (Chapter 153).** Raise a subcutaneous wheal of local anesthetic solution one interspace below the one to be used to insert the chest tube (i.e., the sixth ICS). Infiltrate local anesthetic solution subcutaneously and upward to a point above the fifth ICS. **Ensure the needle goes just above the upper border of the rib to avoid the neurovascular bundle under the bottom edge of the rib above (Figure 51-7).** Redirect the needle to anesthetize the intercostal muscles and parietal pleura of the fifth ICS. Advance the needle into the pleural cavity and inject 2 to 3 mL of local anesthetic solution to adequately anesthetize the pleura (**Figure 51-7**).

TECHNIQUE

The technique described here is an "open" technique as opposed to that employing the use of a trocar. Trocar-aided insertion of chest tubes is associated with a higher incidence of major complications and does not result in any significant saving of time.[1-3] **Never use a trocar for chest tube insertion.**

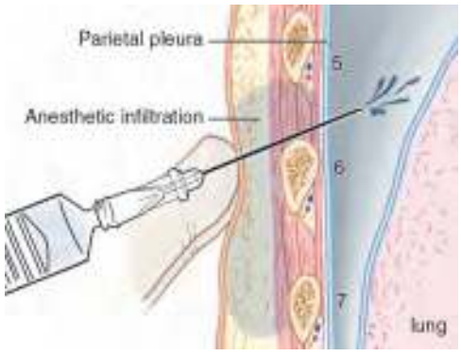


FIGURE 51-7. Infiltration of local anesthetic solution into the chest wall and pleural cavity.

Make a 3 to 5 cm incision with the #10 scalpel blade over the rib one ICS below the desired ICS (i.e., the sixth ICS) (**Figures 51-8 and 51-9 *A***). Bluntly dissect a tract or tunnel with the 6 inch Kelly clamp in the subcutaneous tissue in a cephalic direction to the rib above (**Figure 51-9*B***). Orient the clamp with the tips curved toward the skin. Advance the closed tips of the clamp in 1 cm increments and open the jaws to dissect the tract (**Figure 51-9*B***). The tract should terminate at the upper border of the fifth rib. **This will avoid injury to the neurovascular bundle lying under the inferior border of the fourth rib.** Rotate the clamp 180° such that the tip is aimed just above the superior border of the fifth rib and toward the pleural cavity (**Figure 51-9*C***). Briskly push the closed tips of the clamp through the intercostal muscles and parietal pleura and into the pleural cavity (**Figure 51-9*C***). **This maneuver requires a significant amount of force to enter the pleural cavity.** A twisting motion as the clamp is advanced may facilitate penetration into the pleural cavity. The intercostal muscles will stretch and entering the pleural cavity will be difficult if the clamp is advanced slowly.

A loss of resistance associated with a rush of air or fluid occurs as the pleural cavity is entered with the closed tips of the clamp (**Figure 51-9*C***). The fluid contained within the pleural cavity may exit the tract forcibly if under pressure. **It is important not to plunge too deeply with the clamp as the pleural cavity is entered.** The tips of the clamp can injure the diaphragm, great vessels, heart, or lung. The forward motion of the clamp can be partially opposed by bracing the nondominant hand on the underside of the clamp and applying counterpressure away from the patient as the clamp enters the pleural cavity.

Spread the jaws of the clamp to enlarge the tract through the subcutaneous tissue, intercostal muscles, and parietal pleura. Insert a finger through the tract and into the pleural cavity (**Figure 51-9*D***). Feel the lung as it expands with inspiration and contracts with

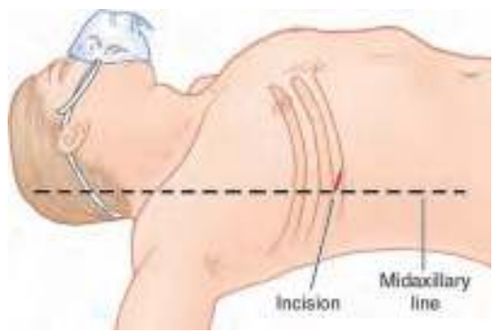


FIGURE 51-8. The initial skin incision is made over the rib one interspace below the desired chest tube insertion site.

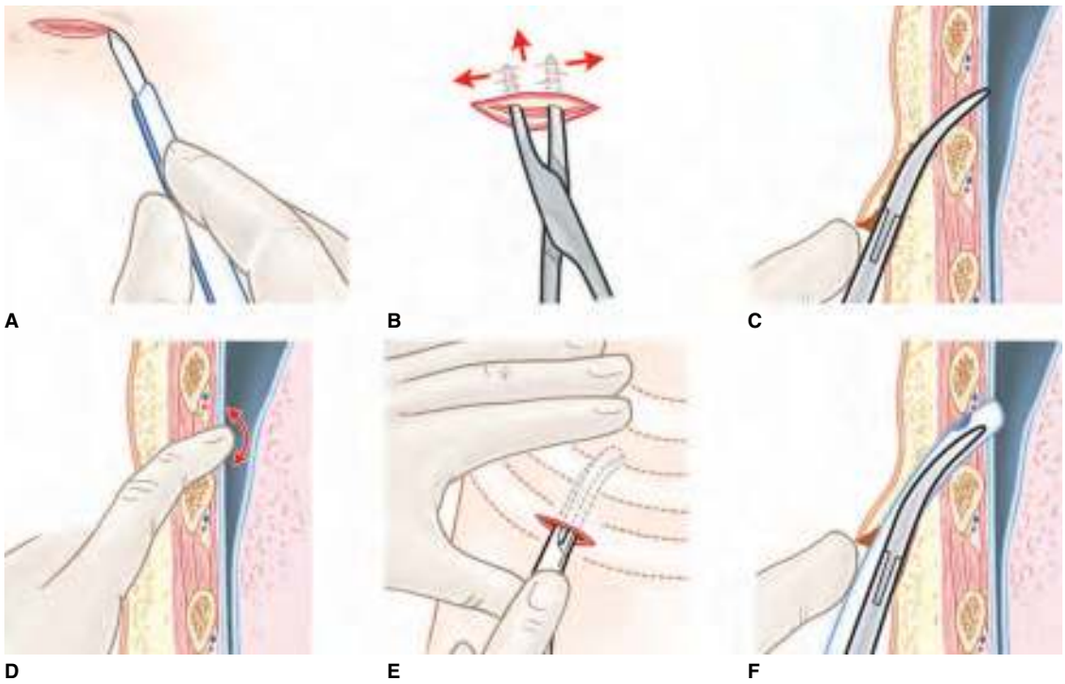


FIGURE 51-9. The tube thoracostomy. ***A.*** The skin incision is made. ***B.*** A tract is bluntly dissected in the subcutaneous tissues. ***C.*** The Kelly clamp is forced into the pleural cavity. ***D.*** A finger is inserted through the tract to feel for adhesions. ***E.*** The chest tube is held in the Kelly clamp and inserted through the tract. ***F.*** The chest tube is guided into the pleural cavity.

expiration. Rotate the finger to ascertain the presence or absence of adhesions (**Figure 51-9*D***). **Gently break any loose adhesions between the lung and thoracic cage with the finger. Dense adhesions require the chest tube to be inserted at another site.**

Prepare to insert the chest tube. Estimate the distance from the skin incision to the apex of the lung by laying the chest tube over the patient. Apply a clamp onto the chest tube at the estimated site at which it should exit the skin incision. This location should be 4 to 5 cm proximal to the fenestrations in the chest tube. Cut off the beveled proximal end of the chest tube just above the bevel.

Grasp and clamp the tips of the large Kelly clamp onto the distal end of the chest tube. Insert the tips of the clamp and chest tube through the tract and into the pleural cavity (**Figures 51-9*E* and *F***).[32] Use the clamp to direct the tip of the chest tube posteriorly and superiorly. Alternatively, the dominant index finger can be placed through the tract to direct the chest tube. **The use of the finger in the tract is the preferred method to guide the chest tube.** The finger will be able to confirm the proper intrapleural placement of the chest tube. Release the Kelly clamp and advance the chest tube until all the fenestrations are within the pleural cavity and the preplaced clamp on the chest tube is at the skin incision. Hold the chest tube securely in place. Remove the Kelly clamp from the incision. Release the clamp on the chest tube.

Secure the chest tube with 0 or 1–0 silk or monofilament nylon suture (**Figure 51-10**). The many techniques that have been described for securing chest tubes are idiosyncratic and probably equivalent. Suffice it to say that the tube should be sewn in such a way that the incision is closed tightly around the tube to assure a better seal and that routine movements of the patient should not dislodge it (**Figure 51-10*A***).[33]

Place the first stitch as a simple interrupted stitch at one end of the skin incision (**Figure 51-10*B***). Leave both ends of the suture long after tying the knot in the first stitch. Wrap the needle end of the suture firmly around the chest tube three or four times. Tie a knot in

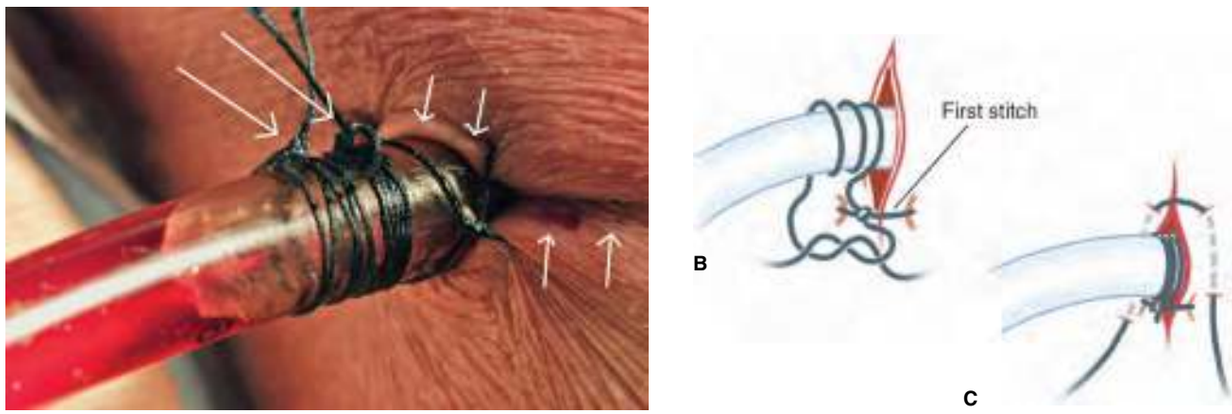


FIGURE 51-10. Securing the chest tube to the thoracic wall. ***A.*** Proper knot tightness and wound approximation. The *long arrows* point to knots that are tied on the chest tube. The *short arrows* are the sealed skin incision and the area around the chest tube. (Used with permission from reference 33.) ***B.*** The stay suture. ***C.*** The purse-string suture.

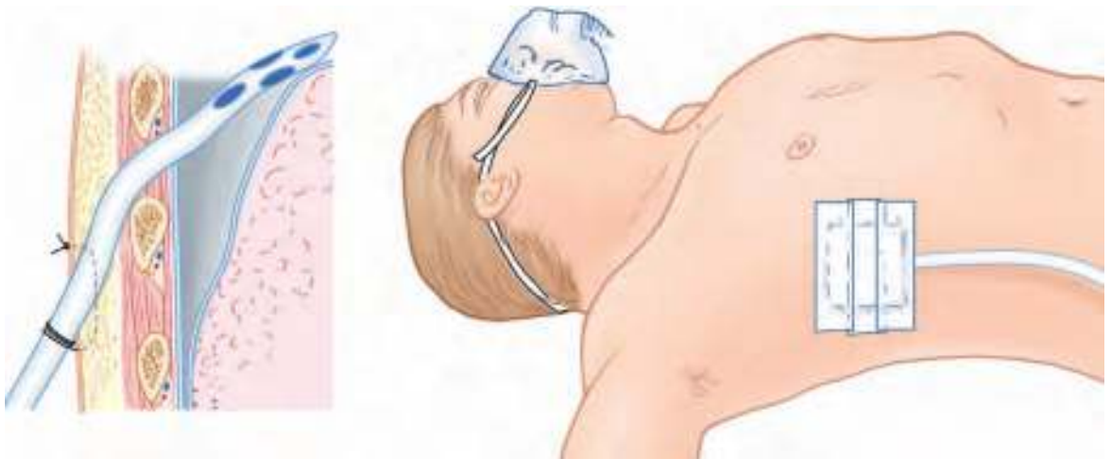


FIGURE 51-11. Securing the chest tube. ***A.*** The chest tube has been secured with suture to the chest wall. ***B.*** An occlusive dressing has been placed over the incision and taped to the chest wall.

the suture to secure the chest tube to the skin (**Figure 51-10*B***). Place the second stitch as a purse-string suture encompassing the chest tube (**Figure 51-10*C***). Leave both ends of the suture long. Wrap both ends of the suture around the chest tube and tie a bow, not a knot. This stitch will be used later to close the skin incision after the chest tube is removed. Place simple interrupted or horizontal mattress sutures to close the remainder of the skin incision.

Apply an occlusive dressing over the incision site (**Figure 51-11**). Apply petrolatum gauze over the incision site and around the chest tube as it exits the incision. It seals the chest wall incision from the atmosphere and prevents air from the atmosphere from entering the pleural space from around the chest tube. Place gauze squares over the incision site and petrolatum gauze. Apply tincture of benzoin to the chest wall surrounding the gauze squares. Tape the gauze and chest tube to the torso. The ends of the tape should be adherent to the tincture of benzoin. **Do not place tape over the patient's nipple. Protect the nipple with a piece of gauze if the tape must cover the nipple.**

An alternative to the above dressing is the Centurion Chest Tube Anchor (Centurion Medical Products, Howell, MI). This is a sterile, adherent patch that surrounds the chest tube as it exits the skin (**Figure 51-12**). It forms an occlusive dressing. It has an attached cable tie to wrap around the chest tube and secure it in place. This cable tie replaces suturing the chest tube in place. The device can be trimmed to a smaller size for pediatric patients. The cable tie will secure all sizes of chest tubes.

Connect the chest tube to a drainage system (**Figure 51-13**), which is a self-contained multichamber device.[3] The first chamber is a collecting chamber that connects directly to the chest tube. The second chamber contains a small amount of saline or water and acts as a one-way valve. This assures flow only in the direction away from the patient. The third chamber controls suction, with a capability of at least 20 cmH_2O suction, and attaches to the wall suction system. Commercially available systems encompass all three chambers in one unit (**Figure 51-5**).

ALTERNATIVE TECHNIQUES

CHEST TUBE LOCATION

Evidence is lacking and the recommendations vary regarding the location of a chest tube.[34] The chest tube tip can be directed toward the pleural cavity apex or base.[35] Direct the chest tube apically with its tip above the aortic notch for a pneumothorax (**Figure 51-14**, area 1). Direct the chest tube basally with its tip just above the diaphragm for a hemothorax or a hemopneumothorax (**Figure 51-14**, area 2). Aiming superiorly decreases the likelihood of chest tube placement in the lung fissure.

PLEURAFLOW

The PleuraFlow Active Tube Clearance (PleuraFlow ATC; ClearFlow Inc., Anaheim, CA) was designed to be used in cardiac surgery. It has been adopted by some for use in trauma patients in the Emergency Department. The device uses a looped guidewire inside the chest tube (**Figure 51-15**). It is available for chest tubes that are pediatric and adult sizes. A magnetic slider on the outside moves the looped guidewire to clear any clots. It is simple to use and does not break sterility. The proximal end attaches to any drainage system. The guidewire can be removed if the chest tube

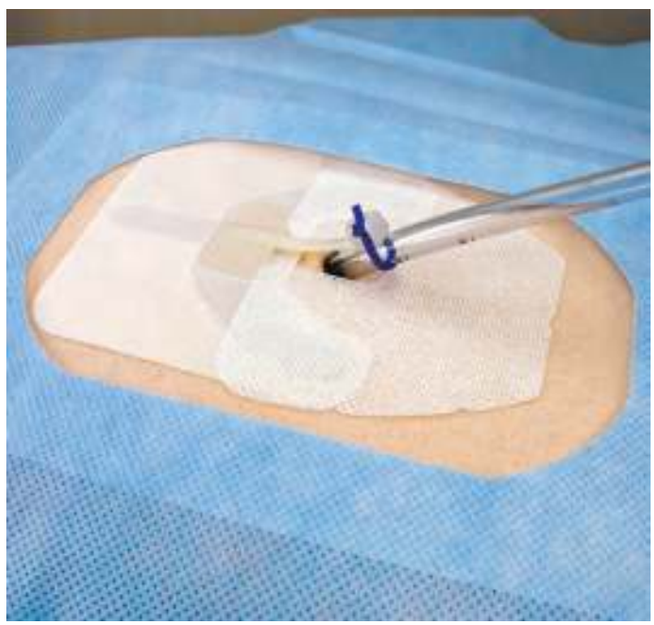


FIGURE 51-12. The Centurion Chest Tube Anchor. (Photo courtesy of Centurion Medical Products, Howell, MI.)

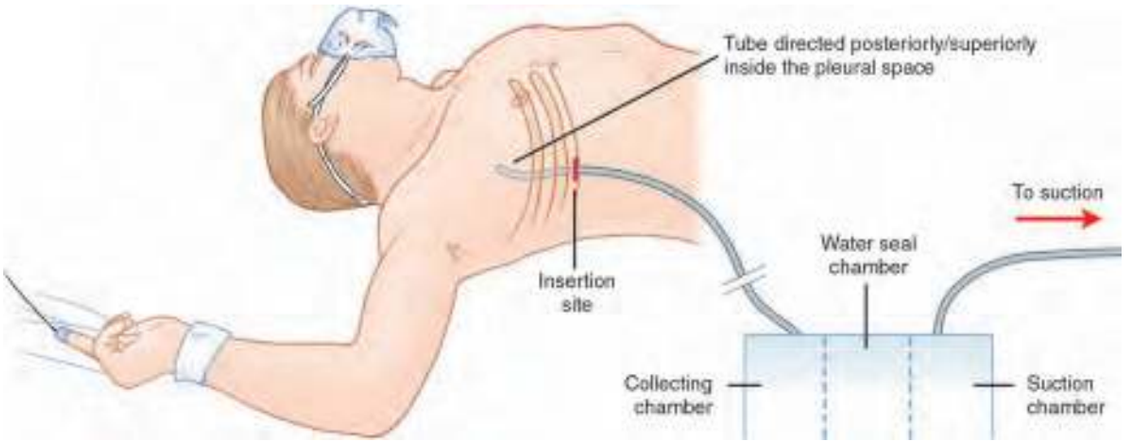


FIGURE 51-13. The chest tube is connected to a drainage system.

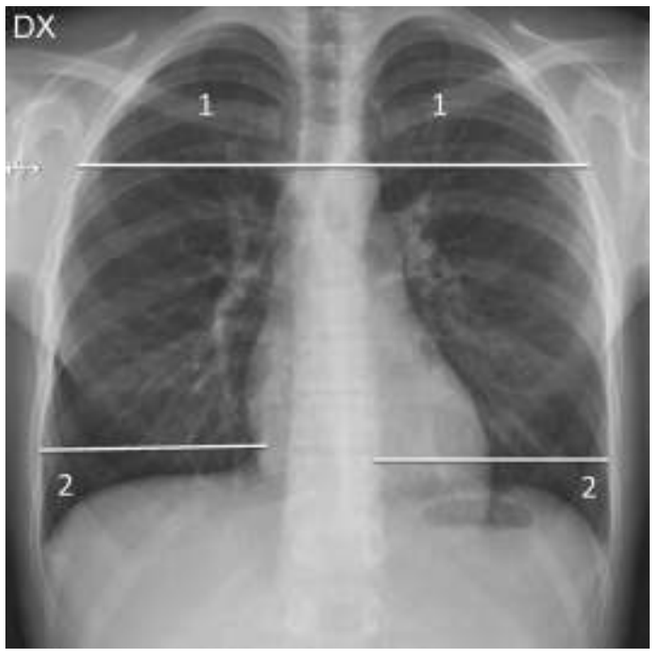


FIGURE 51-14. The chest tube can be inserted into the pleural apex (area 1) or pleural base (area 2). (Used with permission from reference 35.)

remains without the risk of clotting (i.e., resolution of the hemothorax with persistent pneumothorax).

SELDINGER TECHNIQUE FOR SMALL CATHETERS

Drainage of a hemothorax or pneumothorax can be accomplished using a small-bore pigtail or straight catheter in pediatric patients and adults (**Figure 52-19**).[22,36-39] A straight catheter is preferred over a pigtail for traumatic hemothoraces due to the increased failure on insertion and complications.[40] An alternative is to use a central venous catheter if a pigtail catheter is not available.[41] Attach a 16 gauge, 2 inch catheter-over-the-needle to a 5 or 10 mL syringe. Insert the catheter, as described above, aimed superiorly. **Insert the catheter-over-the-needle over the superior border of the rib to avoid the neurovascular bundle located on the inferior border of the rib.**[42] Securely hold the needle and syringe so it does not move. Advance the catheter to the hub. Remove the needle and syringe. **Quickly cover the catheter hub with a gloved finger.** Insert the guidewire through the catheter (**Figure 52-19*A***). **Hold the guidewire securely to prevent it from falling completely into the pleural cavity.** Remove the catheter over the guidewire and leave the guidewire in place (**Figure 52-19*B***). Extend the skin incision with a #11 scalpel blade by 3 to 5 mm to allow the catheter into the pleural space without "crumpling" (**Figure 52-19*C***). Advance the dilator over the guidewire and into the pleural cavity to dilate the tract (**Figures 52-19*C*** and ***D***). A gentle twisting motion of the dilator as it is advanced will aid in its insertion into the pleural cavity (**Figure 52-19*D***). Hold the guidewire securely. Remove the dilator while leaving the guidewire in place. Insert the catheter over the

A

B

FIGURE 51-15. The PleuraFlow ACT system. ***A.*** The system. ***B.*** View of the tip. (Courtesy of ClearFlow Inc., Anaheim CA.)

guidewire and into the pleural cavity (**Figure 52-19*E***). Remove the guidewire catheter. Attach the catheter to a drainage system and secure it as described previously.

DRAINAGE SYSTEMS

Drainage systems for a pneumothorax vary in style but function with the same "one-way valve" principle. The simplest method is a flutter valve. It is best illustrated by the following noncommercial method. Cut a premoistened finger from a sterile glove. Tie the proximal end to the thoracentesis catheter with a silk suture and cut the distal end so that it is open to the air (**Figure 52-20**).[43] This creates a flutter valve and allows air to escape with coughing or expiration and prevents air from reentering the pleural space on inspiration.

Commercial kits are available that can be used for outpatient management of a pneumothorax in some patients. The kit often contains a Heimlich flutter valve (**Figure 52-21**).[43,44] **The arrow on the clear protective tube covering the Heimlich valve must point away from the patient.**[45] Suction is usually not needed. Outpatient management may be considered in stable patients with a primary spontaneous pneumothorax that is small, apical with initial lung reexpansion, and good apposition of the lung with the lateral chest wall.[46,47] **The patient must be able to understand and comply with outpatient instructions, must have access to immediate help, and must return to an Emergency Department if symptoms arise.** Additional requirements include good residual lung function, normal oxygen saturation, and an air leak adequately treated by thoracentesis.[48] **A contraindication to using the flutter valve is a hemothorax.** A closed underwater seal system is recommended in these cases.

The Pneumostat (Maquet Getinge Group, Hudson, NH) is a single-use device (**Figure 52-22**). It attaches to a garment. The Pneumostat has multiple chest tube connectors. The air leak well provides quick and simple air leak confirmation. It has a 30 mL collection chamber and a needleless sampling port. It helps the patient ambulate sooner due to it being small and not cumbersome. It has a one-way valve for patient protection.

The TRU-CLOSE Thora-Vent (UreSil LLC, Skokie, IL) is an alternative device to the pigtail catheter and Heimlich valve combination (**Figure 52-23**). It is a one-piece unit that combines an intrapleural catheter and external one-way antireflux valve that attaches to the chest wall by an adhesive pad. Its insertion is quicker, easier, and simpler than a traditional catheter. It is inserted using a trocar. The diaphragm is used to indicate pleural entry and pneumothorax resolution. The low profile makes ambulation and outpatient management easier for the patient. This device is rarely available in the Emergency Department.

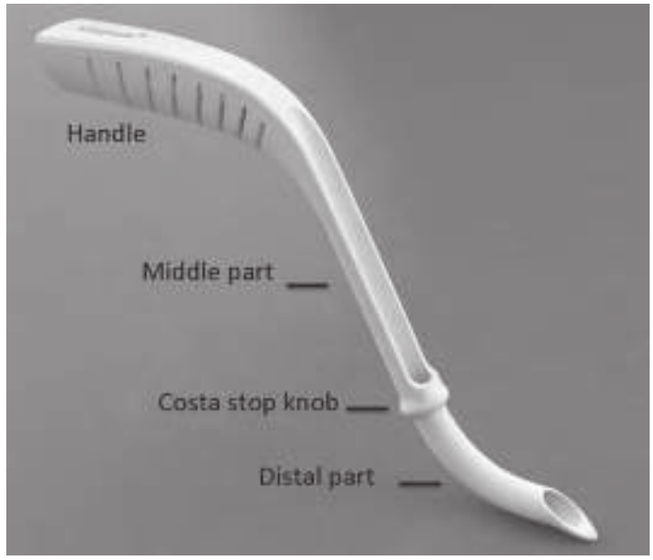
ULTRASOUND-GUIDED TECHNIQUE

The same sterile preparation applies to the ultrasound evaluation. A pneumothorax may be more readily identified with the patient positioned supine rather than upright with ultrasound. A 3.5 to 5.0 MHz phased-array transducer or 7.5 to 10 MHz high-frequency linear transducer is recommended.[49] The orientation of the transducer follows the convention that the marker should correlate to the reference point in the left upper corner of the monitor screen. Ultrasound can identify the safe location for the insertion of a chest tube.[30]

The key US features of a pneumothorax include the absence of inspiration-expiration–related "lung slide," loss of "comet tail" artifact, and broadening of the pleural line to a thick band (**Figure 52-17**).[50,51] Compare the hemithorax suspected of having a pneumothorax to the unaffected side to confirm these findings in the case of a unilateral pneumothorax (**Figure 52-17**). Aspirate a pneumothorax using the ultrasound similar to a pleural effusion (Chapter 52) or insert a chest tube.[52]

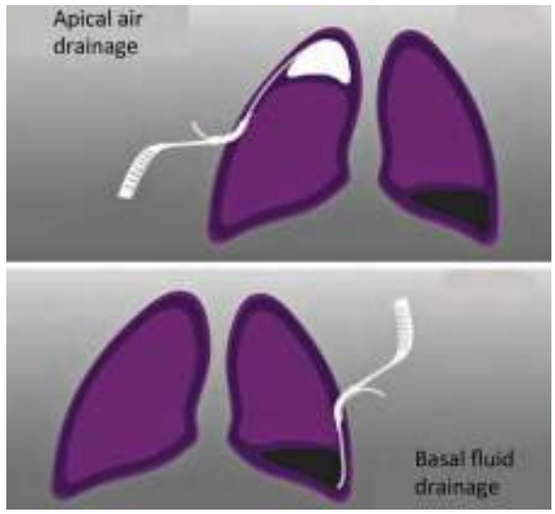
KATGUIDE

The KatGuide was developed for the safe and accurate insertion of a chest tube (Pleuratech, Denmark).[35] The device has a curved shape to place the chest tube (**Figure 51-16**). It consists of a handle,



A

B



C

FIGURE 51-16. The KatGuide. ***A.*** The device. ***B.*** The chest tube loaded. ***C.*** The device to insert the chest tube in the apex or the base. (Photos used with permission from reference 35.)

a middle part to load the chest tube, and a distal part that guides the chest tube. The KatGuide method is a blunt technique (i.e., dissection with a Kelly clamp, digital exploration, insertion of the distal end of the KatGuide pointing to the part of the pleural cavity that needs drainage followed by chest tube insertion). The chest tube is guided into the pleural cavity through the KatGuide instead of using a finger and Kelly clamp. The KatGuide enhances the chance of optimal chest tube positioning and reduces the risk of chest tube misplacement. The rounded tip and the insertion direction parallel to the lung surface prevent lung damage.

ASSESSMENT

Obtain an anteroposterior portable chest radiograph.[53] Observe the position of the chest tube. Remove the chest tube and insert a new one if it is bent, kinked, or in the fissure of the lung. If its tip is against the mediastinum, unsecure the tube, withdraw it a few centimeters, resecure the tube, and obtain a repeat radiograph. Remove the chest tube and insert a new one if it is in the subcutaneous tissue. Observe the fenestrations on the distal end of the chest tube in the radiograph. **They all must be within the thoracic cavity.** If not, remove the chest tube and insert a new one. **Never advance a chest tube further into the thoracic cavity after obtaining a chest radiograph to prevent tracking infectious material into the pleural cavity**.

Persistent bubbling in the system or failure of the lung to reexpand indicates a leak in the system.[54,55] Check the system to ensure that all connections are secure. Place tape over the connections to eliminate leaks and prevent the components from becoming dislodged. Check the tubing for any holes or fissures. Examine the chest tube and the radiograph to confirm that all fenestrations are within the thoracic cavity. Replace the chest tube if all fenestrations are not within the pleural cavity. **An injury to the trachea, mainstem bronchus, a large bronchiole, or the esophagus can cause a persistent air leak.[54,55]** A second chest tube can be inserted to keep up with the leak and prepare the patient for bronchoscopy and/or esophagoscopy to diagnose the etiology of the persistent air leak. Large or persistent air leaks usually represent proximal tracheobronchial injuries and may require urgent surgical intervention. Any persistent air leak should prompt a surgical consult.

AFTERCARE

Patients with chest tubes require close monitoring. If administered, continue prophylactic antibiotics for 24 hours. Obtain daily serial chest radiographs to monitor for resolution of the inciting process. The presence of air leaks from the chest tube indicates that the injury has not completely healed and the seal between the parietal and visceral pleurae has not yet been restored. Maintain suction until there is no evidence of an air leak. The acceptable minimal daily output from a chest tube as a criterion for removal varies according to the institution, the physician, and the reason for insertion. It is also unclear whether a trial period of water seal following suction is strictly necessary. There is literature to suggest that suction and water seal protocols for removing chest tubes are effective and have similar incidences of recurrent pneumothoraces.[56] There is literature supporting an abbreviated trial of water seal following suction as it may allow time for occult pneumothoraces to manifest themselves and alleviate the need for reinsertion of a chest tube.[57] Monitor the chest tube insertion site for signs of infection.

Check the chest tube and collection tubing periodically for blockage. The tubing may be milked or stripped to alleviate the blockage and avoid the need to replace the chest tube. **Milking refers to forcing air, fluid, or clots back into the chest. Stripping refers to creating negative pressure within the tubing to move fluid or clots distally and into the collecting chamber.** To milk the tube, clamp or pinch the tubing shut distally while using the other hand to compress the tubing and move proximally to force the contents back into the thoracic cavity. To strip the tube, clamp or pinch the tubing shut proximally while using the other hand to compress the tubing and move distally followed by the sudden release of the proximal tubing.

CHEST TUBE REMOVAL

Be prepared to replace the chest tube if planning to remove a chest tube. Have all the necessary equipment and supplies readily available in case the patient urgently requires a new chest tube. Don gloves, a gown, a face mask with an eye shield or goggles, and a cap to prevent becoming contaminated when the chest tube is removed.

Place the patient supine or semirecumbent. The use of parenteral sedation and soft restraints is rarely necessary when removing a chest tube. Carefully remove the tape securing the chest tube to the chest wall. Be cautious when removing the tape if it covers the patient's nipple to prevent any injury. Remove the gauze squares and petrolatum gauze covering the incision site. Untie the bow securing the free ends of the purse-string suture that was previously placed. Cut the suture that is holding the chest tube to the chest wall. Remove this suture. Place the first half of a surgeon's knot in the free ends of the purse-string suture. Pass the ends of the suture to an assistant. **Instruct the patient to inhale or exhale fully and hold their breath.[58] This results in a Valsalva-type maneuver and will prevent ambient air from being drawn through the chest wall and into the pleural cavity**. Quickly and smoothly remove the chest tube while the assistant cinches down the knot of the suture to seal the skin incision. Tie additional knots to secure the purse-string suture. Place petrolatum jelly or topical antibiotic ointment over the incision. Cover the site with gauze squares and tape it securely.

Sometimes a purse-string suture is not used in the insertion of a chest tube. Ensure that the chest tube site is completely occluded by the assistant. This is ideally performed with petrolatum and standard gauze as the chest tube is being removed.

Observe the patient for 4 to 6 hours for any signs of cardiovascular or respiratory compromise. Obtain expiratory posteroanterior and lateral chest radiographs if the patient remains asymptomatic. Evaluate the radiograph for the presence or recurrence of a hemothorax, hydrothorax, pneumothorax, or pyothorax.[59,60] The dressing may be removed in 24 to 48 hours. Remove the chest wall incision sutures in 8 to 10 days.

COMPLICATIONS

Tube thoracostomy is often described as a simple procedure. However, a chest tube can result in serious complications (e.g., injuries to thoracic and abdominal organs) if it is not performed with care and attention.[1,2,61-68] An unusual occurrence of sudden death following chest tube insertion has been reported.[69] It was attributed to hemorrhage near the vagus nerve causing irritation and stimulation of the vague nerve and refractory bradycardia. Injury to the thoracic duct from the chest tube being inserted too deeply can result in a chylothorax.[70] Injury to the heart and great vessels can occur if the chest tube is placed anteriorly or a trocar was used to insert the chest tube.[64] Lung injury can occur if the clamp plunges inward on entering the pleural cavity. **It is imperative that the Kelly clamp be controlled as it enters the pleural cavity**. If the lung is adherent to the chest wall, it may be penetrated by the Kelly clamp or the chest tube. **A trocar should never be used to insert a chest tube as it can**

cause significant injury to the heart, lung, or other intrathoracic structures.[64,67]

Other complications associated with chest tube placement and removal include recurrent, residual, and loculated pneumothoraces.[62] These may require the placement of additional chest tubes. Placement of the chest tube too high can result in a Horner's syndrome or subclavian artery occlusion.[71,72] Insertion too far from the left side can result to injury of the right side.[73] The wrong ICS can be identified and the chest tube inserted too high or low.[31] The lung can herniate through the incision site.[64] The chest tube can compress and injure the long thoracic, phrenic, and ulnar nerves.[64,74,75] A retained hemothorax may require decortication and may develop into an empyema.[8,76,77] The lung may not expand due to the bronchus being plugged (e.g., mucus). Allergic reactions can occur to the skin preparation, local anesthetic solution, or tape.

Posttraumatic empyema remains a serious complication of thoracic trauma with incidences ranging from 2% to 25%.[1,2,8,77] The etiology of the infection is not always clear. A break in sterile technique on chest tube insertion, nosocomial pneumonia, superinfected pulmonary contusion, and undrained hemothoraces have all been implicated. **Empyemas attributed to the chest tube insertion are completely preventable complications that can be avoided by strict adherence to aseptic technique.** Other infectious complications include chest wall cellulitis and necrotizing fasciitis.[64,78]

Bleeding can occur from several sites. Incision site bleeding is often due to superficial venules and arterioles. The application of pressure and suturing the incision closed will alleviate this bleeding in most cases. An intercostal artery or vein can be lacerated if the dissection or penetration into the pleural cavity occurs along the inferior surface of a rib. Securing the chest tube against the inferior surface of the rib may tamponade the bleeding. Attempt to tamponade the bleeding with a Foley catheter if it continues. Insert the catheter into the pleural cavity, inflate the cuff, and withdraw the catheter to lodge the inflated cuff against the posterior surface of the rib. Another option is to extend the incision to expose and ligate the bleeding vessel. Lung injury and bleeding from penetration into the pleural cavity are often self-limited and minor. Rarely will an injury be serious enough to warrant surgical intervention. **A trocar should never be used as risk of injury to intrathoracic structures is significantly increased.** An anteriorly placed chest tube should be at least 3 cm from the lateral border of the sternum to prevent injury to the internal mammary artery.

The chest tube can become occluded and stop functioning. A large tube should always be inserted if its purpose it to drain blood, clots, or purulent material. Attempt to milk and/or strip the tubing, as described previously. Obtain a chest radiograph to determine if the chest tube is kinked. Twist the chest tube 180° and release it. If it spins back into its original position, it is kinked.[79] Remove the chest tube and insert a new one if the occlusion cannot be dislodged or the tube is kinked.

Subcutaneous emphysema results from air from an inadequately decompressed pneumothorax that tracks into the subcutaneous tissues or a misplaced chest tube.[62] Ensure that the chest tube, drainage system, and suction source are functioning properly. Replace any component that is not functioning. Verify that the chest tube is within the pleural cavity and not within the subcutaneous tissues using either plain chest radiography or ultrasound.[80] Evaluate the chest radiograph to ensure that all of the distal drainage holes are within the pleural cavity and not in the subcutaneous tissues.

Reexpansion pulmonary edema occurs from the rapid expansion of a lung that has been collapsed for over 48 to 72 hours or from the removal of a large pleural effusion.[4,81] Patients will begin to experience increasing shortness of breath and hypoxemia within a few hours of the procedure. Repeat chest radiographs will show an expanded lung with pulmonary edema. The exact etiology of this complication is unknown. This complication may be prevented by the slow expansion of a lung and the removal of pleural fluid in increments. Treatment includes supportive care, supplemental oxygenation, and positive-pressure ventilation (e.g., bilevel positive airway pressure, continuous positive airway pressure, and/or intubation). Diuretics have no role in relieving the edema. Reexpansion pulmonary edema has an associated mortality rate of up to 20%.[4]

The sources of pain for a patient with a tube thoracostomy are numerous. These include the skin incision, subcutaneous dissection, intercostal muscle transection, the chest tube, and the underlying injury. Pain can usually be managed with a combination of oral, parenteral, and topical analgesics. Sedatives may be required but can often be avoided with adequate pain management. Intrapleural bupivacaine has been found to be effective in reducing pain.[82-84] Administer 20 to 40 mL of 0.25% bupivacaine through the chest tube and into the pleural cavity. Clamp the chest tube or the tubing for up to 10 minutes to allow the bupivacaine to thoroughly coat the pleural cavity. **Carefully monitor and observe the patient to ensure that they do not develop a tension pneumothorax while the chest tube is clamped**. Unclamp the chest tube and allow the excess anesthetic to drain into the collection system. This can provide several hours of pain relief to a patient who may have limits or contraindications to parenteral analgesics.

SUMMARY

Tube thoracostomy is useful in the treatment of thoracic injuries resulting in chylothoraces, empyemas, hemothoraces, hydrothoraces, and pneumothoraces. Attention must be paid to observe sterile technique, choose the proper insertion site, carefully enter the pleura, and verify entry via digital examination. Employ appropriate drainage systems to assure maintenance of a closed, water-tight system. Monitor the patient regularly while the chest tube is in place. Emergency Physicians performing this procedure should be cognizant of the serious complications that may be associated with tube thoracostomies, some of which are directly related to the insertion technique. Adherence to the principles described above will assist in avoiding many of these complications and provide optimal care for victims of thoracic trauma.

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52 Thoracentesis

Marianne Juarez and Jacqueline Nemer

INTRODUCTION

Thoracentesis is a term derived from the Greek meaning "to pierce the chest." It is used today to refer to the removal of air or fluid from the thoracic cavity. Hippocrates first described thoracentesis in the management of an empyema.[1] Thoracentesis was widely used in World War II and the Korean conflict in lieu of a thoracotomy for chest drainage. This practice was replaced by tube thoracostomy by the time of the Vietnam War. Thoracentesis is used today in the diagnosis and therapy of pleural effusions, as an emergent and temporizing treatment of a tension pneumothorax, and in the management of small nontraumatic pneumothoraces.[1-4] Thoracentesis is generally indicated to aid in the evaluation and management of the underlying etiology in newly discovered pleural effusions. **Accumulation of pleural fluid is not a specific diagnosis but rather a reflection of an underlying process.**

A pleural effusion may be identified clinically and radiologically. The patient may develop pain related to irritation of the parietal pleura, compromised pulmonary mechanics, or interference with gas exchange.[3] The pain may be in the abdomen, chest, or ipsilateral shoulder. Cough is a common presenting symptom, although its mechanism is unclear. Dyspnea occurs secondary to the space-occupying effect of the fluid and alterations in gas exchange. Pleural effusions can reduce cardiac output in extreme cases. Physical examination findings will depend on the size and location of the pleural effusion. Tactile fremitus may be absent or attenuated. There may be dullness to percussion. Auscultation may reveal decreased breath sounds over the involved hemithorax.

Radiographic diagnosis of pleural effusions may be made by a posteroanterior (PA) chest radiograph when there is homogeneous opacification in the hemithorax, absent air bronchograms, and clouded vesicular vascular markings.[5] The minimum fluid volume injected into cadavers to blunt the costophrenic angle was between 175 and 500 mL.[6] Chest computed tomography (CT) scan will provide details of the pleural effusion, associated findings, location, and size. Several studies support the routine use of ultrasound (US) to identify pleural effusions and to guide the thoracentesis procedure.

A lateral decubitus film is often necessary and will determine whether the fluid is loculated or free-flowing if CT scan and US are not available. It will be helpful if one of the following signs are present on the PA chest radiograph: blurred contour of the diaphragmatic dome, clear costophrenic angle, elevated hemidiaphragm, or the gastric bubble seen more than 2 cm from the lung border in patients with left-sided pleural effusions.[2] US may be necessary in localizing effusions that are < 10 mm thick on the lateral decubitus film.[7]

The use of US can aid in the localization of pleural effusions that may be small or missed on plain chest films or distinguish between interpretations as possible pneumonia or atelectasis.[8] Pleural fluid can be identified on US as a black hypoechoic area that appears darker than the surrounding diaphragm, liver, and lung.

Thoracentesis performed blindly has a high associated complication rate.[9] US-guided thoracentesis allows the Emergency Physician to map the pleural effusion, choose the best site for thoracentesis, and reduce the rate of complications.[10-14] US guidance is safe in assisting with thoracentesis in intubated and mechanically ventilated patients, both of which are high-risk populations for complications.[12,15]

A pneumothorax may be simple or under pressure (i.e., a tension pneumothorax).[16] A simple pneumothorax may present clinically with chest pain, dyspnea, hypoxia, and tachycardia.[17] Physical examination may reveal decreased breath sounds on auscultation of the affected side. A tension pneumothorax classically presents with absent breath sounds on the affected side, hypotension, and tachycardia. Other symptoms may include an acute change in mental status, air hunger, cardiorespiratory arrest, chest pain, cyanosis, deviated trachea, diaphoresis, and hypoxia.[1,4] Sometimes a patient with a tension pneumothorax may have a normal physical examination due to subtle auscultation findings often missed in a noisy Emergency Department. Tactile fremitus may be absent, and percussion is typically hyperresonant over the hemithorax with the tension pneumothorax. Thoracentesis to relieve a tension pneumothorax is based on the mechanism of lung injury, the patient's symptoms, and physical examination findings. Mechanisms of injury to the lung include chest trauma, instrumentation of the chest, mechanical ventilation, and spontaneous lung rupture.

There are two major indications for performing a thoracentesis.[1-3,16,18] The first is for the evacuation of air for treating a simple pneumothorax and for the emergent temporizing treatment of a tension pneumothorax. The second is for the evacuation of fluid.

This may be done to help diagnose the etiology of a pleural effusion or for the treatment of a symptomatic pleural effusion.

PLEURAL EFFUSIONS

ANATOMY AND PATHOPHYSIOLOGY

The pleura is a serous membrane that covers the diaphragm, lungs, mediastinum, and thoracic cavity. The pleural space is a potential space between the lung and the thoracic cavity. A thin layer of fluid normally exists between the visceral pleura covering the organs and the parietal pleura covering the chest wall. This fluid acts as a lubricant.[3] Pleural fluid originates from the interstitium, parietal capillaries, and visceral capillaries. Hydrostatic and oncotic forces govern the flow of fluid in the pleural space. These forces are summarized in **Figure 52-1**. Protein free fluid enters the pleural space from the parietal pleura and is absorbed by the visceral pleura in a healthy person. Small amounts of protein then leak into the pleural space. Approximately 10% of the pleural fluid and large proteins are removed by the lymphatics at a rate of up to 20 mL/h for each hemithorax.[2] Ventilation and muscular activity facilitate the action of the lymphatics.[3] Alterations in pleural fluid homeostasis will lead to a pleural effusion or the pathologic collection of excess fluid between the visceral and parietal pleura.[19] Hydrostatic changes result in protein-free effusions or transudates. Changes in oncotic pressure in the lung or pleura lead to effusions. The differential diagnosis of transudates and exudates is listed in **Table 52-1**.

TABLE 52-1 Differential Diagnosis of Fluid Exudates and Fluid Transudates in the Pleural Space

Exudates	Transudates
Asbestos exposure	Atelectasis
Collagen vascular disease	Cirrhosis of the liver with ascites
Drug-induced	Congestive heart failure
Empyema	Nephrotic syndrome
Esophageal rupture	Peritoneal dialysis
Idiopathic	Pulmonary embolism
Malignancy	
Pancreatitis	
Parapneumonic	
Pulmonary embolism	
Rheumatoid arthritis	
Systemic lupus erythematosus	
Thoracic duct exposure	
Trauma	
Tuberculosis	
Viral	

INDICATIONS

Thoracentesis may be performed to relieve the patient's symptom of dyspnea or hypoxia. It is performed whenever a pleural effusion interferes with normal respiration, results in respiratory compromise, or results in hemodynamic compromise. Thoracentesis may be performed to remove pleural fluid for analysis to diagnose the etiology of the fluid (e.g., infection or malignancy).

CONTRAINDICATIONS

Absolute contraindications include an uncooperative patient or a patient who refuses to give informed consent for the procedure.[20] Uncooperative patients or patients with altered levels of consciousness who emergently need thoracentesis may require sedation for the procedure.

There are numerous relative contraindications to performing a thoracentesis. Patients receiving anticoagulants, with a known or suspected bleeding diathesis, or with thrombocytopenia have a significant risk of bleeding.[2,20,21] Consider reversing the anticoagulant or the bleeding disorder prior to performing the thoracentesis. A small volume of pleural fluid may make the procedure difficult to perform and increase the risk of complications.[20] Patients undergoing positive-pressure ventilation (e.g., bilevel positive airway pressure, continuous positive airway pressure, or using mechanical ventilation) are at an increased risk of developing a pneumothorax and a tension pneumothorax.[1] Pleural adhesions (i.e., a loculated pleural effusion) may limit the amount of fluid obtained or require multiple thoracenteses to drain the fluid.[1] Drain loculated pleural effusions under US guidance. Avoid areas of cellulitis or other infections on the chest wall unless no alternate site can be identified.[1,3] Unsupervised physicians with little or no experience should not perform this procedure, as the risk of complications is increased.[22] Patients with chronic obstructive pulmonary disease are at increased risk for complications.

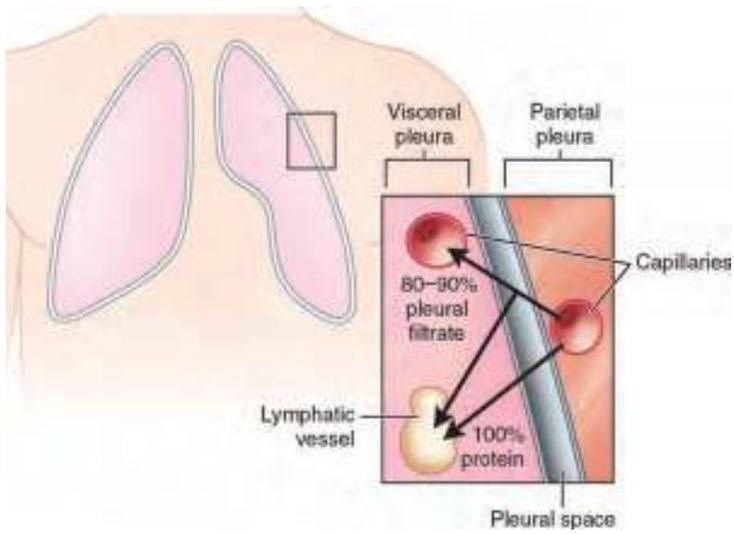


FIGURE 52-1. Schematic of pleural fluid homeostasis in a normal lung.

EQUIPMENT

DIAGNOSTIC THORACENTESIS FOR PLEURAL EFFUSIONS

- Sterile gloves and gown
- Face mask with a face shield or goggles
- Lidocaine, 1% or 2%
- Purple top (EDTA) collection tube
- Heparin, 1000 U/mL
- Atropine
- Alcohol pads
- Povidone iodine or chlorhexidine solution
- Gauze 4 × 4 squares
- Sterile drapes
- Sterile towels
- Sterile gloves
- Band-Aids
- 25 or 27 gauge needle
- 21 and 22 gauge needles, 1.5 inches long
- 10 mL syringes
- 50 mL syringe
- 18 or 20 gauge needle

THERAPEUTIC THORACENTESIS FOR PLEURAL EFFUSIONS

- The supplies listed above
- 16 to 18 gauge catheter-over-the-needle
- Three-way stopcock
- Connector tubing (connects to three-way stopcock and sterile container)
- Sterile container for pleural fluid
- 50 mL syringe
- Intravenous (IV) extension tubing

US GUIDANCE

- US machine
- 3.5 to 5.0 MHz phased-array US transducer
- Sterile US gel
- Sterile US transducer cover

Commercial kits are available to perform a thoracentesis (**Figure 52-2**). The kits are disposable, intended for single-patient use, and usually contain most of the required equipment. They save time in that the equipment does not have to be gathered and set up. Disadvantages include potential increased cost and limited equipment in the kit.

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative and obtain a signed consent form.[1] The position of the patient can vary depending on their clinical condition. Place ambulatory and cooperative patients sitting up at the edge of a bed with their feet on the floor or a stool (**Figure 52-3**). Place the patient's head and arms on an elevated bedside tray. The patient's back should be as vertical as possible so that the lowest part of the hemithorax is posterior. This will ensure that the free-flowing fluid remains posteriorly.[1-3,20]

One of three other positions are recommended in debilitated patients. Place the patient in the lateral decubitus position and lying on the side of the pleural effusion. The patient's back should be along the edge of the bed. The procedure will be performed in the midscapular line or the posterior axillary line. Place the ventilator-dependent patient into the lateral decubitus position and lying on the side with the pleural effusion.[23] Alternatively, place the ventilator-dependent patient supine and elevate the head of the bed as maximally as possible. The patient will be sitting with the assistance of the bed, and the procedure will be performed in midaxillary or posterior axillary line.[3] Finally, place the patient supine and perform the procedure in the midaxillary or posterior axillary line. US may be required to locate the pleural fluid with the patient supine. **Sedation or paralysis may be needed for optimal positioning depending on the patient's clinical condition.**

Prepare the patient. Clean any dirt or debris from the skin. Identify the anatomic landmarks required to perform the procedure. View the chest radiograph or US imaging to estimate the amount of pleural fluid. Percuss from superior to inferior starting at the midscapular or posterior axillary line. **Choose a single interspace below the top of the dullness to percussion for aspiration.** US may be used to locate the fluid.[6,20] US is comparable to CT for diagnosing and managing a pleural effusion.[24]

Place the patient on the cardiac monitor, noninvasive blood pressure cuff, pulse oximetry, and supplemental oxygen to monitor them during and after the procedure as an added level of precaution. Apply povidone iodine or chlorhexidine solution to the skin surface and allow it to dry. Apply sterile drapes around the site of the procedure. Have atropine at the bedside. It can be administered intramuscularly, intravenously, or subcutaneously to patients who develop symptomatic bradycardia during the procedure. Don personal protective equipment to protect from contact with the patient's blood and body fluids as well as protect the patient from an iatrogenic infection.

Prepare the US transducer if using US with the procedure. Have an assistant apply US gel over the footprint of the transducer. Instruct the assistant to place the gel-coated US transducer in the sterile cover while you hold it. Have the assistant place sterile US gel on the patient or the covered transducer.

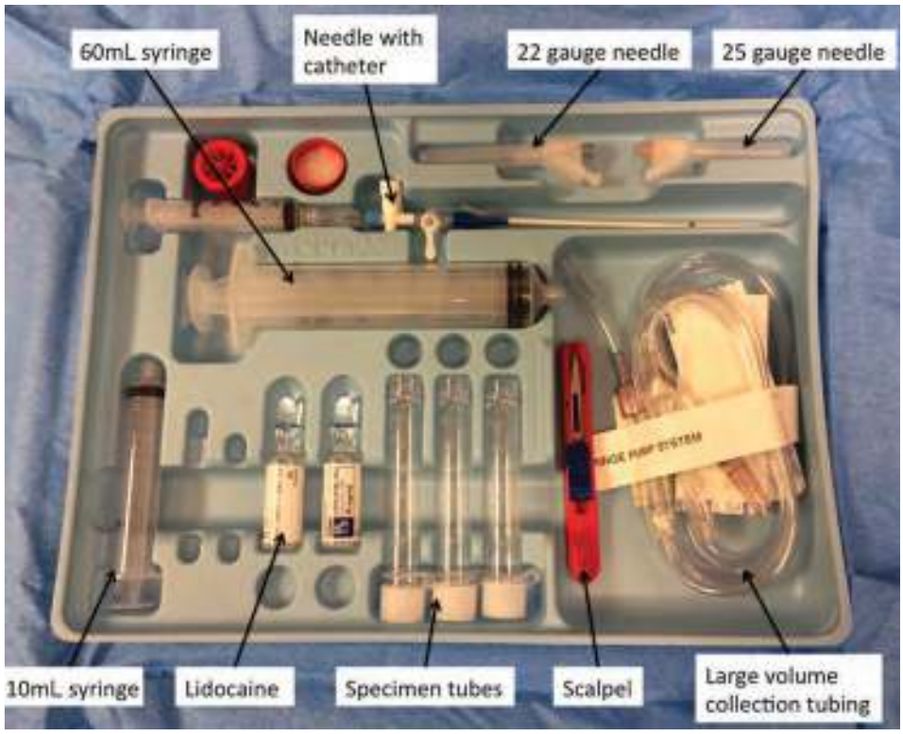
Place a skin wheal of local anesthetic solution over the thoracentesis site using a 25 or 27 gauge needle on a 10 mL syringe. Remove the 25 or 27 gauge needle from the syringe. Apply a 21 or 22 gauge, 1.5 to 3.0 inch needle to the syringe containing the local anesthetic solution. Anesthetize the subcutaneous tissues and the periosteum along the superior aspect of the rib (**Figure 52-4**). Walk the needle up the rib while simultaneously injecting local anesthetic solution. Gently aspirate prior to injecting each time the needle is advanced to ensure that the needle is not injecting within a blood vessel. Slowly and carefully advance the needle over the rib while applying negative pressure on the syringe when the superior border of the rib is located. **Be sure not to insert or advance the needle below the rib to avoid injury to the neurovascular bundle inferior to the rib.** Fluid will flow into the syringe when the pleural space has been entered. Inject and aspirate small volumes (e.g., 1 to 2 mL) while the needle is within the pleural cavity. This will distribute the local anesthetic solution into the pleural fluid and ensure anesthesia of the pleura. Withdraw the needle from the pleural cavity and out of the skin. Hold pressure to the skin puncture site to tamponade any bleeding.

DIAGNOSTIC THORACENTESIS TECHNIQUE FOR PLEURAL EFFUSIONS

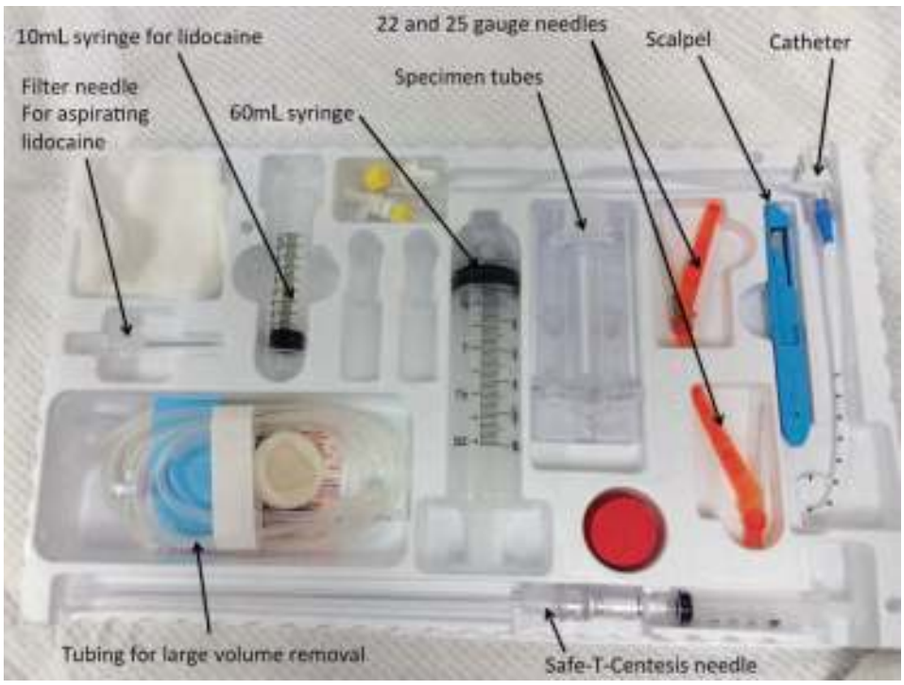
Attach an 18 gauge needle to a 50 mL syringe. Introduce the needle through the anesthetized track and into the pleural cavity (**Figure 52-5*B***). Aspirate up to 50 mL of fluid. Withdraw the

needle and place the fluid into the appropriate sterile containers. One of the containers should be a purple top (EDTA) collection tube to obtain a cell count and differential. An alternative is to collect the sample in a 50 mL syringe containing 1 mL of heparin (1000 U/mL) to ensure accurate cell counts, accurate pH, and prevent the fluid from clotting. **Use proper techniques when transferring the fluid into containers or specimen tubes to prevent a needlestick injury.**

A "dry tap" occurs if pleural fluid cannot be aspirated. Consider that the needle may be too short to reach the fluid, positioned too high to reach the fluid (**Figure 52-5*A***), positioned too low to reach the fluid (**Figure 52-5*C***), or that there may not actually be



A



B

FIGURE 52-2. Examples of thoracentesis kits. ***A.*** Pleura-Seal kit. ***B.*** Safet-kit. ***C.*** Thora-Para catheter drainage system. ***D.*** Emergency thoracentesis kit. ***E.*** Wayne pneumothorax kit. (Photos A and B courtesy of Jason Williams, MD, at www.proceduralist.org.)

C

E

D

FIGURE 52-2. (*Continued*)

an effusion. Repeat the physical examination and review the chest radiograph to reconfirm the presence of a pleural effusion. Use US, if available, to assess for the presence of a pleural effusion. Penetration of the lung with the needle is rarely catastrophic but can result in a pneumothorax.[3]

The principles are the same for debilitated patients except the site for the procedure. Use the midaxillary or posterior axillary line if the patient is supine. **Be cautious of the diaphragm as it can be as high as the fifth interspace on expiration at the anterior axillary line.** Use the midscapular line or the posterior axillary line if the patient is in the lateral decubitus position.

THERAPEUTIC THORACENTESIS TECHNIQUES FOR PLEURAL EFFUSIONS

The same sterile preparation, location of fluid, positioning, and anesthesia considerations apply for therapeutic thoracentesis. **The main difference between a diagnostic and therapeutic thoracentesis is the quantity of fluid removed.** Up to 1.5 L is removed in a therapeutic thoracentesis. A diagnostic thoracentesis requires approximately 10 to 20 mL of pleural fluid.

CATHETER-OVER-THE-NEEDLE TECHNIQUE

The catheter-over-the-needle (i.e., angiocatheter) technique is most commonly used to perform a therapeutic thoracentesis (**Figure 52-6**). Make a small "nick" in the skin with a #11 surgical blade at the anesthetized needle insertion site. Attach a 14 to 18 gauge catheter-over-the-needle to a 10 mL syringe as a handle. Insert the catheter-over-the-needle into the nick and advance it along the anesthetized tract (**Figure 52-6*A***). Apply negative pressure to the syringe as the catheter-over-the-needle is advanced. Stop advancing the catheter-over-the-needle when fluid is aspirated. Angle the catheter-over-the-needle caudally. Securely hold the syringe and needle so they do not move. Advance the catheter until the hub is against the skin (**Figure 52-6*B***). Withdraw the needle and syringe as a unit while the catheter remains in the pleural cavity (**Figure 52-6*B***). **Quickly cover the catheter with a gloved finger when the needle is removed. This will prevent ambient air from entering the pleural cavity.** Attach IV catheter extension tubing to the hub of the catheter. Place a three-way stopcock attached to a 50 mL syringe onto the extension tubing. Hold the catheter hub against the skin securely. Aspirate fluid into the syringe and then advance the fluid into the sterile container by adjusting the three-way stopcock.

An alternative option is to set up a siphon through the three-way stopcock (**Figure 52-7**). Prime the tubing with pleural fluid. Place the end of the tubing into a sterile container that is located below the site of the catheter. Alternatively, apply a needle onto the end of the IV tubing and insert it into a suction container. This allows the fluid to flow freely into the sterile container. Fluid can be removed in 50 mL aliquots up to 1.5 L. Limit the amount removed to 1.5 L due to the risk of postevacuation pulmonary edema and excessive protein loss.[1]

FIGURE 52-3. Recommended positioning of an ambulatory patient for a diagnostic or therapeutic thoracentesis for the evacuation of fluid.[2]

SELDINGER TECHNIQUE

An alternative approach is to use the Seldinger technique to insert a small-bore catheter into the pleural cavity. The major disadvantages to this technique include the time it takes to insert the catheter, the cost of the catheter kit versus a catheter-over-the-needle, and catheter blockage (e.g., from cells, debris, and protein) requiring insertion of a second catheter. Another option is to use a central venous catheter kit as the larger catheter opening may not become obstructed as easily.[25,26] Refer to Chapter 63 for the complete details regarding using a central venous catheter and the Seldinger technique.

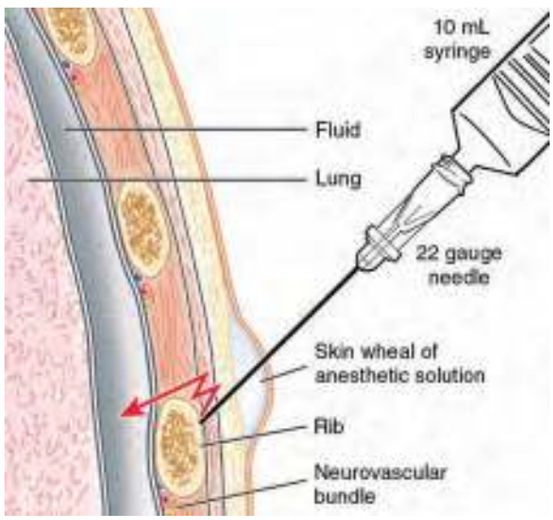


FIGURE 52-4. Administration of local anesthesia. A skin wheal is made. The needle is inserted through the skin wheal, while local anesthetic solution is injected to anesthetize the subcutaneous tissues and the periosteum of the rib. The needle is "walked" above the upper border of the rib (red jagged line) to avoid the neurovascular bundle inferior to the rib. The intercostal muscles, parietal pleura, and pleural space are then infiltrated with local anesthetic solution.

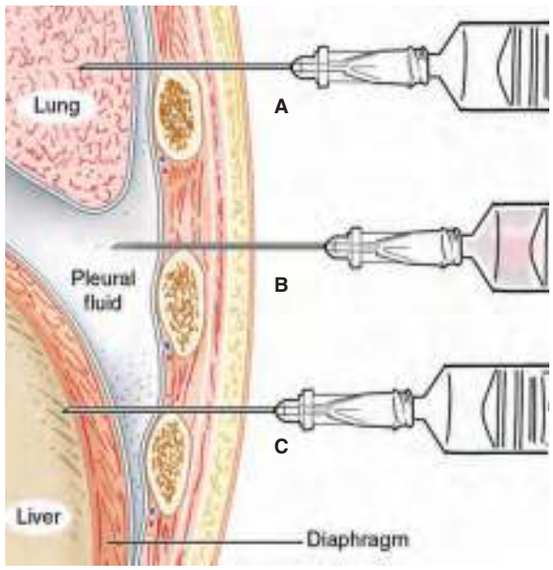


FIGURE 52-5. Needle positioning for a diagnostic thoracentesis. ***A.*** The pleural space is entered above the effusion (too high). ***B.*** The pleural space is entered properly, over the rib and into the fluid. ***C.*** The needle is too low and enters the abdominal cavity below the diaphragm.

ULTRASOUND-GUIDED TECHNIQUE FOR PLEURAL EFFUSIONS

US can be used to map the location and the extent of a pleural effusion.[27,28] It will help identify the appropriate site of needle entry. A 3.5 to 5.0 MHz phased-array transducer or 7.5 to 10 MHz high-frequency linear transducer is recommended.[29] Orientation of the transducer follows the convention that the marker should correlate to the reference point in the left upper corner of the monitor screen.

Percuss and auscultate the posterior thorax to estimate the location of pleural fluid with the patient in an upright sitting position. Apply sterile US gel onto the US transducer cover. Place the transducer at the intercostal space of the estimated level of pleural fluid in the posterior axillary line, usually at the level of ribs 9 to 11. Sweep the transducer inferiorly, superiorly, and transversely to assess the

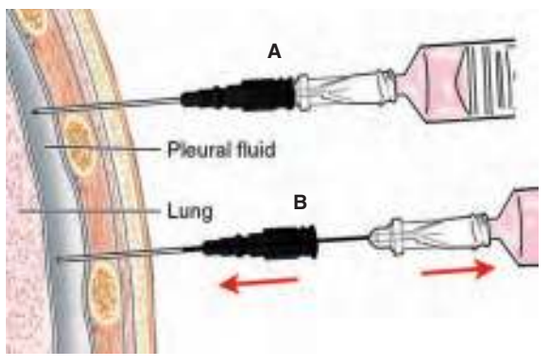


FIGURE 52-6. The catheter-over-the-needle technique. ***A.*** The needle and catheter are inserted over the rib and aimed slightly caudally into the pleural cavity. ***B.*** The catheter is advanced into the pleural cavity, and the needle is removed.

FIGURE 52-7. Equipment for continuous pleural fluid drainage includes a stopcock, IV tubing, empty evacuated suction container, syringes for anesthesia and fluid collection, catheter-over-the-needle, and an 18 gauge needle.

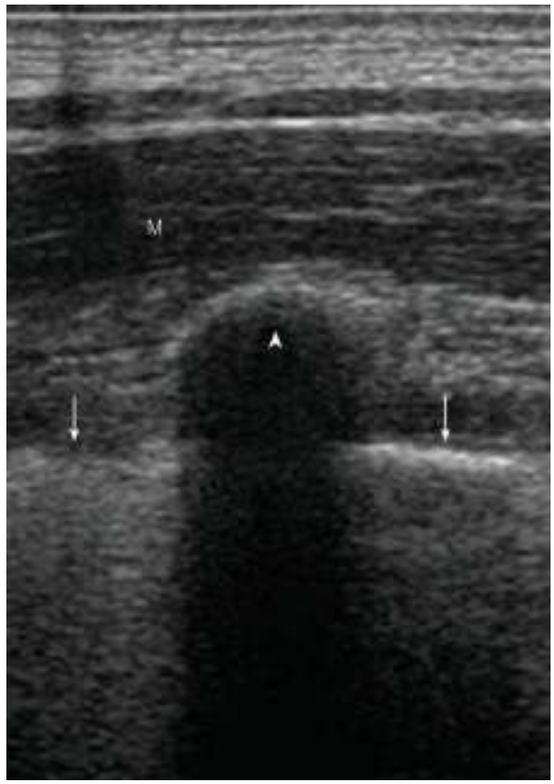


FIGURE 52-8. Ultrasound image of a normal lung. Visualized from top to bottom are the subcutaneous tissues, muscle (M), rib shadow (*arrowhead*), and pleural line (*arrows*).

location and size of the fluid collection. Identify the liver on the right, the spleen on the left, and the diaphragm.

Place the US transducer in the midscapular line or, if possible, the posterior axillary line to locate the fluid in a debilitated patient in the lateral decubitus position. Place the ventilator-dependent patient in the lateral decubitus position. Place the transducer in the posterior or midaxillary line if the patient is supine. **The movement of the diaphragm can be used as a key reference point when examining the pleural space.**[30] The liver may be used as an echogenic reference point for the identification of adjacent hyperechoic or hypoechoic structures. **The best window to view the intrathoracic contents is through the intercostal space as US penetration through the soft tissues is superior to that of rib bone or rib cartilage.**

The US waves will penetrate the skin, subcutaneous tissue, and muscle to produce multiple layers of varying echogenicity. The echogenic ribs cast an acoustic shadow (**Figure 52-8**). The parietal and visceral pleura are encountered posterior to the rib as two hyperechoic lines < 2 mm thick.[30,31] The diaphragm is identified as a hyperechoic transverse structure at the base of the chest wall. The lung is visualized as a bright, hyperechoic structure just cephalad to the diaphragm. The lung becomes more intensely hyperechoic or brighter during inspiration. A pleural effusion is identified as an anechoic to hypoechoic image above the diaphragm that decreases in size with inspiration (**Figure 52-9*A***). The lung swings during the respiratory cycle in the pleural effusion (**Figure 52-9*B***).

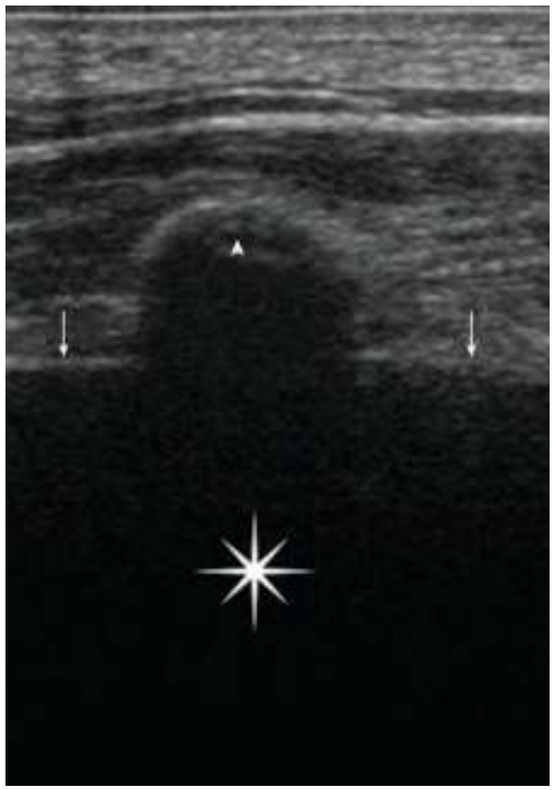
Two additional US findings should be identified on examination. The movement of the lung to the surrounding parietal pleura with inspiration and expiration produces an artifact referred to as "lung slide" or the "seashore sign" (**Figure 52-10**).[32] Normal inspiration produces a "slide" with each breath. This represents movement between the visceral and parietal pleural interface. The thin, hyperechoic sliding line is located approximately 0.5 cm below the surface of the rib and should move back and forth with each inspiration.[33]

A "comet tail artifact" is another normal finding that can be readily identified in a healthy patient (**Figure 52-11**).[32] The comet tail artifact is identified as a hyperechoic vertical artifact that slides transversely and is oriented perpendicular to the transverse "lung slide."[33]

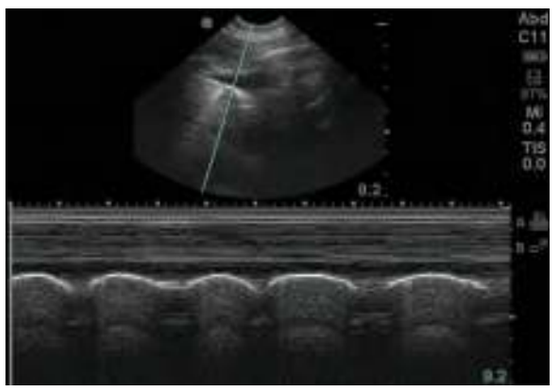
A pleural effusion is easily visualized using US (**Figure 52-9**). It appears black or anechoic. A distinct hyperechoic pleural line is not seen as the parietal and visceral pleura are separated by the effusion. Lung tissue appears hyperechogenic compared with the anechoic effusion (**Figure 52-12**). The pleural effusion can be seen moving during the respiratory cycle.

The posterior approach is the preferred choice in the stable, cooperative patient who sits upright and leans over a table (**Figure 52-13**). Scan the posterior hemithorax from the inferior border of the scapula to the upper lumbar region and from the paravertebral area to the posterior axillary line to survey the lung anatomy and map the effusion. Note the minimum depth of the effusion and the location of other vital structures (i.e., diaphragm, liver, lung, and spleen). Scan with the transducer perpendicular to the ribs and observe the structures during the full respiratory phase. The diaphragm can go as low as the twelfth rib posteriorly and as high as the eighth rib laterally. Determine the location of the skin entry site. Mark the site with a pen, surgical marker, or by indenting the skin with the cap of a needle. **The ideal site should have a large area of pleural effusion and be free of any internal structures (i.e., liver, spleen, or diaphragm) along the needle path.**

The lateral approach is used for mechanically ventilated patients and for those unable to sit up for the procedure. Abduct the ipsilateral arm and place the hand behind the patient's head (**Figure 52-14**). Survey the anterior and lateral thorax with US from the midclavicular line to the posterior axillary line. Note the depth of the effusion and the location of any vital structures to be avoided. Determine and mark the skin entry site.



A



B

FIGURE 52-9. Ultrasound image of a pleural effusion. ***A.*** The pleural effusion appears black or anechoic (*asterisks*). The pleura is not brightly echogenic (*arrows*) due to the separation of the two layers. Note the rib shadow (*arrowhead*) in the upper part of the image. ***B.*** Sinusoidal sign of lung movement in the pleural fluid during respiration. (Photo used with permission from Turner JP et al: Thoracic ultrasound. *Emerg Med Clin N Am* 2012; 30:451-473.)

The lateral decubitus approach is an alternative for patients unable to sit upright. Place the patient on their side with the pleural effusion side down. Use US to map the effusion. Note the distance from the skin to the effusion, the depth of the effusion, and the presence of any important structures to be avoided. Determine and mark the skin entry site.

Do not allow the patient to move once they have been scanned and the skin entry site marked regardless of the patient position or approach. Prep and drape the patient similar to that for the blind thoracentesis approach. Use local anesthetic to anesthetize the skin, rib, and pleura. The catheter-over-the-needle can be inserted using US guidance or blindly as described previously.

FIGURE 52-10. The sliding lung or seashore sign. Real-time US (*left*) and M-mode Doppler US (*right*). The *arrows* are the pleural interface. (Used with permission from reference 32.)

The use of US guidance can be helpful. Place a sterile cover over the US transducer and apply sterile US gel over the cover. Rescan the patient in the area previously identified as the skin entry site (**Figure 52-15**). Verify that no structures are along the needle path between the skin and the pleural effusion. Insert an 18 gauge catheter-over-the-needle into the pleural space using the technique already described while using US for visualization of the needle entering the pleural cavity. The needle will appear as a thin, bright, hyperechoic structure moving through the skin, subcutaneous tissue, parietal pleura, visceral pleura, and finally reaching the hypoechoic pleural effusion (**Figure 52-16**). Aspirate to confirm that the catheter-over-the-needle is within the pleural effusion. Place the US transducer aside. Continue the remainder of the procedure as described previously.

ASSESSMENT

Numerous analyses of the pleural effusion fluid are required to determine its etiology (**Tables 52-2 and 52-3**).[34] Large pleural effusions are more commonly associated with infections and

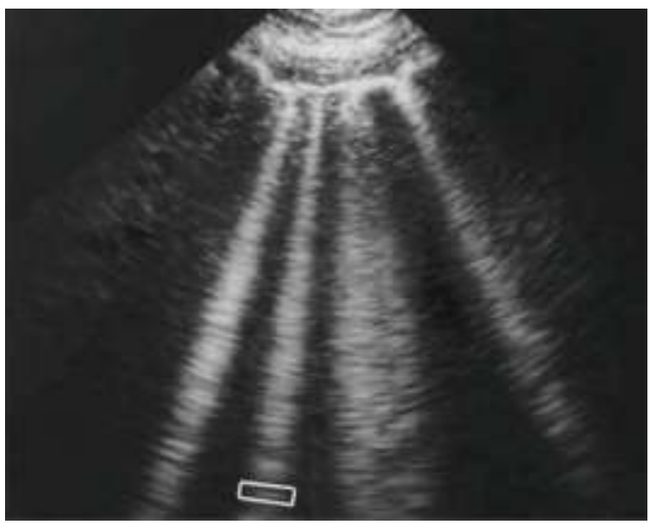


FIGURE 52-11. The lung rockets or comet tail artefacts. (Used with permission from reference 32.)

FIGURE 52-12. Ultrasound image of a pleural effusion (*asterisks*) with the underlying hyperechoic lung tissue (L). Note the rib shadow (*arrowhead*) in the upper left of the image.

malignancy.[35] Fluid analysis criteria have been established to separate transudates and exudates.[10] An exudate is present if the fluid fits one of the criteria in **Table 52-4**.[36] These parameters have been confirmed to have 98% sensitivity and 83% specificity in detecting exudates.[37] Color and odor can be helpful. Consider an infection if the fluid has a putrid odor. White or yellow fluid suggests an empyema or chylothorax. The white blood cell (WBC) count is of limited benefit. The fluid likely represents a parapneumonic effusion if the

FIGURE 52-13. Posterior approach in the sitting patient. A linear ultrasound transducer is seen here, although a phased-array transducer is often preferred to visualize deeper structures.

FIGURE 52-14. Lateral approach in the supine patient. A curvilinear transducer is positioned in the midaxillary line. Note the coronal plane of the transducer.

WBC count is > 10,000. A pleural fluid hemoglobin and hematocrit can be compared to that of the blood. Bloody pleural effusions are usually associated with malignancy, pneumonia, pulmonary embolism with a lung infarction, or trauma.[38] Consider a hemothorax if the fluid is grossly bloody. A hemothorax is likely and consider chest tube placement if the pleural fluid's hematocrit is > 50% of the serum hematocrit. Other helpful tests include a fluid pH. A pH below 7.25 to 7.30 is usually the result of an esophageal rupture, malignancy, parapneumonic process, or rheumatologic process. An elevated amylase level suggests esophageal rupture, malignancy, or pancreatic disease.[39] Elevated triglyceride levels suggest a chylothorax. Cytology is important to search for an underlying malignancy.[3] Bacteriologic information (e.g., Gram stain, acid-fast stain, and fungal stains) is important although the yield can be below 30%.[20]

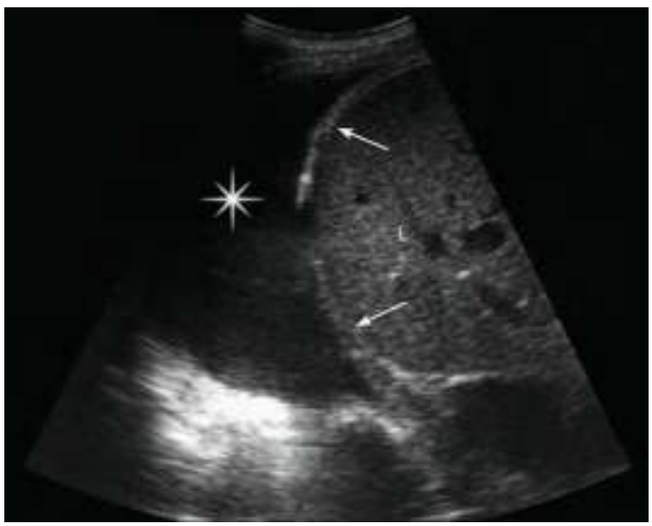


FIGURE 52-15. The pleural effusion (*asterisks*) is visible above the diaphragm (*arrows*). The liver (L) is seen below the diaphragm.

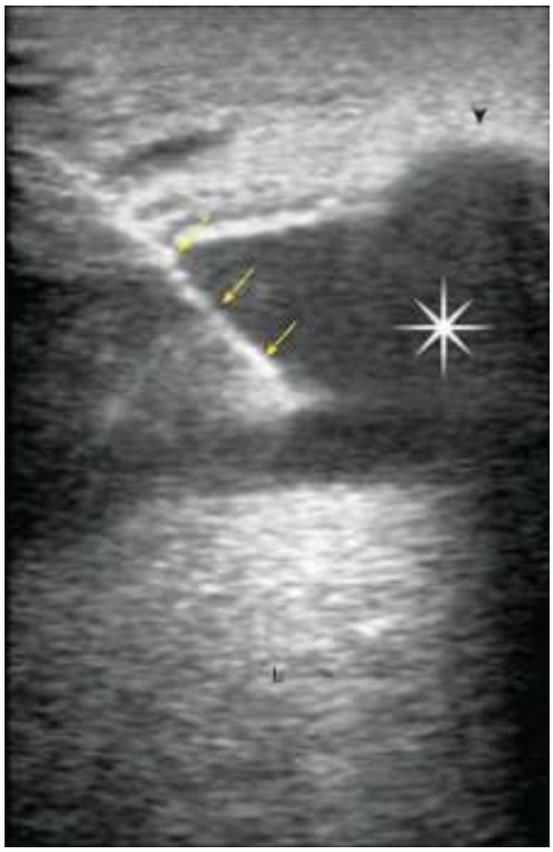


FIGURE 52-16. Long-axis view of a thoracentesis needle (*arrows*) being placed into a pleural effusion (*asterisks*) under US guidance. The rib shadow is denoted by an *arrowhead*.

TABLE 52-2 Laboratory Analysis of Pleural Effusions

Acid-fast stain
Amylase
Cell count and differential
Color
Cultures
Acid-fast
Aerobic
Anaerobic
Fungal
Glucose
Gram stain
Hemoglobin and hematocrit
pH
Triglycerides

TABLE 52-4 Laboratory Features of a Pleural Fluid Exudate[34]

Fluid/serum lactate dehydrogenase (LDH) > 0.6
Fluid/serum protein > 0.5
Pleural fluid LDH > 200 IU/mL
Pleural fluid LDH > 2/3 upper limit of normal for serum

Always send the fluid for aerobic and anaerobic cultures to rule out an infectious etiology for the pleural effusion. The appearance of pleural fluid on US may help identify whether the pleural fluid is a transudate or exudate. Transudates are consistently seen as anechoic, whereas exudates may range from anechoic to hyperechoic.[40,41]

AFTERCARE

Remove the catheter and apply a bandage to the puncture site when done aspirating fluid. Obtain a plain chest radiograph to assess for a pneumothorax. Repeat the chest radiograph in 4 to 6 hours to assess for a delayed pneumothorax.[42,43] An expiratory film is the best option to look for a pneumothorax, especially if it is small. Discharge the patient with good instructions and close follow-up if no pneumothorax is present and if appropriate for the clinical condition.

Instruct the patient to evaluate the procedure site two to three times a day for signs of infection. Educate the patient about the signs, symptoms, and significance of an infection. They should return to their Primary Physician or the Emergency Department immediately if they develop any concerns, fever, chills, shortness of breath, redness, or pus at the puncture site.

COMPLICATIONS

The potential complications of a thoracentesis are listed in **Table 52-5**.[1,3,11,20,22,37,44-47] Most studies report lower rates of postprocedural pneumothoraces with US-guided thoracentesis performed by an experienced operator.[9-11,15,48-54] Other studies have not shown a significant difference in the incidence of postprocedural pneumothoraces with the use of US.[55,56]

The use of the proper technique and Emergency Physician experience are important in reducing the rate of complications.[53,57-60] It is difficult to predict which patients may be at risk of developing a postprocedural pneumothorax.[61] Drainage of large volumes of fluid (i.e., greater than 1.5 to 2 L) may increase the risk of developing excessive protein loss, a pneumothorax, and reexpansion pulmonary edema.[1,62,63] Improper technique or tortuosity of the intercostal artery can result in intercostal artery injury.[64,65] Direct supervision of inexperienced operators, removal of small amounts of fluid, use of small-gauge needles, use of US for small effusions, and use of a needle-catheter system for a therapeutic procedure may all reduce the risk of an associated pneumothorax.[20]

TABLE 52-3 Common Etiologies of a Pleural Effusion and Their Associated Analysis

Etiology	Fluid type	Cholesterol (mg/dL)	Fluid LDH	Fluid/serum LDH	Fluid/serum protein	Glucose (g/dL)	pH
Congestive heart failure	Transudate	< 45	< 2/3 limit of serum	< 0.6	< 0.5	80–120	7.4–7.6
Infection	Exudate	> 45	> 2/3 limit of serum	> 0.6	> 0.5	< 60	< 7.3
Liver disease	Transudate	< 45	< 2/3 limit of serum	< 0.6	< 0.5	80–120	7.4–7.6
Malignancy	Exudate	> 45	> 2/3 limit of serum	> 0.6	> 0.5	< 60	< 7.3
Pulmonary embolism	Exudate	> 45	> 2/3 limit of serum	> 0.6	> 0.5	< 60	< 7.3

LDH, lactate dehydrogenase.

TABLE 52-5 Potential Complications Associated with a Thoracentesis

Cough
Hemothorax
Hypovolemia
Hypoxemia
Inadequate yield
Intrapleural infection
Laceration of an intercostal nerve or vessel
Laceration of the liver or spleen
Pain at the procedure site
Pneumothorax
Reexpansion pulmonary edema
Shortness of breath
Tension pneumothorax
Vasovagal reactions

PNEUMOTHORAX

ANATOMY AND PATHOPHYSIOLOGY

A thoracentesis can be performed to relieve a simple pneumothorax or a tension pneumothorax.[16] A pneumothorax is the presence of air between the visceral and parietal pleura.[16,17,33,66] Primary spontaneous pneumothoraces occur in otherwise healthy people without antecedent trauma. Secondary spontaneous pneumothoraces occur as a complication of underlying lung disease, most commonly chronic obstructive pulmonary disease.[1,3] A traumatic pneumothorax occurs from penetrating or blunt trauma to the thoracic cavity. An iatrogenic pneumothorax is a subcategory of the traumatic pneumothorax. The three most common etiologies for an iatrogenic pneumothorax are pleural biopsy, subclavian vein catheterization, and thoracentesis.[67]

The pressure in the pleural space is negative compared to the atmosphere. This causes the lung to collapse and the chest wall to expand. The alveolar pressure is greater than the pleural space pressure due to the elastic recoil of the lung. A communication between the alveolar and pleural space allows the air to preferentially move into the pleural space until the pressure equalizes. The physiologic consequence is a decrease in vital capacity and PaO_2. This may be well tolerated in healthy people but not in patients with underlying cardiac and/or pulmonary disease.

A one-way valve may allow air to enter the pleural space from the alveolus but not return. The intrapleural pressure will eventually exceed atmospheric pressure. A progressive increase in air occupying the pleural space leads to a tension pneumothorax. Clinical deterioration may occur due to a decreasing PaO_2, decreasing cardiac output, hypercarbia, and hypoxia.[68-71]

US guidance may aid in locating a pneumothorax, with the best viewing window being the intercostal space.[72,73] US can diagnose a pneumothorax within only 2 to 5 minutes compared to 20 to 30 minutes for chest radiography.[72,74] US showed a sensitivity and specificity of 100% and 94% for detection of pneumothoraces, compared to 36% and 100% for chest radiography, respectively.[75]

The presence or absence of lung sliding can be shown using M-mode Doppler US (**Figure 52-17**).[76] The lung normally shows "waves on a beach" or the "seashore." The anterior chest wall is stationary and is represented by a line. The presence of lung motion posteriorly is represented by an irregular and granular pattern. M-mode Doppler US of a pneumothorax is represented by repeating horizontal lines from the lack of lung sliding. This is known as the "barcode sign" or the "stratosphere sign."[76]

Controversy exists regarding the exact management of a spontaneous pneumothorax.[77-83] Options include simple aspiration, tube thoracostomy, simple aspiration followed by a tube thoracostomy if aspiration fails, and outpatient versus inpatient management. Simple aspiration is more likely to fail with larger pneumothoraces.[77-81,84] These studies noted that simple aspiration is associated with a reduction in the percentage of patients requiring hospitalization compared to tube thoracostomy. There were no differences between the two procedures in early failures, immediate success rates, duration of hospitalization, 1-year success rates, and the number of patients requiring a subsequent pleurodesis. Advantages of simple aspiration compared to tube thoracostomy include less equipment costs, easier and quicker to perform, and the potential to avoid hospitalization.

The size of a pneumothorax can be calculated in three ways.[61,85-87] Collins calculated the size as follows: size (%) = 4.2 + 4.7 (distances at apex + midpoint of the upper half of the collapsed lung + midpoint of the lower half of the collapsed lung).[87] The calculation was derived from helical CT scan data. It uses the distances determined on a PA chest radiograph and may overestimate the size of a large pneumothorax. Light determined the volume as follows: volume (%) = 100 – [(average diameter of lung)^3 ÷ (average diameter pneumothorax)^3 × 100].[61] This calculation uses the distances determined on a PA chest radiograph and may underestimate the size of a small pneumothorax. It is accurate for medium and large pneumothoraces. Rhea calculated the size as follows: size = (distances at apex + midpoint of the upper half of the collapsed lung + midpoint of the lower half of the collapsed lung) ÷ 3.[85,86] The pneumothorax size is converted to a volume using a normogram. It uses the

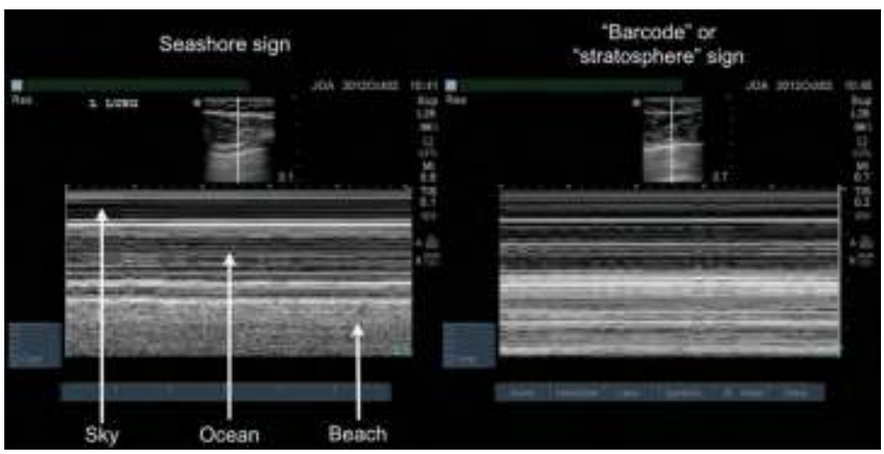


FIGURE 52-17. The normal US versus a pneumothorax. (Courtesy of Jacob Avila, MD, and www.5minsono.com.)

distances determined on a PA and lateral chest radiograph. It may underestimate the size of a large pneumothorax.

INDICATIONS

All tension pneumothoraces require needle drainage (Chapter 50) followed by tube thoracostomy (Chapter 51). Patients usually present with hypotension, neck vein engorgement, respiratory distress, tachycardia, and unilateral absence of breath sounds. These patients have tracheal deviation that is often difficult to assess and is often limited to the thoracic cavity.

Not all simple pneumothoraces require drainage as they may resolve spontaneously. A small pneumothorax in a healthy patient may be treated conservatively with observation alone which has shown a spontaneous resorption rate of 1.25% a day.[60] The application of supplemental inhaled oxygen can facilitate reabsorption of air in the pleural cavity up to fourfold faster.[88] Drain a pneumothorax if the patient complains of dyspnea, dyspnea on exertion, or pain, or if the pneumothorax is estimated to be 15% or greater.[83,89]

CONTRAINDICATIONS

There are no absolute or relative contraindications to relieving a tension pneumothorax since it is a life-threatening emergency.

Contraindications to thoracentesis to relieve a simple pneumothorax are few.[89] Select an alternative site if the primary site has an infection.[3] A traumatic pneumothorax or a pneumothorax associated with a hemothorax or empyema requires a tube thoracostomy. Any pneumothorax that is expanding or expanding despite thoracentesis requires a tube thoracostomy. Any patient on anticoagulation or with a possible bleeding diathesis may require reversal of the condition before the procedure.[1] A patient with minimal symptoms and a small pneumothorax may be observed before deciding to evacuate the pneumothorax.

EQUIPMENT

PNEUMOTHORAX—TENSION

- Alcohol swab, povidone iodine solution, or chlorhexidine solution
- 12 to 16 gauge catheter-over-the-needle, 2 inches long

PNEUMOTHORAX—STABLE

- Sterile gloves and gown
- Face mask with a face shield or goggles and cap
- Povidone iodine or chlorhexidine solution
- Sterile gauze sponges
- Sterile towels
- Sterile basin
- Syringes for anesthesia infiltration, 5 and 10 mL
- 25 gauge needle for anesthesia infiltration of the skin
- 21 or 23 gauge needle for infiltration of subcutaneous tissue, periosteum, and pleura
- 16 or 18 gauge catheter-over-the-needle
- Pigtail or straight catheter kit
- Three-way stopcock
- 50 mL syringe
- Intravenous extension tubing
- Heimlich valve

US GUIDANCE

- US machine
- 3.5 to 5.0 MHz phased-array US transducer
- 7.5 to 10 MHz high-frequency linear US transducer
- Sterile US gel
- Sterile US transducer cover

Commercial kits have been developed and are available to provide the equipment needed to perform a thoracentesis (**Figure 52-2**). These kits are disposable, single-patient use, and contain all the required equipment. They save time in that the equipment does not have to be found and set up. Disadvantages include potentially increased cost and limited equipment in the kit.[68]

Some alternative devices exist that combine the pigtail catheter and Heimlich valve in the same kit. They use a one-piece unit that combines an intrapleural catheter and an external one-way antireflux valve that attaches to the chest wall by an adhesive pad. Its insertion is quicker, easier, and simpler than a traditional catheter. The low profile makes ambulation and outpatient management easier for the patient. These devices are rarely available in the Emergency Department.

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative and obtain a signed consent form.[1] Place the patient supine on the bed. Alternatively, the patient may be supine with the head of the bed elevated to 30° (**Figure 52-18**). Clean any dirt or debris from the skin. Identify the anatomic landmarks required to perform the procedure. It is recommended to place the patient on the cardiac monitor, noninvasive blood pressure cuff, pulse oximetry, and supplemental oxygen, although not required. Apply povidone iodine or chlorhexidine solution to the skin and allow it to dry. Apply sterile drapes around the site of the procedure. Have atropine available at the bedside. It may be administered intramuscularly, intravenously, or subcutaneously to patients who develop symptomatic bradycardia during the procedure. The most common approach is the second intercostal space in the midclavicular line (**Figure 52-18**). An alternate site is the fourth or fifth intercostal space in the midaxillary line. Don full personal protective equipment to protect from contact with the patient's blood and body fluids as well as protect the patient from an iatrogenic infection.

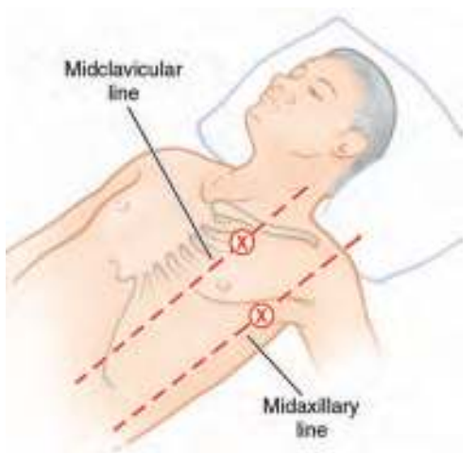


FIGURE 52-18. Relief of a tension pneumothorax. Place the patient supine with the head of the bed elevated 30° if not contraindicated. The second intercostal space in the midclavicular line is the recommended site. For pleural effusions or a debilitated patient, the midaxillary line or posterior axillary line may be used at the level of the fourth or fifth intercostal space.

TENSION PNEUMOTHORAX TECHNIQUE

Thoracentesis is a diagnostic and therapeutic procedure if the patient has a tension pneumothorax. A tension pneumothorax is a true life-threatening condition. Identify the needle insertion site by palpating the second intercostal space in the midclavicular line. US guidance is not necessary prior to needle decompression and should not delay treatment if a tension pneumothorax is clinically evident. **Insert the catheter-over-the-needle over the superior border of the third rib to avoid the neurovascular bundle located on the inferior border of the second rib.**[16] Advance the catheter-over-the-needle into the pleural space. A 5 or 10 mL syringe without the plunger can be attached to the catheter-over-the-needle. The syringe barrel can be used as a handle to advance the catheter-over-the-needle.

A release of pressure and a small "pop" may be felt when the pleural cavity is entered. Stop advancing the needle. Securely hold the needle so it does not move. Advance the catheter until the hub is against the skin. Remove the needle. A continuous rush of air may be heard or felt if the patient has a tension pneumothorax. **Needle thoracentesis for a tension pneumothorax is a temporizing measure. Perform a tube thoracostomy (Chapter 51) immediately after this life-saving procedure.**[90]

SIMPLE PNEUMOTHORAX TECHNIQUES

A primary spontaneous pneumothorax occupying over 15% of the hemithorax is the indication for simple aspiration via a thoracentesis.[3] The same sterile preparation, location of fluid, positioning, anesthesia considerations, and US-guided technique apply to the evacuation of a simple pneumothorax as to a diagnostic thoracentesis. Spontaneous pneumothoraces used to be drained by tube thoracostomy; however, simple aspiration may be just as effective.[77-82]

CATHETER-OVER-THE-NEEDLE TECHNIQUE

The catheter-over-the-needle technique is most commonly used for drainage of a spontaneous pneumothorax (**Figure 52-6**). Anesthetize the site marked for needle insertion by making a small wheal under the skin. Make a small "nick" in the skin with a #11 surgical blade at the needle insertion site. Attach a 14 to 18 gauge catheter-over-the-needle to a 10 mL syringe as a handle. Insert the catheter-over-the-needle into the nick and advance it along the original anesthetized tract (**Figure 52-6*A***). **Insert the catheter-over-the-needle over the superior border of the rib to avoid the neurovascular bundle located on the inferior border of the rib.**[16] Apply negative pressure to the syringe as the catheter-over-the-needle is advanced. Stop advancing the catheter-over-the-needle when air is aspirated. Angle the catheter-over-the-needle superiorly. Securely hold the syringe and needle so they do not move. Advance the catheter until the hub is against the skin. Withdraw the needle and syringe as a unit while the catheter remains within the pleural cavity (**Figure 52-6*B***). **Quickly cover the catheter with a gloved finger when the needle is removed. This will prevent ambient air from entering the pleural cavity.** Attach IV catheter extension tubing to the hub of the catheter. Place a three-way stopcock attached to a 50 mL syringe onto the extension tubing. Hold the catheter hub against the skin securely. Aspirate air into the syringe and then advance the air into the room by adjusting the three-way stopcock.

Air is withdrawn manually. Continue the process until resistance is felt. **It is presumed that reexpansion has not occurred and a continual leak of air exists from the lung into the pleural cavity if no resistance is felt after 2.5 L of aspiration. Perform a tube thoracostomy (Chapter 51).** Close the stopcock after no more air is aspirated and secure it to the chest wall. The success rate for the aspiration of a pneumothorax is approximately 60%.[91-93]

SELDINGER TECHNIQUE

Drainage can be accomplished using a pigtail or straight catheter and the Seldinger technique (**Figure 52-19**). An alternative is to use a central venous catheter if a pigtail catheter is not available.[68,94] Attach a 16 gauge, 2 inch catheter-over-the-needle to a 5 or 10 mL syringe. Insert the catheter, as described above, aimed superiorly. **Insert the catheter-over-the-needle over the superior border of the rib to avoid the neurovascular bundle located on the inferior border of the rib.**[16] Securely hold the needle and syringe so it does not move. Advance the catheter to the hub. Remove the needle and syringe. **Quickly cover the catheter hub with a gloved finger.** Insert the guidewire through the catheter (**Figure 52-19*A***). **Hold the guidewire securely to prevent it from falling completely into the pleural cavity.** Remove the catheter over the guidewire and leave the guidewire in place (**Figure 52-19*B***). Extend the skin incision with a #11 scalpel blade by 3 to 5 mm to allow the catheter into the pleural space without "crumpling" (**Figure 52-19*C***). Advance the dilator over the guidewire and into the pleural cavity to dilate the tract (**Figures 52-19*C* and 19*D***). A gentle twisting motion of the dilator as it is advanced will aid in its insertion into the pleural cavity (**Figure 52-19*D***). Hold the guidewire securely. Remove the dilator while leaving the guidewire in place. Insert the catheter over the guidewire and into the pleural cavity (**Figure 52-19*E***). Remove the guidewire and attach a three-way stopcock to the catheter. Aspirate the air as described previously.

DRAINAGE SYSTEMS

Drainage systems for a pneumothorax vary in style but function with the same "one-way valve" principle. The simplest method is a flutter valve. It is best illustrated by the following noncommercial method. Cut a premoistened finger from a sterile glove. Tie the proximal end to the thoracentesis catheter with a silk suture and cut the distal end so that it is open to the air (**Figure 52-20**).[1] This creates a flutter valve and allows air to escape with coughing or expiration and prevents air from reentering the pleural space on inspiration.

Commercial kits are available that can be used for outpatient management of a pneumothorax in some patients. The kit often contains a Heimlich flutter valve (**Figure 52-21**).[1,66] **The arrow on the clear protective tube covering the Heimlich valve must point away from the patient.**[95] Suction is usually not needed. Outpatient management may be considered in stable patients with a primary spontaneous pneumothorax that is small and apical with initial lung reexpansion and have good apposition of the lung with the lateral chest wall.[96] **The patient must be able to understand and comply with outpatient instructions, must have access to immediate help, and must return to an Emergency Department if symptoms arise.** Additional requirements include good residual lung function, normal oxygen saturation, and an air leak adequately treated by thoracentesis.[22] **A contraindication to using the flutter valve is a hemothorax.** A closed underwater seal system is recommended in these cases. Refer to Chapter 51 for details regarding the use of a closed underwater seal system and the other indications for its use.

The Pneumostat (Maquet Getinge Group, Hudson, NH) is a single-use device (**Figure 52-22**). It attaches to a garment. The Pneumostat has multiple chest tube connectors. The air leak well provides quick and simple air leak confirmation. It has a 30 mL collection chamber and a needleless sampling port. It helps the patient ambulate sooner due to it being small and not cumbersome. It has a one-way valve for patient protection.

The TRU-CLOSE Thora-Vent (UreSil LLC, Skokie, IL) is an alternative device to the pigtail catheter and Heimlich valve combination (**Figure 52-23**). It is a one-piece unit that combines an intrapleural catheter and external one-way antireflux valve that attaches to the chest wall by an adhesive pad. Its insertion is quicker, easier,

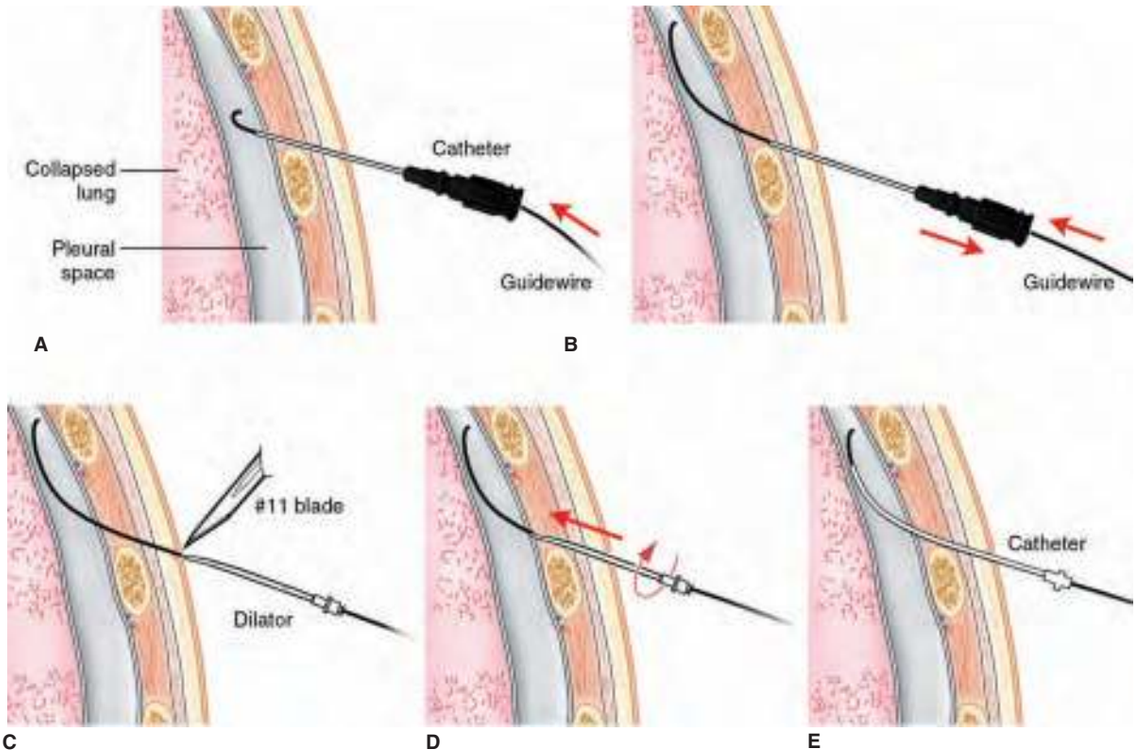


FIGURE 52-19. The Seldinger technique for inserting a catheter to aspirate a pneumothorax. A catheter-over-the-needle has been placed into the pleural cavity and aimed superiorly. The needle has been removed while the catheter remains in the pleural cavity. ***A.*** A guidewire is inserted through the catheter. ***B.*** The catheter is removed while the guidewire remains in the pleural cavity. ***C.*** The skin incision is enlarged with a #11 scalpel blade. A dilator is placed over the guidewire. ***D.*** The dilator is advanced over the guidewire and into the pleural cavity. A gentle twisting motion will help guide the dilator through the tract. ***E.*** The dilator has been removed while the guidewire remains inside the pleural cavity. The catheter is advanced over the guidewire and into the pleural cavity. The guidewire is then removed while the catheter remains inside the pleural cavity.

and simpler than a traditional catheter. It is inserted using a trocar. The diaphragm is used to indicate pleural entry and pneumothorax resolution. The low profile makes ambulation and outpatient management easier for the patient. This device is rarely available in the Emergency Department.

■ ULTRASOUND-GUIDED TECHNIQUE FOR A PNEUMOTHORAX

The same sterile preparation applies to US evaluation of a pneumothorax as for evaluation of a pleural effusion. A pneumothorax may be more readily identified with the patient positioned supine rather than upright with US. A 3.5 to 5.0 MHz phased-array transducer or 7.5 to 10 MHz high-frequency linear transducer is recommended.[29] The orientation of the transducer follows the convention that the marker should correlate to the reference point in the left upper corner of the monitor screen.

The key US features of a pneumothorax include the absence of inspiration-expiration–related "lung slide," loss of "comet tail" artifact, and broadening of the pleural line to a thick band (**Figure 52-17**).[31,76] Compare the hemithorax suspected of having a pneumothorax to the unaffected side to confirm these findings in the case of a unilateral pneumothorax (**Figure 52-17**). Aspirate a pneumothorax using the US similar to a pleural effusion.[97]

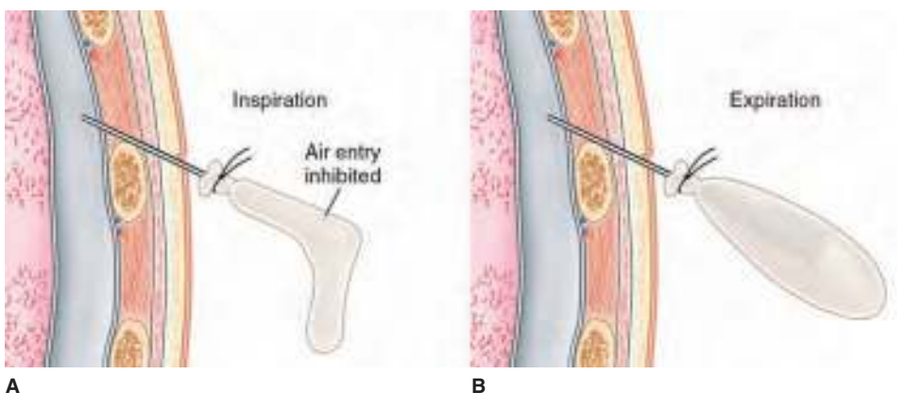
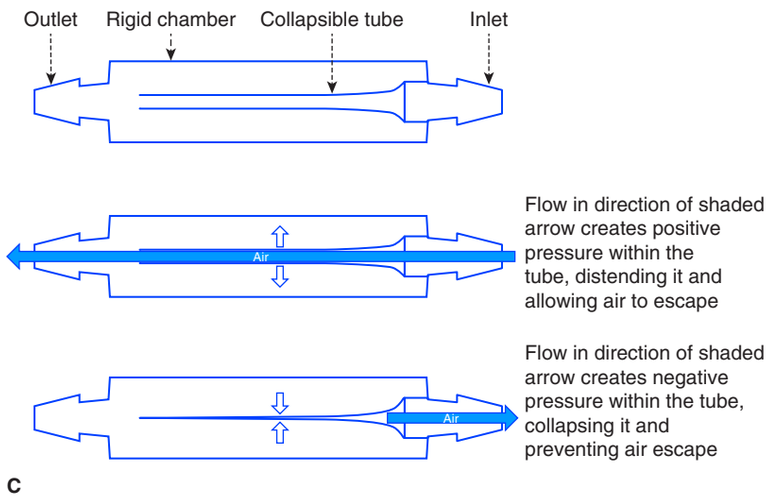


FIGURE 52-20. Use of a finger from a sterile glove as a one-way valve. Place the proximal end of the finger on the drainage system and cut the distal tip. It will act as a one-way valve.

A

B



C

FIGURE 52-21. The Heimlich valve. ***A.*** Picture of the device. ***B.*** Schematic illustration. ***C.*** A collapsed tube is tethered within the rigid cylindrical chamber. Positive pressure within the tube distends it and allows air to flow outward. Negative pressure within the tube collapses it and prevents reversal of flow. (Part C used with permission from reference 95.)

ASSESSMENT

Relief of a tension pneumothorax will equalize the pressure between the atmosphere and the pleural space. The patient will now have a simple pneumothorax. The vital signs should begin to normalize and the respiratory distress improve. The patient will need a tube thoracostomy (Chapter 51) followed by a chest radiograph if they had a tension pneumothorax. The patient may begin to cough when the lung expands. Breath sounds should be present bilaterally upon reexpansion.

There are two possibilities if the patient does not improve clinically after needle decompression for a tension pneumothorax. The pleural space may not have been entered with the needle. This occurs when the chest wall is thick (e.g., patient is obese or very muscular). The procedure may be repeated with a longer needle. The patient may not have had a tension pneumothorax. Reevaluate the patient by physical examination, bedside US, or review of the chest radiograph to determine if a tension pneumothorax is present.

Obtain a chest radiograph after the relief of a simple pneumothorax. It will allow the Emergency Physician to determine the success, or lack thereof, of the procedure. Perform a tube thoracostomy if at any point the pneumothorax is not improving with a thoracentesis or if the patient's symptoms worsen.

FIGURE 52-22. The Pneumostat.

AFTERCARE

Obtain a follow-up chest radiograph upon completion of the procedure to assess for a pneumothorax. An expiratory film may be helpful, especially if the pneumothorax was small. Repeat the chest radiograph 4 to 6 hours after the procedure to look for a delayed pneumothorax. The patient may be discharged with good instructions and close follow-up if no pneumothorax is present and if

A

B

FIGURE 52-23. The Thora-Vent. ***A.*** Kit. ***B.*** The device. (Photos courtesy of Uresil LLC.)

appropriate for their clinical condition.[98] An alternative is placement in an observation unit and discharge within 24 hours.

Instruct the patient to evaluate the procedure site two to three times a day for signs of infection. Educate the patient about the signs, symptoms, and significance of an infection. They should return to their Primary Physician or Emergency Department immediately if they develop any concerns, fever, chills, shortness of breath, redness, or pus at the puncture site.

A patient may be a candidate for outpatient management with a Heimlich valve if they have a stable primary spontaneous pneumothorax, close apposition of the lung to the lateral chest wall, Emergency Physician satisfaction with the position of the catheter, and an air leak that is manageable with one thoracentesis.[99-107] Contraindications for outpatient management include a hemothorax, a large air leak requiring tube thoracostomy, poor residual function, secondary pneumothoraces, traumatic pneumothoraces, unacceptable residual collapse defined as poor apposition of the lateral lung to the lateral chest wall, and unreliable patients.[18]

Instruct the patient to clean the thoracentesis site with mild soap daily, apply a split dressing around the catheter and tape it to the skin, make sure that the tubing is taped firmly to the valve to prevent accidental dislodgment, make sure the arrow on the valve points away from the patient, and make sure the sound of air exiting the valve is expected; showers are permitted, but not baths or swimming. Patients should call 911 and immediately return to the Emergency Department if they experience shortness of breath, chest pain, or difficulty breathing. **Instruct the patient on the removal of the Heimlich valve from the catheter if significant shortness of breath develops as they may have developed a tension pneumothorax.** Instruct the patient on the signs of an infection at the chest wall insertion site.

Arrange daily follow-up for radiographic evaluation. Remove the thoracentesis catheter upon complete inflation of the lung. Observe the patient for 4 to 6 hours after catheter removal and obtain a repeat chest radiograph. Discharge the patient if the lung is completely expanded. Perform a tube thoracostomy if the lung is not completely expanded.

COMPLICATIONS

Complications associated with this procedure can be numerous. A worsening pneumothorax may be caused by lacerating the lung with the needle, inadequate coverage of the hub of the needle or catheter with a gloved finger after entering the pleural space, an air leak in the drainage system, and a lung parenchymal-pleural fistula that can develop from poor lung expansion during large-volume pneumothorax drainage.[101] Puncture of the lung can cause a pulmonary contusion.[102] A tension pneumothorax can occur from a lung laceration along with inadvertent plugging of the drainage system (e.g., fluid in tube or kinking). A hemothorax is possible if the intercostal artery, lung, or mammary artery is lacerated with the needle.[89] Less common complications include cardiac or great vessel perforation due to poor positioning of the needle upon insertion. Infection occurs approximately 2% of the time if sterile technique is observed. Many of these complications can be prevented by using proper and careful technique. Aspiration of a pneumothorax can result in a recurrence and require a chest tube.[16,93,103]

Reexpansion hypotension has been reported following rapid evacuation of persistent unilateral pneumothoraces of at least 1 week in duration.[1,108] The mechanism is unclear. It is associated with reexpansion pulmonary edema that precipitates intravascular volume depletion, myocardial depletion, or large-volume aspiration.[1,108] Reexpansion pulmonary edema may be precipitated by pneumothoraces over a few days in duration before reexpansion or large-volume aspiration.[104-106]

PEDIATRIC CONSIDERATIONS

Thoracentesis in pediatrics is similar to the adult (**Table 52-6**).[107] Most information is extrapolated from adults or small pediatric case studies. A pneumothorax occurs less frequently in pediatric patients than adults. A spontaneous pneumothorax is more often seen in males and teenagers. No single mechanism explains the etiologies of a spontaneous pneumothorax. No equations exist for estimating the size of a pneumothorax. The recurrence rate is unknown.

SUMMARY

A thoracentesis can help differentiate between pleural effusions that are transudates and exudates. Together with the clinical presentation, the information can be useful in diagnosing the patient's

TABLE 52-6 Etiology of Pediatric Pneumothoraces

Barotrauma
Catamenial
Central line placement
Congenital lung malformations
Foreign body aspiration
Infection
HIV
Measles
Necrotizing abscesses
Necrotizing pneumonia
Parasites
Pneumocystis
Pneumonia
Tuberculosis
Inflammatory and connective tissue diseases
Alpha-1-antitrypsin deficiency
Birt-Hogg-Dubé syndrome
Dermatomyositis
Ehlers-Danlos syndrome
Juvenile arthritis
Langerhans cell histiocytosis
Marfan syndrome
Polymyositis
Sarcoidosis
Systemic lupus erythematosus
Lung biopsy
Lung disease
Asthma
Cystic fibrosis
Emphysema
Malignancy
Lymphoma
Metastasis
Mechanical ventilation
Radiation
Thoracentesis
Trauma

condition. It can be lifesaving in the treatment of a tension pneumothorax and offers an alternative to tube thoracostomy for patients with stable primary spontaneous pneumothoraces. Outpatient management can be considered in some cases with the addition of a Heimlich valve. Regardless of what method is used, physicians in training should be supervised until competency with this procedure is demonstrated.

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53 Open Chest Wound Management

Eric F. Reichman

INTRODUCTION

Open chest wounds come in a variety of shapes and sizes. Their one commonality is an open communication between the pleural space and the external environment. The wounds have often been sealed by the soft tissues of the chest wall in the vast majority of patients with penetrating injuries to the chest. The primary concern with these patients is the diagnosis and treatment of underlying thoracic, cervical, and/or abdominal injuries. **Rarely, small perforations may produce a valve-like entry into the pleural space, enabling air to be "sucked in" during inspiration but blocking air egress during expiration.[1-3] Thus air will continue to accumulate, leading to a tension pneumothorax requiring needle decompression followed by a tube thoracostomy**. Larger, more destructive wounds of the chest may also occur. These are most common in combat injuries. In civilian practice, they are often secondary to shotgun injuries. The larger wounds are also caused by high-velocity weapons, explosions, on-the-job injuries, propeller injuries, or fencepost impalements, to name a few. Clothing, wadding, shell fragments, and pieces of the chest wall may all be driven into the thoracic cavity. Such injuries are associated with physical loss of a portion of the chest wall itself, making adequate ventilation impossible.[4,5]

These wounds are known by numerous names including open chest wounds, open pneumothoraces, sucking chest wounds, and communicating pneumothoraces. These specific open chest wounds are the focus of this chapter.

Wounds of the chest are described in the earliest of medical documents, the Edwin Smith papyrus. This document dates from the time of Imhotep (3000 B.C.). During Greco-Roman times, open chest wounds were universally fatal. Galen cared for chest wounds in gladiators. Treatment consisted of a poultice and leaving the wound open. This treatment did not change until the time of Theodoric in 1267 who advised the closing of chest wounds.

Techniques for managing chest wounds have improved with each subsequent war. The most important treatable aspect of these chest wounds was the associated open pneumothorax. The question of whether to manage such injuries open or closed remained controversial. John de Vigo, in 1514, was the first surgeon to present his views on gunshot wounds of the chest. He thought them to be universally fatal and, for the most part, untreatable. William Hewson, in 1767, observed that a patient with a large open chest wound was not able to breathe but could do so easily once the injury was closed. It took another 40 years for Baron Larrey, Napoleon's Surgeon, to confirm Hewson's observation in a wounded soldier. Another famous accounting of an open chest wound was by William Beaumont in 1825. He arrived within one-half hour of the injury and saved the patient's life by closing his chest wound.

By the final years of World War I, the controversy of "to close or not to close" was resolved in favor of immediate wound closure. However, when closing these wounds, it was important to understand the physiology of negative intrathoracic pressure. The German internist Buelau introduced closed underwater seal drainage of an empyema in 1875. In 1889, T. Holmes, a consulting Surgeon at St. George's Hospital in London, introduced intercostal drainage for large chest wounds. It was not until World War II that closed tube drainage was added to the treatment of an open pneumothorax as a routine measure. Positive-pressure ventilation was introduced in the early 1900s. The most recent advancement in the treatment of these injuries came from the Scandinavians, who invented respirators in the early 1950s. Mortality from chest wounds steadily decreased in each war. It was 79% in the Crimean War, 62.5% in the Civil War, 55.7% in the Franco-Prussian War, 24.6% in World War I, and 12% in World War II; in recent civilian experience, it is now 4% to 7%.[5]

ANATOMY AND PATHOPHYSIOLOGY

The pathophysiology of an open pneumothorax has not much been improved upon since the days of Hewson and Larrey. The pathophysiologic changes of a sucking chest wound depend on the size of the wound and the intactness of the pleural space and lung. A defect in the chest wall is usually of no major clinical significance if the pleural space is obliterated.[6] However, most commonly, the pleural space is free and the air and/or blood moves in and out through the chest wall defect, making a "sucking" sound.

A sucking chest wound or open pneumothorax results in a unidirectional flow of air through the wound and into the pleural space. During inspiration, the intrathoracic pressure is negative compared to the extrathoracic pressure. This allows air to flow through the wound and into the pleural space. During expiration, the tissues surrounding the wound come into apposition and decrease the wound diameter. This results in air becoming trapped within the pleural space. As breathing continues, this process progresses and air increasingly accumulates in the pleural space, compresses the lung, and prevents lung expansion during inspiration. The open pneumothorax can become large enough to exert tension on the hemithorax contents and the mediastinal structures.

In larger injuries, this air movement causes the ipsilateral lung to move inward and collapse on inspiration (**Figure 53-1*A***). The lung may expand slightly or remain completely collapsed upon expiration, depending on the size of the chest wall defect (**Figure 53-1*B***). There may also be mediastinal motion toward the noninjured lung during inspiration and toward the injured lung during expiration. This to-and-fro motion compromises the function of the healthy lung as well as the injured lung because it prevents its full expansion

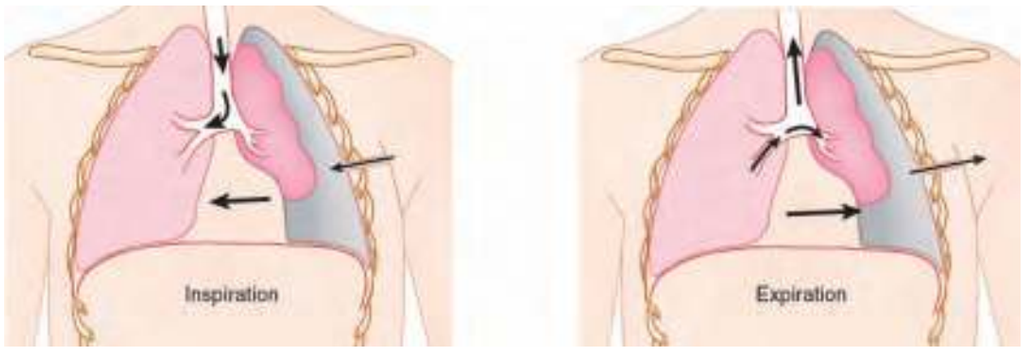


FIGURE 53-1. The effects of an open chest wound. ***A.*** Air moves into the pleural cavity and the lung collapses with inspiration. ***B.*** Air exits the pleural cavity and the lung expands slightly with expiration. The *arrows* represent the direction of airflow.

during inspiration. During expiration, some of the air from the noninjured lung may shift to the injured lung, and the reverse may happen during inspiration (an element of the "pendelluft" phenomenon). This entire mechanism results in a large functional dead space in the noninjured lung and loss of ventilation of the injured lung, causing severe ventilatory derangement, asphyxia, hypoxemia, and hypercarbia.[5-7]

The patient with an open pneumothorax may manifest a spectrum of presentations, ranging from asymptomatic and stable to severely dyspneic and agonal. The presentation depends on the size of the chest wall defect, the extensiveness of the injuries to the lung and other structures, the preinjury pulmonary status, and whether the pleural space is free or has adhesions. The patient may present with progressive respiratory insufficiency leading to a rapid demise if not treated. **The critical diameter of the chest wall wound has been described as two-thirds (or greater) the diameter of the trachea.[8] It is thought that, at this size, air moves preferentially through the chest wall rather than through the trachea**.

Two additional open chest wounds deserve mention. First is the wound that fully penetrates the chest wall but seals itself and closes. It can seal from apposition of adjacent chest wall soft tissues, a hematoma, underlying pleural adhesions that do not allow air into the pleural space, or a combination of these. This type of injury prevents air from entering the pleural space and does not significantly alter respiratory physiology except for the pain of breathing. This type of wound does not indicate that there is no underlying cardiac, intrathoracic, lung, or mediastinal injury. These types of wounds require management of hemorrhage, the application of a simple dressing, and a Surgeon for management of the wound, continued hemorrhage, and any internal injuries.

The final type of open chest wound is one that allows bidirectional airflow. Breathing results in bidirectional airflow through both the trachea and the chest wound, thus decreasing airflow through the trachea. This results in hypoventilation and hypoxemia. These patients require positive-pressure ventilation with either a bag-valve-mask device, continuous positive airway pressure, or bilevel positive airway pressure through a face mask or an endotracheal tube. The wound must be covered with a three-sided dressing or an occlusive dressing after the placement of a chest tube, and a Surgeon is required for management of the wound, continued hemorrhage, and any internal injuries.

INDICATIONS

Diagnosis of these injuries can be made easily based on the obvious presence of the chest wall defect and the noise produced as the air moves in and out through the wound. All open chest wounds should be treated immediately if the patient is symptomatic.[6,9] Treatment techniques should be selected based on the patient's clinical condition and stability (**Figure 53-2**). **The three-sided dressing is only a**

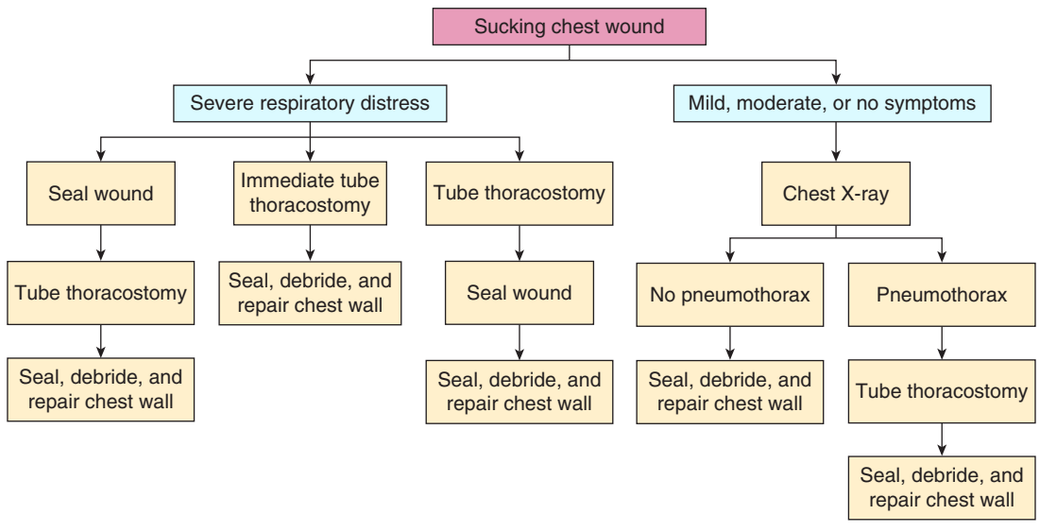


FIGURE 53-2. Algorithm for the diagnosis and treatment of an open pneumothorax. (Modified from reference 6.)

temporary measure, and it must be followed with the placement of a chest tube (Chapter 51). Current guidelines recommend leaving the open chest wound exposed and open or applying a nonocclusive dressing.[10]

Most experts agree that only the symptomatic patients should be treated in the field. An occlusive dressing should be placed over the wound and taped on only three sides. **The three-sided dressing allows air within the pleural cavity to be expelled into the atmosphere while preventing atmospheric air from entering the pleural cavity. If the wound is taped on all four sides, an open pneumothorax may quickly be converted into a tension pneumothorax.**[7,8,11-14]

In the Emergency Department, the very symptomatic patient should be treated as in the field. However, a completely occlusive dressing may be placed over the wound if a tube thoracostomy is subsequently performed. Continuously and closely monitor the patient for the development of a tension pneumothorax if the wound is completely sealed prior to the tube thoracostomy. **If a tension pneumothorax occurs, remove the occlusive dressing on at least one side or perform a needle thoracostomy (Chapter 50) to relieve it**.

Obtain a rapid, portable anteroposterior chest radiograph if the patient is asymptomatic, mildly symptomatic, or moderately symptomatic. If a pneumothorax is present, perform a tube thoracostomy and seal the wound. If the patient becomes severely symptomatic, the Emergency Physician should default to the other limb of the algorithm (**Figure 53-2**).

CONTRAINDICATIONS

There are no contraindications to the placement of a three-sided occlusive dressing, as this is a treatment for a life-threatening emergency. It must be properly placed to prevent the accidental conversion to a totally occlusive dressing and the progression of an open pneumothorax to a tension pneumothorax.[15] Likewise, current guidelines recommend not using an occlusive four-sided dressing.[10]

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Petrolatum gauze
- Gauze 4 × 4 squares
- Reinforcing sterile dressings
- Tincture of benzoin
- Adhesive tape
- Sterile gloves and gown
- Face mask with an eye shield or goggles and cap

The supplies for an occlusive dressing (i.e., petrolatum gauze) may not be readily available within or outside of the Emergency Department. Numerous substitutes for the petrolatum gauze are available. This includes a defibrillator pad. These gel-like pads are large, can be cut to size, and adhere to both dry and wet skin. Other alternatives include coating water-soluble lubricant onto one side of a piece of aluminum foil, plastic food wrap, a piece cut from the plastic packaging of sterile procedure packs, a piece cut from a plastic trash bag, or a piece cut from a zippered sandwich bag.

There are commercially available open chest wound seals.[16] The Hyfin Vent Chest Seal (North American Rescue LLC, Greer, SC) allows one-way airflow out of the chest cavity during exhalation (**Figure 53-3*A***). It is clear to allow visualization of the underlying wound, is latex free, and is peel-and-apply. The SAM Chest Seal (SAM Medical Products, Wilsonville, OR) has a dual-vent technology that enables simultaneous air and blood release from the chest cavity (**Figure 53-3*B***). This transparent dressing has a channel design that minimizes vent occlusion and the need to manually vent the chest wound. It also has a cap allowing it to be used as an occlusive dressing. The domed shape was designed to prevent obstruction of the air channels from blankets and clothing. The Sentinel Chest Seal (Combat Medical Systems, Harrisburg, NC) is also transparent, allows venting of air and blood, and is designed to minimize vent occlusion (**Figure 53-3*C***). The FastBreathe Thoracic Seal (FastTrack Medical Solutions LLC, Eden Prairie, MN) is a transparent, hardened, one-way valve assembly that has a low profile (**Figure 53-3*D***). It allows air to be expelled with minimal intrathoracic pressure. The PractiSeal (S.T.A.T., San Luis Obispo, CA) is a training chest seal that is cost effective and not for use in patients (**Figure 53-3*E***).

PATIENT PREPARATION

The amount and timing of patient preparation will be dictated by the location of the patient and their physiologic status. An informed consent is not required, as this is a noninvasive and lifesaving procedure. Such procedures should occur emergently with minimal patient preparation. If time permits, place the patient on the cardiac monitor and pulse oximeter, and provide supplemental oxygen by face mask. The Emergency Physician should wear full personal protective equipment to protect themselves from contact with the patient's blood and body fluids.

Consider performing orotracheal intubation before or simultaneously with the application of the three-sided occlusive dressing if the patient has severe respiratory insufficiency. Positive-pressure ventilation through the endotracheal tube will expand the collapsed lung and force the intrapleural air out the wound and into the atmosphere.

Prepare the chest wall if the patient is asymptomatic, mildly symptomatic, or moderately symptomatic. Clean the wound and surrounding chest wall of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the skin surrounding the wound and allow it to dry. Do not place the povidone iodine or chlorhexidine into the wound, as this may later inhibit wound healing. Apply the three-sided occlusive bandage as described below. **If the patient is moderately to severely symptomatic, no preparation is required as this wastes valuable time. Immediately apply the three-sided occlusive bandage**.

TECHNIQUE

The safest initial therapy for symptomatic sucking chest wounds is the careful application of a petrolatum gauze–based dressing taped on three sides (**Figure 53-4**). Apply three or four layers of petrolatum gauze over the wound. The dressing should extend 6 to 8 cm beyond the margins of the wound so that it will not be sucked into the pleural cavity in the spontaneously breathing patient. Cover the petrolatum gauze with dry 4 × 4 gauze squares. Apply tincture of benzoin around three sides of the dressing. Apply tape to secure the three sides of the dressing to the chest wall.

ALTERNATIVE TECHNIQUES

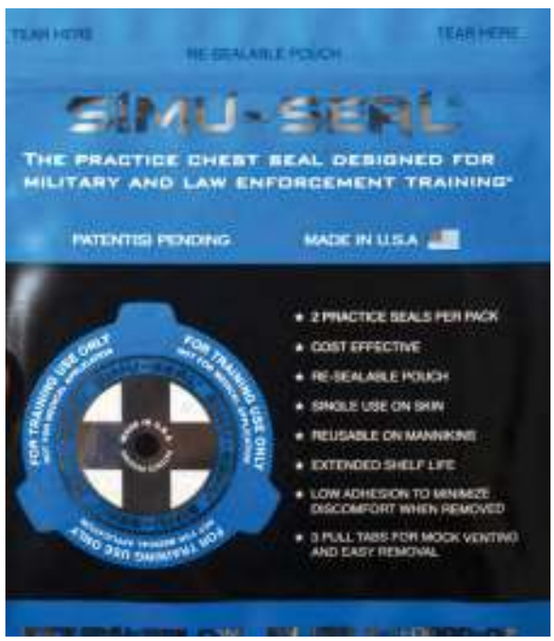
An alternative to the three-sided dressing is one that is totally occlusive or a four-sided dressing.[15,17] There is little to no clinical evidence that a three-sided dressing is superior to a four-sided dressing. **The four-sided dressing should be placed only in the setting where rapid placement of a chest tube will be undertaken**. This occurs most commonly in the Emergency Department. It may be considered in the field when a tube thoracostomy is included in the prehospital standing medical orders.[18]

A

B

C

D



E

FIGURE 53-3. Commercially available chest vents. ***A.*** The Hyfin Chest Seal. (Photo courtesy of North American Rescue LLC.) ***B.*** The SAM Chest Seal. (Photo courtesy of SAM Medical Products.) ***C.*** The Sentinel Chest Seal. (Photo courtesy of Combat Medical Systems.) ***D.*** The FastBreathe Thoracic Seal. (Photo courtesy of FastTrack Medical Solutions LLC.) ***E.*** The PractiSeal. (Photo courtesy of S.T.A.T.)

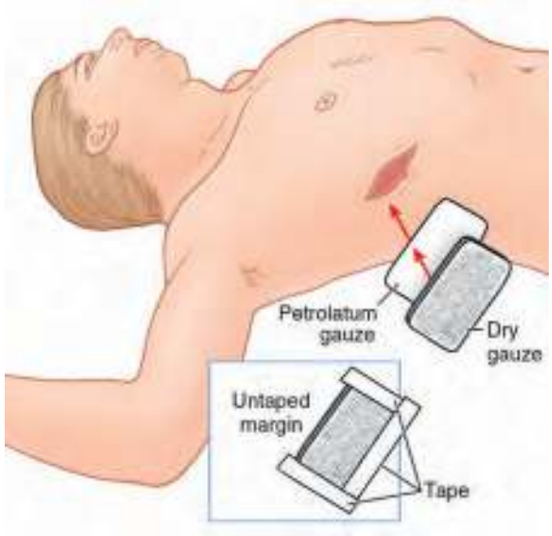


FIGURE 53-4. The three-sided occlusive dressing.

ASSESSMENT

Once the three-sided dressing has been placed, continue to closely monitor the patient for associated complications and evaluate them for underlying injuries. **Paramount among the complications is conversion of a simple pneumothorax to a tension pneumothorax. Do not be led into a false sense of security after placing the three-sided dressing. A patient can still develop a tension pneumothorax.** Periodically evaluate the three-sided dressing to ensure that it is not covered with another dressing or blankets, that it is not positioned between the patient and the bed or another object, and that it has not become occluded on all four sides. **If a tension pneumothorax occurs, remove the occlusive dressing on at least one side or perform a needle thoracostomy (Chapter 50) to relieve it**. Patients with an open chest wound have typically sustained an injury comprising great kinetic energy, whether from a blunt or penetrating event. Associated injuries will be very common. A head-to-toe secondary survey is imperative.

AFTERCARE

The aftercare of these patients consists of a tube thoracostomy followed by aggressive wound care, pain management, pulmonary toilet, continued monitoring, and investigation for other underlying injuries. **Once the wound is closed, the underlying pneumothorax or hemopneumothorax should be treated with the placement of a chest tube placed through an incision away from the injury site and not through the open chest wound**.

Pain will be a major problem for these patients. Trauma to the parietal pleura, bony structures, and intercostal nerves is very painful. It is imperative that these patients be able to make adequate ventilatory efforts, cough, deep breathe, perform incentive spirometry, and have aggressive pulmonary toilet. These are all necessary to prevent atelectasis, retained secretions, and pneumonia.[19] Various pain-relieving techniques may be used. Infiltration of long-acting local anesthetic solution may give initial relief, especially during wound debridement. Parenteral analgesics are the initial treatment modality. Advanced techniques to consider include regional nerve blocks (i.e., intercostal nerve blocks) and indwelling intrapleural or epidural catheters.

More definitive wound care should be considered once the patient is stabilized. Small, clean wounds may be managed simply with routine wound care and changes of the occlusive dressing. Local irrigation and debridement may be added if there is a limited amount of contamination. Large, grossly contaminated, and/or complex wounds are best managed in the Operating Room. Optimally, these wounds should be debrided and closed primarily. However, some wounds may be contaminated or complex, such that closure is not possible initially. In such cases, the wound should be debrided and left open. Large occlusive dressings are placed along with chest tubes for removal of air, fluid, and blood. Multiple operative debridements may be required. When the wound is clean and the patient is optimized, secondary closure may be performed.[19]

Closure of large wounds may require a combination of complex techniques. These may include various skin, subcutaneous tissue, and muscle flaps.[20] Free rib grafts, pectoral muscle flaps, latissimus dorsi muscle flaps, abdominal muscle flaps, omentum flaps, skin grafts, or a myocutaneous flap can be used.[20-27] If using these is not possible, closure may be accomplished with a prosthetic material such as Prolene or Marlex, either temporarily or definitively.[28-30] In wounds of the lower chest, detachment of the diaphragm with reattachment at a higher level may be used. This converts an open chest wound to an intraabdominal wound and alleviates the ventilatory problems.

COMPLICATIONS

Complications may occur acutely or may be delayed. **Occlusion of the chest wall defect and decompensation of the patient from a simple pneumothorax being converted to a tension pneumothorax are the primary early complications.[15] The patient must be closely monitored until a chest tube can be inserted**. A tension pneumothorax can result if the wound is completely occluded by a blood clot, a dressing that has been sucked into the wound, soft tissue, or a bandage that is adherent on all four sides. **Immediately remove the bandage to relieve a tension pneumothorax**. Some physicians and authors remove only one side of the occlusive bandage to relieve the pneumothorax. The choice to remove part or all of the bandage is physician-dependent. Ensure that the wound is not occluded by a blood clot or soft tissue if the patient is still symptomatic after removing part or all of the bandage.

Other complications can ensue from the failure to seek, diagnose, and treat other underlying and potentially life-threatening injuries. The patient may develop respiratory insufficiency secondary to multiple causes, some of which may be preventable with optimal care. These causes include inadequate pulmonary toilet, inadequate pain management, pulmonary contusion, pneumonia, and/or adult respiratory distress syndrome. Wound complications may include infection, fasciitis, osteomyelitis, empyema, hemothorax, and loculated hemothoraces or pneumothoraces. These wounds require frequent evaluation and aggressive care to prevent these sequelae.

SUMMARY

Open chest wounds are easily diagnosed by the evident chest wall defect and the auscultation of air moving into and out of the pleural cavity. This is a true life-threatening emergency. Treatment is dictated by the patient's clinical presentation. Lifesaving therapy should be undertaken with the simple application of a three-sided petrolatum gauze dressing. The three-sided dressing allows air within the pleural cavity to be expelled into the atmosphere while preventing atmospheric air from entering the pleural cavity. The three-sided dressing converts the open pneumothorax to a closed pneumothorax and eliminates the major physiologic derangement. Once the patient is stabilized, more definitive care should be carried out with chest tube placement and appropriate wound care.

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54 Emergency Department Thoracotomy

Kenny Banh

INTRODUCTION

An increase in urban violence combined with better prehospital and transport systems has resulted in the arrival of sicker trauma patients to the Emergency Department (ED). Previously, these patients might not have survived long enough to make it to the ED.[1] The majority of individuals with penetrating chest injuries arrive at the ED in stable condition and are managed without major operative procedures.[2] A subset of individuals, however, arrive in extremis and may require a thoracotomy. The purpose of the ED thoracotomy may be to control hemorrhage within the chest or below, to relieve a pericardial tamponade, to redistribute cardiac output to the brain and the heart, or to provide more effective cardiac massage.[3]

ANATOMY AND PATHOPHYSIOLOGY

The structures within the chest include the heart, esophagus, lungs, bronchi, pulmonary hilar vessels, and numerous other vascular structures. The heart is located in the anterior mediastinum. The aorta and esophagus are located in the posterior mediastinum. The internal mammary arteries course along the posterior aspect of the anterior chest wall just lateral to the sternum. The intercostal vessels run along the inferior aspect of the ribs. The subclavian vessels are at the very superior aspect of the thorax. They course directly under the clavicles and can be very difficult to visualize via an anterolateral thoracotomy. The azygos vein can be found coursing along the posterior right hemithorax and empties into the superior vena cava.

The heart is covered by the tough pericardial sac. **The phrenic nerves run superiorly and inferiorly on each side of the pericardiac sac**. They can be visualized as white or yellow strands on either side of the pericardium. Once the pericardial sac is opened, the left anterior descending coronary artery can be visualized on the anterior surface of the heart. It overlies the interventricular septum. Injuries to the left of this artery usually denote left ventricular damage, whereas injuries to the right usually denote right ventricular damage. The majority of the anterior surface of the heart is occupied by the right ventricle.

The posterior mediastinum contains the aorta. It is located posterior to the esophagus and runs lateral to the vertebral bodies. The thoracic aorta gives off the intercostal vessels. If torn during the mobilization of the aorta, the intercostal vessels can cause troublesome bleeding.

The ED thoracotomy was originally introduced for penetrating chest wounds.[4] This procedure was subsequently used for patients with penetrating abdominal wounds and victims of blunt trauma. **In recent years, several studies have shown an abysmal survival rate associated with an ED thoracotomy in victims of blunt trauma, including children.**[5,6] When vital signs are present in the field, the survival rates for such individuals range from 0.6% to 6.0%.[7-12] Patients who undergo an ED thoracotomy for penetrating abdominal injuries have survival rates of approximately 5%.[11,13] In these cases, the ED thoracotomy is performed for resuscitation purposes.

Blunt trauma arrests are difficult to resuscitate and not agreed upon by expert Surgeons.[14-16] The Western Trauma Association recommends a thoracotomy for adults with blunt cardiac arrest if there are any signs of life or < 10 minutes of cardiopulmonary resuscitation (CPR).[14] Advanced trauma life support does not recommend resuscitation of these patients. A systematic review showed up to a

1.5% overall survival rate with a good neurologic outcome.[16] It also showed survival to be essentially zero if the patient has no signs of life in the field. The review conclusions were that thoracotomy may be useful in patients with vital signs in the ED, 15 minutes of CPR, and no head injury.

Patients presenting to the ED with penetrating chest trauma who present without signs of life in the field have a poor prognosis. Survival ranges from 0% to 9%.[17,18] If the patient sustains penetrating chest trauma and has signs of life in the field, survival averages 14%, with a range of 0% to 36%.[1] The reason for the wide range probably lies in the small numbers of patients in the studies and the varying definitions of "signs of life."

The best survival for penetrating chest injury and an ED thoracotomy is in patients with stab wounds resulting in cardiac tamponade. The use of bedside ED ultrasonography allows better identification of these patients.[19] The survival for this entity ranges from 21% to 71%, averaging 31%.[10,12,20-25] It should be noted that up to 90% of patients who survive an ED thoracotomy for penetrating chest injuries have good neurologic outcomes.[24-27] This must be contrasted with a 50% incidence of a good neurologic outcome in the few survivors of blunt trauma who receive a thoracotomy.[28]

INDICATIONS

There is little consensus among trauma surgeons on the exact indications of an ED thoracotomy.[29-31] **Signs of life present at the scene, signs of life at any time during the transport, or a resuscitation with a short transport time should prompt an ED thoracotomy in patients with penetrating chest trauma.**[20-23,32,33] These signs include a palpable pulse, a blood pressure, pupil reactivity, any purposeful movement, an organized cardiac rhythm, or any respiratory effort. **Thus, the patient must have signs of life on presentation or have lost them en route to the ED if a thoracotomy is to be considered.** A thoracotomy should be performed to control hemorrhage within the thoracic cavity, to decompress a pericardial tamponade, to cross-clamp the aorta and redistribute the cardiac output to the brain and heart, and to provide open cardiac massage.[34] Patients who are in shock or rapidly deteriorating clinically after penetrating chest trauma and are not responding to aggressive fluid resuscitation are also candidates for an ED thoracotomy. It is also indicated to cross-clamp the aorta when the patient is exsanguinating from injuries below the level of the diaphragm.[33,35] Consider a thoracotomy if the patient has a pericardial tamponade not relieved with a pericardiocentesis and cardiac arrest.[36,37] A final indication is the placement of a chest tube (Chapter 51) with > 1500 mL of immediate blood return or a persistent bleed of 150 to 200 mL/h for 2 to 4 hours.[38]

CONTRAINDICATIONS

There are a few well-supported contraindications to performing an ED thoracotomy. **A thoracotomy should not be performed in trauma patients who have no vital signs in the field**.[15,39,40] In the absence of field vitals, the survival rates are extremely low and the few who survive have severe neurologic impairment. **Outside of patients who collapse in the ED, victims of blunt trauma with or without field vitals should not routinely undergo an ED thoracotomy**.[40,41] It is also contraindicated when prehospital CPR exceeds 10 minutes without a return of spontaneous circulation after blunt trauma, when prehospital CPR exceeds 15 minutes without a return of spontaneous circulation after penetrating trauma, and when the patient presents to the ED in asystole without a pericardial tamponade.[39-43] **An ED thoracotomy should not be performed regardless of the indications if a Trauma Surgeon or other qualified Surgeon is not available to take the patient to the Operating Room for definitive management.** Do not perform an ED thoracotomy with the anticipation of transferring the patient to another facility if they can be resuscitated. In the near future, resuscitative endovascular balloon occlusion of the aorta (REBOA) may make some indications for a thoracotomy into contraindications (Chapter 74).

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile towels
- Sterile gloves and gown
- Face mask with an eye shield or goggles and cap
- Sandbags or towels
- #3 scalpel handle
- #10 scalpel blade
- U.S. Army retractors, 1 set
- Curved Mayo scissors, 8¾ and 6¾ inches
- Curved Metzenbaum scissors, 5½ inches
- Toothed forceps
- Satinsky vascular clamp
- Finochietto rib retractor, 12 inch spread
- Suction source
- Suction tubing
- Yankauer suction catheter
- 2–0 silk suture on a large curved needle
- Hemostats needle driver, 10 inches
- Gauze 4 × 4 squares
- Sternal saw, hand-operated
- Lebsche knife (sternal osteotome) and mallet

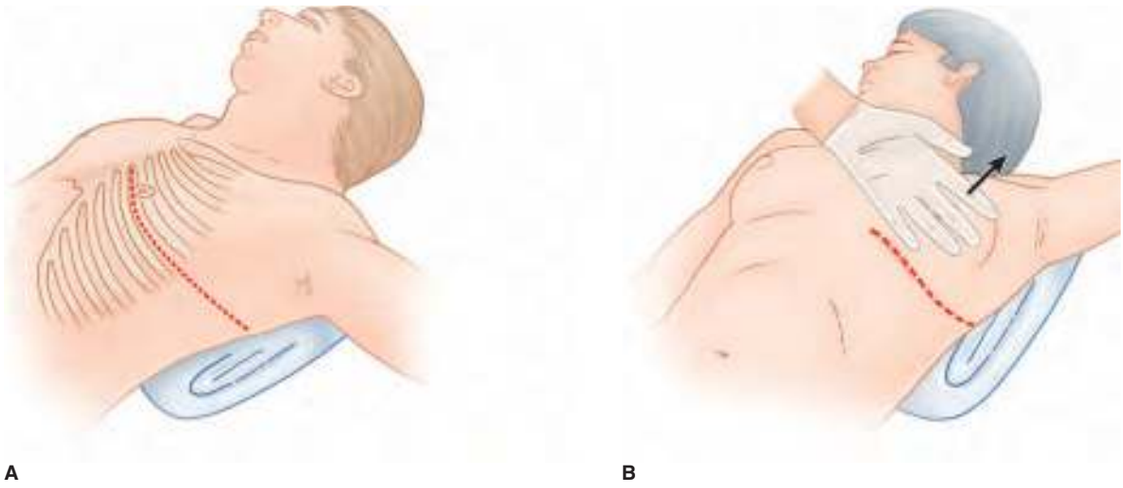
All hospital EDs should have prepackaged and sterile thoracotomy trays. Review the equipment available on the trays at your institution to become familiar with their contents before the tray is required emergently.

PATIENT PREPARATION

The patient should be fully monitored with a noninvasive blood pressure cuff, pulse oximetry, cardiac monitor, and end-tidal carbon dioxide monitor (if available). Explain the risks, benefits, and complications of the procedure to the patient and/or their representative. The patient is usually deteriorating and loses consciousness or is unconscious, and time is of the essence. This may limit obtaining an informed consent. Document the circumstances of not getting consent in the medical record.

The patient should already be supine, intubated, and ventilated. Abduct the left upper extremity 180° (**Figure 54-1**). The extremity should be held in position by an assistant or with the use of a soft restraint. Place sandbags or towels under the patient's left scapula. This will elevate the torso off the bed and allow for more complete access. Apply povidone iodine or chlorhexidine solution to the patient's left chest. Apply sterile drapes over the chest to demarcate a surgical field.

An ED thoracotomy is primarily performed on patients who are unresponsive. As such, there is no immediate need for analgesics, sedatives, or local anesthetic solution. If the patient is resuscitated, they will experience significant postprocedural pain. **Parenteral analgesics and sedatives should be available and administered if the patient survives and there are no contraindications**.



A B

FIGURE 54-1. Patient positioning. Place a sandbag or towel under the left shoulder and abduct the left arm 180°. Identify the fifth intercostal space in the male (*A*) or the inframammary line in the female (*B*).

Set up the required equipment. Open the prepackaged, sterile thoracotomy tray on a bedside table. The tray should contain all the equipment required for the procedure. Sterile technique should be observed and followed by all involved personnel (i.e., cap, mask, sterile gown, and sterile gloves). This will protect the health care personnel from exposure to the patient's blood and body fluids as well as minimize the risk of infection if the patient survives.

TECHNIQUES

LEFT-SIDED THORACOTOMY

Apply the scalpel blade to the handle. Identify the site to be used to make the initial skin incision. This is the left fourth or fifth intercostal space, corresponding to the intercostal space below the nipple in a male and below the inframammary fold in a female (**Figure 54-1**). **Make an incision using one stroke of the scalpel and extending from the sternum to the posterior axillary line or the gurney (Figure 54-2*A*)**. Carry the incision through the skin, subcutaneous tissues, and superficial chest musculature down to and through most of the intercostal muscles (**Figure 54-2*A***). An Army retractor may be used to open and separate the edges of the incision but is not required.

Discontinue mechanical ventilation and advance the endotracheal tube into the right mainstem bronchus. This will allow the left lung to deflate and minimize injury upon entering the left thoracic cavity while still ventilating the right lung. As an alternative, briefly discontinue mechanical ventilation. Puncture through the intercostal muscles in the anterior axillary line with the curved Mayo scissors. Carefully extend the puncture 2 to 3 cm using the curved Mayo scissors. Insert the nondominant index and middle fingers through the incision and separate the lung from the chest wall. Advance the fingers and Mayo scissors simultaneously superiorly and then inferiorly to cut the intercostal muscles along the entire inner space (**Figure 54-2*B***). Resume mechanical ventilation.

Insert the Finochietto retractor with the arm and crank positioned near the gurney (**Figure 54-2*C***). Turn the crank to open the arms of the rib spreader. **Make sure that the lung is not caught between the arms of the rib spreader and the chest wall to avoid tearing the lung.** Clear any blood from the left hemithorax and inspect for any brisk bleeding. If extensive bleeding is observed, it must be controlled. Use digital pressure or hemostats to initially control intercostal artery or other bleeding vessels. Subsequently, place 2–0 silk stitches to tie off the bleeding vessels. For subclavian vessels, digital control must be followed by rapid transport to the Operating Room, since these vessels are difficult to control through an anterolateral thoracotomy.

OPENING THE PERICARDIUM

Move the left lung superiorly and laterally to expose the pericardial sac. **A pericardiotomy should be routinely performed if blood is seen within the pericardial sac, if the heart cannot be visualized through the pericardium, or if there is no other obvious injury within the chest and a potential cardiac injury may exist. Many Emergency Physicians and Trauma Surgeons will routinely open the pericardium when they are performing a thoracotomy, as a pericardial tamponade is difficult to detect visually and a potential cardiac injury may exist**.

Open the pericardial sac anterior to the phrenic nerve. Grasp and elevate the pericardium with a toothed forceps anterior to the phrenic nerve, which appears as a white or yellow strand along the lateral aspect of the pericardium (Figure 54-2*D*). Make an incision in the pericardium near the apex of the heart using a curved Mayo scissors. On occasion, a patient with pericardial tamponade physiology will have a tense pericardium that can be grabbed with the forceps and a small incision will have to be made with the Mayo scissors or a scalpel blade facing upward. Normally a small amount of straw-colored fluid is expressed from the pericardium if no cardiac trauma has occurred. Carefully insert one of the jaws of the Mayo scissors into the pericardial sac. **Extend the incision with the Mayo scissors parallel to the phrenic nerve, from the apex of the heart to the root of the aorta.** Deliver the heart from the pericardium and inspect it for any injury (**Figure 54-2*E***). Internal cardiac massage (Chapter 55) may be performed for asystole, bradycardia, and/or hypotension. Any cardiac wounds identified should be temporarily plugged (Chapter 56).

RIGHT-SIDED THORACOTOMY

If there is a high index of suspicion of injury in the right hemithorax with minimal or no injury found in the left hemithorax, the thoracotomy must be extended to the right side. Extend the incision through the sternum using a curved Mayo scissors and continue it to the right posterior axillary line (**Figure 54-3**). Remove the Finochietto rib spreaders from the left side and apply them to the patient's right fifth intercostal space. Examine the right hemithorax

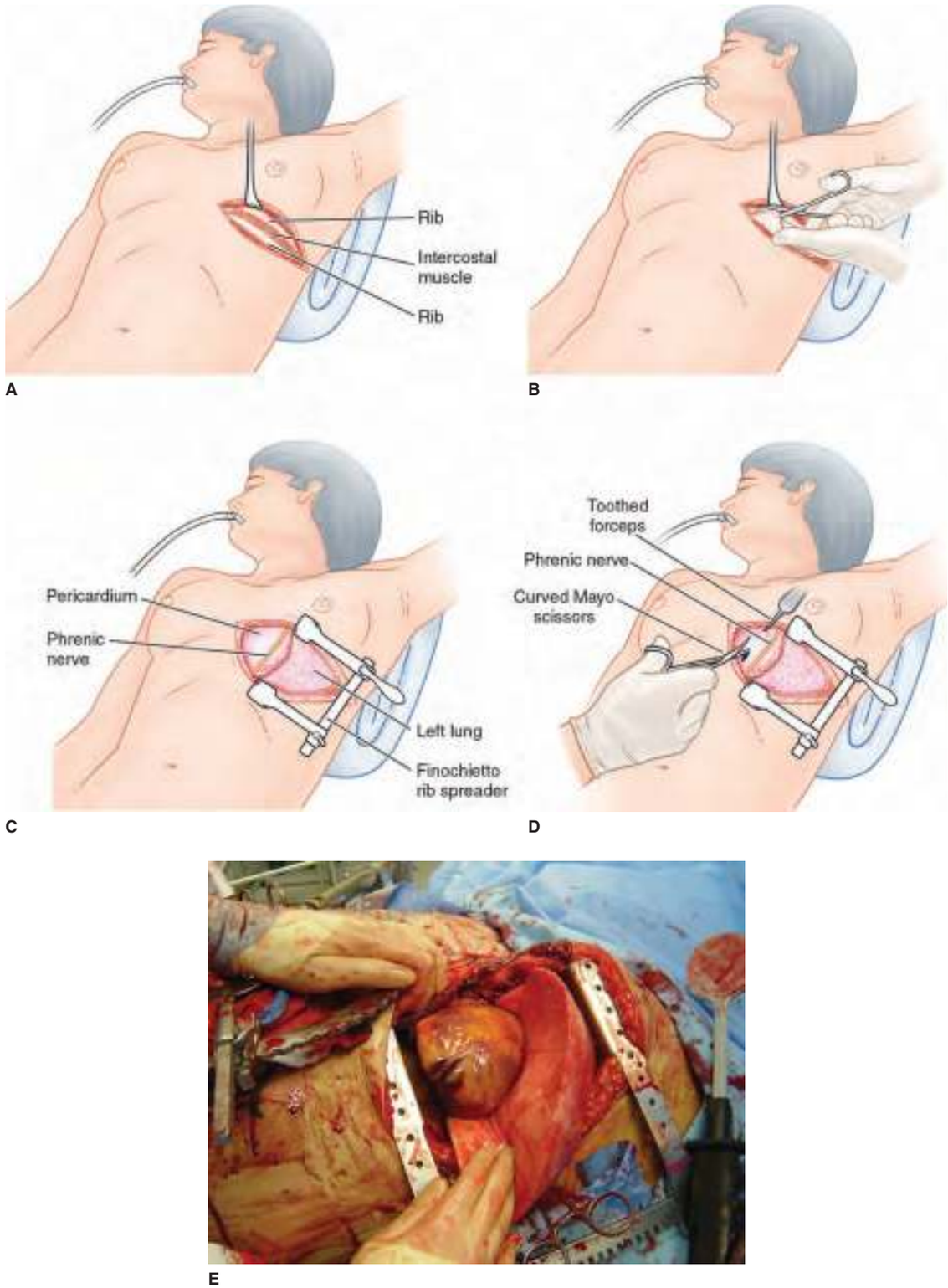


FIGURE 54-2. ED thoracotomy. ***A.*** The initial incision is made through the skin, subcutaneous tissue, and superficial muscles. ***B.*** The intercostal muscles are incised with Mayo scissors. ***C.*** The Finochietto rib spreader has been inserted and opened. ***D.*** The pericardium is grasped and opened. ***E.*** A thoracotomy with the pericardium opened. (Part E used with permission from Cothren CC, Moore EE: Emergency department thoracotomy for the critically injured patient: objectives, indications, and outcomes. *World J Emerg Surg* 2006; 1:4.)

for injury. The internal mammary arteries on both sides will be lacerated when the sternum is cut. Apply hemostats to the transected vessels to obtain hemostasis. They may later be tied off if the patient is resuscitated.

Occasionally, the large curved Mayo scissors cannot transect the calcified sternum. Perform a right-sided thoracotomy similar to that on the left. If further access is required, cut the sternum with a handheld sternal saw or Lebsche knife (**Figure 54-4**). Pass one end of the

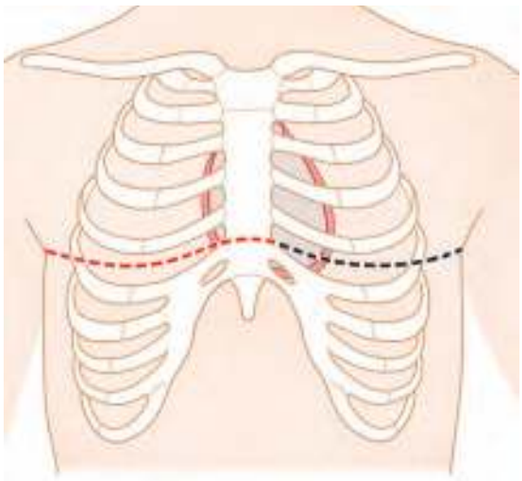


FIGURE 54-3. The left-sided incision is continued across the sternum and the right fifth intercostal space to perform a right-sided thoracotomy.

sternal saw from the left fifth intercostal space, behind the sternum, and out of the right fifth intercostal space (**Figure 54-4*A***). Place an index finger through the loop on each end of the sternal saw. Move the hands toward and away from the patient in a to-and-fro motion until the sternum is transected. Alternatively, a Lebsche knife can be used to transect the sternum. Place the hooked portion of the knife under the sternum with the sharp blade against the lateral border of the sternum (**Figure 54-4*B***). Lift up the handle of the Lebsche knife to lock it against the posterior surface of the sternum. This will prevent the tip of the Lebsche knife from cutting the heart. Hit the flat knob on the back of the Lebsche knife with a mallet to drive the knife through the sternum (**Figure 54-4*B***).

ASSESSMENT

Any bleeding should be controlled with the application of pressure, hemostats, and/or sutures. Any injuries to the heart (Chapter 56) or the hilum and great vessels (Chapter 57) should be managed. Cross-clamping of the proximal aorta will prevent further exsanguination from more distal injuries (Chapter 58). Open cardiac massage can be performed while the resuscitative efforts continue (Chapter 55).

AFTERCARE

The patient should be immediately transported to the Operating Room for definitive care, as soon as the Anesthesiologist and Surgeon are available, if resuscitated in the ED. Continue the administration of fluids, packed red blood cells, platelets, plasma, and inotropic agents as necessary until the patient is hemodynamically stable. Administer broad-spectrum antibiotics intravenously if the patient is resuscitated and survives. Administer parenteral analgesics and/or sedation (Chapter 159) if not contraindicated.[44]

COMPLICATIONS

The ED thoracotomy has many potential and serious complications. Fortunately, this procedure is often performed as a last effort for the resuscitation of a "dead patient." The complications are, therefore, not significant when the alternative to this procedure is death.

The ED thoracotomy is rarely ever performed under truly sterile conditions. Time is often of the essence in performing this procedure. Povidone iodine or chlorhexidine solution and sterile drapes are rarely applied before an emergent ED thoracotomy. If applied, the povidone iodine or chlorhexidine solution does not have time to dry before the skin incision is made. The complications of the thoracotomy include vascular and organ injury. Lacerations of the internal mammary or intercostal arteries can be ligated with silk suture. There is also the possibility of inadvertent laceration of the lung or the myocardium during the initial incision. By temporarily halting mechanical ventilation while performing the thoracotomy, injury to the underlying lung can often be prevented. These injuries will need subsequent repair at the appropriate time.

A pericardiotomy can result in significant complications. The left phrenic nerve may be transected. The myocardium or a coronary artery can be lacerated. The heart may be fixed by adhesions to the pericardium from prior pericardial disease or pericarditis. Attempting to remove the heart from the pericardium can result in avulsion of the atrial or ventricular myocardium. Performance of the pericardiotomy adds another delay in initiating cardiac compressions.

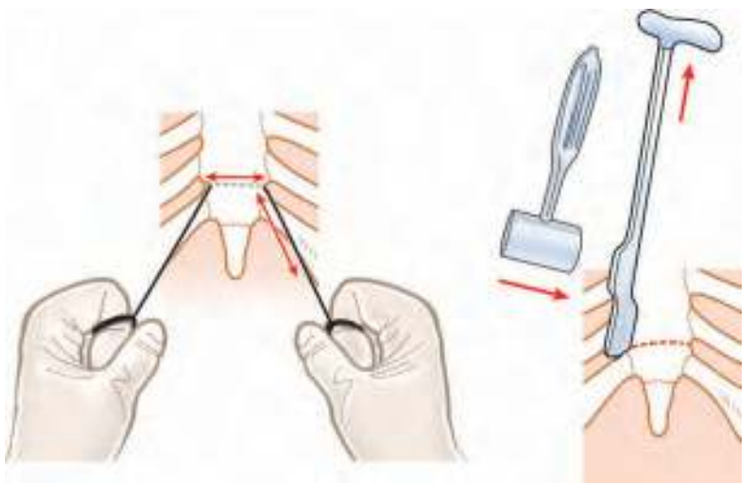


FIGURE 54-4. Cutting the sternum. ***A.*** The sternal saw is moved to-and-fro while maintaining upward pressure. ***B.*** The Lebsche knife is hooked under the sternum and lifted upward to secure it in place. The mallet strikes the Lebsche knife and drives it through the sternum.

Care should be taken to prevent injury to any of the health care providers. Universal precautions should be followed by all personnel. The use of caps, gloves, goggles, masks, and gowns will protect against exposure to the patient's blood. All needles, scalpels, and scissors should be returned to the bedside tray immediately after use and not left on the patient or the bed. **Use extreme caution when placing your hands inside the patient's hemithorax.** Fractured ribs from the trauma or the Finochietto rib spreader can easily penetrate gloves and skin. The costs associated with an occupational exposure associated with an ED thoracotomy are quite significant, especially if the health care provider develops hepatitis C or HIV.[5,42,45]

SUMMARY

An ED thoracotomy is a lifesaving procedure when used on select patients in extremis secondary to chest or abdominal trauma. To be considered for this procedure, the patient must have signs of life when brought to the ED, must have lost them en route, or must have lost them on the scene with a short transport time. This procedure should rarely be used in patients with blunt trauma. The Emergency Physician should have the appropriate surgical backup before performing a thoracotomy since this procedure does not provide definitive therapy. In the near future, REBOA may change some thoracotomy indications into contraindications.

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55 Open Cardiac Massage

Eric F. Reichman

INTRODUCTION

The purpose of cardiopulmonary resuscitation (CPR) during cardiac arrest or hypovolemic shock is to provide adequate cardiac output. This can be done using either closed or open chest cardiac massage. Open cardiac massage may, on rare occasions, be performed in the Emergency Department. It is performed on patients who have had an emergent thoracotomy after penetrating chest trauma and have inadequate cardiac activity. It may also be performed, in rare instances, after a thoracotomy to decompress a pericardial tamponade in a medical patient. Open cardiac massage is considered a heroic procedure in the Emergency Department that can be lifesaving if performed on the appropriate patient.[1-12]

Open cardiac massage was routinely performed before the introduction of closed chest CPR. This technique was primarily performed in hospitals by Surgeons. Most of the patients were surgical, and open cardiac massage had a high success rate. After the development of closed chest CPR in the early 1960s, there was a dramatic decline in open cardiac massage in the mid-to-late 1960s and 1970s. The exceptions were cardiac arrests due to trauma or in the Operating Room.

ANATOMY AND PATHOPHYSIOLOGY

The efficacy of cardiac massage can be established by measuring the cardiac output, coronary perfusion pressure, and cerebral perfusion pressure. A higher cardiac index can be achieved with open than with closed cardiac massage.[1] A minimal coronary perfusion pressure of 15 mmHg must be maintained for return of spontaneous circulation. While not all patients with this pressure will have a return of spontaneous circulation, a pressure of less than 15 mmHg predicts a uniformly fatal outcome.[2] While closed chest CPR generated only 1 to 9 mmHg of pressure, Boczar et al found that their patients all had a coronary perfusion pressure of almost 20 mmHg throughout open chest massage.[3] Open chest CPR produces improved cerebral perfusion and better neurologic recovery.[4,10] Open cardiac massage can generate near normal cerebral blood flow and improve cardiac perfusion pressure.

INDICATIONS

Open cardiac massage is indicated if absent or inadequate cardiac activity is noted after a thoracotomy for penetrating trauma, a thoracotomy to decompress a pericardial tamponade (spontaneous, postsurgical, or from an aortic dissection), or a cardiac arrest after recent chest surgery.[7,9,11] Other possible indications include abnormal chest wall anatomy that prevents closed chest CPR, hypothermic cardiac arrest, refractory ventricular fibrillation, massive air embolism, and if standard CPR is not effective.

CONTRAINDICATIONS

The only absolute contraindication to performing open cardiac massage is the presence of a palpable pulse. Open cardiac massage is ineffective if the patient has a pericardial tamponade. Perform a thoracotomy and pericardiotomy to remove any clots from the pericardial sac. The heart may then begin to beat spontaneously. If not, repair any lacerations to the myocardium prior to performing cardiac compressions. Refer to Chapter 54 for a discussion in which a thoracotomy is contraindicated, as open cardiac massage is also contraindicated. It is also contraindicated if the patient is clearly deceased.

EQUIPMENT

No equipment is required to perform open cardiac massage other than that needed to perform the thoracotomy and pericardiotomy (Chapter 54).

PATIENT PREPARATION

The preparation and positioning of the patient are exactly the same as those for a thoracotomy. A thoracotomy must first be performed (**Figure 55-1**). Refer to Chapter 54 for the complete details of a thoracotomy. A pericardiotomy should be performed only if absolutely necessary—that is, if blood is seen within the pericardial sac or cardiac tamponade is suspected. Remove any blood and clots from the pericardial sac, deliver the heart from the pericardial sac, and repair any myocardial lacerations (refer to Chapter 56).

An intact pericardium is preferable if open cardiac massage is to be performed. It prevents the fingertips from inadvertently rupturing the atria or ventricles should the heart be grasped incorrectly. The pressure from open cardiac massage is distributed over a larger area if the pericardium remains intact. The heart will move within the pericardial sac, which will prevent the fingers from compressing one spot for a prolonged time and may decrease the chance of myocardial rupture.

TECHNIQUES

Several important principles must be kept in mind prior to performing open cardiac massage. The heart must be angled not more than 20° to 30° into the left hemithorax (Figure 55-2*A*). Angulation of more than 30° may crimp the pulmonary veins and vena cava closed and thus minimize cardiac output. The compressive forces should be applied perpendicular to the interventricular septum. Place your hands directly behind and in front of the heart. The left anterior descending artery can be used as a landmark, as it runs above the interventricular septum. Do not place the fingers over the coronary arteries so that their flow is occluded. The fingertips should never be used to compress the heart, as they may rupture the myocardium. Use only the palm, volar surfaces of the fingers, and pads of the fingers to perform cardiac compressions. The fingers should always be held tightly together to form a flat surface. This allows the force of compression to be spread out and not concentrated over one finger. Each compression of the heart must be followed by complete relaxation of the heart. It is during this relaxation phase (diastole) that the cardiac chambers fill with blood and the coronary arteries perfuse the myocardium.

Three techniques have been described to perform open cardiac massage (**Figures 55-2 and 55-3**). These include one-handed massage with sternal compression, one-handed compression, and two-handed compression.[12] Two-handed compression has been shown to be consistently superior to the other two techniques in generating cardiac output.[5]

ONE-HANDED MASSAGE WITH STERNAL COMPRESSIONS

Tightly adduct the fingers of the dominant hand to make a flat surface. Insert the hand into the thoracotomy incision and against the posterior surface of the heart (**Figures 55-2*A* and *B***). Angle the heart 20° to 30° into the left hemithorax. Compress the heart against the sternum, beginning with the heel of the hand, followed by the

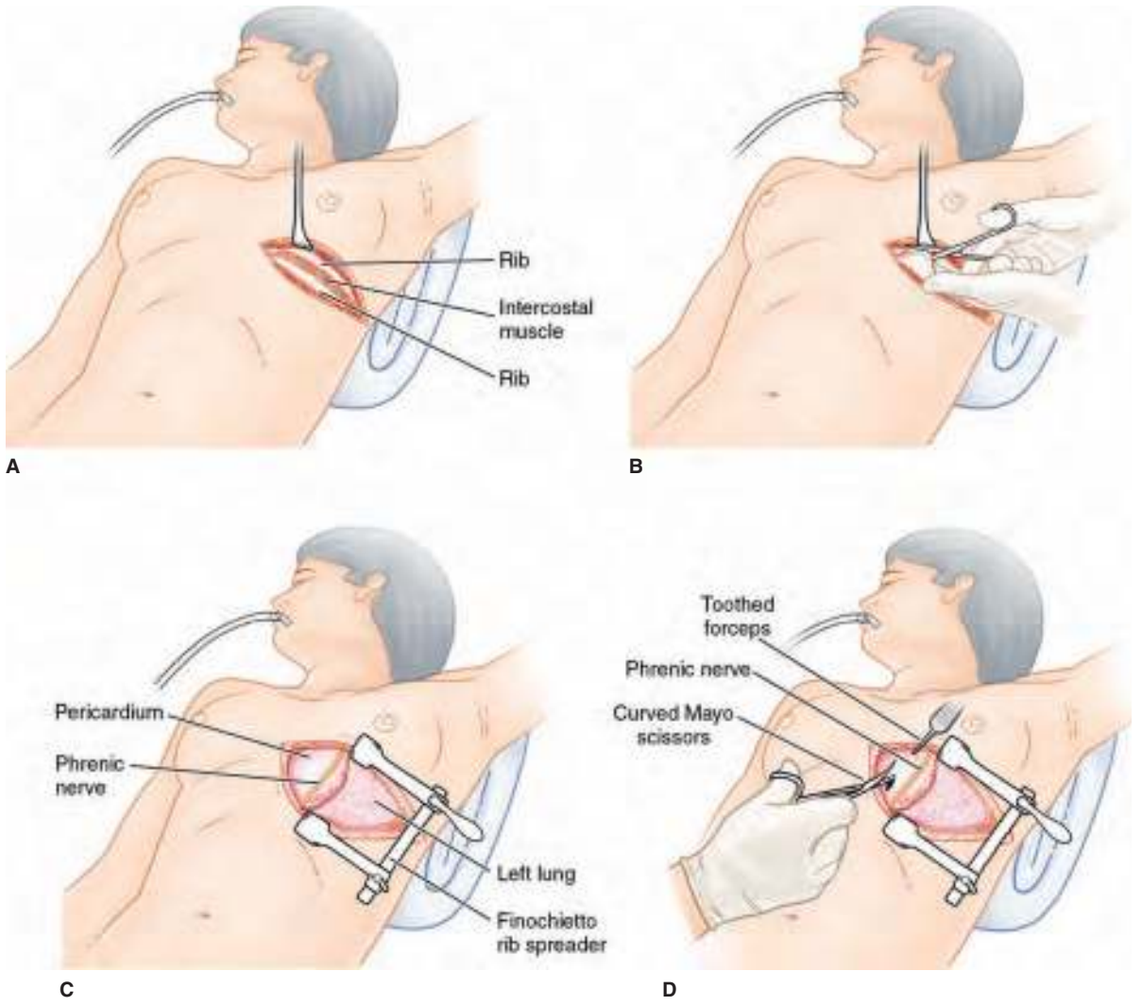

FIGURE 55-1. The anterolateral thoracotomy.

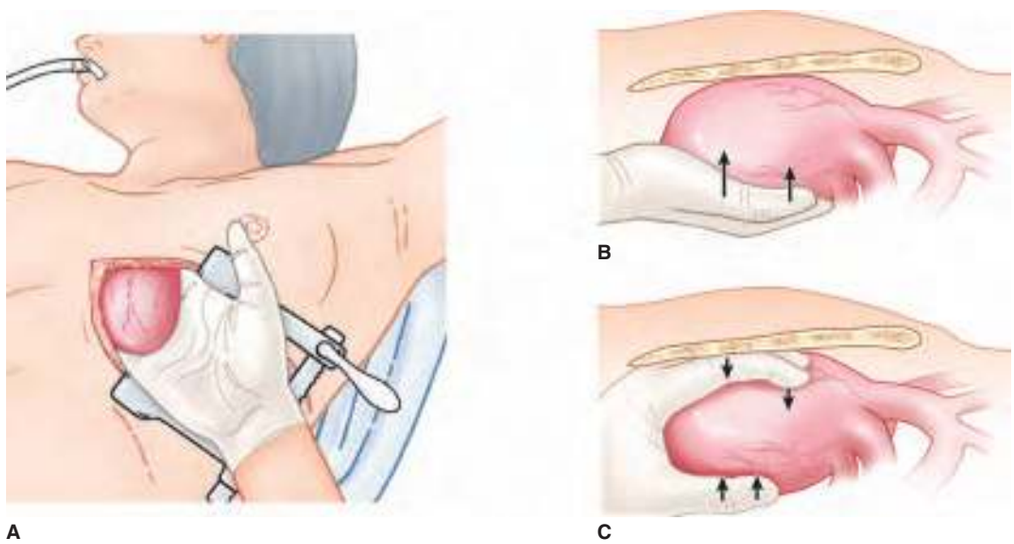

FIGURE 55-2. One-handed open cardiac massage. ***A.*** The dominant hand angles the heart 20° to 30° into the left hemithorax. ***B.*** One-handed cardiac massage with sternal compression. ***C.*** The one-handed compression technique.

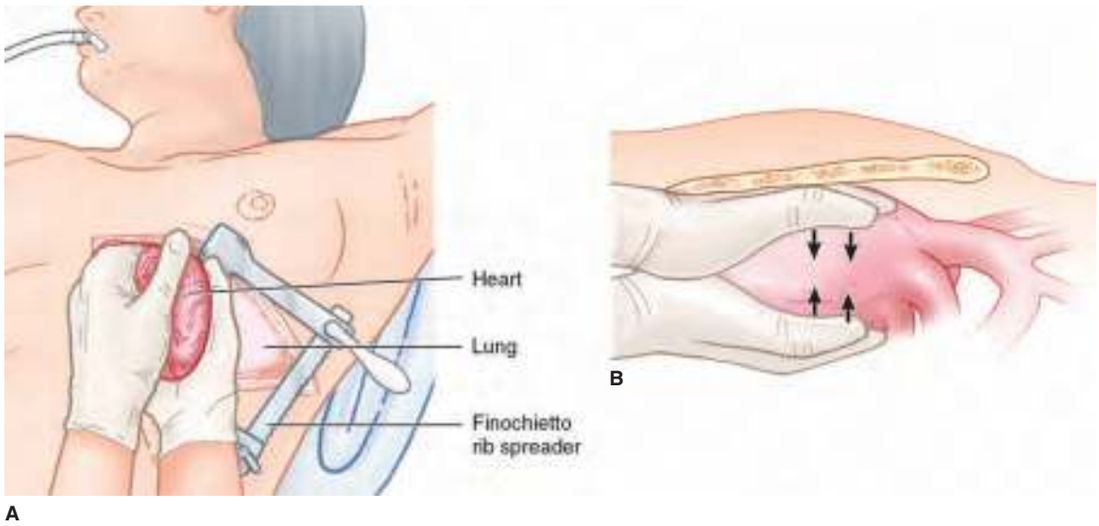

FIGURE 55-3. Two-handed open cardiac massage. ***A.*** The hands are positioned on the anterior and posterior surfaces of the heart. ***B.*** Compressions begin at the cardiac apex with the palms and progress toward the base of the heart with the fingers.

palm, and then followed by the fingers in sequence. This method is difficult to perform in children because of the plasticity of the sternum.[6]

ONE-HANDED COMPRESSIONS

Tightly adduct the fingers of the dominant hand to make a flat surface. Insert the hand into the thoracotomy incision and against the anterior surface of the heart (**Figure 55-2*C***). Place the thumb against the posterior surface of the heart. The apex of the heart will lie in the palm of the hand (**Figure 55-2*C***). Angle the heart 20° to 30° into the left hemithorax. Appose the thumb and fingers to compress the heart. This is not the preferred method, as the thumb can place significant pressure on the left ventricle and possibly cause it to rupture.

TWO-HANDED COMPRESSIONS

This is the technique of choice in children. The child's sternum, the costochondral tissues, and the ribs are mostly cartilage. This makes their sternum very pliable. This decreases effectiveness of the one-handed massage with sternal compression technique. The one-handed compression technique can rupture the thin myocardial walls.

Tightly adduct the fingers and cup the left hand. Insert the left hand into the thoracotomy incision and against the anterior surface of the heart (**Figure 55-3*A***). Tightly adduct the fingers of the right hand to make a flat surface. Insert the right hand into the thoracotomy incision and against the posterior surface of the heart (**Figure 55-3*A***). Angle the heart 20° to 30° into the left hemithorax. Appose both of the hands to compress the heart (**Figure 55-3*B***). Compressions should ideally begin with the heel of the hands and progress toward the fingers.

ASSESSMENT

Compressions should begin at a rate appropriate for the patient's age as specified by the American Heart Association's Pediatric Advanced Life Support and Advanced Cardiac Life Support guidelines.[13,14] Ideally, compress the heart at a rate of at least 100 times a minute if possible. This compression rate is based on consensus and not clinical research. Assess the effectiveness of the cardiac compressions by noting a palpable carotid pulse. A radial or femoral arterial line can be placed to monitor the effectiveness of the compressions. The arterial line allows for repeated blood and blood gas sampling. The arterial line will also allow the Emergency Physician to ensure that the heart is completely relaxed between compressions.

AFTERCARE

If a spontaneous cardiac rhythm and a peripheral pulse return, cover the thoracotomy incision with a sterile saline-moistened gauze and a simple dressing. Administer intravenous antibiotics whose spectrum covers skin flora. The patient must be taken emergently to the Operating Room by a Trauma Surgeon or Cardiothoracic Surgeon for definitive treatment and closure of the chest wall.

COMPLICATIONS

The major complication of open cardiac massage is myocardial rupture or perforation. This can occur from compression of the heart against a jagged and fractured rib or sternum. Incorrect technique with too vigorous a massage can result in perforation of the ischemic myocardium by the fingertips. Other complications include decreased cardiac output with compressions if the heart is angled more than 30° into the left hemithorax and kinks shut the vena cava or pulmonary veins. Infection is possible if the patient survives. Parenteral antibiotics should be administered to prevent infection and sepsis. The complications associated with the thoracotomy and the pericardiotomy are discussed in Chapter 54. The quality of life of survivors can be poor to normal.[8]

SUMMARY

Open cardiac massage is a more efficient way of maintaining circulation than closed chest massage. Cardiac compressions can be performed with one or two hands, depending on physician preference. The two-handed technique is preferred, as it generates greater cardiac output than the one-handed techniques. Once a cardiac rhythm and blood pressure are restored, definitive treatment for injuries must be provided expeditiously by a Surgeon in the Operating Room.

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56 Cardiac Wound Repair

Eric F. Reichman

INTRODUCTION

Wounds of the heart are highly lethal. Traumatic cardiac penetration carries a 70% to 80% fatality rate.[1] Major factors determining survivability include whether or not cardiac standstill has occurred as well as the amount of tissue destruction sustained from the injury.[2] A repair of a cardiac wound was first described in the literature in 1893.[3]

Penetrating wounds can be caused by knives, bullets, ice picks, and (infrequently) rib or sternal fragments. **Regardless of the offending agent, repair must be done as expeditiously as possible.** The right ventricle is the most frequently injured chamber.[4] However, injury to the heart may occur at more than one site. This is especially true with gunshot wounds.[1]

The treatment goal in the Emergency Department is temporary hemostasis. Many different techniques of cardiorrhaphy have been described. We will limit our discussion to five possible approaches to dealing with these injuries (i.e., digital or Foley catheter occlusion, vascular clamps, staples, and sutures).

ANATOMY AND PATHOPHYSIOLOGY

The heart is contained within the pericardial sac. Numerous portions of the heart are exposed behind the anterior chest wall (**Figure 48-2**). This includes the right ventricle, left ventricle, right atrium, left atrium, aorta, pulmonary artery, and inferior vena cava. These structures are vulnerable to injury behind the anterior chest wall.[5,6] The surface areas that each of these structures contributes to the anterior cardiac silhouette are as follows: 55% right ventricle, 20% left ventricle, 10% right atrium, 10% aorta and pulmonary artery, 4% inferior vena cava, and 1% left atrium.[7] These numbers also reflect, roughly, the anatomic incidence of injury with cardiac trauma.[7] Traumatic injury to any of these structures can result in a pericardial effusion and cardiac tamponade. Injuries to more than one cardiac chamber can occur from a single gunshot or stab wound.[4,8] Cardiac wall injuries can also occur following blunt trauma.[9]

INDICATIONS

Any penetrating injury to the heart requires immediate and temporary repair to prevent the patient from exsanguinating. A bluish hue behind the pericardium or a tense pericardial sac after penetrating trauma suggests an underlying cardiac injury. A pericardiotomy should be performed, any blood and clot removed from the pericardial sac, and the heart explored for the site of injury. The indications to perform a thoracotomy are described in Chapter 54.

CONTRAINDICATIONS

The only absolute contraindication to performing a cardiorrhaphy is if the patient has obvious signs of death. It should not be performed if the patient has not had any vital signs for over 15 minutes, as anoxic brain injury is irreversible. It is also contraindicated in patients with penetrating chest trauma who do not meet the criteria for performing an anterolateral thoracotomy. Refer to Chapter 54 for a discussion in which a thoracotomy is contraindicated, as any cardiac repair is also contraindicated. There is a less than 1% chance of survival if the patient has multiple gunshot wounds to the torso.[10,11] It is up to the discretion of the treating physician in cases of multiple gunshot wounds to the torso if a thoracotomy is to be done due to the generally accepted unsalvageability.[10,11]

EQUIPMENT

- Sterile gloves and gown
- Face mask with an eye shield or goggles and cap
- Silk suture, 4–0 and 5–0 (or Prolene)
- 2–0 silk, on a semicircular atraumatic needle
- 10 inch needle driver
- Foley catheter, sizes 14 to 20 French
- Satinsky, or other, atraumatic vascular clamp (**Figure 56-1**)
- Allis clamps
- Defibrillator with internal cardiac paddles
- Mayo scissors
- Metzenbaum scissors, curved
- Teflon pledgets
- Sterile saline
- 20 mL syringe

FIGURE 56-1. Examples of several atraumatic vascular clamps.

- Standard skin stapler, 6 mm wide staples
- Laparotomy pads
- Gauze 4 × 4 squares
- Hemostats

PATIENT PREPARATION

No preparation is required other than that of performing a thoracotomy and a pericardiotomy (Chapter 54). The patient should be intubated, ventilated with 100% oxygen, and fully monitored (i.e., telemetry, a noninvasive blood pressure cuff, and pulse oximetry). Instruct a nurse to administer intravenous broad-spectrum antibiotics that cover skin flora, gram-positive organisms, and gram-negative organisms. The Emergency Physician should wear full personal protective equipment to protect themselves from contact with the patient's blood and body fluids. Although time is of the essence and this is an emergent procedure, aseptic technique should be followed. A thoracotomy must first be performed. Refer to Chapter 54 for the complete details of a thoracotomy.

TECHNIQUES

Bleeding from a cardiac wound should ideally be stopped by placing a finger over the wound and immediately transporting the patient to the Operating Room for definitive repair. Unfortunately, the Anesthesiologist, Surgeon, and/or Operating Room may not be immediately available. If the Emergency Physician performs a thoracotomy, they should be versed in methods of temporary cardiac wound repair. Several techniques are available to control hemorrhage from a cardiac wound.[12]

CONTROL OF THE HEART

It is extremely difficult to maintain a finger over a cardiac wound or to stitch a cardiac wound if the heart is beating. An apical traction suture known as Beck's suture can be placed to control the heart (**Figure 56-2**). The apex of the heart is the ideal site because it is often away from most lacerations, is a thick portion of the heart, and is away from any major coronary arteries. Place a 2–0 silk suture through the apex of the heart and remove the needle from the suture. The ends of the suture can be grasped by hand or with a hemostat to elevate and control the heart while it is beating.

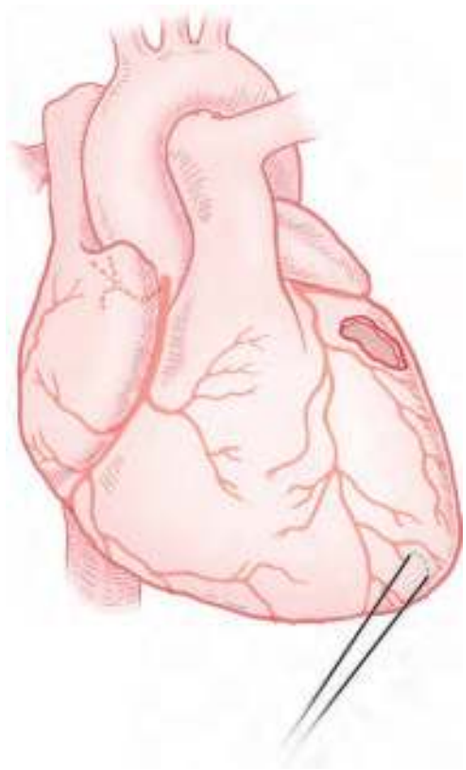


FIGURE 56-2. The apical traction suture known as Beck's suture may be placed to control the heart.

SAUERBRUCH MANEUVER

Large cardiac wounds and wounds with significant bleeding are difficult to repair, as the blood obscures the surgical field. Experienced Surgeons may temporarily clamp the inferior and superior vena cava to maintain a bloodless field. **Clamping the vena cava should not be performed by an Emergency Physician, as it is time-consuming and can injure other structures.** A quicker and safer alternative is the Sauerbruch maneuver, or grip, to partially occlude venous inflow through the inferior and superior vena cava (**Figure 56-3**). This technique will stabilize the heart for wound repair and allow the bleeding site to be identified and repaired.

Insert the nondominant hand into the pericardial cavity and toward the vena cava. Place the middle finger behind the vena cava and the index finger in front of the vena cava (**Figure 56-3**). The thumb should be resting on the anterior surface of the heart. The ring and little fingers should be resting on the posterior surface of the heart. Use the thumb and the ring and little fingers to cradle the heart while apposing the middle and index fingers to partially occlude the vena cava. This technique can be used to control hemorrhage from the heart, great vessels, or hilum. **While effective at controlling hemorrhage, this technique will significantly reduce cardiac output and may result in cardiac arrest. The grip should be released every 30 to 60 seconds to ensure coronary artery perfusion.**

DIGITAL OCCLUSION

Digital occlusion of small cardiac wounds can provide excellent hemostasis while awaiting definitive repair. Once a finger is placed on or over the defect, it must remain there until the appropriate materials to perform cardiorrhaphy are available. **Do not place a finger into the defect as this may increase the size of the wound.** Unfortunately, the fingertip often slips off the wound

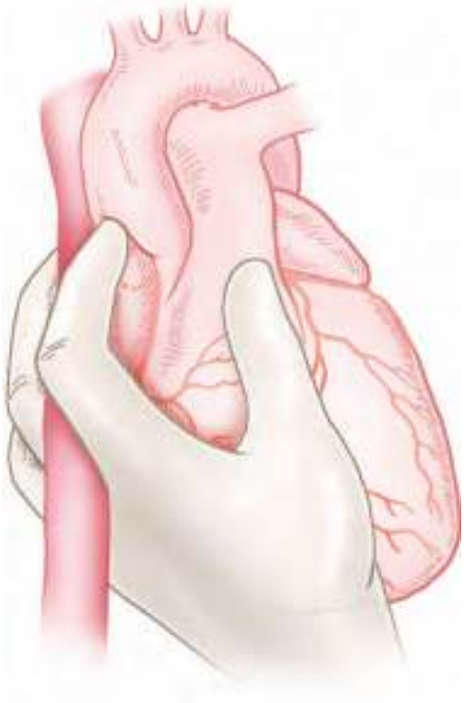


FIGURE 56-3. The Sauerbruch maneuver to partially occlude venous inflow from the superior and inferior vena cava.

if the heart is beating. The fingertip interferes with visualization and repair of the wound. It also places the Emergency Physician at risk for a needle-stick injury while attempting to repair the wound.

FOLEY CATHETER TECHNIQUE

A Foley catheter may be used to provide temporary hemostasis (**Figure 56-4**). As with digital occlusion, this is only a temporizing measure. Inflate the cuff of the Foley catheter with sterile saline to check its integrity and look for leaks. **Deflate the cuff. Place a hemostat on the Foley catheter 3 to 4 cm proximal to the cuff. This will prevent air from being drawn through the catheter and causing an air embolism.**

Identify the location of the cardiac wound (**Figure 56-4*A***). Insert the Foley catheter through the cardiac wound until the balloon is within the cardiac chamber (**Figure 56-4*B***). Release the hemostat, inflate the cuff with 10 to 20 mL of sterile saline, and reclamp the Foley catheter (**Figure 56-4*C***). **This step should be done quickly to prevent air from being drawn through the Foley catheter and into the heart.**

Apply gentle traction to the catheter (Figure 56-4*D*). Apply just enough traction to mostly occlude the wound and slow the bleeding. A small amount of bleeding is adequate and acceptable to visualize the wound and perform a temporary repair. Do not try to provide complete hemostasis. Providing complete hemostasis can result in excessive traction on the Foley catheter causing the cuff to pull through the wound, enlarge the wound, and further lacerate the myocardium.

Repair the cardiac wound. Place a purse-string suture around the wound (**Figure 56-4*E***). **Use caution when placing the suture**

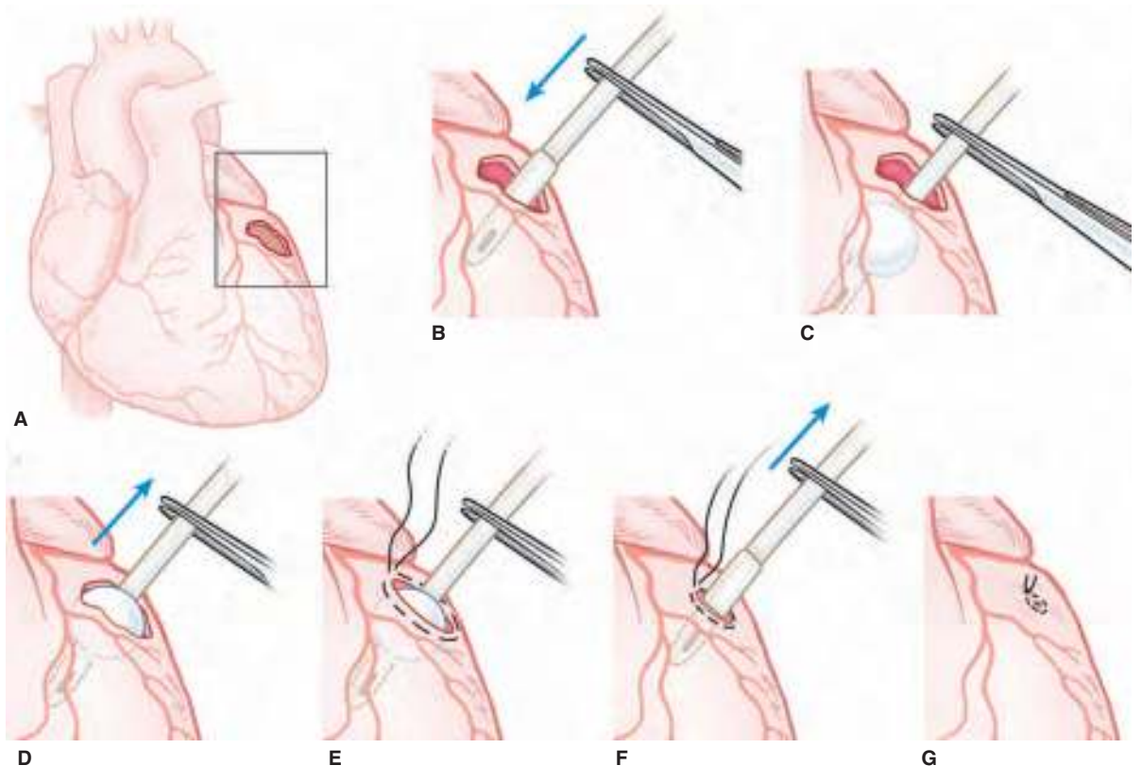


FIGURE 56-4. The Foley catheter technique to occlude and repair a cardiac wound. ***A.*** The cardiac wound is identified. ***B.*** The Foley catheter is inserted through the wound. ***C.*** The cuff is inflated. ***D.*** Gentle traction is applied to occlude the wound with the cuff. ***E.*** A purse-string suture is placed around the wound. ***F.*** The cuff is deflated, and the Foley catheter is removed. ***G.*** The purse-string suture is tightened and tied to occlude the wound.

so that the needle does not pierce and rupture the cuff of the Foley catheter. The Foley catheter may be advanced into the cardiac chamber temporarily while the suture is being placed. This will prevent the needle from piercing the cuff. **Do not advance the cuff too far into the heart and for too long a time period. The cuff may occlude blood flow through the valves and result in cardiac arrest.** After the purse-string suture is placed, deflate the cuff and quickly remove the Foley catheter (**Figure 56-4*F***). Tie the ends of the suture to close the cardiac wound (**Figure 56-4*G***). Place additional knots to secure the suture.

The Foley catheter technique is simple and effective. It avoids the problems associated with digital occlusion. The catheter may be placed in posterior cardiac wounds that are difficult to visualize and where it is hard to maintain digital occlusion. The Foley catheter does not interfere with visualization of the wound, wound repair, or the simultaneous performance of cardiac massage. Intravenous catheter tubing may be inserted into the lumen of the Foley catheter to infuse crystalloid solutions or red blood cells directly into the heart and central circulation. This technique is especially valuable in repairing wounds at the junction of the right atrium and vena cava.

CLAMP TECHNIQUE

Atrial wounds can bleed profusely and are difficult to control. The thin walls do not allow digital occlusion to be effective. The atrial wound can be grasped and compressed between the thumb and index finger. An atraumatic Satinsky vascular clamp, or other atraumatic vascular clamp (**Figure 56-1**) in the thoracotomy tray, can be placed around the wound to provide hemostasis (**Figure 56-5**). The wound may then be repaired with 4–0 or 5–0 interrupted silk sutures.

Allis clamps may be substituted if an atraumatic vascular clamp is not available. Place an Allis clamp on each of the opposing edges of the atrial wound. Apply upward traction and then cross the Allis clamps to approximate the wound edges. The wound may now be sutured closed. The Allis clamps do not provide as bloodless a field as does the atraumatic vascular clamp.

STAPLE TECHNIQUE

Skin staplers provide a quick and easy method to repair cardiac wounds. Careful approximation and stapling of the wound will rapidly close the defect and aid in controlling massive blood loss. Staples may be used to close small, large, or multiple lacerations. Its use avoids the potential complication of a needle stick to the Emergency Physician.

Cardiac wounds are closed similarly to skin lacerations (**Figure 56-6**). Obtain a standard skin stapler with 6 mm wide staples. These are readily available in most Emergency Departments for wound closure. Use the nondominant hand to appose the wound edges. Grasp the stapler with the dominant hand. Place staples at 5 mm intervals until the wound is closed. **Avoid any visible epicardial coronary vessels or use an alternate technique. Closing or damage to the vessels can result in myocardial ischemia, myocardial infarcts, and cardiac arrest.**

SUTURE TECHNIQUES

Suturing of cardiac wounds is more time consuming than the other methods previously described. It requires technical proficiency and understanding of the heart's surface anatomy. Place horizontal mattress sutures using 2–0 or 3–0 silk or Prolene suture material (**Figure 56-7**). **Do not tie the sutures tightly. It is easy to inadvertently tear through the myocardium.** Use Teflon pledgets to reinforce the repair and prevent cutting through the heart tissue (**Figures 56-7*A* and *B***). This is especially important when the wound edges are irregular or tattered. Use care to avoid injury to the coronary vessels, which may lie in close proximity to a cardiac wound. Ensure that the sutures are placed adjacent to and underneath the coronary vessels (**Figure 56-7*C***).

Large cardiac wounds are difficult to repair. They bleed profusely. The digital occlusion and Foley catheter techniques are ineffective on large wounds. The wound edges are difficult to grasp and appose so that they can be repaired with a skin stapler. Place an incomplete horizontal mattress stitch on each side of the wound (**Figure 56-8**). Grasp the free ends of the sutures and cross them across the wound to appose the wound edges. Instruct an assistant to hold the suture ends while the cardiac wound is repaired

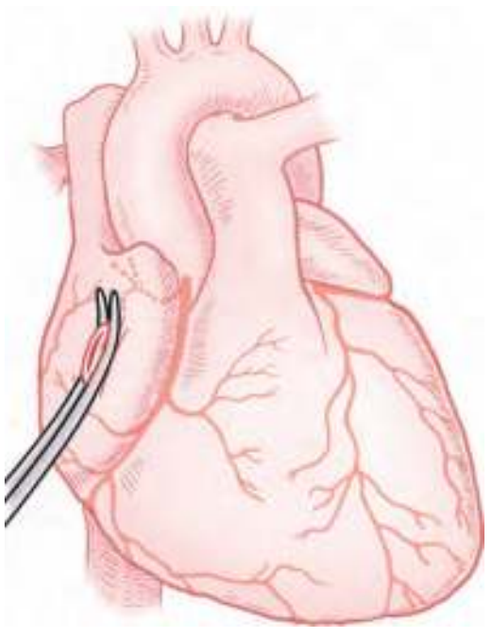


FIGURE 56-5. A Satinsky vascular clamp provides hemostasis for atrial wounds.

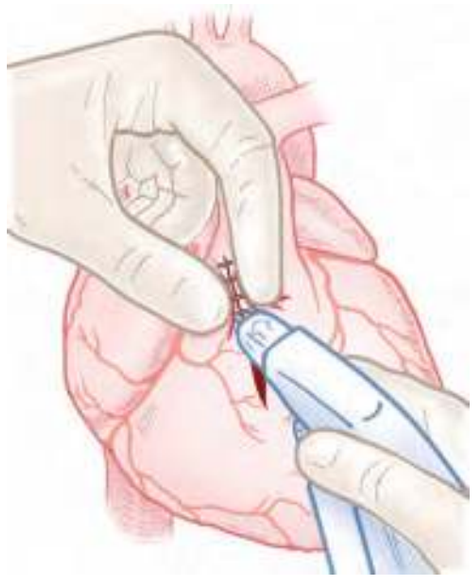


FIGURE 56-6. The cardiac stapling technique provides temporary hemostasis.

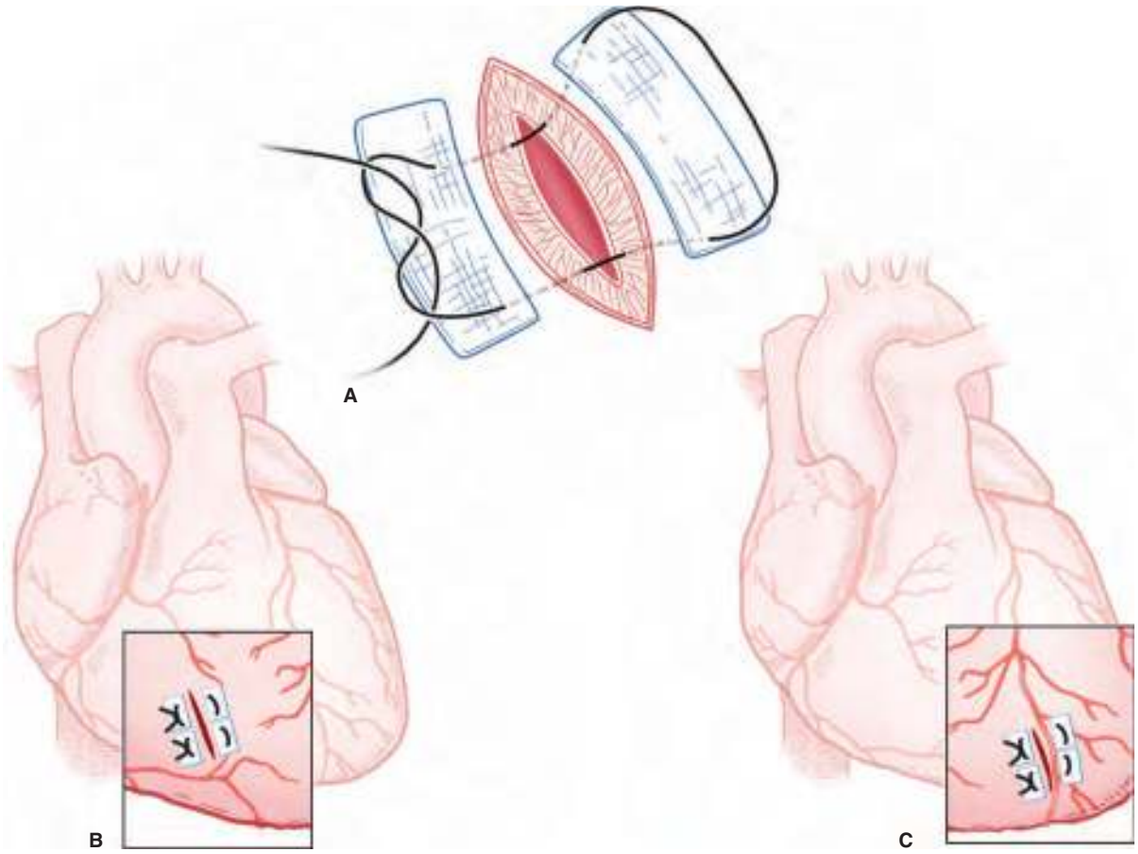


FIGURE 56-7. Horizontal mattress sutures used to close cardiac wounds. ***A.*** Teflon pledgets are placed on either side of the wound to prevent the suture from pulling through the myocardium. ***B.*** Wounds are closed with multiple horizontal mattress sutures. ***C.*** When suturing near a coronary artery, ensure that the sutures pass completely below the artery.

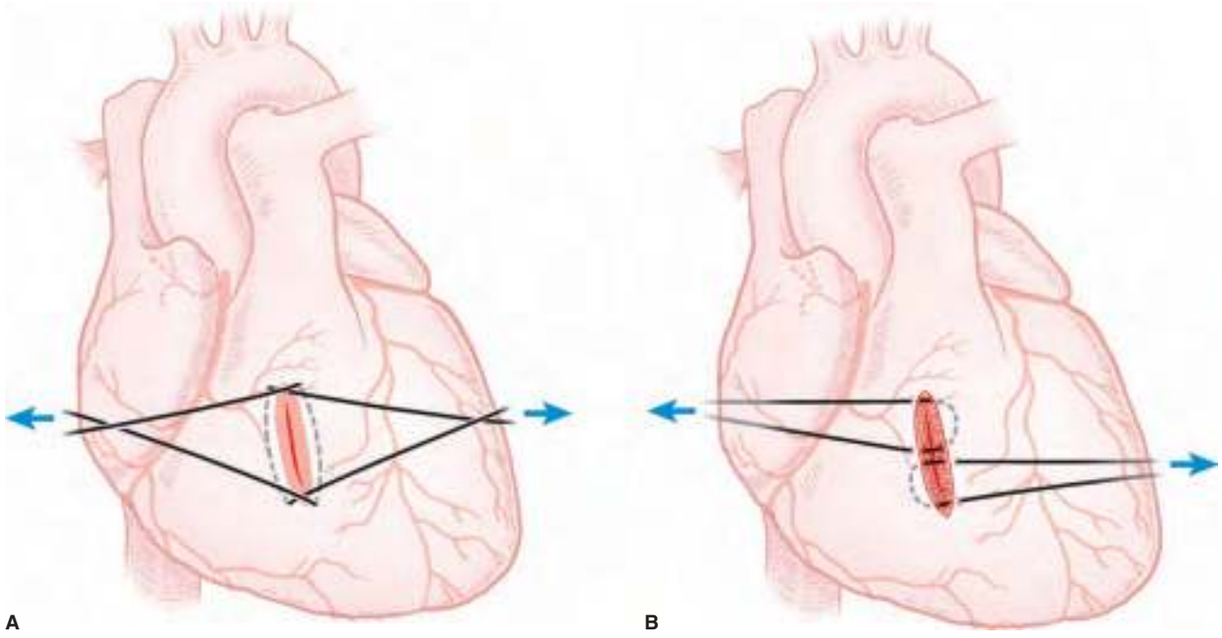


FIGURE 56-8. Incomplete horizontal mattress stitches may be placed on each side of a large wound. Tension applied to the sutures will appose the wound edges (*arrows*).

FIGURE 56-9. The internal cardiac paddles inserted into the handles.

with 2–0 or 3–0 silk, Prolene, or staples. The incomplete mattress sutures may then be removed or tied to each other.

FIBRILLATION

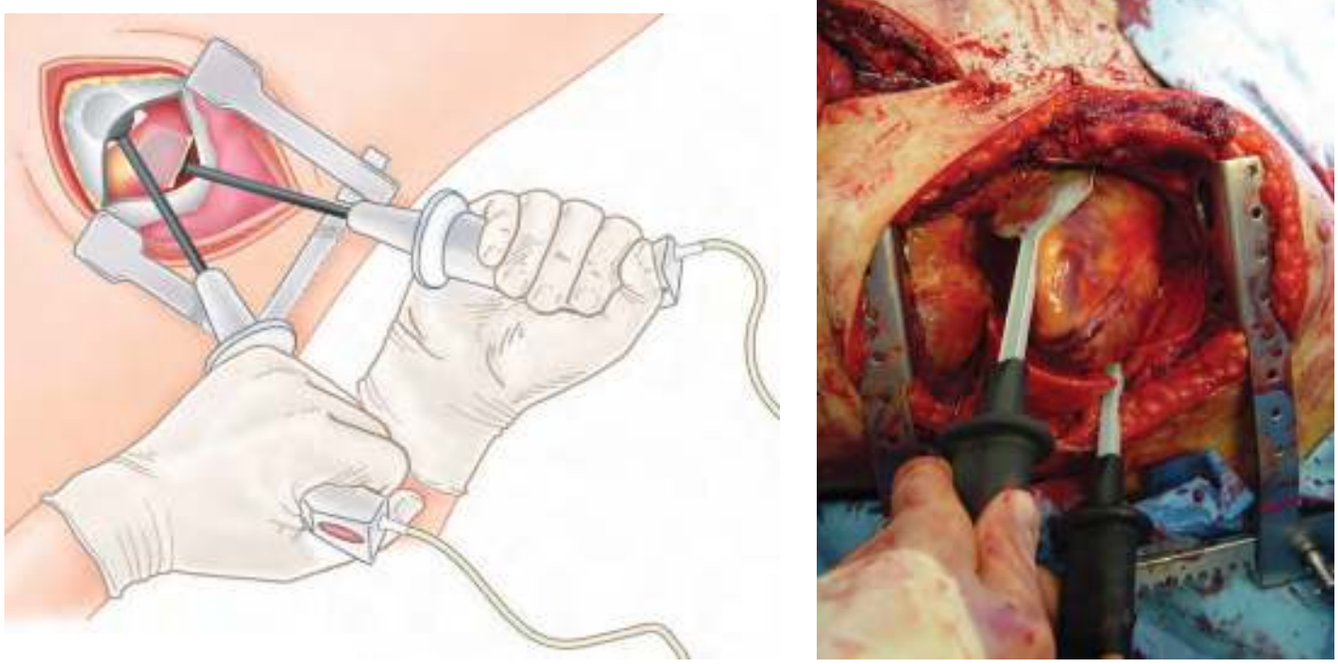
The myocardium can be deliberately placed in fibrillation to halt myocardial contractions and repair large ventricular wounds. **This should be performed only if other techniques of cardiac wound repair are unsuccessful.** Choose the proper size internal cardiac paddles. The paddle size is 2 cm for an infant, 4 cm for a child, and 6 cm for an adolescent or adult. Insert the sterile paddles into the sterile handles (**Figure 56-9**). Pass the cables exiting the base of the handles to an assistant to insert into the defibrillator machine ports. Instruct the assistant to turn on the defibrillator.

Place the internal cardiac paddles on the anterior and posterior surfaces of the heart (**Figure 56-10**). While the paddles can be placed directly on the heart, it is recommended by some to place sterile saline-moistened gauze squares between the paddles and the heart to aid in conduction. Apply 20 Joules (J) of energy through the paddles to fibrillate the adolescent or adult heart. Begin with 5 J of energy to fibrillate the infant or child heart. Proceed upward incrementally as required to a maximum of 20 J to fibrillate the infant or child heart. **Quickly repair the cardiac wound.**

Perform intermittent open cardiac massage (Chapter 55) while repairing the fibrillating myocardium to maintain cardiac output. Do not allow the heart to fibrillate for more than 3 minutes. Defibrillate the heart with the internal paddles using 20 J of energy for the adult and adolescent heart. Repeat the defibrillation with 20 J of energy until the heart begins beating. **In general, do not use more than 20 J to defibrillate the adult or adolescent heart with the internal cardiac paddles as myocardial necrosis can occur.** It may be occasionally necessary to increase the energy in the adolescent or adult to 40 J then 60 J if multiple attempts at 20 J are ineffective. Begin with 5 J of energy to defibrillate the infant or child heart. Proceed upward incrementally as required to a maximum of 20 J to defibrillate the infant or child heart.

ALTERNATIVE TECHNIQUES

There are several alternative techniques for cardiac wound repair.[13-15] They cannot be recommended for use in the Emergency Department at this time due to lack of information. They may be adopted in the future with some modification. Pericardial drainage for tamponade may stabilize the patient long enough to get them to the Operating Room for surgical repair.[13] BioGlue (CryoLite Inc.,



A B

FIGURE 56-10. Application of the internal cardiac paddles. ***A.*** Sterile saline-moistened gauze can be placed between the paddles and the heart to increase conductivity. ***B.*** The paddles are properly positioned in a patient. (Used with permission from Cothren CC, Moore EE: Emergency department thoracotomy for the critically injured patient: objectives, indications, and outcomes. *World J Emerg Surg* 2006; 1:4.)

Kennesaw, GA) has been used to repair penetrating cardiac wounds successfully in the Operating Room.[14] This BioGlue is made of synthetic bovine albumin and glutaraldehyde. The Wyss Institute at Harvard University developed a specialized catheter to fix holes in the heart.[15] It uses a biodegradable adhesive and patch with ultraviolet light to patch the beating heart.

A new device has recently been developed.[16] It consists of a flexible plastic shaft (approximately 3 mm in diameter and 12 cm long), 4 cm diameter silicone suction cup, and a circular low-profile collapsible blood flow blocking membrane (**Figure 56-11*A***). The suction cup is adjustable to different positions along the shaft. The collapsible membrane is made of natural rubber and is 3.5 cm in diameter and 1 mm in width. The collapsible blood flow blocking membrane is inserted through the cardiac injury (**Figure 56-11*B***). The silicone suction cup is then moved down to the surface of the heart to abut the ventricle wall, creating a firm seal (**Figures 56-11*C* and *D***). The device is removed by the Surgeon by sliding the suction cup along the plastic shaft and away from the surface of the ventricle and then pulling the device out of the wound. This device is not yet available commercially but will make cardiac wounds easier to manage in the Emergency Department.

AFTERCARE

If the patient is resuscitated, cover the thoracotomy wound with saline-moistened gauze and a simple dressing. Administer broad-spectrum antibiotics if not done previously. Immediately transport the patient to the Operating Room for definitive repair of the cardiac wound and any other injuries by a Trauma or Cardiovascular Surgeon.

COMPLICATIONS

The complications associated with digital pressure include further destruction of tissue. Foley catheters can become dislodged and restart troublesome bleeding if pulled too tightly. They can also

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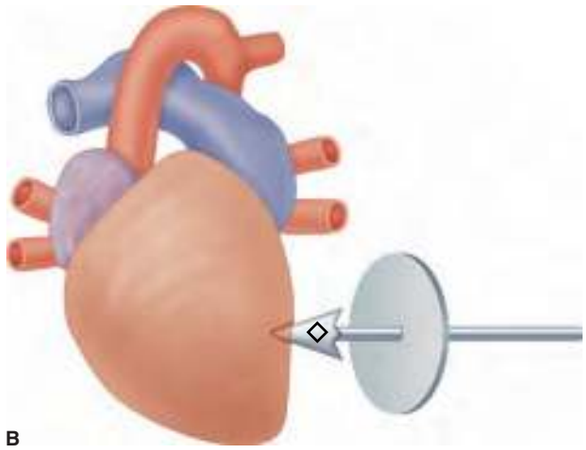
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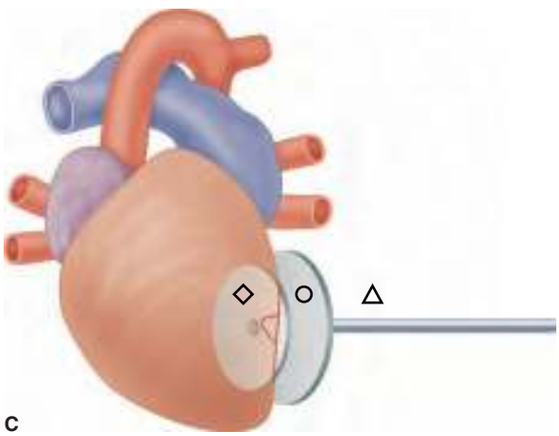
C

D

FIGURE 56-11. A new device for the stopping of cardiac wound bleeding ***A.*** The components include the silicone suction cup (O), flexible shaft (Δ), and low-profile collapsible blood flow locking membrane (◊). ***B.*** Insertion of the low-profile membrane into the cardiac defect. ***C.*** The device in place. ***D.*** The device in use. (Photos courtesy of Dr. Rezende-Neto.)

enlarge a cardiac wound if the cuff pulls through the wound. A drop in cardiac output can result if the cuff obstructs the cardiac valves, impinges on the chordae tendineae, or occupies too much space within the cardiac chamber. Decrease the cuff size by withdrawing some of the saline from the cuff. Suturing is probably associated with the greatest rate of complications. Tearing of the myocardium is a frequently encountered problem. The suture should be tied just tight enough to stop the bleeding and not necessarily achieve complete hemostasis. Care should be exercised to avoid ligation of the major coronary vessels and their branches. Dysrhythmias can result from occlusion of venous inflow (Sauerbruch maneuver) or injury to the coronary vasculature. If the patient is resuscitated, administer broad-spectrum antibiotics to prevent any potential infectious complication.

SUMMARY

Injuries to the heart can be devastating. The role of the Emergency Physician is to rapidly and temporarily control bleeding and ensure the transport of the patient to the Operating Room for definitive repair. The Emergency Physician must be versed in the techniques used to repair cardiac wounds and resuscitate the patient if there is a delay in transporting the patient to the Operating Room.

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57 Hilum and Great Vessel Wound Management

Eric F. Reichman

INTRODUCTION

Injuries to the thoracic great vessels can be a significant cause of morbidity and mortality. Large vessels in the hilum of the lung include the pulmonary artery and vein. The great vessels also include the vena cava, aorta, innominate artery, subclavian artery, and subclavian vein. The mortality from injuries to the subclavian artery is approximately 5% if patients who are moribund on admission to the Emergency Department are excluded.[1] However, the mortality from injury to the vena cava and the pulmonary vessels is over 60%.[2] While over 85% of patients with penetrating injuries to the thorax are stable, the remainder present in varying levels of hypovolemic shock. They may have bled externally or into the chest. Each hemithorax can hold up to one-half of an individual's blood volume. In these cases, an Emergency Department thoracotomy may be performed for hypovolemic shock.

ANATOMY AND PATHOPHYSIOLOGY

Injury to the thoracic great vessels may be due to blunt trauma, diagnostic procedures, iatrogenic causes, or penetrating trauma. Crush injuries, deceleration injuries, motor vehicle versus pedestrian collisions, and penetrating thoracic injuries may all signify an injury to a thoracic great vessel. The vessels that are most commonly injured include the aorta, innominate artery, pulmonary vein, and venae cavae.

The portable anteroposterior chest radiograph is the initial radiographic screening. It may reveal loss of the aortic knob contour, left-sided pleural effusions, mediastinal widening, nasogastric tube deviation, or tracheal deviation, all of which suggest injury to a great vessel. Other findings suggestive of great vessel injury include depression of the left mainstem bronchus, left apical capping, narrowing of the carinal angle, sternal fractures, opacification of the aortopulmonary window, and widening of the paraspinous stripe.

Numerous physical examination findings are suggestive of a thoracic great vessel injury. Asymmetric pulses or unequal blood pressures between the extremities are quick and simple to evaluate. Hypotension may be due to internal or external hemorrhage. Steering wheel contusions, sternal fractures, thoracic spine fractures, and a left-sided flail chest signify potential intrathoracic injury. A thoracic outlet hematoma or a hoarse voice can occur from injury to the aorta or one of its major branches. Paraplegia may be due to hypotension or an aortic disruption.

INDICATIONS

Attempts should be made to control any laceration or rupture of the thoracic great vessels.[3,4]

CONTRAINDICATIONS

There are no absolute contraindications to temporarily controlling any hemorrhage from a thoracic great vessel after performing a thoracotomy (Chapter 54). The thoracotomy should not be performed if the patient has obvious signs of death, no vital signs in the field, or no vital signs for over 15 minutes. Refer to Chapter 54 for a discussion in which a thoracotomy is contraindicated, as any hilum or great vessel repair is also contraindicated. A pericardial tamponade or cardiac injury may require management prior to managing

FIGURE 57-1. Examples of several atraumatic vascular clamps.

a great vessel injury. There is a less than 1% chance of survival if the patient has multiple gunshot wounds to the torso.[5,6] It is up to the discretion of the treating physician in cases of multiple gunshot wounds to the torso if a thoracotomy is to be done due to the generally accepted unsalvageability.[5,6] It is also contraindicated if the patient is clearly deceased.

EQUIPMENT

- 3–0 Prolene suture
- Foley catheters, sizes 14 to 20 French
- 10 inch needle driver
- Satinsky, or other, atraumatic vascular clamp (**Figure 57-1**)
- Sterile saline
- 20 mL syringe
- Laparotomy pads
- Gauze 4 × 4 squares
- Umbilical clamp
- Hemostats

PATIENT PREPARATION

There is no preparation required other than performing an anterolateral thoracotomy (Chapter 54). The patient should be intubated, ventilated with 100% oxygen, and fully monitored (i.e., telemetry, a noninvasive blood pressure cuff, and pulse oximetry). Instruct a nurse to administer intravenous broad-spectrum antibiotics that cover skin flora, gram-positive organisms, and gram-negative organisms. The Emergency Physician should wear full personal protective equipment to protect themselves from contact with the patient's blood and body fluids. Although time is of the essence and this is an emergent procedure, aseptic technique should be followed. A thoracotomy must first be performed. Refer to Chapter 54 for the complete details of a thoracotomy.

TECHNIQUES

DIGITAL OCCLUSION

Digital pressure may be used to control small lacerations of the thoracic vena cava. It will provide adequate hemostasis while repairing the wound. **Place a fingertip over the defect**. **Do not place a finger in the defect.** Place horizontal mattress sutures using 3–0 Prolene to close the defect. Unfortunately, the fingertip interferes with visualization of the wound and places the Emergency Physician at risk for a needlestick injury. The fingertip will have to be intermittently removed to place the sutures.

Digital pressure and rapid transport to the Operating Room is the most practical method of dealing with injuries to the subclavian vessels. These vessels are extremely difficult to control through a traditional anterolateral thoracotomy incision. If digital pressure is ineffective, pack the apex of the thoracic cavity with laparotomy pads or gauze squares and apply compression from below.

FOLEY CATHETER TECHNIQUE

One may occlude a wound to a great vessel by inserting a Foley catheter into the vessel, inflating the cuff, and placing it under gentle traction (**Figure 57-2**). As with digital occlusion, this is only a temporizing measure. Inflate the cuff of the Foley catheter with sterile saline to check its integrity and look for leaks. Deflate the cuff. **Place a hemostat on the Foley catheter proximal to the cuff. This will prevent air from being drawn through the catheter and causing an air embolism.**

Identify the location of the vascular injury. Insert the catheter through the wound until the cuff is within the vessel (**Figure 57-2*A***). Open the hemostat, inflate the cuff with 5 to 10 mL of sterile saline, and reclamp the Foley catheter. **This step should be completed quickly to prevent air from being drawn through the catheter. Do not overinflate the cuff so that it occludes flow through the vessel. Apply gentle traction to the catheter (Figure 57-2*B*). Apply just enough traction to mostly occlude the wound and slow the**

FIGURE 57-2. The Foley catheter technique to occlude an injury to a great vessel. ***A.*** The catheter is inserted through the wound and into the vessel. ***B.*** The cuff is inflated and gentle traction (*arrow*) is applied to occlude the wound with the cuff. ***C.*** An umbilical clamp is placed to prevent the catheter from migrating inward.

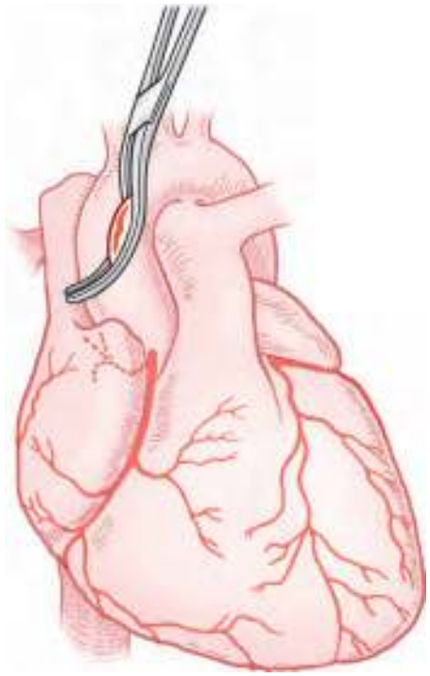


FIGURE 57-3. A Satinsky vascular clamp may be used to partially occlude the great vessel and isolate the injury.

bleeding. Do not try to provide complete hemostasis. Providing complete hemostasis can result in excessive traction on the catheter causing the cuff to pull through the wound. This can enlarge the wound and further injure the vessel. Place an umbilical clamp or hemostat on the catheter just outside the vessel to prevent it from migrating into the vessel (**Figure 57-2*C***).

The Foley catheter technique is simple and effective. It avoids the problems associated with digital occlusion. It does not interfere with visualization of the wound or the simultaneous performance of cardiac massage. Intravenous catheter tubing may be inserted into the lumen of the Foley catheter to infuse crystalloid solutions or red blood cells directly into the heart and central circulation.

CROSS-CLAMPING TECHNIQUE

Injuries to the thoracic great vessels can bleed profusely and are difficult to control. Digital occlusion is often ineffective. An atraumatic Satinsky vascular clamp, or other atraumatic vascular clamp (**Figure 57-1**) in the thoracotomy tray, can be placed to partially occlude a great vessel and isolate the injury (**Figure 57-3**). This will provide temporary hemostasis until definitive repair in the Operating Room.

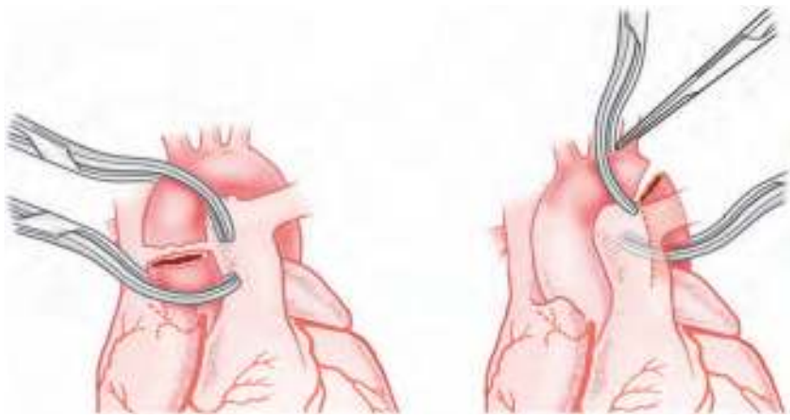
Large wounds and complete transections are difficult to manage. Injuries to the pulmonary vasculature in the region of the hilum are most expeditiously controlled by placing an atraumatic vascular clamp across the respective hilum.[7] Grasp the hilum between the thumb and the forefinger. Place the clamp carefully around the entire hilum. Take care not to injure the pulmonary parenchyma or the vessels any further. Vascular injuries may be controlled by placing a cross-clamp proximal to the injury and occluding the backbleeding with additional clamps (**Figure 57-4**). These patients should be immediately transported to the Operating Room to be placed on bypass and repair the injuries. **It is not recommended for the Emergency Physician to suture great vessel injuries in an attempt to repair them.**

AFTERCARE

If the patient is resuscitated, cover the thoracotomy wound with a saline-moistened gauze and a simple dressing. Administer broad-spectrum antibiotics if not done previously. Immediately transport the patient to the Operating Room for definitive repair of the great vessel injury and any other injuries by a Trauma or Cardiovascular Surgeon.

COMPLICATIONS

The complications associated with digital pressure include extending the injury if the procedure is not performed carefully. Inaccurate digital control can lead to unnecessary loss of blood during transport of the patient to the Operating Room. Foley catheters, if pulled too tightly, can become dislodged and restart troublesome bleeding. They can also enlarge a wound if the cuff pulls through the wound. The cuff may obstruct flow through the vessel and compromise cardiac output. Decrease the cuff size by withdrawing some of the saline from the cuff. An overly rough mobilization and clamping can increase the size of the injury and cause massive bleeding. Cross-clamping of the aorta and/or pulmonary artery will obstruct peripheral blood flow. The vessel must be repaired or the patient placed on bypass to prevent anoxia and permanent neurologic dysfunction.



A B

FIGURE 57-4. Cross-clamping of vascular injuries. ***A.*** Cross-clamp the great vessels to provide temporary hemostasis. ***B.*** Distal vessels must also be cross-clamped to prevent backbleeding.

SUMMARY

Injuries to the thoracic great vessels carry a high mortality as bleeding occurs unimpeded into the pleural space. The survival of the patient depends on their presenting condition as well as the speed and accuracy with which the intrathoracic hemorrhage is controlled.

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58 Thoracic Aortic Occlusion

Eric F. Reichman

INTRODUCTION

Temporary thoracic aortic occlusion should be performed during an Emergency Department thoracotomy for hypovolemic shock. It preserves cerebral and coronary artery perfusion pressure.[1] The blood flow to the viscera below the cross clamp, however, falls to less than 10% of baseline flow.[2] This can be advantageous since it stops distal hemorrhage, but it can later result in the undesired metabolic consequences of acidosis, hyperkalemia, and multiple organ system failure.[3,4] The use of the Emergency Department thoracotomy is likely to decrease as the resuscitative endovascular balloon occlusion of the aorta (REBOA) technique (Chapter 74) becomes more established and available.

ANATOMY AND PATHOPHYSIOLOGY

The aorta begins at the left ventricle and gives rise to the arteries of the body, directly or indirectly (**Figure 58-1**). It leaves the ventricle and is directed upward as the ascending aorta. It arches to the left and backward at the level of the sternal angle to become the aortic arch. The arch gives rise to the brachiocephalic trunk, left common carotid artery, and left subclavian artery. The aortic arch is directed inferiorly after giving rise to the left subclavian artery and is known as the descending aorta. The descending aorta is subdivided into the thoracic portion above the diaphragm and the abdominal portion below the diaphragm. It descends through the posterior mediastinum, lying first against the left side of the fifth thoracic vertebral body. As it descends, it gradually approaches the midline of the 12th thoracic vertebral body, at which point it passes through the diaphragm.

The esophagus is a thin, muscular tube measuring approximately 2.0 to 2.5 cm in diameter. It descends along the vertebral bodies. It travels forward, away from the vertebral bodies, and to the right at the level of the ninth thoracic vertebral body. It traverses the diaphragm at the level of the 10th thoracic vertebral body. It lies posterior and medial to the descending thoracic aorta throughout most of its course. It migrates as it travels distally, so that its lower part lies in front of the aorta just above the diaphragm (**Figure 58-1**).

INDICATIONS

The primary reason to occlude the descending thoracic aorta is to temporarily direct blood flow from below the diaphragm to preserve flow to the brain and heart. The descending thoracic aorta may be occluded in patients with penetrating thoracic or abdominal trauma in which hypovolemic shock and clinical deterioration are

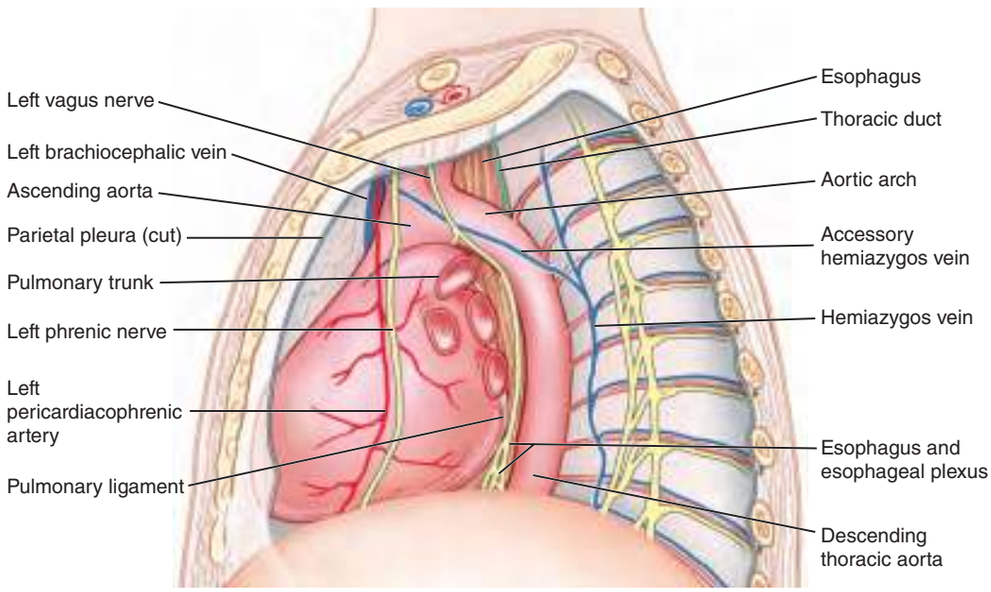

FIGURE 58-1. Anatomy of the aorta and surrounding structures of the mediastinum and left hemithorax. The mediastinal pleura has been removed to visualize the underlying structures.

not responsive to aggressive fluid resuscitation and blood transfusion. These patients should have the appropriate indications to perform an anterolateral thoracotomy (Chapter 54). The thoracic aorta may also be occluded immediately prior to laparotomy if the patient has a tense abdomen filled with blood. The abdominal incision will decompress the abdomen and result in hypotension, decreased coronary and cerebral perfusion pressure, exsanguination, and death. Uncontrollable hemorrhage anywhere below the diaphragm can be controlled by temporarily occluding the descending thoracic aorta.

CONTRAINDICATIONS

There are no absolute contraindications to temporarily occluding the descending thoracic aorta after performing an anterolateral thoracotomy. The thoracotomy should not be performed if the patient has obvious signs of death, no vital signs in the field, or no vital signs for over 15 minutes. Refer to Chapter 54 for a discussion in which a thoracotomy is contraindicated, as thoracic aortic occlusion is also contraindicated. It is also contraindicated if the patient is clearly deceased.

EQUIPMENT

- Satinsky, or other, atraumatic vascular clamp (**Figure 58-2**)
- Metzenbaum scissors
- DeBakey or large Kelly clamp
- Nasogastric tube
- Aortic compressor, Conn or homemade
- Gauze 4 × 4 squares

PATIENT PREPARATION

No preparation is required other than that of performing a thoracotomy and a pericardiotomy (Chapter 54). The patient should be intubated, ventilated with 100% oxygen, and fully monitored (i.e., telemetry, a noninvasive blood pressure cuff, and pulse oximetry). Insert a nasogastric tube. Instruct a nurse to administer intravenous broad-spectrum antibiotics that cover skin flora, gram-positive organisms, and gram-negative organisms. The Emergency Physician should wear full personal protective equipment to protect themselves from contact with the patient's blood and body fluids. While time is of the essence and this is an emergent procedure, aseptic technique should be followed. Perform the left lateral thoracotomy (Chapter 54).

FIGURE 58-2. Examples of several atraumatic vascular clamps.

TECHNIQUE

Identify the aorta by palpation. It is often easier to identify and isolate the aorta just above the diaphragm. In this location, the aorta is slightly separated from the adjacent esophagus. Elevate the left lung with the nondominant hand superiorly and medially. Instruct an assistant to maintain the lung out of the way. Place the dominant hand through the thoracotomy incision and into the posteroinferior recess of the thoracic cavity. Advance the hand along the diaphragm and toward the midline. The fingers will first encounter the vertebral bodies. The next palpable structure is the aorta. It lies anterior to the vertebral bodies. The aorta may be difficult to palpate if it is collapsed in the patient with hypovolemic shock. In the elderly, the aorta may be significantly calcified, which helps to identify it despite hypovolemia. The aorta is covered by the mediastinal pleura. Place the thumb and index finger of the nondominant hand over the aorta just above the diaphragm.

Isolate the aorta. Bluntly dissect open the mediastinal pleura overlying the aorta with a DeBakey clamp or a large curved Kelly clamp. **Never use a scalpel to open the mediastinal pleura, as it may lacerate the aorta.** Some physicians may prefer to use a Metzenbaum scissors to dissect and open the mediastinal pleura. Identify the aorta by palpation. Bluntly separate the aorta from the esophagus with the dominant hand. It may be extremely difficult to separate the aorta from the esophagus in the patient with hypotension, hypovolemia, and/or shock. Place a nasogastric tube if this has not been done previously. The nasogastric tube will be palpable within the esophagus and can be used to identify the esophagus. Hook the dominant index finger around the aorta. **Use the finger to separate the aorta from the vertebral bodies. The dissection should not be extensive. It should free approximately 3 to 4 cm of the descending thoracic aorta.**

DIRECT COMPRESSION

Direct compression of the aorta is fast and simple, it does not interfere with the operative field, and it causes less damage than the application of a clamp. Digital compression is often ineffective. Aortic compression devices have a unique shape to occlude the aorta atraumatically by compressing it against the vertebral bodies. It may be applied before or after the aorta is isolated. Homemade compression devices may use rubber tubing to occlude the aorta (**Figure 58-3*A***).[5] The Conn compressor is commercially available and uses a metal plate to occlude the aorta (**Figure 58-3*B***). Place the distal end of the compression device against the distal descending thoracic aorta (**Figure 58-3*C***). Apply downward pressure to occlude the aorta. The degree of occlusion can be controlled by increasing or decreasing the pressure applied to the aorta.

CROSS-CLAMPING

The descending thoracic aorta is most commonly occluded with an atraumatic or Satinsky vascular clamp. Aortic compression devices are rarely available in Emergency Departments or on thoracotomy trays. The aorta must first be separated and isolated from the esophagus, as described above. Place an index finger behind the descending thoracic aorta to elevate it away from the underlying esophagus (**Figure 58-4*A***). Place the Satinsky or other atraumatic vascular clamp over the aorta. One jaw should be posterior to the aorta and adjacent to the index finger while the other jaw is anterior to the aorta. Clamp the aorta and remove the index finger (**Figure 58-4*B***).

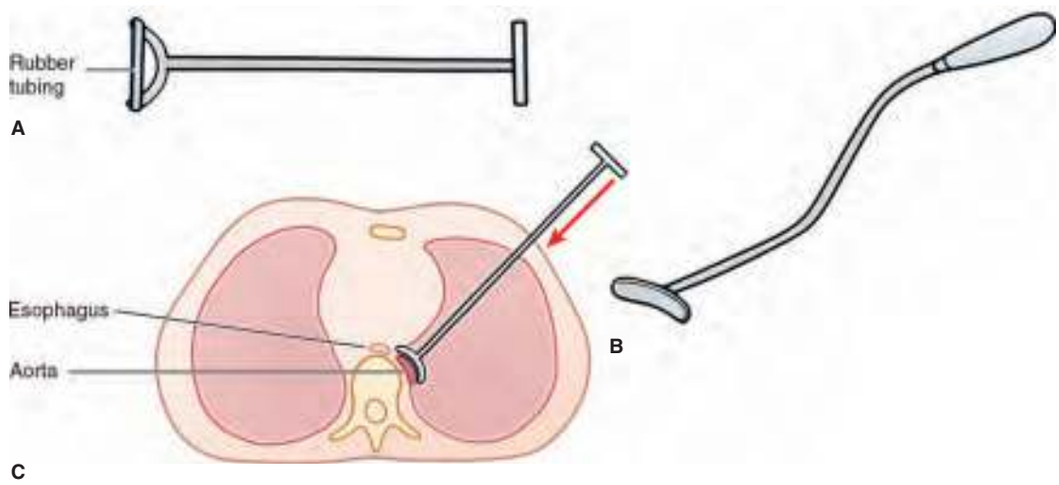

FIGURE 58-3. Aortic compression. ***A.*** The homemade aortic compression device.[5] ***B.*** The commercially available Conn compressor. ***C.*** The aorta is compressed between the distal end of the compression device and the thoracic vertebral body.

It is imperative not to clamp the esophagus. Do not clamp the aorta before it is dissected from the esophagus. Esophageal injury can lead to perforation, ischemia, and sepsis if the patient is resuscitated. Ideally, the clamp should be placed under direct visualization of the aorta. Unfortunately, this is not always practical. The index finger under the aorta can confirm the proper isolation of the aorta and the proper position of the jaws of the clamp before the aorta is cross-clamped.

AFTERCARE

If—after the thoracotomy, open cardiac massage, and aortic cross-clamping—there is return of a cardiac rhythm and a carotid pulse, the patient must be taken immediately to the Operating Room for definitive treatment. Cover the thoracotomy wound with a saline-moistened gauze and a simple dressing. The patient's blood pressure in the upper extremity should be monitored every 30 to 60 seconds after the aorta is occluded. This is best accomplished with the use of an arterial line (Chapter 72). An elevated blood pressure can result in a hemorrhagic stroke or left ventricular failure. Elevated blood pressure will require intermittent release of the aortic occlusion and/or pharmacologic management. Parenteral broad-spectrum antibiotics should be administered to prevent infection if not done previously.

COMPLICATIONS

Intercostal arteries arising from the thoracic aorta can be damaged during mobilization of the aorta. This will result in troublesome bleeding that requires operative control. The aorta, vena cava, or the esophagus can be damaged by the clamp. Aortic cross-clamping can precipitate hypertension, stroke, and left-sided heart failure. If the patient is successfully resuscitated, this should be dealt with by periodically releasing the clamp. Lack of blood flow through the artery of Adamkiewicz will cause ischemia of the distal spinal cord. There is a 5% incidence of paraplegia when the blood supply to the distal aorta and spinal cord is disrupted.[6,7] This incidence increases dramatically when the spinal cord is ischemic for more than 30 minutes.[7]

Aortic cross-clamping causes visceral ischemia. The gut loses its barrier function and becomes a cytokine-generating organ, which leads to a systemic inflammatory response and multiple organ failure. Renal and liver failure can result from a lack of blood flow.

The organs distal to the aortic clamp become severely ischemic and receive only 10% of the basal cardiac output. The anaerobic

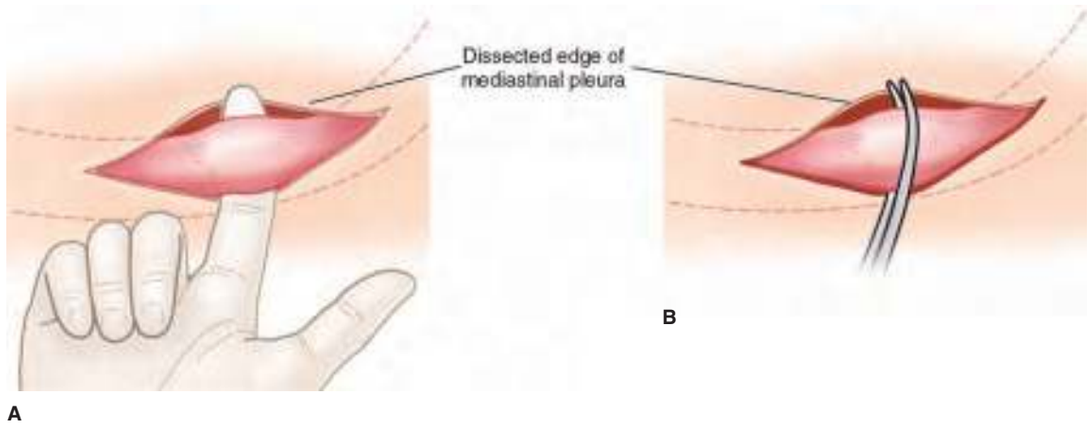

FIGURE 58-4. Aortic cross-clamping. ***A.*** The mediastinal pleura has been bluntly opened and the aorta isolated from the esophagus. ***B.*** The Satinsky, or other atraumatic, vascular clamp is placed across the aorta to occlude distal blood flow.

metabolism in these organs generates lactic acid. When the aortic clamp is released, acid and potassium are released into the central circulation and can cause a cardiac arrest. Thus, bicarbonate must be given at this time and the cardiac rhythm monitored carefully.

SUMMARY

Aortic cross-clamping is a useful adjunct to open cardiac massage in hypovolemic shock. It can help salvage patients by increasing coronary and cerebral perfusion. It may be performed as a life-saving and temporizing measure until the patient can be taken to the Operating Room for definitive management. The use of the Emergency Department thoracotomy is likely to decrease as the REBOA technique (Chapter 74) becomes more established and available.

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SECTION 4

Vascular Procedures

59 General Principles of Intravenous Access

Daniel Belmont

INTRODUCTION

The practice of Emergency Medicine frequently requires access to a patient's venous circulation. Venous access allows sampling of blood as well as administration of medications, nutritional support, and blood products. Devices such as cardiac pacing wires and pulmonary artery catheters can be introduced into the patient's central venous circulatory system.

Percutaneous, as opposed to surgical, venous access is usually rapid, safe, and well tolerated. An understanding of the various techniques available, the venous anatomy, and the indications for the procedure allows the Emergency Physician to choose the appropriate site, equipment, and method of venous access.

ANATOMY AND PATHOPHYSIOLOGY

Blood vessels have a three-layered wall composed of an internal endothelium, muscle, and a layer of connective tissue (**Figure 59-1**).[1] The muscular layer of a vein is much smaller than that of an artery. Veins can dilate and constrict in response to the pressure within them. Veins with high pressures become engorged and are easier to access. The use of venous tourniquets, dependent positioning, "pumping" via muscle contraction, and the local application of heat or nitroglycerin ointment all contribute to venous engorgement.[2] These maneuvers can be used to aid in the identification of a peripheral vein.[3]

The connective tissue surrounding veins can be a help or a hindrance during attempts at peripheral venous access. Deficient connective tissue permits the vein to "roll" from side to side and evade the needle. Tough connective tissue can impede the entry of a flexible catheter through the soft tissues and into the vein. Connective tissue serves to stabilize the vein and prevent its collapse.

Venous valves are an important aspect of peripheral venous anatomy (**Figure 59-2**).[1] They encourage unidirectional flow of blood back towards the heart. Venous valves prevent blood from pooling in the dependent portions of the extremities due to gravitational forces. **Valves can impede the passage of a catheter through and into a vein. Forcing a catheter past venous valves may damage them and contribute to later venous insufficiency.** Valves are more numerous at the points where tributaries join larger veins and in the lower extremities. Valves are largely absent within the large central veins and the veins of the head and neck.

Veins can be subdivided into central veins and peripheral veins. The important central veins for intravenous access are the internal jugular, subclavian, and femoral veins. Central veins are usually larger than peripheral veins and have fewer tributaries.

Superficial peripheral veins are generally visible beneath the surface of the skin of the extremities and neck. They are often tortuous and continually merge and divide. Peripheral veins are easiest to access at the apex of the "Y" formed when two tributaries merge into a larger vein or where the vein is straight and free of branches (and hence valves) for approximately 2 cm proximal to the site of puncture (**Figure 59-3**). These sites tend to be anchored and "roll" less than other sites. The superficial veins of the upper extremity

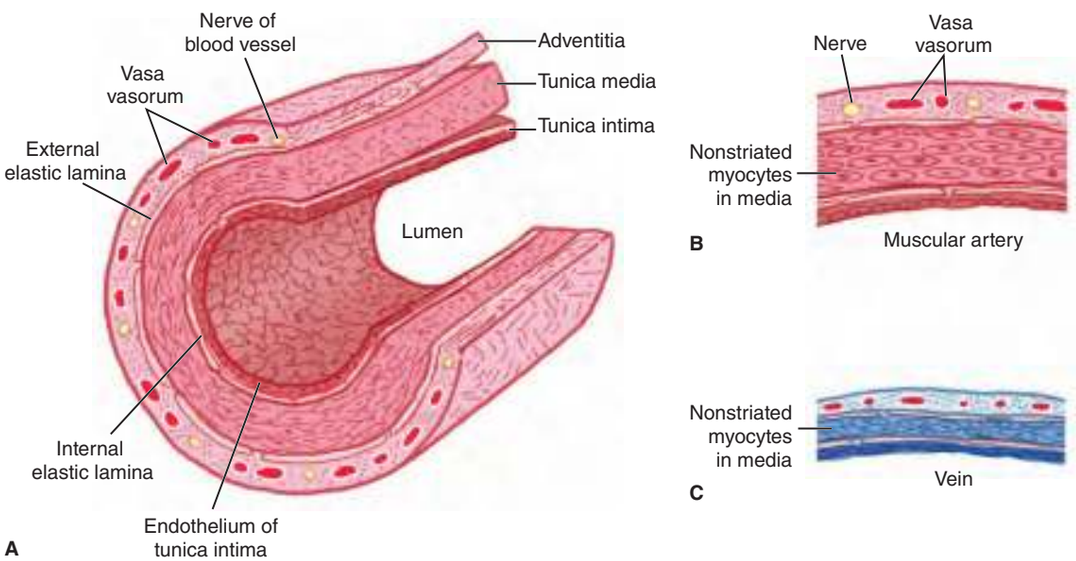

FIGURE 59-1. Comparative anatomy of an artery and a vein. ***A.*** The generic blood vessel. ***B.*** A muscular artery. ***C.*** A vein. Note the vein's thinner wall with fewer myocytes and elastic fibers. This is indicative of the lower pressure within veins compared to arteries.

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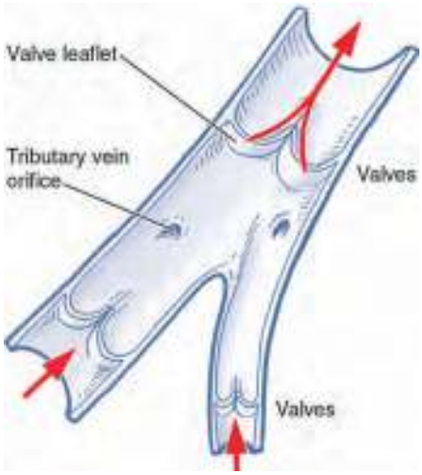


FIGURE 59-2. Venous valves. Cross-section of converging veins demonstrating the valve leaflets that only permit forward flow, proximally, toward the right heart. The *arrows* represent the directional flow of blood.

are preferred to those of the lower extremity for peripheral venous access. Indwelling catheters in the upper extremity interfere less with patient mobility and the risk of phlebitis is lower.[1]

The depth of the vein beneath the epidermis will affect the ease with which it may be accessed. Very superficial veins are often small, fragile, and easily passed "through-and-through" with a needle forming a hematoma. The deeper veins are often not visible and must be located by surface landmarks, palpation, or ultrasound (US).[4] The angle of insertion of the needle must be varied depending on the depth of the vein being punctured (**Figure 59-4**). A shallow angle of approximately 30° to 45° should be used for most small and superficial veins (**Figure 59-4*A***). A more

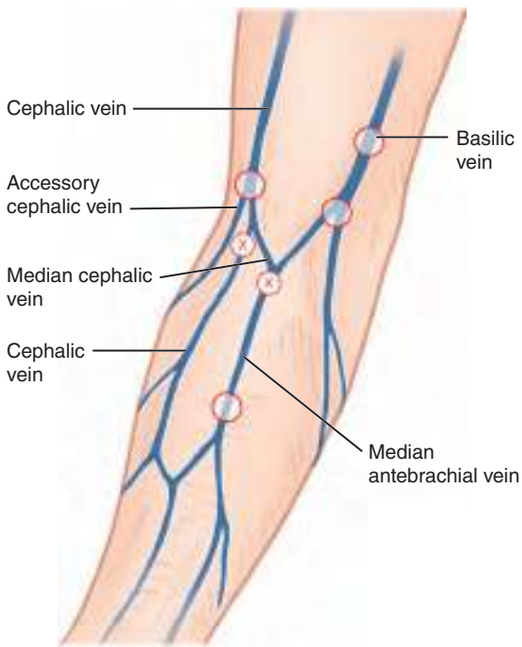


FIGURE 59-3. Preferred venous access sites. Preferred sites (*open circles*) are at the apex of converging veins or in the middle of a long straight vein. Sites just distal to branching or convergence of veins (red ⊗) are best avoided due to the presence of valves and the difficulty in threading a cannula.

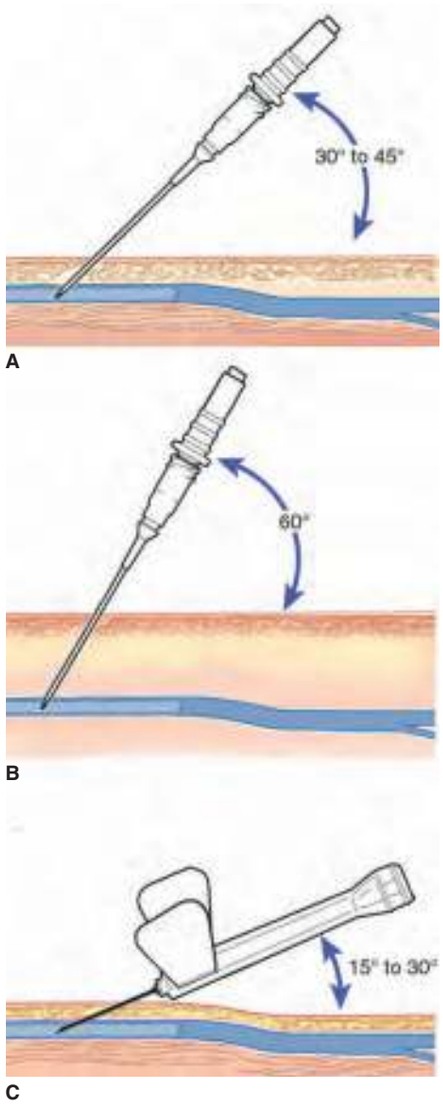


FIGURE 59-4. The angle between the needle and the skin must be varied based on the depth and diameter of the target vein. ***A.*** A shallow angle must be used for small and superficial veins. ***B.*** A steeper angle must be used for deeper veins. ***C.*** A butterfly-type needle permits the shallowest angle of entry for very small and superficial veins.

obtuse angle of approximately 60° should be used to access deeper veins (**Figure 59-4*B***). This steeper angle allows the vein to be penetrated within a reasonable horizontal distance from the skin puncture site. Very small and superficial veins should be entered at a very acute angle of approximately 15° to 30° (**Figure 59-4*C***).

INDICATIONS

Venous puncture (i.e., venipuncture) with a needle is indicated only for the sampling of venous blood. Medications may be administered as a one-time dose via this technique. The risk of medication extravasation with this technique is high and has fallen out of favor.

Venous cannulation is indicated for repeated sampling of venous blood. It is also performed for the administration of intravenous medications, fluid solutions, blood products, and nutritional support. The specific indications for peripheral venous access, central venous access, and the various techniques of venous cannulation are discussed below and in Chapters 60 through 64.

CONTRAINDICATIONS

Veins should not be accessed through infected skin. A vein proximal to a running venous infusion should not be used for venous blood sampling. The blood sample will be tainted or diluted by the infused solution. The hole in the vein may allow blood, infused solutions, and medications to extravasate into the surrounding tissues.

Venipuncture and venous cannulation of veins in an extremity with an arteriovenous fistula should be avoided. Veins in the upper extremity that may be needed for arteriovenous fistula construction for hemodialysis in the near future should not be punctured unless absolutely necessary. Scarring of the vein may complicate later surgical procedures.

CATHETER MATERIALS

Indwelling catheters are made of flexible polymers that are less likely to break or erode through the blood vessel wall than more rigid materials (e.g., steel or glass). Polymer resins, Teflon, and polyurethane are commonly used materials. Latex-containing products should be avoided due to the risk of allergic reactions. Polyurethane catheters may be weakened by alcohol-based solutions.[1] Do not infuse these solutions through polyurethane catheters.

All catheters are potentially thrombogenic. They should be left in place only as long as needed. Catheters impregnated with antiseptics (e.g., chlorhexidine and silver sulfadiazine) are commercially available and may decrease the incidence of catheter-related sepsis.[5] Chlorhexidine has been associated with immediate hypersensitivity reactions, most commonly in persons of Japanese descent.[6] **Avoid silver sulfadiazine in sulfa-sensitive patients.** Catheters are available that, when immersed briefly in an antibiotic solution prior to insertion, allow an antibiotic to bind to the catheter surfaces. These catheters may reduce the risk of infection with organisms susceptible to the chosen antibiotic.

FLUID-FLOW CONSIDERATIONS

The diameter and the length of the infusion device will affect the flow rate through the catheter.[7] Viscous fluids (e.g., blood products and albumin) will infuse more slowly than less viscous fluids (e.g., saline). These relationships can be seen in the solution of Poiseuille's equation for ideal fluid flow through a cylindrical tube:

$$\text{Flow rate } \alpha \frac{(\pi \times \text{catheter radius}^4 \times \text{pressure gradient along the tube})}{(8 \times \text{tube length} \times \text{dynamic fluid viscosity})}$$

The pressure gradient and resistance to flow are inversely proportional to the length of the tubing. **Changes in catheter diameter (i.e., catheter size) will have the most effect on flow rates.** The flow rate increases to the fourth power as the catheter's internal radius increases. Flow rates can be maximized by using the largest internal diameter (i.e., smallest gauge) catheter that will fit inside the chosen vein. Large-bore venous catheters are preferred for the highest-volume rapid fluid resuscitations, particularly of viscous blood products. Flow rates decrease as catheter length increases. **Use of the shortest possible catheter to access the chosen vein will permit the highest fluid infusion rates.** External pressure applied to the bag of infusion solution will linearly increase the flow rate.

Fluids must be administered quickly in some patients (i.e., acute blood loss, hypothermia, hypovolemia, and shock). Many devices have been developed to increase the fluid management to these patients quicker than the drip of the intravenous (IV) tubing. These include level 1 infusers, IV pumps, the LifeFlow device, and the pressure bag (**Figure 59-5**). Some of these devices have a fluid warmer in line.

Level 1 devices infuse blood products and fluids very quickly (**Figure 59-5*A***). Flow rates range from 30 to 1400 mL/min. They usually contain a heat exchanger that can be turned on and off with a switch. The heating of fluid uses a countercurrent of 42°C to deliver fluid of 36°C to 40°C. A gas vent filter removes microbubbles of gas from the IV line due to heating of the fluids. Some devices have an integrated air detector that detects the presence of air, alarms, and automatically stops the flow of fluid to the patient. One nurse or technician is usually dedicated to operate the level 1 infuser.

In 2017, the 410 Medical Company (Durham, NC) released the LifeFlow Rapid Infuser (**Figure 59-5*C***). It infuses 500 mL in 2.5 minutes. It is a hand-powered device. The dedicated person squeezes the trigger to pump 10 mL of fluid into the IV catheter. Release of the trigger loads 10 more mL into the device in anticipation of pressing the trigger again. The fluid flow resistance is felt through the handle. The disadvantages to this device are the manual hand pumping, no fluid warming capacity, and fatigue of the operator. The advantages of this device are its costs, single use, small size, and portability.

LOCATING A PERIPHERAL VEIN

Identifying a peripheral vein can sometimes be quite difficult. Dilating a vein (i.e., venodilation) can make a vein larger, easier to identify, and easier to access. These techniques are easy to perform.

Several venodilation techniques do not require any special equipment.[8] Place the extremity in a dependent position.[9] This position allows gravity to decrease venous return and dilate the veins.[10] Gently tap the skin in the area to dilate the underlying veins.[9,10] The exact mechanism of how this works is not known. It may be due to the release of chemical mediators, stimulation of nerve fibers, or a combination of both. Instruct the patient to open and close their hand. The muscular contractions of the forearm muscles increases arterial inflow distally while venous outflow is inhibited by the tourniquet.[9,11-13] Try to "milk" the veins in a distal direction. Apply your fingers over the skin, press downward, and then move the fingers distally to "milk" or back-flow the blood and dilate the distal veins.[9] Apply a warm compress, heat pack, or warm towel or submerge the extremity in warm water to dilate the veins. Be careful to not use too hot of a temperature to prevent burning the patient.

Several commercially available devices can be used to produce venodilation.[8,14] The Esmarch Bandage is a tourniquet system used to exsanguinate a limb prior to surgery. Apply it to the upper extremity starting proximally and wrap it distally, the reverse of applying it to exsanguinate the extremity. It will result in blood pooling in the distal veins and subsequent venodilation. The Rhys-Davies Exsanguinator can also be placed proximal to distal to pool blood distally and cause venodilation.[15] This device is usually not available in the Emergency Department. A vacuum device was developed to aid in obtaining venous access.[16,17] It is cumbersome and often not available in the Emergency Department.

Bedside US is being taught in all Emergency Medicine residencies. It is also taught in medical schools, paramedic programs, and air ambulance services. There are many advantages to using US to facilitate IV access. The use of US has been shown to make finding a vein easier.[4,18] US allows faster IV access in patients less than 3 years of age.[18] It can be used in patients with difficult IV access.[19,20] US use avoids central venous line placement.[21] Refer to Chapter 64 for more information on US-guided vascular access.

Another technique to finding a larger peripheral vein to cannulate is to place a small gauge intravenous catheter in a distal vein (typically the hand), keep the tourniquet in place, and infuse a small amount of crystalloid (e.g., 60 to 300 mL) through the catheter. The

FIGURE 59-5. Examples of IV fluid flow devices. ***A.*** Level 1 infuser. ***B.*** IV infusion pump. ***C.*** LifeFlow Rapid Infuser. ***D.*** Pressure bag.

fluid will flow through the canalized and collateral veins distending all the veins distal to the tourniquet.[22]

Topically applied pharmaceuticals can be used to dilate the dorsal hand veins.[23-25] Topical nitroglycerine ointment has been used for many years. Apply 1 inch of the ointment and rub it into the skin on the dorsal hand. Use gloves while applying the ointment to prevent the side effects of a headache or hypotension to the healthcare provider. Allow the ointment to sit for 2 minutes and then completely wipe it off the skin. Clean any residual nitroglycerine ointment from the skin with an alcohol swab. Identify a vein by palpation. Topical nifedipine is an alternative but is not currently available in the United States.

In recent years, numerous commercial devices have become available to visualize veins. These devices reduce the number of attempts and increase the success rate but increase the time to obtain IV access.[26] The major drawbacks of these devices are their cost. The VeinViewer (Christie Medical Holdings, Inc., Memphis, TN) uses polarized near-infrared light from light-emitting diodes to penetrate the skin and subcutaneous tissues (**Figure 59-6**).[27-29] It is available in hand-held and mounted versions. The device projects the underlying blood-filled veins onto the skin using a visible green light. The AccuVein AV300 (AccuVein LLC, Cold Spring Harbor, NY)[30] and the Veinlite (TransLite, Sugar Land, TX)[31] are similar devices but are only available in the hand-held version (**Figures 59-7 and 59-8**). The Veinlite is available in several sizes and versions. The Venoscope (Venoscope LLC, Lafayette, LA) is a hand-held device that is also available in a neonatal version (**Figure 59-9**). Evena Medical (Roseville, CA) has developed a glasses vein finder

A

C

B

FIGURE 59-6. The VeinViewer. ***A.*** Hand-held version. ***B.*** Mounted versions. ***C.*** The projection of the veins on the skin. (Photos courtesy of Christie Medical Holdings Inc.)

FIGURE 59-7. The AccuVein. (Photo courtesy of AccuVein LLC.)

and a mounted version (**Figure 59-10**). Oxy-Amp (2AI Labs, US Virgin Islands) makes several versions of glasses (**Figure 59-11**). The VeinSite (VueTek, Gray, ME) fits over the head and leaves both hands free (**Figure 59-12**).

ANESTHESIA

The use of anesthesia prior to venipuncture or venous cannulation is much appreciated by the patient, especially if the patient is a child. The use of local anesthetic solution will decrease the pain of venous access. The pain of injection can be just as uncomfortable as the venous access procedure. The application of a topical anesthetic (e.g., ELA-Max [Ferndale Laboratories, Ferndale, MI] or EMLA [AstraZeneca, Wilmington, DE]) is designed to enhance transdermal absorption and requires approximately 30 to 60 minutes to adequately anesthetize the skin and subcutaneous tissues. This time delay limits their use in the Emergency Department (Chapter 154).

Numerous alternative anesthesia methods can be considered (Chapter 154). These are rarely available or used in the Emergency Department. Local anesthetic absorption through the skin can be enhanced to decrease the time it takes to provide anesthesia. These methods include using sound energy (sonophoresis), electrical energy (iontophoresis), and epidermal tape stripping.[32-34] A portable, hand-held laser used prior to the application of topical anesthesia for 5 minutes effectively reduces the pain of venous access.[35,36] Topical vapocoolant spray will briefly anesthetize the skin long enough to allow for venous access.[37-43] Local anesthetic patches that are heat activated to increase transdermal absorption of the local anesthetic agent

A

B

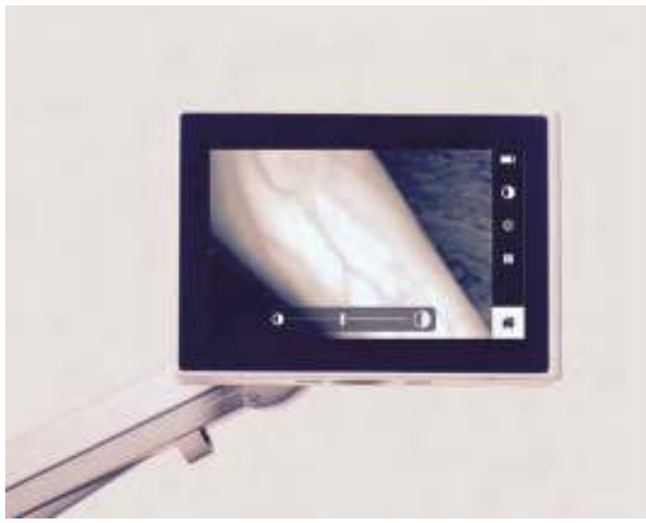
C

FIGURE 59-8. The Veinlite. ***A.*** The device. ***B.*** The underside light. ***C.*** Finding a vein. (Photos courtesy of Translite Inc.)

A B

FIGURE 59-9. The Veinoscope. ***A.*** Regular version. ***B.*** Neonatal version. (Photos courtesy of Venoscope LLC.)

A



B

FIGURE 59-10. Evena Medical's vein finders. ***A.*** Glasses. ***B.*** The OWL. (Photos courtesy of Evena Medical.)

in approximately 20 minutes have been found to be effective.[44] Needleless jet injections of local anesthetics effectively anesthetize the skin and subcutaneous tissues.[45-47] One study questioned if the anesthesia was due to the local anesthetic agent or the jet injection procedure itself as the placebo group was just as effective in terms of anesthesia.[46] **The main advantages of these techniques, compared to injectable anesthetics, are the lack of pain during the application and the lack of tissue distortion making identification of the vein difficult.**

A

B

FIGURE 59-11. The Oxy-Amp vein finders. ***A.*** An example of the glasses. ***B.*** The different versions. (Photos courtesy of 2AI Labs.)

A

B

FIGURE 59-12. The Veinsite. ***A.*** The device. ***B.*** Using the device. (Photos courtesy of VueTek Scientific.)

FIGURE 59-13. Venous access devices. *From top to bottom:* butterfly needle with extension tubing, hollow needle, catheter-over-the-needle (i.e., angiocatheter), catheter-through-the-needle, and the guidewire-guided catheter.

VENIPUNCTURE

Five types of devices are used for vascular access (**Figure 59-13**). There are numerous variations of these devices. The butterfly needle and hollow needle are used for venipuncture. Blood may be withdrawn using a butterfly-type needle (**Figure 59-14**) or a standard hypodermic needle. The butterfly needle, attached to a short length of plastic tubing, allows for greater control while accessing small and superficial veins. It is often too short to reach deeper veins. Versions with an integral sheath to minimize accidental needle-stick injuries are available (**Figure 59-15*A***). A butterfly needle appears to decrease the hemolysis when compared to IV catheters.[48,49]

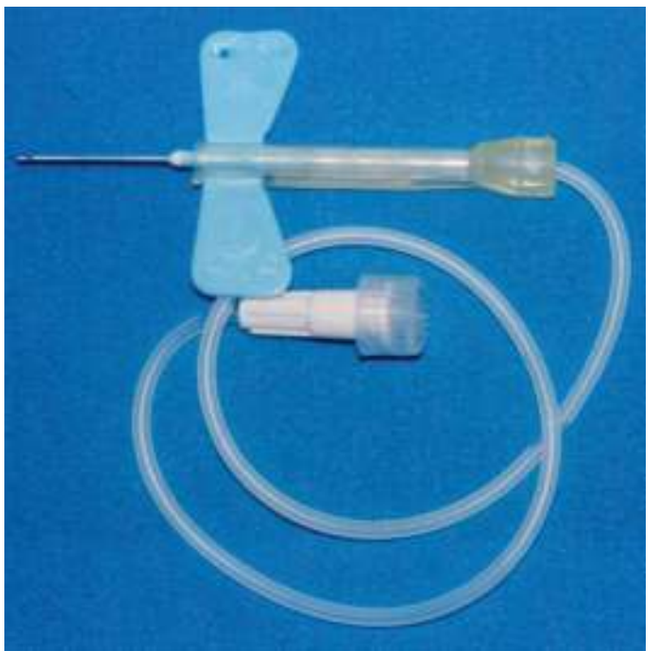
Venous blood sampling can be accomplished by one of several methods. Blood may be allowed to drip from the open end of the butterfly extension tubing into small-volume collection tubes for pediatric patients. Some form of suction is used to withdraw the blood more rapidly in older children and adults. A syringe or vacuum tube may be used (**Figure 59-16**). Vacuum tubes reduce the risk of needle-stick injuries. The amount of suction provided is fixed and may result in hemolysis of the specimen and cause small veins to collapse. Syringe aspiration allows greater control over the amount of suction applied. Large syringes can be difficult to manipulate while maintaining the tip of the needle within the vein. Use a 5 to 10 mL syringe, as larger syringes result in hemolysis of the specimen and collapse of the vein. **The needle used to transfer the specimen from the syringe to the laboratory tubes can cause a needle-stick injury.**

The Touch Activated Phlebotomy (TAP) device (Seventh Sense Biosystems, Medford, MA) was developed for venipuncture (**Figure 59-17**). The base of the device uses adhesive to stick and form an airtight seal on the skin of the upper arm. Pushing the button on the top of the device activates 30 thin needles that penetrate the skin. This micropuncture causes no pain, take 2 minutes, and obtains 100 μL of blood. Blood is collected in a reservoir with anticoagulant. The device has an indicator for when the blood collection is complete. The laboratory can extract the sample from the TAP device and analyze the blood using regular methods.

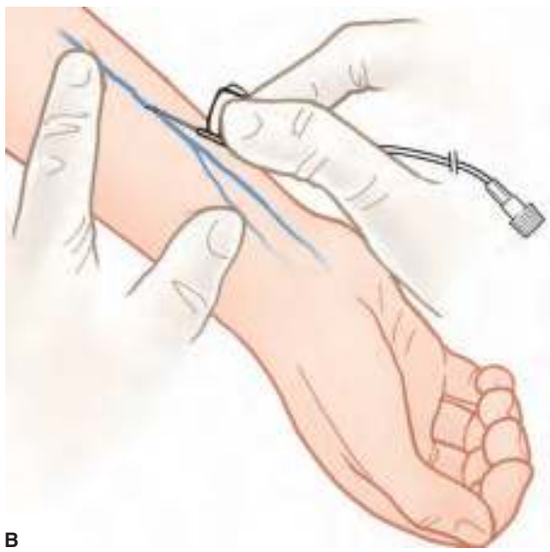
VENOUS CANNULATION

There are four main techniques of vein cannulation.[50-52] The first is the needle-only technique using a butterfly-type needle. This is seldom used today. The catheter-over-the-needle technique is most commonly used for peripheral venous cannulation. The catheter-through-the-needle technique is occasionally used but not very popular. The Seldinger wire-guided technique is most commonly used for central venous access. The major advantages and disadvantages of each technique are summarized in **Table 59-1**.

Identify the vein to be cannulated and the site of the skin puncture. Clean the area of any dirt and debris. Cleanse the skin with isopropyl alcohol, chlorhexidine solution, or povidone iodine solution and allow it to dry. Apply a tourniquet to the extremity, proximal to the venous cannulation site, to engorge the vein.[53] Additional engorgement of the vein or the use of a device to locate a vein can aid in the identification of a peripheral vein. **Do not attempt cannulation if the vein cannot be seen, palpated, or otherwise visualized (e.g., US) in the engorged state.** Place a small subcutaneous wheal of local anesthetic solution or some alternative at the skin

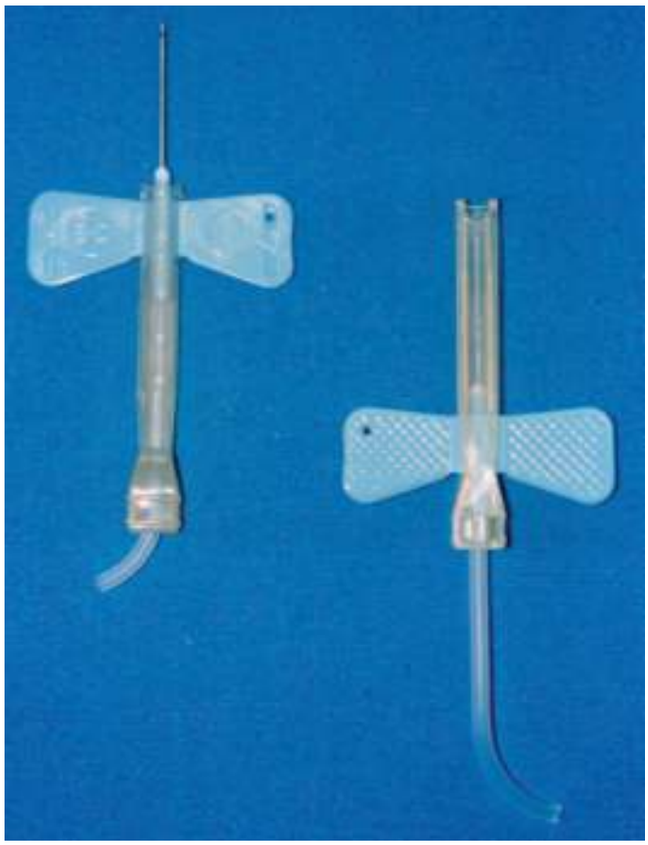


A



B

FIGURE 59-14. The butterfly-type needle. ***A.*** Butterfly needle with attached extension tubing. ***B.*** The wings of the catheter are folded together and used to direct the needle into the superficial vein. The needle may be secured within the vein and used as an infusion cannula or removed after blood samples are collected.



A

B

FIGURE 59-15. Needle-stick prevention devices. ***A.*** Butterfly needle with an integral needle sheath. The left figure demonstrates the sheath retracted and the needle exposed. The right figure demonstrates the needle safety sheathed. ***B.*** The spring-loaded catheter-over-the-needle system. The top figure demonstrates the catheter-over-the-needle. The bottom figure demonstrates the needle inside the safety handle.

puncture site to provide some comfort to the patient. The next step is to cannulate the vein by one of the methods described below.

NEEDLE-ONLY TECHNIQUE

This technique is used occasionally for short-term venous access in young children and elderly patients with fragile veins. This system is prone to malposition and infiltration. **The tip of the needle can easily lacerate the vein if the needle is not secure and allowed to move.**

Grasp and fold the wings of the butterfly needle with the dominant index finger and thumb (**Figure 59-14*B***). Briskly insert the needle with the bevel facing upward. Insert it through the skin and into the vein.[54] A flash of blood will be seen in the tubing when the tip of the needle enters the vein. Carefully advance the needle an additional 3 to 5 mm into the vein. Attach a 5 mL syringe to the extension tubing and aspirate blood. The flow of blood into the syringe confirms proper intravascular placement of the needle. Remove the tourniquet from the extremity. Securely tape the wings of the butterfly needle to

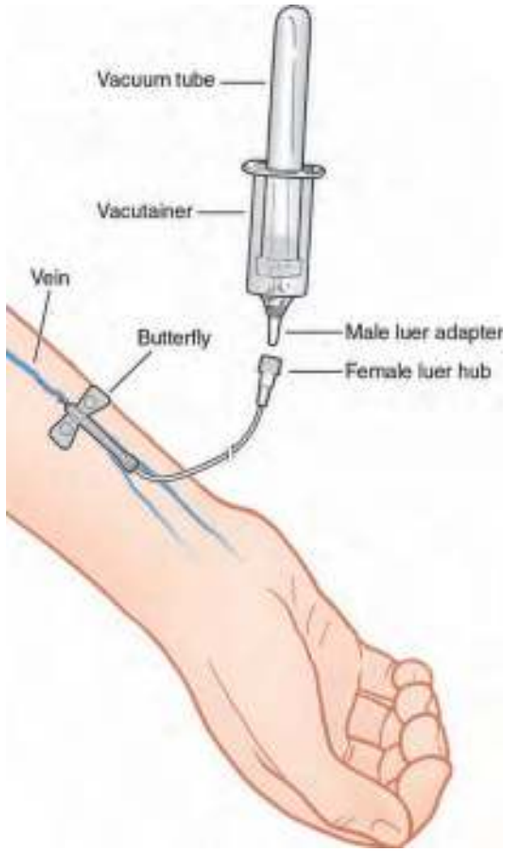


FIGURE 59-16. The Vacutainer.

the patient's skin. Remove the 5 mL syringe, attach intravenous tubing to the catheter, and begin the intravenous infusion.

CATHETER-OVER-THE-NEEDLE TECHNIQUE

The catheter-over-the-needle systems are the ones most commonly used for venous access. The infusion catheter fits closely over a hypodermic needle. The needle and the catheter are advanced as a unit into the vein. These devices are inexpensive (e.g., $1 to $4 each), come in a variety of diameters (12 to 24 gauge) and lengths, and are widely available. Versions designed to minimize accidental needle-stick injuries are available, and their use is encouraged (**Figure 59-15*B***).[55]

FIGURE 59-17. The TAP device. (Photo courtesy of AccuVein LLC.)

TABLE 59-1 Features of Venous Catheterization Techniques

	Butterfly needle	Catheter-over-the-needle	Catheter-through-the-needle	Seldinger
Diameter of vein punctured compared to catheter diameter	Same	Slightly smaller	Larger	Smaller
Catheter length compared to needle	Same	Slightly shorter	Longer, up to 61 cm (24 inches)	Unlimited
Speed of insertion	Rapid	Rapid	Slower	Slowest
Risk of extravasation	Highest	Low, higher with shorter catheters	Low	Very low
Security of catheter with patient movement	Lowest	Fair to good	Good	Excellent when sutured
Best choice for	Peripheral venous sampling	Peripheral venous infusion	Central venous Access	Central venous access
Can change catheter without new venous puncture?	No	Can use small guidewire and Seldinger technique	No	Yes

Insert the catheter-over-the-needle, with the bevel facing upward, through the skin and into the vein (**Figure 59-18*A***).[54] A flash of blood in the hub of the needle confirms that the tip of the needle is within the vein. Advance the unit an additional 2 to 3 mm to ensure that the catheter is within the vein. Hold the hub of the needle securely. Advance the catheter over the needle until its hub is against the skin (**Figure 59-18*B***). Apply pressure with the nondominant index finger over the skin above the catheter to prevent blood from exiting the catheter. Remove the tourniquet from the extremity. Securely hold the hub of the catheter against the skin. Withdraw the needle (**Figure 59-18*C***). Remove the tourniquet from the extremity. Attach intravenous tubing to the hub of the catheter and begin the infusion (**Figure 59-18*D***). Secure the catheter to the skin with tape.

Placement of these catheters is usually quick and simple. Several considerations should always be kept in mind when using the catheter-over-the-needle technique. Intravascular placement of the system is indicated by a flash of blood in the hub of the needle. Both sides of the vessel may be traversed (i.e., through-and-through) before the practitioner realizes that the needle was within the vein if the patient's venous pressure is very low or if the needle is long and narrow (**Figure 59-19*A***). The catheter will not advance if the tip of the needle is withdrawn from the vein. The catheter will not advance if the tip of the needle but not the catheter is within the vein. The catheter will push the vein off the needle (**Figure 59-19*B***). Place a finger just distal to the puncture site. Depress the skin and pull it distally to prevent the vein from "rolling" as the catheter-over-the-needle is inserted into the vein (**Figures 59-19*C* and 59-19*D***).

CATHETER-THROUGH-THE-NEEDLE TECHNIQUE

This technique eliminates the need for a needle that is as long as the catheter and eliminates the possibility of pushing the vein off the end of the needle when the catheter is advanced.[56] This system is used most commonly for central, rather than peripheral, venous access. Catheters up to 61 cm (24 inches) long are available and allow central venous access from the antecubital vein or the femoral vein. Select a catheter size that is appropriate for the patient and the site of entry. Packaged with each catheter is a needle and a needle guard. The needle will have an inner diameter that is slightly larger than the outer diameter of the catheter. The needle guard has a beveled channel in which the needle resides. The needle guard hinges closed over the needle to hold it securely and prevent the needle from shearing the catheter. Holes in the corners of the needle guard allow it to be sewn to the patient's skin.

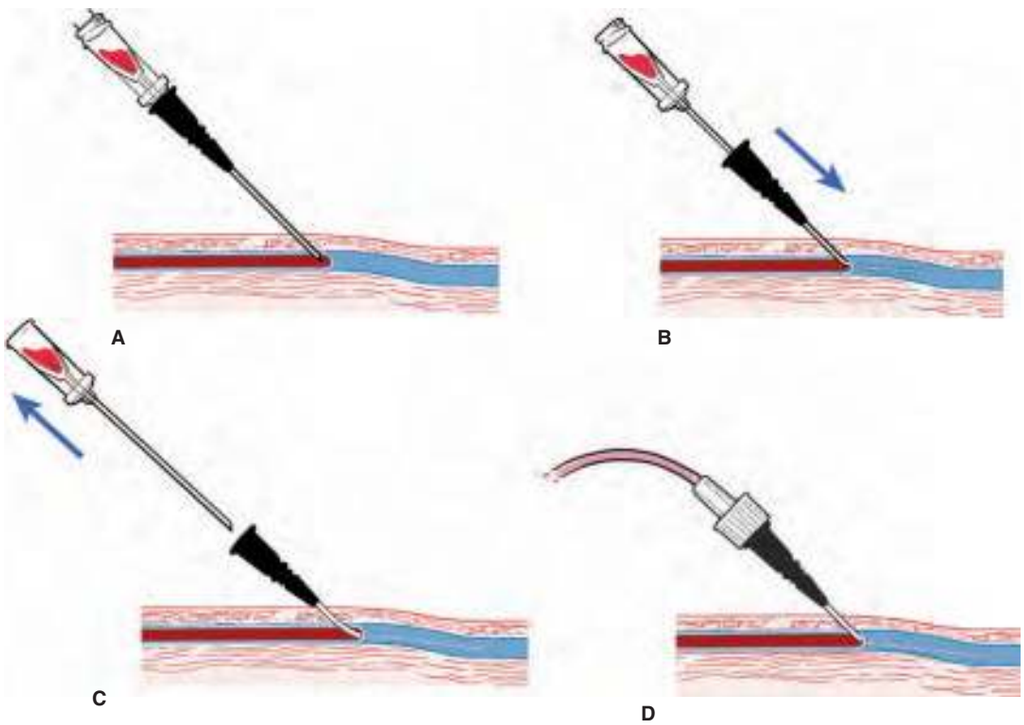


FIGURE 59-18. The catheter-over-the-needle technique. ***A.*** The vein is punctured and blood returns in the needle hub. ***B.*** The catheter is advanced over the needle and into the vein. ***C.*** The needle is removed. ***D.*** Intravenous extension tubing is attached to the catheter.

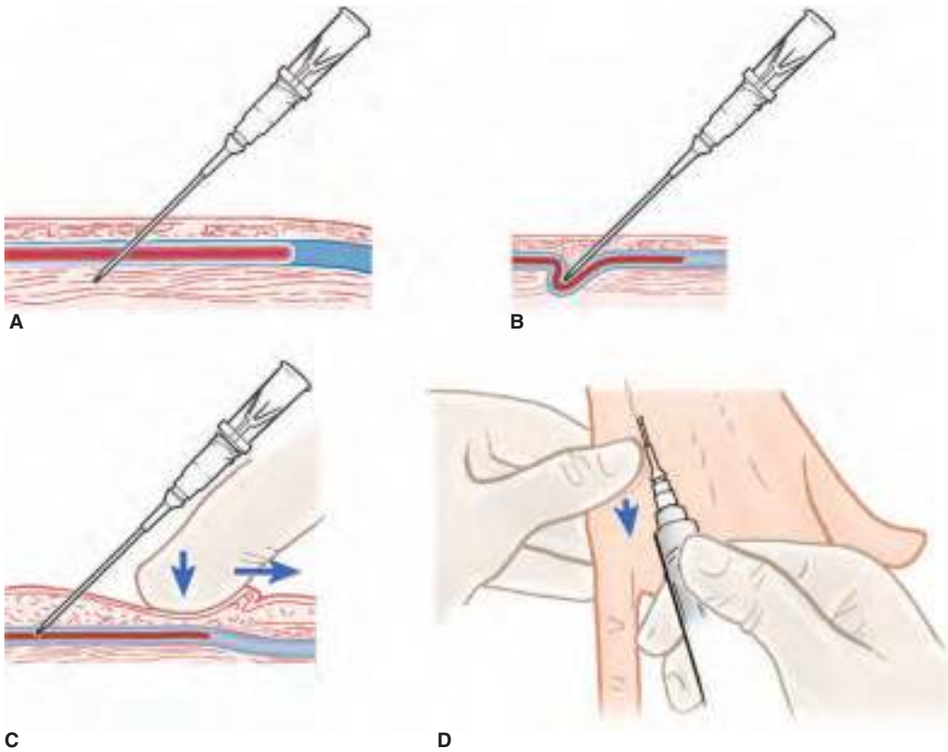


FIGURE 59-19. Pitfalls of the catheter-over-the-needle technique. ***A.*** Through-and-through puncture of the vein. ***B.*** The catheter can push the vein off the needle and prevent cannulation. ***C.*** Push a finger into the skin distal to the puncture site and pull back (*arrow*) to keep the vein straight and prevent it from moving. ***D.*** The nondominant thumb is used to pull the skin and stabilize the vein.

Place the needle on a tuberculin syringe. Insert the needle, with the bevel facing upward, through the skin and into the vein while applying negative pressure to the syringe (**Figure 59-20*A***).[54] A flash of blood in the syringe confirms that the tip of the needle is within the vein. Advance the needle an additional 2 mm to ensure that the tip of the needle is completely within the vein. Grasp and hold the needle securely with the nondominant hand. Remove the syringe with the dominant hand. Immediately place the nondominant thumb over the needle hub to prevent air from entering the vein. Remove the tourniquet from the extremity.

Insert the catheter through the hub of the needle (**Figure 59-20*B***). Advance the catheter through the needle until the desired length of catheter is within the vein. **If the catheter will not advance, remove the catheter and needle as a unit. Never withdraw the catheter through the needle. The sharp bevel of the needle may cut the catheter as it is being withdrawn and result in a catheter embolism in the central venous circulation.** Remove the tourniquet from the extremity.

Withdraw the needle over the catheter (**Figure 59-20*C***). **Do not allow the catheter to be withdrawn through the needle.** Continue to withdraw the needle until the tip is completely outside the skin. Apply the needle guard over the needle (**Figure 59-20*D***). Attach intravenous tubing to the hub of the catheter and begin infusing fluids through the catheter. Secure the catheter and needle guard to the skin with tape and/or sutures.

The main disadvantage of this technique is the possibility of the needle tip shearing off the catheter causing a catheter embolism in the venous circulation. **This can be prevented by not withdrawing the catheter through the needle and applying the needle guard immediately after the needle is withdrawn from the skin.** The contaminated needle must be handled to some extent, creating a potential risk for a needle-stick injury. The needle used for the venipuncture must be larger in diameter than the catheter. This limits the practical diameter of the catheter. The needle punctures a hole in the vessel larger than the catheter and increases the risk of hematoma formation.

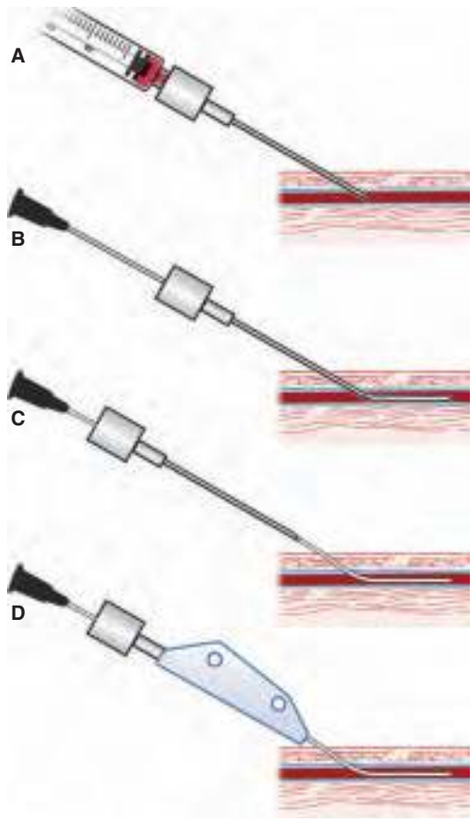


FIGURE 59-20. The catheter-through-the-needle technique. ***A.*** The vein is punctured with the needle. ***B.*** The syringe has been removed. The catheter is inserted through the needle and into the vein. ***C.*** The needle is withdrawn over the catheter and completely outside the skin. ***D.*** The needle guard is attached to secure the needle and prevent it from shearing the catheter.

SELDINGER TECHNIQUE

First described by Seldinger in 1953, this technique allows for the placement of a catheter over a guidewire rather than directly over a needle.[57-59] The guidewire used must be longer than the catheter. The needle used to insert the guidewire can be short and of a smaller gauge than the catheter. The catheter type may be changed later without the need for a new venous puncture. Materials needed for catheter insertion are commercially available in a prefabricated kit (Teleflex Medical, Cleveland, OH; Cook Medical Inc., Bloomington, IN).

The Seldinger technique is most commonly used for central venous catheter insertion. It can be used for peripheral venous access if a short, thin guidewire is available. US may be a useful adjunct with this technique (Chapter 64).[60] All-in-one arterial line kits are commercially available. They are intended for peripheral arterial line placement but can also be used to place catheters in a peripheral vein, the brachial vein, and the external jugular vein.

The Seldinger technique for venous catheter insertion is described briefly here. Refer to Chapter 63 (central venous access techniques) for a more complete discussion. Choose the puncture site. Prepare the patient for the procedure. Cleanse the skin with isopropyl alcohol, chlorhexidine solution, or povidone iodine solution and allow it to dry. Apply a tourniquet to the extremity, proximal to the venous cannulation site, to engorge the vein.[53] The vein may first be located with a small "finder" needle if there is doubt about its exact location. Insert a 25 or 27 gauge needle attached to a 5 mL syringe, with the bevel facing upward, through the skin. Advance the needle while applying negative pressure to the syringe. A flash of blood signifies that the tip of the needle is within the vein. Note the depth and location of the vein based on the depth and direction of the "finder" needle.

Insert the thin-walled introducer needle while applying negative pressure to the syringe. The introducer needle has a tapered hub on the proximal end to guide the guidewire into the needle lumen. **Do not use a standard hypodermic needle as it does not allow for the passage of the guidewire.** A flash of blood in the needle hub signifies that the tip of the needle is within the vein (**Figure 59-21*A***). Advance the needle an additional 1 to 2 mm into the vein. Hold the needle securely in place and remove the syringe.

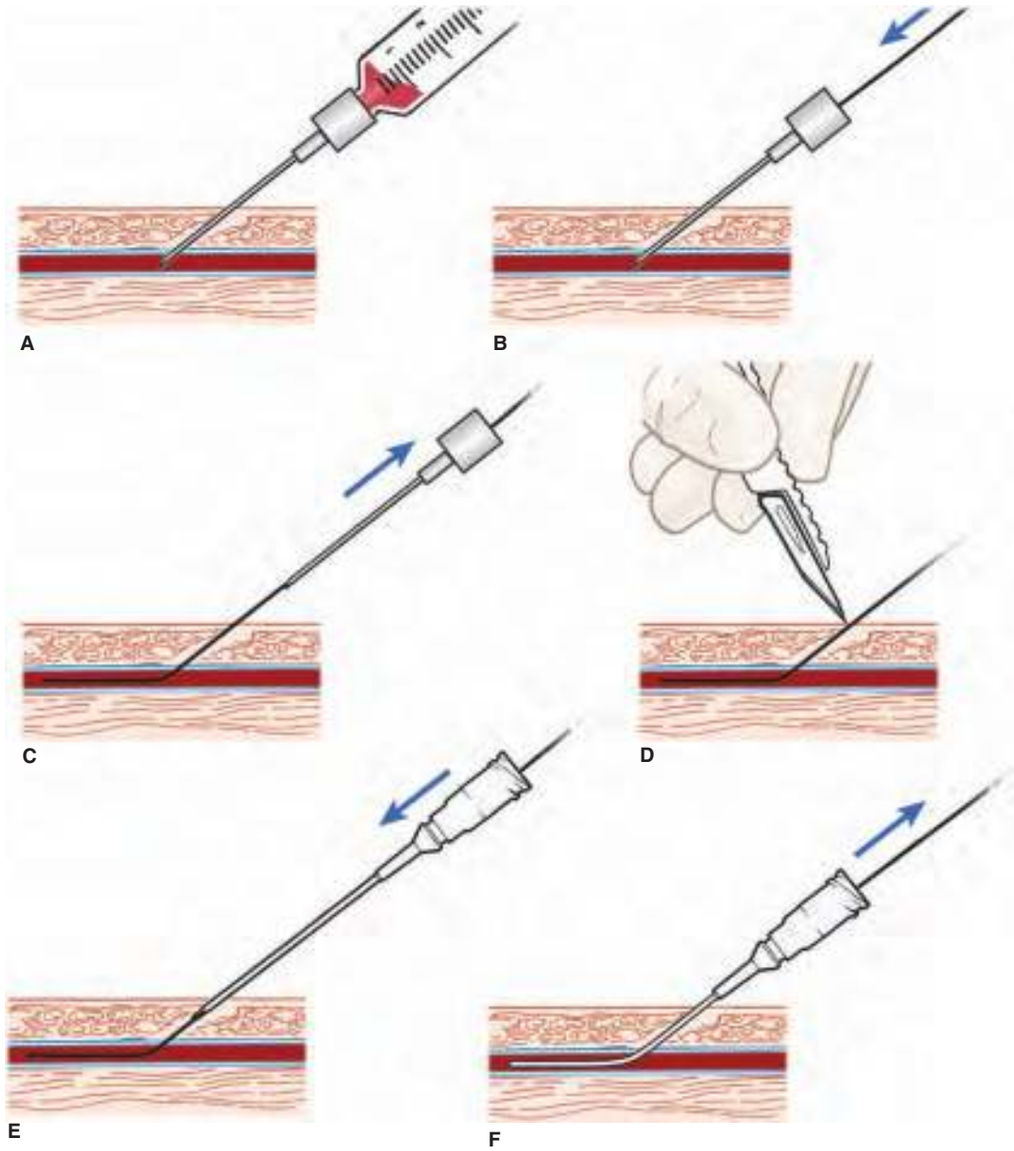


FIGURE 59-21. The Seldinger technique. ***A.*** The vein is punctured by the needle and blood is aspirated. ***B.*** The syringe has been removed. The guidewire is inserted through the needle and into the vein. ***C.*** The needle is withdrawn over the guidewire. ***D.*** The skin puncture site is enlarged to permit catheter passage. ***E.*** The catheter is advanced over the guidewire and into the vein. ***F.*** The guidewire is withdrawn through the catheter.

Occlude the needle hub with a sterile gloved finger. This will prevent air from entering the venous system. Insert the guidewire through the hub of the needle (**Figure 59-21*B***). Advance the guidewire to the desired depth, ensuring that it is at least several centimeters beyond the beveled end of the needle. **Never let go of the guidewire with both hands at the same time to prevent loss of the guidewire into the venous circulation.** Hold the guidewire securely in place. Remove the needle over the guidewire (**Figure 59-21*C***). Make a small nick in the skin adjacent to the guidewire with a #11 scalpel blade (**Figure 59-21*D***). **Direct the sharp edge of the scalpel blade away from the guidewire to prevent nicking the guidewire.**

Place the dilator over the guidewire. Advance the dilator over the guidewire to enlarge the subcutaneous passage for the catheter. Continue to advance the dilator until its hub is against the skin. A twisting motion of the dilator may aid in its advancement through the subcutaneous tissues and into the vein. Withdraw the dilator over the guidewire while leaving the guidewire in place. Advance the catheter over the guidewire until its hub is against the skin (**Figure 59-21*E***). A twisting motion of the catheter may aid in its advancement through the subcutaneous tissues and into the vein. Securely hold the hub of the catheter. Remove the tourniquet from the extremity. Remove the guidewire through the catheter (**Figure 59-21*F***).

Aspirate blood from the catheter with a syringe to confirm intravenous placement. Flush the catheter with sterile saline or begin an infusion. Secure the catheter to the skin with sutures and tape. While this technique seems complicated at first glance, it is easy to learn and can be performed in a few minutes by an experienced Emergency Physician.

MIDLINE CATHETERS

Midline catheters are extended dwell catheters (i.e., up to 30 days) that are longer in length than peripheral IV catheters and often shorter than central venous line catheters. They are available in a variety of sizes and lumens (**Figure 59-22**).[61] These catheters can be used for blood sampling, fluid administration, medication administration, and power injection of computed tomography contrast material. They are mostly inserted using the Seldinger method. The flow rate is printed on the hub of the catheter. These catheters are designed to withstand the pressure injectors.

AFTERCARE

The IV catheter needs to be secured. The use of tissue adhesive at the skin puncture site can prevent the catheter from moving. The tissue adhesive prevents dislodgement, infection, occlusion, and phlebitis.[62] Removal may be problematic. Cover the IV and skin puncture site with a clear dressing.[63] Tegaderm dressings are most often used because of convenience. Special clear dressings with cutouts are available (**Figure 59-23**). These come in a variety of sizes and cost more than a Tegaderm.

FIGURE 59-22. Examples of midline catheters. ***A.*** The Powerwand kit. (Photo courtesy of Access Scientific, San Diego, CA.) ***B.*** Medcomp single and dual lumen. (Photo courtesy of Medical Components Inc., Harleysville, PA.) ***C.*** Nexus CT dual lumen. (Photo courtesy of Health Line Medical Products, Salt Lake City, UT.) ***D.*** Bard Powerglide single lumen. (Photo courtesy of Bard Access Systems, Salt Lake City, UT.)

C

D

FIGURE 59-22. (*Continued*)

The IV Pro (WV IV Pro Inc., Fairmont, WV) can be applied (**Figure 59-24**). This shield was designed to protect the IV site from buildup of bacteria and needle dislodgement. It is a protector and securement device and not a dressing. The clear dome allows site monitoring. Its holes allow airflow and prevent condensation. The device can be used multiple times in the same patient.

Other devices may be added for patient safety. The ivWatch (ivWatch, Williamsburg, VA) monitors the IV site to detect leaks, extravasation, and infiltration (**Figure 59-25**). It is comprised of the patient monitor, a reusable sensor cable, single-use disposable sensor applied next to the IV site, and an AC adapter. The monitor gives an audible and visual alert. The FIVA (FIVAMed Inc., Halifax, Canada) attaches in-line and alerts to the empty IV fluid bag (**Figure 59-26**).[64] The FIVA clips onto the IV line and uses infrared to detect air bubbles in the IV tubing and stop the flow. The FIVA gives an audible and visual alert. It is meant to be used for gravity drips and pressure bags and not pumps. The device is only approved for use in Canada for now. The ClearLine (ClearLine MD, Woburn, MA) prevents air bubbles in the IV tubing from entering the patient (**Figure 59-27**). The device uses US to monitor the IV tubing for air bubbles as small as 25 μm. The bubbles are directed into a disposable bag. It works with gravity drips, pumps, and fluid warmers.

A

B

FIGURE 59-23. Examples of clear coverings for IV lines. ***A.*** IV Clear. (Photo courtesy of Convalon Technologies Ltd, Ontario, Canada.) ***B.*** The Sorbaview shield. (Photo courtesy of Centurion Medical Products, Howell, MI.)

A

B

C

FIGURE 59-24. The IV Pro. ***A.*** Top view. ***B.*** Underneath view. ***C.*** The device on an arm. (Photos courtesy of WV IV PRO Inc.)

A

B

FIGURE 59-25. The ivWatch. ***A.*** The complete device. ***B.*** The sensor applied to an IV site. (Photos courtesy of ivWatch.)

FIGURE 59-26. The FIVA. (Photos used with permission from reference 64.)

COMPLICATIONS

Complications specific to each technique and site are discussed more fully in the following chapters. Venous catheters should be assessed immediately after their placement and reassessed frequently. The assessment must include the skin puncture site, catheter function, the extremity distal to the catheter, and the patient's overall condition. Some of the common problems are noted in **Table 59-2**. Other specific complications of peripheral and central venous access are discussed in the following chapters.

SUMMARY

Venous access is an essential skill for all providers of care to the acutely ill and injured. As with most procedures, success rates increase and complication rates decrease with experience. Successful

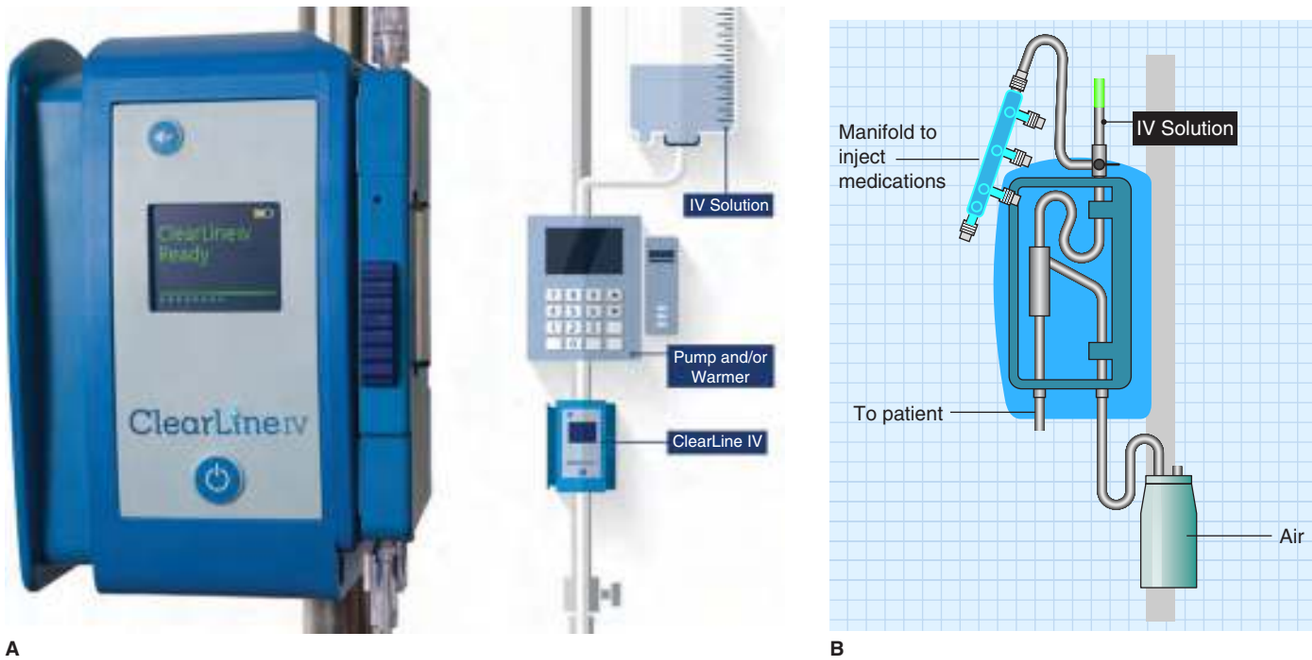


FIGURE 59-27. The ClearLine IV. ***A.*** The complete device. ***B.*** The inside of the device. (Photos courtesy of ClearLine MD.)

TABLE 59-2 Complications of Venous Catheterization

	Observation	Complications	Errors in technique	Response
Skin puncture site	Immediate swelling	Hematoma	Laceration or through-and-through puncture	Remove catheter, apply pressure
Skin puncture site	Delayed swelling	Extravasation or hematoma	Catheter dislodged or damaged, vein lacerated	Remove catheter, apply pressure
Skin puncture site	Erythema or discharge	Infection	Catheter in place too long, catheter or skin contaminated	Remove catheter, give parenteral antibiotics
Catheter	Cannot infuse	Thrombosis or kinking	Catheter not flushed enough, catheter not secured properly	Flush catheter, check position, remove catheter
Catheter	Blood runs up IV tubing	Arterial placement	Arterial puncture not recognized	Remove catheter, apply pressure
Catheter	Cannot aspirate from proximal lumens of multi-lumen line	Extravascular placement or migration of proximal lumens	Not enough catheter inserted; patient movement; catheter not properly secured	Change catheter over a guidewire if distal port is intravascular
Systemic	Fever	Line sepsis	Catheter left in place too long or contaminated	Remove catheter once infection is verified
Systemic	Hemodynamic or respiratory compromise	Pneumothorax, pericardial tamponade	Pleura punctured during insertion; catheter tip malpositioned	Chest radiograph, auscultate chest; pericardial or pleural drainage

venipuncture or cannulation is not the end of the Emergency Physician's obligation to the patient. Frequent reassessment of the venous access site, the equipment, and the patient is essential to prevent complications.

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60 Heel Stick Blood Sampling

Kimberly Fugok and Christopher J. Russo

INTRODUCTION

Obtaining blood samples in newborns or infants can be a challenging task. Venous access in the patients can be difficult or limited. The vessel size and volume status of these patients can prove to be a challenge for the most skilled Emergency Physician.[1] Capillary blood sampling is the most frequently used method to obtain blood samples from infants. This chapter reviews the basic principles and techniques of heel stick blood sampling.

ANATOMY AND PATHOPHYSIOLOGY

The selection site for capillary sampling is usually based on age and weight of the patient. The heel is the most common location for capillary blood sampling because the junction between arterial and venous blood supply is located just below the dermis.[2] The depth of this capillary bed in newborns and infants is consistently between 0.35 and 1.6 mm and significantly increases with weight gain.[3] A thick callus can form as the child ambulates and can interfere with adequate capillary blood collection at this site. Heel blood sampling is recommended in children weighing up to 10 kg. This equates to an approximate age of 6 months. Finger prick can be an alternative modality in patients over 6 months of age and weighing greater than 10 kg. The fingers or ear lobes are recommended in adults for capillary blood sampling. The third or fourth digits of the nondominant hand are the recommended site as they are less likely to have calluses. It is suggested to draw samples from the medial or lateral aspects of the heels and fingers (**Figure 60-1**).[2] These areas avoid the blood vessels, bone, nerves, and tendons.

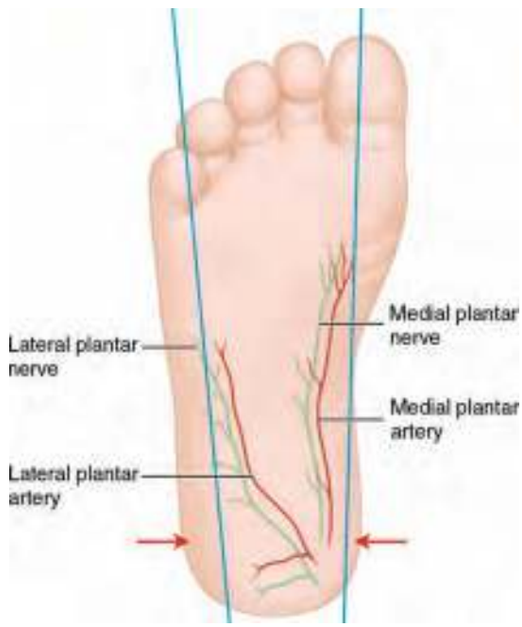


FIGURE 60-1. The topographic anatomy of the sole of an infant's foot. The *arrows* represent the areas devoid of structures to perform the heel stick.

INDICATIONS

This technique is useful for small samples of blood or repeated small-volume blood samples. Commonly drawn samples include hemoglobin, hematocrit, blood glucose, and electrolytes.[2] This modality is ideal for premies, newborns, and infants. Heel sticks are often used up to 12 months of age. This is a source of blood when other sources (e.g., arterial lines, central venous access, and intraosseous access) are not available or appropriate.

CONTRAINDICATIONS

A heel stick is not recommended during poor peripheral perfusion or states of compromised blood flow to an extremity. Blood samples should not be collected through local infections, significant edema, or hematomas. Capillary blood gas collection is not the recommended specimen if a precise PaO_2 is needed.[2] Heel sticks are not the collection method of choice for blood cultures due to the likelihood of bacterial contamination.

EQUIPMENT

- Warm packs
- Latex-free gloves
- Alcohol swab
- Sterile gauze
- Capillary blood collection tubes (e.g., capillary tube microtainers)
- Incision device (**Figure 60-2**)

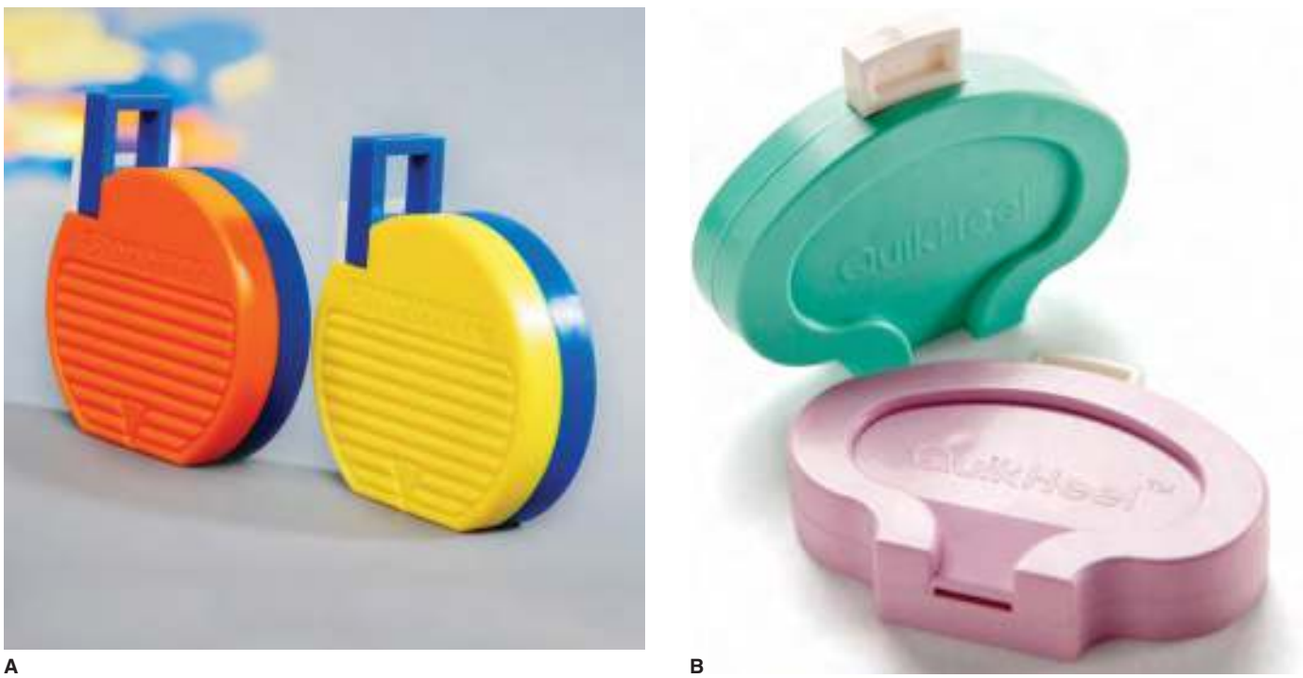
Heel stick incision devices and capillary blood collectors are subject to variability depending on the brand and model.[2] Lengths of incisions from devices vary by manufacturer. The recommended maximum depth is 2.4 mm in the heel. The devices are available in multiple sizes for different-sized patients (e.g., premies, neonates, and infants). **Do not use a lancet.**[4,5] A lancet has been shown to increase bruising, inflammation, the number of punctures to obtain a sufficient blood quantity, and the time required to obtain a sufficient blood quantity.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an oral informed consent for the procedure. It is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Warm the heel prior to the procedure to increase local blood flow. Apply a warm towel or a disposable single-use heating pack for approximately 5 minutes. Clean any dirt and debris from the skin. Cleanse the area with an alcohol swab (i.e., 70% isopropyl alcohol). **Allow the area to dry as the alcohol may artificially raise blood glucose levels.** Immobilize the extremity. This may be done by the Emergency Physician or an assistant to prevent the patient moving after the device is activated making blood collection more challenging. It is recommended that neonates and infants are positioned supine with the leg held in a dependent position and the heel at an 80° to 90° angle to the leg.[2] This position allows gravity to naturally bring blood to the heel. The patient may be held in the arms of a parent or assistant or supine on the gurney with their leg bent at the knee to attain this position. Don gloves after hand washing to prevent contamination of the patient or exposure of the Emergency Physician.

Many physicians use no anesthesia for the heel stick procedure. We know neonates and infants recognize pain from this procedure. Consider the use of heel warming, mechanical vibration, swaddling, or sweet solutions.[6-10] All these have been shown to reduce the pain associated with heel sticks. Sweet solutions are shown to induce endogenous opioid mechanisms. Sweet solutions reduce crying time and pain. There are no untoward effects of the above techniques to provide analgesia.



A B

FIGURE 60-2. Examples of some heel stick devices. ***A.*** The Steriheel Incision Device. (Photo courtesy of SteriLance Medical.) ***B.*** The Quickheel Lancet. (Photo courtesy of Becton Dickinson.) ***C.*** The Tenderfoot Lancet. (Photo courtesy of Accriva Diagnostics.) ***D.*** The Baby Lance. (Photo courtesy of Clinical Innovations.)

FIGURE 60-2. (*Continued*)

TECHNIQUE

Remove the device from its protective wrapping. Keep the area of the blade sterile. Place the device perpendicular to the skin at the medial or lateral portion of the heel (**Figures 60-1 and 60-3**). **Do not pinch the heel skin, stretch the heel skin, or stretch the skin on the back of the leg to tighten the heel skin.** These maneuvers force blood from the heel, make obtaining the sample difficult, and cause the skin puncture to be too deep. Activate the device to puncture the skin. **Avoid excessive force on the device to prevent it from puncturing too deep.**[2,11] **Avoid squeezing or "milking" the heel.** These maneuvers may inhibit

FIGURE 60-3. The heel stick. ***A.*** Placement of the device. ***B.*** The result of a heel stick. (Photos courtesy of Accriva Diagnostics.)

capillary filling, decrease the blood flow, activate clotting, and cause hemolysis.[2]

Wipe away the first drop of blood with the sterile gauze. Collect the blood sample by filling the capillary blood collector to the demarcated line. **Obtaining an inadequate (i.e., too little) blood volume may produce inaccurate results.** Apply pressure to the puncture site with sterile gauze for 2 to 3 minutes to stop any bleeding. Cover the puncture site with a light adhesive dressing.

COMPLICATIONS

Capillary blood sampling has a low rate of complications. The following have been reported: hematoma, subcutaneous bruising, soft tissue infection, calcaneal puncture with osteomyelitis of the heel, scarring at the incision site, calcified nodules, nerve damage if fingers of neonates are punctured, collapse of the veins if an adjacent artery is lacerated, localized necrosis, and skin break down from repeated dressings after procedures.[2,12-15] The choice of a proper incision device will limit uncontrolled bleeding and prevent lacerations.

There are discussions regarding the procedural pain of heel stick blood collection compared to venipuncture performed by a skilled phlebotomist. Recent studies have suggested that the heel stick procedure in newborns may cause significant discomfort, which should be taken into account.[16]

SUMMARY

Heel stick blood sampling is a simple, safe, and efficient way to collect blood in infants where standard blood draws may be challenging. The quickness of this procedure can obtain blood sooner than placing a central line, intraosseous line, or intravenous line.

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61 Venipuncture and Peripheral Intravenous Access

Daniel Belmont

INTRODUCTION

Puncture of a peripheral vein is the most common invasive procedure performed in the Emergency Department. While some newer point-of-care testing techniques require only capillary blood, the vast majority of laboratory studies require venous blood. Cannulation of a peripheral vein is performed on a daily basis and is the cornerstone of circulatory resuscitation. It is an essential skill for all emergency personnel. A variety of approaches for obtaining peripheral venous access are described in this chapter.

ANATOMY AND PATHOPHYSIOLOGY

Veins and arteries are composed of a three-layered wall of internal endothelium, muscle, and a layer of connective tissue (**Figure 61-1**).[1] The muscular layer of a vein is much thinner and weaker than an artery. Veins can dilate and constrict in response to the pressure within them. Veins with high internal pressures become engorged and are easier to access. The use of venous tourniquets, dependent positioning, "pumping" via muscle contraction, and the local application of heat or nitroglycerin ointment all contribute to venous engorgement (Chapter 59).[2] These maneuvers can be used to aid in the identification of a peripheral vein.

The connective tissue surrounding veins can be a help or a hindrance during attempts at gaining peripheral venous access. Deficient connective tissue permits the vein to "roll" from side to side and evade the needle. Tough connective tissue can impede the entry of a flexible catheter through the soft tissues and into the vein. Connective tissue serves to stabilize the vein and prevent its collapse.

Venous valves are an important aspect of peripheral venous anatomy (**Figure 61-2**).[1] Venous valves encourage unidirectional flow of blood back to the heart. Gravitational forces prevent blood from pooling in the dependent portions of the extremities. Valves can impede the passage of a catheter through and into a vein. **Forcing a catheter past venous valves may damage them and contribute to later venous insufficiency.** Valves are more numerous at the points where tributaries join larger veins and in the lower extremities. Valves are almost totally absent within the large central veins and the veins of the head and neck.

Veins can be subdivided into central veins and peripheral veins. The important central veins with regard to venous access are the internal jugular, subclavian, and femoral veins. Central veins are usually larger than peripheral veins and have fewer tributaries.

Superficial peripheral veins are generally visible beneath the surface of the skin of the extremities and neck. They are often tortuous and continually merge and divide. Peripheral veins are easiest

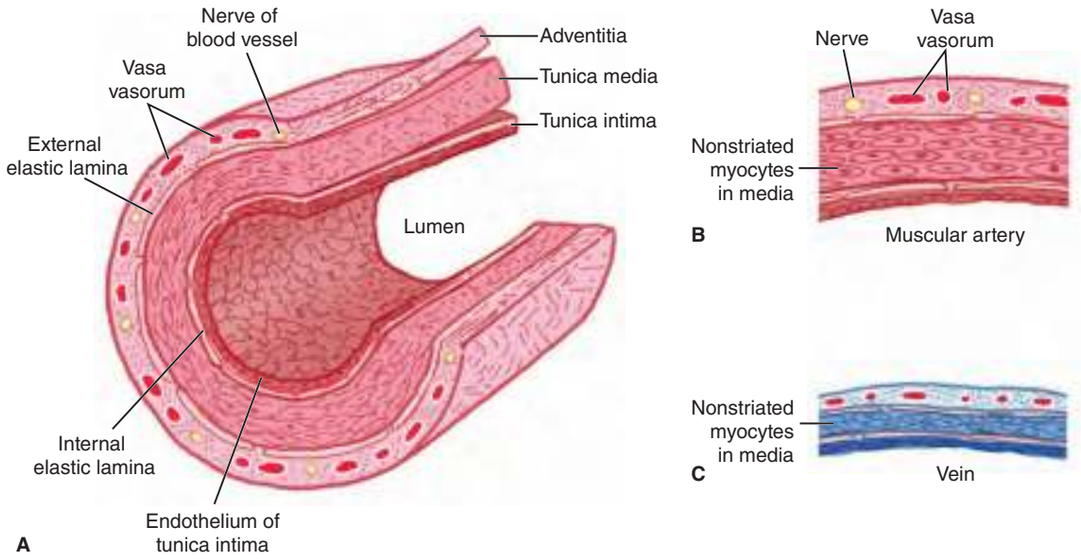

FIGURE 61-1. Comparative anatomy of an artery and a vein. ***A.*** The generic blood vessel. ***B.*** A muscular artery. ***C.*** A vein. Note the vein's thinner wall with fewer myocytes and elastic fibers. This is indicative of the lower pressure within veins compared to arteries.

to access at the apex of the "Y" formed when two tributaries merge into a larger vein, where the vein is straight and free of branches for 2 cm, or more proximal to the site of puncture (**Figure 61-3**). These sites tend to be anchored and hence "roll" less than other sites. The superficial veins of the upper extremity are preferred to those of the lower extremity for peripheral venous access. Indwelling catheters in the upper extremity interfere less with patient mobility, and they pose a lower risk of phlebitis.[2] The superficial veins of the extremities are shown in **Figures 61-4 and 61-5**. The veins most commonly used for venipuncture and venous access are the basilic and cephalic veins as well as their branches and tributaries (**Figure 61-4**). The veins of the dorsal foot and the distal saphenous veins are the most commonly used veins in the lower extremity (**Figure 61-5**).

The depth of the vein beneath the epidermis will affect the ease with which it may be accessed. Very superficial veins are often small, fragile, and easily passed "through-and-through" with a needle resulting in a hematoma. The deeper veins are often not visible and must be located by surface landmarks, palpation, and ultrasound (US). The angle of insertion of the needle must be varied depending on the depth of the vein being punctured (**Figure 61-6**). A shallow angle of approximately 30° to 45° should be used for small and superficial veins (**Figure 61-6*A***). A more obtuse angle of 45° to 60° should be used to access deeper veins (**Figure 61-6*B***). This angle allows the vein to be penetrated within a reasonable horizontal distance from the skin puncture site. Very small and very superficial veins should be entered at a very acute angle of approximately 15° to 30° (**Figure 61-6*C***).

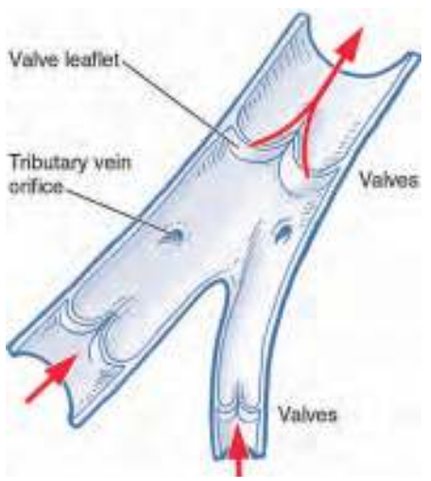

FIGURE 61-2. Venous valves. Cross section of converging veins demonstrating the valve leaflets that permit only forward flow, proximally, toward the right heart. The *arrows* represent the directional flow of blood.

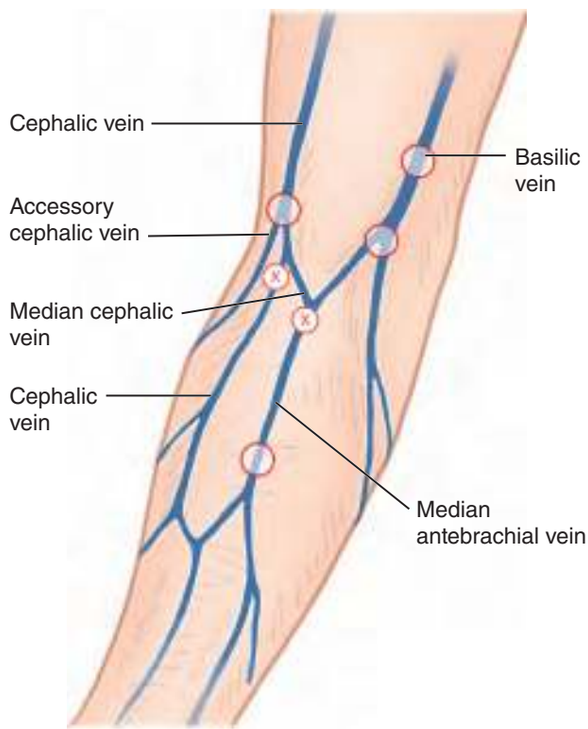

FIGURE 61-3. Preferred vein entry points. Preferred sites (*open circles*) are at the apex of converging veins or in the middle of a long straight vein. Sites just distal to branching or convergence of veins (red ⊗) are best avoided due to the presence of valves and the difficulty in threading a cannula.

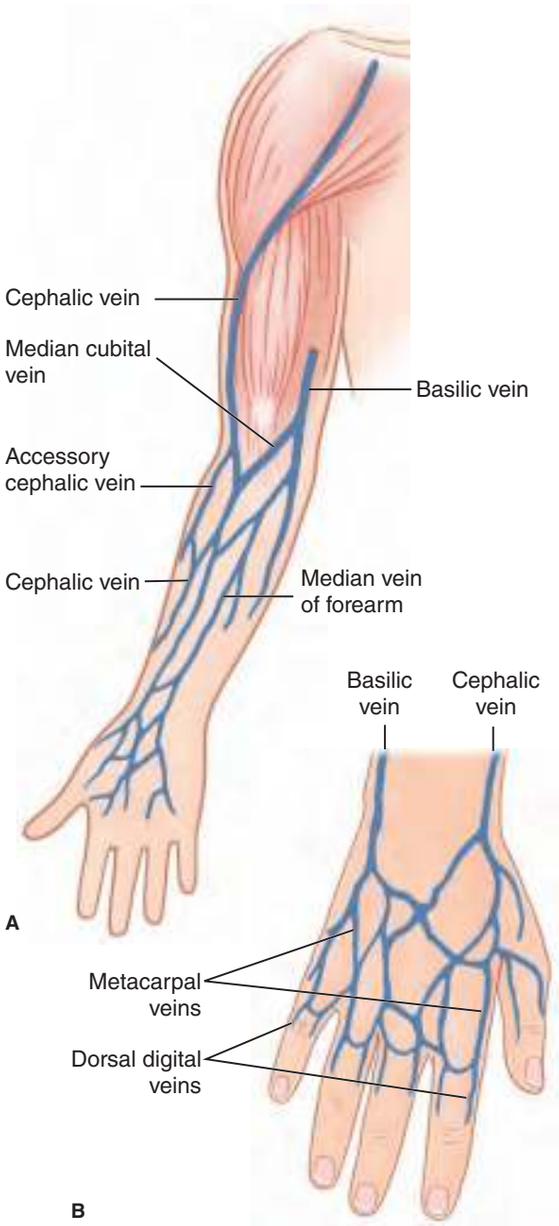

FIGURE 61-4. Superficial veins of the upper extremity. ***A.*** Volar surface of the upper extremity. ***B.*** Dorsal surface of the hand and wrist.

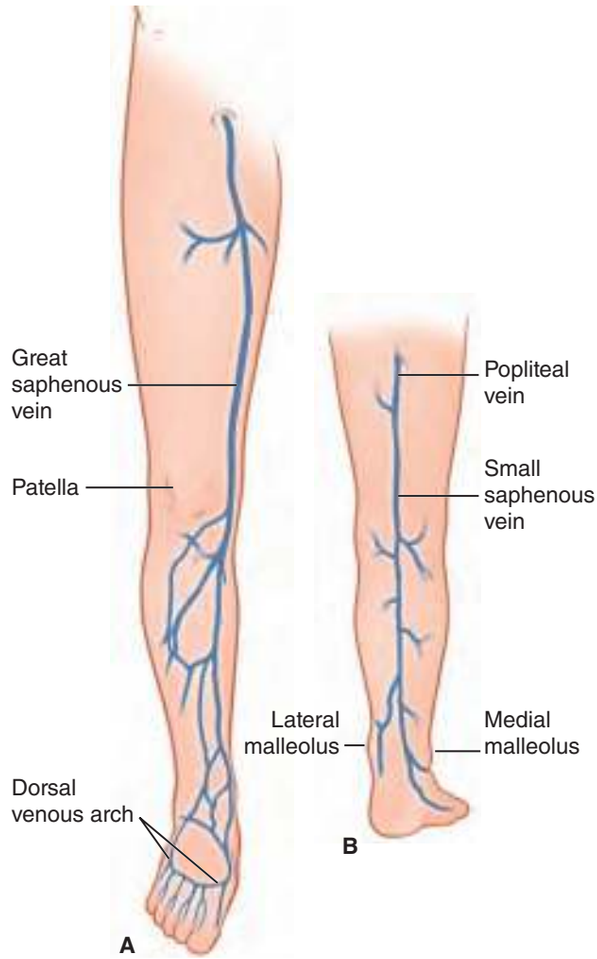

FIGURE 61-5. Superficial veins of the lower extremity. ***A.*** Anterior surface. ***B.*** Posterior surface.

The upper extremity is preferred to the lower for venous cannulation, and distal placement should be attempted before moving proximally.[3] **Avoid veins overlying a joint if possible.** Adherence to these simple principles will allow the patient maximum mobility and increase the chance of successfully cannulating and maintaining access in the vein. **Any solutions or medications infused distally can extravasate and injure the surrounding tissues once a proximal vein has been punctured unsuccessfully.**

It is easiest to insert a venous cannula where two tributaries merge and form a "Y." Choose a straight portion of vein without branches to minimize the chance of hitting valves within the vein. This also makes it easier to thread the catheter (**Figure 61-3**).

The deep brachial veins are variably located alongside the brachial artery, running lateral and/or medial to the artery (**Figure 61-7**).[4,5] The brachial veins are relatively small, deep, not visible, have a close relationship to the brachial artery, and are thus not normally accessed. **It is important to prevent injury to the brachial artery when cannulating or puncturing the brachial veins.** The brachial artery is the sole arterial supply of the forearm, hand, and median nerve. The deep brachial veins may be used when superficial veins have been destroyed by scarring due to intravenous drug abuse, chemotherapy, or prior infusions. The location and cannulation of the brachial veins can be aided through the use of US.[6]

The neck is an important potential site for peripheral venous access through the external jugular vein (**Figure 61-8**). This vein begins at the level of the mandible and runs obliquely across the sternocleidomastoid muscle. It dives beneath the fascia in the subclavian triangle in the neck to join with the subclavian vein.[5] Some patients have two external jugular veins on one or both sides as an anatomic variant. The external jugular vein has two sets of valves (**Figure 61-8**). One is located where the external jugular vein joins the subclavian vein, and the other is located approximately 4 cm above the clavicle. These valves are not fully competent but may prevent the passage of a guidewire or catheter.[5,7]

INDICATIONS

Venous puncture (i.e., venipuncture) with a needle is indicated only for the sampling of venous blood. Medications may be administered as a one-time dose via this technique. The risk of medication extravasation with this technique is high and has fallen out of favor.

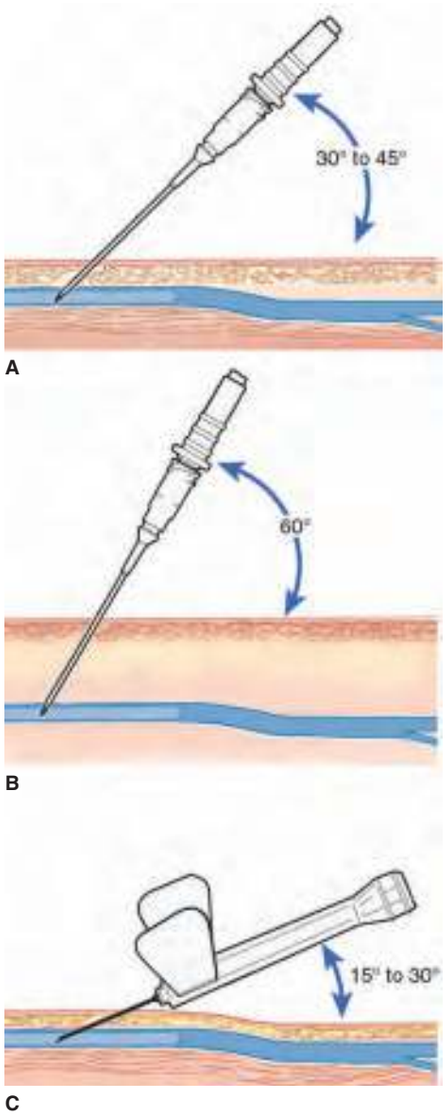

FIGURE 61-6. The angle between the needle and the skin must be varied based on the depth and diameter of the target vein. ***A.*** A shallow angle must be used for small and superficial veins. ***B.*** A steeper angle must be used for deeper veins. ***C.*** A butterfly-type needle permits the shallowest angle of entry for very small and superficial veins.

Venous cannulation is indicated for sampling of venous blood. It is also performed for the administration of intravenous medications, fluid solutions, blood products, and partial nutritional support. Full nutritional support requires central venous access. The specific indications for peripheral venous access, central venous access, and the various techniques of venous cannulation are discussed below and in Chapters 60 through 64.

CONTRAINDICATIONS

Sclerosing solutions, vasopressors, concentrated solutions of electrolytes or glucose, and chemotherapeutic agents are more safely infused into a central vein. Avoid peripheral venous access in an injured extremity to not interfere with care of the injury and venous drainage of the limb. Do not perform intravenous cannulation and venipuncture through infected or burned skin if possible.

Do not use a vein proximal to a running venous infusion for venous blood sampling. The blood sample will be tainted or diluted by the infused solution. The hole in the vein may allow blood, infused solutions, and medications to extravasate into the surrounding tissues.

Avoid venipuncture and venous cannulation in an extremity with an arteriovenous fistula. Do not puncture veins in the upper extremity that may be needed for arteriovenous fistula construction for hemodialysis in the near future unless absolutely necessary. Scarring of the vein may complicate later surgical procedures.

EQUIPMENT

- Alcohol swabs
- Povidone iodine or chlorhexidine solution
- Local anesthetic solution
- 25 gauge needle
- 1 mL syringe
- Gloves
- Immobilization supplies, if necessary
- 1 inch tape, waterproof or plastic
- Transparent dressing
- Gauze 4×4 squares
- Tourniquet
- Desired venous access device
- Saline lock or infusion set
- Intravenous fluids
- Vacuum blood collection tubes
- Medicine cup
- Scissors
- Gauze roll or stockinette

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. Obtain verbal consent for the venipuncture unless it is an emergency. Select a site for the venipuncture. A site in the upper extremity is preferred. While a satisfactory vein is usually evident upon inspection, the placement of a venous tourniquet will aid the process greatly. It is easiest to place the tourniquet a few inches proximal to the elbow when first trying to locate a vein in the upper extremity. This restricts venous return from the entire extremity distal to the tourniquet and allows rapid inspection of the entire limb. Release the tourniquet once the site is chosen.

The use of dependent positioning, "pumping" via muscle contraction, and the local application of heat or nitroglycerin ointment will all contribute to venous engorgement if difficulty is encountered in finding a vein (Chapter 59).[2] These maneuvers can be used to aid in the performance of a venipuncture or peripheral venous access. A device specifically designed to identify veins may also be used (Chapter 59).

Clean the puncture site of any dirt and debris. Apply an alcohol swab, povidone iodine solution, or chlorhexidine solution to the area overlying the identified vein and allow it to dry.[8] Wipe off the povidone iodine with an alcohol swab after it dries to lessen the chance of a local skin reaction. Infiltrate a small amount of local anesthetic solution subcutaneously with a 25 gauge needle over the puncture site. **Take care not to puncture the target vein**

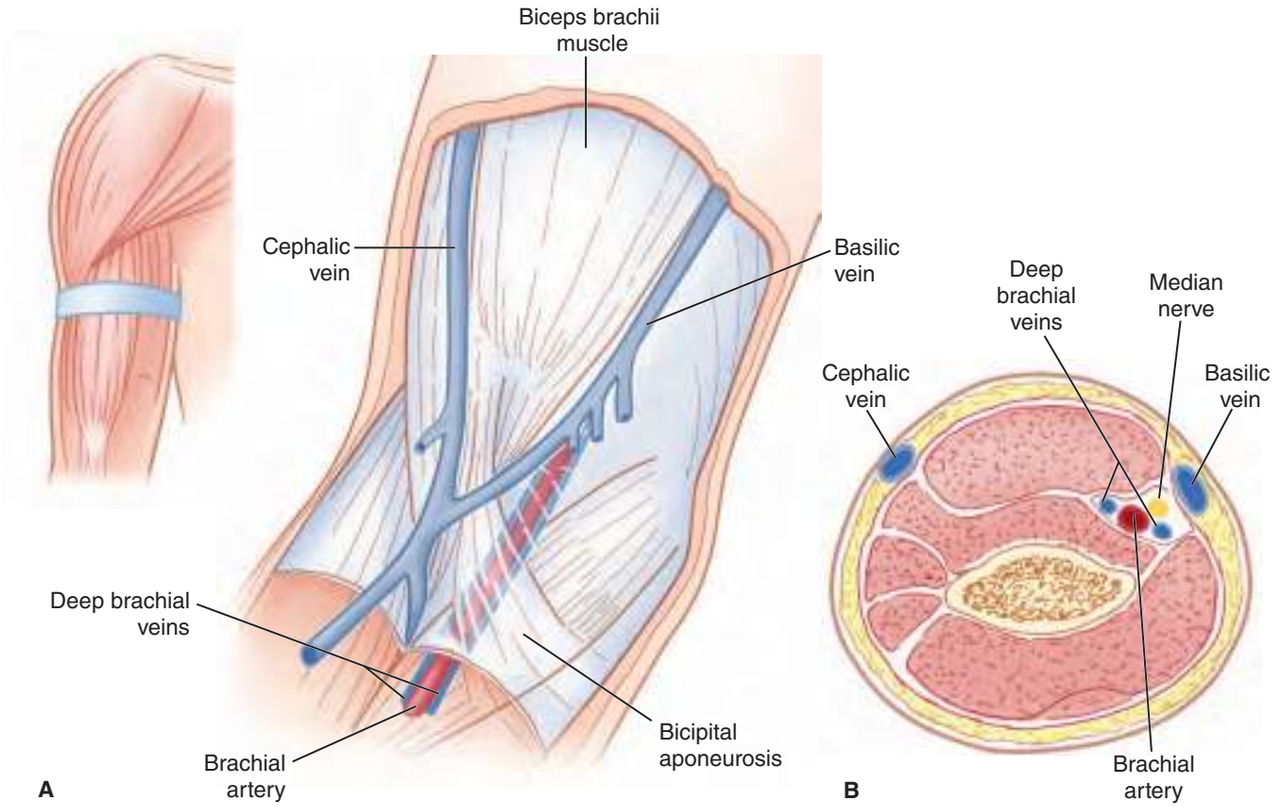

FIGURE 61-7. The deep brachial veins. ***A.*** The deep brachial veins are located deep to the biceps tendon and muscle, and adjacent to the brachial artery. ***B.*** Cross section of the arm 2 cm above the elbow. Note the two deep brachial veins, one on each side of the brachial artery.

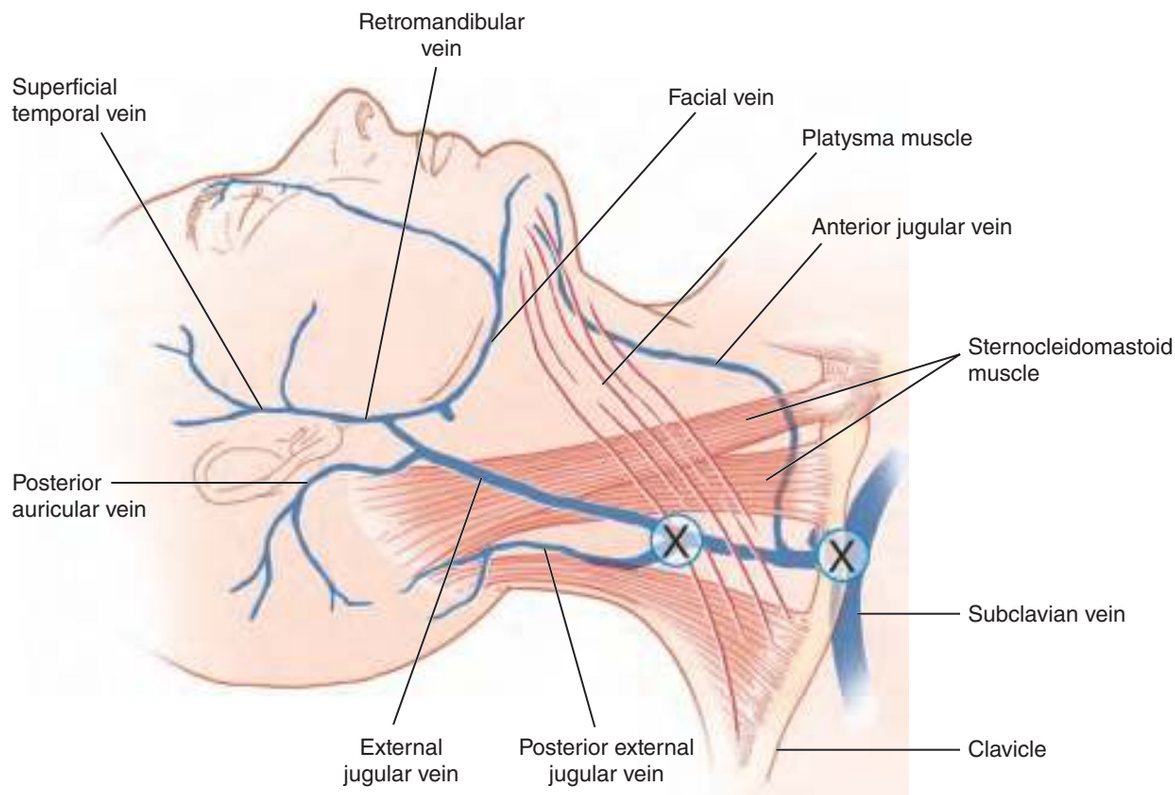

FIGURE 61-8. The external jugular veins. The anterior external jugular vein is usually larger than the posterior and runs deep to the platysma muscle. Note its relationship to the internal jugular vein. Valves (noted by the ⊗) are normally present in the external jugular vein where it enters the subclavian vein and approximately 4 cm superior to the clavicle.

accidentally. Other methods of anesthesia are available and do not cause pain (Chapter 154). Reapply the tourniquet.

TECHNIQUES

PERIPHERAL VENIPUNCTURE

Stabilize the vein with the nondominant hand (**Figure 61-9**). Insert the needle attached to a syringe or vacuum tube adapter with the bevel upward and into the vein at a 30° to 45° angle (**Figure 61-6*A***).[9] Lower angles, at times nearly parallel to the skin, may be needed to enter very narrow or superficial veins. This is easiest to achieve with a butterfly-type needle (**Figure 61-6*C***). Apply negative pressure to the syringe. A flashback of blood in the hub of the needle indicates that the tip of the needle is within the vein. Pulsatile blood that pushes back the syringe plunger indicates an arterial puncture. **Collect the necessary samples before removing the intraarterial needle unless venous blood is specifically needed for a test.** No additional harm will be done by withdrawing a blood sample from an artery that has already been punctured.

Slowly advance the needle until it is deeper than the judged depth of the vein if no blood is obtained. Apply negative pressure to the syringe and slowly withdraw the needle. The vein will often have been punctured through-and-through (**Figure 61-10*B***), and the specimen will be obtained as the needle is withdrawn (**Figure 61-10*C***). Redirect the needle and make another attempt at puncturing the vein if no blood is obtained by the time the needle is withdrawn to just beneath the skin. **Never sweep the point of the needle around without withdrawing it as the sharp bevel of the needle can lacerate nearby structures.** Remove the tourniquet and apply direct pressure for several minutes if swelling develops indicating a hematoma formation. Search for another venous access site. Accidental peripheral arterial punctures should have direct pressure applied for at least 5 minutes.

The Touch Activated Phlebotomy or TAP device (Seventh Sense Biosystems, Medford, MA) was developed for venipuncture (**Figure 59-17**). The base of the device uses adhesive to stick and form an airtight seal on the skin of the upper arm. Pushing the button on the top of the device activates 30 thin needles that penetrate the skin. This micropuncture causes no pain, takes 2 minutes, and obtains 100 µL of blood. Blood is collected in a reservoir with anticoagulant. The device has an indicator for when the blood collection is complete. The laboratory can extract the sample from the TAP device and analyze the blood using regular methods.

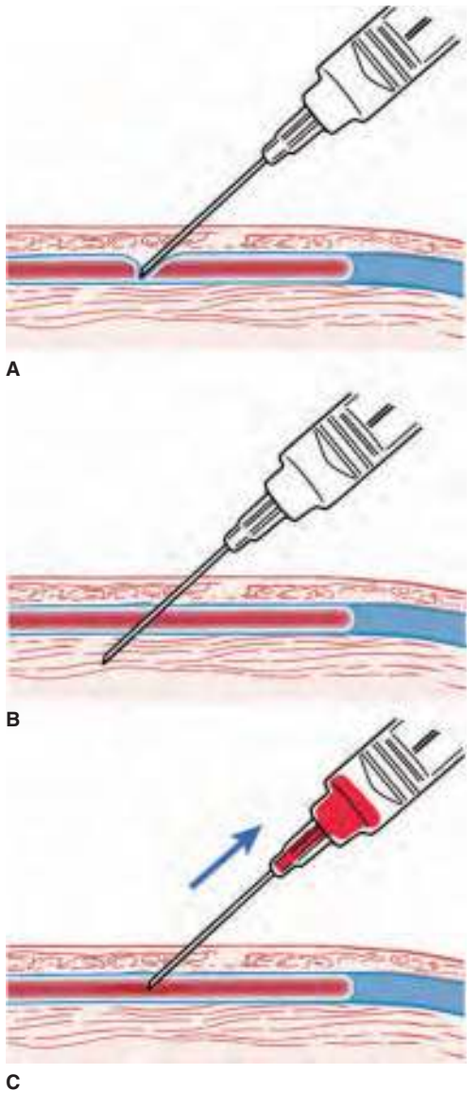

FIGURE 61-10. Through-and-through puncture of the vein. ***A.*** The tip of the hypodermic needle can collapse the vein and prevents a flashback of blood in the syringe. ***B.*** The vein may be punctured through-and-through. ***C.*** Slow withdrawal of the needle permits the vein to open and blood to return into the syringe.

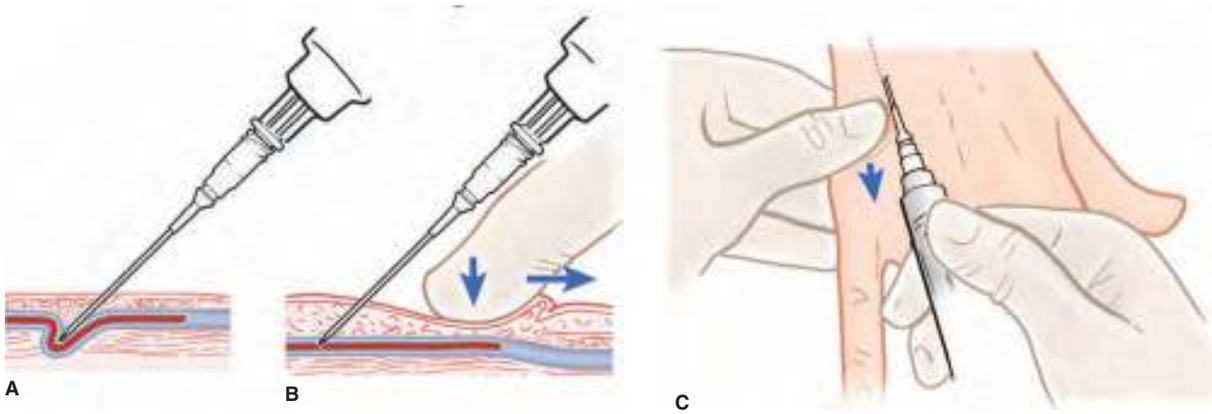

FIGURE 61-9. Stabilization of the vein during venipuncture. ***A.*** The vein may kink or roll away from the tip of the hypodermic needle without stabilization. ***B.*** Gentle stabilizing pressure applied with the fingertips allows entry into the vein. ***C.*** The nondominant thumb is used to stabilize the skin.

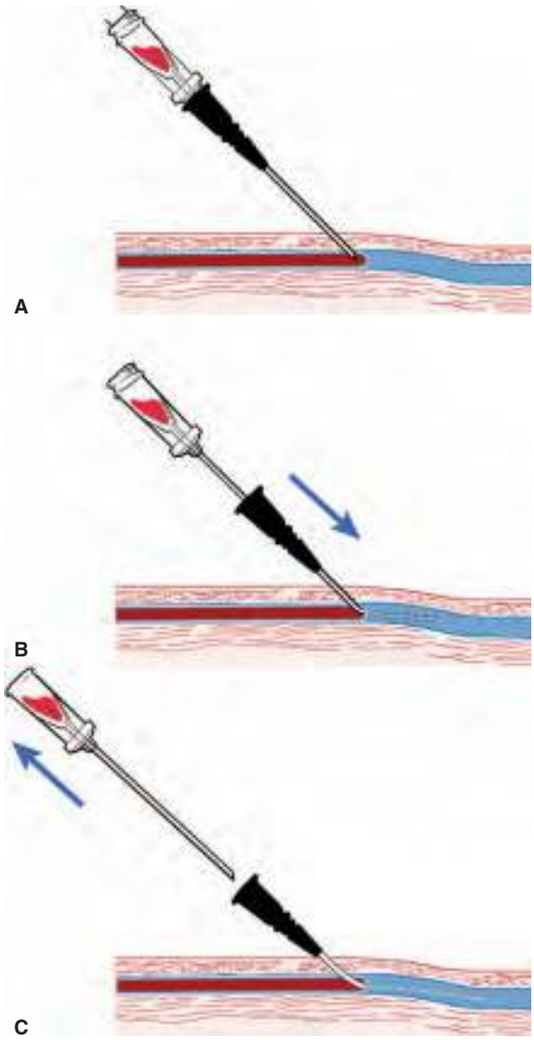

FIGURE 61-11. The catheter-over-the-needle technique. ***A.*** The vein is punctured and blood returns in the needle hub. ***B.*** The catheter is advanced over the needle and into the vein. ***C.*** The needle is removed.

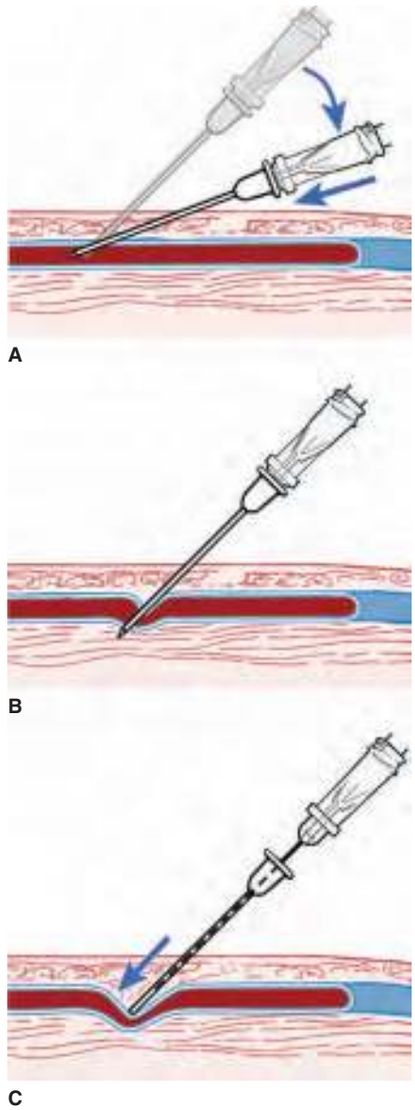

FIGURE 61-12. Advancing the catheter-over-the-needle. ***A.*** Drop the catheter hub toward the skin and then advance the catheter-over-the-needle 2 to 3 mm into the vein. ***B.*** The far wall of the vein may be punctured if the needle is advanced at the original angle to the skin. ***C.*** The catheter may push the vein off the end of the needle if the catheter is advanced over the needle as soon as the vein is entered.

PERIPHERAL INTRAVENOUS CANNULATION

Cannulation of a vein begins with a successful venipuncture with the desired device, as described above. This section focuses on the use of the catheter-over-the-needle technique of peripheral venous access. This is the most commonly used method for venous cannulation.

Insert the catheter-over-the-needle bevel upward through the skin and into the vein (**Figure 61-11*A***).[9] A flash of blood in the hub of the needle confirms that the tip of the needle is within the vein. Advance the catheter-over-the-needle an additional 2 to 3 mm to ensure that the catheter is within the vein. An alternative is to drop the hub of the needle nearly parallel to the skin before advancing the catheter-over-the-needle (**Figure 61-12*A***). This will prevent the needle from puncturing the far wall of the vein (**Figure 61-12*B***) and the catheter from pushing the vein away from the needle (**Figure 61-12*C***). Hold the hub of the needle securely. Advance the catheter over the needle until its hub is against the skin (**Figure 61-11*B***). Securely hold the hub of the catheter against the skin. Withdraw the needle (**Figure 61-11*C***). Apply pressure with the nondominant index finger over the skin above the catheter to prevent blood from exiting the catheter (**Figure 61-13*A***).

Apply a device or intravenous tubing to the hub of the catheter while applying digital pressure over the catheter. A syringe or vacuum device may be attached to the catheter to draw blood samples (**Figure 61-13*B***). Intravenous tubing can be attached to the catheter to begin a fluid infusion (**Figure 61-13*C***). A saline lock may be attached to the catheter to be used later for intravenous access (**Figure 61-13*D***). Remove the tourniquet.

Secure the intravenous catheter to the skin (**Figure 61-14**). There are numerous methods to tape the catheter to the skin, but only two are described here. Place a 2 inch piece of adhesive tape sticky side up under the catheter hub (**Figure 61-14*A***). Fold the ends of the adhesive tape over the catheter and onto the skin to form a "chevron" (**Figure 61-14*B***) or a "U" (**Figure 61-14*C***). Apply a 2 inch piece of adhesive tape over the catheter hub (**Figure 61-14*D***). Place a transparent dressing over the catheter hub and distal intravenous tubing (**Figure 61-14*E***). Some prefer to use the transparent dressing without the adhesive tape. The catheter may be sutured to the

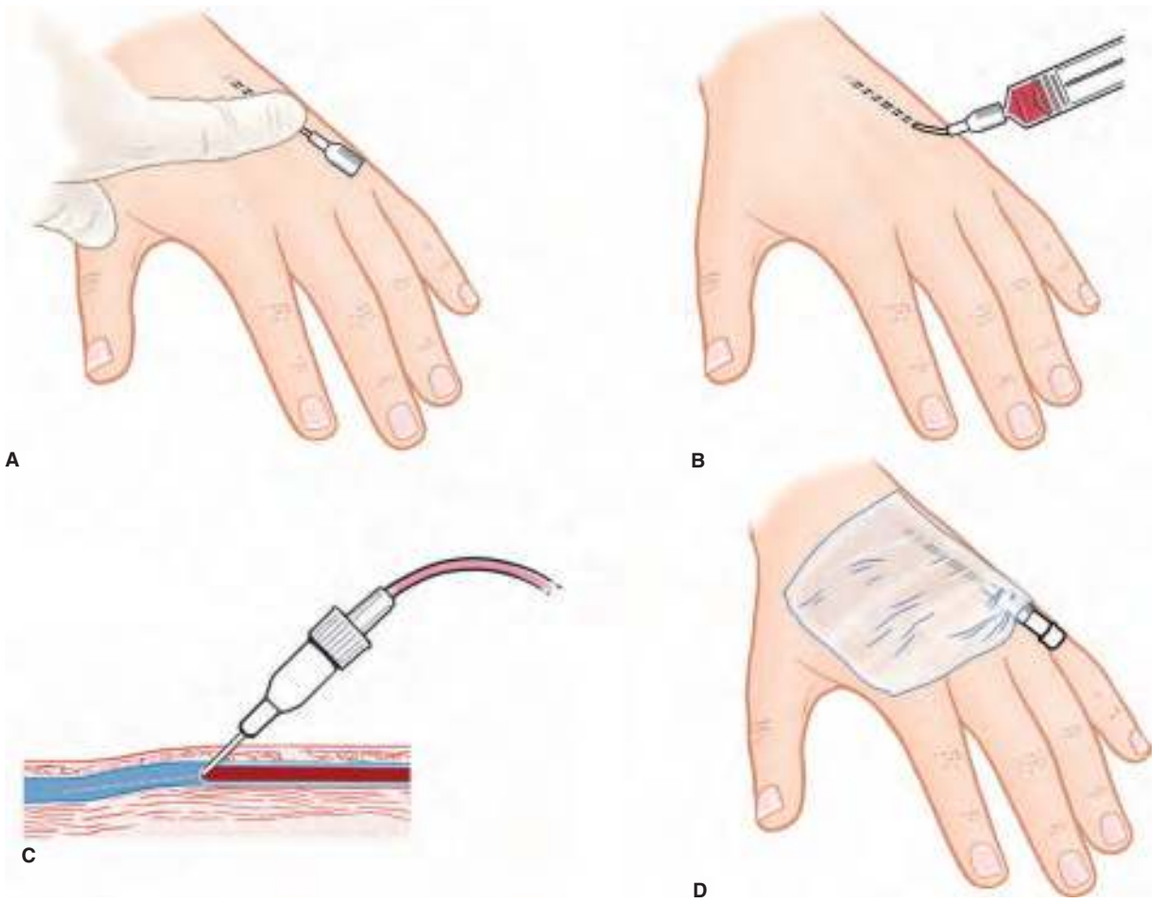

FIGURE 61-13. Peripheral intravenous cannulation. ***A.*** Apply gentle pressure over the catheter with a gloved finger to prevent hemorrhage from the catheter hub. ***B.*** Blood samples may be withdrawn from the cannula via syringe or vacuum tubes. ***C.*** Intravenous infusion tubing is attached to the catheter. ***D.*** A saline lock is attached to the catheter.

skin when vascular access is essential and when the catheter may be pulled out by a young child or combative patient. It is very rare that a peripheral intravenous catheter must be sewn into place.

ALTERNATIVE TECHNIQUES

ARTERIAL LINE KIT

The modified Seldinger technique with a QuickFlash radial artery catheterization set (Arrow International, Bloomington, IN) is very useful for the catheterization of deep brachial and external jugular veins. The unit is commonly available in Emergency Departments and Intensive Care Units. It consists of a one-piece unit that incorporates a catheter-over-the-needle, a guidewire in a feed tube, and a lever to advance the guidewire (**Figure 61-15**). The black mark on the feed tube is a reference mark. The tip of the guidewire is positioned at the tip of the needle when the advancement lever is at the reference mark. The unit is also available without the catheter-over-the-needle as the Positive Placement Spring-Wire Guide (Arrow International, Bloomington, IN). It can be attached to a standard stock catheter-over-the-needle. A similar device is The WAND (Access Scientific, San Diego, CA).

The integral guidewire and soft, 2 inch long, 20 gauge catheter found in the QuickFlash set can ease the process of catheterization considerably. The depth of the deep brachial vein combined with overlying skin that is often scarred from previous venipunctures makes catheterization with the usual 1¼ inch catheter difficult. The external jugular vein is quite mobile, and the overlying tissues are fairly tough. This can make it quite difficult to thread an over-the-needle-catheter into the vein without pushing the vein off the end of the needle.

Select a vein to cannulate. Clean and prep the skin overlying the puncture site. Place a tourniquet on the extremity. Open the package and remove the unit. Advance the guidewire through the needle and then retract it. Do not use the catheterization unit if the guidewire does not advance and retract smoothly. Ensure that the guidewire advancement lever is retracted as far as possible so that the guidewire is not within the needle. **The flashback of blood will not be seen if the guidewire is not fully retracted and out of the needle.**

Stabilize the vein with the nondominant hand. Insert the catheter-over-the-needle with the bevel upward through the skin and into the vein (**Figure 61-16*A***).[9] A flash of blood in the hub of the needle indicates that the tip of the needle is within the vein. Hold the needle hub securely. Advance the guidewire, using the advancement lever, into the vein (**Figure 61-16*B***). Continue to advance the guidewire as far as possible into the vein. **The advancement lever must be distal to the reference mark to ensure that the guidewire is past the tip of the needle. Stop advancing the guidewire if resistance is encountered. Do not force the guidewire against resistance. Do not retract the guidewire if resistance to advancement is encountered. Doing so may damage the vein or shear off a piece of the guidewire. Withdraw the entire unit and repeat the procedure with a new unit.**

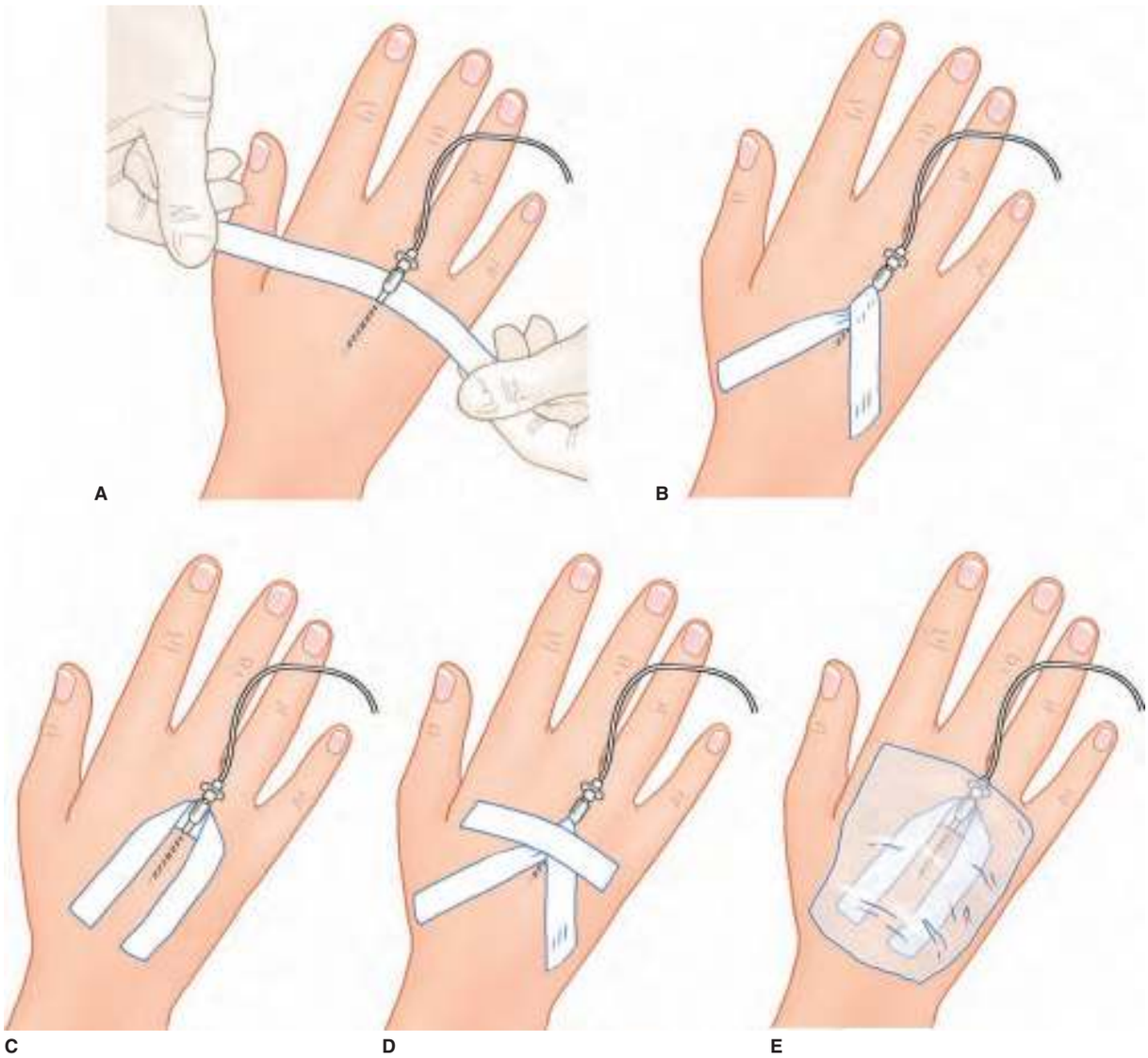

FIGURE 61-14. Securing the intravenous catheter. ***A.*** Place a narrow strip of adhesive tape sticky side up under the catheter hub. ***B.*** Fold the tape over the catheter to form a "chevron." ***C.*** Alternatively, the tape with the sticky side up can be folded to form a "U." ***D.*** A second piece of tape is applied to better secure the catheter to the skin. ***E.*** A transparent dressing is applied over the catheter.

Advance the guidewire as far as possible into the vein. Advance the catheter-over-the-needle an additional 1 to 2 mm into the vein. This will ensure that the tip of the catheter is within the vein. Hold the hub of the needle securely. Advance the catheter over the needle and guidewire until its hub is against the skin (**Figure 61-16*C***). A twisting motion may help to advance the catheter against resistance. Release the tourniquet. Hold the catheter hub firmly against the skin. Remove the needle and guidewire, through the catheter, as a unit. Attach a syringe, vacuum blood collection system, intravenous line, or saline lock onto the catheter hub. Secure the catheter with adhesive tape.

FIGURE 61-15. The Arrow QuickFlash radial artery catheterization set. Note the different positions of the guidewire. The guidewire is within the feed tube (*top*). The tip of the guidewire is at the tip of the needle when the advancement lever is at the reference mark (*middle*). The guidewire is advanced through the catheter-over-the-needle (*bottom*).

EXTERNAL JUGULAR VEIN CANNULATION

Place the patient in the Trendelenburg position to distend the external jugular vein (**Figure 61-17**). Turn the patient's head to the opposite side. This will gently stretch the vein and prevent it from rolling. Do not overturn the patient's head and compress the external jugular vein. Clean and prep the skin of the neck. Place the nondominant thumb or index finger above the midportion of the clavicle to obstruct outflow and distend the external jugular vein. Align the catheter-over-the-needle parallel to the vein with the bevel of the needle upward and the tip of the needle pointing toward the clavicle (**Figure 61-17**).[9] Enter the vein midway between the angle of the mandible and the mid-clavicle. Insert the catheter-over-the-needle during inspiration when the valves of the external jugular vein are open. **Be sure to cover the open hub of the needle and/or catheter**

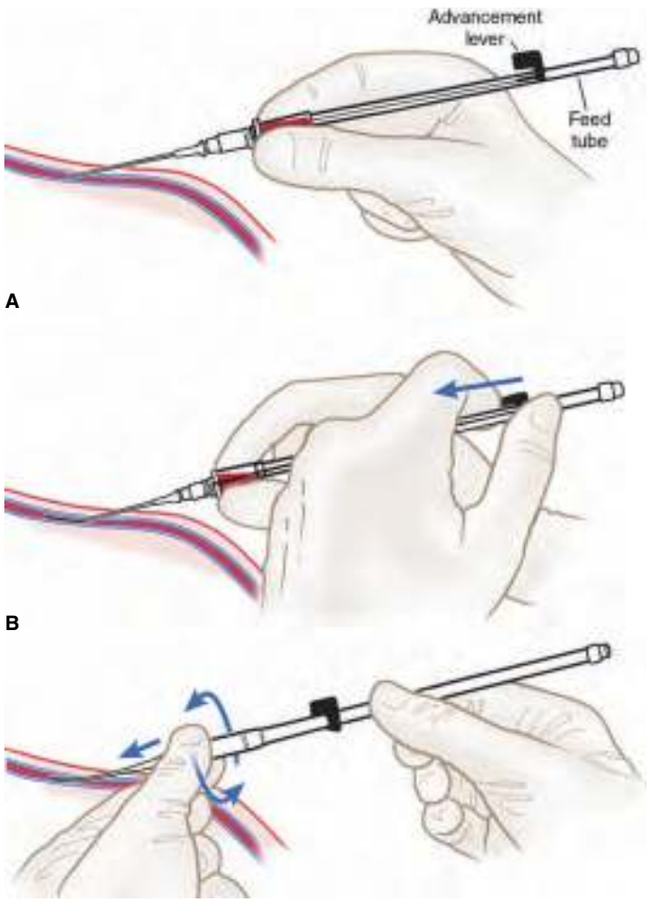

FIGURE 61-16. The Arrow QuickFlash radial artery catheterization set. ***A.*** The vein is punctured and blood returns into the hub of the needle. ***B.*** The guidewire is advanced into the vein. ***C.*** The catheter is advanced over the needle and guidewire with a back-and-forth rotating motion. The needle and guidewire are then removed as a unit.

with a finger at all times to prevent an air embolism. Attempt to insert the catheter-over-the-needle obliquely into the vein if it rolls when trying to access it. Another option is to cannulate the vein in the area where a tributary joins it. These areas are often anchored in the subcutaneous tissue. The remainder of the technique is similar to that described previously.

FIGURE 61-17. External jugular vein cannulation.

DEEP BRACHIAL VEIN CANNULATION

Extend the patient's arm. Identify the brachial artery pulse by palpation or US in the antecubital fossa. Clean and prep the skin of the antecubital fossa. Place a tourniquet on the upper arm. Reidentify the brachial artery pulse. Place a 2 to 3 inch long catheter-over-the-needle onto a 5 mL syringe and insert it bevel upward just medial or lateral to the brachial artery pulse and at a 30° to 45° angle to the skin with the tip of the needle pointing cephalad.[9] Advance the catheter-over-the-needle while applying negative pressure to the syringe. A flash of blood in the syringe indicates that the vein has been entered. Slowly withdraw the catheter-over-the-needle if a flash is not seen. It may have gone "through-and-through." A flash will be seen as the catheter-over-the-needle is withdrawn and the tip of the needle reenters the vein. The remainder of the technique is similar to that described previously. The use of US can significantly improve the success rate of deep brachial vein cannulation (**Figure 61-18**). Complications include brachial artery puncture, clotting of the brachial artery, hand ischemia, hematoma formation, loss of vascular access, and transient paresthesias.[3,10]

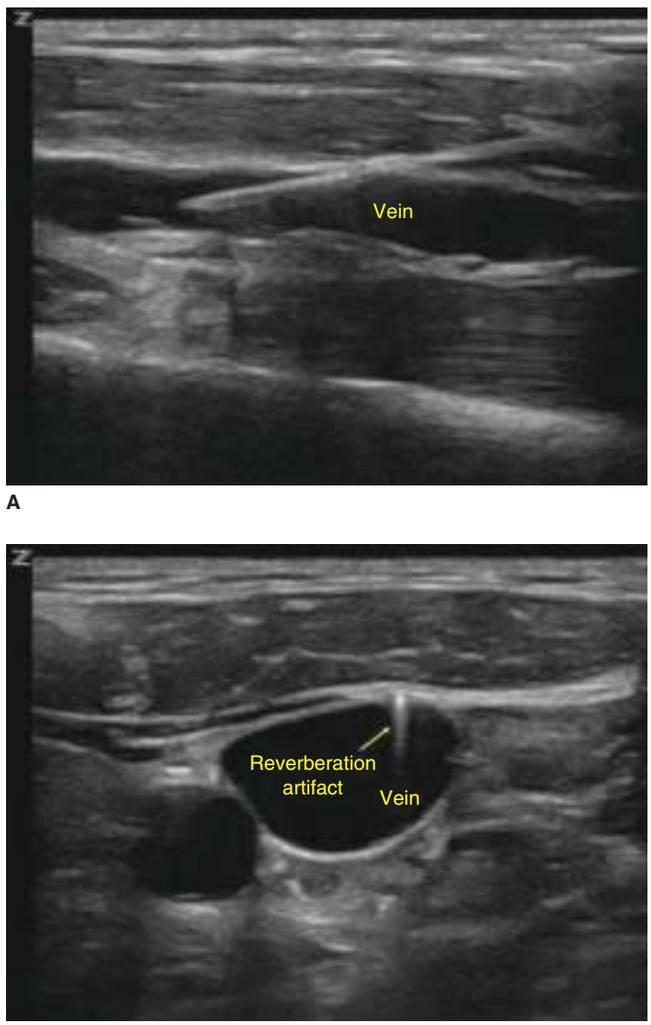

FIGURE 61-18. The use of US to cannulate a vein. ***A.*** Longitudinal view of a vein with a catheter in the vein. ***B.*** Transverse view of a vein with a catheter in the vein. (Photos courtesy of Sriker Adhikari, MD.)

ULTRASOUND-GUIDED PERIPHERAL VEIN CANNULATION

A deep peripheral vein, often the brachial or basilic vein, may be cannulated under direct visualization using ultrasonography (**Figure 61-18**).[3] US may be used to identify a superficial vein when one is not palpable or visible. Place a tourniquet on the upper arm. Identify the vein by placing the US transducer perpendicular to the vein. Identify the vein as a thin-walled, nonpulsatile, vascular structure. Move the US transducer along the vein to identify its most superficial and easiest accessed point. Insert the catheter-over-the-needle under direct visualization.[11,12]

The easiest way to gage the depth of the vein is to note its depth on the US screen. Move the US transducer distally this same distance while still maintaining visualization of the vein. Insert the catheter-over-the-needle at a 45° angle and advance it until the tip of the needle is seen on the US screen. Puncture the vein wall using a quick and short jabbing motion. Take care to not puncture through the posterior wall of the vein. Once the tip of the needle is seen within the vein, advance the catheter-over-the-needle and into the vein. Refer to Chapter 64 for the complete details of US-guided vascular access.

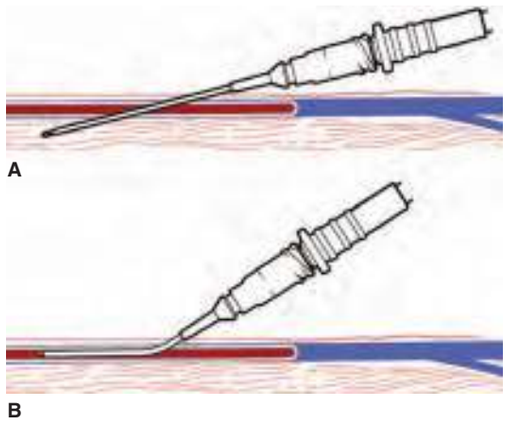

FIGURE 61-20. Cannulation of a small superficial vein with a catheter-over-the-needle. ***A.*** The "shoulder" of the catheter hub prevents the needle from being placed nearly parallel to the skin. ***B.*** A slight bend at the base of the needle permits the needle to run nearly parallel to the skin surface, enabling the subcutaneous vein to be cannulated. Always make sure that the catheter can be advanced over the needle before puncturing the patient's skin.

PEDIATRIC CONSIDERATIONS

Venipuncture and peripheral venous access can be quite a challenge in the infant, neonate, and small child. Proper restraint of the extremity with a board or an assistant will greatly aid the process. Aside from the techniques discussed above, there are a few techniques that can facilitate pediatric vascular access.[13,14] The scalp veins can be used for venous access in newborns, infants, and children up to 1 year of age (**Figure 61-19**). They are most easily cannulated with a small (i.e., 23 or 25 gauge) butterfly needle or catheter. Place a rubber band around the baby's head as a tourniquet (**Figure 61-19**). Other veins commonly used include those of the antecubital fossa, dorsal hand, dorsal foot, external jugular vein, and saphenous vein at the knee or groin.[15] It can be helpful to place a small bend at the hub of the catheter-over-the-needle assembly for small and superficial veins (**Figure 61-20**).

FIGURE 61-19. Scalp vein cannulation in the neonate. A rubber band makes a convenient tourniquet.

This allows for the use of a less acute angle and easier entry into the vein without puncturing the far wall.

The best guide to the gauge (i.e., diameter) of catheter to use is to compare the catheter to the vein. The catheter should be slightly smaller than the vein. The smallest readily available catheters are usually 24 gauge. A 22 gauge catheter will allow a much higher flow rate if it can be inserted successfully.

Keeping a peripheral intravenous line from being pulled out by an active child is quite a challenge. Each institution has its own "recipe" for securing pediatric intravenous lines. The catheter is secured to the skin in the usual manner (**Figure 61-14**). Several strips of tape can be placed to secure the intravenous tubing to the skin and act as "strain reliefs" to prevent traction applied to the intravenous tubing from being transmitted to the catheter. Half of a medicine cup can be used as a shield for the catheter (**Figure 61-21*A***). Apply tape over the cut edges of the cup to prevent the sharp edges from cutting the child's delicate skin. The clear medicine cup acts as a window so the catheterization site can easily be inspected without removing all the dressings. The whole assembly, or just the taped intravenous line, can then be covered with a gauze roll or stockinette (**Figure 61-21*B***). A cup with one-quarter or one-third cut away may be used to protect a scalp vein access site (**Figure 61-21*C***). A splint applied to the extremity will increase the duration of intravenous line patency.[16]

ASSESSMENT

The intravenous line should flush easily. Any infusions should flow by gravity alone. Progressive swelling at the catheterization site indicates the formation of a hematoma or extravasation of infused fluids. Peripheral infusions of vasopressors and caustic solutions require the skin puncture site to be assessed frequently and carefully, since extravasation may lead to extensive local soft tissue necrosis. Pain at the intravenous access site must be taken seriously and should prompt a search for the cause. Pinched skin, extravasation, or thrombosis must be looked for and ruled out.

AFTERCARE

The intravenous (IV) catheter needs to be secured. The use of tissue adhesive at the skin puncture site can prevent the catheter from moving. The tissue adhesive prevents dislodgement, infection, occlusion, and phlebitis.[17] Removal may be problematic. Cover the IV and skin puncture site with a clear dressing.[18] Tegaderm dressings are most often used because of convenience. Special clear dressings with cutouts are available (**Figure 59-23**). These come in a variety of sizes and cost more than a Tegaderm.

Most authorities recommend changing peripheral infusion sites at least every 3 days, although this is probably overly cautious.[19,20] It may be impractical if the patient has poor superficial veins. Any cannula with signs of venous thrombosis, skin erythema, or puncture site discharge must be removed at once. Heparin or saline locks that have not been accessed should be flushed regularly, usually every 8 hours. Saline works as well as heparin solutions for most applications.[21] Approximately 1 to 2 mL of sterile saline to flush is adequate for peripheral venous catheters. Heparin flushed cannulations have an increase in duration of patency.[13]

Dressings should be inspected and changed if they have become moist or contaminated. Routine dressing changes are probably unnecessary.[21] Transparent dressings are widely used despite some studies suggesting that transparent occlusive dressings are associated with higher rates of infection than plain gauze dressings.[10] Transparent dressings have the advantages of allowing easy inspection of the catheter site and holding the catheter securely in place. Individual institutions often have their own nursing guidelines and infection control statistics to support their choice of dressing.

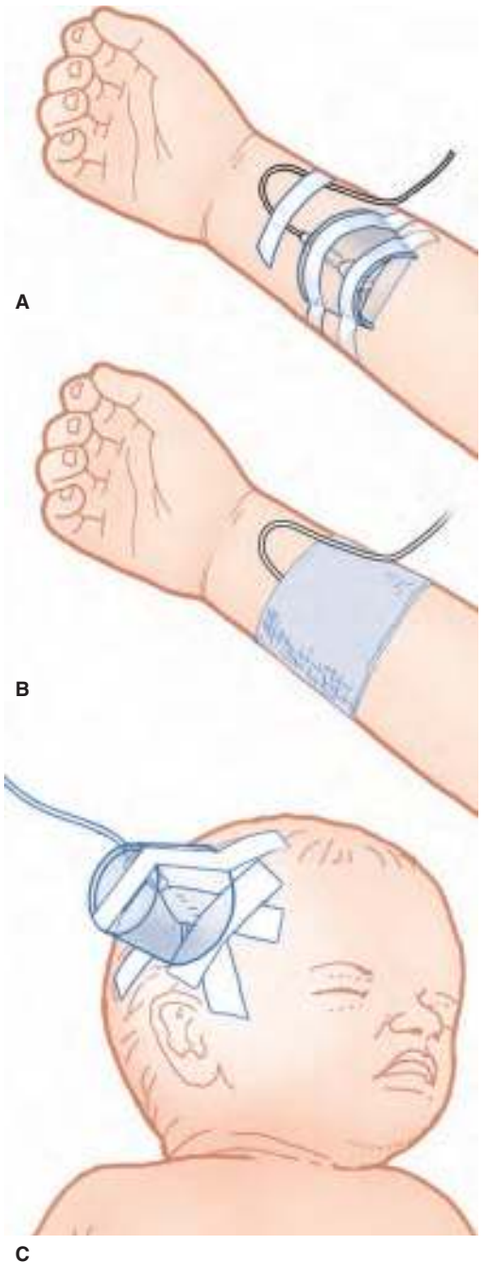

FIGURE 61-21. Securing pediatric intravenous lines. ***A.*** Use a clear plastic medicine cup, cut in half lengthwise, to protect the skin puncture site. ***B.*** A stockinette or gauze roll can be used to further protect the site from manipulation. ***C.*** Protecting a scalp vein cannula with tape and a clear plastic medicine cup.

The IV Pro (WV IV Pro Inc., Fairmont, WV) can be applied (**Figure 59-24**). This shield was designed to protect the IV site from buildup of bacteria and needle dislodgement. It is a protector and securement device and not a dressing. The clear dome allows site monitoring. Its holes allow airflow and prevent condensation. The device can be used multiple times in the same patient.

Other devices may be added for patient safety. The ivWatch (ivWatch, Williamsburg, VA) monitors the IV site to detect leaks, extravasation, and infiltration (**Figure 59-25**). It is composed of the patient monitor, a reusable sensor cable, single-use disposable sensor applied next to the IV site, and an AC adapter. The monitor gives an audible and visual alert. The FIVA (FIVAMed Inc., Halifax, Canada) attaches in-line and alerts to the empty IV fluid bag

(**Figure 59-26**).[22] The FIVA clips onto the IV line and uses infrared to detect air bubbles in the IV tubing and stop the flow. The FIVA gives an audible and visual alert. It is meant to be used for gravity drips and pressure bags and not pumps. Currently, the device is only approved for use in Canada. The ClearLine (ClearLine MD, Woburn, MA) prevents air bubbles in the IV tubing from entering the patient (**Figure 59-27**). The device uses US to monitor the IV tubing for air bubbles as small as 25 μm. The bubbles are directed into a disposable bag. The device works with gravity drips, pumps, and fluid warmers.

REMOVAL OF INTRAVENOUS CATHETERS

Turn off any IV infusions and clamp the tubing. Place the infusion site in a dependent position below the right atrium to prevent a venous air embolism. Remove any tape and dressings from the infusion site. Apply direct pressure to the skin puncture site with a gauze pad. Briskly remove the catheter or needle. Hold firm direct pressure for several minutes. Apply a bandage. Instruct the patient to check for signs of thrombophlebitis, cellulitis, and an infection for the next several days.

COMPLICATIONS

PERIPHERAL VENIPUNCTURE

The main complications of venipuncture are pain and hematoma formation. Minimize pain during venipuncture by using small gauge needles, clearly identifying a vein before attempts at venipuncture, and minimizing the number of attempts at venipuncture. Prevent hematomas and bleeding by removing the tourniquet before removing the needle and applying direct pressure after the needle is removed. Hematomas are self-limited and easily treated with nonsteroidal anti-inflammatory drugs, cool compresses for analgesia, and warm compresses to hasten hematoma resorption. Other complications include nerve injury, usually reversible paresthesias. Arterial puncture is common with deep brachial lines and may rarely be catastrophic if it causes thrombosis of the brachial artery, the sole arterial supply of the forearm and hand.[10] Infection from simple venipuncture is uncommon.

PERIPHERAL INTRAVENOUS CANNULATION

In addition to the complications described for venipuncture, indwelling venous catheters pose additional risks (**Table 59-2**). The steel needle cannula of the butterfly needle can move easily and cause lacerations of the vein and neighboring structures. The risk of infection is greater the longer a catheter is left in place. IV catheters increase the risk of superficial venous thrombosis and thrombophlebitis. This may be prevented by limiting catheter manipulation during tubing changes and by using extension tubing at the catheter hub. Complications are more common when the intravenous catheter is placed in the hand or forearm when compared to other sites.[23] This may be the result of the higher frequency of catheters placed in these two sites in this small study. It is possible to injure a number of structures in the neck during external jugular vein cannulation. This includes the carotid artery, internal jugular vein, and trachea. It is also possible to cause a pneumothorax. Avoid deep penetration with the needle to prevent these complications.

Extravasation of vasopressors or caustic solutions can cause local skin necrosis (Chapter 141). Extravasation of large volumes into a muscle compartment can lead to a compartment syndrome (Chapter 93). This is rare with superficial peripheral venous lines. Prevent extravasation and tissue injury by using a small gauge catheter in a large vein, diluting medications before administration, and observing IV access sites frequently.

Infections can often be prevented by using aseptic technique and sterile dressings and changing peripheral catheters every 48 to 72 hours. The complications of peripheral nerve palsies, pressure necrosis, and compromised peripheral circulation are rare but do occasionally occur. Prevent these with frequent neurovascular checks to any restrained extremity, by padding all pressure points, and by avoiding the placement of circumferential tape on an extremity.

SUMMARY

Venipuncture and peripheral venous access are essential skills for nearly all medical practitioners. The only way to become proficient at these techniques is to master them during training and to practice them regularly. Equally important is frequent reassessment of venous cannulas so that complications can be detected and treated early before they become major problems.

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62 Peripheral Inserted Central Catheter (PICC) Lines

Elizabeth Barrall Werley

INTRODUCTION

There are a variety of short-, moderate-, and long-term vascular access devices available (i.e., midline catheters, nontunneled central venous catheters, peripheral inserted central catheters [PICCs], peripheral intravenous [IV] catheters, ports, tunneled central venous catheters, and ultrasound [US]-guided peripheral IV catheters). PICC lines are commonly used in adults and children.[1-3] PICC lines provide options that routine peripheral IV access or other devices do not provide and without the risks associated with direct puncture of central vessels.[4] PICC lines are a cost-effective option that can often be inserted at the bedside.[5,6] A PICC line reduces health care costs by permitting the patient to receive care as an outpatient.[7,8]

The insertion of a PICC line is infrequently performed in the Emergency Department (ED) and rarely by an Emergency Physician (EP). A PICC line is inserted more commonly by an Interventional Radiologist and their team or a skilled nursing team dedicated to placing PICC lines. An EP may request PICC line placement for a patient given the appropriate clinical scenario. Patients with indwelling PICC lines may present to an ED with complications associated with the PICC line. **Be prepared to recognize the device, determine its use and functionality, and troubleshoot any potential complications.**

ANATOMY AND PATHOPHYSIOLOGY

The veins of the upper extremities are the typical sites for PICC lines to be placed (**Figures 61-3** and **61-4**). The vessels most frequently used are the basilic, brachial, and cephalic veins proximal to the antecubital fossa to avoid occlusion or damage caused by elbow flexion.[9-12] Rarely, the axillary vein, scalp veins, superior vena cava (SVC), a transhepatic approach, or a translumbar approach may be used.[13-15] The veins may be accessed by palpation or some form of diagnostic imaging to guide vein selection. US is often used prior to PICC line placement to determine the patency and size of the veins potentially being accessed (**Figure 62-1**). US use decreases the time needed for insertion, decreases PICC line manipulation, and decreases the need for ionizing radiation when compared to standard blind landmark or blind length techniques.[16] A tourniquet may be used to impede venous return and make the target veins more dilated.[9] Contrast venography of the upper extremity performed under fluoroscopy may be used to select the target vessels. This requires peripheral IV access in a distal superficial vein of the arm and injection of venous contrast to opacify the vessels during fluoroscopy. A tourniquet for venous dilatation will enhance opacification of the vessels.[9]

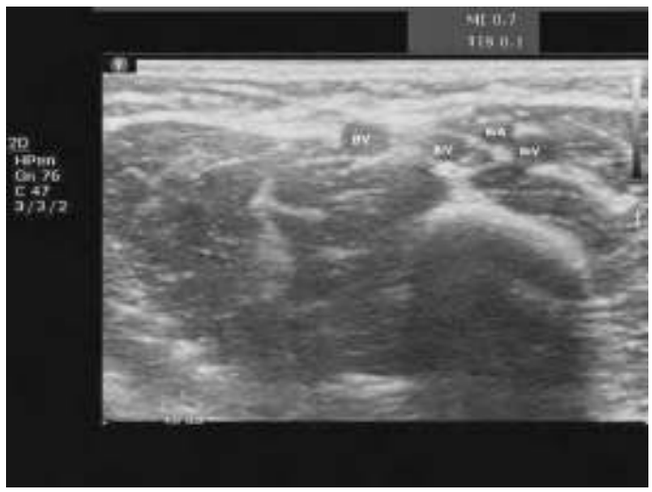


FIGURE 62-1. Transverse ultrasound image of the veins proximal to the antecubital fossa in the right upper extremity. BrA, brachial artery; BrV, brachial vein; BV, basilic vein. (Courtesy of Mark R. Werley, MD.)

PICC LINES VERSUS MIDLINE CATHETERS

Physicians are using midline catheters more frequently for venous vascular access.[17] The main differences are how far the catheter is inserted (**Figure 62-2**). The midline catheter is much shorter and does not require radiography to confirm it is in the proper location and not inserted too far. The midline catheter has its tip positioned at or below the axillary vein. It does not dwell in the central circulation like a PICC line or central line. There are numerous similarities between these two types of catheters. They are inserted in the same location. Both are available in a variety of lengths and lumens. Both are available in power injection models. The indications, contraindications, and insertion are similar. US can be used to insert the catheters. The literature primarily compares midline catheters to peripheral IVs and central lines. The use of midline catheters may eventually be used in the ED.

INDICATIONS

Some indications for PICC lines are clearer than others. The duration and type of treatment impact the decision to use a PICC line. A multispecialty panel used the RAND Corporation/University of California Los Angeles (RAND/UCLA) Appropriateness Method to develop criteria for appropriate use of PICC lines and other venous access devices.[4] The Michigan Appropriateness Guide for Intravenous Catheters (MAGIC) is one of the most comprehensive reviews of the literature combined with expert opinion.[4] Irritant or vesicant infusions (e.g., chemotherapy or total parenteral nutrition [TPN]) are incompatible with peripheral veins. This is deemed an appropriate indication for a PICC line regardless of the proposed duration of treatment. The risk of thrombosis needs to be considered in patients with active malignancy or prior clotting history.[18] A summary of the indications for PICC lines according to the MAGIC guidelines can be found in **Table 62-1**.

PICC lines have the advantages of decreased cost, increased durability, lower infection rates, and outpatient or bedside insertion/removal when compared to other venous access devices. The PICC line is easier to maintain than other venous access devices. The procedure only requires local anesthesia and no procedural sedation or general anesthesia.[7,8,12] This is beneficial in some high-risk patient populations.[7,8,12] Newer PICC lines often tolerate higher flow rates, permit hemodynamic monitoring, and allow

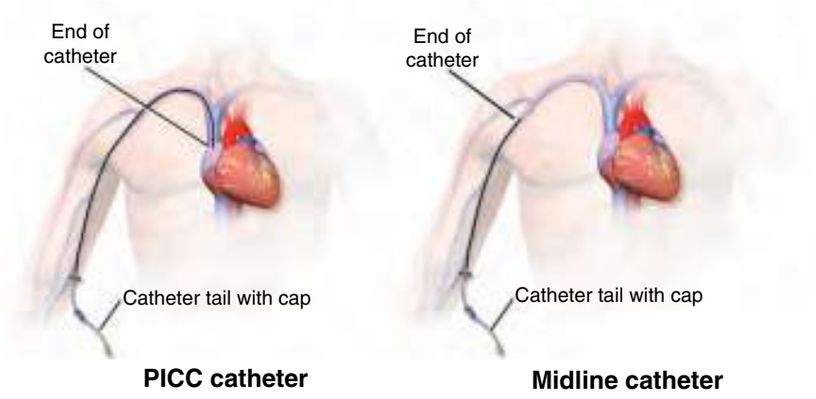


FIGURE 62-2. The different placement of the PICC line and the midline catheter. (Used from www.commons.wikimedia.org.)

for the power injection of contrast material for computed tomography (CT) imaging. Power injectable PICC lines negate the need for larger-bore peripheral IV access to accommodate the power injection of IV contrast.[19,20] Power injectable PICC lines tolerate contrast injection rates up to 5 mL per second and up to 300 psi (**Figure 62-3**). This allows studies that may have otherwise been cancelled or performed without IV contrast. PICC lines are particularly advantageous in the chronically ill, especially the pediatric population. Multiple repeated peripheral IV catheter attempts are not required for access or blood sampling.

PICC lines are used to administer parenteral antimicrobial therapy in the outpatient setting (e.g., private homes or skilled nursing facilities).[2] These patients no longer need inpatient monitoring and treatment but require prolonged courses of IV antimicrobial therapy. PICC lines are the most convenient option to administer concentrated parenteral antimicrobials due to their central location.[7] Outpatient parenteral antimicrobial therapy is used for bacteremia, bone infections (i.e., osteomyelitis), endocarditis, joint infections, skin infections, and soft tissue infections. There is increasing use of PICC lines for device infections, intraabdominal infections, line infections, and prosthesis infections.[7]

PICC lines are commonly used in patients with active malignancy. They can be used to administer IV chemotherapy. The PICC line provides the benefits of administration of antimicrobial therapy, administration of blood products, easy blood draws, fluid hydration, palliative medications, and parenteral nutrition.[8,12,21]

TABLE 62-1 Summary of Appropriate Indications for PICCs

Situation	Proposed duration
Peripherally compatible infusate	• If ≥ 6 days up to 14 days, a midline catheter or ultrasound-guided peripheral IV catheter is preferred, but a PICC line is considered an appropriate option • If > 15 days, a PICC line is preferred.
Non–peripherally compatible infusate	• Indicated regardless of proposed duration.
Peripherally compatible cyclical/episodic chemotherapy	• ≥ 3 months. • The risk of thrombosis must be considered.
Central venous access/invasive hemodynamic monitoring (critically ill)	• ≥ 15 days. • If < 14 days or urgent venous access is needed, a non-tunneled central venous catheter is preferred.
Frequent phlebotomy (inpatient)	• ≥ 6 days.
Intermittent infusions/infrequent phlebotomy (patients with poor peripheral IV access)	• ≥ 6 days. • A midline catheter or ultrasound-guided peripheral IV catheter is preferred.
Infusions/palliative treatment during end-of-life care	• PICC line considered appropriate if it facilitates end-of-life goals.
Peripherally compatible infusions (residents of skilled nursing facilities or transitioning from inpatient to home)	• ≥ 15 days.

Source: Adapted from reference 4.

CONTRAINDICATIONS

The MAGIC guidelines do not address the contraindications to PICC placement. They discuss the consensus agreement on inappropriate indications for PICC lines.[4] Examples of inappropriate indications include any infusion of substances other than those incompatible with peripheral veins or infrequent blood sampling if less than 6 days. A PICC line is not considered appropriate if chemotherapeutic agents are compatible with a peripheral vein and estimated to be administered cyclically for less than 3 months. The request for patient comfort if the patient is not under hospice care or actively dying is considered inappropriate. A PICC line is considered inappropriate for an infusion that is compatible with a peripheral vein and the duration of treatment is "short" or in a hemodynamically unstable patient who requires central venous access. Other contraindications to PICC line placement include an overlying skin or soft tissue infection at the intended insertion site, impaired venous return (e.g., lymphedema or previous axillary node dissection), and thrombophlebitis.[22]

A PICC line is not appropriate for patients with advanced kidney disease or those already receiving renal replacement therapy. It is advised to preserve the veins of the upper extremities for future dialysis access sites whenever possible.[4,9] Alternate sites (e.g., neck veins) or alternate venous access devices may need to be considered.[10]

PICC lines may not be the ideal venous access device in the critical care setting. Volume resuscitation is not always feasible due to lower flow rates and higher resistance from the dimensions of the catheter lumens. Many PICC lines have only a single or double lumen. This does not provide enough options for the multiple infusions critically ill patients often receive.[14]

There are socioeconomic issues to consider. PICC line placement is unlikely to be a good option if the patient lives alone or without access to assistance, if the patient is without communication, if the patient lives in isolated areas, if there is a language barrier that cannot be overcome by translators or family, or if the patients are aggressive, do not cope well when ill, or are active substance abusers.[7] Consider placement in a skilled nursing facility for treatment with indwelling vascular access methods.

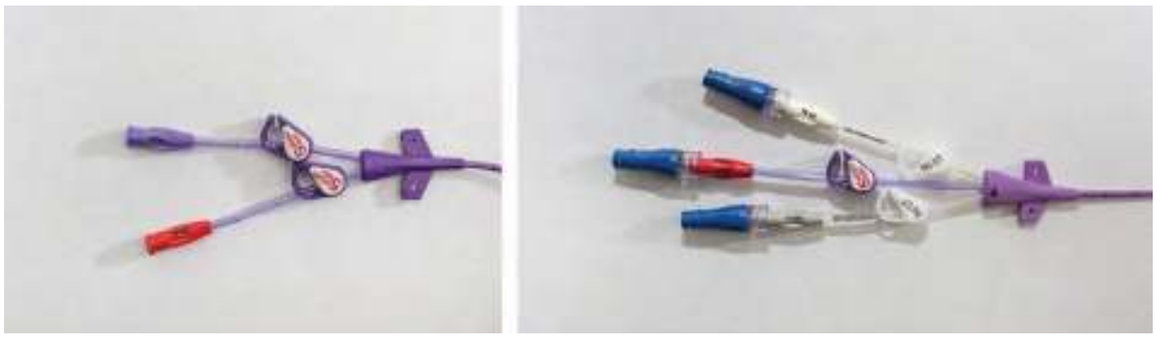


FIGURE 62-3. Power injectable PICCs. Lumens are labeled individually if they support power injection. Clamps showing a flow rate of 5 mL/sec also signify compatibility with power injection. (Courtesy of Joshua Kamnetz, MD.)

EQUIPMENT

- Distal peripheral IV catheter on arm to be accessed if using venography
- Fluoroscopy unit
- Syringes prefilled with nonionic contrast
- US machine
- US vascular transducer
- Sterile US transducer cover
- Sterile US gel
- US needle guide, optional
- Arm board for children
- Tourniquet
- Povidone iodine or chlorhexidine solution
- Hat
- Mask
- Sterile gown
- Sterile gloves
- Local anesthetic solution
- 21 gauge micropuncture needle
- #11 scalpel
- PICC guidewire, catheter, and peel-away sheath
- Needleless catheter caps
- Syringes for aspiration
- Saline flushes
- Heparin locking solution
- Suture material or adhesive dressing

PICC access kits are commercially available and include all the required supplies (**Figure 62-4*A***). PICC lines are typically made of polyurethane or silicone, measure 50 to 60 cm in length, and vary from 2 to 7 French (**Figures 62-4*A* through 62-4*D***).[10,15,23] The PICC line is inserted through a peel-away sheath (**Figure 62-4*C***). The catheter will usually need its length trimmed (**Figure 62-4*D***). This is done via external measurements prior to obtaining venous access or by measuring with an intravascular guidewire placed after obtaining venous access. A good external estimate is to measure from the intended entry site, along the arm to the midclavicular line, and then down to the third intercostal space.[21] The goal for the catheter tip is the high right atrium, the lower third of the SVC, or the junction of the SVC and right atrium (**Figure 62-5**).[10,21-23] Caution needs to be maintained regarding positioning. The ultimate location of the catheter tip may change from when the catheter is placed in the abducted arm with the patient supine to when the patient stands.[6,10]

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

The procedure for PICC line placement can be done at the bedside by specially trained nurses with US guidance or other localization techniques. Placement by a physician in a procedure suite is indicated if this option is not available or unsuccessful.[6,11,24]

PICC line insertion is considered a lower risk procedure compared to the insertion of other vascular access devices. Preprocedure laboratory values are not required. It may be helpful to know the patient's baseline coagulation profile, hemoglobin, and platelet count on a case-by-case basis. It is advisable to obtain the patient's renal function if contrast will be used for venography.[21]

Prepare for the procedure. Place the procedural access arm, ideally the nondominant arm, abducted with the palm facing up. Identify the target vein on the arm by palpation or US. Clean the area of any blood, debris, and dirt. Apply chlorohexidine or povidone iodine to the area and let it dry. **Maintain sterility from this point forward.** Don full personal protective equipment (i.e., hat, mask, sterile gloves, and sterile gown). This protects from exposure to the patient's blood and the patient from iatrogenic infections. Apply sterile drapes onto the patient to demarcate a sterile field.

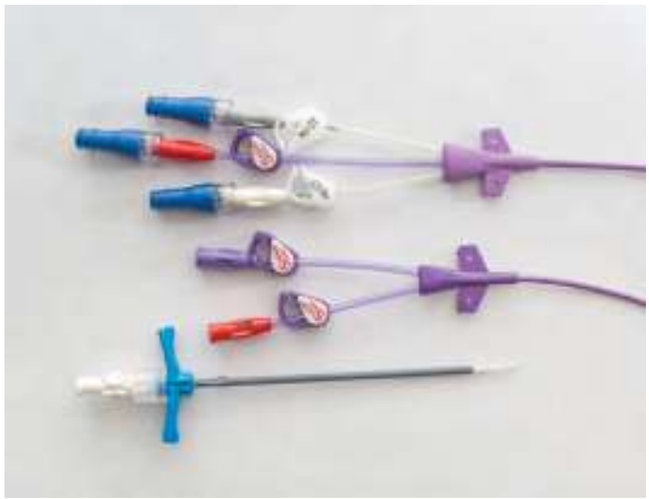
Prepare the US transducer if using US with the procedure. Have an assistant apply US gel over the footprint of the transducer. Instruct the assistant to place the gel-coated US transducer in the sterile cover while you hold it. Have the assistant place sterile US gel over the transducer or on the patient. Place a sterile tourniquet near the shoulder. Reidentify the vein. Apply a 25 gauge needle onto a 10 mL syringe. Fill the syringe with local anesthetic solution. Inject local anesthetic solution subcutaneously and over the vein.

TECHNIQUE

The process of placing a PICC line uses the Seldinger technique (**Figure 62-6** and Chapter 63). **Observe several cardinal rules for the insertion of the PICC line. Always occlude the open hub of a needle or catheter to prevent an air embolism. Never let go of**

A

B



C

D

FIGURE 62-4. Examples of PICC lines. ***A.*** An insertion kit. ***B.*** A variety of available PICC lines. (Courtesy of Cook Medical, Bloomington, IN.) ***C.*** From top to bottom: triple lumen PICC, double lumen PICC, and a peel-away sheath. (Courtesy of Joshua Kamnetz, MD.) ***D.*** A view of the complete PICC line. (Used from www.commons.wikimedia.org.)

the guidewire to prevent its embolization into the central venous circulation. Never apply excessive force to the guidewire on insertion or removal. Doing so may injure the vessel, break the guidewire, and/or embolize the guidewire.

FIGURE 62-5. Chest radiograph confirming PICC line position. The catheter tip is located at the upper right atrium and slightly below the cavoatrial junction. (Courtesy of Mark R. Werley, MD.)

Attach the introducer needle to a 5 mL syringe containing 1 mL of sterile saline or local anesthetic solution. Use the specially designed introducer needle included with the PICC line as it has a relatively thin wall and a larger internal diameter relative to its external diameter. It has a shorter bevel than a conventional hypodermic needle. It has a tapered hub to guide the guidewire into the needle proper.

Insert the introducer needle at a 30° to 60° angle to the skin. Shallower angles are generally necessary in children whose vessels are smaller. Apply negative pressure to the syringe by withdrawing the plunger. Advance the introducer needle into the vein (**Figure 62-6*A***). The vein's distance will vary depending on the patient's size and the target vessel's location. Veins have very low pressures within them and are easily collapsed by external pressure. If no blood is aspirated while withdrawing the needle, withdraw the introducer needle to the subcutaneous plane and redirect it. Avoid putting continuous pressure on the vein as gentle pressure may collapse the vein.

Stabilize and hold the introducer needle perfectly still with the nondominant hand once blood returns in the syringe. Remove the syringe. **Blood should flow slowly and freely from the hub of the needle.** The introducer needle is in an artery if blood squirts out the introducer needle hub. **Occlude the open hub of the introducer needle with the thumb of the nondominant hand while keeping the small finger of the hand in contact with the patient's skin.** The EP's proprioceptive reflexes will prevent movement of the

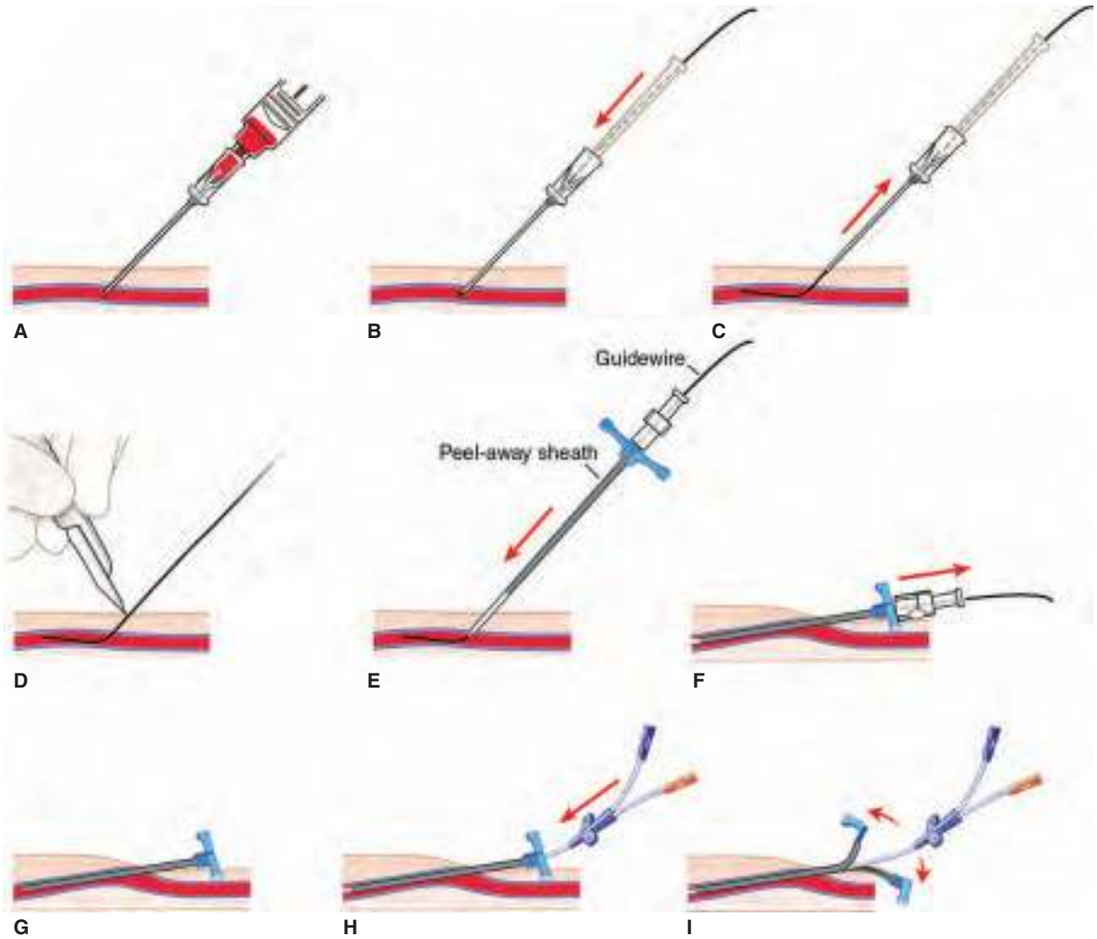


FIGURE 62-6. Insertion of the PICC line using the Seldinger technique. ***A.*** The vein is punctured by the introducer needle and blood is aspirated. ***B.*** The syringe has been removed. The guidewire is inserted through the introducer needle and into the vein. ***C.*** The introducer needle and guidewire sleeve are withdrawn over the guidewire. ***D.*** The skin puncture site is enlarged. ***E.*** The sheath-dilator unit is advanced over the guidewire until the hub is against the skin. ***F.*** The sheath-dilator unit and guidewire are within the vein. ***G.*** The guidewire and dilator have been removed and the sheath remains within the vein. ***H.*** The PICC line is inserted and advanced through the sheath. ***I.*** The peel-away sheath is removed, leaving the PICC line.

introducer needle by maintaining contact with the patient's skin. Even a millimeter of movement may result in failure to stay within the lumen of the vein.

Advance the guidewire through the introducer needle and into the vein (**Figures 62-6*B* and 62-6*C***). The guidewire should advance easily into the vein. **Never force the guidewire.** Guidewire resistance may indicate that the introducer needle is not within the vein, is against the wall of a vessel, or is caught as the vessel bends. Slightly withdraw the guidewire, rotate it slightly, and readvance it. **The use of force will kink the guidewire and may cause it to damage the vein and adjoining tissues.** Advance the guidewire to the SVC or right atrium using fluoroscopy, length measurement estimates, or US.

Withdraw the introducer needle while securely holding the guidewire (**Figure 62-6*C***). Grasp the guidewire as soon as the guidewire is visible between the tip of the introducer needle and the skin. Finish removing the needle over the guidewire.

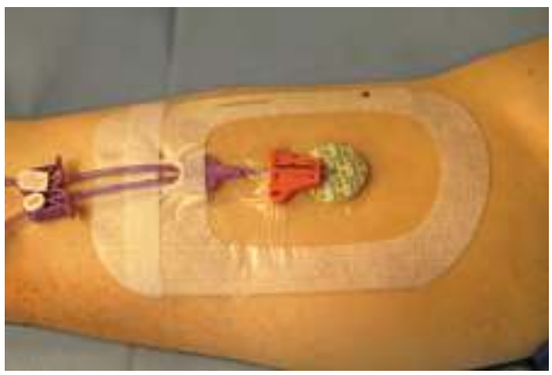
Make a small incision in the skin adjacent to the guidewire using a #11 scalpel blade (**Figure 62-6*D***). Place the peel-away sheath-dilator unit over the straight end of the guidewire (**Figure 62-6*E***). Advance the peel-away sheath-dilator unit over the guidewire, through the skin, and into the vein (**Figure 62-6*F***). A slight twisting motion as it is advanced may aid in its insertion. Continue to advance the peel-away sheath-dilator unit until its hub is against the skin. **Do not release hold of the guidewire at any time.** Remove the dilator and guidewire as a unit and leave the peel-away sheath (**Figure 62-6*G***).

Place the PICC line through the peel-away sheath (**Figure 62-6*H***). Advance the PICC line and into the SVC or right atrium to the desired depth (**Figure 62-5**). Gently rolling or twisting the PICC line between the thumb and forefinger may aid in its advancement. Occlude the open PICC line lumen with the clamp attached to the PICC catheter to prevent an air embolism and excessive blood loss. Remove the peel-away sheath (**Figure 62-6*I***).

Attach a syringe to the PICC line and aspirate blood to confirm that the catheter is within the vein. Withdraw any necessary blood samples from the catheter. Remove the syringe. Apply needleless caps to the ports of the PICC line (**Figure 62-3**). Securely attach the catheter to the skin with an adhesive dressing, nylon suture, a securing device, or silk sutures.[6,10,11,23] Cover the skin puncture site with a sterile dressing.

A securing device is the Secure-A-Cath (Interrad Medical, Plymouth, MN). It securely holds the PICC line at the skin junction (**Figure 62-7**). It minimizes PICC line movement so the distal end does not migrate. It is faster than suturing with no potential needle stick. The device allows cleaning of the insertion site and repositioning of the PICC line. Several other securing devices are available from other companies.

A



B

FIGURE 62-7. Secure-A-Cath device. ***A.*** Schematic of the device attached to a catheter and secured subcutaneously. ***B.*** The device attached to a PICC line and covered with a clear dressing. (Courtesy of Interrad Medical, Plymouth, MN.)

PEDIATRIC CONSIDERATIONS

The procedure for PICC line placement can be done at the bedside by specially trained nurses, either with US guidance or other localization techniques. Placement by a physician in a procedure suite is indicated if this option is not available or unsuccessful.[6,11,24] The complications in pediatrics are the same as those seen in an adult.[25]

ASSESSMENT

Obtain a chest radiograph upon completion of the procedure to document and assess the position of the PICC line tip unless tip placement is visualized under fluoroscopy (**Figure 62-5**). Electrocardiogram (ECG) devices have been proven safe to use for catheter localization and have been implemented at the bedside during PICC line placement.[26-28] Not all patients are candidates for ECG localization as it requires a rhythm with a normal-appearing P wave. It also requires additional training in ECG interpretation for the provider placing the PICC line. Newer technologies incorporate multiple modalities (e.g., ECG and intravascular Doppler in conjunction with an algorithm) to accurately guide PICC line placement.[27]

AFTERCARE

There is an immediate risk of thrombus formation, which can be a source of infection. Use saline flushing and heparin locking after each use, when not routinely being accessed, and at routine intermittent times. There is variation in the recommended dose of heparin and frequency of the flushes. Heparin solution can alter laboratory testing performed on blood drawn from the PICC line. Waste blood when drawing labs from an indwelling PICC line to account for any residual heparin.[12,22] Draw blood from the lumen(s) of a PICC line with a syringe. Avoid using a vacuum tube collection device as it can collapse the catheter.[15,22] Flushing regimens, dressing changes, permissible activities, and routine PICC line care are typically guided by institutional guidelines.[21,22] **A PICC line can be used for weeks or months if it is cared for meticulously.[22]**

Instruct the patient or caretaker to check the insertion site daily. It is easier to inspect if a transparent dressing is used. Instruct the patient to wash their hands and apply gloves before touching the insertion site or using the PICC line. Palpate the insertion site for focal tenderness. Educate the patient about the signs, symptoms, and significance of an infection. The patient should return to their primary physician or ED immediately if they develop any concerns, fever, chills, shortness of breath, redness, or pus at the puncture site. The patient should not use prefilled syringes of heparin or saline if they have leaks, the fluid appears cloudy or discolored, particles are seen in the fluid, or they are past the expiration date. Change the needleless connectors to the PICC line ports no less than every 72 hours or according to the instructions provided by the institution and manufacturer to reduce the risk of infection. Check the catheter for signs of breakage. **Use only compatible attachments.** The patient is often given extensive instruction sheets made by the institution that cover care and using the PICC line.

Scrub the needleless access caps with an appropriate antiseptic solution prior to being accessed.[29] It is imperative to ensure the appropriate withdrawal of blood prior to any infusion.[15] Clamp the catheter to prevent the rare possibility of an air embolism when needleless hubs are not in place or are being changed.[15]

PICC LINE REMOVAL

A PICC line is typically removed by a representative of the insertion team. The process is simple if an EP needs to remove a PICC line.[15] Remove any adhesive dressing or suture material holding the PICC line to the patient's skin. Clean the skin at the insertion site. Withdraw the PICC line from the patient. Apply pressure to the insertion site to tamponade any bleeding. Apply a bandage to the site.

COMPLICATIONS

There will always be complications associated with any procedure or invasive device. Some of the more common issues encountered with PICC lines are discussed below.

MALPOSITION

Difficulty in placing the PICC line or malposition can occur. PICC lines sometimes do not pass centrally and can get caught in the axilla or pass upward into the internal or external jugular veins.[20]

It can be repositioned if caught early enough during the insertion. Delayed malposition of the catheter can occur if the PICC line is not secured correctly, especially at times of dressing changes. Frequent sliding of the catheter at the insertion site can be a source of phlebitis or infection. **Ensure the proper securing of the catheter to minimize this risk.**[19,22] There is some movement of the PICC line with normal arm motion.[12] PICC line dislodgement may be due to iatrogenic issues, patient manipulation, or inappropriate patient movement.[15]

CATHETER DAMAGE

Damage to the PICC line from instruments, needles, or suture can occur during insertion and aftercare.[30] Damage external to the patient may be potentially repaired and salvaged. Consult the PICC line placement team for possible repair. There is the risk that the catheter can fracture and a fragment could migrate if damage occurs internally.[30] Apply a tourniquet to prevent proximal venous migration if it is witnessed. Removal of the broken piece may require a venous cut-down or retrieval by an Interventional Radiologist or Surgeon.[22]

INFECTION

Any indwelling device is a possible source of infection. Infection rates for venous access devices peak within the first 3 months of placement, decrease some, and then plateau by 5 to 6 months after placement.[15] PICC lines have a lower infection rate than some other venous access devices (e.g., nontunneled central venous catheters), but they do have a higher rate of infection than peripheral IV catheters.[22,31,32] PICC line use in the inpatient setting has demonstrated higher catheter-associated infections than in the outpatient setting.[22,32]

There are three major routes of PICC line contamination that can cause bacteremia. The infused solution can be contaminated, the skin at the insertion site can become contaminated, and direct contamination of the hub and lumen can occur. Intraluminal contamination is the most common etiology of catheter-related bacteremia.[33]

Insertion-site infections rarely necessitate removal of the PICC line. These can often be treated with antibiotics in the absence of systemic symptoms or sepsis.[22] It is often unclear initially if the source of a fever or sepsis is related to the indwelling PICC line. Obtain cultures from the insertion site if there are signs of a localized infection. Obtain blood cultures from the PICC line and a separate peripheral source. **The PICC line may remain in place with cultures pending if another source of fever is found.**[22]

Decisions regarding catheter-related bloodstream infection rely on comparison of PICC line blood cultures to peripheral blood cultures or the time differential to positivity between PICC line and peripheral blood cultures. It can be presumed that the PICC line is the source of infection if the PICC line blood culture has colony counts higher than a predetermined threshold compared to the peripheral blood culture or the peripheral blood culture is negative. **Culture all PICC lines removed.**[12,15]

The most common organisms associated with catheter-related bloodstream infection, in descending order of frequency, are coagulase-negative staphylococci, *Staphylococcus aureus*, enterococci, and *Candida* species.[22,33] There are certain organisms in catheter-related bloodstream infections that require the catheter to be removed (i.e., *S aureus*, *Xanthomonas*, *Pseudomonas* species, mycobacteria, *Bacillus* species, *Corynebacterium* species, and *Candida* species). Other indications to remove the PICC line include endocarditis, osteomyelitis, sepsis with significant and persistent end-organ dysfunction, septic shock, and septic thrombophlebitis.[15,22]

PICC lines are available as single-, double-, or triple-lumen catheters (**Figures 62-3 and 62-4**). PICC lines with more lumens and ports allow for the possible introduction of infection each time a port is accessed.[12] There is evidence to support an increased risk of infection with increased lumens. Use a PICC line with the minimum number of lumens needed for the goals of care to minimize the risk of infection.[12,22,34] Antibiotic-coated PICC lines can decrease the rates of bacteremia and infection.[35]

Some intraluminal infections can potentially be treated. This permits the PICC line to remain in place if it is the only remaining viable option for the patient, the risk of replacement is too great, or the risk of insertion of another venous access device is too great. Antibiotic-lock therapy is an option, alone or in conjunction with parenteral therapy. This permits high concentrations of antibiotics to be instilled into the catheter hub and remain in the catheter for a predetermined time. This so-called salvage helps eliminate bacteria that generate a biofilm (i.e., in and around the catheter) and prolong the use of the PICC line.[15,22,32]

OCCLUSION AND THROMBOSIS

Nonthrombotic causes of occlusion include kinking of the catheter, abutment of the catheter against the wall of the vessel, precipitations of infusions within the lumen, and catheter migration leading to tip malposition. A chest radiograph can indicate the proper location of the catheter tip, kinking, or radiographic evidence of damage.[12,22] Abutment of the catheter against the vessel wall can often be resolved by a Valsalva maneuver or positional changes to the patient. Pinch-off of the catheter occurs when it is placed too medially and becomes compressed between the clavicle and the first rib. Patient positioning can be diagnostic and temporarily resolve the problem. The PICC line needs to be repositioned or replaced for long-term management.[12]

Thrombus is the most common cause of catheter occlusion (Figure 62-8).[22] There are two main thrombotic causes of catheter

FIGURE 62-8. Digital subtraction venogram demonstrating complete occlusion of the subclavian vein (*circled*) with collateral formation. (Courtesy of Mark R. Werley, MD.)

occlusion. A fibrin sheath present on the tip or surrounding the catheter may act like a one-way valve. This permits flow of fluid through the catheter but not blood return. A complete occlusion is usually due to a collection of fibrin and platelets preventing the infusion of fluid and the return of blood. These two circumstances are considered appropriate indications for treatment with a small dose of a thrombolytic agent (Chapter 65).[12,15] The agent of choice is guided by institutional guidelines.

Venous thrombosis is another complication that may arise.[18] The risk of thrombosis has been documented to be as high as 50% and is one of the drawbacks to PICC lines.[12] Use of a PICC line with fewer lumens results in a smaller PICC line. Evidence supports decreased rates of thrombosis with fewer lumens.[12,34] Venous thrombosis occurs more frequently if the tip is higher in the SVC and less frequently if it is in the lower SVC, right atrium, or cavoatrial junction.[12,22] PICC lines have a higher rate of upper extremity deep vein thrombosis when compared to other forms of central venous access.[14,18] Current recommendations for a catheter-associated deep vein thrombosis of the upper extremity include leaving the catheter in place and treating with anticoagulation therapy. Consider removal of the PICC line if it is malfunctioning, there are signs of infection, the patient has a contraindication to anticoagulation therapy, there are persistent or worsening signs of deep vein thrombosis despite appropriate therapy, or it is no longer required for treatment.[36]

Another consideration is the long-term sequelae of prior PICC line placement. Stenosis or occlusion of the venous system may have developed along with the formation of collateral vessels in patients who have had previous PICC lines placed in the same anatomic location (**Figure 62-8**). This may lead to increased difficulty in placing subsequent PICC lines and require additional imaging to guide placement or insertion by an Interventional Radiologist. This is an important consideration in pediatric patients or other young patients with chronic illnesses who may require multiple repeated PICC lines over time.[37]

ARRHYTHMIA

Cardiac conduction may be altered if the catheter contacts specific portions of the right atrium. **It is important to ensure the appropriate location of the PICC line and obtain an ECG upon completion of the procedure to avoid this complication.[12]**

SUMMARY

It is very unlikely that an EP may place a PICC line. It is feasible that a request may be placed to insert a PICC line for a patient in the ED. It is even more likely that an EP may deal with a patient presenting with a PICC line that needs to be accessed or presents with a complication. It is imperative that the EP be familiar with these catheters.

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63 Central Venous Access

Arun Nagdev, Craig Sisson, Benjamin Thomas, and Peter Wroe

INTRODUCTION

Percutaneous cannulation of the central veins is an essential technique for both long-term and emergent medical care. Access to the major veins of the torso allows rapid high-volume fluid resuscitation, administration of concentrated ionic and nutritional solutions, and hemodynamic measurements.

Obtaining venous access is an essential skill for the Emergency Physician. Indications for peripheral venous access are broad, ranging from simple fluid and medication administration to delivery of intravenous (IV) contrast for imaging studies. Central venous access is less often compulsory but still remains an indispensable procedure in the practice of Emergency Medicine. Central venous access allows for multiple critical actions to be performed from the administration of blood products and vasoactive medications to transvenous cardiac pacing. Central venous access is often undertaken when peripheral IV access cannot be obtained.

Even the most experienced Emergency Physician can have difficulty securing rapid and functional access to the venous system in specific situations (e.g., cardiac arrest, injection drug users with sclerosed veins, obesity, and severe dehydration). The classical "blind" technique is based on anatomic and vascular landmarks. The growing integration of bedside ultrasound (US) into the practice of Emergency Medicine has slowly changed the way Emergency Physicians are choosing to perform central venous access. US visualization of the patient's vascular anatomy allows the specific advantage of determining the ideal location to access the central venous circulation. A thrombosed vein can be identified, which allows one to preemptively choose another site. The visualization of the overlap of the right internal jugular vein and the carotid artery may prevent inadvertent arterial puncture and the resultant sequelae in the anticoagulated patient. US guidance for central venous access has altered the clinical algorithm of obtaining vascular access, making the procedure easier to perform, and safer for the patient.

Evidence supporting US guidance for central vascular access is fairly robust.[1-5] Convincing data from the Critical Care and Emergency Medicine literature indicates an increased success rate and a decrease in the complication rates. The Agency for Healthcare Research and Quality and National Institute for Clinical Excellence recommended US guidance for central venous access. The availability of small, low-cost, and portable US machines has made US guidance for central venous access a requisite skill for all Emergency Physicians. A brief description of US-guided central venous access is discussed in the following sections as appropriate. Refer to Chapter 64 for the complete details.

ANATOMY AND PATHOPHYSIOLOGY

The tip of the central venous catheter must lie in the superior or inferior vena cava and never in the right atrium. The thin wall of the right atrium may easily be perforated by the catheter tip and result in complications (e.g., hemorrhage and cardiac tamponade). The central venous anatomy is shown in **Figure 63-1**. The superior vena cava is accessed through the internal jugular veins, the subclavian veins, and less commonly the external jugular veins. The inferior vena cava is accessed through the femoral veins. These access routes are discussed in greater detail in the corresponding sections below. The advantages and disadvantages of each route for central venous access are summarized in **Table 63-1**.

INTERNAL JUGULAR VEIN

The internal jugular vein is not directly visible from the surface of the skin. A thorough knowledge of its anatomic relationships is essential for successful cannulation. The internal jugular vein is a direct continuation of the sigmoid sinus and exits the skull through the jugular foramen, just anteromedial to the mastoid process.[6] It joins the subclavian vein deep and just lateral to the head of the clavicle (**Figure 63-2**).[6] The internal jugular vein drains blood back to

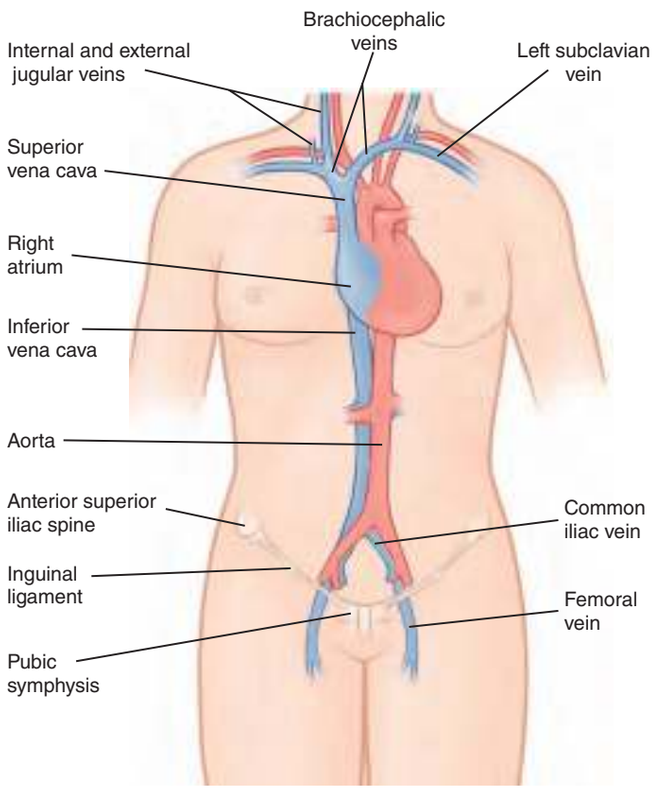


FIGURE 63-1. The anatomy of the central venous system.

TABLE 63-1 Characteristics of the Different Routes for Central Venous Cannulation

	Internal jugular vein	External jugular vein	Subclavian vein	Femoral vein
Risk of infection	Low	Low	Low	High
Patient mobility	Fair	Poor	Good	Bedridden
Trendelenburg required	Yes	Yes	Yes	No, best for congestive heart failure or dyspnea
Need to stop CPR	Probably	Probably	Yes	No, may continue CPR
Suitable for long-term use	Yes, but not if ambulatory	No	Yes—best choice	No, remove within 2–3 days
Risk of venous thrombosis	Low	Low	Low	High

the heart from the brain, face, and neck. There is a proximal and distal dilatation of the vein known as the superior and inferior bulbs of the internal jugular vein. The inferior bulb contains a bicuspid valve to prevent retrograde flow. The surface projection of the internal jugular vein runs from the earlobe to the medial clavicle, between the sternal and clavicular heads of the sternocleidomastoid muscle. The internal jugular vein increases in diameter as it descends. It is joined by tributary veins in the upper neck, which make it easier to cannulate below the level of the cricoid cartilage. The left internal jugular vein is smaller than the right internal jugular vein.[7-9] This is one reason the right internal jugular vein is the choice for vascular access.

The internal jugular vein is collapsible (Figure 63-3). Its overall diameter is dependent on the intravascular volume status and patient position. It has a very small diameter in low-flow states (e.g., during cardiopulmonary resuscitation [CPR] and when the patient is upright). The vein is easily compressible and will collapse with gentle external pressure from a palpating finger or a large-diameter needle indenting the skin (**Figure 63-3*B***). Local masses (e.g., a goiter, hematoma, or tumor) can easily collapse the internal jugular vein. The internal jugular vein is very distensible. Placing the patient in the Trendelenburg position or having the patient perform the Valsalva maneuver will distend the vein and make it easier to locate and cannulate (**Figure 63-3*C***).

The internal jugular vein lies in proximity to the carotid artery, vagus nerve, phrenic nerve, brachial plexus, cervical sympathetic plexus, thyroid gland, and the pleural cupula of the lung along its course. The inferior portion of the left internal jugular vein lies in proximity to the thoracic duct. **The location of these structures places them at risk for injury during central venous cannulation of the internal jugular vein.**

The position of the internal jugular vein in relation to the common carotid artery within the carotid sheath can vary considerably between individuals. The simple assumption that the internal jugular vein is always lateral to the carotid artery has not born out in ultrasonographic studies. Overlap with the carotid artery can vary from 0% to 100% depending on individual anatomic variation, patient positioning, and where along its course the internal jugular vein is imaged.[10-15] It can lie medial to the carotid artery in some patients, making blind needle puncture nearly impossible and extremely dangerous.[10,11,15]

This variability in location is mirrored by the size variability of the internal jugular vein.[8,16,17] The size of the internal jugular vein can vary widely for anatomic and physiologic reasons. A vein diameter of less than 0.7 cm may be an independent risk factor for unsuccessful venous cannulation.[18] The internal jugular vein can vary in size and location when comparing the right side to the left side and the proximal vein to the distal vein.[8,10,13,16] Each of these individual factors forms a strong argument for ultrasonographic assistance during internal jugular vein central line placement.

The common carotid artery travels alongside the internal jugular vein and is an important anatomic landmark for locating the internal jugular vein. The carotid artery runs deep and slightly anterior to the internal jugular vein. The left internal jugular vein usually overlaps the carotid artery in the lower neck (**Figure 63-3*A***). The right internal jugular vein and the right carotid artery are usually separated slightly.

The right internal jugular vein is generally preferred to the left internal jugular vein as the site of central venous cannulation. The right internal jugular vein provides a nearly direct route to the superior vena cava. The dome of the right lung is somewhat lower than that of the left lung and decreases the chance of an iatrogenic

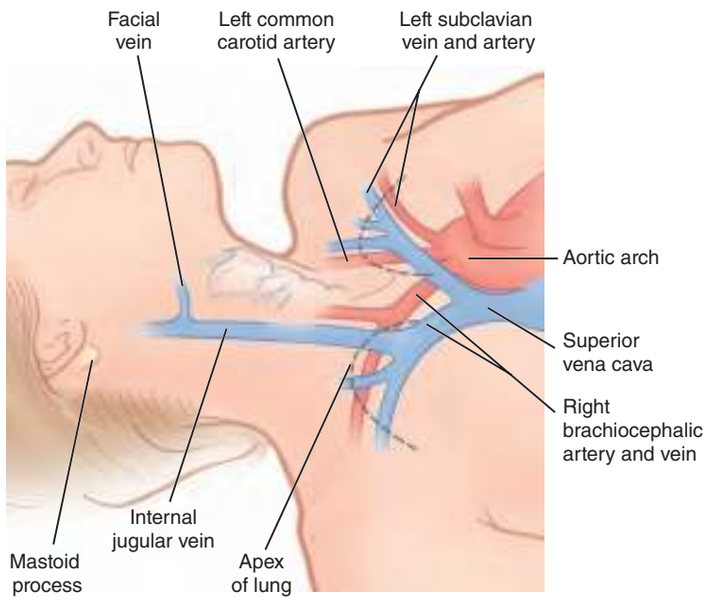
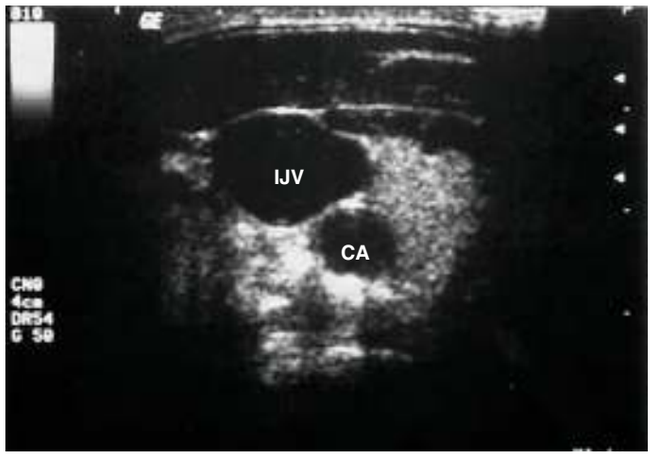
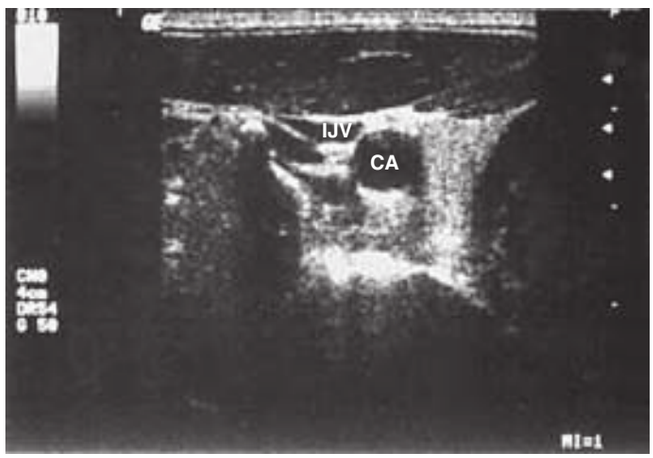


FIGURE 63-2. Anatomy and surface relationships of the internal jugular vein.



A



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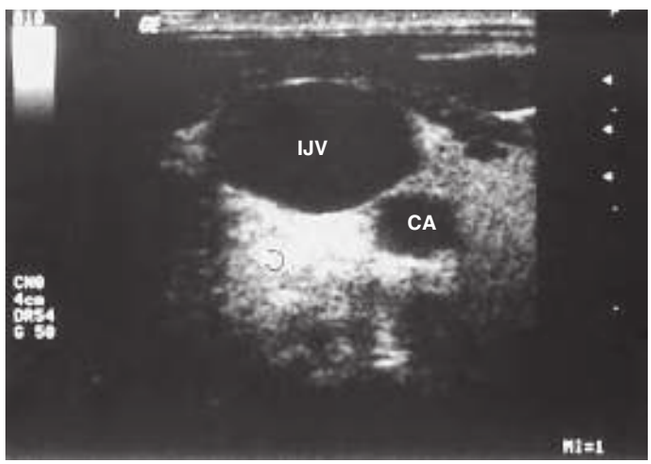


C

FIGURE 63-3. Transverse US images of the left internal jugular vein (IJV) and carotid artery (CA). ***A.*** The patient is supine. ***B.*** The low-pressure internal jugular vein collapses easily while the carotid is still patent with gentle external pressure. ***C.*** The Valsalva maneuver or placement of the patient in the Trendelenburg position dilates the internal jugular vein.

pneumothorax. The thoracic duct is relatively large and lies high in the left chest. These favor the right internal jugular approach to central venous cannulation to minimize complications.

There are three main "blind" or landmark approaches to the internal jugular vein as defined by their relationship to the sternocleidomastoid muscle. These are the anterior, central, and posterior approaches (**Figures 63-4, 63-5, and 63-6**). The central approach is most commonly used. These three approaches are summarized in **Table 63-2** and described later in this chapter.

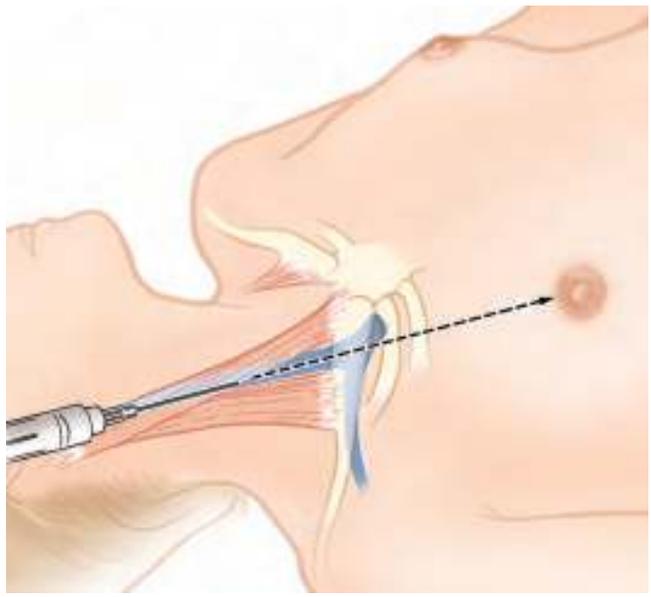


FIGURE 63-4. Central approach to the right internal jugular vein.

SUBCLAVIAN VEIN

The subclavian vein begins as the continuation of the axillary vein at the lateral edge of the first rib (**Figure 63-7**). The subclavian vein courses anterior to the anterior scalene muscle, which separates it from the subclavian artery. The subclavian vein descends to join the internal jugular vein and form the brachiocephalic trunk, which empties into the superior vena cava. **US visualization of the vein**

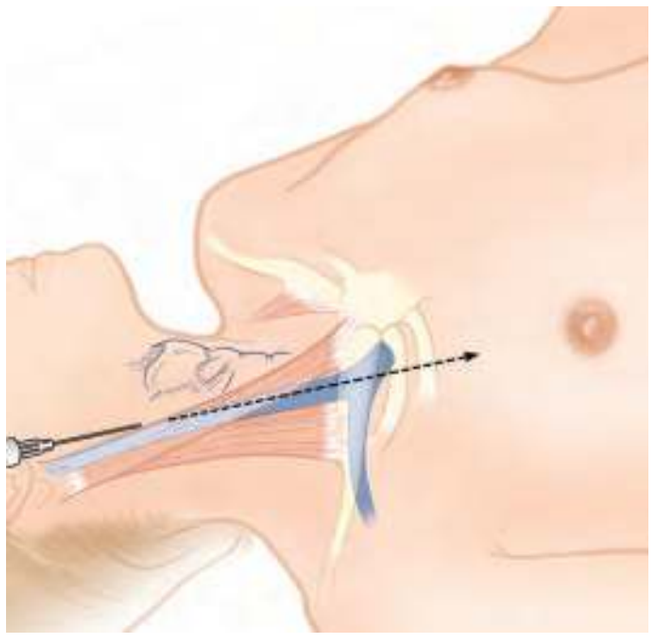


FIGURE 63-5. Anterior approach to the right internal jugular vein.

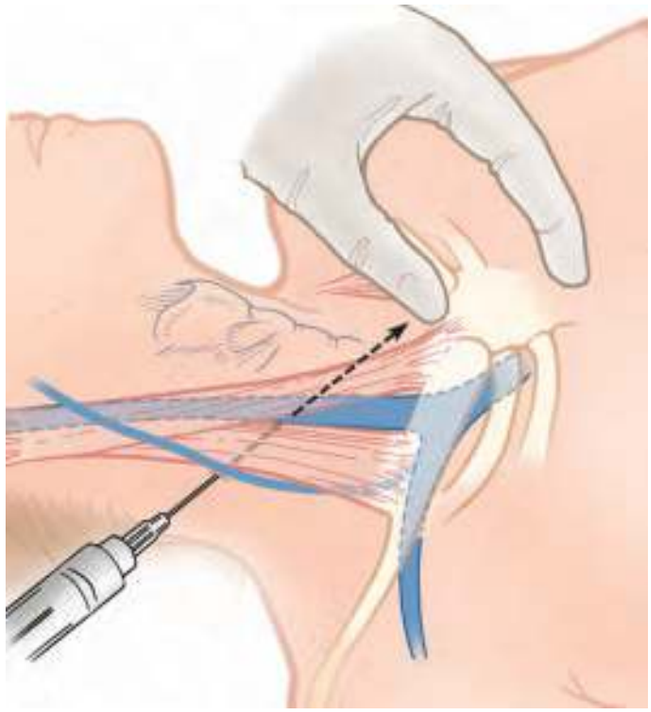


FIGURE 63-6. Posterior approach to the right internal jugular vein.

is not usually possible because the subclavian vein lies directly underneath the clavicle.

The subclavian veins are 1 to 2 cm in diameter in an adult. Fibrous connective tissue joins the subclavian vein to the clavicle and first rib, preventing collapse of the vessel even in the event of a low-flow state. Anatomically associated structures include the thoracic duct which joins the left subclavian vein at its junction with the left internal jugular vein. The right subclavian vein is preferred to the left for central venous access for this reason. The domes of the pleura lie posterior and inferior to the subclavian veins and medial to the anterior scalene muscles. The subclavian arteries lie immediately posterior to the veins (**Figure 63-7**).

Subcutaneous fatty tissue, chest morphology, the close proximity of the pleura, and the close proximity of the subclavian artery make the subclavian vein the least favored site for central venous access in children. This is especially true in infants. An experienced Emergency Physician must perform the procedure if this route must be used in a neonate, infant, or small child.

AXILLARY VEIN

The axillary vein is defined as the continuation of the brachial vein from the medial border of the teres major to the lateral border of the first rib. The axillary vein continues under the clavicle as the subclavian vein, which then joins the internal jugular vein to form the brachiocephalic vein (**Figure 63-2**). Cadaver-based and radiologic studies have demonstrated a great variation in the anatomic relationship between the axillary vein and artery.[19,20] This makes the blind or landmark technique difficult for even the most experienced Emergency Physician.[19,20] A US study of the axillary vein in volunteers demonstrated a decrease overlap between the axillary vein and artery and a farther distance from the pleura as it was followed laterally.[21]

FEMORAL VEIN

The femoral vein is a continuation of the iliac vein after it crosses the inguinal ligament (**Figure 63-8**). The femoral vein lies within the femoral sheath and just medial to the femoral artery in the groin. This relationship can be remembered by the mnemonic "toward the NAVEL." This describes the contents of the femoral sheath from medial to lateral (i.e., femoral **N**erve, femoral **A**rtery, femoral **V**ein, **E**mpty space, and **L**ymphatics). The femoral artery lies at the midpoint of the line connecting the symphysis pubis and the anterior superior iliac spine.[22,23] The femoral vein lies approximately 1 cm medial to the femoral artery pulse in an adult and approximately 0.5 cm medial in infants and young children.[24,25]

Recent anatomic US surveys on adult and pediatric populations have demonstrated variation in the classical anatomic teaching. There is an increased overlap between the femoral vein and artery as they move distally to the inguinal ligament.[26] A study evaluated the amount of venous and arterial overlap in euvolemic children 1 cm distal to the inguinal ligament.[27] The femoral vein had either partial or complete overlap with the femoral artery in 12% of the patients. **These studies and others have noted the great variability of the vascular venous anatomy and recommend US visualization before femoral venous cannulation attempts.**[26-30]

The puncture site for femoral vein cannulation lies medial to the femoral artery and inferior to the inguinal ligament (**Figure 63-8**).[22,23,31] **Perform femoral venous cannulation 1 to 2 cm inferior to the inguinal ligament to prevent inadvertent intraabdominal external iliac vein puncture.** The femoral vein becomes the external iliac vein superior to the inguinal ligament (**Figure 63-8**). **Blood can flow freely into the retroperitoneal space, forming a potentially large and externally invisible hematoma if the posterior wall of the femoral vein is punctured by a through-and-through needle track above the inguinal ligament. It is imperative to puncture the femoral vein inferior to the inguinal ligament.**

The femoral vein remains a popular route for access to the central circulation due to its relative ease of placement.[32] A large, randomized, multicenter study questioned the classically held notion that the femoral venous cannulations have higher rates of infection.[33] The study did not demonstrate a higher rate of infection between catheter tips removed from the femoral vein and the internal jugular vein. The rate of catheter-related bloodstream infection between femoral vein cannulation and internal jugular vein cannulation was similar.

TABLE 63-2 Approaches to the Internal Jugular Vein

	Central	Anterior	Posterior
Insertion landmark	Superior apex of the triangle formed by the two heads of the sternocleidomastoid muscle and the clavicle	Medial edge of the sternocleidomastoid muscle at level of thyroid cartilage	Lateral edge of sternocleidomastoid muscle, 1/3 of the way from the clavicle to the mastoid process
Angle with skin	30° (child), 45–60° (adult)	30° (child), 45° (adult)	30–45°, dive under the border of the sternocleidomastoid muscle
Aim toward	Ipsilateral nipple	Ipsilateral nipple	Sternal notch
Internal jugular vein depth in an adult	Within 3 cm	Within 3 cm	Within 5 cm

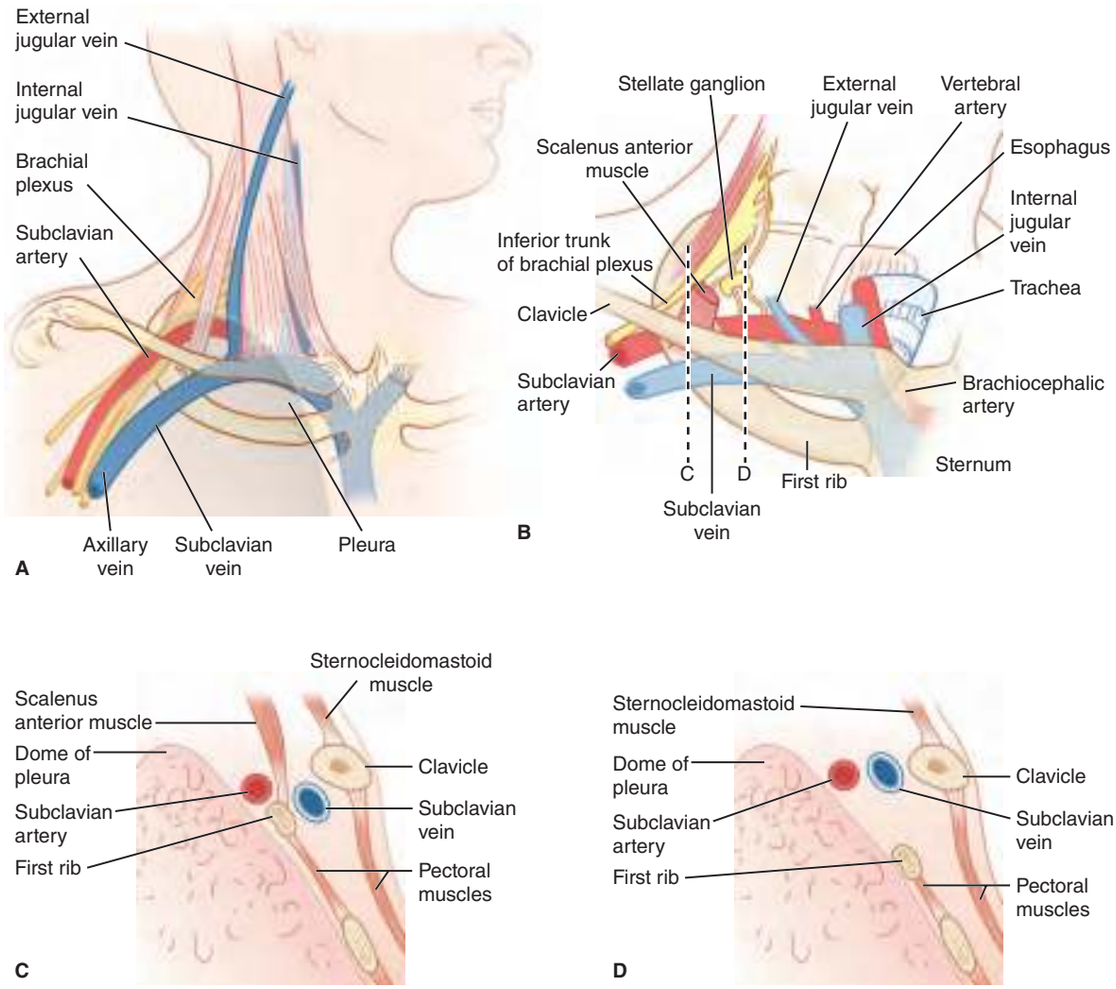


FIGURE 63-7. The anatomy of the subclavian vein. ***A.*** The right subclavian vein. ***B.*** Magnified view of the right subclavian vein demonstrating adjacent structures that may be injured during attempted cannulation. ***C.*** Sagittal section through the mid-clavicle. Note that the first rib protects the subclavian artery during an infraclavicular approach to the subclavian vein. ***D.*** Sagittal section through the medial third of the clavicle. Note the proximity of the subclavian artery and pleural dome.

CENTRAL VENOUS ACCESS IN THE OBESE PATIENT

Central venous access is more complicated in the obese patient.[34] Surface landmarks may be obscured by adipose tissue and skin folds. The clavicle, carotid artery pulses, thyroid cartilage, and trachea can be difficult to palpate. This makes internal jugular and subclavian venous access problematic. The anterior superior iliac spine, femoral artery pulse, and pubic symphysis can be difficult to palpate. This makes femoral venous access problematic. Femoral venous access is often performed lower than normal due to an overhanging pannus. Placing the obese patient in the Trendelenburg position may not be possible. They may decompensate in this position due to their poor pulmonary reserves, decreased lung and tidal volumes, increased intraabdominal pressure against the diaphragm, and difficult airway anatomy. All these factors may increase the rate of complications in the obese patient.

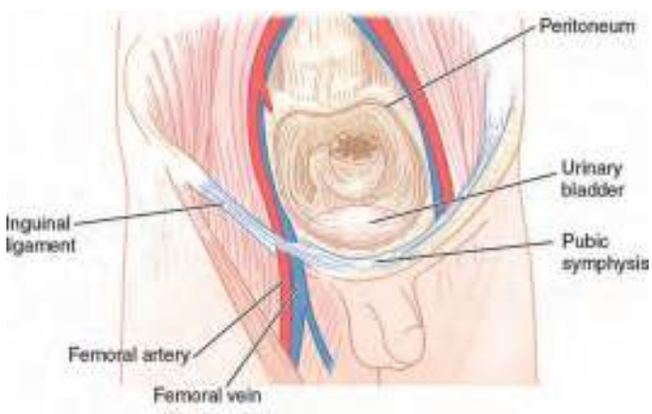


FIGURE 63-8. Anatomy of the femoral vein.

INDICATIONS

The internal jugular route is acceptable for central venous access in most cases. Take into consideration the patient's age, build, expected duration of line placement, health status, medical history, and risk of infection when determining the site of placement. It allows ready access to the superior vena cava for long-term central venous access, caustic infusions, and monitoring of central venous pressure. Pulmonary artery catheters and transvenous pacing wires can be introduced through the right internal jugular vein. The internal jugular vein is accessible without terminating CPR efforts, although chest compressions and the lack of carotid pulsations make accessing it

difficult. The risk of an iatrogenic pneumothorax is probably less with internal jugular vein cannulation as opposed to the subclavian vein, although patient mobility is less and discomfort is greater. The internal jugular vein puncture site is compressible in the coagulopathic patient, but a hematoma formation may lead to airway compromise. Patients historically labeled as having "difficult access" had very low complication rates using US-guided central venous access of the internal jugular vein.[35] Approximately 28% of the total procedures performed were on patients with disorders of hemostasis. Access to the internal jugular vein and subclavian vein is safe in patients with a coagulopathy.[36,37]

The subclavian vein is the preferred route for long-term central venous access. This site allows for ambulation (unlike a femoral line) and neck movement without discomfort (unlike an internal jugular line). The catheter is concealable under clothing and makes outpatient use more acceptable.

The axillary vein is not a commonly used venous access site. It is more commonly used in children for long-term access. The use of US guidance has allowed the axillary vein to be an alternate access site. **The axillary vein should only be cannulated in thin patients and as an alternative site when other sites are not available, have failed, or are contraindicated due to the depth of the vessel and its proximity to the brachial plexus.**

The femoral vein is often the preferred route for emergent central venous cannulation. The indications are the same as for any other site with a few exceptions. The femoral vein is not a suitable route for ambulatory patients beyond the initial resuscitation and stabilization period. Patients with femoral central venous lines must be confined to bed. Femoral venous access is easily obtained in patients with respiratory distress and pulmonary edema since they do not need to be placed in the Trendelenburg position. Femoral venous access is relatively easy during CPR and does not require the cessation of chest compressions. The femoral vein is easily compressible. This makes it preferable to the subclavian vein in coagulopathic patients or those undergoing thrombolysis, although peripheral access would be preferred in these cases.[6] There is no risk of injury to the airway, pleura, or carotid arteries in very young or combative patients. Femoral central venous lines are generally preferred for initial central venous access in the very young or combative patient if deep sedation or neuromuscular paralysis is contraindicated or otherwise unnecessary.

CONTRAINDICATIONS

The usual contraindications to any invasive procedure apply to central venous access. Cellulitis or overlying infection at the puncture site is a contraindication to central venous access. A mass or hematoma causing external compression and/or obliteration of the vessel lumen, an intraluminal thrombosis, or a small size vein when visualized by US are contraindications. Seek an alternative if the patient is combative, agitated, or uncooperative. These patients require sedation and/or paralysis prior to insertion of the central venous line. Distorted anatomic landmarks (i.e., deformities, fractures, obesity, previous catheterization at the site, surgery, or trauma) are relative contraindications. There is a small but real risk of serious morbidity and even death due to the procedure. **Do not place a central venous line unless a peripheral IV line is inadequate or unobtainable and unless personnel capable of managing the procedural complications are immediately available.**

INTERNAL JUGULAR VEIN CANNULATION

Anatomic distortion of the neck (e.g., subcutaneous emphysema or a hematoma) may make placement of an internal jugular line difficult and hazardous. Known severe carotid artery stenosis or atherosclerosis on the desired side of cannulation is a relative contraindication to internal jugular vein cannulation. Accidental carotid artery puncture during line placement may result in plaque rupture and a subsequent stroke. The vein may be collapsed and difficult to access in the hypovolemic patient. Other contraindications to cannulating the internal jugular vein include actual or suspected cervical spine fractures and penetrating neck injuries. Do not cannulate the ipsilateral internal jugular vein if the patient has an implanted pacemaker or defibrillator.[38] Central venous access can result in needle injury to the leads, lead displacement, and thromboembolism.

The subclavian or femoral route may be preferable in some circumstances. The subclavian route is a better choice for long-term lines in ambulatory patients and for hemodialysis. The limited neck mobility due to an internal jugular line is very uncomfortable. Ongoing or impending thrombolytic administration is a relative contraindication to internal jugular puncture. A femoral central venous line is preferable. Successful internal jugular cannulation requires the patient to be placed supine and preferably in 15° to 30° of Trendelenburg tilt. This may be impossible in a patient with severe pulmonary compromise, and the femoral route is preferred. Internal jugular vein cannulation is difficult in children under 1 year of age due to poor landmarks and a very short neck. Internal jugular cannulation is contraindicated in any child who cannot be adequately immobilized or is paralyzed after insertion of the central venous line. Internal jugular cannulation will be more difficult if the patient's neck cannot be turned. The more rotation applied to the neck, the greater is the vascular overlap between the internal jugular vein and carotid artery. This will increase the risk of arterial puncture. Head rotation should be minimized if a blind technique is used. US guidance can minimize head rotation and free the Emergency Physician from the historical anatomic approaches. This allows for any needle approach to be used depending on the location of the internal jugular vein in relation to the carotid artery.

A left bundle branch block is a relative contraindication to central venous cannulation. The guidewire can induce complete heart block when it enters the right ventricle.[39] Extreme caution should be used if an internal jugular or subclavian central line is necessary. **Avoid inserting the guidewire into the heart.** Continuous cardiac monitoring should be used, and transcutaneous and transvenous cardiac pacing equipment should be readily available.

SUBCLAVIAN VEIN CANNULATION

The subclavian vein is incompressible and should be accessed with care in any patient who is coagulopathic. Current or imminent systemic thrombolysis is an absolute contraindication to placing a subclavian vein catheter.[6] Perform subclavian vein cannulation on the contralateral side if the patient is relying on a single lung. Chest wall deformities, distorted anatomy, and suspected vascular injury to the chest or ipsilateral upper extremity are contraindications. This route should be avoided if the patient has had prior surgery or trauma to the clavicle, the first two ribs, or the subclavian vessels. Do not cannulate the ipsilateral subclavian vein if the patient has an implanted pacemaker or defibrillator.[38] Central venous access can result in needle injury to the leads, lead displacement, and thromboembolism.

AXILLARY VEIN CANNULATION

US-guided axillary vein cannulation can be difficult for the Emergency Physician inexperienced in accessing this vein. **Use the axillary vein as an access point only when other central veins are not accessible or contraindicated and the Emergency Physician is comfortable with the procedure of US-guided vascular access.** The depth of the axillary vein, commonly > 3 cm, makes access difficult in patients who are not thin. The proximity of the brachial

plexus can result in significant injury if the needle penetrates it. The length of the catheter may be too short to reach the superior vena cava if placed in the left axillary vein.

FEMORAL VEIN CANNULATION

Contraindications to femoral line placement include infection, venous thrombosis, or significant trauma to the ipsilateral lower extremity or groin area. Abdominal trauma may result in an interruption of the inferior vena cava, allowing any infused fluid or blood to flow into the abdomen rather than into the central circulation. Blood return below the diaphragm is reduced during CPR, and a femoral catheter must end near the level of the diaphragm for medications to be most effective.

Catheterization via the internal jugular vein or subclavian vein is usually easier if the purpose of central venous access is pulmonary artery catheterization or transvenous cardiac pacing. These procedures often require fluoroscopy when they are performed through the femoral vein. Central venous pressures measured through a femoral vein catheter may be inaccurate unless the patient is perfectly supine.

EQUIPMENT

- Sterile gloves and gown
- Facemask and cap
- Povidone iodine or chlorhexidine solution
- Sterile drapes or towels
- Local anesthetic solution
- 25 gauge needle
- 5 mL syringes
- "Finder" needle, usually 22 gauge for an adult
- 5 mL syringe with a nonlocking hub
- Thin-walled introducer needle or catheter-over-the-needle
- Guidewire
- Gauze 4×4 squares
- Central venous line
- Dilator
- #11 scalpel blade
- Nylon or silk suture, 3–0 or 4–0
- Sterile saline
- Needle driver
- Tape and catheter site dressing material
- Catheter clamp, if supplied with the kit

A variety of standard kits are commercially available (**Figure 63-9**). They contain all required equipment. The appropriate catheter should be chosen based on the patient's needs. The optimal catheter lengths for patients of different ages are summarized in **Table 63-3**.

Catheters with between one and four lumens are available. Multiple-lumen catheters are available in a variety of sizes and allow simultaneous venous pressure measurement, administration of medications, and venous sampling without disconnecting the infusion apparatus. Disadvantages of multiple-lumen catheters over single-lumen catheters include smaller lumen sizes for a given catheter's outside diameter, greater cost, and the need to maintain unused lumens to prevent them becoming thrombosed. There is probably no increased risk of infection in using triple-lumen versus single-lumen catheters.[40-42] Some comparisons between these devices are summarized in **Table 63-4**.

A

B

FIGURE 63-9. Equipment needed for central venous catheterization. ***A.*** A commercially available central venous line kit. ***B.*** Examples of different catheter types available. From left to right: single-lumen, double-lumen, triple-lumen, and introducer sheath (Cordis).

Percutaneous sheaths are intended primarily for the introduction of intravascular devices (e.g., pulmonary artery catheters and transvenous pacing wires). They are most often used in the Emergency Department as a large-bore line for the rapid resuscitation of hypotensive and hypovolemic patients. Sheaths are available in many sizes and configurations. Many models have an adjustable hemostasis valve that may be removed and a side port that allows infusion while the main lumen is being used for monitoring.

The equipment required for subclavian vein cannulation is the same as that for internal jugular vein cannulation. Subclavian vein catheters must be slightly longer or inserted farther than internal jugular vein catheters. Left-sided catheters must be a few centimeters longer or inserted farther than right-sided catheters. The longer

TABLE 63-3 Catheter Sizes, Types, and Characteristics

Catheter size (French)	Number of lumens	Patient size	Venous access site	Minimum catheter length (cm)*
2	1	Infant	Femoral, internal jugular, external jugular, or subclavian	5
3	1	< 5 kg	Femoral, internal jugular, external jugular, or subclavian	5
4	1,2	5–10 kg 10–15 kg	Femoral Femoral, internal jugular, external jugular, or subclavian	5 8–12
5	1, 2, 3	> 15 kg	Femoral, internal jugular, external jugular, or subclavian	12–25
7	1 (sheath), 2, 3	>40 kg	Femoral, internal jugular, external jugular, or subclavian	15–25
8 and larger	1 (sheath), 2, 3, 4	Adult	Femoral, internal jugular, or subclavian	15–25

*The longer end of the catheter length range is for use in the subclavian veins, with the longest catheters needed for the left subclavian vein.

needle should be used for subclavian vein cannulation if the kit used has two different lengths of introducer needles.

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative. Obtain an informed consent for the procedure unless it is being performed emergently. Place the patient in the Trendelenburg position if catheterization of the internal jugular vein is being attempted. Position the patient in at least 15° of Trendelenburg to prevent an air embolism. Slightly rotate the patient's head toward the side opposite that to be cannulated. A large degree of head rotation has been shown to increase the overlap between the internal jugular vein and carotid artery, theoretically increasing the risk of carotid artery puncture.[43]

The subclavian vein is fixed to the surrounding tissues and will neither collapse nor distend. **The Valsalva maneuver or the extreme Trendelenburg position is not necessary.** Head rotation is neither necessary nor helpful. Slightly abduct the patient's arm on the side to be cannulated. Avoid placing rolled towels between the shoulder blades as this can decrease the distance between the clavicle and first rib, compress the subclavian vein, and make the procedure more difficult.[44] Placement of rolled towels between the shoulder blades decreases the overlap between the carotid artery and internal jugular vein.[45]

TABLE 63-4 Comparisons between Central Venous Catheter Types

	Single lumen	Multiple lumen	Sheath (Cordis)
Minimum outer diameter	Smallest	Intermediate	Largest
Infusion rate	Moderate	Lowest (resuscitation catheters with larger lumen available)	Fastest (for central lumen; side port is slower)
Simultaneous infusions, or infusion while monitoring	No	Yes	Yes, if central lumen and side port both used
Length	Varies, fairly long	Long	Short
Allows device insertion (pulmonary artery lines and transvenous pacemakers)	No	No	Yes

Place the patient supine or in slight reverse Trendelenburg if femoral vein catheterization is being attempted. The reverse Trendelenburg position will increase the cross-sectional area of the femoral vein.[46] The Trendelenburg position is contraindicated due to the risk of venous air embolism. Slight external rotation and abduction of the extremity may increase the amount of femoral vein accessible for cannulation.[47] It is easier for a right-handed Emergency Physician to perform the procedure on the patient's right side, regardless of which femoral vein is being accessed. The opposite is true for those who are left-handed. Do not abduct or externally rotate the patient's hip as this may increase the overlap of the femoral vessels.[48]

Identify the anatomic landmarks for the procedure after positioning the patient. Clean any dirt and debris from the area of the puncture site. Apply povidone iodine or chlorhexidine solution and allow it to dry.[49] Chlorhexidine solutions may be more effective at preventing infections.[50] **It is recommended to prepare the entire neck and clavicular area if the internal jugular or subclavian routes are attempted so that, if access to one site is unsuccessful, another site may be accessed without repeat prepping and draping. Due to the risk of inducing a pneumothorax, attempts at contralateral internal jugular or subclavian vein cannulation after an unsuccessful attempt must be delayed until a chest radiograph is checked to prevent bilateral pneumothoraces.**

Infiltrate the subcutaneous tissues at the needle puncture site with a generous volume of local anesthetic solution, including any areas that will be used for suturing the catheter in place. This allows the local anesthetic to diffuse throughout the area and take effect before the main procedure begins. Any distortion of anatomic landmarks caused by anesthetic infiltration decreases as the anesthetic is absorbed into the subcutaneous tissues.

Apply electrocardiographic monitoring, pulse oximetry, and noninvasive blood pressure monitoring to the patient and administer supplemental oxygen. Electrocardiographic monitoring during insertion of a central line is recommended due to the risk of ventricular dysrhythmias if the guidewire or catheter enter the right ventricle. It is preferable to have a designated person whose only job is to watch the monitoring equipment. The patient's face and chest will be draped for the internal jugular or subclavian vein cannulation procedure. The Emergency Physician will be focused on the procedure and is often unaware of any sudden patient deterioration, ventilator disconnect, or other irregularities. Resuscitation equipment should be immediately available. A postinsertion chest radiograph to verify line placement and the lack of a pneumothorax must be immediately available. Bedside ultrasound can be used to ensure lack of a pneumothorax as well as proper placement of the catheter tip.

Prepare for the procedure. Apply sterile gloves, a sterile gown, a hat, and a face mask. Some Emergency Physicians prefer to double-glove. If one glove becomes contaminated, it can be discarded and the procedure continued without interruption. Open the desired venous access kit using aseptic technique on a bedside table. Perform a quick inventory and identify all necessary equipment before beginning the procedure. Set up a sterile field next to the patient and within easy reach. Place the equipment that must be immediately at hand on the sterile field. This includes a sterile drape, syringe, large-bore hollow needle, guidewire, and gauze squares. Any other equipment, including the catheter itself, may be temporarily left in the kit. **Never use the patient as a table.** Everything can fall onto the floor if the patient moves. A needle-stick injury can occur from the falling needles or if the instinct to grab the falling equipment occurs.

A difficulty arises in how far the central venous line is inserted. A guideline using equations was determined to predict the length of catheter to insert, reduce complications, and save time.[51] The equations for insertion length are simple to remember for the right subclavian vein [i.e., (height ÷ 10) – 2 cm], left subclavian vein [i.e., (height ÷ 10) + 2 cm], right internal jugular vein (i.e., height [cm] ÷ 10), and left internal jugular vein [i.e., (height ÷ 10) + 4 cm].

INTERNAL JUGULAR VEIN CATHETERIZATION TECHNIQUES

CENTRAL APPROACH TO THE INTERNAL JUGULAR VEIN

While an internal jugular vein cannula can be inserted using the over-the-needle and through-the-needle techniques, the Seldinger technique is often preferred.[52,53] See Chapter 59 and **Table 63-5** for a more complete discussion. The Seldinger technique uses a flexible guidewire inserted through a thin-walled hollow needle to guide a catheter of any desired length through the skin and into the central circulation. This technique is described below and summarized in **Table 63-6**.

Clean, prep, and drape the area as described previously. Place the patient in the Trendelenburg position with their head down 15° to 30°. Slightly rotate the patient's head away from the side that will be cannulated. Excessive rotation will distort the anatomic landmarks and may bring the internal jugular vein closer to the carotid artery.[43,54,55]

Observe several cardinal rules for the insertion of the catheter. Always occlude the open hub of a needle or catheter in a central vein to prevent an air embolism. Never let go of the guidewire to prevent its embolization into the central venous circulation. Never apply excessive force to the guidewire on insertion or removal. Doing so may injure the vessel, break the guidewire, and/or embolize the guidewire.

Attach the introducer needle to a 5 mL syringe containing 1 mL of sterile saline or local anesthetic solution. The specially designed introducer needle included with the catheter should be used, as it has a relatively thin wall and a larger internal diameter relative to its external diameter. It has a shorter bevel than a conventional hypodermic needle. It has a tapered hub to guide the guidewire into the needle proper.

If there is doubt about the exact location of the vein, it may first be located with a small "finder" needle. Insert a 25 or 27 gauge needle attached to a 5 mL syringe through the skin puncture site previously chosen. Advance the needle at a 30° to 60° angle to the skin while applying negative pressure to the syringe. A flash of blood signifies that the tip of the needle is within the vein. Note the depth and location of the vein. Remove the finder needle. Alternatively, the finder needle may be left in place for reference.

TABLE 63-5 Comparison of Central Venous Catheterization Methods

	Seldinger	Catheter-over-the-needle	Catheter-through-the-needle
Insertion needle	Small	Large	Largest
Speed	Slowest	Fastest	Fast
Number of steps	4+	1	2
Risk of catheter shear	None	Low	Highest
Catheters and lumens available	Single- or multiple-lumen, sheath/introducer	Single-lumen only	Single-lumen only
Rate of infusion	Highest (with sheath)	Moderate	Low to moderate

Insert the introducer needle at a 30° to 60° angle at the apex of the triangle formed by the sternal and clavicular heads of the sternocleidomastoid muscle and the clavicle (**Figure 63-4**). This point is just lateral to the carotid artery pulse. Direct the introducer needle toward the ipsilateral nipple. Shallower angles make it necessary to traverse a greater amount of subcutaneous tissues and structures before entering the vessel. Steeper angles make insertion of the catheter over the guidewire difficult, as the guidewire tends to kink. Shallower angles are generally necessary in children whose vessels are smaller. Inject a small amount of the fluid in the syringe to remove any skin plug that may block blood return once the vein has been penetrated.

Apply negative pressure to the syringe by withdrawing the plunger. Advance the introducer needle into the vein (**Figure 63-10*A***). The vein's distance will vary depending on the patient's size and the target vessel's location. Stop advancing the introducer needle if the vein is not located within 3 to 5 cm of the skin. Withdraw the needle slowly while continuing to aspirate. The vessel will often have been completely traversed, and no blood will return due to collapse of the vein by the pressure of the skin being forced inward as the introducer needle passes through it. Veins have very low pressures within them and are easily collapsed by external pressure. If no blood is aspirated while withdrawing the needle, withdraw the introducer needle to the subcutaneous plane and redirect it slightly medially. Avoid putting continuous pressure on the carotid artery pulse, as even gentle pressure may collapse the internal jugular vein (**Figure 63-3*B***).

Stabilize and hold the introducer needle perfectly still with the nondominant hand once blood returns in the syringe. The carotid artery has been entered if the blood is bright red and/or forces the plunger of the syringe back. Remove the syringe. **Blood should flow slowly and freely from the hub of the needle.** The introducer needle is in the carotid or subclavian artery if blood squirts out the introducer needle hub. If blood dribbles out or does not flow from the hub and the patient has spontaneous circulation, reattach the syringe and reposition the introducer needle until free flow is obtained. Occlude the open hub of the introducer needle with the thumb of the nondominant hand while keeping the small finger of the hand in contact with the patient's skin. The Emergency Physician's proprioceptive reflexes will prevent movement of the introducer needle by maintaining contact with the patient's skin. Even a millimeter of movement may result in failure to stay within the lumen of the vein.

Prepare the guidewire (**Figure 63-11**). Grasp the guidewire and its sleeve with the dominant hand. The tip of the guidewire has a "J" shape when the sleeve is retracted (**Figure 63-11*A***). Slide the sleeve forward to straighten out the "J" of the guidewire (**Figure 63–11*B***). Insert the guidewire sleeve into the hub of the introducer needle (**Figures 63-10*B* and 63-11*C***). Advance the guidewire through the sleeve and into the introducer needle. **Never let go of the guidewire. One end of the guidewire must always be held to prevent its loss and embolization into the central circulation.**

Do not simply reverse the guidewire if the sleeve used to straighten the curved end of the guidewire is lost. The straight end of the guidewire can puncture the wall of the vein. Grasp the guidewire between the fourth and fifth fingers and the palm of the dominant hand (**Figure 63-12*A***). Apply gentle traction on the curved guidewire tip with the thumb and the second fingers to straighten the guidewire (**Figure 63-12*B***). The guidewire can then be inserted into the introducer needle hub without the use of the sleeve.

Advance the guidewire through the introducer needle and into the vein (**Figure 63-10*B***). The guidewire should advance easily into the vein. **Never force the guidewire.** Guidewire resistance may indicate that the introducer needle is not within the vein, is against the wall of a vessel, or is caught as the vessel bends. Slightly withdraw

TABLE 63-6 Summary of the Seldinger Method of Central Venous Cannulation*

Step	Action	Tips and caveats
1	Prep and drape the skin puncture site.	For internal jugular vein, prepping down to the clavicle and up to the jaw will enable an attempt at the ipsilateral subclavian vein (or vice versa).
2	Anesthetize the puncture site if not already done.	Anesthetize the suture sites also.
3	Uncap the distal lumen.	Additional lumens may be flushed at this point or after insertion, as desired.
4	Locate the vein using the finder needle and aspirating syringe.	Internal jugular vein should be reached within 3 cm. Stop advancing after 4–5 cm if the vein is not located.
5	Remove the finder needle, noting the direction and depth of the internal jugular vein. Or withdraw the needle slightly so it is outside the internal jugular vein and leave it in place as a guide.	A few drops or a line of blood may be left on the skin as the finder is withdrawn to show the proper direction.
6	Insert introducer needle on a syringe along the "finder's" path until venous blood is aspirated. Alternatively, an introducer catheter and needle assembly can be used to cannulate the internal jugular vein; the needle is then withdrawn.	Syringe must have a nonlocking hub. A little saline in the syringe allows any occluding skin plug to be ejected. The vein is often located on withdrawal of the needle, since the friction of the large needle in the tissues can compress the internal jugular vein.
7	Disconnect the syringe from the needle, immediately occluding the open needle hub to prevent air embolism.	Do not move the needle at all! Keep the hand holding the needle in contact with the patient's skin to prevent movement.
8	Insert the guidewire through the introducer needle and into the vein.	Do not move the needle! Do not force the guidewire—it should pass easily!
9	Advance the guidewire into the vein to the desired depth or until ventricular ectopy is seen on the ECG monitor.	The guidewire must be securely in the vein, not just in the subcutaneous tissue.
10	Withdraw the introducer needle a few millimeters and use the scalpel to enlarge the puncture site slightly.	Keeping the needle in place eliminates any possibility of cutting the guidewire.
11	Remove the introducer needle.	Never let go of the guidewire!
12	Thread the dilator over the guidewire until it can be grasped outside the hub, and then insert and withdraw the dilator.	Always keep a firm grip on the guidewire!
13	Thread the catheter tip over the guidewire and withdraw the guidewire from the skin until it can be grasped at the infusion hub.	Never let go of the guidewire.
14	Insert the catheter to the desired depth; most catheters are marked in centimeters, with larger markings every 5 and 10 cm. Introducer sheaths should be inserted completely.	The tip of the catheter should be in the superior vena cava, at the level of the manubriosternal angle.
15	Holding the catheter in place, remove the guidewire. Occlude the open hub with a gloved finger to prevent air embolism.	Do not apply excessive force to the guidewire. If it is trapped, withdraw the catheter a few centimeters and try again. Do not break the wire!
16	Attach a syringe to the catheter hub and aspirate blood, taking samples as desired; then flush the lumen with saline and begin the desired venous infusion.	Other lumens may be aspirated, flushed, and clamped.
17	Verify intravenous placement before suturing the catheter in place.	If the patient's blood travels up the intravenous tubing, the catheter is in the carotid artery!
18	Remove the patient from the Trendelenburg position.	
19	Suture the catheter to the skin with sutures and tape.	Take care not to puncture the catheter or to occlude it with a tight suture.
20	Apply a dressing to the catheter site.	
21	Verify catheter tip position by chest radiograph.	Catheter tip must be in the superior vena cava, not in the right atrium. Tip should be above the azygos vein and the carina, with the tip parallel to the vessel wall.

*The central approach to the internal jugular vein is used as an example, although the same technique is used for other approaches and central veins.

the guidewire, rotate it slightly, and readvance it. **The use of force will kink the guidewire and may cause it to damage the vein and adjoining tissues.** Advance the guidewire 5 to 10 cm into the vessel or until ectopic beats are seen on the cardiac monitor. **Do not advance the guidewire more than 16 cm into the right internal jugular vein and 18 cm into left internal jugular or subclavian veins.**

Withdraw the introducer needle and guidewire sheath while securely holding the guidewire (**Figure 63-10*C***). Grasp the guidewire with the nondominant hand as soon as the guidewire is visible between the tip of the introducer needle and the skin. Finish removing the needle over the guidewire.

Make a small incision in the skin adjacent to the guidewire using a #11 scalpel blade (**Figure 63-10*D***). Place the dilator over the straight end of the guidewire (**Figure 63-10*E***). Advance the dilator over the guidewire, through the skin, and into the vein. A slight twisting motion of the dilator as it is advanced may aid in its insertion. Continue to advance the dilator until its hub is against the skin. **Do not release hold of the guidewire at any time.** Remove the dilator over the guidewire.

Place the catheter tip over the guidewire. Advance the catheter over the guidewire and into the vein to the desired depth (**Figure 63-10*F***). **Do not release hold of the guidewire.** Gently rolling or twisting the catheter between the thumb and forefinger may aid in its advancement. Insert the catheter to the predetermined desired depth. This is approximately 16 to 18 cm for right-sided lines and 18 to 20 cm for left-sided lines. Hold the catheter securely in place and remove the guidewire (**Figure 63-10*G***). Occlude the open catheter lumen with a sterile-gloved finger to prevent an air embolism and excessive blood loss.

Attach a syringe to the catheter hub and aspirate blood to confirm that the catheter is within the vein. Withdraw any necessary blood samples from the catheter. Remove the syringe. Attach infusion tubing or a heparin lock to the port and flush the catheter to prevent a blood clot from obstructing the lumen. If a multilumen catheter is inserted, flush any other lumens after first withdrawing any air (**Figure 63-13**). Securely attach the catheter to the skin with nylon or silk sutures. Cover the skin puncture site with a sterile dressing.

ANTERIOR APPROACH TO THE INTERNAL JUGULAR VEIN

The skin puncture site is at the anterior border of the sternal head of the sternocleidomastoid muscle, just lateral to the carotid artery and at the level of the cricoid cartilage (**Figure 63-5**). Enter the skin at a

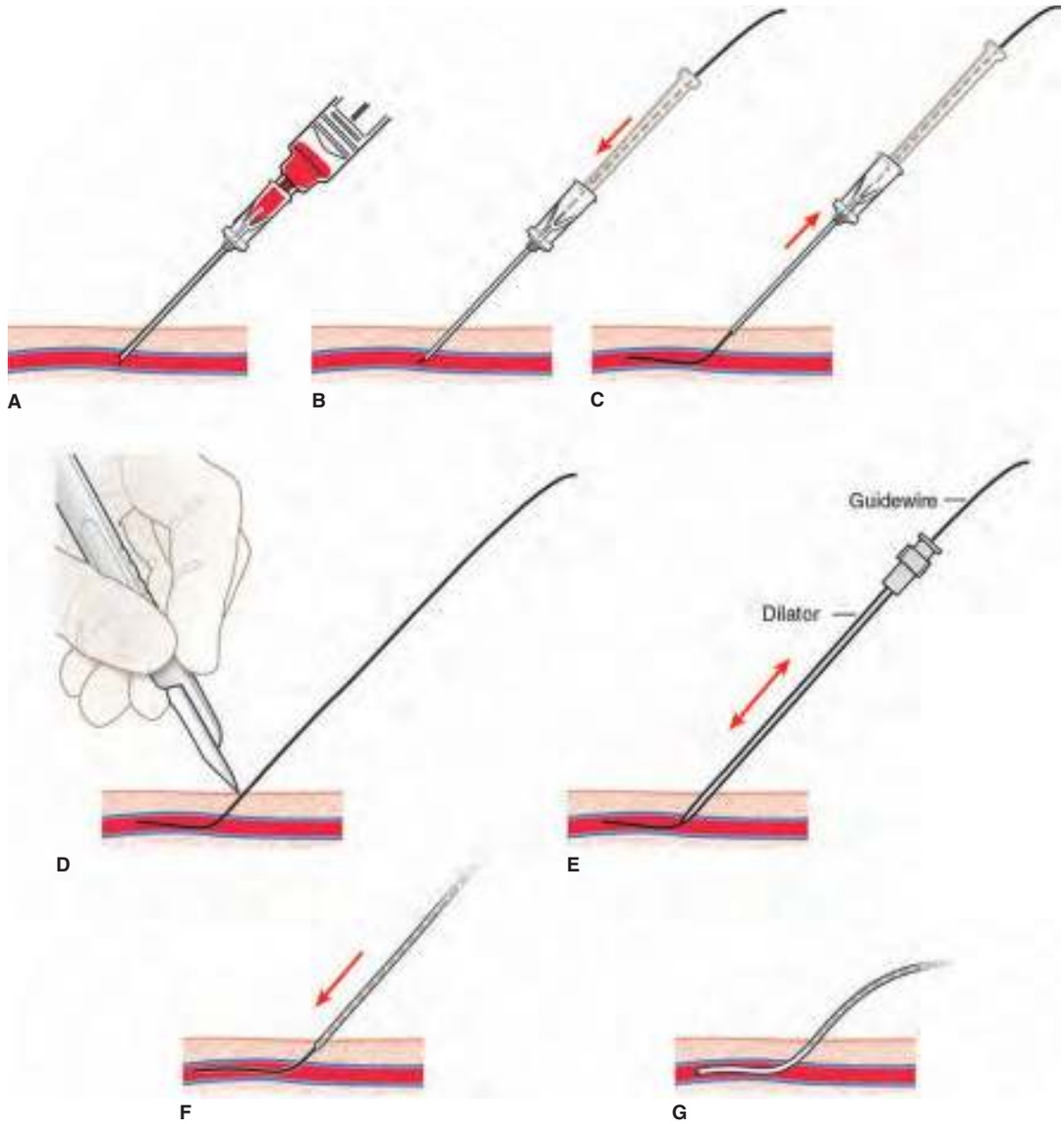


FIGURE 63-10. The Seldinger technique. ***A.*** The vein is punctured by the introducer needle, and blood is aspirated. ***B.*** The syringe has been removed. The guidewire is inserted through the introducer needle and into the vein. ***C.*** The introducer needle and guidewire sleeve are withdrawn over the guidewire. ***D.*** The skin puncture site is enlarged. ***E.*** The dilator is advanced over the guidewire until the hub is against the skin; then it is removed. ***F.*** The catheter is advanced over the guidewire and into the vein. ***G.*** The guidewire is withdrawn through the catheter.

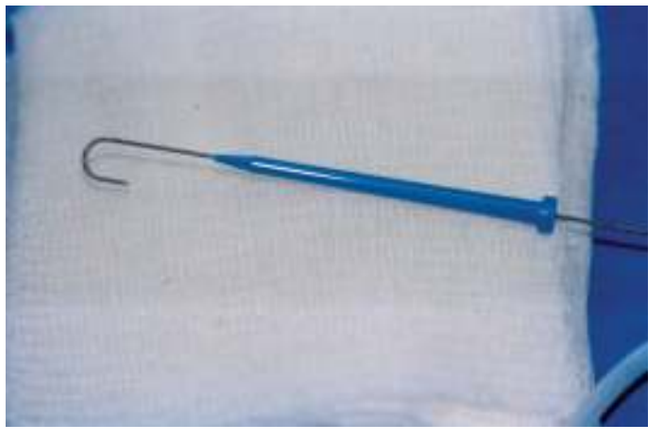
45° to 60° angle. Direct the introducer needle toward the ipsilateral nipple. **The internal jugular vein in an adult should be encountered within 3 to 5 cm of the skin.** If the vein is not encountered by 5 cm, withdraw the tip of the introducer needle to the subcutaneous space and redirect it slightly medially. The remainder of the procedure is as described for the central approach and in **Table 63-6**.

POSTERIOR APPROACH TO THE INTERNAL JUGULAR VEIN

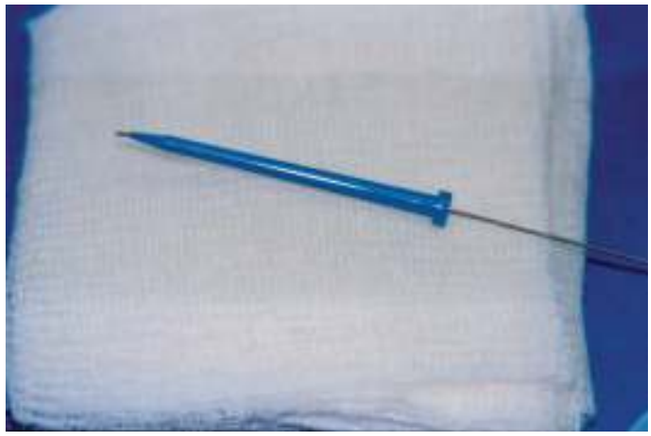
Enter the skin at the posterior edge of the sternocleidomastoid muscle, one-third of the way from the clavicle to the mastoid process (**Figure 63-6**). Alternatively, the point where the external jugular vein crosses the lateral border of the sternocleidomastoid muscle can be used. Direct the introducer needle under the sternocleidomastoid muscle at a 30° to 45° angle to the skin and toward the sternal notch. Place the index finger of the nondominant hand in the sternal notch to provide a landmark with the patient draped. **The internal jugular vein should be encountered within 5 cm from the skin. This approach is not recommended in children.** The remainder of the procedure is as described for the central approach and in **Table 63-6**.

NOVEL AND EMERGING APPROACHES TO THE INTERNAL JUGULAR VEIN

The sternocleidomastoid muscle and the carotid artery are the most commonly used landmarks for internal jugular vein catheterization. These landmarks can be difficult to palpate in certain patient populations. The sternocleidomastoid muscle can be obscure in unconscious or anesthetized patients and the carotid artery pulse is weak in hypotensive or arrhythmic patients. Other landmarks that are often more easily palpated may be ideal when classic external landmarks are difficult to identify.



A



B

C

FIGURE 63-11. Guidewire preparation. ***A.*** The plastic sleeve is retracted showing the "J" tip. ***B.*** The plastic sleeve is advanced to cover the guidewire tip and allows the guidewire to be threaded into the introducer needle. ***C.*** The sleeve is inserted into the hub of the introducer needle.

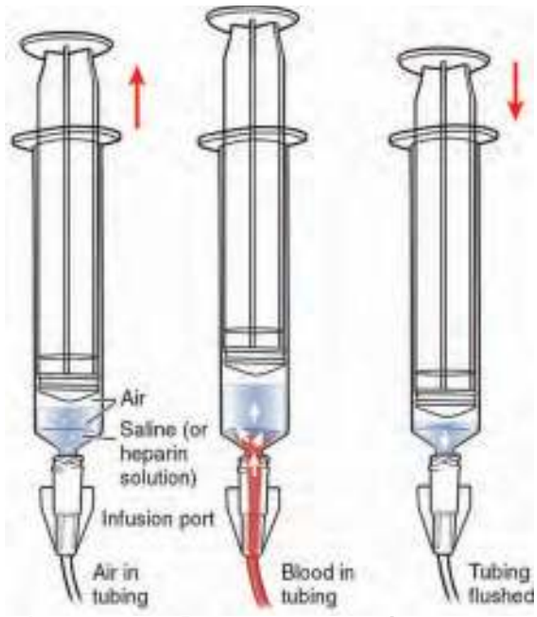


FIGURE 63-13. Aspiration and flushing of catheters. ***A.**** Any air in the lumen of the tubing is aspirated into the syringe of flush solution. The syringe must be held upright, as shown. ***B.*** Stop aspirating once all the air is removed from the catheter and blood begins to enter the syringe. ***C.*** Flush solution is injected until the lumen is filled and contains no blood. This usually requires 2 to 4 mL of flush solution.

A method using the cricoid cartilage and external jugular vein as landmarks resulted in a 99% success rate in right internal jugular vein catheterization, an average of 1.75 attempts, and no carotid artery puncture.[56] Respiratory jugular venodilation in mechanically ventilated patients could be favorably used as the primary landmark for right internal jugular vein catheterization.[57] A modified anterior approach in pediatric patients uses the cannulation needle inserted along the anterior border of the sternocleidomastoid muscle at the level of hyoid bone.[58] A peripheral IV catheter (i.e., angiocatheter) can be placed with or without a guidewire inserted in the angiocatheter and the Seldinger technique.[59-61] The guidewire in an angiocatheter is faster and safer than the traditional Seldinger technique with US guidance.[62] This may be useful in emergencies.

A

B

FIGURE 63-12. Straightening the "J" tip. ***A.*** Grasp the guidewire between the ring and small fingers and the palm. ***B.*** Apply traction using the thumb and index fingers, stretching the outer coil of the guidewire over the solid core to straighten the "J" tip.

TABLE 63-7 Comparison of Subclavian Vein Cannulation Routes

	Infraclavicular approach	Supraclavicular approach
Entry site	Just inferior to the clavicle at the midclavicular line	1 cm lateral to the clavicular head of the sternocleidomastoid muscle, 1 cm posterior to the clavicle
Needle orientation	Keep as close to the coronal plane as possible	Tip aimed 10° anterior to the coronal plane
Needle bevel and "J" wire directed	Medially and caudally	Medially
Aim toward	Just posterior to the sternal notch	Contralateral nipple, needle bisects angle formed by the clavicle and the clavicular head of the sternocleidomastoid muscle
Distance from skin to subclavian vein	3–4 cm	2–3 cm

SUBCLAVIAN VEIN CATHETERIZATION TECHNIQUES

The technique is identical to that described above for internal jugular vein cannulation except for the puncture site. Two techniques, infraclavicular and supraclavicular, are described below and summarized in **Table 63-7**.

INFRACLAVICULAR APPROACH TO THE SUBCLAVIAN VEIN

The infraclavicular approach to the subclavian vein is most often used. It is commonly thought to be easier to perform and less likely to result in a pneumothorax than the supraclavicular approach, although data for this belief are lacking in adult and pediatric populations.[63-66] Some Emergency Physicians prefer not to use a finder needle for infraclavicular subclavian vein cannulation as there is no danger of penetrating the carotid artery. This also makes as few needle passes near the pleura as possible to decrease the risk of an iatrogenic pneumothorax. Estimate the distance from the skin puncture site to the superior vena cava (i.e., the manubriosternal junction).

Several different skin entry sites are described in the literature. Some feel that the preferred entry site is 1 cm caudal to the junction of the medial and middle thirds of the clavicle. The subclavian vein lies just posterior to the clavicle at this site (**Figure 63-14**). The first rib lies between the pleural dome and the subclavian vein. Direct the introducer needle just superior and posterior to the suprasternal notch while staying as close to the frontal (i.e., coronal) plane as possible. The needle and syringe should be parallel to the bed (**Figure 63-14**). Place the nondominant index finger in the sternal notch to help guide placement (**Figure 63-14**).

Some Emergency Physicians prefer to enter the skin inferior to the clavicle at the deltopectoral groove, or the point just lateral to the midclavicular line along the inferior surface of the clavicle. This is the point where the skin may be maximally depressed. Direct the introducer needle parallel to the bed and toward the sternal notch. This entry site may make it easier to keep the introducer needle in the coronal plane. The distance before entering the subclavian vein is longer than in the preceding approach and the protection offered by the first rib is lost.

One additional landmark can be used to identify the skin puncture site. Palpate the bony tubercle, or protrusion, on the inferior surface of the clavicle and approximately one-third to one-half the length of the clavicle from the sternoclavicular joint. The advantage of this site is that it is a definitive landmark and avoids approximating distances, as described for the other sites above. Insert the introducer needle parallel to the bed and aimed just posterior to the sternal notch.

Orient the bevel of the introducer needle caudally, as with the "J" in the guidewire (**Figure 63-15**).[67] This position will allow the guidewire to enter the innominate vein and superior vena cava rather than being directed upward into the internal jugular vein or across to the contralateral subclavian vein (**Figure 63-15**).[67] Manual

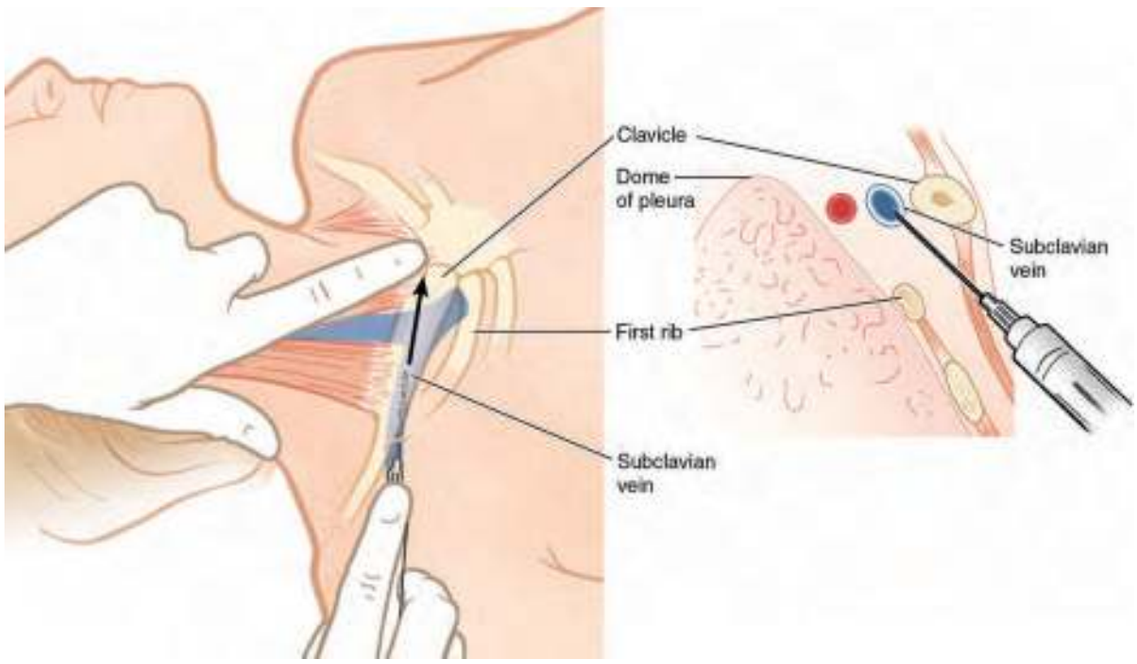


FIGURE 63-14. Infraclavicular approach to subclavian vein cannulation. ***A.*** Frontal (oblique) view of the procedure. ***B.*** Sagittal section through the medial third of the clavicle. Note the proximity of the pleura and subclavian artery.

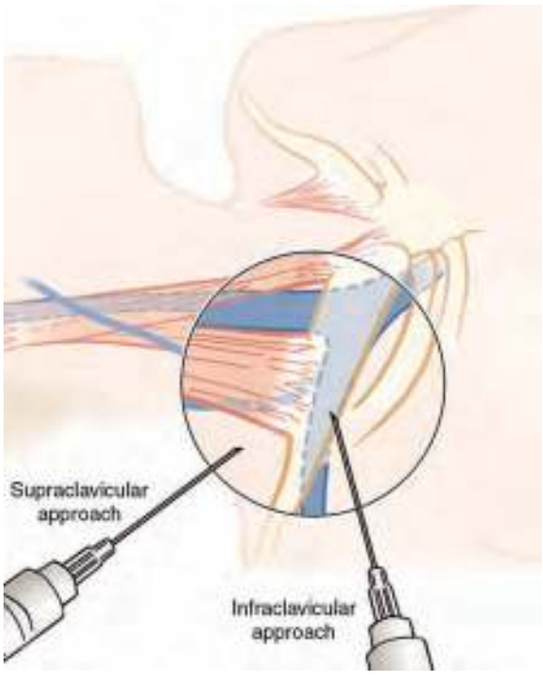


FIGURE 63-15. Introducer needle bevel orientation for subclavian vein cannulation. Varying the orientation of the introducer needle bevel for infraclavicular and supraclavicular techniques helps guide the "J"-shaped guidewire into the superior vena cava.

occlusion of the internal jugular vein by compression just above to the clavicle prevents misplacement of the catheter in the internal jugular vein.[68] The Seldinger technique for catheter insertion is otherwise the same as previously described for internal jugular vein cannulation. Aspiration of bright red blood under pressure indicates subclavian artery puncture, which will be incompressible. Remove the introducer needle and observe the patient for signs of significant hemorrhage over the next several hours. Aspiration of air indicates penetration of the pleura. Observation with serial chest radiographs for at least 6 to 24 hours is essential to evaluate the size of the resulting pneumothorax.

SUPRACLAVICULAR APPROACH TO THE SUBCLAVIAN VEIN

The supraclavicular approach offers some distinct advantages.[69-71] The supraclavicular subclavian vein is closer to the skin. The route from a right-sided skin puncture site to the superior vena cava is more direct. It allows easier access to the superior vena cava while avoiding the hazards of a left-sided puncture (i.e., the thoracic duct). The skin entry site is more accessible during CPR and requires less interruption of external chest compressions.[72] With experience, the complication rate for the supraclavicular approach is probably lower than that for the infraclavicular approach.[63,69,73]

Estimate the distance from the skin puncture site to the superior vena cava to guide the catheter insertion depth. The skin is entered at a point 1 cm lateral to the lateral border of the clavicular head of the sternocleidomastoid muscle and 1 cm superior to the clavicle (**Figure 63-16**).[74] The introducer needle should bisect the angle formed by the clavicle and the lateral border of the sternocleidomastoid muscle (**Figure 63-16*A***). Direct the introducer needle toward the contralateral nipple or a point just superior and posterior to the sternal notch. Orient the introducer needle bevel medially (**Figure 63-15**).[67] The subclavian vein should be entered within 2 to 3 cm in an adult. The length of catheter inserted will be 2 to 4 cm less than that for the infraclavicular approach.

Alternative skin entry sites and approaches have been described. Enter the skin 1 cm medially and 1 cm superiorly to the midpoint of the clavicle with the introducer needle directed toward the ipsilateral sternoclavicular joint.[75] The skin can be entered just posterior to the clavicle, at the junction of the medial and middle third of the

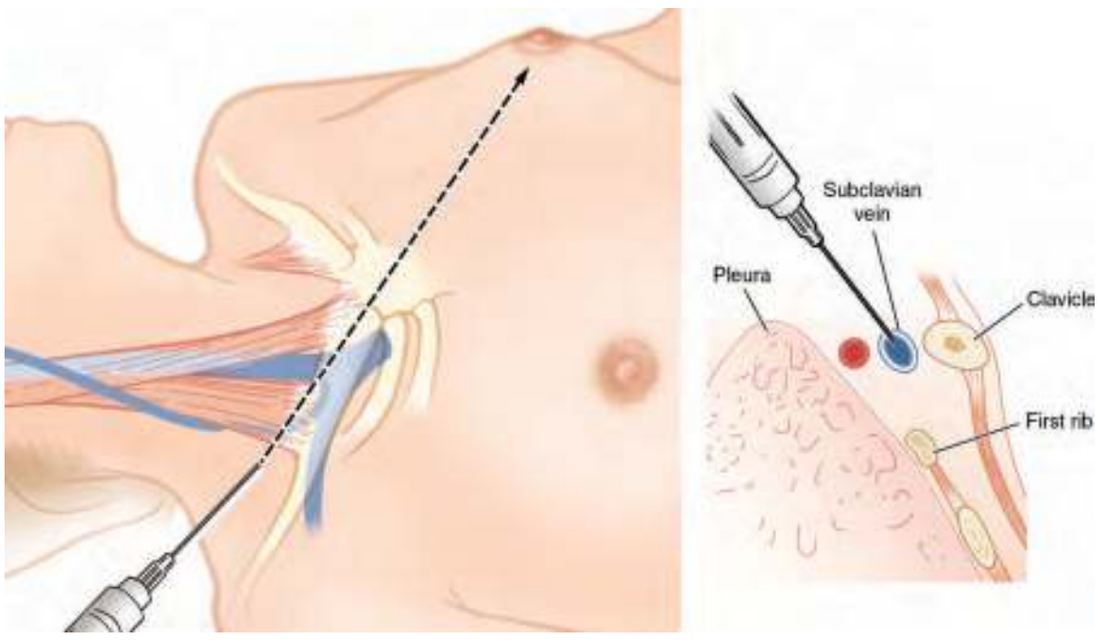


FIGURE 63-16. Supraclavicular approach to subclavian vein cannulation. ***A.*** The insertion point is 1 cm superior to the clavicle and 1 cm lateral to the border of the sternocleidomastoid muscle. Direct the introducer needle tip at a 45° angle to the transverse and sagittal planes and slightly anterior toward the contralateral nipple. ***B.*** Sagittal section through the medial third of the clavicle. Note that the introducer needle track must be directed anteriorly to avoid the subclavian artery and the dome of the pleura.

clavicle, with the introducer needle directed toward the ipsilateral sternoclavicular joint and parallel to the coronal plane.[76] This last approach is probably the simplest, although the study cited was performed on cadavers rather than live patients.

AXILLARY VEIN CATHETERIZATION TECHNIQUE

The axillary vein must be accessed using US guidance and not blindly using landmarks. Place the patient supine with their arm abducted in a comfortable position. One US study demonstrated no benefit of arm abduction in increasing axillary vein size or decreasing arteriovenous overlap.[21] There is no change in size of the axillary vein with the Valsalva maneuver or the Trendelenburg position.[77] Locate the axillary vein using a high-frequency (i.e., 5 to 10 MHz) US transducer. Place the US transducer, with the marker pointing cephalad, just below the clavicle at approximately its middle third. Identify the axillary artery, axillary vein, and the pleura to obtain proper orientation. Manual compression and color flow Doppler can be used to differentiate between the vein and the artery. Follow the axillary vein laterally on the chest until its overlap with the artery is minimized and the pleura is out of the view of the transducer. Rotate the US transducer into a parallel orientation in respect to the axillary vein with the transducer marker toward you to obtain a long axis view of the axillary vein. Gently pivot the US transducer in a cephalad and caudal direction to clearly differentiate the axillary vein from the axillary artery. Slight movements of the transducer will alter the ultrasound image between the vein and artery. It is recommended to stabilize the US transducer by resting the distal forearm and wrist against the anterior chest. Note the depth of the vein to ensure that the central venous catheter needle will be able to puncture the vessel. The remaining details of the technique are as described in Chapter 64 on US-Guided Vascular Access.

FEMORAL VEIN CATHETERIZATION TECHNIQUE

This technique is often performed blind using landmarks. It can also be performed under US guidance and serves as another viable option for central venous catheterization during CPR and trauma resuscitations.[78,79]

The use of an electrocardiogram monitor is still recommended even though the short guidewire may not reach the heart. Care must be taken if the patient has a preexisting left bundle branch block, as complete heart block may result if the guidewire or catheter enters the right ventricle.[39] Premeasuring from the insertion site to the xiphoid process will give the maximum depth of catheter insertion.

The introducer needle should enter the skin 2 to 4 cm inferior to the midpoint of the inguinal ligament and 1 cm medial to the femoral artery pulse (**Figure 63-17**). The introducer needle should enter the skin 1 to 2 cm inferior to the inguinal ligament and 0.5 cm medial to the femoral artery pulse in an infant or young child. The cannulation technique is as described previously for the internal jugular vein.

Two site-specific considerations deserve mention. The use of a finder needle is unnecessary since there are no vital structures in the area other than the femoral artery that is compressible if it is punctured. The introducer needle is directed at a 45° to 60° angle to the skin and parallel to the long axis of the thigh. Shallower angles may be necessary in very small and thin patients. **Use caution to avoid puncturing the posterior wall of the vein above the inguinal ligament as this can result in a retroperitoneal hemorrhage.**

ALTERNATIVE TECHNIQUES

USE OF THE SELDINGER-HUB INTRODUCER CATHETER

Some central venous access kits include a catheter-over-the-needle with a tapered hub that can be used in place of the thin-walled introducer needle. This technique has the advantage of allowing the introducer catheter to remain in place while venous placement is verified. It provides less likelihood of the vein being lost as the aspiration syringe is removed and the guidewire advanced. A guidewire advanced through the introducer catheter cannot become sheared off as when it is inserted through the needle.

The vein is entered with the catheter-over-the-needle assembly attached to an aspirating syringe as described previously. Once the flashback of blood is obtained, advance the catheter-over-the-needle 2 mm further into the vein. This will ensure that the tip of the introducer catheter is within the vein. Hold the hub of the needle securely. Advance the catheter into the vein until its hub is against

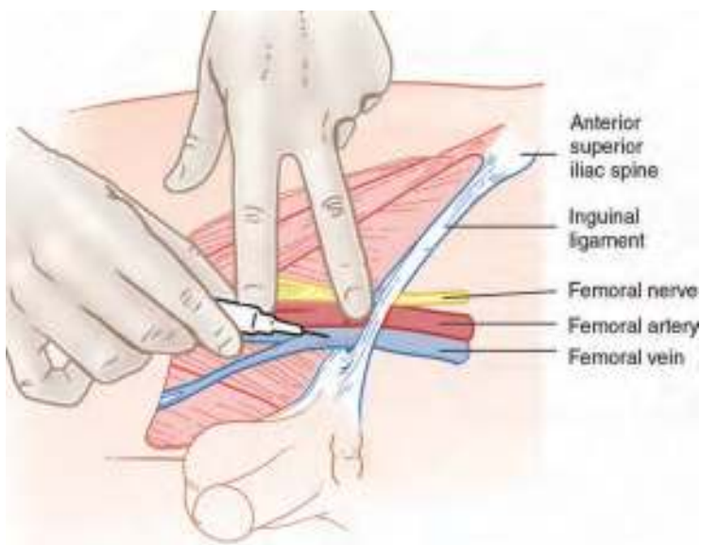


FIGURE 63-17. Femoral vein cannulation. The skin puncture site is 1 cm medial to the femoral artery pulse and 2 to 4 cm inferior to the inguinal ligament. Direct the introducer needle posteriorly at a 45° to 60° angle while aspirating.

the skin. Withdraw the needle. If necessary, the introducer catheter may be attached to a pressure transducer and the venous waveform verified to confirm venous rather than arterial placement. Blood gas measurements may also be performed. Advance the guidewire through the introducer catheter and into the vein. The remainder of the procedure is as previously described.

MULTIPLE-LUMEN CATHETERS

Remove the cap from the distal port's injection hub prior to skin puncture. It is usually marked "distal." The other lumens may be flushed with saline or heparin solution and recapped or left capped and flushed later (**Figure 63-13**). **Heparin concentrations no higher than 100 U/mL should be used to avoid temporarily anticoagulating the patient.**[80]

The introducer needle and guidewire are inserted as described previously. Place the multiple-lumen catheter tip over the guidewire. Advance the catheter until the guidewire emerges from the distal port hub (**Figure 63-18**). Insert the catheter to the desired depth. Remove the guidewire. Flush the distal lumen and connect it to the desired infusion. Aspirate and flush the other lumens with the desired solution if not done previously (**Figure 63-13**).

PERCUTANEOUS INTRODUCER SHEATH (CORDIS)

The insertion technique differs slightly from those described above (**Figure 63-19**). Locate the vein, insert the needle followed by the guidewire, and remove the needle, leaving the guidewire in place as described previously. Insert the plastic dilator into the lumen of the sheath. The entire assembly must be advanced over the guidewire as a unit rather than using separate dilation and insertion steps (**Figure 63-19*C***). A correspondingly larger skin nick must be made with the scalpel, since the sheath is usually of larger diameter than a catheter. Advance the dilator-sheath unit over the guidewire (**Figure 63-19*C***) and into the vein (**Figure 63-19*D***). A twisting motion may aid in its advancement. Continue to advance the unit until the hub of the sheath is against the skin (**Figure 63-19*E***). Remove the guidewire and dilator as a unit (**Figure 63-19*E***). The remainder of the procedure is as described previously.

ULTRASOUND-GUIDED CENTRAL VENOUS ACCESS

The use of bedside US to guide central venous access is becoming more common. The availability of US, its low cost, portable units, and training during residency are making its use standard procedure for central venous line placement. Higher procedural quality, consistency, and lower complication rates support its increasing use. Refer to Chapter 64 for the complete details of US-guided vascular access.

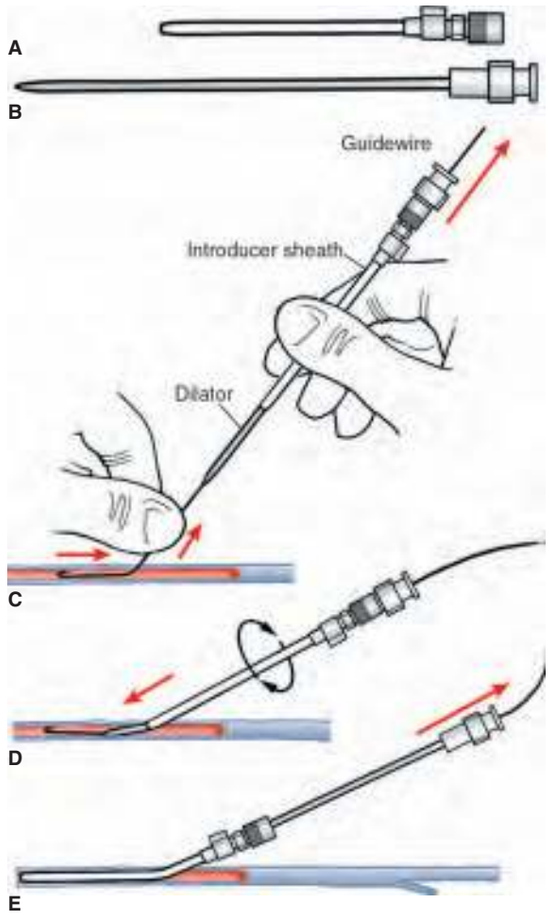


FIGURE 63-19. Inserting an introducer sheath. ***A.*** The sheath. ***B.*** The dilator. ***C.*** The dilator is inserted into the sheath and the unit is threaded over the guidewire. ***D.*** Advance the unit over the guidewire and into the vein using a twisting motion. ***E.*** The dilator and guidewire are removed as a unit, leaving the sheath in place.

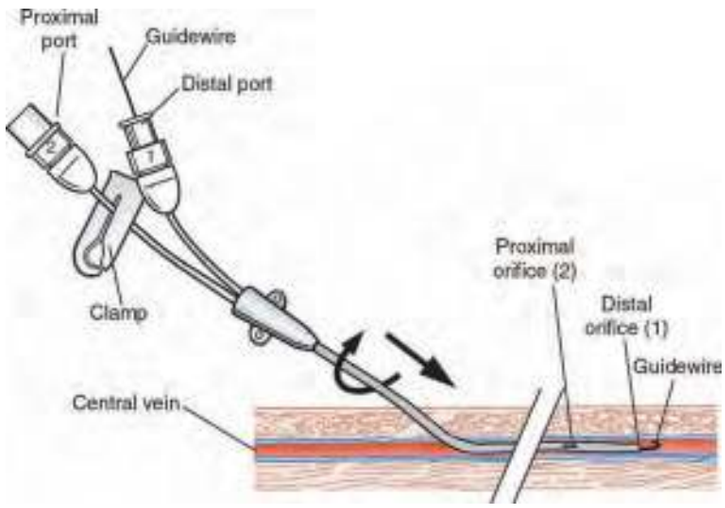


FIGURE 63-18. Inserting a multiple-lumen catheter. The guidewire exits through the uncapped distal port. The proximal port(s) must be clamped or capped to prevent air embolism.

PEDIATRIC CONSIDERATIONS

The anterior or central approach to the internal jugular vein is preferred for children. Appropriate catheter sizes and lengths are shown in **Table 63-3**. The child must be sedated and immobilized prior to attempts at cannulation of the internal jugular or subclavian vein. The femoral vein is the vein of choice if central venous access is needed in a combative child who cannot be completely restrained. The patient need not be in Trendelenburg position, the consequences of a misdirected needle are less severe, and the procedure is less threatening as the face is not draped. A shallower angle of skin entry than in an adult is necessary to access the femoral vein. Enter the skin 1 to 2 cm inferior to the inguinal ligament and 0.5 mm medial to the femoral artery. Subclavian vein access in the small child can be difficult due to the anatomic relationships of the vessels as previously described. Despite this, the subclavian vein and the femoral vein can be safely and effectively accessed in children less than 1 year of age. The use of the frog leg position decreases the overlap of femoral vessels.[28,30]

Several researchers evaluated methods of the depth of central venous lines in children. The size of the catheter is listed in **Table 63-8**. It can be inserted into the carina.[81,82] The problem with this is that a chest radiograph is needed after the catheter is inserted. It was determined that the distance to the carina can be calculated.[82] The sternal head of the right clavicle was labeled "point A." The midpoint of the perpendicular line from "point A" to the line connecting the patient's nipples was labeled "point B." The insertion point was labeled as "point I." The distance to insert the catheter was determined by the following formula: (I to A, in cm) + (A to B, in cm) – 0.5. This formula determined the correct catheter length to insert 95% of the time.

Others looked at the distance to insert the catheter. Insertion depth was calculated using the patient's height in centimeters.[83] If the patient's height was ≤ 100 cm, insertion depth (cm) = [height (cm) ÷ 10] – 1. If the patient's height was > 100 cm, insertion depth (cm) = [height (cm) ÷ 10] – 2. These formulae resulted in the catheter being at the superior vena cava–right atrial junction 97% of the time.[83] The left internal jugular vein insertion depth (in cm) was calculated from 0.07 × height (cm), and the left subclavian insertion depth (in cm) was calculated as 0.08 × height (cm).[84] This predicted 98.5% of correctly located catheters. Another formula was calculated for patients between the height of 40 and 140 cm using the right internal jugular vein.[85] They correctly located the catheter position in 97.5% of patients using the formula 1.7 + (0.7 × height in cm).

Methods were developed to increase the success of cannulation in children. The use of US increased the likelihood of successful placement and at the correct depth.[86] The central line can be inserted through the external jugular vein with good success rates.[87]

ASSESSMENT

Examine the patient. Examine the lung fields carefully to exclude a significant iatrogenic pneumothorax. Recheck the patient's vital signs frequently after the procedure. Obtain a portable anteroposterior chest radiograph to verify line tip placement in the superior vena cava and rule out an iatrogenic pneumothorax.[88,89] Check the catheter site for hematoma formation or hemorrhage along the dilated catheter track. Control any hemorrhage with direct pressure.

US can be used to evaluate positioning. It can be used to evaluate guidewire position prior to dilation and insertion of the catheter.[90,91] This is helpful if the guidewire is intraarterial so it is not dilated. US can be used after insertion to determine the presence or absence of an iatrogenic pneumothorax.[92] Detection of an anterior pneumothorax in a supine patient has been shown to be more sensitive using US than plain radiographs.[92-94] **The use of US does not negate the need to determine catheter location by plain radiographs, even though there is a growing base of literature that demonstrates the ability of US to detect the catheter tip location.**[88] US may be a useful tool when postprocedural chest radiographs are delayed or the patient suddenly decompensates during or immediately after central line placement. US can be used with or without bubbles during a saline flush to detect the flow out of the catheter tip and into the vessel.[92,95,96]

Check the function of the catheter by aspiration and infusion through all ports. A proximal lumen may be extravascular if it fails to aspirate blood easily. A catheter may be exchanged over a guidewire if the distal tip of the catheter is intravascular. Do not attempt to advance the catheter once the guidewire has been removed.

Check the position of the catheter tip on the chest radiograph.[96,97] The catheter must not be in the heart due to the risk that erosion through the thin right atrial wall will result in a pericardial hemorrhage and tamponade.[98,99] Landmarks for an internal jugular or subclavian vein catheter tip include above the level of the carina, above the azygos vein, at the level of the right tracheobronchial angle, and at/above the manubriosternal junction. The tip of the catheter should be parallel to the vein to prevent erosion through the wall of the vein. The catheter may be used for IV infusion if it crosses over to the opposite subclavian vein and the patient cannot tolerate an attempt at repositioning. **Lines placed from the subclavian vein into the jugular system must be replaced. Catheters in the right atrium must be pulled back immediately to prevent any arrhythmias and perforation of the myocardium.**

It is important to ensure that the catheter is within a vein and not an artery. The aspiration of dark blood and absence of pulsatile flow from the syringe hub at the time of line insertion confirms its located within a vein. Confirmation of line placement can be accomplished by US (Chapter 64), blood gas measurement, and/or pressure transduction. An arterial line monitoring set-up can be attached to the catheter to measure intravascular pressures but can be time consuming and expensive.

One alternative mode of pressure transduction is the Compass Vascular Access pressure measurement device (Mirador Biomedical, Seattle, WA).[100] This disposable, single patient use, digital manometer attaches between the syringe and introducer needle to rapidly provide a digital pressure measurement of the vasculature at the location of the needle tip (**Figure 63-20**). It can be used to provide pressure measurements while the guidewire is being inserted through the needle.

Most femoral vein catheters can be fully inserted. Premeasurement is recommended to make sure that the catheter tip will not reach the right atrium. Obtain postprocedural abdominal and chest radiographs if there is any doubt about the catheter position. The tip of the catheter must be at or below the xiphoid process of the sternum. Reassess the distal neurovascular status of the lower extremity after line placement.

TABLE 63-8 Pediatric Catheter Sizes

Age (years)	Internal jugular vein	Subclavian vein	Femoral vein
Birth–0.5	3 French	3 French	3 French
0.5–2	3 French	3 French	3–4 French
3–6	4 French	4 French	4–5 French
7–12	4–5 French	4–5 French	5–8 French

AFTERCARE

The catheter must be sutured in place to prevent malpositioning of the line (**Figure 63-21**).[101] Tie a surgeon's knot at the skin and then secure the suture to the hole(s) provided in the catheter wings. The

A

B

FIGURE 63-20. The Compass Vascular Access device. ***A.*** The devices. ***B.*** The device placed between a needle and syringe to measure the pressure during central venous access. (Photos courtesy of Centurion.)

straight needle contained within most central venous access kits can be difficult to use and poses a needle-stick risk (**Figure 63-22**). One option is to use nylon suture on a curved needle and a laceration repair kit. This requires additional equipment at an additional cost.

FIGURE 63-22. Using a straight needle with the finger can result in a needle stick. (Used with permission from reference 102.)

Another option is to use a needle cap or syringe contained within the kit to protect against the advancing straight needle puncturing a finger (**Figures 63-23, 63-24, and 63-25**).[102,103] Use the cap to apply counter pressure against the skin as the straight needle tip exits the skin. A catheter clamp is often provided in the central line kit for longer catheters. It too should be sutured in place. The clamp holds the catheter in place by friction. It is not a guarantee that the catheter will not move. **Catheter depth should be checked daily by inspection and frequent chest radiographs.** Movement of the patient's head and neck may move the tip of the internal jugular vein catheter by as much as 4 cm.[104]

An alternate method to secure the central venous catheter is using a SecureAcath (Interrad Medical, Plymouth, MN). This device

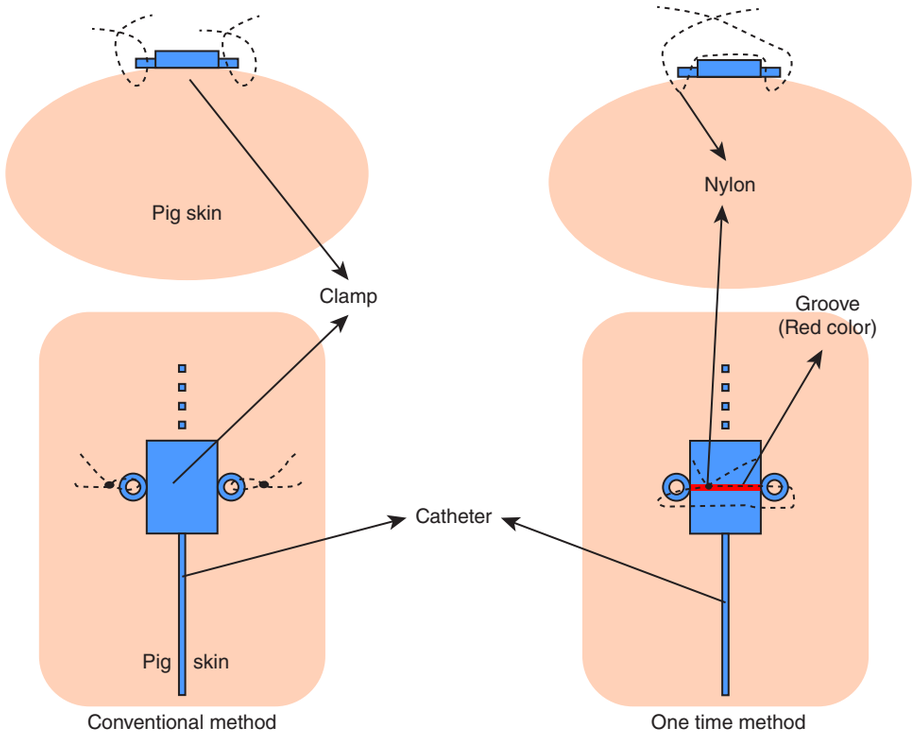
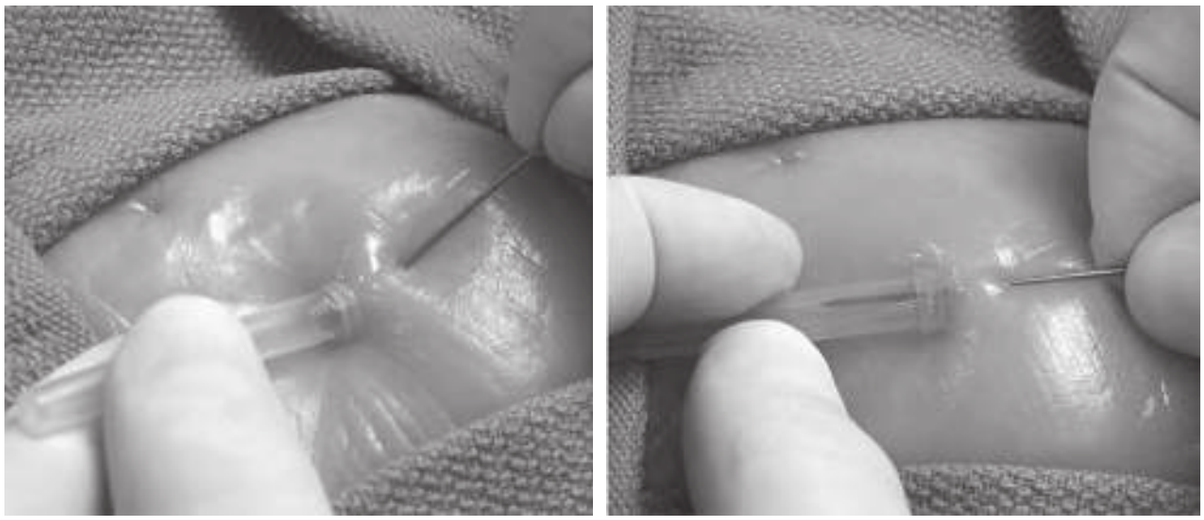
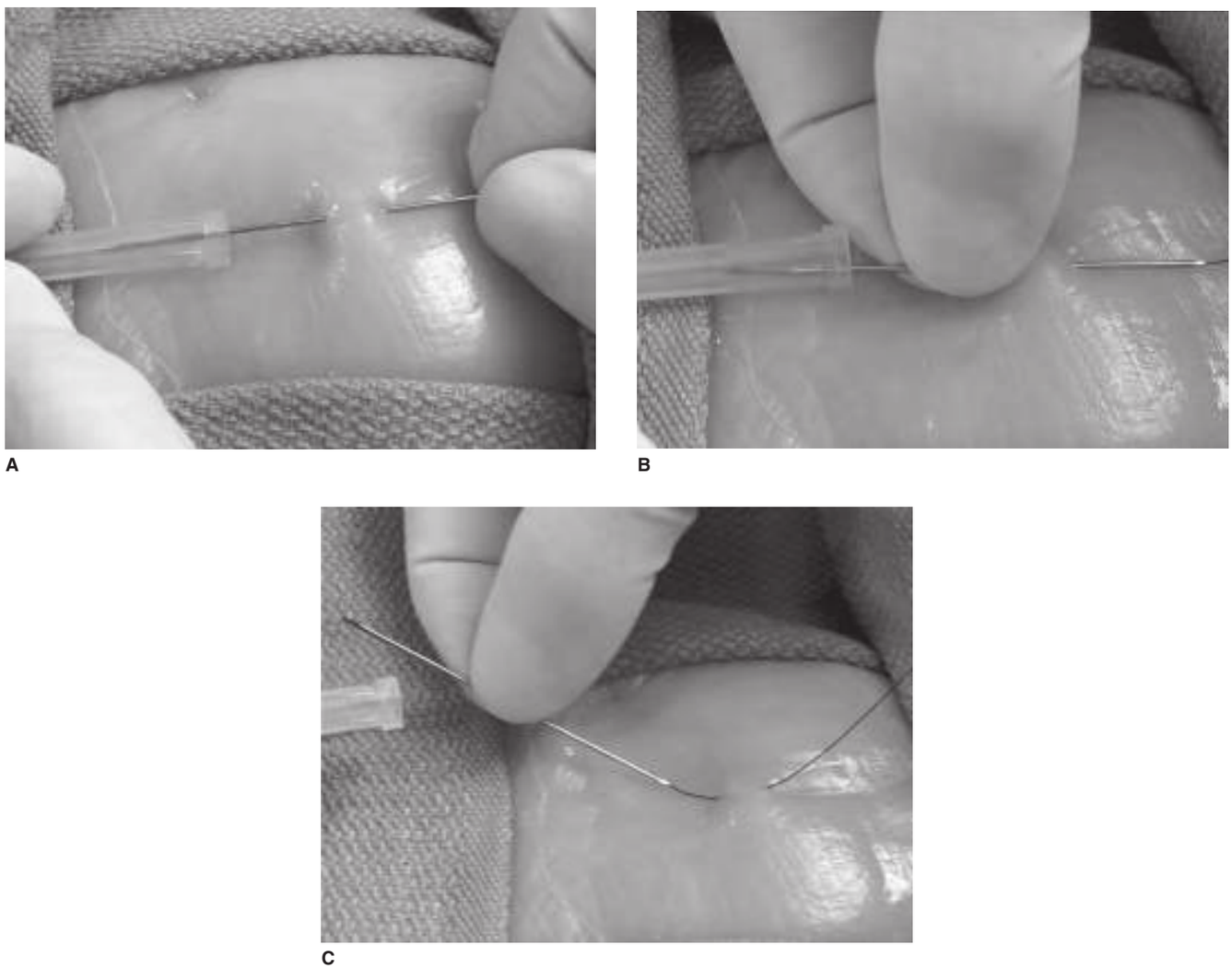


FIGURE 63-21. Securing a central venous line with suture. (Used with permission from reference 101.)



A B

FIGURE 63-23. The use of a needle cap instead of a finger. ***A.*** The cap is used to provide counter pressure instead of a finger. ***B.*** The needle exits the skin and enters the cap. (Used with permission from reference 102.)



A B C

FIGURE 63-24. The use of a needle cap instead of a finger. ***A.*** The sharp end of the needle is enclosed within the cap. ***B.*** The straight needle is grasped in a safe spot without the risk of a needlestick. ***C.*** The straight needle is pulled through the skin. (Used with permission from reference 102.)

A

B

FIGURE 63-25. The use of a syringe instead of a finger. ***A.*** Using a straight needle with the finger can result in a needlestick. ***B.*** The syringe is used to provide counter pressure instead of a finger. The needle exits the skin and enters the syringe. (Used with permission from reference 103.)

attaches to the skin-catheter junction (**Figure 63-26**). The device minimizes catheter movement and prevents needlesticks. It is quick to use, no sutures are required, and the catheter can be repositioned if inserted too deep. The SecureAcath allows cleaning the access site and keeps the catheter securely positioned during the cleaning.

Introducer sheaths have large lumens and present a significant risk of causing an air embolism. Cap the main lumen if it is not being used for an infusion. Any built-in diaphragm is not a reliable means of preventing an air embolism.[105] **Do not use the dilator as an occluder or infusion port, as the stiff plastic can easily erode through the wall of the vein.** An occlusive dressing can be used if no occluder is available.

Check the skin puncture site regularly for signs of infection. Cellulitis or purulent drainage requires a new central venous line at another site. Restrain any patient who is uncooperative to prevent inadvertent removal of the central line.

While the short-term infection rate of femoral lines compares favorably with that of other central venous line location sites, some precautions are necessary to prevent soilage.[25,106] Consider the judicious use of bladder catheterization in patients incontinent of urine and rectal tubes in patients with loose stools. Patients with

A

B

FIGURE 63-26. The SecurAcath. ***A.*** The device. ***B.*** The device on a central line. (Photos courtesy of Interrad Medical.)

percutaneous femoral vein catheters must be confined to bed to prevent catheter dislodgment and hemorrhage around the catheter. **Frequent assessment for venous thrombosis in the lower extremity is essential.**[107]

REMOVAL OF THE CENTRAL VENOUS CATHETER

It has been, and sometimes still is, routine policy in some institutions to change all central venous lines placed in the Emergency Department when the patient arrives in the Intensive Care Unit. It was believed that these lines placed in the Emergency Department were "dirty" and at a higher risk of infection. This practice results in the additional time, cost, associated patient discomfort, and the potential for complications associated with a repeat procedure. The infection rate of central venous lines placed in the Emergency Department using aseptic technique were no different than those placed in the Intensive Care Unit.[108]

Place the patient in the Trendelenburg position when removing a central venous catheter from the internal jugular or subclavian vein. Place the patient supine to remove a femoral vein catheter. Remove the dressing overlying the skin puncture site. Cut the suture securing the catheter to the skin. Ask the patient to exhale and hold their breath. Briskly remove the catheter and cover the puncture site with a gauze dressing. The track from the skin surface to the vein can be a source of a fatal venous air embolism.[109] Apply an occlusive dressing to the site for the first 1 to 2 days after the catheter has been removed. Observe the skin puncture site for signs of an infection twice a day for 48 hours.

COMPLICATIONS

Central line complications usually cause pain to the patient (i.e., from the complication or the resolution procedures). Central line complications are terrifying for patients, bothersome to the Emergency Physician, and costly for the facility. **Table 63-9** lists the risk factors for central venous line complications. Complications can be minimized by following some general rules (**Table 63-10**)

Mechanical complications can occur.[110] The needle and/or guidewire may puncture the lateral or posterior wall of the vein. This can result in a hematoma formation, arterial puncture, arterial cannulation, and an arteriovenous fistula.[111] The catheter can cause atrial thrombi that may become infected.[112-114] A catheter in the heart can result in a perforation and cardiac tamponade.[89,115-117] A thrombus can form on the catheter that may eventually embolize to the lungs.[118-120] Catheters that abut the vein wall can erode through with time.[121] The catheter tip can be malpositioned in a vein that connects to the vena cava.[122]

The use of US guidance reduces but does not eliminate these complications.[123-128] The guidewire or catheter can become lost, break, fragment, or become knotted intravascularly.[129-131] The guidewire can become a venous embolus if it completely enters the vein.[132-138] The tip of the catheter can cause arrhythmias if it is located within the heart.[139-141]

The guidewire can become caught on an inferior vena cava filter.[142-145] This commonly occurs with guidewires inserted through a femoral vein. It can happen from internal jugular and subclavian vein guidewires that are inserted too far. An inferior vena cava filter is the only reason to consider using the straight end of the guidewire. **Stop moving the guidewire if any resistance is felt. Do not use any force to remove the guidewire.** Tape the guidewire to the patient so it does not further enter the patient and obtain an abdominal radiograph. Try to advance the entrapped guidewire to disengage it from the filter. Consult an Interventional Radiologist for removal under fluoroscopy. Overinsertion of the guidewire can dislodge an inferior vena cava filter. Avoid complications with inferior vena cava filters by inserting the guidewire a small distance (e.g., 10 cm)

Antibiotic-coated catheters have been developed and used.[146-148] Their use is controversial. The catheters reduce line infections and sepsis. The evidence in children is mostly extrapolated from studies of adults. There is a question regarding whether they result in antibiotic-resistant organisms. They can expose patients to unnecessary antibiotics, can cause allergic reactions, and are more expensive to use. Leave these catheters for use by consultants to insert in the Intensive Care Unit, hospital floor, or as an outpatient for long-term therapy. The infection rate of uncoated catheters is lowest in subclavian lines followed by femoral and internal jugular lines.[149]

TABLE 63-9 Risk Factors for Complications of Central Venous Lines

Coagulopathy
Hypovolemia
Inexperience of physician
Large catheter size
Number of needle punctures
Obesity
Operations at site
Previous central lines at site
Radiation therapy at site
Small size children
Severe dehydration
Unsuccessful attempts at site

TABLE 63-10 Ways to Avoid Central Venous Line Complications

Access central line catheters sterilely and with sterile devices
Adhere to aseptic technique
Always use full sterile barrier precautions
Apply a sterile dressing to the skin puncture site
Avoid the central line placement
Attempt a US-guided peripheral IV line
Cleanse the central line port immediately before use
Perform skin antisepsis and allow this to dry before starting the procedure
Replace soiled dressings at the skin insertion site using aseptic technique
Remove the central line as soon as it is no longer needed
US-guided peripheral IV placement can eliminate the need for a central line
Use a central line insertion checklist to make fewer mistakes
Use an insertion checklist to allow ED personnel to adhere to the proper care techniques
Use a central line only when indicated
Use a central line when alternative options have been unsuccessful
Use hand hygiene before the procedure
Use US to place a central line to decrease complications
Select the optimal insertion site

INTERNAL JUGULAR VEIN CATHETERIZATION

Internal jugular venous access has a myriad of potential complications.[110] Some of them are listed above. Infection can be either local at the site of insertion or systemic (i.e., bacteremia and sepsis). A pneumothorax can occur during line placement. A hemothorax may be life-threatening, especially if a venopleural fistula is created. A chylothorax occurs if the thoracic duct is lacerated. Airway compromise can occur due to the formation of a hematoma and compression of the airway. An air embolism can occur if the catheter lumens are left open to the air during insertion, if connections loosen and separate later, or if air enters the system while tube manometry is being used for line confirmation.[150,151] Right ventricular irritation from the catheter tip can cause cardiac dysrhythmias. Puncture of the right atrial wall can lead to pericardial tamponade and death. Malposition of the needle and guidewire can rupture the cuff of an endotracheal tube.[152] The guidewire can become entrapped and require surgical or interventional radiology removal. Embolization of the guidewire or catheter parts occurs with improper use of the equipment. Anaphylactic reactions to antibiotic-impregnated catheters have been reported.[153] The cardiac monitors should be observed during the procedure to prevent

the death of a critically ill patient from being unnoticed while the catheter is being inserted. Thrombosis of the catheter or vein may lead to pulmonary embolism.

Carotid artery puncture can occur while accessing the vein. It may be complicated by a stroke if the blood supply to the brain is interrupted or if a plaque embolizes. Devastating complications may result if arterial puncture is unrecognized and a large-bore dilator and/or catheter is then inserted arterially. **Do not remove the catheter if arterial dilation or cannulation accidentally occurs.** Consult a Vascular Surgeon for consideration of surgical or endovascular treatment to minimize risk of major complications (e.g., hemorrhage, major stroke, arteriovenous fistula).[154]

Many of these complications can be prevented or minimized with the use of US guidance. US guidance resulted in lower rates of arterial puncture, hematoma formation, hemothorax, pneumothorax, and infection.[155] Multiple smaller studies have shown similar results in favor of US guidance.[2,156-160] A comprehensive review published in 2015 reported a 71% reduction in overall complications with US guidance compared to a landmark technique.[161] There is no specific report of complication rates involving the thoracic duct, nerve injury, or thyroid injury. This is likely due to the extremely rare incidence of damage to these structures. **US guidance has been shown to significantly reduce complications and improve patient safety for internal jugular vein access.** Complications during catheterization occur in proportion to inexperience.[162] The most experienced person available should perform the procedure if the patient is unlikely to survive a mistake. The left jugular vein is smaller than the right and may account for more complications.[7]

SUBCLAVIAN VEIN CATHETERIZATION

Potential complications of subclavian vein cannulation are similar to those of internal jugular vein cannulation. There is minimal risk of carotid artery injury if the procedure is performed correctly. The pericardium, subclavian artery, or pulmonary vessels can be lacerated if the needle is advanced too deeply.[111,163-166] Malposition of the catheter tip, usually due to overinsertion of the catheter, is common. Lacerations of the thoracic duct can be avoided by performing the procedure on the right side, avoiding overpenetration with the introducer needle, and avoiding directing the needle too superiorly toward the junction of the subclavian vein and internal jugular vein.[167] Other complications (e.g., injury to the brachial plexus and phrenic nerve) are uncommon but possible.[111,168-170] They can be prevented by avoiding overinsertion of the needle during the procedure and avoiding needle paths superior and posterior to the subclavian vein.

A pneumothorax is a very real risk with subclavian vein catheterization.[164,171] The procedure should not be performed unless personnel are immediately available who can deal with this complication.[172] The risk of a pneumothorax is probably higher in obese patients who may have distorted anatomic landmarks, and a more acute angle is required to enter the subclavian vein. Patients with emphysema may have higher pleural domes and less pulmonary reserve in the event of a pneumothorax.

Subclavian vein cannulation results in fewer complications when performed with US guidance. Higher rates of hematoma formation, hemothorax, pneumothorax, brachial plexus injury, phrenic nerve injury, and cardiac tamponade are seen with the use of the landmark technique when compared to US guidance.[173] Multiple meta-analyses showed lower rates of arterial puncture, hematoma formation, and pneumothorax with US guidance.[174,175] Current evidence cannot elucidate whether the infraclavicular or supraclavicular approach to subclavian vein cannulation is safer.[176]

FEMORAL VEIN CATHETERIZATION

Deep venous thrombosis of the femoral and more distal veins is a recognized complication of femoral venous lines (**Figure 63-27**).[107,177-179] Inadvertent cannulation of the femoral artery may occur. This is particularly true during an episode of severe hypotension or cardiac arrest. Infusion of vasopressors into the artery may result in ischemic injury to the distal limb if an episode goes unrecognized.

Early studies suggested a higher risk of catheter-related bloodstream infections associated with the femoral vein cannulation compared to the internal jugular and subclavian sites. Recent studies demonstrate no significant difference in the infection rate between the three sites.[33,177,178,180]

US guidance for femoral vein cannulation does not currently have large studies with robust data in the literature. Anatomic visualization can be useful in multiple situations. There is a trend toward fewer adverse events with US guidance.[181,182] A meta-analysis showed better first-attempt and overall success at femoral vein cannulation with US guidance but did not demonstrate a difference in rates of complications.[175] The anatomic variation and overlap between the femoral artery and the femoral vein in normal patients brings to question the safety of blind or landmark-based venous cannulation.[29] US guidance may aid in identifying a very small femoral vein and/or significant arteriovenous overlap in a stable patient with a palpable

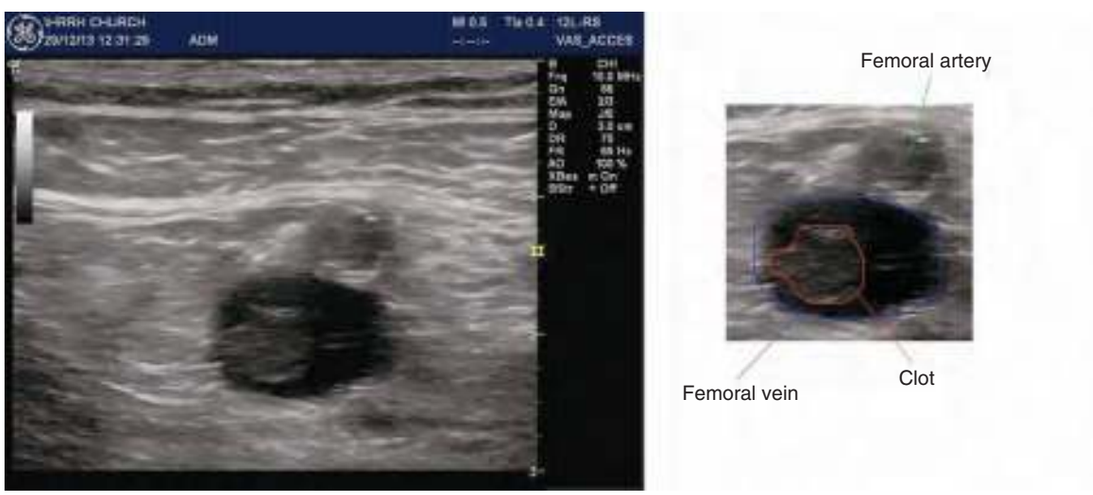


FIGURE 63-27. Ultrasound of a femoral deep venous thrombosis. (Photo courtesy of Lloyd Gordon, MD.)

pulse.[29] Correction with reverse Trendelenburg or proper leg positioning under US guidance may allow the Emergency Physician a better target for cannulation and reduce complications. Palpation of the femoral artery may not be feasible during periods of severe hypotension or cardiac arrest. This can lead to failed access or arterial cannulation. A higher rate of success and decreased rate of arterial cannulation was shown with US guidance during CPR.[183] US guidance for femoral vein cannulation may not yet be a standard practice but may be a useful technique that can reduce the complications.

SUMMARY

Central venous access is often necessary in critically ill patients and in those with poor peripheral veins. Mastery of these techniques is essential for anyone caring for acutely ill and unstable patients. While all approaches to the central circulation have acceptably low complication rates, they all carry real risks to the patient.[110] Be certain that there is no peripheral access alternative before placing a central venous line.

The internal jugular vein is a good choice for central venous access in nonambulatory patients. The right internal jugular vein provides easy access to the superior vena cava for monitoring and for infusion of solutions too concentrated or irritating for peripheral veins. This route poses a slightly lower risk of complications than the subclavian route when done with landmarks.[162,184]

The subclavian vein provides easy access to the central circulation. Subclavian vein catheters are more easily tolerated by awake and ambulatory patients than are internal jugular or femoral catheters. Subclavian vein cannulation does present very real risks to the patient that must be balanced against the need for the procedure. Subclavian vein access is the least preferred route in young children due to their small size, the proximity of the pleura, and the proximity of the subclavian artery.

Femoral vein cannulation is an essential emergency skill. It allows the easiest central venous access in most patients with the lowest risk of catastrophic immediate complications compared to jugular and subclavian access procedures.

US guidance for central venous access has rapidly become integrated into the practice of Emergency Medicine. Familiarity with US guidance has become a mandatory skill for the Emergency Physician when placing central venous lines.

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64 Ultrasound-Guided Vascular Access

Srikar Adhikari and Lori Stolz

INTRODUCTION

Central venous catheterization (CVC) is essential in the management of critically ill patients seen in the Emergency Department (ED). It allows for administration of vasoactive medications, central venous pressure monitoring, fluid resuscitation, and pacemaker placement. Complications (e.g., air embolism, arterial puncture, hematoma, hemothorax, and pneumothorax) have been reported to occur in 5% to 20% of patients.[1-3] Unsuccessful CVC has been reported in up to 20% of cases.[4,5] **CVC has traditionally been performed using surface anatomic landmarks as a guide to locate the veins.** CVC is not always successful using the landmark method due to anatomic variations or obscured landmarks. Factors (e.g., congenital deformities, dehydration, intravenous drug abuse, obesity, scarring, shock, and thromboses) can complicate the procedure. Ultrasound (US)-guided vascular access is widely supported in current medical practice. The use of US guidance for CVC has been endorsed by several medical societies and supported by numerous clinical trials in the literature. **US guidance has been shown to improve CVC success rates, reduce mean insertion attempts, and reduce placement failure rates.**[6-15] US guidance allows the

Emergency Physician to more precisely locate target vessels and provide real-time visualization of needle placement.

Peripheral venous access is more commonly performed in the ED than CVC. Patients lack easily located peripheral venous sites in many cases (e.g., chronic kidney disease, intravenous drug abuse, obesity, organ transplantation, and vascular disease). Obtaining peripheral intravenous (IV) access in these patients can be a challenge, even for the most experienced medical personnel. Multiple studies have shown that US-guided peripheral IV access is safe and successful in these patients.[16-22] US-guided peripheral IV access prevents the need for CVC and the pain of multiple needle sticks in many "hard-to-stick" patients.[23]

ANATOMY AND PATHOPHYSIOLOGY

It is important to recognize the differences in sonographic appearance between arteries and veins to perform US-guided vascular access. Arteries and veins can be distinguished by their ability to be compressed, Doppler mode signal, location, shape, size, and spectral Doppler waveforms. Arteries have relatively thick and hyperechoic (i.e., white) walls and anechoic (i.e., black) lumens. Veins have relatively thin and hypoechoic (i.e., gray) walls and anechoic (i.e., black) lumens. The thin-walled veins are usually easily compressible, have no pulsations on Doppler mode, and are oval-shaped (**Figures 64-1 and 64-2**). Arteries are typically round in appearance and pulsatile on Doppler mode (**Figures 64-1 and 64-2**). **Arteries and veins are often found adjacent to each other.** Veins are usually larger in diameter than arteries in a well-hydrated patient (**Figure 64-1**). The anatomy relevant to the sonographic evaluation of central and peripheral veins is described in the following sections.

CENTRAL VEINS

The internal jugular, subclavian, and femoral veins are commonly used for US-guided CVC. The internal jugular vein traverses the neck and is unopposed by bone. This makes it an ideal vein to visualize with US. The internal jugular vein lies underneath the bifurcation of the sternal and clavicular heads of the sternocleidomastoid muscle. It continues to run vertically downward in the neck. It lies at first lateral to the internal carotid artery and then lateral to the common carotid artery (**Figure 64-1**). It eventually joins the subclavian vein.

The subclavian vein lies anterior to the subclavian artery along a course from the lateral border of the first rib to the medial border of the anterior scalene muscle. It joins the internal jugular vein medially to form the brachiocephalic vein. Subclavian vein cannulation is technically more challenging because it runs for a significant distance under the clavicle, which produces a large acoustic shadow. Cannulation is most frequently performed from an infraclavicular approach and just lateral to the clavicle at the intersection of the axillary vein and subclavian vein (**Figure 64-3**). This approach allows visualization of the vein without the acoustic shadowing of the clavicle and a greater distance from the pleura.

The common femoral vein lies medial to the common femoral artery and inferior to the inguinal ligament. The superficial femoral and deep femoral veins join in the upper thigh to form the common femoral vein. The greater saphenous vein joins the anteromedial aspect of the common femoral vein below the inguinal ligament (**Figure 64-4**). The common femoral vein changes its name to become the external iliac vein after it passes under the inguinal ligament.

FIGURE 64-1. US image of the neck vessels. The internal jugular vein (IJV) is thin-walled and oval. The carotid artery (CA) is thick-walled and round.

FIGURE 64-2. The US transducer is used to apply external pressure to the soft tissues of the neck. The internal jugular vein (IJV) collapses with gentle compression. CA, carotid artery; SCM, sternocleidomastoid muscle.

PERIPHERAL VEINS

The three veins of the upper extremity that are most commonly used for US-guided vascular access are the basilic, brachial, and cephalic veins. The venous drainage of the upper extremity

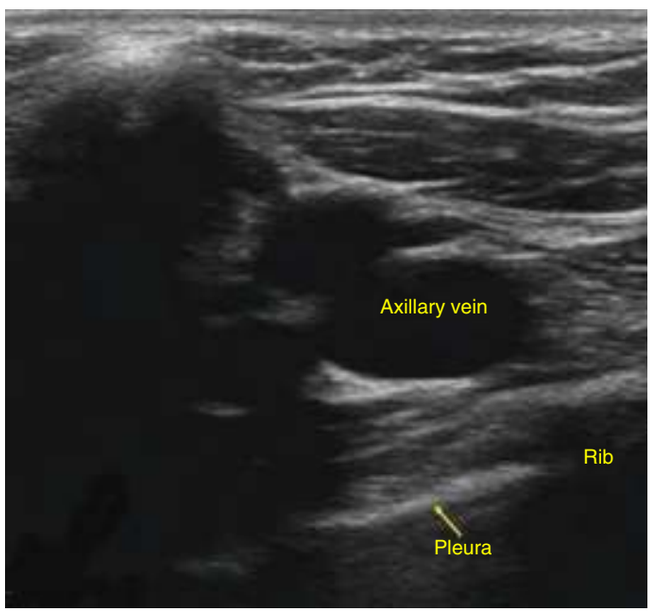


FIGURE 64-3. US image of the right axillary vein. The US transducer is placed in the right infraclavicular region.

FIGURE 64-4. Transverse US image of the femoral vessels. The common femoral vein (CFV) lies medial to the common femoral artery (CFA). The superficially located greater saphenous vein (GSV) enters the common femoral vein 2 to 3 cm below the inguinal ligament.

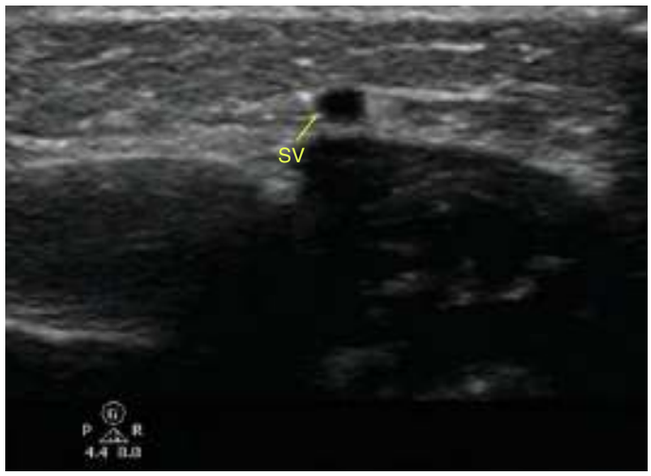


FIGURE 64-6. Transverse US view of the saphenous vein (SV) above the ankle.

consists of a deep system and a superficial system. The deep system includes radial and ulnar veins in the forearm that unite to form the brachial vein, the deep vein in the upper arm. The brachial vein lies next to the noncompressible and pulsatile brachial artery (**Figure 64-5**) and median nerve. It is not uncommon to find paired superficial and deep brachial veins (**Figure 64-5**). The two major superficial veins of the upper arm are the basilic and cephalic veins. The basilic vein begins in the ulnar side of the dorsal hand venous network, ascends along medial aspect of forearm and upper arm, and joins with the brachial veins to form the axillary vein. The cephalic vein arises from the radial aspect of the dorsal hand venous network, ascends proximally along the anterior border of the brachioradialis muscle, runs lateral to biceps muscle, and terminates in the axillary vein.

The great saphenous vein can be accessed in the lower extremity using US guidance. The vein runs along the medial aspect of the lower extremity, anterior to the medial malleolus of the ankle, and posterior to the medial epicondyle of the femur. The saphenous vein can be cannulated anywhere along its course. It is most commonly accessed just proximal to the ankle (**Figure 64-6**).

STATIC APPROACH TO VASCULAR ACCESS

The static approach to vascular access involves confirming the location of the vessel prior to performing the cannulation. Place the US transducer perpendicular to the skin to visualize the vessel and verify compressibility. Adjust the transducer so that the target vessel is in the center of the US monitor screen. **This puts the vessel directly under the center of the US transducer.** Place a mark on the skin at the midpoint of the US transducer to mark the vessel location. Repeat this process to assess the path of the vessel at a point 2 cm away and along the vessel. **Marking these two points on the skin will serve as a guide to direct the needle or catheter-over-the-needle. Determine how deep the vessel is located.** Look at the depth markers on the side of the US monitor screen. Remove the transducer and prepare the patient.

Proceed with the catheterization. Insert the needle or catheter-over-the-needle approximately 0.5 cm above or below the skin mark. Advance and direct it to the appropriate depth underneath the skin markings. **The patient must be reimaged and the skin markings repeated prior to inserting the needle or catheter-over-the-needle if the patient changes position after marking the skin but before needle puncture.** The advantages of the static approach are its simplicity and that the US transducer does not need to be prepared for the sterile portion of the procedure. The disadvantage of this technique is that the needle is not visualized in real time while performing the procedure.

FIGURE 64-5. Transverse US view of the upper arm veins. BA, brachial artery; BAV, basilic vein; BV, paired brachial veins.

DYNAMIC APPROACH FOR VASCULAR ACCESS

The dynamic approach to vascular access involves inserting the needle or catheter-over-the-needle into the vein under real-time US guidance. Three techniques are used for the dynamic approach.[24,25] They refer to the orientation of the US transducer and target vein during the venipuncture. The short axis approach visualizes the vessel in a cross section. The long axis approach visualizes the vessel longitudinally. The oblique approach visualizes the vessel at an oblique angle.

Each approach has its own advantages and disadvantages. The short axis approach provides better medial to lateral orientation of the needle or catheter-over-the-needle in relation to the target vessel and allows for visualization of adjacent structures in the same image. Novice sonographers can learn the short axis approach more quickly, and it can be performed in anatomic locations where space is limited (e.g., the groin or neck in an obese patient). One potential danger with the short axis approach is unseen penetration of the posterior wall of the target vessel and adjacent structures. The long axis approach allows better depth and needle or catheter-over-the-needle slope information. The entire needle or catheter-over-the-needle can be tracked with this approach.[26] The

long axis approach provides much better visualization of the tip of the needle and its trajectory, thus avoiding inadvertent puncture of the posterior wall of the vessel. The long axis approach requires more hand-eye coordination and is technically more difficult to learn. The oblique approach is a hybrid between transverse and longitudinal approaches. The transducer is placed at an oblique angle to the vessel. The needle or catheter-over-the-needle is advanced toward the vessel in-plane with the US transducer. The advantage of this approach is the ability to visualize structures adjacent to the target vessel and observe the needle tip continuously throughout the procedure. The disadvantage is that the needle contacts the vessel at an oblique angle, and this technique may be more difficult to learn.[27]

INDICATIONS

US can be used to mark the site of needle entry or provide real-time guidance.

Use US guidance for all CVCs if the equipment is available. Use US for peripheral IV access in all "hard-to-stick" patients, in children, when a peripheral vein is not visible, or when a peripheral vein is not palpable.[28-34] The use of US-guided peripheral IV access in the patient with difficult peripheral IV access can prevent the need for CVC.[35,36] It is highly recommended in patients with anticoagulant use, in children, and in patients with coagulopathies, disseminated intravascular coagulation, a history of difficult IV access, hypotension, IV drug abuse, obesity, poor anatomic landmarks, prior surgical interventions or radiation in the area, scarring on the skin above veins, severe dehydration, or thrombocytopenia.[37-39]

CONTRAINDICATIONS

There are no absolute contraindications to the use of US for central or peripheral venous access except lack of US training and experience.

EQUIPMENT

- US machine
- US gel
- High-frequency (5 to 10 MHz) linear array US transducer
- Sterile US transducer cover (i.e., clear plastic cover or sterile glove, sterile US gel, and rubber bands)
- Povidone iodine or chlorhexidine solution
- Equipment for peripheral IV access (Chapters 59 and 61)
- Equipment for CVC (Chapter 63)
- Water-soluble lubricant, sterile
- Translucent dressing (e.g., Tegaderm)
- 2 to 3 inch long catheter-over-the-needle (i.e., angiocatheter), various sizes

PATIENT PREPARATION

CENTRAL VENOUS ACCESS

Position the patient the same as if performing CVC using the landmark method (**Figure 64-7**). Place the US machine across from where standing and in a direct line of sight to minimize the body movement required to view the US image. Stand at the head of the patient with the US machine at the patient's hips for internal jugular vein access (**Figure 64-7*A***). Place the patient in the Trendelenburg position with their head turned to the contralateral side or kept in a neutral head position (**Figure 64-7*A***). The relationship of the internal jugular vein to the carotid artery changes with head position. **The internal jugular vein assumes a more lateral position to the carotid artery in the neutral position.[40] This minimizes the risk of arterial puncture while accessing the target vein.[41]** The right internal jugular vein is easier to find and cannulate due to its

A

B

FIGURE 64-7. Patient and US machine positioning for central venous access. ***A.*** Internal jugular vein access. ***B.*** Subclavian vein access using the infraclavicular approach. ***C.*** Subclavian vein access using the supraclavicular approach. ***D.*** Femoral vein access.

C

D

FIGURE 64-7. (*Continued*)

increased size.[42,43] The use of shoulder rolls decreases the overlap of the internal jugular vein and carotid artery.[44] Place the patient in the Trendelenburg position with the shoulders and head in a neutral position for subclavian vein access. Position the US machine on the contralateral side of the patient from the Emergency Physician (**Figures 64-7*B* and 64-7*C***). Caudal traction of the ipsilateral arm may assist in exposure of the subclavian vein for a supraclavicular approach.[45,46] Abduction of the arm to 90° can improve visualization of vessels for the infraclavicular approach.[47,48] Stand at the bedside adjacent to the patient's hip with the US machine above the patient's shoulder for femoral vein access (**Figure 64-7*D***). Place the patient in reverse Trendelenburg with their hip abducted and externally rotated (**Figure 64-7*D***). This position may enhance the success rate of catheterization by decreasing the overlap with the femoral artery, increasing the surface area of the femoral vein, and increasing the vein diameter.[49-53]

Perform a preliminary US scan prior to preparing the patient for the sterile procedure. Survey the underlying vasculature to confirm the patency of the target vein, determine the optimal site for venipuncture, and adjust the US machine settings to optimize the image. The ability of the vein to be compressed distinguishes an artery from a vein, confirms the patency of the vein, and reduces the risk of cannulating a thrombosed vein (**Figure 64-8**). **Do not assess compressibility in the long axis.** The US transducer may slide off the vein and the vein lumen disappears, misinterpreting the vein as being compressible. Clean and prep the skin at the access

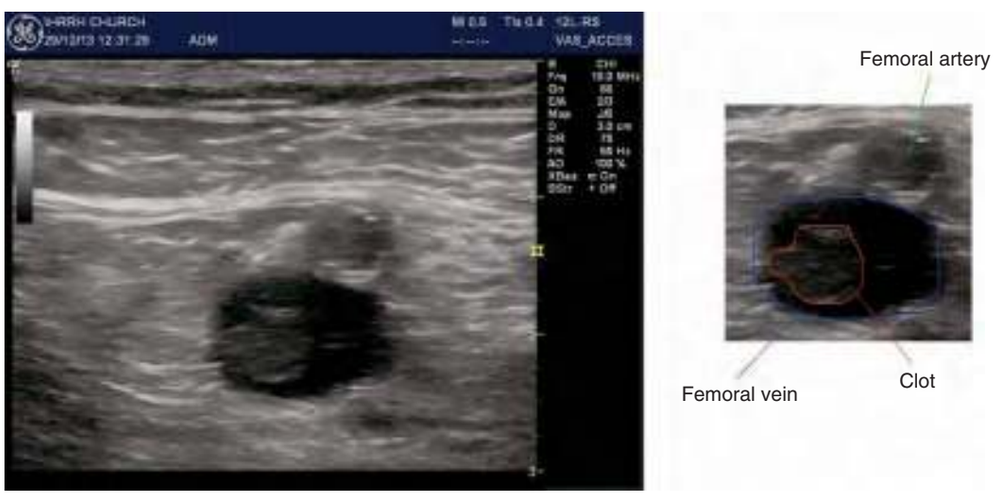


FIGURE 64-8. A clot or thrombus in the femoral vein. The image on the left is labeled on the right. (Courtesy of Lloyd Gordon, MD.)

site as if performing a traditional CVC. Prepare the US transducer as discussed below. **Follow strict aseptic technique and all barrier precautions during the procedure.**

The procedure can be performed by either one person (i.e., the Emergency Physician) or two people (i.e., the Emergency Physician and an assistant). The assistant stabilizes the US transducer over the vein, and the Emergency Physician performs the procedure using the two-person technique.

ULTRASOUND TRANSDUCER PREPARATION

A high frequency (i.e., 5 to 10 MHz) linear array US transducer is typically used for US-guided vascular access. **These US transducers produce linear images and are ideal for visualization of superficial structures such as veins. Always use a sterile US transducer cover and sterile US gel when performing CVC.** Set up a sterile field on a bedside table. Open the US transducer cover set onto the sterile field.

Instruct an assistant to hold the US transducer upright and place standard or sterile US gel on the footprint of the US transducer (**Figure 64-9*A***). Apply the sterile transducer cover over the US transducer using aseptic technique (**Figure 64-9*B***). **Smooth all the air bubbles away from the footprint of the US transducer to prevent imaging artifacts.** Secure the cover with rubber bands to prevent it from sliding off the US transducer (**Figure 64-9*C***). Place the US transducer on the sterile field (**Figure 64-9*D***). Apply sterile US gel onto the cover over the transducer footprint just before scanning.

A

B

C

D

FIGURE 64-9. Preparing the US transducer. ***A.*** An assistant holds the US transducer upright and US gel has been applied. ***B.*** The sterile transducer cover is applied. ***C.*** The sterile transducer cover is secured with sterile rubber bands. ***D.*** The prepared US transducer on a sterile field.

FIGURE 64-10. The US transducer marker and the marker on the US machine screen are aimed in the same direction.

ULTRASOUND TRANSDUCER ORIENTATION

The marker on the US transducer and the marker on the US machine screen should be aimed in the same direction in the short axis approach (**Figure 64-10**). The needle moves to the right on the US machine screen if it moves to the right side of the US transducer. This helps to accurately move the needle right to left while directing the needle toward the vein. This makes performing the procedure less complicated. It is important to know which way the transducer marker is directed in the long axis or oblique approach so that the Emergency Physician knows which side of the screen the needle will come from (i.e., the right or left of the image) (**Figure 64-11**).

IDENTIFICATION OF VEINS

Identify and verify the depth, direction, and patency of the vein prior to the procedure. Place the US transducer in the triangle formed by the two heads of sternocleidomastoid muscle and clavicle to locate the internal jugular vein (**Figure 64-12*A***). Scan from the apex of the triangle to the clavicle. Visualize the internal jugular vein, carotid artery, and thyroid gland (**Figure 64-12*B***). The internal jugular vein is an irregular and oval-shaped structure lateral to the carotid artery

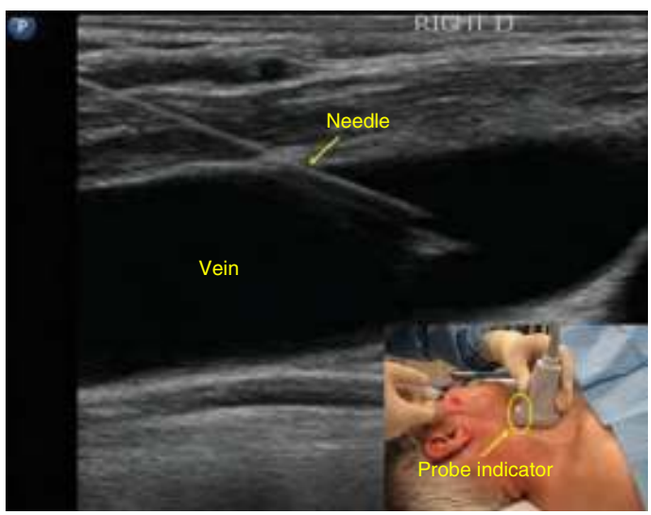
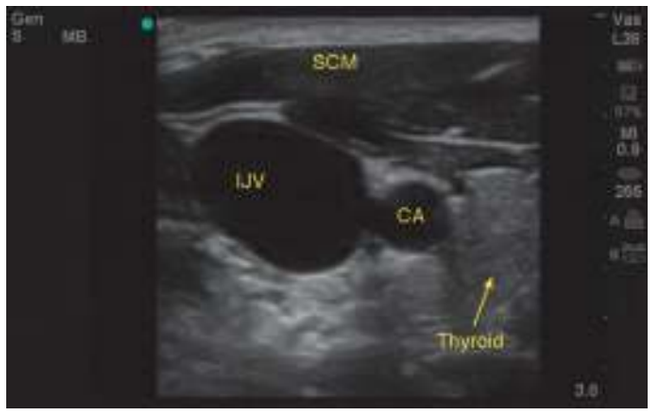


FIGURE 64-11. Long axis approach for internal jugular vein access. Note transducer indicator and needle.

A



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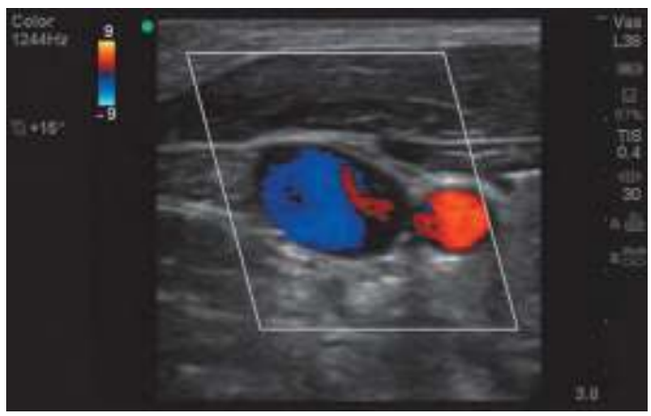


C

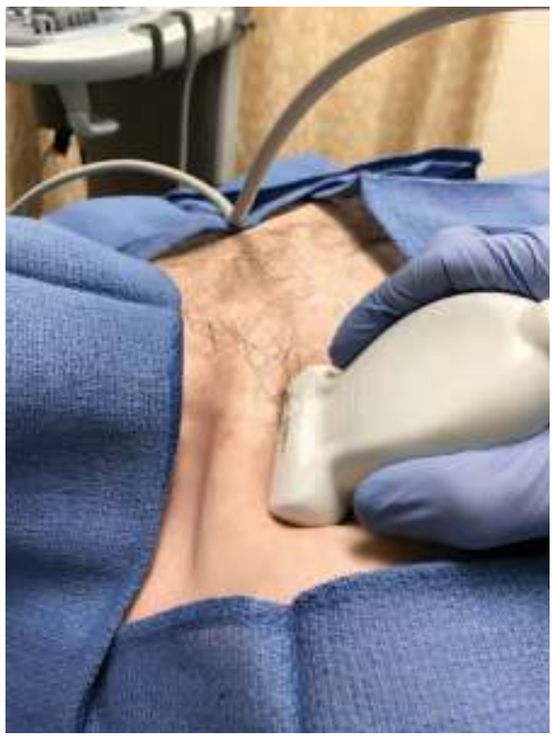
FIGURE 64-12. Identification of the internal jugular vein. ***A.*** Patient and US transducer positioning. ***B.*** The transverse or short axis view in B-mode. ***C.*** Short axis view in color Doppler mode. The large blue internal jugular vein (IJV) is clearly visible adjacent to the red carotid artery (CA). SCM, sternocleidomastoid muscle.

(**Figures 64-12*B* and 64-12*C***). Visualize the internal jugular vein and identify its widest diameter, the depth from the skin surface, and its relationship to the carotid artery. Instruct the patient to perform a Valsalva maneuver to increase venous flow and distend the internal jugular vein while observing the US machine screen to note the changes in the US appearance of the vein.

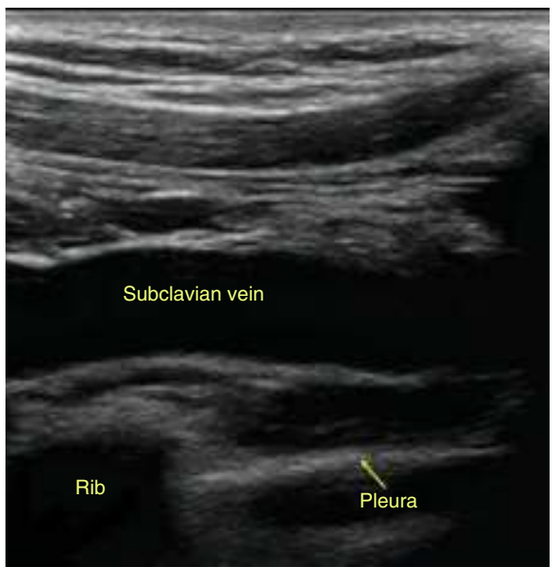
Identify the vein using US. The subclavian vein can be cannulated using the infraclavicular or supraclavicular approach.[54] The supraclavicular approach has some advantages (i.e., can be performed simultaneously with cardiopulmonary resuscitation or tube thoracotomies, decreased risk of arterial puncture, decreased risk of pleura puncture, larger target, more direct path to the superior vena cava, shorter skin to vein distance, and well-defined landmarks). Place the US transducer inferior and lateral to the midportion of the clavicle to locate the subclavian vein using the infraclavicular approach (**Figure 64-13**). The clavicle and first rib are identified by a hyperechoic line with an acoustic shadow (**Figure 64-13*B***). Visualize the subclavian and axillary veins in the short axis view. Position the target vein in the middle of the screen and rotate the transducer, maintaining visualization of the vein until a long axis view is obtained. This maneuver enables visualization of axillary vein, distal subclavian vein, and the pleura underneath the vessels. The pleura is seen just deep to the vein and is identified by a hyperechoic line with associated sliding movement, characteristic of pleural sliding (**Figure 64-13*B***). It might be difficult to demonstrate compressibility due to the slightly deeper location of the veins. Use Doppler to confirm the appropriate vessel (**Figure 64-13*C***). Trace the internal jugular vein caudally into the supraclavicular fossa and locate the confluence of the internal jugular vein and subclavian vein using the supraclavicular approach (**Figure 64-14*A***). Angle the transducer from a posterior to anterior position to locate the subclavian vein, which lies anterior to the subclavian artery (**Figure 64-14*B***). Use an endocavity transducer with a smaller footprint instead of a linear array transducer if there is limited space in the supraclavicular area.

Scan below the midpoint of inguinal ligament to locate the common femoral vein (**Figure 64-15*A***). The common femoral artery is visible next to the common femoral vein (**Figure 64-15*B***). The greater saphenous vein merges with the common femoral vein on the medial aspect. Visualize the common femoral vein and identify its widest diameter, the most superficial location, the depth from the skin surface, and a location that does not overlap with the common femoral artery.

Do not apply excessive pressure with the US transducer when assessing the vein. Excessive pressure can collapse the vein and makes it difficult to identify. Verify that the identified vessel is compressible and truly a vein. **The ability of the vein to be compressed distinguishes artery from vein, confirms the patency of the vein, and reduces the risk of cannulating a thrombosed vein. Do not assess vein compressibility in the long axis.** The US transducer may slide off the vein and the vein lumen will disappear, misinterpreting the vein as being compressible.



A



B

C

FIGURE 64-13. Identification of the right subclavian vein using the infraclavicular approach. ***A.*** Patient and US transducer positioning. ***B.*** Long axis view of the subclavian vein. Note the rib shadow and pleura in the image. ***C.*** Spectral Doppler waveform of the subclavian vein.

A

B

FIGURE 64-14. Identification of the right subclavian vein using the supraclavicular approach. ***A.*** Patient and US transducer positioning. ***B.*** Long axis view of the subclavian vein.

Doppler can be used to identify vascular structures. Color Doppler ensures the patency of blood vessels (**Figure 64-12*C***). Spectral Doppler distinguishes blood flow patterns. Spectral Doppler demonstrates respiratory phasicity in the femoral veins and biphasic flow patterns in the internal jugular and subclavian veins because of their proximity to the heart (**Figure 64-16*A***). Triphasic pulsatile flow is seen on spectral Doppler of the femoral and subclavian arteries, and low resistance flow is seen in the common carotid artery (**Figure 64-16*B***).

TECHNIQUES

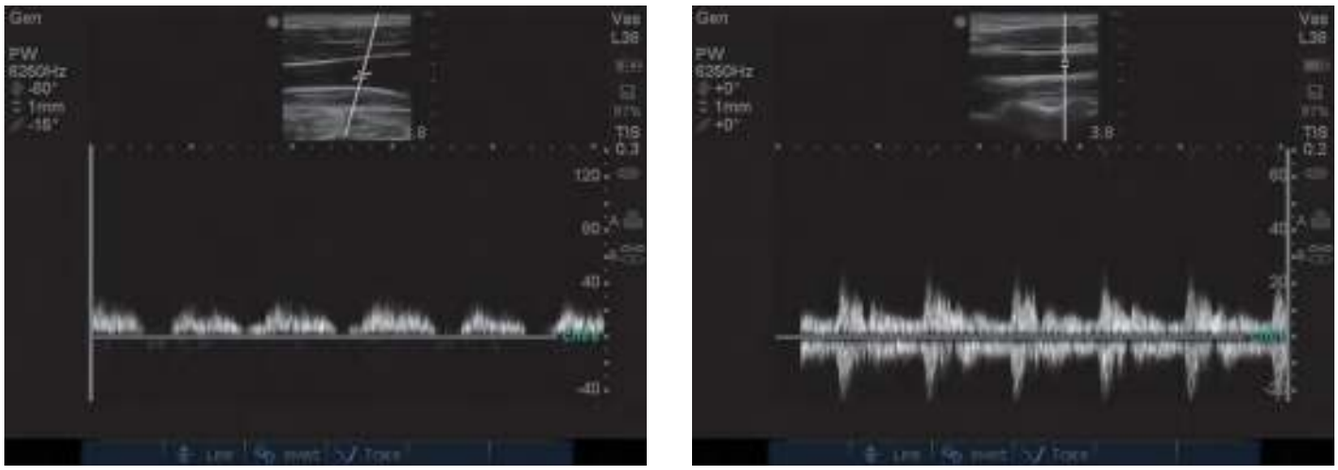
SHORT AXIS APPROACH TO CENTRAL VENOUS ACCESS

The short axis approach allows visualization of the vein in cross section. This view is obtained by placing the US transducer perpendicular to the long axis of the vein. The vein appears oval-shaped in this view (**Figures 64-1, 64-12*B*, and 64-15*B***). Place sterile US

A

B

FIGURE 64-15. Identification of the common femoral vein. ***A.*** Patient and US transducer positioning. ***B.*** Transverse or short axis view in B-mode. CFA, common femoral artery; CFV, common femoral vein.



A B

FIGURE 64-16. Long axis US images of spectral Doppler waveforms. ***A.*** The internal jugular vein. ***B.*** The carotid artery.

gel on the skin above the vein and on the footprint of the sterile US transducer. Grasp the US transducer with the nondominant hand and the needle or catheter-over-the-needle with the dominant hand. Reidentify the vein and the optimal site of insertion. **Determine the best location to puncture the vein in relation to the adjacent artery by assessing the percentage of posterior free wall of the vein in contact with the artery (Figure 64-17).** Adjust the US transducer to center the target vein on the US machine screen. The midpoint of the US transducer now becomes the reference point for needle insertion.

Select the skin entry site to maximize the chances that the tip of the needle punctures the vein and intersects the US transducer scan plane. The geometry of the Pythagorean Theorem (i.e., $a^2 + b^2 = c^2$) can be used to assess the distance to insert the needle into the skin and away from the US transducer (**Figure 64-18**). Measure the distance from the skin surface to the center of the vein. This distance is equal to the distance from the transducer to where the needle punctures the skin at a 45° angle. For example, if the distance from the skin surface to the center of the vein is 1 cm, make the skin puncture 1 cm from the midpoint of the US transducer along the trajectory of the vein (**Figures 64-18*A* and 64-18*B***). Insert and advance the needle at a 45° angle. The vein will then be punctured after the needle is inserted 1.4 cm. **It is very useful to assess the distances using this method before venipuncture to avoid complications.** Reassess the needle trajectory if the vein is not punctured within the expected inserted needle length.

The US beam is very narrow and only 1 to 2 mm wide despite the US transducer being approximately 1 cm wide. The needle will be visualized only when it is within this narrow beam. The cross section of the needle in the short axis approach will be seen only when it crosses the US scan plane that is oriented perpendicular to the needle. The needle tip or shaft will appear as a hyperechoic dot within the vein (**Figure 64-19*A***). It will be seen in association with either an acoustic shadow (i.e., black) or a reverberation or ring down artifact (i.e., white echoes) posterior to the needle (**Figure 64-19*B***).

Clean, prep, and anesthetize the skin after estimating the distance to the vein. Insert and advance the needle at a 45° angle (**Figure 64-20**). **The needle location can only be identified by signs of it pushing through the tissue as soft tissue movement during the initial portion of the needle path.** This is because the needle has not yet crossed the US transducer scan plane. Small, rapid, and in-line movements of the needle are helpful to locate the needle tip on the US image. **Do not mistake the needle shaft for the needle tip.** Always try to locate the needle tip by angling or fanning the US transducer. The needle will tent or buckle the vein wall as it contacts the anterior vein wall (**Figure 64-21**). **The wall of the vein will first collapse downward as it is being punctured and then return to normal after the wall is punctured.** Visualize the hyperechoic needle tip within the lumen of the vein to confirm the needle entered the vein (**Figure 64-19*A***). A concurrent flash of blood will be seen in the syringe. Place the US

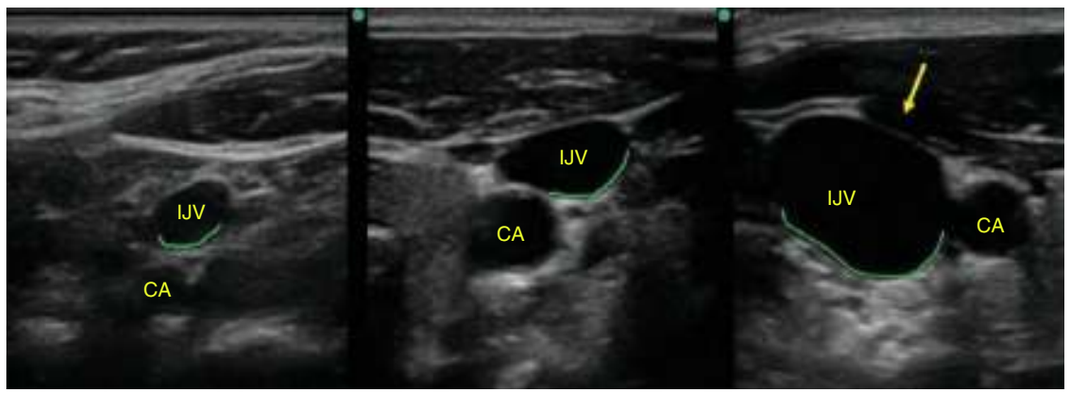


FIGURE 64-17. Short axis views of the neck vessels. Note the percentage of the posterior free wall of the vein (marked in green) in relation with the artery in three different locations in the same patient and the best location (*arrow*) for venipuncture. CA, carotid artery; IJV, internal jugular vein.

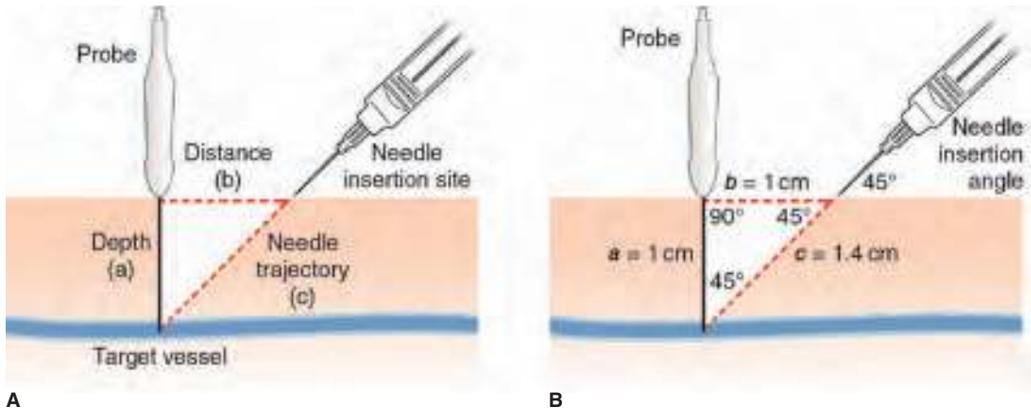


FIGURE 64-18. The short axis approach for needle insertion is based on the Pythagorean theorem. ***A.*** The depth (a) equals the distance from the US transducer (b) to insert the needle at a 45° angle. ***B.*** A sample calculation where $a^2 + b^2 = c^2$.

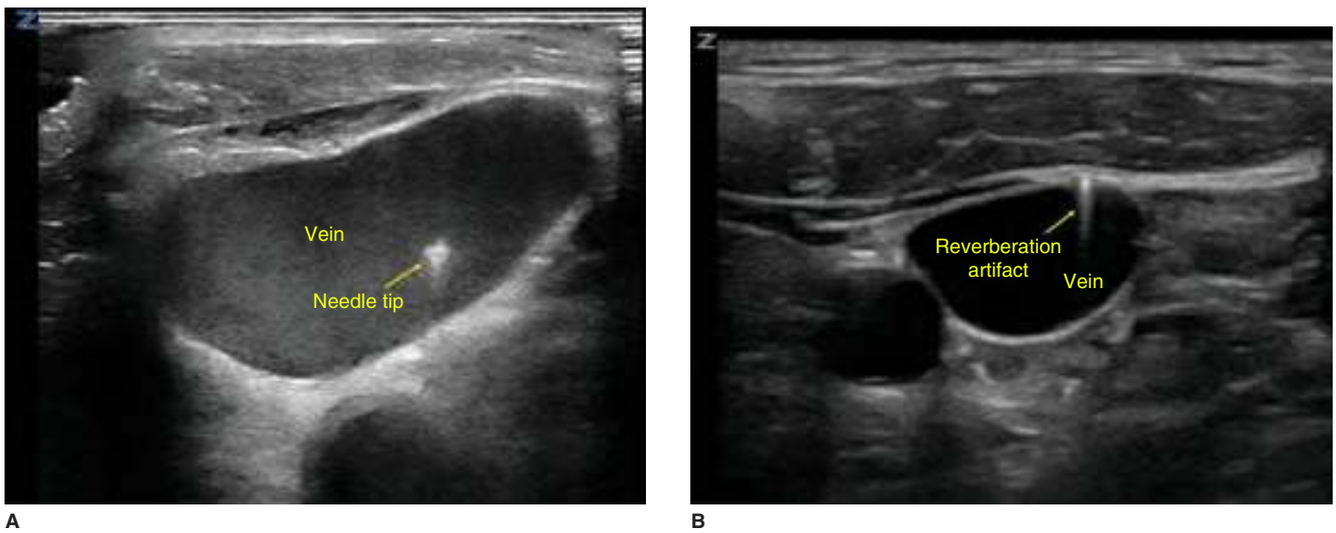


FIGURE 64-19. Short axis view of a needle within a vein. ***A.*** The needle tip is visible as a hyperechoic dot inside the lumen. ***B.*** The reverberation or ring down artifact from the needle.

FIGURE 64-20. The short axis approach for venous access.

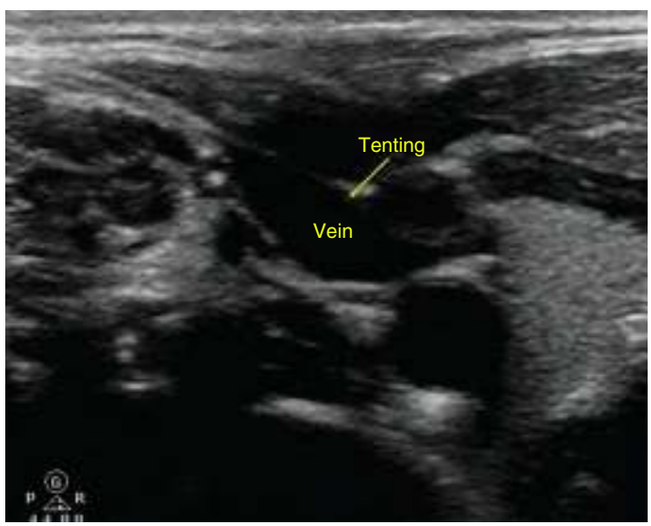


FIGURE 64-21. Short axis view demonstrating tenting of the anterior wall of the vein as it is penetrated by the needle.

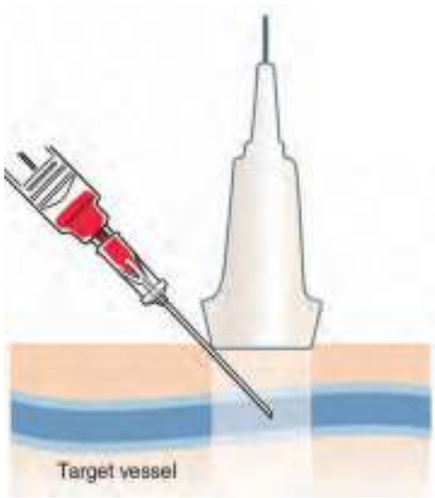


FIGURE 64-22. The long axis orientation and needle insertion.

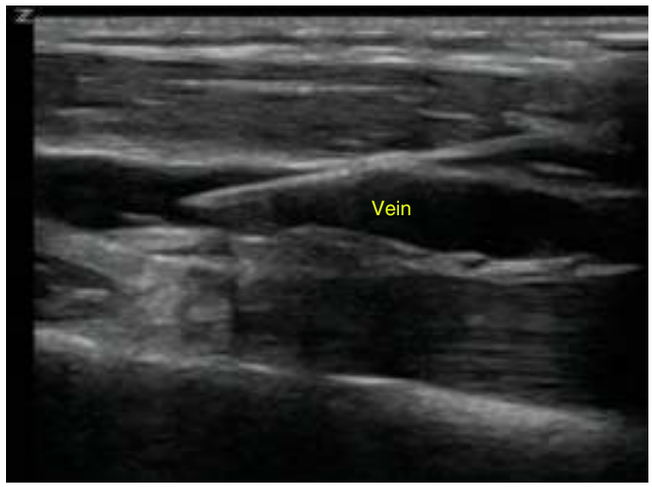


FIGURE 64-24. Long axis view of the needle shaft in the subcutaneous tissues and the tip inside the lumen of the vein.

transducer back onto the sterile field. The remainder of the procedure is as described for the landmark technique of CVC (Chapter 63).

LONG AXIS APPROACH TO CENTRAL VENOUS ACCESS

The long axis approach targets the vein along its length in the longitudinal plane (Figure 64-22). Grasp the US transducer in the nondominant hand and the needle with the dominant hand. Locate the target vein using the short axis approach. Verify it is compressible. Slowly rotate the US transducer to visualize the vein in the long axis. The US transducer should be positioned directly over the vein so that its long axis is parallel to the long axis of the vein (**Figure 64-22**). Adjust the US transducer to visualize the vein at its greatest anteroposterior diameter.

Insert the needle through the skin at approximately a 30° angle adjacent to one end of the US transducer (**Figures 64-22 and 64-23**).

FIGURE 64-23. The long axis approach for venous access.

The needle should be in the plane that is in-line with the long axis of the US transducer and in the exact same plane as the US beam. **Keep the US transducer steady and over the vein with this approach.** Advance the needle. The needle tip and shaft will be visualized in real time as it travels through the subcutaneous tissues and into the lumen of the target vein (**Figure 64-24**). The needle plane and US transducer scan plane are not aligned if the needle is not visualized in the US image. Do not advance the needle any further. Withdraw the needle toward the skin and redirect it to align it with the US beam and the long axis of the vein.

OBLIQUE APPROACH TO CENTRAL VENOUS ACCESS

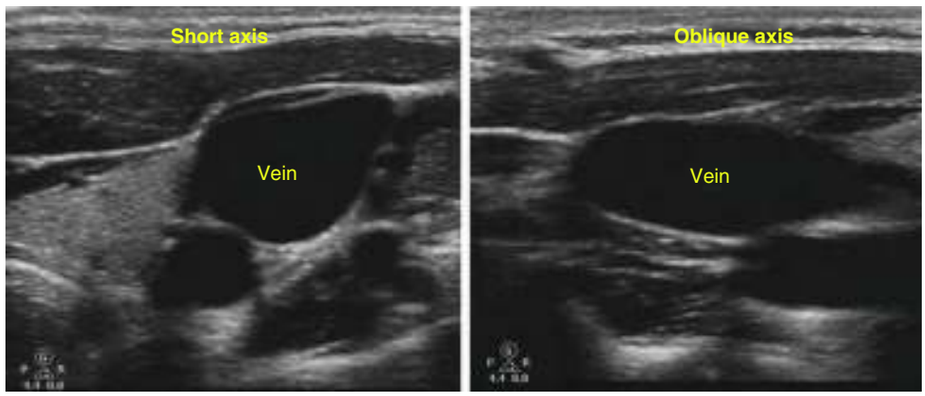
The oblique approach allows visualization of the structures that are adjacent to the target vessel and full needle-tip evaluation throughout the procedure. This approach combines the advantages of the long and short axis approaches. It may allow a higher first-pass success rate and decrease the complications compared to other approaches.[25] The oblique approach is not commonly used and may be difficult to learn.

Place the US transducer on the skin above the target vessel and identify the vein in the short axis. Confirm that the vein is compressible. Rotate the transducer approximately 45° to almost midway between the short and long axis views (**Figure 64-25*A***). The vein will be visualized as oval-shaped (**Figure 64-25*B***). Insert the needle into the skin on one end of the US transducer in-plane with the US beam. The needle tip and needle shaft will be fully visualized as the needle travels at an oblique angle toward the target vessel. Stop advancing the needle if the needle tip is not visualized. Retract the needle and realign it with the US beam. The needle will enter the vessel at a 45° angle. **Do not continue to advance the needle once it is within the vessel walls.** When using a Seldinger technique, the guidewire will be threaded through the needle and will be required to advance along this angle. This may complicate the procedure.

PERIPHERAL VENOUS ACCESS

Place the patient supine with their upper extremity abducted to expose the anteromedial aspect of the upper arm (**Figure 64-26**). Place a tourniquet on the upper arm and just below the axilla to distend the veins.[55] Do not use a blood pressure cuff as a tourniquet.[55] Sit or stand facing the patient. Position the US machine so that the

A



B

FIGURE 64-25. The oblique approach for venous access. ***A.*** The transducer is rotated almost midway between the short and long axis views. ***B.*** Ovoid appearance of the vein using the oblique approach (right). The view can be compared to the short axis (left).

screen is easily visible (**Figure 64-26**). Scan the entire upper arm from just below the tourniquet to the elbow to locate the veins and find the ideal venipuncture site. Identify the basilic vein along the medial aspect of the upper arm (**Figure 64-27**). Move the US transducer slightly laterally to visualize the brachial vein adjacent to the brachial artery. Scanning further laterally will identify the cephalic vein. Place the tourniquet at the calf with patient supine to locate the saphenous vein (**Figure 64-28**).

Place the transducer anterior and superior to the medial malleolus to locate the saphenous vein (**Figure 64-28**). This location has been successfully used in children.[56,57] The saphenous vein in children less than 3 years of age is bigger than hand and forearm veins and equal in size to the antecubital vein.

Peripheral veins collapse very easily. Minimize the pressure placed upon the skin surface with the US transducer. **Too much pressure can collapse these veins and make them difficult to identify.** Identify the segment of vein that is widest in diameter, closest to the skin surface, and not adjacent to an artery or nerve. Verify that the vein is compressible and patent as described previously.

Prepare the skin insertion site. There is no need to follow any special precautions (e.g., a sterile US transducer cover). Apply a large transparent dressing (e.g., Tegaderm) onto the footprint of the linear array US transducer (**Figure 64-29**).[58] **Make sure that no air bubbles are trapped between the transparent dressing and the**

FIGURE 64-26. Patient and US machine positioning for upper extremity peripheral venous access.

FIGURE 64-27. Short axis US image of the basilic and brachial veins. BA, brachial artery; BAV, basilic vein; BV, brachial vein.

FIGURE 64-28. US transducer positioning for saphenous venous access. (Used with permission from reference 56.)

US transducer. The transparent dressing keeps the field sterile and prevents contamination of the US transducer with blood. Use water-soluble lubricant (e.g., Surgilube) instead of US gel for scanning. Use a 2.5 to 3.0 inch long catheter-over-the-needle for US-guided peripheral IV access. Standard-length peripheral IV catheters are too short to reach or stay within the lumen of the vein. Identify the vein in the long axis (**Figure 64-30*A***), short axis (**Figure 64-30*B***), or oblique axis using the methods described previously. Insert the catheter-over-the-needle under US guidance. Advance the catheter-over-the-needle into the vein lumen (**Figure 64-31**). Put down the US transducer. Withdraw the needle, attach IV tubing onto the hub of the catheter, and secure the catheter.

ARTERIAL ACCESS

US guidance can be used to assist in obtaining an arterial blood gas and the placement of an arterial catheter in children and adults.[24,59-64] Identify the artery by its round shape, relatively thick walls, noncompressibility, pulsatile contractions, and Doppler flow. The procedure of cannulating an artery is the same as described above for a vein. Refer to Chapter 72 for the complete details regarding arterial puncture and cannulation.

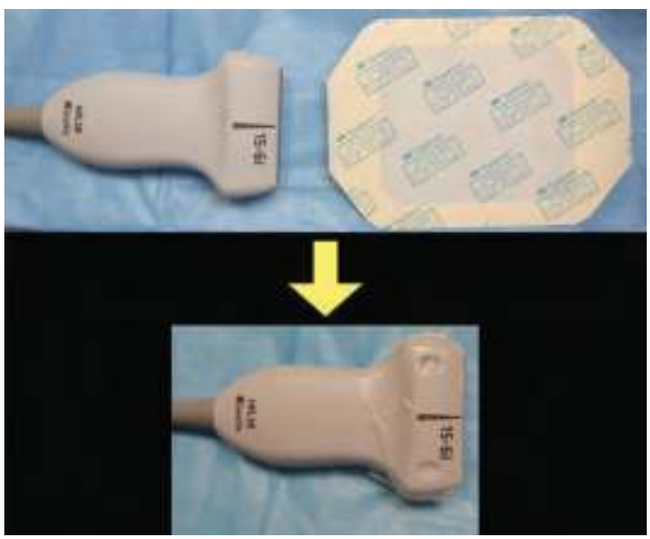


FIGURE 64-29. A sterile Tegaderm is used to cover the US transducer. (Used with permission from reference 58.)

A

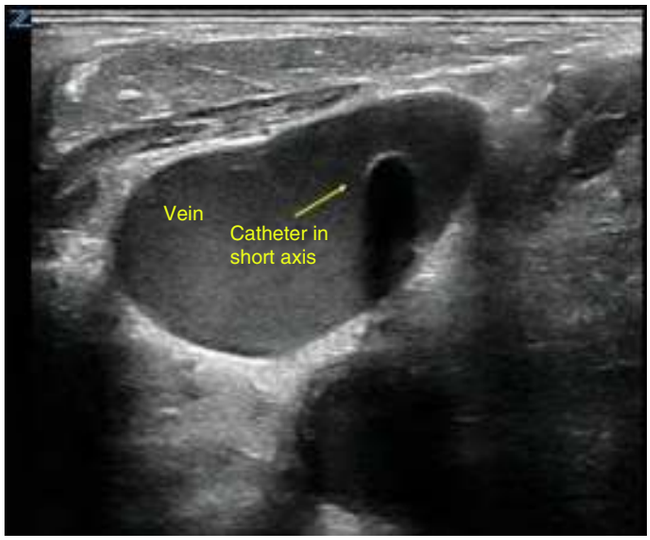
B

FIGURE 64-30. US transducer and needle insertion for upper extremity venous access. ***A.*** Long axis approach. ***B.*** Short axis approach.

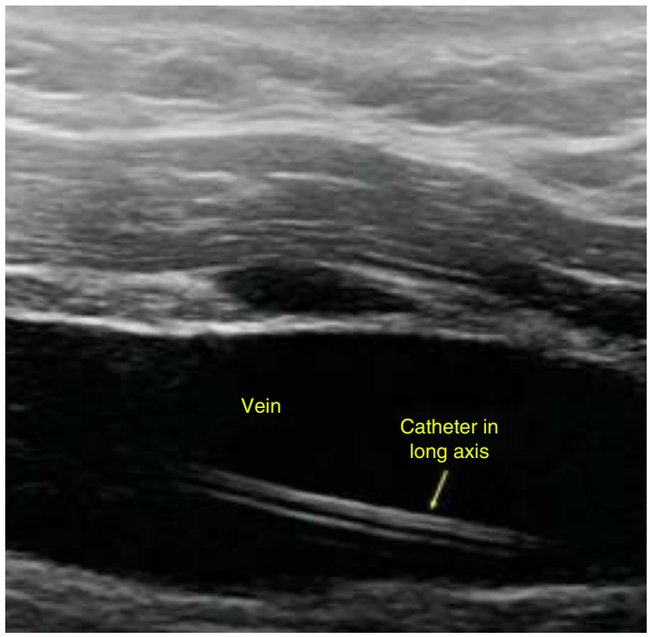
ASSESSMENT

Successful placement of venous access is traditionally confirmed by withdrawing venous blood, easy flushing of saline solution, and radiographs for CVC.[65] US can be used to confirm the catheter placement by scanning the length of the cannulated vein in the long axis and short axis views to visualize the catheter within the lumen of the vein (**Figure 64-31**).[66-69] The advantages of US are that it can be performed at bedside and is quicker than radiographs.

Real-time visualization of a saline flush within the venous system can be used as an adjunct to confirm accurate catheter placement.[70] Saline flushed into the distal port of a central venous catheter can be visualized within the right atrium. Use the phased array or curvilinear US transducer to obtain a subxiphoid four-chamber image. Visualize the right heart using US and have an assistant flush 10 mL of agitated sterile saline through the distal port. Visualize punctate hyperechoic microbubbles from the flush within 2 seconds in the right atrium (**Figure 64-32**).[71]



A



B

FIGURE 64-31. Confirmation of venous catheter placement. ***A.*** Short axis view ***B.*** Long axis view.

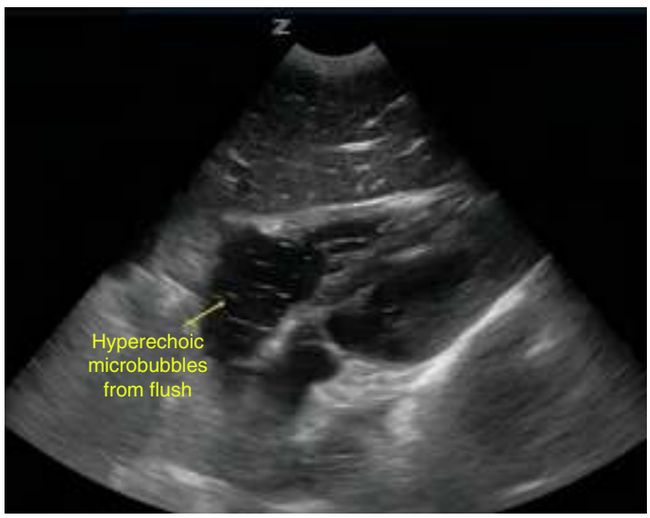
The saline flush can be used for confirmation of peripheral venous catheters (**Figure 64-33**). Use a linear US transducer. Identify the tip of the venous catheter in the vein using the short or the long axis view. Advance the US transducer to visualize the area just beyond the tip of the catheter while flushing normal saline through the catheter. Hyperechoic microbubbles will be visualized in the vessel (**Figure 64-33**).

AFTERCARE

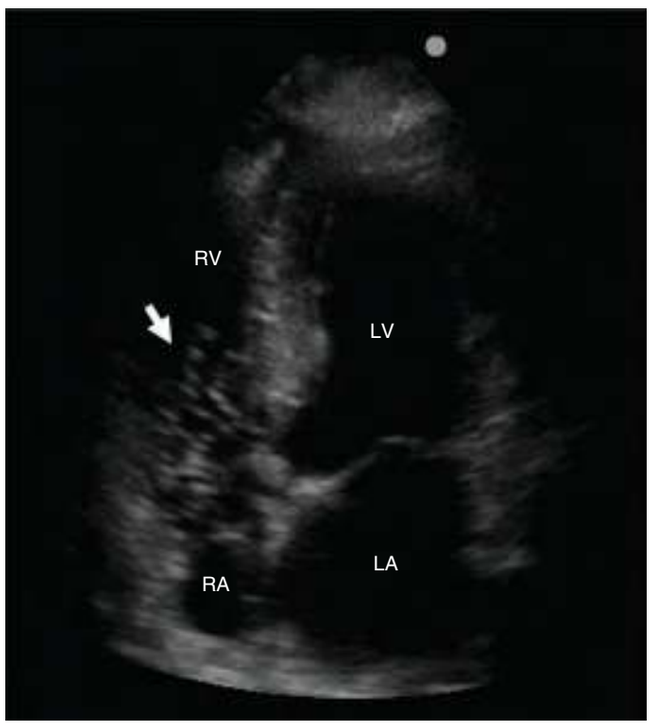
No specific aftercare is required following the US guidance. The aftercare procedures are related to the central venous line (Chapter 63) and peripheral IV (Chapters 59 and 61) placement.

COMPLICATIONS

Complications related to using US for vascular access are generally due to poor technique or misinterpretation of images. The inability to continuously visualize the needle tip during the procedure can result in inadvertent puncture of the adjacent artery or nerve. An artery can be misinterpreted as a vein. There is no exposure to



A



B

FIGURE 64-32. Flush test for confirming central venous catheter placement. ***A.*** Punctate hyperechoic microbubbles (*arrow*) are seen in the right atrium from the flush. ***B.*** Another patient with microbubbles (*arrow*) seen going from the right atrium to the right ventricle. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle. (Used with permission from reference 75.)

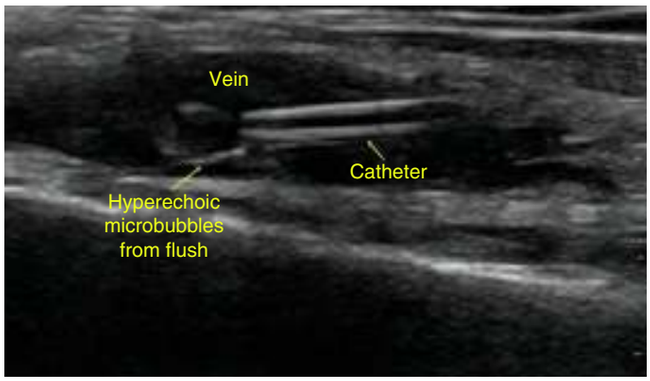


FIGURE 64-33. Flush test for confirming peripheral venous catheter placement. Hyperechoic microbubbles are seen in the vein leaving the tip of the catheter.

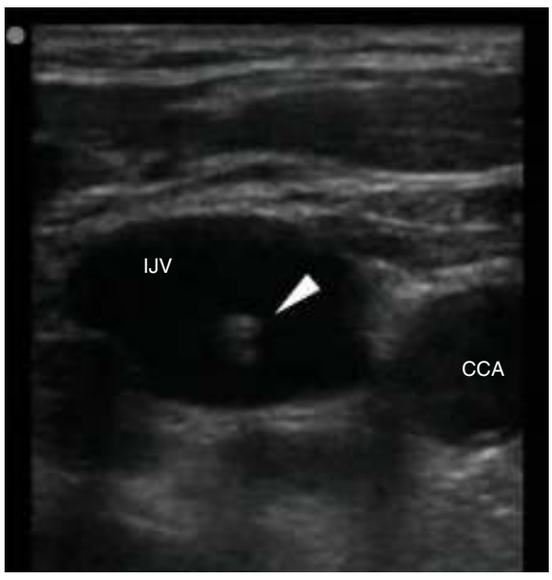
ionizing radiation. No increased risk of infections has been reported with the use of US when aseptic technique was followed. US guidance can be more time consuming when used for routine peripheral IV access. Its use may save time in the patient with difficult venous access. Using too short of a peripheral IV catheter can result in it being pulled out of the vein and the infusion solution extravasating.[72] This can be prevented by using a longer (i.e., 2 to 3 inch) catheter-over-the-needle and/or inserting the catheter at a steeper angle to minimize the distance it travels in the subcutaneous tissues.

Use US to confirm the position of the central venous catheter. It can identify whether the central venous catheter is malpositioned by following the catheter's course. The catheter can go to into the contralateral internal jugular vein instead of the superior vena cava (**Figure 64-34**). This requires changing it over a guidewire or inserting one at a different site. The catheter can be inserted too far and be located within the right atrium instead of the superior vena cava (**Figure 64-35**). Withdraw the catheter back slightly and repeat the imaging.

FIGURE 64-35. Malposition of the central venous catheter in the right atrium (*arrow*). Ao, aorta; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle. (Used with permission from reference 75.)

FUTURE CONSIDERATIONS

Many companies are planning or make a guidance system for central venous access.[73-75] One popular device is the AxoTrack (Soma Access Systems, Greenville, SC). The AxoTrack is a US probe with a built-in needle guidance system (**Figure 64-36**). It incorporates real-time information of the needle direction and location. The needle is visualized from the skin puncture to the vein. It includes an US transducer with a needle guide. The transducer has magnetic



A

B

FIGURE 64-34. Malposition of the central venous catheter in the contralateral internal jugular vein. ***A.*** Short axis view. ***B.*** Long axis view. CCA, common carotid artery; IJV, internal jugular vein. (Used with permission from reference 75.)

A

B

C

FIGURE 64-36. The AxoTrack (Soma Access Systems, Greenville, SC). ***A.*** The device. ***B.*** Positioning the device. ***C.*** Insertion of the guidewire through the needle.

sensors, monitors needle depth and projects this on the US monitor screen. This allows the Emergency Physician to know the needle path and where the needle tip is located without adjusting the transducer. A clamping system secures and stabilizes the needle once inserted into the vessel.

SUMMARY

US-guided vascular access is widely supported in current clinical practice. It can be used for central and peripheral IV access. US guidance has been shown to reduce failure rates and many of the complications associated with the traditional landmark technique. The benefits of US guidance include precise localization of the target vein, visualization of adjacent structures, identification of anatomic variations, avoidance of veins with thrombus, and real-time visualization of venipuncture. Knowledge of the US anatomy and basic US technology combined with hand-eye coordination are essential to perform this procedure. Understanding the principles discussed in this chapter will enhance the success rate of vascular access.

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65 Troubleshooting Indwelling Central Venous Lines

James J. McCarthy

INTRODUCTION

Indwelling central venous lines are an essential part of the care of acute and chronically ill patients (**Figure 65-1**). These patients may require implanted venous access devices due to their poor peripheral venous access or for long-term intravenous therapies (**Figures 65-2 through 65-4**). **The Emergency Physician must act quickly and thoughtfully to diagnose and correct the malfunctioning indwelling central venous line without further damaging the device or exposing the patient to increased risk.** Understanding the various etiologies and a thorough assessment are critical to the successful management of a central venous catheter malfunction.

ANATOMY AND PATHOPHYSIOLOGY

Indwelling central venous catheters allow access to the central venous circulation from a peripheral site. This access to the central circulation is via the end of a partially implanted catheter that protrudes from the body or through the skin into a subcutaneous reservoir of a fully implanted catheter (**Figure 65-1**).[1,2] The proximal tip of the central venous line resides in the inferior vena cava, the right

FIGURE 65-2. A Mahurkar short-term vascular access catheter. (Courtesy of Medtronic, Minneapolis, MN.)

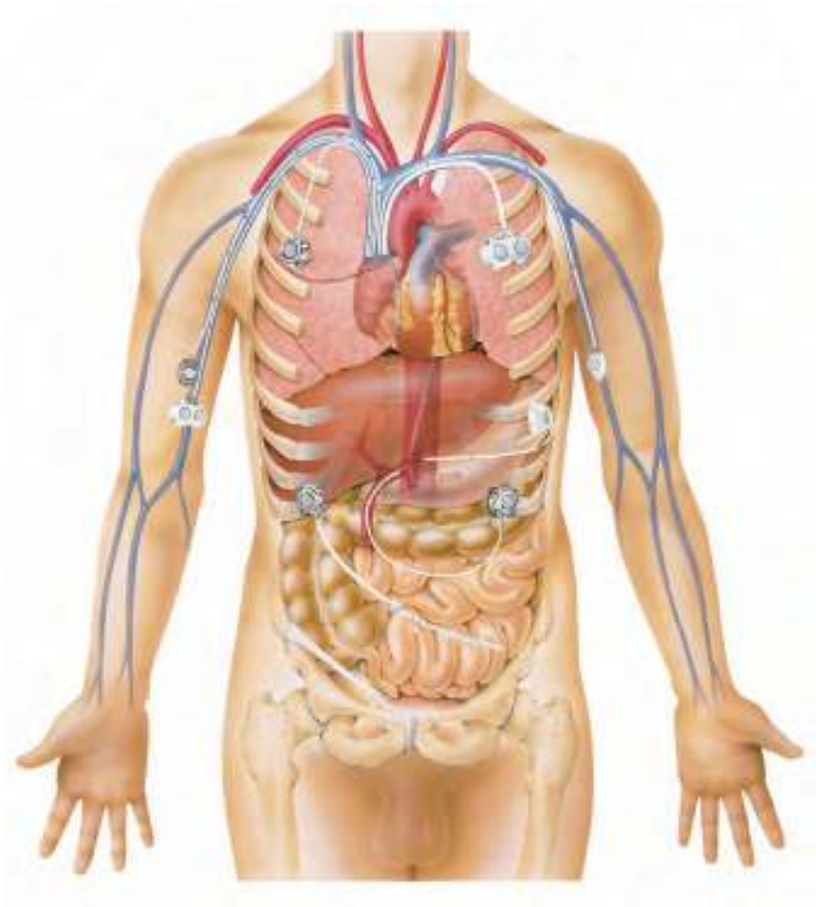
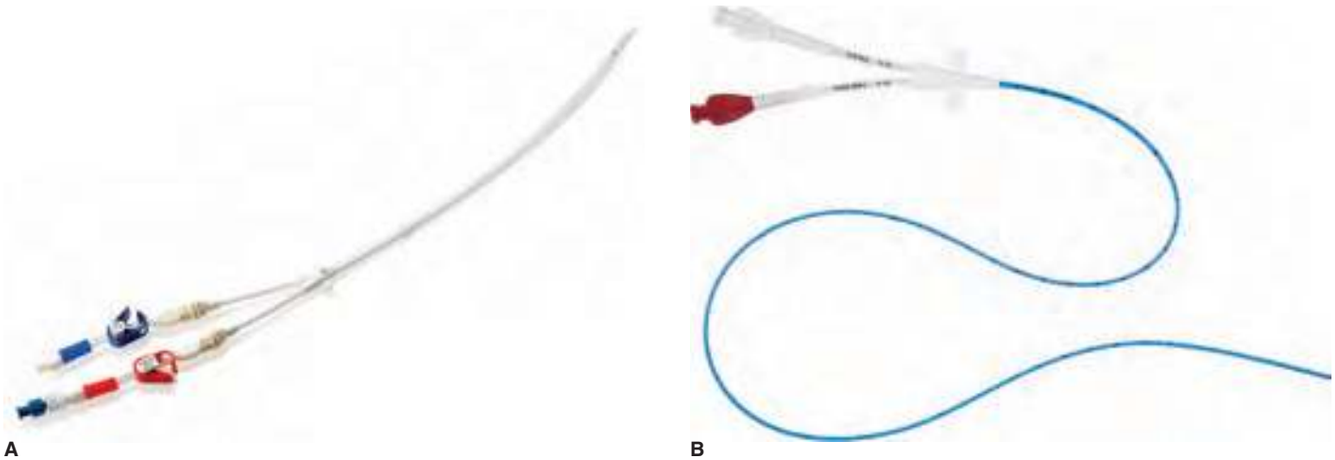


FIGURE 65-1. A variety of indwelling access devices. (Courtesy of Smiths Medical, St. Paul, MN.)

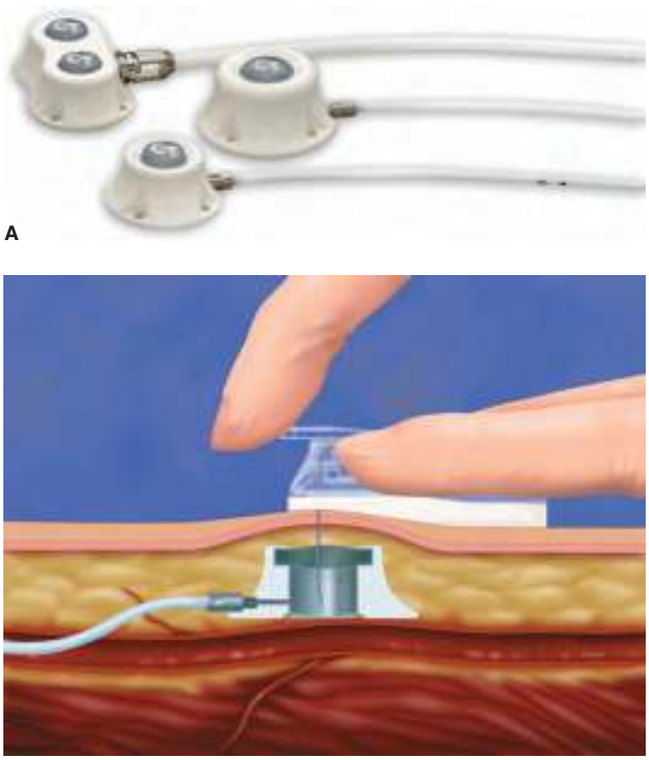


A B

FIGURE 65-3. Long-term vascular access catheters. ***A.*** The Eschelon. (Courtesy of Medical Components, Harleysville, PA.) ***B.*** The Groshong. (Courtesy of C.R. Bard, Murray Hill, NJ.)

atrium, or the superior vena cava. Distance from the right atrium is an important risk factor for catheter occlusion, with the incidence of occlusion increasing the more distal the catheter tip.[3]

Indwelling central venous access devices can malfunction for a variety of reasons. The two most common types of vascular catheter complications are thrombotic occlusions and infections.[4] The etiology of the malfunction can be divided into external to the catheter and internal to the catheter. External malfunctions are, for the most part, mechanical malfunctions (e.g., catheter migration, the catheter tip abutting a vessel wall, a mural thrombus, and kinked catheters). Internal malfunctions can be divided into thrombotic (e.g., intraluminal thrombus, fibrin sheath, and fibrin tail) and nonthrombotic (e.g., drug-drug precipitate, drug-solution precipitate, insoluble salts, and lipid precipitate).[4-6] Phenytoin and diazepam cannot be given through silicone indwelling lines as they can crystallize and permanently obstruct the catheter lumen.[5] Calcium and phosphate can form an insoluble precipitate within the catheter lumen. Other medications that can precipitate are etoposide, heparin, imipenem, oxacillin, ticarcillin, and vancomycin. Infused lipids can form waxy casts within the catheter lumen.



A

B

FIGURE 65-4. Subcutaneous buried ports. ***A.*** Examples. ***B.*** Access using the Huber needle. (Courtesy of Smiths Medical, St. Paul, MN.)

INDICATIONS

Any catheter that cannot be easily flushed or aspirated requires further investigation. Defer catheter troubleshooting to the Primary Care Provider if peripheral venous access is readily available and the patient is not acutely ill due to catheter sepsis or central venous thrombosis. **It is important to consider that delaying troubleshooting may make management more difficult and therefore necessitate line replacement.** The Emergency Physician will have to address the problem if emergent or urgent access to the patient's central vascular system is required.

CONTRAINDICATIONS

Do not inject or use any device that is obviously displaced from the central circulation as it is not salvageable. Dislodging a clot or septic thrombus from a catheter tip can lead to a fatal pulmonary embolism. Avoid catheter manipulation if signs of sepsis or central venous thrombosis are present.[7] The use of indwelling dialysis lines for purposes other than dialysis is discouraged. **Only manipulate a dialysis line in a true emergency or if the line is malfunctioning and is needed for urgent hemodialysis.**

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile alcohol prep pads
- Thrombolytic agent
- Syringes, 5 mL and 10 mL
- 18 gauge needles
- Noncoring (i.e., Huber) needle
- 70% ethanol solution
- 0.1 N hydrochloric acid (HCl) solution
- Sterile saline

- Heparinized saline flush solution (100 U/mL)
- Sterile gauze squares
- Sterile gloves
- Sterile gown
- Face mask with an eye shield or goggles
- Cap
- Sterile towels or drapes

Repair kits are available to repair some externally damaged partially implanted catheters. The kits avoid the need to remove the device and implant a new catheter. Apply a smooth catheter clamp proximal to the damaged area and arrange to have the device repaired if the external tubing is damaged. The use of these kits is beyond the scope of this chapter.

THROMBOLYTIC AGENTS

The use of a specific thrombolytic agent is institution and physician specific. Streptokinase, recombinant tissue plasminogen activator (i.e., t-PA or alteplase), reteplase, and urokinase have all been successfully used to dissolve a clot within a central venous catheter.[5,8-13] **Volumes and doses of the drugs differ significantly. Pay careful attention to the medication administration. Urokinase and recombinant tissue plasminogen activator are most commonly used.** Streptokinase is more likely to lead to a hypersensitivity reaction, especially if the patient has prior exposure to it, and is therefore not recommended. Urokinase is much less expensive than t-PA. Urokinase may be purchased in concentrations of 5000 U/mL and 250,000 U/5 mL (50,000 U/mL). The concentration of 5000 U/mL is used to dissolve a clot within a catheter.

Some institutions prefer to use t-PA for this process. The Pharmacist dilutes a 50 mg vial of t-PA with 50 mL of sterile water to produce 25 syringes containing 2 mg of t-PA in 2 mL (1 mg/mL). The syringes of t-PA are then frozen until needed. It is available from the manufacturer in 2 mg single-use vials and sold as Cathflo (Genentech, South San Francisco, CA). Alteplase or t-PA (i.e., Cathflo) is currently the only Food and Drug Administration-approved thrombolytic agent in the United States indicated for the restoration of function to central venous access devices.

Standard dosing for catheter obstructions is 1 mL of urokinase (5000 U/mL), 2 mL of t-PA (1 mg/mL), or 0.4 U of reteplase.[14] The Groshong catheters (**Figure 65-3*B***) have a slit valve at their proximal end that alleviates the need for heparin. Heparinized saline is used less often today in central venous lines due to heparin-induced thrombocytopenia.

PATIENT PREPARATION

Discuss the procedure with the patient and/or their representative. Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Most patients with indwelling central venous access devices are familiar with their use and idiosyncrasies. The patient will often be able to tell the Emergency Physician if the line has had problems in the past and the method used to correct the problem. Some patients will be able to suggest postural changes (e.g., raising an arm or lying in the Trendelenburg position) that will help the catheter function better.

The chest radiograph is a good screening tool that often allows the Emergency Physician to diagnose catheter complications. Obtain a posteroanterior and lateral chest radiograph to confirm that the tip of the catheter is in a proper location. Routine chest radiographs can be eliminated in pediatric oncologic patients with asymptomatic occlusion due to their low diagnostic yield.[15] **A malpositioned catheter or one with the tip abutting the vessel wall must be removed and replaced.** A catheter whose flow changes with patient arm position may be subject to the "pinch-off" phenomenon. This may be visible as a kinking of the catheter as it passes between the clavicle and first rib. A series of chest radiographs with the arm at the patient's side and elevated may reveal the kinking or pinching-off of the catheter. These catheters require removal. A catheter may migrate intravascularly to the contralateral subclavian vein. An Interventional Radiologist may be able to insert a wire or snare into the femoral vein to grasp and reposition the catheter. Arrhythmias associated with a catheter tip positioned within the heart require the catheter to be removed and a new one inserted. The distal catheter can curve upward so that it lies within the jugular vein. An Interventional Radiologist may be able to reposition it. Otherwise, it must be removed and replaced.

Any aspiration, injection, or manipulation of a central venous line must be done using strict sterile techniques. Clean any blood, dirt, and debris from the distal port of a partially implanted device or the skin overlying the reservoir of a fully implanted device. Apply povidone iodine or chlorhexidine and allow it to dry. Wipe off the iodine (if used) with a sterile alcohol prep pad. **This process must be performed every time a needle is inserted into a central venous access device.**

TECHNIQUES

An algorithmic approach to the occluded indwelling central venous catheter is summarized in **Figure 65-5**.[10] **Never perform forced irrigation of the catheter, especially with a 1 mL syringe, to prevent the catheter from rupturing.**

A catheter that flushes easily but cannot be aspirated may have a fibrin sheath around the catheter tip forming a one-way valve. The tip may also be lodged against the wall of the superior vena cava or the right atrium. Repositioning the patient may alleviate the problem. The catheter may be cautiously used for an infusion if there are no signs of infection (e.g., discharge at the catheter or subcutaneous reservoir site, erythema, fever, or a new heart murmur) and the catheter tip is in good position. Refer the patient to their Primary Care Provider or a consultant for follow-up of the malfunction.

The problem is more serious if the catheter does not easily flush. Attempt to obtain peripheral intravenous access while attempting to correct the problem with the central venous catheter. **The Emergency Physician must decide if a prolonged effort at resolving the occlusion is necessary if there are no signs of infection and the catheter is not ruptured or malpositioned.** If so, proceed as described below and in **Figure 65-5**.

PARTIALLY IMPLANTED CATHETERS

A clot or small amount of precipitate within the partially implanted catheter (**Figures 65-1 through 65-3**) may be able to be aspirated if the catheter bore is large enough to permit passage. Remove the Luer-lock cap from the catheter. Connect a 10 mL syringe with 2 to 3 mL of sterile saline directly to the occluded port's Luer adapter. **Any clot large enough to occlude the catheter will not pass through a needle.** Apply negative pressure to the syringe. The catheter is probably occluded by a clot or a precipitate if the obstruction cannot be aspirated. Remove the syringe and attach a new Luer-lock cap.

Determine if a precipitate seen in the catheter aspirate is waxy or solid. Waxy precipitates are due to the lipid component of parenteral nutrition fluids. Waxy precipitates may be dissolved with a solution of 70% ethanol in water. Inject 1 to 2 mL of an alcohol-water solution and allow it to dwell in the catheter for 1 hour. Aspirate the catheter to determine patency. Inject 1 to 2 mL of 0.1 N hydrochloric acid

(HCl) solution if the catheter is still occluded. Allow the solution to dwell in the catheter for 20 minutes. Aspirate the catheter to determine patency. Attempt to infuse 0.1 N HCl solution two more times. The next step is to infuse a thrombolytic agent, as described below, or to replace the catheter.

Solid precipitates are due to precipitation of medications or minerals. Dilute HCl solution may be used to dissolve precipitated calcium and phosphate crystals. Infuse 1 to 2 mL of 0.1 N HCl and allow it to dwell in the catheter for 20 minutes. Aspirate the catheter to determine patency. The process may be repeated up to three times. Inject 1 to 2 mL of 70% ethanol in water and allow it to dwell in the catheter for 1 hour if it is still occluded after HCl administration. Aspirate the catheter to determine patency. The next step is to inject a thrombolytic agent, as described below, or to replace the catheter.

A clot may be present within the catheter lumen if no precipitate is present or if efforts to clear the precipitate fail. Clots probably form to some extent in most implanted central venous catheters. The clots may obstruct the catheter lumen partially or totally.[16] Thrombosis of the central veins, superior vena cava, or right atrium may occur. Suspect a major vein thrombosis if there is swelling, pain, or edema of anatomic structures that are drained by the cannulated vein(s).

Small clots may be dissolved by a bolus or infusion of a thrombolytic agent. Inject 1 mL of urokinase (5000 U/mL), 2 mL of t-PA (1 mg/mL), or 0.4 U of reteplase into the catheter. Allow the solution to dwell in the catheter for 30 minutes. Aspirate the catheter to determine patency. This process may be repeated up to three times. The medication dosing should remain the same but be divided into equal doses between the ports for catheters with multiple occluded ports. Inject 2 mL of intravenous contrast dye under fluoroscopy or inject the dye and obtain a radiograph if the catheter is still occluded after thrombolytic administration. Attempt to clear the catheter up to three times with HCl and 70% ethanol in water if no clot is present or if the contrast material will not infuse.

Consider a continuous thrombolytic infusion if a clot is present within the catheter. The infusion should occur through an intravenous line equipped with a 0.22 or 0.45 μm filter. A urokinase infusion may be begun over a 24-hour period. Administer the urokinase at a dose of 200 U/kg/hour mixed to run at a rate of at least 20 mL/hour.[9,10] A continuous t-PA infusion may also be started. Use 2 mg/20 mL as a low dose for ports, 4 mg/20 mL as a high dose for ports, or 5 mg/50 mL as a high dose for tunneled catheters. Set the t-PA infusion rate at 10 mL/hour for low and high doses for ports or 20 mL/hour for high-dose tunneled catheters. Perform thrombolytic infusions in consultation with the patient's Primary Care Provider as the patient will require hospital admission.

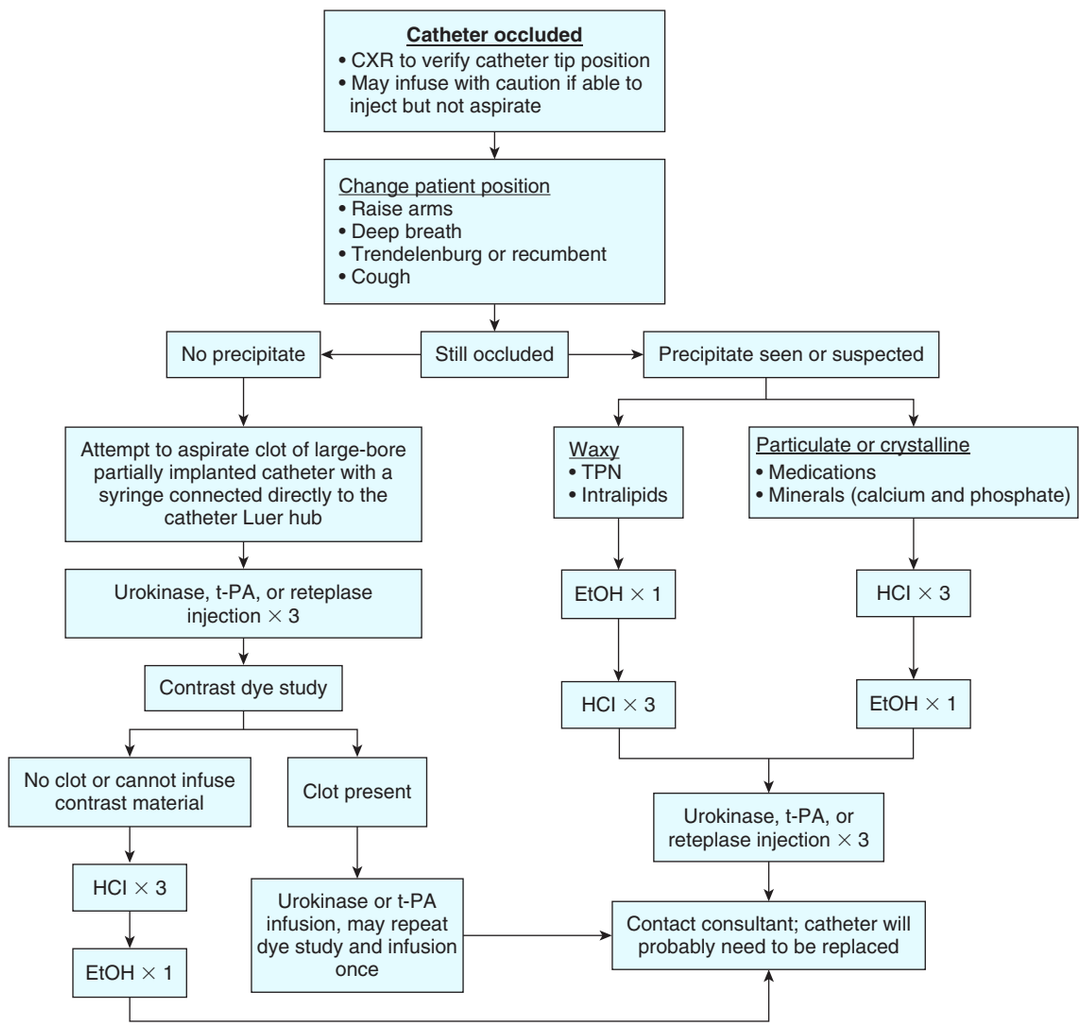


FIGURE 65-5. An algorithmic approach to the occluded central venous catheter. Continue to work down the protocol until the occlusion is resolved. CXR, chest radiograph; EtOH, 70% ethanol in water; HCl, 0.1 normal hydrochloric acid solution; TPN, total parenteral nutrition.

Consider consulting an Interventional Radiologist if these methods do not successfully clear the catheter or if a thrombolytic infusion is contraindicated. They can perform percutaneous fibrin sheath stripping, exchange the catheter over a wire, or remove and replace the malfunctioning catheter. Alternatively, the Emergency Physician may place a new central venous catheter at another site if the patient requires immediate vascular access.

FULLY IMPLANTED CATHETERS

The procedure is the same for a fully implanted central venous access device (**Figure 65-4**) with one exception. The subcutaneous reservoir will not be able to be initially cleared by aspiration. Any clot or precipitates large enough to occlude a catheter will not pass through a noncoring (Huber) needle. **Always use a noncoring (Huber) needle when aspirating or injecting through a fully implanted catheter (Figure 65-4*B*).**

AFTERCARE

Indwelling central venous lines must be flushed with saline. Do not use heparin solutions.[17,18] Assess patients given thrombolytics for bleeding at the catheter site and elsewhere prior to discharge if they do not require hospital admission. The success rate for thrombolytics restoring catheter patency are greater than 80%.[19,20] Admit patients receiving prolonged thrombolytic infusions for the infusion and for monitoring of potential bleeding complications. **Patients and their care providers must be made aware of any catheter malfunctions and the attempts made at restoring catheter patency.**

COMPLICATIONS

The complications associated with attempts to resolve an occluded catheter include catheter rupture, disconnection of the catheter from any implanted reservoir, hemorrhage, and contamination of the catheter with subsequent infection. No major bleeding episodes or deaths have been associated with intracatheter thrombolysis.[14,19,20] Assessment for the complications associated with a central venous line is discussed in Chapter 63.

SUMMARY

Numerous patients with indwelling central venous access devices will present to the Emergency Department with malfunctioning catheters. Familiarity with the strategies for diagnosing and correcting the catheter dysfunction will enable the Emergency Physician to restore the function of some, but not all, indwelling lines. Early consultation with the Primary Care Provider, Vascular Surgeon, or Interventional Radiologist is essential if the procedures outlined do not promptly restore the catheter's function.

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66 Accessing Indwelling Central Venous Lines

John Cruz and Chad Gorbatkin

INTRODUCTION

Venous access for blood sampling, hemodialysis, hydration, medication administration, and nutritional support is essential for the management of many chronic diseases.[1,2] A variety of indwelling central venous access devices have been developed to avoid frequent venipunctures and permit direct access to the central circulation. Approximately 150 million intravascular devices are currently in use in the United States.[3] These devices may be partially or completely embedded under the patient's skin (**Figures 66-1 and 66-2**). The Emergency Physician must be able to access these devices to administer medications and withdraw blood samples without causing catheter damage or catheter thrombosis. Familiarity with this process can be potentially lifesaving if the need for resuscitation becomes imminent in a patient with an indwelling line. The necessary procedures for successfully accessing indwelling devices are described in this chapter.

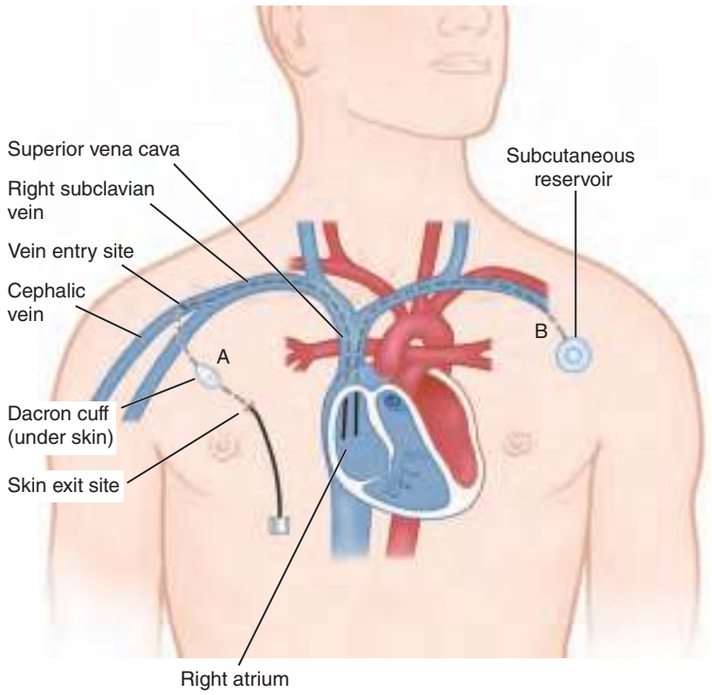


FIGURE 66-1. Indwelling central venous lines. ***A.*** The partially implanted central venous line. The distal end of the line emerges from the chest wall. Contamination of the implanted portion is prevented by a subcutaneous tunnel and a Dacron cuff around the catheter. ***B.*** The fully implanted central venous line. The catheter is connected to a reservoir that is contained in a subcutaneous pocket.

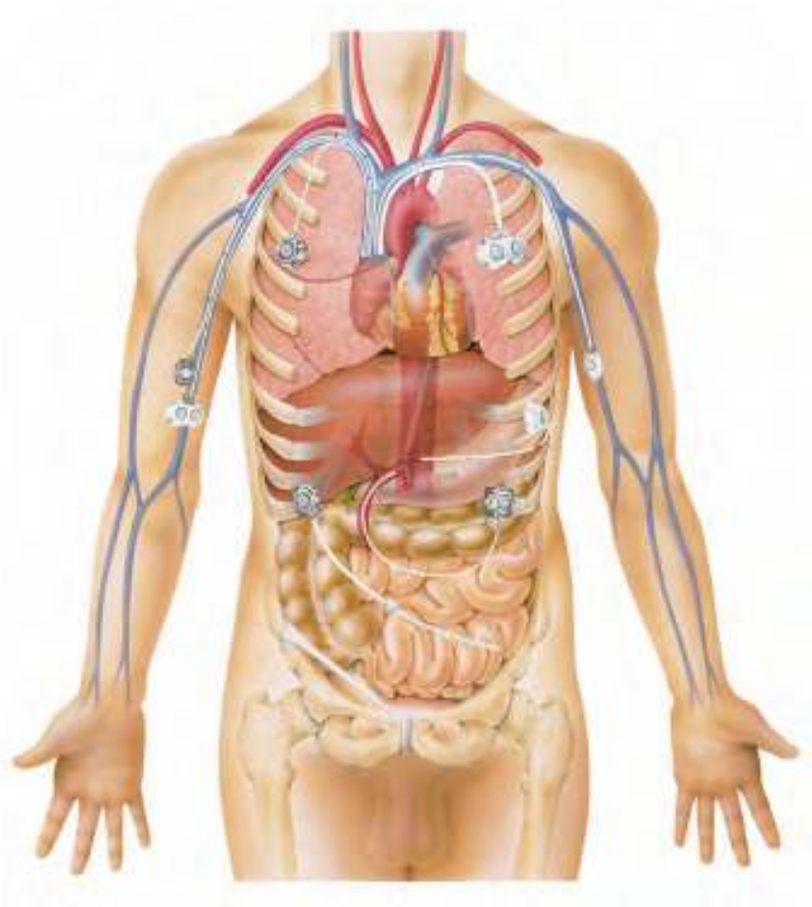


FIGURE 66-2. Indwelling subcutaneous fully implanted devices are used to access the body cavities and veins. (Courtesy of Smiths Medical, Dublin, OH.)

ANATOMY AND PATHOPHYSIOLOGY

Indwelling central venous lines allow access to the central venous circulation from a peripheral site.[4,5] This is accomplished through the end of a partially implanted catheter or through the skin into a subcutaneous reservoir of a fully implanted catheter (**Figures 66-1 and 66-2**). The proximal tip of the central venous line may lie in the superior vena cava or in the right atrium. Catheters designed for right atrial placement are made of softer and more pliable material than catheters used for short-term transcutaneous central venous access. These catheters are less likely to erode through or perforate the thin wall of the right atrium or veins.

The internal jugular, subclavian, and femoral veins can all be used as a route for a central venous line to access the vena cava or right atrium. Subclavian veins are most commonly used to maximize patient comfort and mobility. The vein is punctured transcutaneously when the line is initially inserted. The catheter is inserted into the vein, and its distal segment is tunneled under the skin. The distal end of the catheter is pulled through a small puncture at the skin exit site if the line is partially implanted (**Figure 66-1*A***). Its distal end is connected to a subcutaneous reservoir if the line is fully implanted (**Figures 66-1*B* and 66-2**).

PARTIALLY IMPLANTED CATHETERS

Partially implanted central venous catheters (**Figures 66-1*A* and 66-3**) are those in which the distal end emerges from the skin via a subcutaneous tunnel.[6] This tunnel is designed to prevent the spread of skin flora along the outside of the catheter and ultimately toward the central circulation. Most partially implanted catheters use a subcutaneous Dacron cuff to further insulate the proximal catheter from skin flora and help anchor the catheter in place.[7]

A variety of models are in use, including the most commonly used Broviac, Hickman, and Groshong catheters.[8-10] All are available in single-lumen or multiple-lumen versions. Broviac and Hickman catheters must be flushed with heparin solution twice weekly. The Broviac catheter is generally longer and thinner.[4] The larger internal diameter of the Hickman catheter is designed to allow more frequent accessing without the risk of compromising line patency.[4] The design of the Groshong catheter is unique in that it has a slit valve at its proximal end that prevents the reentry of blood once it has been flushed (**Figure 66-4**). Groshong catheters need only a weekly saline flush to prevent clot formation.

The Hemo-Cath or Perm-A-Cath is the largest bore of the right atrial catheters. It is used for chronic analgesia, hemodialysis, long-term nutritional support, and plasmapheresis. These catheters are typically flushed during dialysis. They are otherwise flushed three times per week with heparinized saline. See **Table 66-1** for review of recommended frequency and volume of flushes.

FULLY IMPLANTED CATHETERS

Fully implanted central venous catheters are entirely embedded and do not exit the skin (**Figures 66-1, 66-2, 66-5, and 66-6**). The catheter's distal end attaches to a subcutaneously implanted reservoir.[7] Various catheters (e.g., Groshong and Hickman catheters) can be attached to a subcutaneous reservoir. Most fully implanted central venous access systems are known by the brand names (e.g., Infuse-A-Port and Port-A-Cath). Most manufacturers recommend flushing these catheters with a heparin solution monthly when not in use.[10] Flushing of the port system can be done every 3 months without any increase in complications.[10] The reservoir's infusion port is covered with a self-sealing silicone rubber membrane (**Figures 66–5 and 66–6**). **Specially designed noncoring Huber needles must be used when accessing the subcutaneous reservoir to avoid permanent fenestration of the self-sealing membrane (Figure 66-7).**

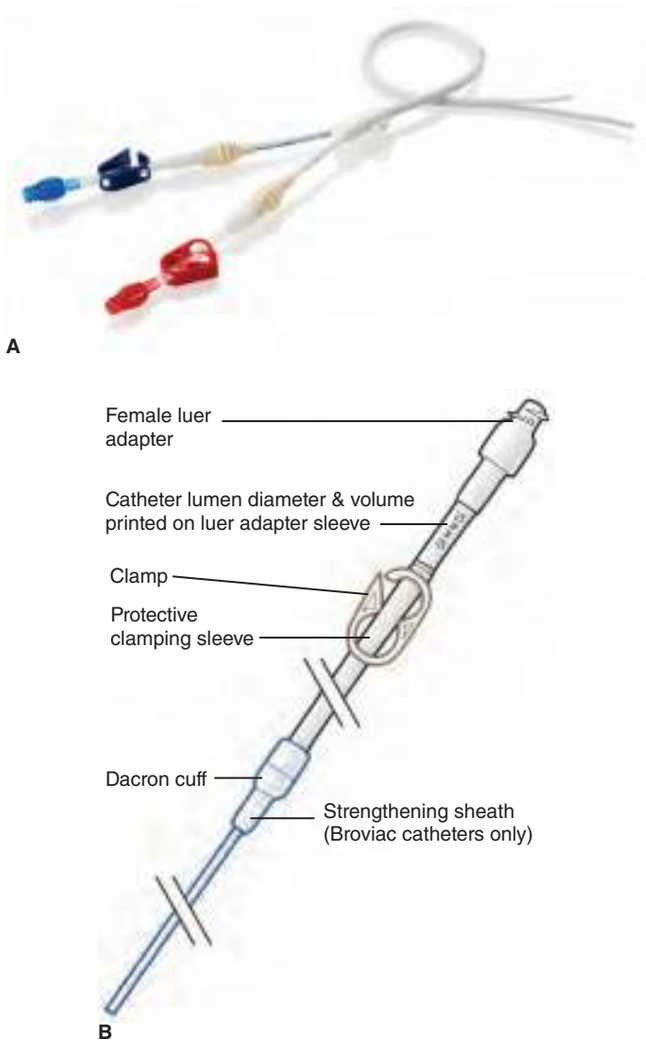


FIGURE 66-3. The partially implanted central venous catheter. ***A.*** An example of a dual-lumen catheter. (Courtesy of Medical Components Inc., Harleysville, PA.) ***B.*** A single-lumen catheter is shown schematically. The Dacron cuff lies in the subcutaneous tissue just proximal to the skin entry site. The catheter tip lies in either the proximal superior vena cava or the right atrium.

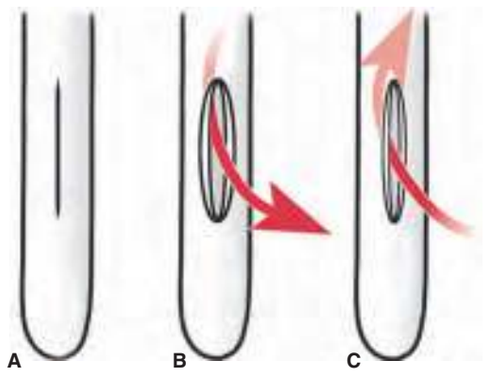


FIGURE 66-4. The Groshong catheter tip. Detail of the Groshong three-position slit valve that prevents venous blood from passively entering the catheter when it is not in use. ***A.*** The closed or resting position of the slit valve. ***B.*** The valve opens outward from positive pressure when the catheter is flushed or infused. ***C.*** The valve opens inward from negative pressure when the catheter is aspirated.

TABLE 66-1 Volumes and Concentrations of Heparin Flushes for Common Indwelling Catheter Devices in Adults

Type of central line	Volume of recommended flush	Recommended frequency of flush
Broviac Catheter	3–5 mL of heparinized saline (100 U/mL)	After each use Twice per week
Groshong Catheter	5 mL of heparinized saline (100 U/mL)	After each use Once per week
Hemo-Cath/Perm-A-Cath	≤ 2 mL of heparinized saline (1000 U/mL)	After each use 3 times per week
Hickman Catheter	3–5 mL of heparinized saline (100 U/mL)	After each use Twice per week
Port-A-Cath/Infuse-A-Port	5 mL of heparinized saline (100 U/mL)	After each use Monthly

PERIPHERALLY INSERTED CENTRAL CATHETERS

Peripherally inserted central catheters (i.e., PICC lines, **Figure 66-2**, and Chapter 62) are inserted into the antecubital, brachial, or cephalic vein of the upper extremity and advanced into the subclavian vein. PICC lines are available in single- and double-lumen configurations based on the indication for placement. PICC lines are made of either silicone or polyurethane. They are 50 to 60 cm long and have an outside diameter of 2 to 7 French, with the 5 French being most commonly used. Some PICC catheters are equipped with a Dacron cuff. Advantages to PICC placement include their ability to be inserted in outpatient settings, ease of placement, ease of maintenance, and ultimately, removal upon cessation of the indication for which it was placed. The greatest drawback for the use of PICC lines is the variable rate of catheter-related upper extremity thrombosis. Greater than 50% thrombosis rates have been noted in patients receiving chemotherapy.[11]

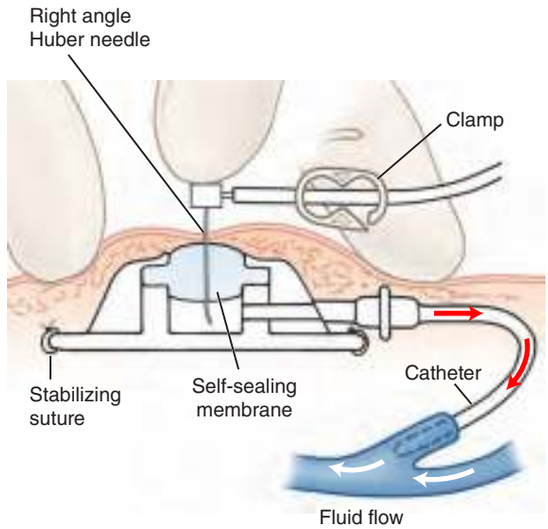
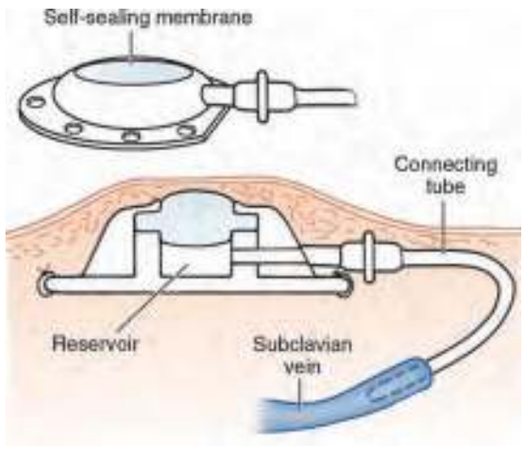


FIGURE 66-6. Accessing the fully implanted central venous catheter system. The reservoir is stabilized between the fingers of the nondominant hand as a noncoring (Huber) needle is used to penetrate the skin and reservoir.

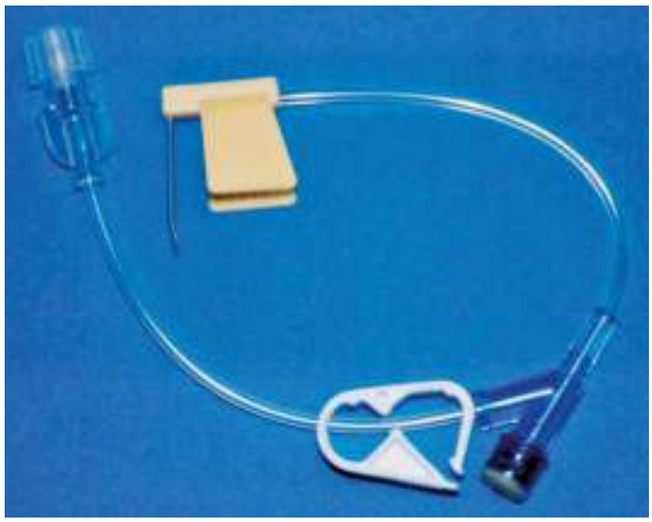


A

B

C

FIGURE 66-5. The fully implanted central venous catheter. ***A.*** The reservoir lies in the subcutaneous tissue and is anchored with sutures to keep the diaphragm facing the skin surface. ***B.*** An example of a single reservoir. ***C.*** An example of a dual reservoir. (Photos courtesy of Medical Components Inc., Harleysville, PA.)



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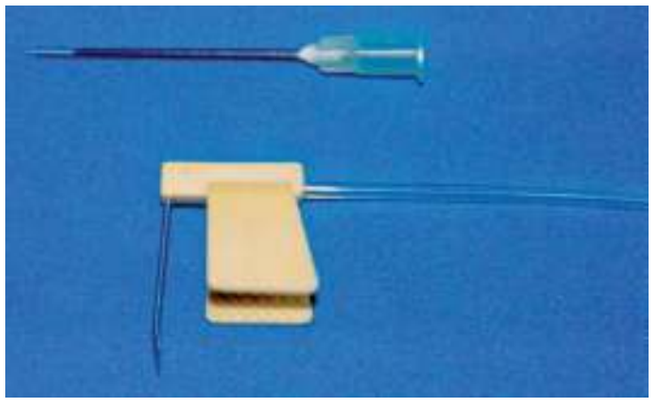

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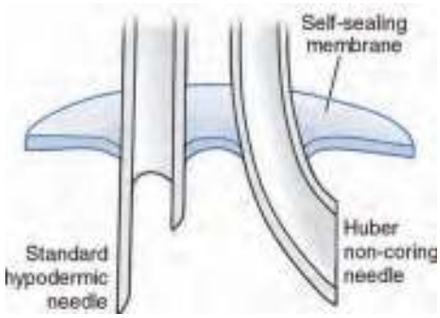

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FIGURE 66-7. The Huber needle. ***A.*** Photo of a complete right-angle Huber needle set including the extension tubing and clamp. ***B.*** Photo showing the right-angle Huber needle compared with a standard hypodermic needle. ***C.*** Drawing showing the key difference between a standard hypodermic needle and the Huber needle. The hypodermic needle tip can cut a cylindrical core of subcutaneous tissue and the diaphragm sealing the subcutaneous reservoir. The Huber needle pushes the subcutaneous tissue and the diaphragm material aside without removing any of it.

PERCUTANEOUS CENTRAL VENOUS CATHETER

A device similar to the PICC that is used exclusively in the neonate is the percutaneous central venous catheter (PCVC). This catheter is primarily used in the Neonatal Intensive Care Unit and is uncommonly encountered in the Emergency Department. PCVCs are often implanted for nutritional support in very-low-birth-weight infants, infants for whom feedings are contraindicated, and prolonged vascular access in critically ill patients.[12] They are available in three sizes (i.e., 1.1, 1.9, and 2.8 French). The 2.8 French size is used more often because the smaller sizes tend to become occluded with a thrombus. Accessing the device follows the same general principles outlined below. This includes adhering meticulously to aseptic technique by cleansing the port with alcohol or chlorhexidine prior to attaching a syringe and ensuring the extension tubing is clamped when not withdrawing or infusing to prevent development of an air embolism. Additional considerations include ensuring a syringe greater than 10 mL is used to infuse or withdraw to prevent damage to the catheter or the underlying vessel by generating too great of a pressure gradient. Emergency Physicians are advised to utilize pulsatile motion when flushing for similar reasons.[13] The authors recommend an expert consultation for accessing this device if encountered.

INDICATIONS

PATIENTS REQUIRING INDWELLING CENTRAL VENOUS LINES

Patients of all ages and with a variety of diagnoses may present with an indwelling central venous line. Examples include chronic conditions requiring frequent parenteral analgesia (e.g., sickle cell disease), chronic infections requiring long-term parenteral antibiotics (e.g., endocarditis, osteomyelitis), and the need for prolonged and frequent access (e.g., chemotherapy, frequent blood sampling, frequent medication infusion, and hyperalimentation). Any patient who requires several weeks of repeated intravenous blood sampling and/or drug administration may be a candidate for an indwelling central venous line.

ACCESS OF INDWELLING CENTRAL VENOUS LINES

Fully or partially implanted central venous access devices may be accessed routinely when intravenous fluids must be administered, medications must be administered, or phlebotomy is required.

CONTRAINDICATIONS

Do not access an indwelling central venous device if peripheral intravenous access can be obtained. The use of bedside ultrasound may facilitate peripheral intravenous access and avoid accessing the indwelling device (Chapter 64). **Always consider the potential risk of damage to the implanted line prior to proceeding with accessing it.** Do not access fully implanted devices through infected overlying skin. Use partially implanted catheters with known or suspected infection cautiously as they may become a source of septic emboli. It is sometimes possible to treat catheter-related infections without requiring removal of the device.[14,15] **Phenytoin and diazepam cannot be given via silicone indwelling central venous lines.** They can crystallize and permanently obstruct the catheter lumen.[16]

Access devices used for hemodialysis only in a true emergency and if no other venous access can be readily obtained. This guideline is intended to prevent loss of the patient's dialysis access due to device damage, infection, or thrombosis. An inability to dialyze the patient in a timely manner may lead to significant morbidity and mortality.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile gauze, 4×4 squares
- 10 mL syringes
- 20 gauge needles
- Sterile 0.9% saline
- Heparinized saline flush (e.g., 100 and 1000 U/mL)
- Adhesive tape
- Luer-lock caps
- Blood collection tubes
- Infusion set
- Huber needle, noncoring right angle or straight angle
- Topical anesthetic (e.g., EMLA cream, ethyl chloride spray, or ice)
- Injectable anesthetic without epinephrine (e.g., 1% lidocaine)
- Sterile alcohol prep pads
- Sterile gloves
- Sterile drapes

PATIENT PREPARATION

Discuss the necessary procedure with the patient and/or their representative. Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Consider involving the patient in the decision to perform the procedure. Patients with indwelling lines are usually quite familiar with the care and use of their devices. Patients can often advise the Emergency Physician on the correct procedure, the appropriate flush solution, and any anatomic manipulations necessary to optimize flow through the line (e.g., raising the arms, turning the head). **Sterile technique is required when accessing indwelling central venous catheters**.

Prepare the skin over implanted catheters or the distal portion of partially implanted catheters. Remove any tape from the area. Clean the skin or distal catheter of any blood, debris, or dirt. Apply chlorhexidine solution or povidone iodine and allow it to dry. Drape around the area to delineate a sterile field. Remove any iodine residue with an alcohol pad.

TECHNIQUES

PARTIALLY IMPLANTED CATHETERS

Accessing a partially implanted central venous catheter is simple and similar to accessing a heparin-locked peripheral intravenous catheter.[10,16] Remove any adhesive tape and gauze wrapped around the distal end of the lumen to be accessed. Fasten the catheter clamp on the desired lumen (**Figure 66-3**). Clean the catheter cap and Luer adapter with povidone iodine or chlorhexidine solution and allow it to dry. The technique for accessing the catheter will vary depending on whether blood sampling with or without a subsequent infusion is required. Both techniques are described below.

BLOOD SAMPLING FROM PARTIALLY IMPLANTED CATHETERS

Blood samples may be withdrawn through the catheter cap using a 20 gauge hypodermic needle attached to a 10 mL syringe if blood is to be sampled without beginning an infusion. **Avoid using syringes smaller than 5 mL as they create a larger pressure gradient upon infusion and risk potential damage to the catheter.**[4] Insert the needle through the Luer cap. Open the catheter clamp. Withdraw 5 mL of blood from the catheter. Discard the blood sample, needle, and syringe. This blood sample is considered contaminated by the catheter contents (i.e., saline or heparinized saline) and does not represent circulating blood. This step is essential when accessing dialysis catheters as they may contain concentrated heparin (e.g., 1000 U/mL). Gently flush the catheter with 2 to 3 mL of sterile saline if unable to aspirate blood. Refer to Chapter 65 for further troubleshooting instructions if the catheter does not flush easily.

Withdraw the required blood samples using a new needle and syringe inserted through the Luer cap. Carefully transfer the blood samples into collection tubes for the laboratory. The catheter must now be flushed to prevent clot formation. Flush the catheter with the appropriate solution using a 10 mL syringe armed with a 20 gauge needle. Flush the indwelling line with the appropriate volume and concentration of heparin solution to prevent thrombosis (**Table 66-2**). Wipe off the cap with an alcohol pad.

Secure the free end of the catheter. Tape the catheter to the patient's chest wall to prevent accidental traction on the catheter. Evaluate the skin puncture site to ensure the line has not been dislodged. Reapply a dressing over the skin puncture site if necessary.

TABLE 66-2 Volumes and Concentrations of Heparin Flushes for Pediatric Indwelling Devices

	Children < 6 months old	Children > 6 months old
Partially implanted device	3 mL heparin (10 U/mL)	5 mL heparin (100 U/mL)
PICC line	3 mL heparin (10 U/mL)	5 mL heparin (100 U/mL)
Fully implanted device	3 mL heparin (10 U/mL)	5 mL heparin (100 U/mL)

BLOOD SAMPLING AND INFUSION THROUGH PARTIALLY IMPLANTED CATHETERS

The Luer catheter cap can be removed entirely and the catheter lumen accessed directly with a Luer-hub syringe if an infusion is to be subsequently started. **Ensure that the catheter clamp is securely closed**. Remove the cap from the catheter. Attach a 10 mL syringe to the hub of the catheter. Open the catheter clamp. Withdraw 5 mL of blood into the syringe. Close the catheter clamp. Remove and discard the syringe with the contaminated blood sample. Apply a new syringe, open the catheter clamp, and withdraw the required aliquot of blood. Close the catheter clamp before removing the syringe. Continue this sequence of events until all required blood samples are obtained. **It is imperative to ensure that the catheter is clamped each time the cap or syringe is removed to prevent a potentially life-threatening air embolism**. Securely attach primed intravenous tubing to the hub of the catheter, open the catheter clamp, and begin the infusion.

Clamp the catheter lumen when terminating the infusion or if no infusion is to be started. Remove the intravenous tubing or the syringe from the catheter. Attach a syringe containing the appropriate flush solution, open the catheter clamp, and flush the catheter. Close the catheter clamp. Remove the syringe. Apply a new sterile cap onto the hub of the catheter. **Never use "needle-less" caps as they are a potential source of air emboli**. Upon completion of the infusion, open the catheter clamp to prevent catheter damage from prolonged clamping. Secure the catheter to the patient's chest wall.

FULLY IMPLANTED CATHETERS

A noncoring Huber-type needle (Figures 66-6 and 66-7) must be used to access subcutaneous injection ports to prevent permanent fenestration. A small-gauge standard hypodermic needle should only be considered for emergent indications if a noncoring needle is not immediately available. Damage to the diaphragm can lead to subcutaneous hemorrhage and necessitate surgical replacement of the implanted device.

Clean and prep the skin overlying the injection port with povidone iodine or chlorhexidine solution and allow it to dry. The application of a topical or injectable anesthetic over the reservoir is optional but often greatly appreciated by the patient. Remove the iodine solution from the skin with an alcohol swab. Flush the Huber needle and extension tubing with normal saline using a 10 mL syringe. Leave the syringe attached. Locate the center of the self-sealing membrane or diaphragm. Stabilize the reservoir with the nondominant hand (**Figure 66-6**). Slowly and steadily insert the needle through the skin and into the reservoir (**Figure 66-6**). Stop advancing the needle when it touches the far wall of the device. Gently flush 2 to 3 mL of saline through the needle to ensure proper placement. Refer to Chapter 65 for troubleshooting instructions if the catheter does not flush easily.

Secure the Huber needle in place if the catheter can be flushed easily. Stabilize it with gauze squares and tape. Withdraw 5 to 10 mL of blood. Close the clamp on the extension tubing of the Huber needle (**Figure 66-6**). Remove and discard the syringe. Attach a new syringe, open the clamp, and withdraw the required blood samples. Clamp the extension tubing and remove the syringe. Attach the primed intravenous tubing if an infusion is to be started and begin the infusion. Clamp the extension tubing if no infusion is desired or when discontinuing an infusion and disconnect the intravenous tubing. Attach a 5 or 10 mL syringe containing heparinized saline (e.g., 100 to 200 U/mL) to the Huber needle extension tubing. Open the clamp and flush the device with 3 to 5 mL of heparinized saline. Flush with saline if a Groshong catheter is attached to the reservoir. Remove the Huber needle from the skin. Control any skin bleeding with direct pressure. Apply a sterile dressing.

An alternative to the Huber needle is the Gripper Safety Needle (Smiths Medical, Dublin, OH). This is a noncoring safety needle mounted in a plastic base (**Figure 66-8**). The sharp inserter is removed to leave the small blunt cannula in the reservoir. The foam pad against the skin is convenient for health care personnel and comfortable for the patient. The device comes in numerous sizes and needle gauges.

PEDIATRIC CONSIDERATIONS

The management of pediatric indwelling devices is similar to that in adults with a few exceptions. The catheters, reservoirs, and tubing are often smaller than those used in adults or adolescents. The volumes and concentrations of heparin flushes differ in these smaller catheters (**Table 66-2**). The external portion of partially implanted devices and PICC lines must be properly secured with a dressing to ensure that the child does not dislodge the device, intentionally or otherwise. Consider expert consultation in the unlikely event that a PCVC is encountered in the Emergency Department.

OTHER INDWELLING DEVICES

Subcutaneous reservoirs may be attached to peripheral veins and body cavities for the same indications (**Figure 66-2**). These devices may have to be accessed in the Emergency Department. The method for access is the same as described above.

ASSESSMENT

Assessment of line function after accessing a central venous access device will not occur until the next access attempt. Troubleshooting nonfunctioning indwelling central venous lines is discussed in Chapter 65.

AFTERCARE

Secure the partially implanted catheter to the skin with tape to minimize the risk of dislodgement. Give the patient a written record of how the access device was used and flushed in the Emergency Department to convey to their Primary Care Physician should problems with the line become evident later. Document this also in the medical record. Patients must regularly assess their indwelling access sites for signs of infection (e.g., erythema, pain, purulent discharge, or serous discharge). Instruct the patient to immediately contact their Primary Care Provider or return to the Emergency Department if they develop a fever or other signs concerning for infection.

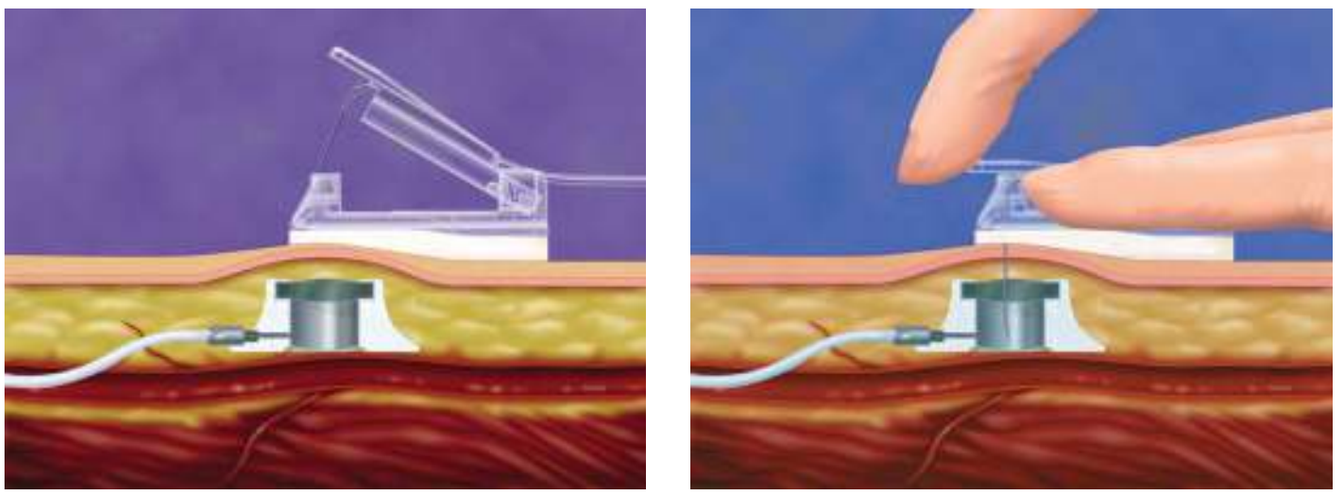

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FIGURE 66-8. The Gripper Safety Needle. ***A.*** Positioning over the reservoir. ***B.*** The safety needle inserted into the reservoir. (Courtesy of Smiths Medical, Dublin, OH.)

TABLE 66-3 Recommendations to Prevent Infections

An assistant watches procedure and stops it if aseptic technique is breached
Change administration sets per hospital policy
Change dressing if dirty, loose, or wet
Cover with transparent dressings
Daily chlorhexidine baths for the patient
Disinfect using mechanical friction
Educate the patient on proper care
Maintain aseptic technique
Minimize access
Only access devices with proper equipment
Only access if necessary
Only insert if necessary
Remove for complications
Remove for signs of local skin infection
Remove if no longer needed
Use an insertion checklist
Use approved skin disinfectant solutions
Use chlorhexidine-impregnated dressings
Wash hands before accessing, dressing, inserting
Wash hands before touching the skin access site

COMPLICATIONS

Two of the most important considerations while accessing indwelling lines are to reduce the risk of thrombus formation and infection. Strict adherence to the procedures described above will minimize the chances of these problems. There are many recommendations to prevent an infection (**Table 66-3**).[1] Complications associated with the presence of the catheter include catheter-related infections, pulmonary embolism, right atrial erosion with pericardial tamponade (rare with implanted lines), and right atrial thromboses.[10,15,17-19] Fully implanted catheters may become disconnected from the subcutaneous reservoir or may leak into the subcutaneous tissue due to diaphragm failure. Any hematoma formation near the reservoir must be assessed promptly to prevent major hemorrhage.

Suspect an air embolism immediately if the patient becomes confused, hypotensive, and/or tachycardic while accessing the central venous access device. This complication is preventable by ensuring that the catheter tubing is clamped whenever the end of the tubing is being accessed or uncapped. Never attach needle-less tubing to the device. Immediately place the patient in the Trendelenburg position with the left lateral decubitus position if air embolism is suspected. This positioning will hopefully cause air emboli to collect in the apex of the right ventricle instead of entering the pulmonary artery.

Repair kits are available to repair damaged partially implanted catheters. These kits may obviate the need for device removal and replacement if successful. Apply a smooth catheter clamp proximal to the damaged area if the external tubing of the partially implanted catheter is damaged. Arrange to have the device repaired as soon as possible. Instructions for the use of these kits are beyond the scope of this chapter.

SUMMARY

The Emergency Physician will encounter many patients with indwelling central venous catheters. Careful adherence to sterile technique, proper blood sampling technique, and proper infusion techniques will allow access for fluid administration, medication administration, and phlebotomy while preserving the indwelling line for future use and minimizing risk to the patient.

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67 Pulmonary Artery (Swan-Ganz) Catheterization

J. Elizabeth Neuman and Jessica Mann

INTRODUCTION

The concept of invasive hemodynamic monitoring was first pioneered in the mid-1800s. After that time, various devices and techniques evolved until Swan and Ganz developed the balloon-tipped pulmonary artery catheter (PAC) in 1970.[1-3] The "Swan-Ganz catheter" has been used almost exclusively in the intensive

care setting ever since, with an estimated 1 million PACs placed annually at a cost of approximately 2 billion dollars.[4,5] The use of the PAC has allowed clinicians to measure right atrial, pulmonary artery, and pulmonary capillary wedge pressures at the bedside.[6] Although advanced hemodynamic monitoring would seem intuitively to help clinicians in the management of unstable patients, the PAC has not been found to improve survival, and its use has declined significantly over the past 20 years.[7] While the utility of the PAC has become less clear, particularly with advances in less invasive monitoring techniques, it is still used in complex cases.[8] This chapter focuses on the actual technique of placement of the PAC and is predicated on the ability of the Emergency Physician (EP) to gain central venous access (Chapters 63 and 64).

ANATOMY AND PATHOPHYSIOLOGY

Central access is generally obtained via the right internal jugular or left subclavian vein. Using either of these access points allows the easiest passage of the PAC into the right atrium (RA) from the superior vena cava (SVC) (**Figure 67-1**). The left internal jugular, right subclavian, or femoral veins may be used but are more technically challenging. Use of the flexible balloon-tipped catheter allows it to "float" with the blood flow through the tricuspid valve into the right ventricle (RV). From there, the catheter travels through the RV outflow tract, through the pulmonic valve, and into the pulmonary artery (PA). Direct measurements of central venous pressure (CVP), right ventricular pressure, pulmonary artery pressure (PAP), pulmonary artery wedge pressure (PAWP), and mixed venous saturation can be made.[9] The ability to measure the central venous oxygen saturation ($ScvO_2$) provides of the most crucial pieces of information to the EP. This represents the balance of oxygen delivery versus consumption and is critically important in the management of patients in shock.

The balloon of the PAC is inflated and "wedges" into a branch of the PA to occlude blood flow into that segment (**Figure 67-2**). The catheter, therefore, measures the downstream pressure in the vessel. This PAWP has been shown to correlate closely with left atrial pressure (LAP).[7] During diastole, the mitral valve is open and the LAP

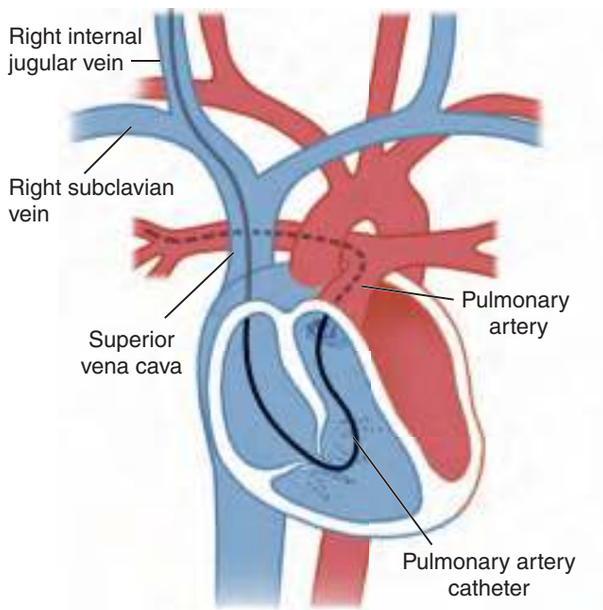

FIGURE 67-1. Cardiac anatomy as it pertains to PAC insertion. The PAC enters the right atrium from the superior vena cava, crosses the tricuspid valve into the right ventricle, and then crosses the pulmonic valve into the pulmonary artery. The catheter tip lies in a branch of the right pulmonary artery.

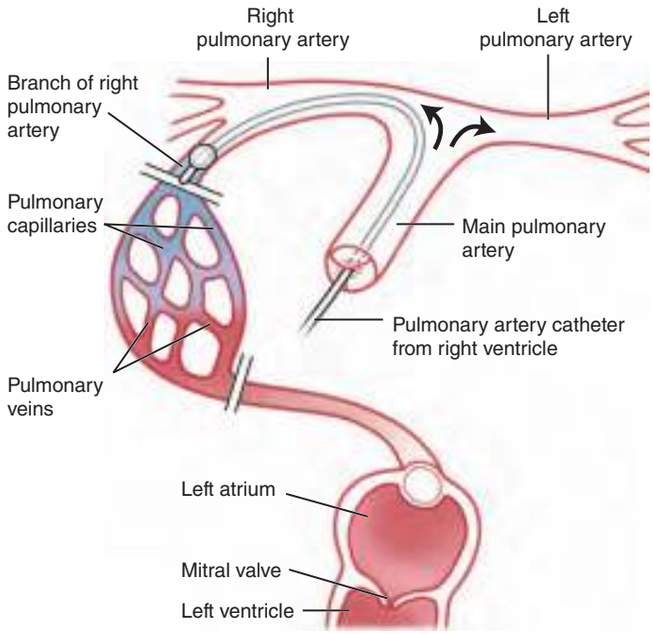

FIGURE 67-2. Diagram of the principle underlying pulmonary capillary wedge pressure. Balloon inflation blocks transmission of the forward pulmonary artery pressure to the tip of the catheter. The catheter tip therefore measures the downstream pressure of the pulmonary circulation. Because the pulmonary circulation has no valves, the pressure measured at the catheter tip is equal to the pressure in the left ventricle when the mitral valve is open (diastole).

is equal to left ventricular end-diastolic pressure (LVEDP). Knowing the LVEDP is important because it provides the best estimate of preload, allows for the assessment of left ventricular filling, and can theoretically help guide the resuscitation.[9]

The PAC may also be used to measure the cardiac output using the thermodilution method. The tip of the PAC has a temperature-sensitive probe. When a given volume of cold saline is injected into the right atrial port of the PAC, it cools the temperature of the blood flowing past the catheter tip. If the cardiac output is high, the cold saline is mixed with and carried along by a larger flow of blood so that the temperature change detected at the PAC tip is smaller and dissipates faster. If cardiac output is low, the cold saline mixes with a smaller volume of blood, and the temperature change is more apparent and slower to dissipate.

INDICATIONS

Classically, the PAC has been used in the critical care setting to aid in the management of patients with sepsis, left ventricular dysfunction, myocardial ischemia, and multiple other causes of hemodynamic instability. Measuring cardiac output (CO) and $ScvO_2$ helps guide therapy, but there are no data to suggest that the use of the PAC improves outcomes.[10-14] A 2013 Cochrane review found 13 randomized controlled trials totaling 5686 patients.[15] It found no difference in death, intensive care unit length of stay, or hospital length of stay between patients in whom the PAC was and was not used.[15] As with any intervention, the EP must carefully assess the potential benefits and risks in making the decision to place a PAC. **Table 67-1** lists the most widely accepted indications for the use of a PAC.

CONTRAINDICATIONS

Contraindications to actual placement of a PAC include refusal of consent, infection at the insertion site, and the presence of a right ventricular assist device. Placement of the PAC is contraindicated

TABLE 67-1 Common Clinical Indications for Pulmonary Artery Catheter Placement in Medical and Surgical Patients

Cardiac

A. Complicated myocardial infarction
   i. Management of refractory hypotension or left ventricular failure
   ii. In presence of hemodynamic deterioration due to a mechanical complication, to differentiate mitral regurgitation from acute ventricular septal defect
B. Other cardiac conditions
   i. Diagnose/manage cardiac tamponade
   ii. Distinguish cardiogenic from noncardiogenic pulmonary edema
   iii. Management of severe cardiomyopathy
   iv. Diagnose/manage severe pulmonary hypertension

Medical/Surgical

In the setting of sepsis, trauma, burns, multiple organ failure, pulmonary embolus, or drug overdose.

If any of the following is found to be unresponsive to conventional medical management:

A. Hypotension
B. Low urine output
C. Hypoperfusion (evidenced by cool skin, mental obtundation, lactic acidosis)
D. Severe hypoxemia requiring high levels of positive end-expiratory pressure (> 10 cm)

Preoperative

A. High-risk cardiac surgery (e.g., coronary artery bypass graft in elderly patients, multiple valve replacement, ventricular aneurysm resection)
B. Complicated vascular surgery (dissecting aneurysm, resection of thoracic or abdominal aneurysm)
C. Other surgical patients with multiple risk factors
   i. Myocardial infarction within 6 months
   ii. Poor left ventricular function
   iii. Elevated Goldman or American Society of Anesthesiologists score

if less invasive monitoring options (e.g., echocardiography) will provide the EP with the information needed. Competence of the EP to place, use, and interpret the data provided by the PAC and the availability of required equipment are prerequisites. Laboratory evaluation should be undertaken prior to the procedure because electrolyte abnormalities and severe acid-base disturbances can predispose to dysrhythmias. Risk versus benefit must be weighed carefully in cases of coagulopathy, bleeding diathesis, or known right heart thrombus. Routine placement of PAC prior to low-risk operative procedures is not recommended per the latest American Society of Anesthesiologists task force on PA catheterization.[16]

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile gloves and gown
- Face mask and cap
- Saline or dextrose solution with or without heparin (1 to 2 U/mL)
- Pressure bag with manometer
- Pressure tubing
- Pressure transducer for distal port (CVP port optional)
- Stopcocks and occlusive caps for each port of the PAC
- Fluid infusion tubing for sheath sideport and for PAC ports
- PAC
- Balloon inflation syringe
- Catheter sleeve
- Sterile dressing for site
- Electrocardiogram (ECG) and pressure monitor

Insertion of the PAC requires placement of a percutaneous introducer sheath, typically 8.5 French in size (Chapters 63 and 64).[17]

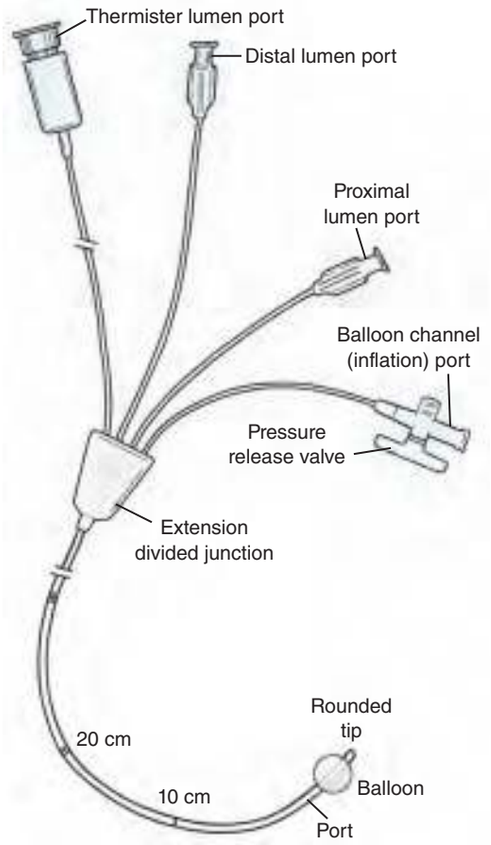

FIGURE 67-3. The pulmonary artery catheter.

Please refer to Chapter 63 for a detailed review of introducer sheath placement. The PAC is placed in a sterile sleeve that allows for aseptic manipulation of the catheter.[14] The PAC itself (**Figure 67-3**) is a flexible, balloon-tipped catheter that typically is able to accommodate a cardiac pacing wire. The catheter is a triple-lumen catheter with a proximal port/lumen at about 30 cm from the tip, a thermistor used to calculate CO, a balloon just proximal to the tip, and a distal lumen that opens at the end of the catheter. The catheter is marked in 5 cm increments to guide insertion. Each catheter comes with a 3 mL syringe that is used to inflate the balloon.

PATIENT PREPARATION

Preparation plays a very important role in any procedure. Routine laboratory studies are advisable prior to PAC insertion in nonemergent circumstances. Hematologic abnormalities (e.g., severe anemia, thrombocytopenia, and coagulation system deficiencies) can increase the risk or adverse consequences of bleeding. Electrolyte derangements (e.g., hyperkalemia, hypokalemia, and hypomagnesemia) that may predispose to arrhythmias should be identified and corrected when possible.

Explain to the patient and/or their representative the risks, benefits, and complications of the procedure. Obtain informed consent for the procedure if possible. The use of mild sedation may be advantageous in some patients. Sedation may be required in the appropriate clinical setting if the patient cannot lie still for the procedure. Place the patient supine if possible. Continuous ECG monitoring is essential. Pulse oximetry should be routinely monitored. Arterial pressure monitoring is often desirable in patients receiving a PAC. Apply supplemental oxygen. Equipment and personnel

necessary for assisting with the PAC insertion procedure and for managing potential complications should be immediately available. This should include equipment for emergency airway management and emergency cardiac pacing.

The choice of which central venous access site to use for PAC insertion must be individualized. The preferred sites are the right internal jugular vein or the left subclavian vein. The PAC tends to float into the desired position more easily from these two sites. The left internal jugular vein and the right subclavian vein are acceptable alternatives. The femoral vein may also be used. The femoral approach may be quite difficult without fluoroscopic guidance of the catheter. The external jugular veins, basilic veins, and axillary veins are additional alternatives that carry the same difficulty.

It is essential that strict sterile technique be maintained throughout the insertion procedure. The EP and their assistant should be wearing sterile gloves, a sterile gown, a face mask, and a cap. A large sterile field is necessary, as is close attention to the long PAC, which can easily become contaminated. It is also very important to take all necessary precautions with syringe needles, scalpel blades, and suture needles to prevent a needlestick injury.

Insert a percutaneous introducer sheath into the central venous system (Chapters 63 and 64). If the patient already has a single-lumen or multilumen central venous catheter inserted, it may be exchanged for an introducer sheath. Remove any bandages and dressings on the catheter and skin access site. Thoroughly prep the catheter, skin access site, and surrounding skin with povidone iodine or chlorhexidine solution and allow it to dry. Drape a sterile field. Discontinue any infusions through the catheter. Open an introducer sheath kit. Cut any sutures securing the catheter to the skin. Insert a guidewire through the hub of the distal port and into the central venous circulation. Withdraw the catheter over the guidewire. Insert and secure the percutaneous sheath over the guidewire.

Take the time to reassure the awake patient and explain what will be happening. Patients in general will be draped and their faces covered, which can be frightening. Verbal reassurance should be frequent, and anxiolysis may be necessary to ensure that they stay still.

TECHNIQUE

Set up a bedside sterile table and open the PAC kit. Remove the protective sleeve. It allows later repositioning of the PAC while maintaining sterility. Place the sleeve over the catheter and slide it far back (> 60 cm) from the catheter tip. Attach the balloon inflation syringe to the PAC. Inflate the balloon with 1.5 mL of air to confirm the integrity of the balloon. It is a good idea to inflate the balloon in a full bowl of sterile saline. Observe for air bubbles to ensure that there are no leaks or gross eccentricities. Check the balloon to ensure it inflates properly and symmetrically and deflates adequately.[18] Allow the balloon to deflate passively. **Deflation by aspirating air from the balloon should be avoided as it places undue stress on the balloon and increases the risk of rupture.**

Flush the PAC ports with sterile saline and attach a stopcock to each port. Attach the pressure tubing to the distal port. Flush the entire apparatus, including the PAC and the pressure monitoring system, with sterile saline to ensure that no air remains in any part of the system. Have an assistant set up, calibrate, and level the transducer. The transducer must be leveled to ensure pressures are measured against a standard reference level.[18] Hand the proximal end of the PAC to an assistant to attach to the ECG monitor. Finally, shake the tip of the PAC while observing the pressure waveform on the monitor to confirm that the monitoring system is operative.[18]

Prior to the insertion, allow the PAC to rest in its natural curvature. Insert the PAC through the diaphragm on the introducer sheath, taking care to orient the natural curve of the catheter toward the RV outflow tract. Continue to advance the PAC until it is inserted 10 to 15 cm and exits the sheath. **This ensures that the balloon is not inflated within the sheath. Stop advancing the PAC.** Inflate 1.5 mL of air into the balloon and lock the pressure release valve. **The PAC should never be advanced through the central venous system with the balloon deflated, as this may provoke ectopy, injure the heart, or injure other vascular structures. Conversely, the PAC should always be withdrawn with the balloon deflated.**

Pay close attention to the distance markings on the PAC and to the pressure waveform on the monitor when advancing the PAC. Typical pressure waveforms are illustrated in **Figure 67-4**. The average distances from the different catheter insertion sites into each chamber of the heart are listed in **Table 67-2**. Advance the PAC

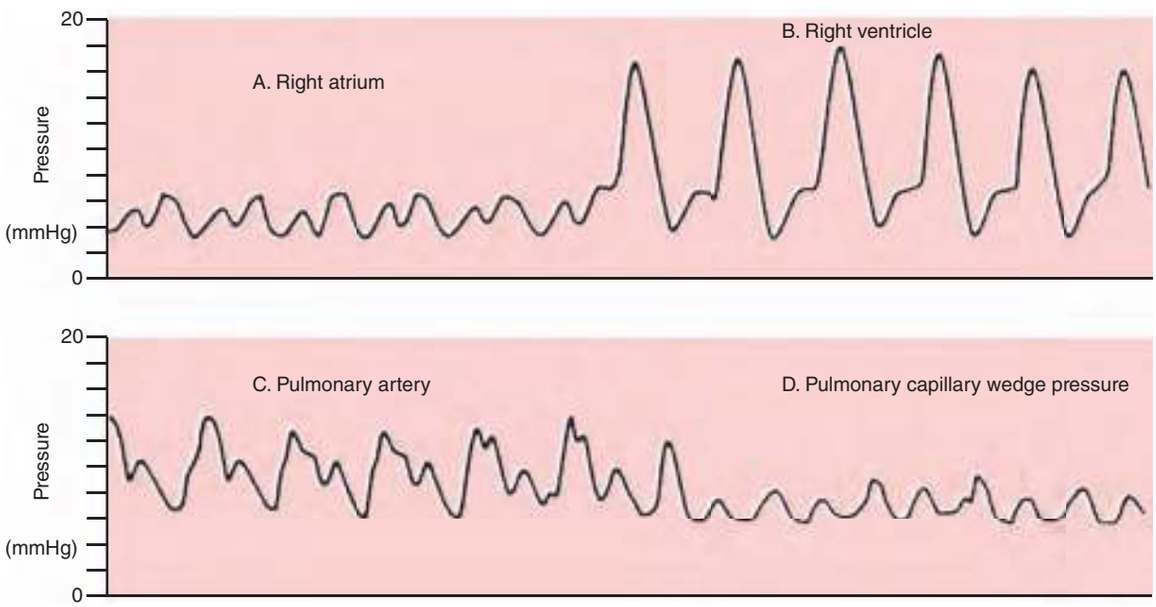

FIGURE 67-4. Typical pressure waveforms recorded by the PAC during insertion.

TABLE 67-2 Average Distances from the Catheter Insertion Site to the Catheter Tip Position*

Catheter insertion site	Right atrium distance (cm)	Right ventricle distance (cm)	Pulmonary artery distance (cm)	Wedge distance (cm)
Subclavian vein	10	20	30–40	40–45
Right internal jugular vein	15	25	35–45	45–50
Left internal jugular vein	20	30	40–50	50–55
Right antecubital vein	45	60	70–80	80–85
Left antecubital vein	50	65	75–85	85–90

*These distances are considered the common estimates for uncomplicated PAC placement in patients with normal-sized hearts. The distances will vary in patients and may be greater, especially in the patient with a dilated right ventricle. Any time there is a gross discrepancy between these distances and the actual observed placement distance, the physician should consider catheter misplacement, catheter looping, or catheter knotting. An immediate portable anteroposterior chest radiograph should be obtained to evaluate the situation.

into the low-pressure right atrium (**Figure 67-4*A***). Prior to the insertion, allow the PAC to rest in its natural curvature. Insert the PAC such that its natural curvature follows in the direction of the RV outflow tract. Continue to advance the PAC into the right ventricle, which will be apparent by an abrupt change in the pressure waveform (**Figure 67-4*B***). This occurs at approximately 30 cm of insertion. There is a larger amplitude waveform with a sharp upstroke in the higher pressure RV (**Figure 67-4*B***). **The PAC may be coiled in the RA or migrated into the inferior vena cava if this waveform is not obtained. Continuing to insert the catheter further can increase the risk of knotting.**[17]

Advance the PAC if the classic RV waveform is obtained. At about 40 to 45 cm, the PA is reached, yielding a waveform with a sharp upstroke and downstroke and the classic "dicrotic notch" (**Figure 67-4*C***).[17] The PA is occluded by advancing the balloon a few centimeters further, yielding the PAWP. This final waveform will be similar to that observed when the catheter is in the RA (**Figure 67-4*D***).[18] Passing the PAC through the right ventricle may produce some ventricular ectopy, which is generally uncomplicated.

Deflate the balloon, and the PAP waveform should again be visualized. **If the PAP waveform does not return and the PAWP waveform persists, withdraw the PAC with the balloon deflated until the PAP waveform is again recognized.** This problem can be encountered when the PAC migrates far enough that the catheter itself occludes the PA without the balloon inflated. **Leaving the PAC wedged in this position can cause pulmonary infarction**. **The PAC balloon should never be left inflated.** Correct PCW positioning can also be confirmed by withdrawing a blood sample and checking a blood gas. A sample from the wedge position should have an oxygen saturation of > 94%.[17]

Whenever it is unclear where the tip of the PAC is located, deflate the balloon and withdraw the PAC to a spot where the waveform is recognizable. Take notice of the distance marking. Inflate the balloon and advance the PAC until the desired tracing is obtained.

Difficulties in passing the PAC into the PA may occur in patients with pulmonary hypertension, significant tricuspid regurgitation, or markedly dilated right heart chambers. Instruct the patient to inspire slowly and deeply to increase venous return to the right heart. This may allow the PAC to be advanced successfully. Tilting the patient's head upward and repositioning the patient on their left side may also be helpful. Fluoroscopic guidance may be necessary if repeated attempts are unsuccessful.

Pull up the protective sleeve over the catheter and secure it to the introducer sheath. It is important not to advance the PAC itself during this manipulation. Begin any infusions through the PAC. The PAC should be secured to the skin with sutures. Apply a sterile dressing. Care of the catheter and skin site are covered in Chapters 63 and 64. Obtain a chest radiograph to confirm placement and evaluate for a pneumothorax. The tip of the PAC should be at or below the level of the left atrium, generally no more than 2 cm from the hilum and 3 to 5 cm from the midline (**Figure 67-5**). This position corresponds to West Zone 3 of the lung, which is the most dependent portion in which the PAP and PAWP exceed alveolar pressure.[19]

FIGURE 67-5. The PAC in position as viewed on the anteroposterior chest radiograph. (Image courtesy of Dr. Alex Yartsev.)

DATA INTERPRETATION

A detailed discussion of PAC data interpretation cannot be undertaken here. However, anyone who places or uses PACs in the management of critically ill patients should be familiar with the standard information provided by the PAC. To obtain and interpret data from the PAC, the monitoring system must be calibrated and zeroed and the transducer leveled. **The PAWP will not be accurate if these steps are not taken.** Some studies have cited that only 30% to 55% of physicians and nurses can correctly identify the PAWP.[20-23]

The data generated by the PAC can be categorized as that which is directly observed, obtained, or extrapolated.[17] The PAC allows real-time continuous display of heart rate (HR), mean arterial pressure (MAP), CVP, right atrial pressure (RAP), and PAP (**Table 67-3**). PAWP can be obtained by inflating the balloon. Mixed venous oxygen saturation (SvO_2) can be obtained by taking a PA blood sample, and CO can be obtained using thermodilution. These data, including normal values, are listed in **Table 67-3**. The next set of data comprises the variables that are mathematically derived from the measured data (**Tables 67-4 and 67-5**). These provide information crucial to understanding cardiac and pulmonary physiology and pathology.

TABLE 67-3 Variables Obtained from the Pulmonary Artery Catheter Through Direct Measurement

Variable	Normal values	Main utility	Comments
Cardiac output (CO)	4–6 L/min	Diagnosis of shock (high output versus low output); titration of vasoactive medications	Measurement is prone to error; should be indexed to patient's size
Pulmonary capillary wedge pressure (PCWP)	5–15 mmHg	Volume status, diuresis, fluid challenges	Often overinterpreted; must be used with other values
Right atrial pressure (RAP)	0–10 mmHg	Status of right ventricle	Less useful than PCWP
Pulmonary artery pressure (PAP)	15–25 mmHg systolic; 8–15 mmHg diastolic	Status of right ventricle and pulmonary circuit	Pulmonary artery diastolic can be substituted for PCWP in most patients
Mixed venous oxygen saturation (SvO_2)	70–80%	Evaluation of oxygen delivery; pulmonary shunt fraction	Best obtained by blood gas from distal pulmonary artery

TABLE 67-5 Formulas for the Derivation of Variables

SVR = (mean arterial pressure – mean arterial pressure) × 80 cardiac output
PVR = (mean pulmonary artery pressure – pulmonary capillary wedge pressure) × 80 cardiac output
LVSWI = SV × (mean arterial pressure – pulmonary capillary wedge pressure) × 0.0136 body surface area
RVSWI = SV × (mean arterial pressure – right atrial pressure) × 0.0136 body surface area
DO_2 = [(cardiac output × hemoglobin) × (13.4) × (% O_2 saturation)] + (PO_2 × 0.0031)
VO_2 = (cardiac output × hemoglobin) × (13.4) × (SaO_2 – SvO_2)
Q_s/Q_t = (pulmonary capillary O_2 content – CaO_2) ÷ (pulmonary capillary O_2 content – CvO_2)

CaO_2, arterial oxygen content; CvO_2, mixed venous oxygen content; PO_2, partial pressure of oxygen; SaO_2, arterial oxygen saturation; SV, stroke volume. Other terms are defined in Tables 67-3 and 67-4.

Today, this information is routinely available on an instantaneous basis as part of computer software packages that accompany the monitoring equipment. PAC users should become familiar with the information from the PAC and its application in different clinical situations. The reader is advised to consult a current textbook of cardiology or critical care medicine for a more detailed description of the hemodynamic data provided by the PAC.

COMPLICATIONS

Complications related to the use of PAC are related to three general areas: obtaining central access (Chapters 63 and 64), the catheterization process itself, and those related to the indwelling PAC (**Table 67-6**). Obtaining central access can result in arterial puncture, pneumothorax, hemothorax, bleeding, and potentially cardiac perforation.[1] The complications directly related to the PAC may be divided into those that are associated with catheter insertion and those associated with maintenance (**Table 67-6**). Both sets of complications can be further divided into those problems where there has been systematic study and the incidence of complications has been published and those that have been observed and published as case reports but the actual incidence of which is unknown.

Problems with tracing quality may occur due to problems involving the catheter itself or other parts of the system. Catheter problems include positioning too distal or not distal enough, balloon rupture, or clot formation at the tip. Problems elsewhere in the system include air in the lines, loose connections, failure of the transducer, failure of the wires, or failure of the monitor. The system should be zeroed and calibrated again to confirm the accuracy of the pressure values if abnormally high or low values are obtained. Sometimes it is just not possible to obtain a good wedge tracing despite repeated attempts. In such cases, the pulmonary diastolic pressure may be used as a surrogate for the wedge.

A right bundle branch block may occur in 5% due to impact of the PAC with the right side of the septum during insertion. This is usually transient. Even if it persists for hours, it is well tolerated in most patients. **However, the superimposition of a right bundle branch block in the presence of a preexisting left bundle branch block leaves the patient with a complete heart block. This complication can result in severe bradycardia and hemodynamic embarrassment. It is important to be prepared to institute temporary transcutaneous or transvenous pacing when placing a PAC in patients with a left bundle branch block.**[24]

Arrhythmias during insertion, most commonly premature ventricular beats, are usually due to irritation of the right ventricle. This is especially true of the outflow tract. Premature ventricular contractions are usually well tolerated unless sustained ventricular tachycardia or ventricular fibrillation occurs. Slight withdrawal and redirection of the PAC are usually adequate. Arrhythmias after insertion may be due to catheter loops in the right heart, which will be apparent on the chest radiograph and can be corrected by careful withdrawal of the PAC until the loop is removed. Arrhythmias may also occur if the PAC tip slips back into the right ventricle. In this case, the pressure tracing will show a typical RV waveform. Readvancement of the PAC into the PA should eliminate the arrhythmias.

Other potentially serious but rare complications include injuries to the great vessels, trachea, lymphatic duct, vagus nerve, or phrenic nerve as well as a pneumothorax, hemothorax, hemomediastinum, misplacement, and cardiac perforation.[25,26] On occasion, the PAC can become knotted intravascularly or within the heart during placement. This requires an Interventional Radiologist to unknot it or a Surgeon to remove the PAC if this fails.[1]

The most serious complications related to insertion or maintenance of the PAC are PA rupture, PA infarction, and catheter infection. PA rupture can occur at the time of placement or later secondary

TABLE 67-4 Derived Variables Obtained from the Pulmonary Artery Catheter

Variable	Normal values	Main utility	Comments
Systemic vascular resistance (SVR)	800–1600 dynes/sec/cm^{-5}	Shock states, vasodilator versus vasopressor therapy (afterload)	Unclear whether value should be indexed to patient's size
Pulmonary vascular resistance (PVR)	20–200 dynes/sec/cm^{-5}	Pulmonary hypertension; acute and chronic lung disease	Unclear whether value should be indexed to patient's size
Left ventricular stroke work index (LVSWI)	56 ± 6 gm-m/m^2	Left ventricular performance	Clinical utility uncertain
Right ventricular stroke work index (RVSWI)	8.8 ± 0.9 gm-m/m^2	Right ventricular performance	Clinical utility uncertain
Oxygen delivery (DO_2)	900–1100 mL/min	Shock states, anemia, low cardiac output	Often amenable to therapy, but controversial
Oxygen consumption (VO_2)	200–250 mL/min	Sepsis, burns, trauma, ventilator patients	Affected by many variables; use is controversial
Pulmonary shunt fraction (Q_s/Q_t)	3–5%	Acute and chronic lung disease	Underused in the evaluation of pulmonary disease

TABLE 67-6 Recognized Complications of Pulmonary Artery Catheterization

Related to catheter insertion (published incidence)
Air embolism (0.1%)
Complete heart block (0–2.6%)
Hematoma (0–3%)
Inability to place catheter with multiple attempts (1.7%)
Injury to great vessels (0.1–13%)
Pneumothorax (0.1–1.5%)
Ventricular arrhythmias (20–50%)
Ventricular arrhythmias requiring treatment (0–3%)
Related to catheter insertion (reported, but incidence not published)
Cardiac perforation
Catheter knotting (requiring surgical removal)
Guidewire embolism
Hemomediastinum
Hemothorax
Injury to phrenic or vagus nerve
Injury to trachea
Lymphatic duct perforation
Related to long-term maintenance (published incidence)
Catheter infection (1–5%)
Pulmonary artery rupture (0–0.5%)
Pulmonary infarction (0–0.5%)
Related to long-term maintenance (reported, but incidence not published)
Catheter shearing with embolization
Misreading or misunderstanding of data provided by catheter

to migration of the catheter, with a mortality ranging from 30% to 70%.[27] PA rupture is usually the result of the catheter becoming overwedged and/or the balloon overinflated.[27] Pulmonary infarction has been seen primarily in patients with mitral regurgitation or pulmonary hypertension and may be avoided if the duration of balloon inflation is kept to a minimum. Catheter infection occurs in 1% to 5% of PAC placements and can be minimized by strict sterile maintenance of the PAC as well as continually reevaluating the need for the PAC and keeping the placement time as short as possible.[28] Equipment malfunction or failure can also occur. The presence of the indwelling catheter for prolonged periods of time (> 48 hours) increases risk of PA rupture, deep venous thrombosis, pulmonary infarction, and infection.[28] The majority of PACs should be used for 72 hours or less.

SUMMARY

At the time of its introduction four decades ago, the PAC was initially embraced by EPs and Critical Care Physicians for its ability to provide real-time information regarding variables such as CVP and pulmonary capillary wedge pressure at the bedside, and PAC use became common practice. However, over the past 15 years, this has come into significant question. At this point, the utility of PAC is unclear. The PAC has not led to improved outcomes, decreased mortality, or decreased intensive care unit length of stay. Thus, its use as common practice cannot be supported. However, there are still many clinical situations for which the use of a PAC may benefit the EP to make crucial decisions. The PAC is a tool in the armamentarium of any clinician taking care of critically ill patients. Therefore, it is important to be well versed in the information that it provides and its application in the clinical context of the disease state that EPs are managing.

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68 Noninvasive and Minimally Invasive Cardiac Output Monitoring

Caleb Canders, Daniel W. Weingrow, and Alan Chiem

INTRODUCTION

More than 1,000,000 cases of shock present to the Emergency Department per year. Many critically ill patients remain in Emergency Departments for extended periods of time. Delays in diagnosis and/or therapy may increase morbidity and mortality.[1] The underlying physiologic abnormality for all types of shock remains inadequate oxygen delivery. The main contributors to oxygen delivery are cardiac output (CO) and oxygen content. Early aggressive management of patients with shock leads to improved outcomes.[2]

The goals of CO monitoring in shock are to aid in the diagnosis of undifferentiated shock, to accurately measure the patient's hemodynamics, to treat patients more precisely, and to improve patient outcomes. CO and volume status have traditionally been measured by physical examination and invasive monitors (e.g., pulmonary artery catheter and central venous pressure monitoring).[3] Noninvasive CO monitoring aims to give the Emergency Physician accurate dynamic monitoring information to guide clinical decision making to optimize preload, administer fluid resuscitation, and administer vasopressors.

CO determination has conventionally been obtained by thermodilution or dye dilution measurements that are invasive and associated with potential risks. Pulmonary artery (PA) catheters (Chapter 67) are considered the reference standard for hemodynamic assessment. PA catheters have complication rates of up to 7%.[4-6] Complications of PA catheters include sepsis, arrhythmias, valvar damage, cardiac tamponade, intermittent CO determinations, expense, and restricted use to monitored sites within a hospital (i.e., Emergency Department, Intensive Care Unit, or Operating Room).

Noninvasive devices that measure CO are being used more often and show agreement with the reference standard thermodilution technique. An ideal noninvasive CO monitor should be accurate, reliable, continuous, and compatible with adult and pediatric patients. No single device fits all the criteria despite considerable advancements in CO monitoring. The commercially available modalities for noninvasive CO monitoring fall into three categories: ultrasound (US)-based technology, pulse contour analysis, and thoracic electrical bioimpedance. This chapter reviews the available technology and its applications and limitations.

ULTRASOUND-BASED TECHNOLOGY

Christian Doppler identified that the velocity of a moving object is proportional to the shift in reflected frequency of an optic wave of known frequency. This principle has been adapted to sound waves and is now the basis for Doppler devices that can continuously measure the velocity of blood flow and related hemodynamic variables. The first use of Doppler to measure the velocity of red blood cells in humans or animals occurred in 1969.[5] Commercial devices are available that measure blood velocity in the thoracic aorta via a transcutaneous or transesophageal approach. The USCOM 1A (Ultrasonic CO Monitor, Uscom Ltd., Sydney, Australia) and the Oesophageal Doppler Monitor (ODM; CardioQ, Deltex Medical Inc., Greenville, SC) display continuous hemodynamic data (e.g., CO, peak velocity, and corrected flow time) and the characteristic flow-velocity waveform (**Figures 68-1 and 68-2**). **Point-of-care US can be used to assess CO and fluid responsiveness.**

A

B

FIGURE 68-1. The USCOM US monitor. ***A.*** The Monitor. ***B.*** Close-up of monitor screen. (Photos courtesy of Uscom Ltd.)

POINT-OF-CARE ULTRASOUND

ANATOMY AND PATHOPHYSIOLOGY

CO is the product of heart rate (HR) and stroke volume (SV) (i.e., CO = HR × SV). The left ventricular outflow tract (LVOT) lies proximal to the ascending aorta. Velocity in the LVOT changes with factors that influence stroke volume (e.g., respiratory variation, fluid administration, or passive leg raise [PLR]). Any changes in LVOT velocities will be directly linked to changes in CO or SV.

Fluid responsiveness is an important concept in the management of septic patients. Fluid responsiveness has been traditionally defined as an increase in CO or SV by more than 10% after an isotonic fluid bolus of 500 mL.[6] PLR is a maneuver that results in an intrathoracic autotransfusion of blood from the lower extremities that is hemodynamically similar to a 300 to 500 mL intravenous fluid bolus. PLR has been well validated combined with a CO monitor

FIGURE 68-2. The CardioQ ODM monitor. (Photo courtesy of Deltex Medical Inc.)

FIGURE 68-4. Changes in CBF with PLR to predict fluid responsiveness. See text for details.

and is a good predictor of response to subsequent volume challenges in studies involving mechanically ventilated and spontaneously breathing patients.[7-9] A pre-to-post PLR CO change of ≥ 15% or SV change ≥ 13% has been shown to predict fluid responsiveness.[10-13]

SV can be calculated by multiplying the sum of velocities in one systolic ejection cycle (i.e., known as the velocity-time integral [VTI]) by the cross-sectional area (CSA) of the aortic valve or the LVOT. The Emergency Physician can strictly focus on the changes in VTI given that CSA does not change. A change in VTI and the maximal velocity (V_{max}) with respiratory variation, fluid administration, or PLR can be used to assess fluid responsiveness. LVOT-derived stroke volume variation ≥ 13% with PLR and respiratory variation in septic shock patients in the Intensive Care Unit has been shown to be highly predictive of fluid responsiveness.[14-16]

Figure 68-3 shows the measurement of LVOT ΔVTI and the change in (Δ) V_{max} using an apical five-chamber window. Each negatively deflected peak represents the sum of velocities of blood (or VTI) traveling through the LVOT during systole. The V_{max} is the fastest blood velocity recorded in the peak. The changes in V_{max} or VTI are due to respiratory variation or PLR. A ΔVTI > 20% or ΔV_{max} ≥ 13% predicts fluid responsiveness.

Changes to carotid blood flow (CBF) have been shown to be associated with fluid responsiveness when compared to a combined PLR and bioreactance method. A ΔCBF ≥ 20% with PLR predicts fluid responsiveness with a high sensitivity and specificity.[13] CBF measurement has the advantage that many Emergency Physicians are already able to identify the carotid artery due to experience using US for internal jugular central venous catheterization. Carotid flow time (CFT) is derived from carotid Doppler tracings and has been proposed as a method to assess for fluid responsiveness. CFT reflects the duration of systole in the cardiac cycle. Animal models of hemorrhage show that the CFT decreases when intravascular volume is depleted and CFT increases after fluid administration.[17]

Figure 68-4 shows the assessment changes in CBF with PLR to predict fluid responsiveness. The common carotid artery is identified in the long axis, and a pulsed wave Doppler (PWD) tracing is obtained. The diameter of the vessel is measured. The interval (**Figure 68-4**, *inset*) represents one cardiac cycle. The left marker indicates the beginning of systole. A dicrotic notch (*arrow*) indicates the beginning of diastole. The right marker indicates the end of diastole. The tracing of the velocity between this interval is referred to as the time-average peak (TAP), which is conceptually similar to the VTI. CBF is obtained by multiplying the TAP with the diameter, making it a surrogate estimate for SV.

Figure 68-5 shows the assessment of corrected CFT. The carotid artery is imaged in its long axis, and a PWD tracing is obtained. The systole time and the entire cardiac cycle (i.e., cycle time) are

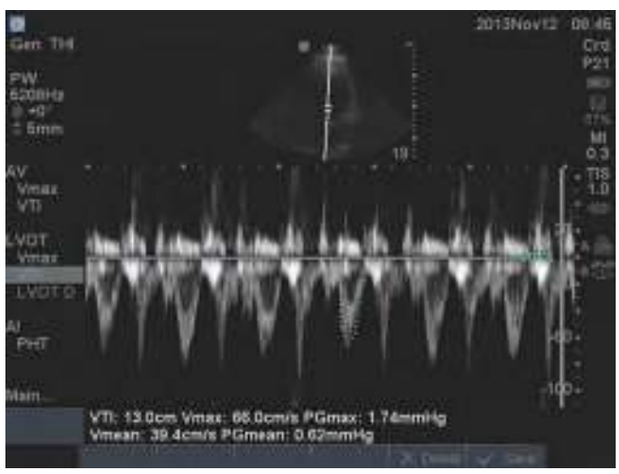

FIGURE 68-3. Measurement of LVOT ΔVTI and ΔV_{max} using an apical five-chamber window. See text for details.

FIGURE 68-5. Assessing the corrected CFT. See text for details. CT, cycle time; ST, systolic time.

measured. A CFT is calculated by dividing systole time with the square root of the cycle time. A ΔCFT ≥ 20% is suggested to predict fluid responsiveness.

INDICATIONS

Point-of-care US is indicated in patients in whom estimations of the CO and fluid responsiveness are needed and noninvasive methods are desired that can be readily repeated to evaluate response to treatment.

CONTRAINDICATIONS

A major disadvantage to point-of-care US, especially when performing advanced Doppler echocardiography, is the skill required of the Emergency Physician. LVOT VTI and V_{max} measurements require more advanced skill in acquiring images and performing calculations. An adequate apical five-chamber view may not be obtainable in some cases. There can be 20% to 30% variation from actual flow if the angle of the US beam (i.e., the insonation angle) is > 30% off flow.[17] Despite these issues with image acquisition, point-of-care US has good agreement between expert sonographers and Emergency Physicians, with optimal VTI measurements obtained nearly 80% of the time by Emergency Physicians.[18] The LVOT VTI may not be accurate when the cardiac rhythm is irregular because of beat-to-beat variation in diastolic filling.

EQUIPMENT

The only equipment required is a portable point-of-care US machine and transducer that can acquire two-dimensional images and Doppler measurements. A phased array probe is ideal for LVOT measurements. A linear probe is ideal for carotid artery measurements. Cardiac calculations software must be installed on the machine for LVOT measurement.

TECHNIQUE

The LVOT diameter is measured in the two-dimensional mode using the parasternal long axis. Measure the diameter 0.5 cm proximal to the aortic valve leaflet insertions in systole (i.e., when the semilunar valve leaflets are open). Most US machines will calculate the cross-sectional area of the LVOT based on the diameter. Obtain an apical three- or five-chamber view. Place the PWD gate in the LVOT as close to the aortic valve as possible without including it. The PWD gate should be 2 to 4 mm. Acquire the PWD waveform. Choose LVOT VTI from the calculation menu, and manually trace the PWD waveform. Some US machines can calculate this automatically. The US machine will calculate the area under the curve and represent it as a VTI. Perform the VTI measurement three times, and average the results to reduce sampling bias. Calculate the SV by multiplying the LVOT VTI and the LVOT CSA. Calculate the CO by multiplying the SV and HR.

For carotid Doppler flow measurements, visualize the carotid artery in its long axis using a linear probe. Measure the diameter of the carotid artery within 0.5 cm of the common carotid bulb during systole. Place a PWD gate at the same location in the middle of the artery to determine the VTI. The angle of insonation should be 45° to 60°. Calculate the SV by multiplying the LVOT VTI and the LVOT CSA. Calculate the CO by multiplying the SV and HR.

SUMMARY

Point-of-care US performed at the bedside can be used to determine CO. CSAs and VTIs can be measured at the LVOT or major arteries. Point-of-care US is noninvasive, readily available, and relatively inexpensive compared to other cardiac monitoring devices. The major drawback is that image acquisition and interpretation are operator dependent. Point-of-care US has the potential to be an effective adjunct in assessing fluid responsiveness and guiding resuscitation in critically ill patients.

ESOPHAGEAL DOPPLER MONITORING

ANATOMY AND PATHOPHYSIOLOGY

SV can be determined by multiplying the spatial average blood velocity within the aorta during systole (V), ejection time (ET), and the CSA of the aorta (i.e., SV = V × ET × CSA). The descending aorta CSA is related to the height, age, and weight of the patient. Determination of CO using this formula is an approximation based on patient variables rather than a precise measurement.

The device displays the peak velocity and flow time (FT) once the esophageal Doppler monitor (EDM) is inserted and the flow-velocity waveform is obtained. The peak velocity is the apex of the waveform, and the FT is the length of the waveform base (**Figure 68-6**).[19-21] The FT is HR dependent. The FT must be corrected (FTc) given that most hemodynamic interventions will affect HR.

The normal range for peak velocity is age dependent: 90 to 120 cm/sec for a 20-year-old, 70 to 100 cm/sec for a 50-year-old, and 50 to 80 cm/sec for a 70-year-old.[22,23] Peak velocity is an indicator of cardiac contractility. Values above the normal ranges (i.e., tall waveforms) correspond to hyperdynamic states. Values below the normal values (i.e., short waveforms) are consistent with hypodynamic states. The FTc represents left ventricular filling (i.e., preload) and systemic vascular resistance (SVR). A normal FTc is approximately 330 to 360 milliseconds. Values below this range are indicative of hypovolemia and/or systemic vasoconstriction.[24] Values above this range are consistent with vasodilatation.[24] Improving the FTc (i.e., preload) will result in greater peak velocity when applying Starling principles. Normal volunteers were monitored for their hemodynamic response to increasing infusions of positive inotropes, medications affecting afterload, and plasma removal (i.e., preload reduction) to corroborate the predicted hemodynamic measurements on the Doppler readings.[25] The waveform changes associated with alterations in preload, inotropy, and afterload are seen in **Figure 68-7**.

INDICATIONS

Use of EDM has been predominantly in the Intensive Care Unit and Operating Room, although it has been used in the Emergency Department for trauma resuscitation and management of septic shock.[25-29] EDM allows the Emergency Physician to recognize circulatory compromise at the earliest possible time, to gain a better understanding of pathophysiologic conditions, and to provide real-time monitoring of interventions applied to patients with hemodynamic compromise or at high risk for deterioration.

CONTRAINDICATIONS

The esophageal Doppler probe is similar in size to a nasogastric tube but more rigid and less tolerated by the awake patient. Do not use EDM in patients with oropharyngeal or midface injuries. Use extreme caution in patients with portal hypertension complicated by esophageal varices. EDM is relatively contraindicated in patients with esophageal strictures, recent esophagogastric operations, or caustic ingestions because of the risk of esophageal perforation.

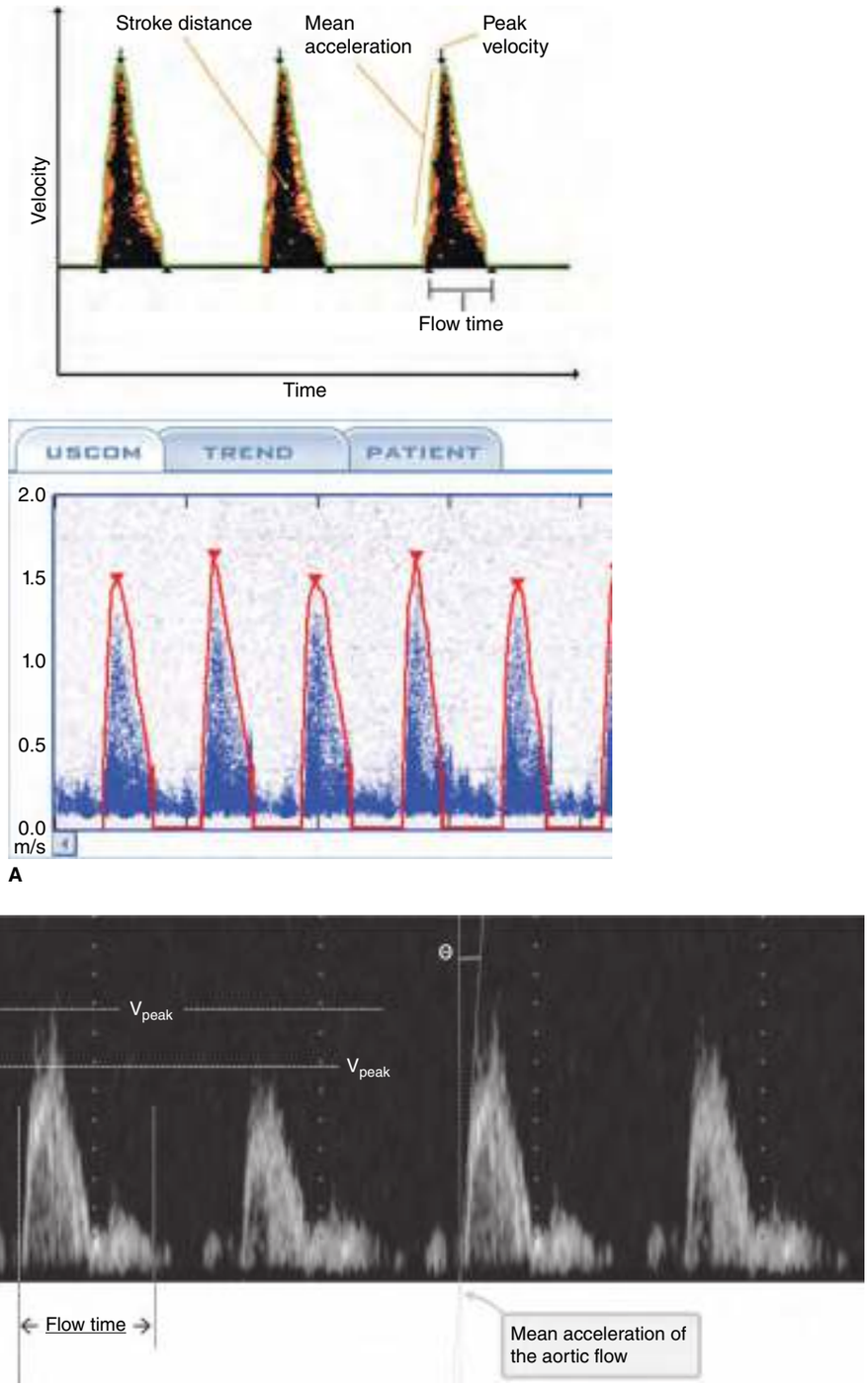

FIGURE 68-6. Velocity versus time assessed with Doppler. ***A.*** Top image is a graphic representation. Bottom image is a monitor view. (Used with permission from reference 19.) ***B.*** Doppler waveform. (Used with permission from www.derangedphysiology.com.)

EQUIPMENT

The only equipment required is the EDM machine and a probe. The beveled end of the probe emits the Doppler signal. The use of a nasal decongestant (e.g., cocaine, oxymetazoline, or phenylephrine) is recommended prior to EDM probe insertion if not contraindicated.[30] Some EDM probes are disposable. A sterile cover is used as a barrier method to prevent contamination with the mucosal surface of the esophagus for reusable probes. There is approximately a 1% chance of failure with the cover. The EDM probe requires high-level disinfection after each use.[31]

TECHNIQUE

The EDM probe can be inserted via the orogastric or nasogastric approach (Chapter 75). The nasogastric approach is preferred in the awake patient. Apply a nasal decongestant prior to insertion.[32] Apply lubricant to the probe. Gently insert the probe through either

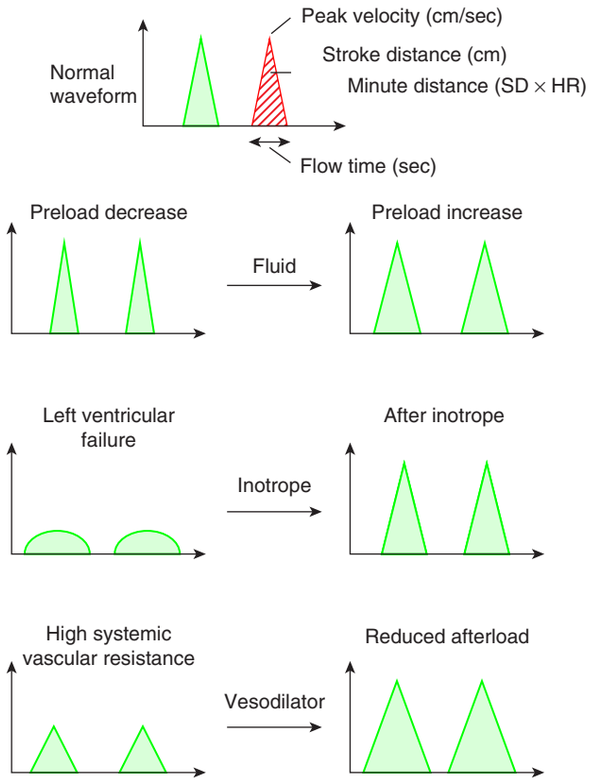

FIGURE 68-7. Waveforms associated with changes in preload, inotropy, and afterload. (Used with permission from reference 20.)

naris until it reaches the nasopharynx. **The beveled surface of the probe must be facing the posterior surface of the esophagus during insertion to capture the blood flow in the descending aorta.** Gradually advance the probe while maintaining its correct position until a distinct flow-velocity curve is obtained on the EDM output display. The image quality may be improved by slight rotation of the probe in either direction. Secure the probe to the patient once a proper waveform is obtained. Orogastric insertion is similar. Procedural sedation (Chapter 159) may help facilitate placement if the patient has difficulty tolerating placement.

AFTERCARE

Roentgenographic confirmation of EDM probe placement is not necessary, as the waveform output indicates proper positioning. An esophageal Doppler probe may be used for many days provided a proper waveform is maintained. This is in contrast to invasive CO measurement devices. Simply remove the probe in a manner similar to removing a nasogastric tube (Chapter 75) to discontinue monitoring with the EDM.

COMPLICATIONS

Minor bleeding from the nares may occur and can generally be controlled with direct pressure or packing. Sinusitis may develop with prolonged use. Prevention with nasal decongestants may minimize this complication. Chapter 75 provides a more detailed discussion of the complications associated with the use of a nasogastric tube.

SUMMARY

CO measurement obtained by EDM correlates well with that obtained by thermodilution.[29-33] The FTc provides a measure of preload. The peak velocity is correlated with inotropy and to a lesser extent with SVR. The device is relatively inexpensive compared to a PA catheter and has a better safety profile. EDM provides continuous CO readings that have less interrater variability than transthoracic Doppler and allows more effective trending of resuscitative interventions. Drawbacks include its poor tolerability in the awake patient and its inaccurate readings in patients with valvular abnormalities due to turbulent blood flow. Calculated measurements of the CO are derivations from these estimations because the descending aorta receives only a percentage of the total CO. EDM is not intended to serve as a replacement for the PA catheter. The EDM may prove to be an important adjunct in the resuscitation of critically ill patients and for the identification of occult hemodynamic compromise.

USCOM: ULTRASONIC CARDIAC OUTPUT MONITORING

ANATOMY AND PATHOPHYSIOLOGY

The USCOM device incorporates height-index regression equations to determine SV from the VTI and CSA of the aortic valve. The SV is then multiplied by the HR to determine the CO. The USCOM device directly measures the percent ejection time (ET%), HR, mean pressure gradient, peak velocity, and VTI. The device uses the VTI and CSA to calculate the SV, CO, and cardiac index. The machine calculates the SVR of the patient with the addition of the blood pressure and central venous pressure. The monitor displays these variables in a continuous format and allows trending with the therapies given (e.g., intravenous fluids or vasoactive agents).[22]

INDICATIONS

The USCOM device has been used in the prehospital, Emergency Department, Intensive Care Unit, and Operating Room settings. It has been used in diverse patient populations (e.g., neonates, trauma patients, cardiac surgery patients, and the critically ill). There have been comparisons of the USCOM device to the PA catheter for a reference standard. Studies have evaluated the precision of Emergency Physicians using the device. The USCOM device shows a fair level of agreement with invasive methods. The trends obtained with the device may help the Emergency Physician to further differentiate shock states and assess responses to therapy.[22-24] The device's noninvasive and portable nature makes it an attractive alternative to invasive means of CO measurement in a variety of settings.

CONTRAINDICATIONS

There are no major contraindications to using this device. Large body habitus and indwelling tracheostomy tubes may make it difficult to obtain views of the aortic root to measure the CSA. The device is best used on supine patients.

EQUIPMENT

The monitor and attached probe are the only required equipment. The transducer is curved at the tip to allow for the insonation angle to be in-line with blood flow and increase the accuracy of the measurements.

TECHNIQUE

The optimal placement of the transducer is in the suprasternal notch or the left supraclavicular space to get the aortic flow profile in the ascending aorta. There is no direct visualization of the aortic arch as would be possible in a standard US machine. The aortic CSA is

estimated by the patient's height and ideal body weight. An operator achieves confidence obtaining an adequate view after approximately 30 patients per the device manufacturer.

SUMMARY

US offers a noninvasive, portable, and cost-effective device for CO monitoring in a variety of patients and clinical settings. The CO measurements correlate well with thermodilution and have been used to assess shock states and responses to treatments. The major limitation of this technique is in patients in whom it is difficult to identify the aortic root. There is user variability based on level of experience.

PULSE CONTOUR ANALYSIS

METHODOLOGY

Otto Frank first proposed analyzing arterial pressure waveform to estimate CO in 1899.[3] He used the concept of calculating the total peripheral resistance (TPR) from the time constant of diastolic aortic pressure decay. He calculated the CO from the TPR and mean arterial pressure, or MAP (i.e., CO = MAP ÷ TPR).[3]

The Windkessel model of blood flow was used to determine the area under the curve of the arterial waveform and has two fundamental aspects. First is conservation of mass. This means that the amount of blood entering a blood vessel is equal to the amount of blood leaving the blood vessel over the cardiac cycle. The second principle is that the compliance of the blood vessel affects the flow through it in a predictable manner. The blood vessel expands and absorbs some of the blood that would have otherwise passed through it as the pressure rises during systole. The blood vessel contracts and ejects the previously absorbed blood as the pressure drops in diastole. Wesseling and colleagues further developed the Windkessel model by incorporating the concept of impedance (i.e., the resistance to pulsatile flow). This model, known as the characteristic impedance model, incorporates the MAP and HR into the calculation of resistance.

Wesseling and colleagues subsequently developed the three-element Windkessel model, which includes peripheral resistance, compliance, and characteristic impedance. This model assumes that the impedance and compliance can be estimated by a patient's demographic data and the resistance can be calculated by incorporating the blood pressure data into the three-element model. This methodology still requires CO measurement by another method for calibration due to the variation in empiric estimates in aortic dimensions.[25,34-36]

A variety of pulse contour analysis devices use the above models to measure CO. We will discuss the LiDCO, PiCCO, Flotrac/Vigileo, and ClearSight systems in this chapter.

LIMITATIONS

All pulse contour analysis systems use pulse pressure analysis in one form or another. **It is imperative that one has an optimal arterial waveform to analyze for a reliable estimation of the CO.** This requires a proximal arterial catheter. Use of a peripheral location results in decreased accuracy of the pulse contour.[31] It is very important to pay close attention for overdampened or underdampened waveforms. Arrhythmias limit the accuracy of these devices because there is beat-to-beat variation in the waveform. The presence of an intraaortic balloon pump also alters the arterial waveform and limits the use of these devices. Frequent recalibration is required to ensure accurate readings during hemodynamic instability. Each system has incorporated methods to compensate for these limitations. Trend analysis in these situations may be more useful than the absolute values of CO.

LiDCO Plus AND LiDCOrapid SYSTEMS

METHODOLOGY

These systems use the PulseCO system algorithm to analyze the arterial waveform and determine continuous changes in SV. The PulseCO system incorporates the concept of characteristic impedance into its algorithm. The system modifies the characteristic impedance approach by incorporating the difference between the peripheral and central blood pressures.

The LiDCO Plus system (LiDCO, London, UK) requires calibration using a transpulmonary lithium indicator dilution technique. This requires a disposable lithium sensor, arterial line, lithium chloride, and monitor. A lithium sensor is attached to an arterial line to calibrate the system. The lithium sensor is a flow-through cell that is sterilized by gamma radiation. The lithium sensor attaches through a stopcock, and flow is restricted to 4.5 mL/min with a small peristaltic pump. Lithium chloride is injected via a peripheral or central venous line, and blood is drawn past the lithium sensor. The resulting arterial lithium concentration-time curve is recorded on a monitor (**Figure 68-8**). This value is the calculated CO for the patient and serves as the calibration point for the pulse contour algorithm.[34,37] The arterial pressure transforms into a volume-time waveform. The raw data can be displayed if you want the numbers and to see the hemodynamic changes associated with interventions.

The LiDCOrapid uses nomograms to estimate CO (**Figure 68-9**). The key step is the conversion of blood pressure volume for aortic compliance and aortic capacitance. A patient-specific factor adjusts the aortic compliance for each patient using the PulseCO algorithm. This factor is determined by comparing the CO with the PulseCO estimate. The factor does not change over time. The device has multiple windows. The hemodynamic window shows the trends in MAP, diastolic pressure, systolic pressure, HR, and scaled SV. The dynamic preload window displays preload response values or volume status indicators (i.e., pulse pressure variation and SV variation). The blood pressure window displays the arterial pressure waveform, which is converted through the software to SV and CO. The event response window evaluates the patient response to interventions (e.g., fluid boluses and vasopressors). Finally, the data download window is used to review the patient's history.

LIMITATIONS

In addition to the general limitations of pulse contour analysis devices, the lithium chloride used for calibration of the LiDCO Plus system should not be used in patients who take lithium-containing medications, patients weighing less than 40 kg, or women in the first trimester of pregnancy. Some muscle relaxants cross-react with the

FIGURE 68-8. The LiDCO Plus monitor. (Photo courtesy of LiDCO.)

FIGURE 68-9. The LiDCOrapid monitor. (Photo courtesy of LiDCO.)

lithium sensor and make readings unreliable. This problem can be overcome by performing the calibration prior to or 5 to 10 minutes after succinylcholine, 15 minutes after pancuronium, or 15 to 30 minutes after vecuronium is administered. Rocuronium use is problematic due to the large doses administered to cause paralysis and longer plasma levels. Because the LiDCOrapid system uses estimations for the CO, it is limited in its calibration. It is contraindicated in patients with aortic regurgitation, intraaortic balloon pumps, and peripheral vascular disease. Arrhythmias provide data that cannot be analyzed by the system.

PiCCO SYSTEM

METHODOLOGY

The PiCCO system (Pulsion Medical Systems, Munich, Germany) uses a dedicated thermistor-tipped catheter to assess stroke volume on a beat-to-beat basis (**Figure 68-10**). The PiCCO algorithm includes an estimate of aortic compliance derived from analyzing the waveform distal to the dicrotic notch. Place a catheter in the femoral, radial, or brachial artery. The system measures aortic compliance and instantaneous pressure change integrated over systole. The advantage of this calculation is that it treats compliance as a dynamic variable that changes with time. This system does require calibration with transpulmonary thermodilution via a central venous line. The calibration is required for adjustment of individual aortic impedance. Compliance is a dynamic variable that should be recalculated every 8 hours. This system has been shown to be valid in variety of patient populations.[36,37]

FIGURE 68-10. The PiCCO monitor. (Photo courtesy of Pulsion Medical Systems.)

LIMITATIONS

The greatest limitation of the PiCCO system is that it needs to be calibrated much more frequently in hemodynamically unstable patients. This system has been the most validated in different patient populations.

FloTrac/Vigileo SYSTEM

METHODOLOGY

The FloTrac/Vigileo System (Edwards Lifesciences, Irvine, CA) requires a proprietary FloTrac transducer. It attaches to any standard arterial line catheter and connects to the Vigileo monitor (**Figure 68-11**). This system uses a significantly different approach in determining SV than the Windkessel-based devices and does not require calibration. This system assumes that the SV is related to the standard deviation of the arterial pressure and chi. The arterial pressure (σ_{AP}) represents the relationship between pulse pressure and SV. The chi (χ) is the effect of vascular tone on waveform morphology. These variables are determined from correlations developed from a proprietary database. The standard deviation of pulse pressure

A

B

FIGURE 68-11. The FloTrac transducer connects to a standard arterial line catheter and the Vigileo monitor. ***A.*** The Vigileo monitor. ***B.*** The FloTrac transducer. (Photos courtesy of Edwards Lifesciences.)

(σ_{AP}) is sampled over a 20 second period and correlated with a "normal SV" based on patient demographic data stored in the built-in database. Impedance (χ) is determined from this data.[34,35,38,39] The vascular compliance and resistance are determined from the arterial waveform analysis.[34,35,38,39]

LIMITATIONS

The major limitation of the FloTrac/Vigileo system is that it may be inaccurate in patients with rapid hemodynamic changes. The algorithm has gone through significant modifications after its initial release and has subsequently shown improved performance.

ClearSight SYSTEM

METHODOLOGY

The ClearSight system (BMEYE, Edwards Lifesciences, Irvine, CA), previously known as the NexFin system, is a completely noninvasive pulse pressure analysis device that assesses pulse pressure using photoelectric plethysmography in combination with a volume clamp technique (inflatable finger cuff) (**Figure 68-12**). This system uses the three-element Windkessel model. The system continuously measures finger blood pressure via a cuff applied to the second, third, or fourth digit. It converts the finger blood pressure curve into a brachial arterial waveform. This system generates continuous CO measurements based on the brachial arterial waveform analysis and the patient's demographic data.[35,40]

LIMITATIONS

The ClearSight system frequently fails to measure pressure at any of the fingers with extreme low flow in the arteries of the finger. This limits its feasibility in critically ill patients who suffer from hypoperfusion.[17] This is a critical limitation because CO monitoring makes the greatest clinical impact in this patient group. This device performs adequately in healthy patients and has been recommended for use in outpatient clinics and in perioperative management. Its use in patients with suspected shock states should be cautioned until further validation.

THORACIC ELECTRICAL BIOIMPEDANCE

INTRODUCTION

Thoracic electrical bioimpedance (Z) was developed by NASA in the 1960s to monitor hemodynamic changes in astronauts. The first clinical use of bioimpedance to determine CO was by Nyober.[41] Kubicek used the first derivative of impedance (dZ/dt) to better estimate CO calculations in astronauts and labeled it the "Kubicek equation."[42,43] The Sramek-Bernstein equation accounts for the noncylindrical shape of the thorax and calibration factors compensating for patient demographics. This equation has been incorporated in the software of current bioimpedance monitoring devices such as the NCCOM3-R7 (Biomed Corp., Irving, CA). The Kubicek equation is the foundation for the CIC-1000 (Sorba Medical Systems Inc., Brookfield, WI).

The application of bioimpedance cardiography is minimally invasive, is affordable, and has very good reproducibility.[44] CO determination by thermodilution provides only a "snapshot" of a patient's hemodynamic status. Bioimpedance allows for continuous, real-time data acquisition that enables the Emergency Physician to follow a response to therapy. Such a device can be used in the Intensive Care Unit, Operating Room, and Emergency Department.

ANATOMY AND PATHOPHYSIOLOGY

The patient interfaces with the transducer through a series of disposable skin electrodes that allow for measurements of current flowing parallel to the spine. The electric current transmitted is a low-frequency (e.g., 50 to 100 kHz) and high-amplitude (e.g., 0.2 to 5.0 mA) alternating current. The voltage is sensed by pairs of electrodes at the beginning and end of the thorax (**Figure 68-13**). These electrodes can sense the electrocardiogram (ECG), which is necessary to determine hemodynamic variables (e.g., the ventricular ejection time determined by the distance between QRS intervals).

The electrical resistance (Z = impedance) to this current is directly proportional to the content of fluid within the thoracic cavity. The lungs and thoracic wall are of low conductance for electric current. Much of the impedance is derived from the plasma within the heart and great vessels. The average baseline impedance (Z_o) of the thorax changes due to respiration and the accumulation of interstitial fluid. Air is a poor conductor of electricity compared to plasma, and Z_o will rise with inspiration due to increased resistance within the chest. Increased interstitial volume (i.e., pulmonary edema) will cause Z_o to fall. The bioimpedance device will monitor Z_o and has an inverse relationship with thoracic intravascular fluid volumes (i.e., thoracic fluid volume index [TVFI]) where $TVFI = 1/Z_o$. This index provides a way to assess central fluid status of a patient noninvasively and continuously.

The change in electrical bioimpedance (ΔZ) primarily reflects velocity and volumetric changes of aortic blood flow during each

FIGURE 68-12. The ClearSight system finger cuff. (Photo courtesy of Edwards Lifesciences.)

FIGURE 68-13. An illustration of electrode placement and monitor output on the Cheetah-Nicom bioimpedance device. (Photo courtesy of Cheetah Medical.)

cardiac cycle. The first derivative of impedance (dZ/dt) is reflective of aortic blood flow. The maximum rate of change [$(dZ/dt)_{max}$] is proportional to the peak velocity of aortic blood flow. The timing of dZ/dt in correlation with the ECG allows for measurement of systolic time intervals such as the ventricular ejection time and the preejection period (PEP).

The SV determined by Nyober used ΔZ but was found to have excessive respiratory variability. Kubicek used dZ/dt in the SV equation, which was calculated as SV = Rho $L^2/Z_o^2 \times (dZ/dt)_{max} \times$ LVET, where Rho is the specific resistance of blood, L is thorax length, and LVET is left ventricular ejection time. Sramek proposed that Rho L^2/Z_o^2 be replaced by $VEPT/Z_o$, where VEPT is volume of thoracic electrically participating tissue and is equal to $L^3/4.25$. This is based on the premise that the thorax is a truncated cone as opposed to a cylinder and the resistance of blood is negligible in the total resistance. Bernstein further elaborated on the equation by correcting the VEPT calculation due to differences in body habitus and using a nomogram that incorporates the patient's sex, height, and weight.[45]

The bioimpedance device can be used to determine other hemodynamic variables (e.g., systolic and diastolic time intervals). The description of the cardiac cycle was pioneered by Lababidi et al.[46] The A wave is at or shortly after the beginning of the fourth heart sound and just before the ECG Q wave. The B wave occurs at the first heart sound at the apex coincident with aortic valve opening. The X wave signals aortic valve closure. The O wave occurs at mitral valve opening. The PEP represents isovolumic contraction (A→B). The LVET begins at the end of the PEP and ends at the closure of the aortic valve (B→X). The PEP and LVET compose the systolic interval. The isovolumic relaxation time (IVRT) begins when the aortic valve closes and ends on the beginning of mitral flow (X→O). The filling time (FT) is the interval between mitral valve flow and the beginning of the next cardiac cycle (O→A). The diastolic time interval comprises both the IVRT and the FT.

Contractility was first determined using bioimpedance over 30 years ago using the ratio of PEP/LVET to reflect cardiac ejection fraction.[47] Currently, there are several formulas used to calculate contractility. The Heather index [HI = $(dZ/dt)_{max}$/QZ1, where QZ1 = time from beginning of the Q wave to peak dZ/dt], has been shown to be the most accurate for determining contractility.[48,49] The ability of physicians to categorize patients with congestive heart failure into those with systolic or diastolic dysfunction has been problematic. With the use of bioimpedance, Summers et al identified patients with an HI < 5 to have predominantly systolic dysfunction, whereas an IVRT > 0.125 correlated with diastolic dysfunction.[50] The use of thoracic electrical bioimpedance may facilitate the management of this difficult subset of patients.

INDICATIONS

The indications for the use of thoracic electrical bioimpedance include the management of patients with hemodynamic compromise (i.e., shock) and the recognition of occult hemodynamic compromise. Thoracic electrical bioimpedance can be used to recognize diastolic dysfunction, determine hypovolemia during tilt testing, monitor exercise tolerance, or gauge the adequacy of hemodialysis. It may become a useful adjunct in the resuscitation of critically ill patients prior to the insertion of a PA catheter due to its noninvasive nature.

CONTRAINDICATIONS

There are no contraindications to the use of a bioimpedance device. There are situations that will result in inaccurate measurements. Anything that creates noise within the system (i.e., shivering, excessive movement, and extreme obesity) will affect SV determination and measurement of other hemodynamic variables. Current systems incorporate waveform averaging to minimize the effect of system noise. Poor skin electrode contact has been problematic, notably with excessive diaphoresis. The CIC-1000 (Sorba Medical Systems Inc., Brookfield, WI) has devised electrodes that minimize noise and optimize impedance waveform analysis.

Other limitations include valvular abnormalities and extremes in HR or rhythm. Aortic regurgitation can lead to falsely elevated SV estimates. Bradycardia less than 60 beats per minute or tachycardia over 140 beats per minute can lead to inaccurate results. Patients with paced rhythms can alter calculation of the PEP due to an altered QRS morphology. Patients with irregular rhythms will have errors in calculation of ventricular ejection time, leading to an inaccuracy in estimation of the CO. Algorithms have been created to overcome many of these flaws. It is nonetheless important to identify circumstances when thoracic electrical bioimpedance measurements may be less reliable. Extreme alterations in tissue water content, as may occur in pulmonary or chest wall edema, can modify bioimpedance analyses unrelated to changes in CO. Electrode placement can also significantly alter hemodynamic measurements. A 1 cm difference in thoracic length has been shown to result in a 10% error in stroke volume.[51]

EQUIPMENT

The amount of equipment necessary is limited regardless of the thoracic electrical bioimpedance device used. The device itself and the electrodes are required for measurement. Other materials needed include electrode preparation gel and a tape measure to improve accuracy of the readings.

SUMMARY

Given the growing safety concerns regarding the PA catheter, thoracic electrical bioimpedance has been found to be safe, inexpensive, and simple to use. There is good correlation between thoracic electrical bioimpedance and thermodilution-derived CO measurements.[52] The thoracic electrical bioimpedance values tend to underestimate slightly because these are indirect measurements compared to CO obtained via a PA catheter. The ability to provide continuous and beat-to-beat output of hemodynamic data is a distinct advantage. The ability to follow trends in real time gives the Emergency Physician an opportunity to monitor disease progression as well as the response to therapeutic interventions. The ability to use thoracic electrical bioimpedance as a diagnostic tool may prove to be of great benefit in differentiating between systolic and diastolic dysfunction or identifying hypovolemia during tilt testing.

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69 Peripheral Venous Cutdown

Jason M. Rotoli and Flavia Nobay

INTRODUCTION

Venous access in the critically ill patient is of the utmost importance. The literature regarding peripheral venous cutdowns extends back to 1940 when Keeley introduced this technique as an alternative to venipuncture in patients with shock.[1] There has been a noticeable lack of recent investigations regarding venous cutdowns. This is most likely due to the focus on central venous access with ultrasound guidance and intraosseous access. Recent editions of the Advanced Trauma Life Support (ATLS) text refer to the saphenous venous cutdown as an optional skill to be taught at the discretion of the instructor.[2] The importance of obtaining venous access in critically ill patients supports the need to know a wide variety of techniques in order to be successful in every situation.[3] The steps outlined in 1940 by Keeley to expose and cannulate the saphenous vein remain mostly unchanged.[1]

Peripheral venous access can be extremely difficult due to vascular collapse from shock, vascular injury, obesity, anatomically altered central venous sites, sclerotic veins, or cutaneous scars. Direct visualization of the peripheral vein in these patients can be more fruitful than indirect visualization.[4] An additional advantage of the venous cutdown is that it does not interfere with concurrent resuscitative efforts.[5] A cutdown may be the procedure of choice in resuscitating critically ill patients where resources are limited or nonexistent.

Familiarity with this procedure allows for large-bore access and the rapid infusions required in the critically ill patient.[5] This technique can only be successfully performed if one understands the anatomy and details of venous cannulation. Practicing the cutdown technique before its critical need will help one perform optimally in the emergent setting.

ANATOMY AND PATHOPHYSIOLOGY

There are three critical areas for venous cutdowns (**Figure 69-1**). All Emergency Physicians should be knowledgeable of the anatomy of the saphenous vein at the ankle, the saphenous vein at the groin, and the basilic vein at the elbow. **The potential injury to the patient can be significant if one approaches this procedure without regard to the clinical anatomy.**

GREATER SAPHENOUS VEIN

The greater saphenous vein is the longest vein in the body. It is the ideal vein for a peripheral venous cutdown due to its anatomic regularity and superficiality (**Figure 69-2**).[3] **The superficial and consistent position of the saphenous vein, in both adults and children, makes this the ideal vessel for a peripheral venous cutdown.**[6] The saphenous vein begins at the medial dorsal venous arch of the foot. It passes upward and 1.5 to 2.5 cm directly anterior to the medial malleolus (**Figure 69-2*A***). The saphenous vein lies just above the periosteum of the tibia at the level of the medial malleolus.[7] It continues to ascend in the leg, along with the saphenous nerve, in the superficial fascia over the medial aspect of the leg. The vein passes posteromedially to the knee. It curves forward above the knee onto the anteromedial thigh. It passes over the falciform margin of the deep investing fascia to join the femoral vein approximately 4 cm below and 3 cm lateral to the pubic tubercle (**Figure 69-2*B***).

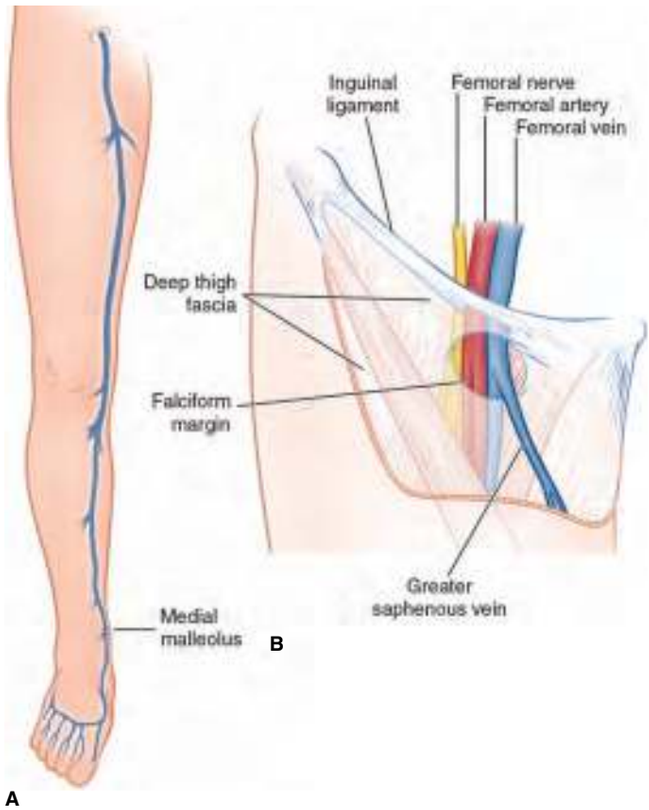


FIGURE 69-2. Anatomy of the greater saphenous vein. ***A.*** The subcutaneous course of the vein in the lower extremity. ***B.*** Detail of the greater saphenous vein at the groin.

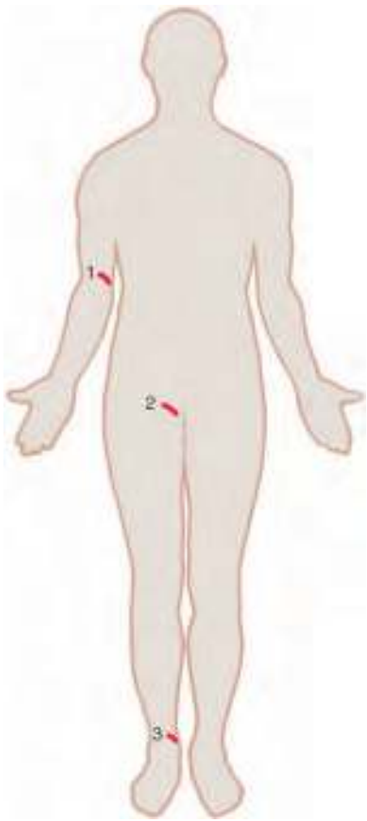


FIGURE 69-1. Common sites for peripheral venous cutdowns include the inner arm above the elbow (1), the inner thigh (2), and the inner ankle (3).

The greater saphenous vein is easily identified at the ankle. **It will be found approximately 2.5 cm anterior and 2.5 cm superior to the medial malleolus.** It may be palpable if the patient is not hypovolemic or obese. The saphenous nerve, a branch of the femoral nerve, travels with the greater saphenous vein. It supplies sensory innervation to the skin of the medial leg and foot as far as the first metatarsal. This nerve is of minimal clinical significance and is often transected when isolating the greater saphenous vein at the ankle.

The saphenous vein in the thigh travels on the anteromedial surface and enters the fossa ovalis to join the femoral vein (**Figure 69-2*B***). The femoral vein is at its largest diameter 3 to 4 cm distal to the inguinal ligament. This is approximately 2 cm distal to the approach for the placement of a femoral central venous line, at the same level where the scrotal or labial fold meets the thigh. The greater saphenous vein is also at its largest diameter in this location. The greater saphenous vein is easily isolated from the surrounding subcutaneous tissue at the level of the scrotal or labial fold meeting the thigh. **The dissection has progressed deeper than the position of the greater saphenous vein if the deep investing fascia of the thigh muscles is visible.**

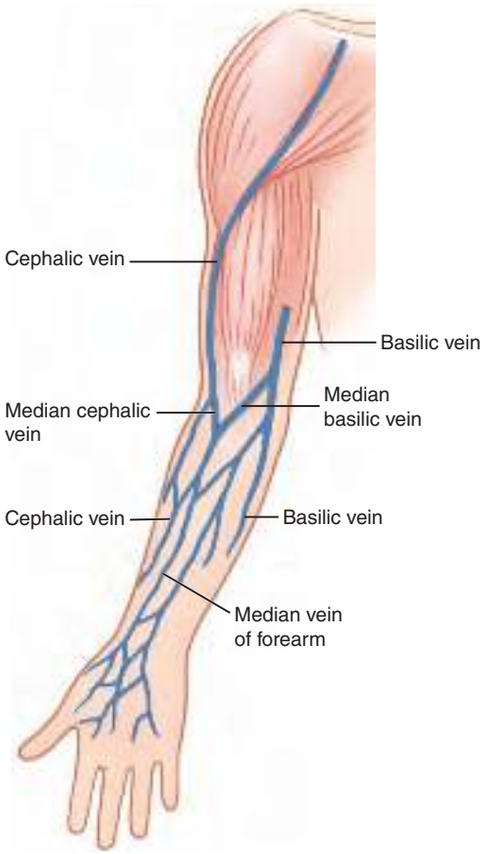


FIGURE 69-3. The superficial veins of the upper extremity.

BASILIC VEIN

The basilic vein is the site of choice for a peripheral venous cutdown in the upper extremity (**Figure 69-3**). It starts from the dorsal venous arch of the hand. It ascends on the posteromedial forearm to become anteromedial on the mid-to-upper forearm. It continues to ascend to the midportion of the arm where it pierces the deep fascia. The basilic vein runs in the groove between the biceps and triceps muscles in the distal one-third of the arm. **The basilic vein can always be found in the groove between the biceps and triceps muscles.** A peripheral venous cutdown should be performed in this location. The basilic vein is more consistently found 2 cm cephalad and 1 to 2 cm lateral to the medial epicondyle of the humerus on the volar (anterior) surface of the arm. **The brachial artery and median nerve lie deep to the basilic vein in this location and are unlikely to be injured if the dissection remains superficial.**

It has been recommended that the approach to the basilic vein is through the groove between the biceps and triceps muscles in the distal one-third of the arm.[8,9] The editor feels that locating the vein above and lateral to the medial epicondyle of the humerus will result in difficult cannulation secondary to the surrounding dense venous plexus. There is a significant risk of injury to the medial antebrachial cutaneous nerve and the deep brachial artery if one tries to find and isolate the basilic vein in the middle third or proximal third of the arm. Injury to the median antebrachial cutaneous nerve will result in sensory loss to the ulnar aspect of the forearm.

BRACHIAL VEIN

Brachial vein cutdowns should not be performed in the Emergency Department. The brachial vein is small in diameter. It is located relatively deep and would require significant and time-consuming steps to locate it. The anatomic structures surrounding the brachial vein include major arteries and nerves that can easily become injured while isolating the vein. Patients in hypovolemic shock will often not have a brachial artery pulse. This can lead to confusion as to which vessel is the artery and which is the vein.[7] Inadvertent cannulation of the brachial artery can result in a brachial artery thrombosis and upper extremity ischemia.

AXILLARY VEIN

The axillary vein is rarely used as a site for a venous cutdown. It is contained within the axillary sheath along side the axillary artery and brachial plexus. A venous cutdown for the axillary vein can injure these structures and is associated with a high rate of complications. **It is not recommended to perform a cutdown for the axillary vein unless the Emergency Physician has specific experience with this technique.**

INDICATIONS

The primary indication for a peripheral venous cutdown is the need for venous access in a patient with no peripheral access and in whom intraosseous access or central access is not immediately obtainable or is contraindicated.[2] This is the ideal procedure for the intravenous drug user with no peripheral veins and scarred central access sites, the burn patient with peripheral venous collapse and scarring, the patient in cardiorespiratory arrest, or the hypovolemic trauma patient who requires definitive and lifesaving volume resuscitation.[10,11] A unit of blood can be infused in less than 3 minutes with intravenous extension tubing inserted directly into the vein.[5,7]

This is an excellent technique for emergent pediatric vascular access. Direct visualization of the vein will aid in cannulation of a vessel that may be collapsed secondary to undifferentiated shock from hemorrhage, hypovolemia, and/or shock. This technique should only be used after other access attempts (i.e., interosseous access, ultrasound-guided central venous access, and peripheral venous access—including scalp veins) have failed.[2,12]

CONTRAINDICATIONS

The absolute contraindications to a peripheral venous cutdown are vascular injury, saphenous vein removal, or long bone fractures proximal to the cutdown site. Relative contraindications include infection overlying the cutdown site, bleeding disorders, or severely distorted anatomy from congenital, blunt, or penetrating trauma to the area of the cutdown or the limb. A cutdown should not be performed unless peripheral intravenous access has failed and intraosseous access equipment is not available or has failed.[2]

EQUIPMENT

- Povidone iodine or chlorhexidine solution*
- Local anesthetic solution*
- 10 mL syringe*
- 22 gauge needle
- #10 scalpel blade*
- #11 scalpel blade*
- #3 scalpel handle*
- Curved Kelley hemostat*
- Small mosquito hemostat*
- Vein pick

- Fine tooth forceps*
- Iris scissors
- Sharp tissue-cutting scissors
- Sterile drapes
- Towel clips
- Sterile polyethylene intravenous tubing*
- Sterile intravenous extension tubing
- Central line kit (for Seldinger method)
- Catheter-over-the-needle, 16 or 18 gauge*
- Sterile 4×4 sponges
- 5 mL syringe
- 18 gauge needles
- Self-retaining skin retractors
- Small rake, two
- Needle driver*
- Silk suture, 3–0 and 4–0*
- Injectable sterile saline
- Intravenous tubing and solution
- Wound dressing supplies
- Antibiotic ointment

The comprehensive list of equipment is listed above (**Figure 69-4**). The equipment is contained in a cutdown kit. Many Emergency Departments do not have cutdown kits available. The basic equipment (**Figure 69-5**) denoted by an asterisk is contained in a chest tube tray or thoracotomy tray. Neither **Figure 69-4** nor **Figure 69-5** shows the intravenous tubing, normal saline bag, and dressing supplies that are available in every Emergency Department.

PATIENT PREPARATION

The patient is usually in extremis and positioned supine if a peripheral venous cutdown is to be performed. They may be in the Trendelenburg position, although this is not ideal for the procedure. Secure the selected extremity to the bed with a restraint, tape, or an assistant holding it in place. **Perform a cutdown in as sterile a manner as possible.**

Identify the landmarks for the procedure. Clean the skin of any dirt and debris. Anesthetize the area of the cutdown if the patient is conscious. Infiltrate local anesthetic solution into the subcutaneous tissue where the incision will be made. Prepare the skin with povidone iodine or chlorhexidine solution and allow it to dry. Apply sterile drapes to isolate a surgical field. Collect and set up all the required equipment on a bedside table covered with a sterile drape.

FIGURE 69-4. Comprehensive equipment.

FIGURE 69-5. Basic equipment.

TECHNIQUES TO ISOLATE THE VEINS

The technique of cannulation is the same regardless of the vein chosen. The methods to isolate the saphenous vein and the basilic vein will be discussed in this section. A discussion of the different techniques to cannulate the isolated vein will be presented in the following section.

GREATER SAPHENOUS VEIN ISOLATION AT THE ANKLE

The saphenous vein is reliably found and easily isolated at the ankle (**Figures 69-6 and 69-7**). Extend and externally rotate the lower extremity. Identify the medial malleolus of the tibia. Find the location 2.5 cm anterior and 2.5 cm superior to the medial malleolus. The greater saphenous vein can often be found at this site (**Figures 69-6*A* and 69-7*A***). Alternatively, place the index and middle fingers at the level of the malleolus. The vein will be found two finger breadths cephalad and two finger breadths anterior to the medial malleolus. The vein may be palpable if the patient is not hypovolemic or obese. The vein may be visualized in patients with thin skin, superficial veins, minimal subcutaneous tissue, or dark vessels.

Stretch the skin taught over the distal tibia with the nondominant hand (**Figures 69-6*B* and 69-7*B***). Note the position of the hands in the illustration. **The nondominant hand is placed with the fingertips pointing toward the Emergency Physician.** This will prevent inadvertent injury while making the skin incision. Transversely incise the skin overlying the great saphenous vein using a #10 scalpel blade. Make the incision from the anterior tibial border to the posterior tibial border after appropriate anesthesia (**Figures 69-6*B* and 69-7*B***). **This incision should be superficial so that the subcutaneous tissue is barely exposed.** A deep incision may inadvertently transect the vein causing significant bleeding, difficulty visualizing the surgical field, and difficulty completing the procedure due to subcutaneous venous retraction. Apply tension to the skin on either side of the incision to expose the underlying structures. This can be

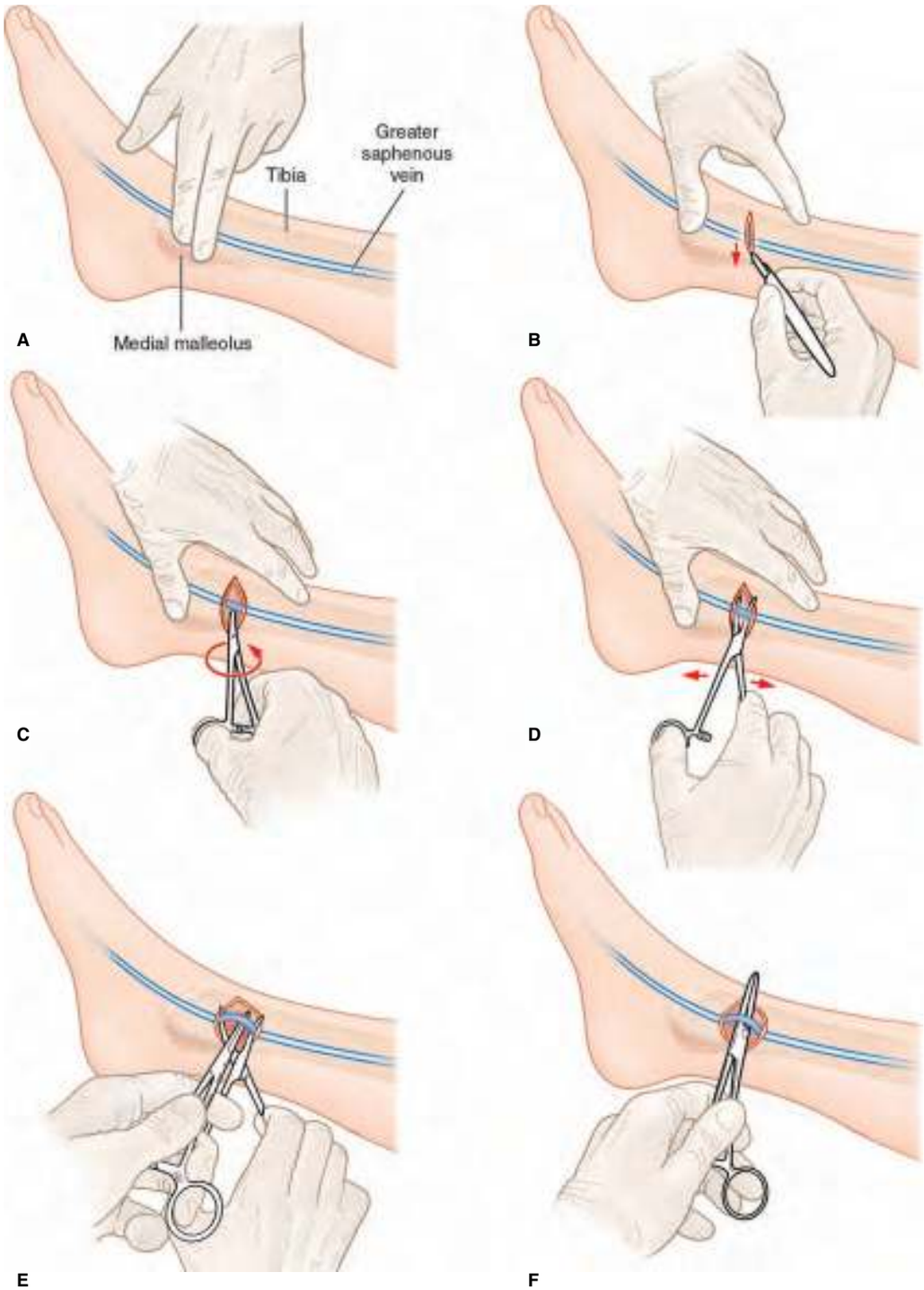
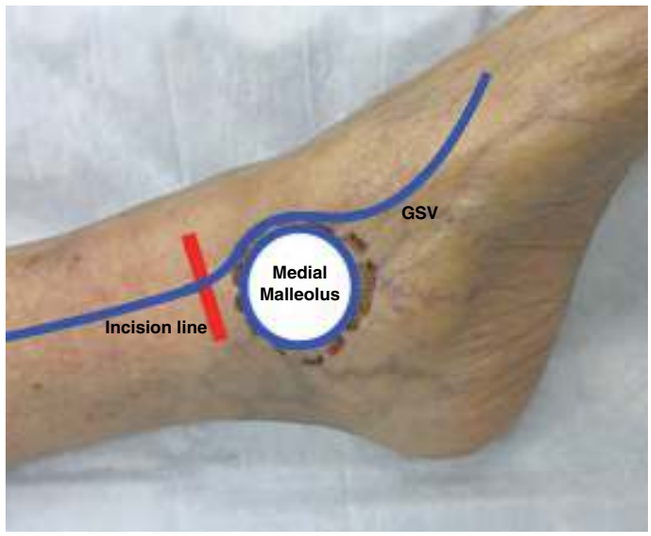


FIGURE 69-6. Artist illustration of isolation of the greater saphenous vein at the ankle. ***A.*** Identifying the vein. ***B.*** A transverse skin incision is made from the anterior to the posterior border of the medial tibia. ***C.*** The tip of a curved hemostat is scraped along the tibia and then rotated 180°. ***D.*** The hemostat is spread to separate the tissues. ***E.*** A straight hemostat is inserted between the jaws of the curved hemostat to elevate the vein. ***F.*** The curved hemostat has been removed.

accomplished with the nondominant hand, a self-retaining retractor, or skin rakes held by an assistant.

Isolate the greater saphenous vein.[8] This may be difficult in some patients in shock or patients in the Trendelenburg position as the vein may be poorly perfused. Grasp and hold a curved hemostat or a Kelly clamp with the tip facing downward. Insert the hemostat along the posterior border of the tibia and scrape the tip anteriorly along the tibia. If done properly, all the tissue between the skin and the tibia will be above the hemostat. If not visible, cautiously blunt dissect with the curved hemostat down to the vein. When identified, insert the hemostat under the vein and rotate the hemostat 180°. The tip of the hemostat will be facing upward (**Figures 69-6*C* and 69-7*C***). Widely open the arms of the hemostat (**Figures 69-6*D* and 69-7*D***). This will open the jaws of the hemostat and separate the saphenous vein from the saphenous nerve and fibrous strands of connective tissue. The saphenous vein should be visible between



A

B

C

D

E

F

FIGURE 69-7. Isolation of the greater saphenous vein at the ankle in a cadaver. ***A.*** Identifying the vein. The long blue line is the greater saphenous vein (GSV). ***B.*** A transverse skin incision is made. ***C.*** The greater saphenous vein is isolated. ***D.*** The hemostat is spread to separate the tissues. ***E.*** A straight hemostat is inserted between the jaws of the curved hemostat to elevate the vein. ***F.*** The curved hemostat has been removed.

the jaws of the hemostat. If there is difficulty identifying the vein, squeeze the foot to fill the vein with blood. Insert a straight hemostat or a Kelly clamp between the jaws of the curved hemostat and below the greater saphenous vein (**Figures 69-6*E* and 69-7*E***). Remove the curved hemostat to leave the straight hemostat elevating the greater saphenous vein (**Figures 69-6*F* and 69-7*F***). The straight hemostat will be useful as a "cutting board" to later transect the vein and allow more control of the vein.

An alternative technique to isolate the vein is used by some physicians. **This technique is not recommended by the editor but is briefly described for the sake of completeness.** Make the transverse skin incision. Place the jaws of a curved hemostat parallel to the greater saphenous vein. Open the arms of the hemostat to allow the jaws to dissect through the subcutaneous tissue. Continue placing the hemostat and opening the jaws until the vein is isolated. This technique is harder to perform because the vein is less likely to be identified given the white background of the periosteum.[1,12,13] In addition, this technique takes significantly longer to find and isolate the vein.

GREATER SAPHENOUS VEIN ISOLATION AT THE GROIN

The groin vasculature offers the potential for massive infusion of blood or fluids in a matter of minutes.[5] These vessels are closer to the central circulation and large enough to easily accommodate intravenous tubing cut off at a 45° as a catheter. The greater saphenous vein is superficial at the groin and lies in a meshwork of subcutaneous tissue. It is superficial to the femoral artery and vein. The saphenous vein travels on the anteromedial surface of the thigh and enters the fossa ovalis to join the femoral vein (**Figure 69-2*B***). The greater saphenous vein is at its largest diameter 3 to 4 cm distal to the inguinal ligament. This is approximately 2 cm below the site for placement of a femoral central venous line and level with where the scrotal or labial fold meets the thigh.

Identify the location where the scrotal or labial fold meets the thigh (**Figure 69-8**). Identify the lateral edge of the mons pubis. Identify the point where a vertical line from the lateral edge of the mons pubis meets a horizontal line from the scrotal/labial fold. Make a transverse, medial to lateral, incision with a #10 scalpel blade on the patient's thigh starting where the scrotal or labial fold meets the thigh after appropriate anesthesia. Extend the incision laterally until it meets the vertical line from the lateral edge of the mons pubis.

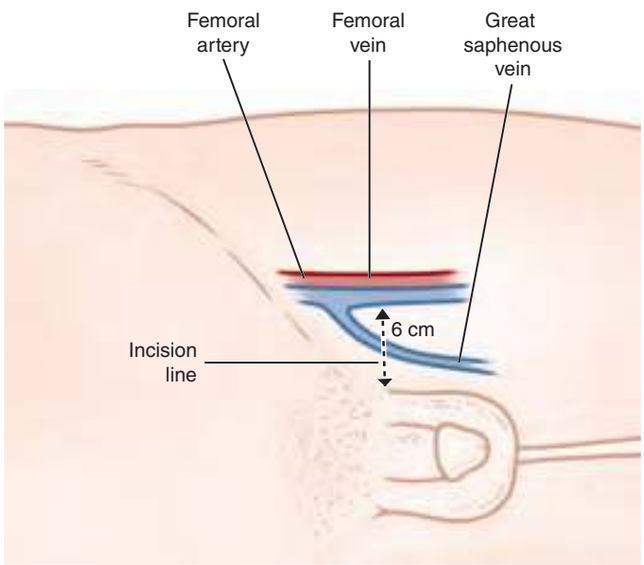


FIGURE 69-8. Isolation of the greater saphenous vein at the groin. The skin incision should begin where the scrotal or labial fold meets the thigh. Extend the incision laterally until it meets a vertical line from the lateral edge of the mons pubis.

Dissect the subcutaneous tissue to locate the greater saphenous vein. Place the jaws of a curved hemostat parallel to the greater saphenous vein. Open the arms of the hemostat to allow the jaws to dissect through the subcutaneous tissue. Continue placing the hemostat and opening the jaws until the vein is isolated. **The dissection is too deep if the deep investing fascia or the muscle bellies of the thigh muscles are encountered.** Stop, reidentify the landmarks, and adjust the skin incision and dissection as necessary.

The subcutaneous tissues can alternatively be bluntly dissected using 4×4 gauze squares. Grasp two or three gauze squares in each hand. Put the fingertips of both hands, covered with gauze, in the center of the incision. Move the hands in opposite directions (i.e., cephalad and caudad) while scraping the subcutaneous tissue with the gauze. Reapply the hands in the incision and repeat the motion until the greater saphenous vein is exposed. This technique applies pressure parallel to the greater saphenous vein and will not injure the vein.

BASILIC VEIN ISOLATION AT THE ELBOW

The basilic vein may be used for a peripheral venous cutdown. This is often performed when the greater saphenous vein cannot be accessed due to lower extremity amputation, deformity, injury, or trauma, or if it has been used previously (e.g., in a coronary bypass). This site is not ideal as it may interfere with resuscitative efforts while the basilic vein is being exposed. The basilic vein is consistently found 2 cm cephalad and 1 to 2 cm lateral to the medial epicondyle of the humerus on the volar surface of the arm. It may also be found in the groove between the biceps and triceps muscles. There is controversy in the literature as to where the incision for the basilic vein cutdown should be performed. The simple answer is that if one fails in isolating the vein in one location, make an incision in the second location to isolate the vein.

Position the patient to allow exposure of the basilic vein. Abduct the patient's arm 90° with the elbow flexed 90° and the palm facing upward (**Figure 69-9**). This positioning is required to access the basilic vein at either location.

Identify the point 2 cm cephalad and 2 cm lateral from the medial epicondyle of the humerus. This is the location of the basilic vein. Make a 4 to 6 cm transverse incision with a #10 scalpel blade centered on the reference point after appropriate anesthesia. The incision should only cut through the epidermis. Bluntly dissect the subcutaneous tissue with a curved hemostat or 4×4 gauze squares, as described previously, to locate the basilic vein. **The dissection is too deep if the brachial artery, median nerve, or muscle fibers are encountered.** Stop, reidentify the landmarks, and adjust the skin incision as necessary.

Alternatively, the basilic vein can be isolated in the middle of the distal third of the arm. Palpate the groove between the biceps and triceps muscles. This is the location of the basilic vein. Make a 4 to 6 cm horizontal incision centered about the groove after appropriate anesthesia. Bluntly dissect with a curved hemostat until the vein is located. The basilic vein is superficial to the muscle fascia and the brachial artery.

TECHNIQUES FOR CANNULATION OF THE VEIN

There are numerous techniques to cannulate a vein after it has been isolated. Any of the following techniques can be used to cannulate the greater saphenous vein or the basilic vein. The rapid and definitive cannulation of the vessel is the primary goal and not the chosen technique.

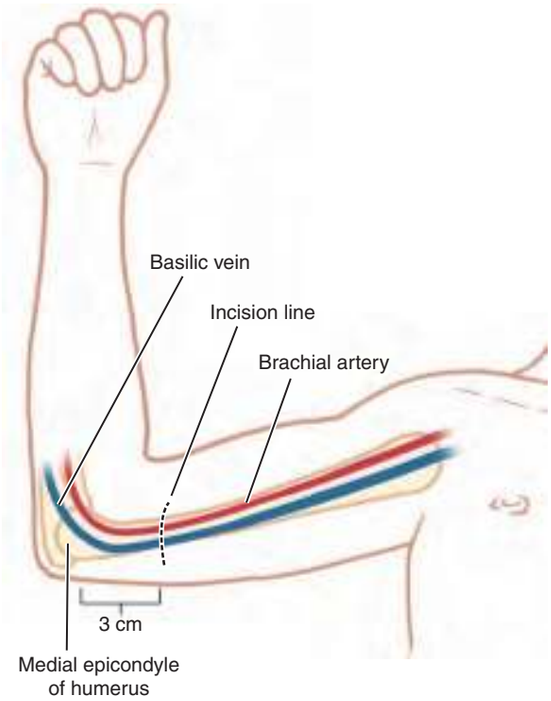


FIGURE 69-9. Isolation of the basilic vein.

GENERAL ACCESS TO THE VEIN

Isolate the chosen vein as described previously. Place a straight hemostat or a Kelly clamp under the midportion of the vein (**Figure 69-10*A***). Slightly elevate the hemostat. Pass a silk suture under the vein at its proximal end and a second silk suture at its distal end (**Figures 69-10*B* and 69-11*A***). Grasp the silk sutures with a hemostat to maintain the position of the tie and to allow for manipulation of the vein. Tie the distal suture to occlude inflow of blood from distal veins (**Figures 69-10*C* and 69-11*B***). **Do not cut either of these two sutures. The proximal suture will be left untied to allow for control and manipulation of the vein.**

Incise the vein. Grasp the hemostat holding the proximal suture with the nondominant hand. Raise the hemostat to flatten the vein and prevent back bleeding. Make a small incision through a third to half of the vein with the tip of a #11 scalpel blade (**Figures 69-10*D*, 69-11*C*, and 69-11*D***).[12,14] **Do not cut the entire vein as this will cause significant bleeding and loss of the proximal end. If the incision is too large, greater than one-half the vein's diameter, the vessel may be torn completely and retract from the surgical field.[12,14] If the incision is too small, the catheter will enter a false lumen in the vein wall.[12,14]** The straight hemostat below the vein will act as a "cutting board" and prevent injury to underlying structures from the scalpel blade. As an alternative, the jaws of the straight hemostat can be opened and the vein cut with an iris scissors (**Figure 69-10*E***).

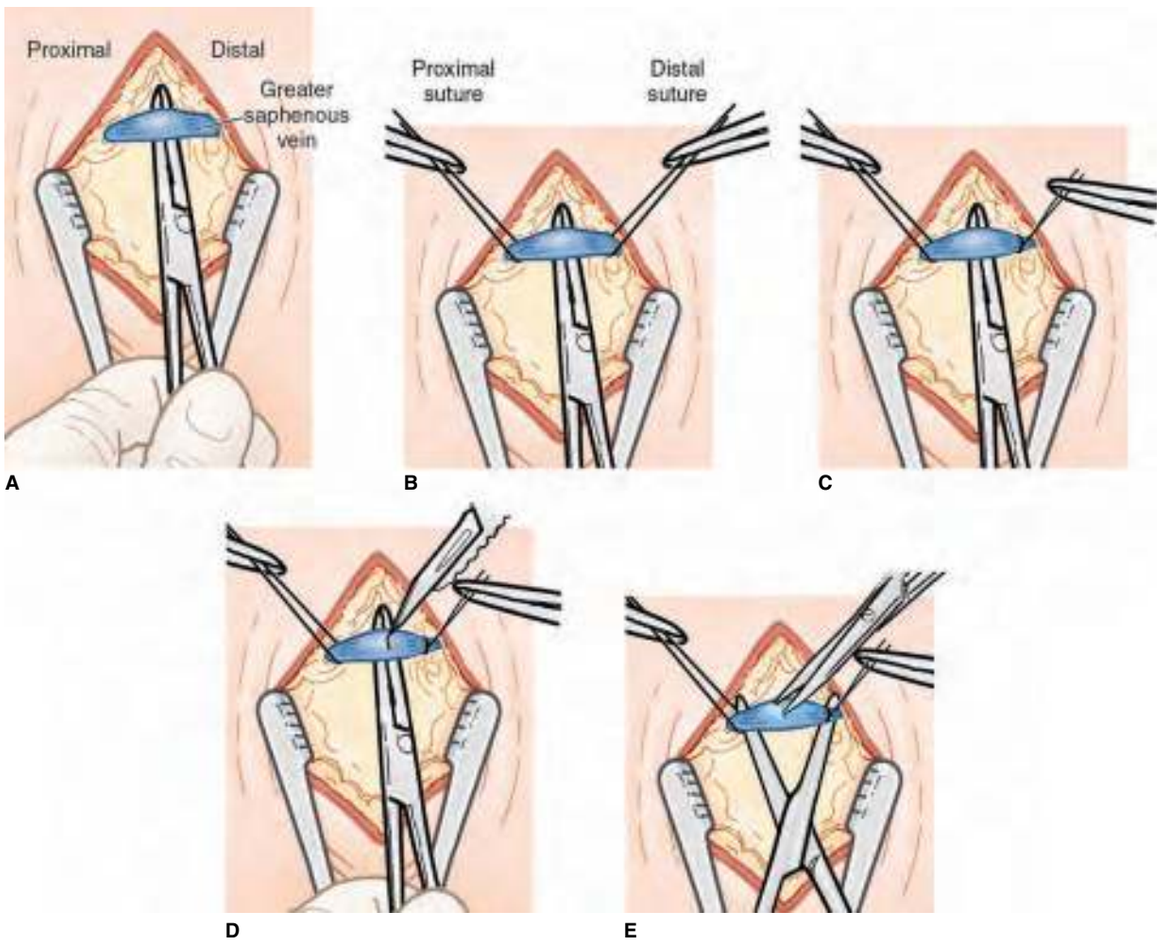


FIGURE 69-10. Artist illustration of general access to the vein. ***A.*** The vein has been isolated. ***B.*** A silk suture has been placed proximally and distally around the vein. ***C.*** The distal suture is tied. ***D.*** An incision is made through the vein with a #11 scalpel blade. ***E.*** Alternatively, the hemostat is opened and an iris scissors is used to cut the vein.

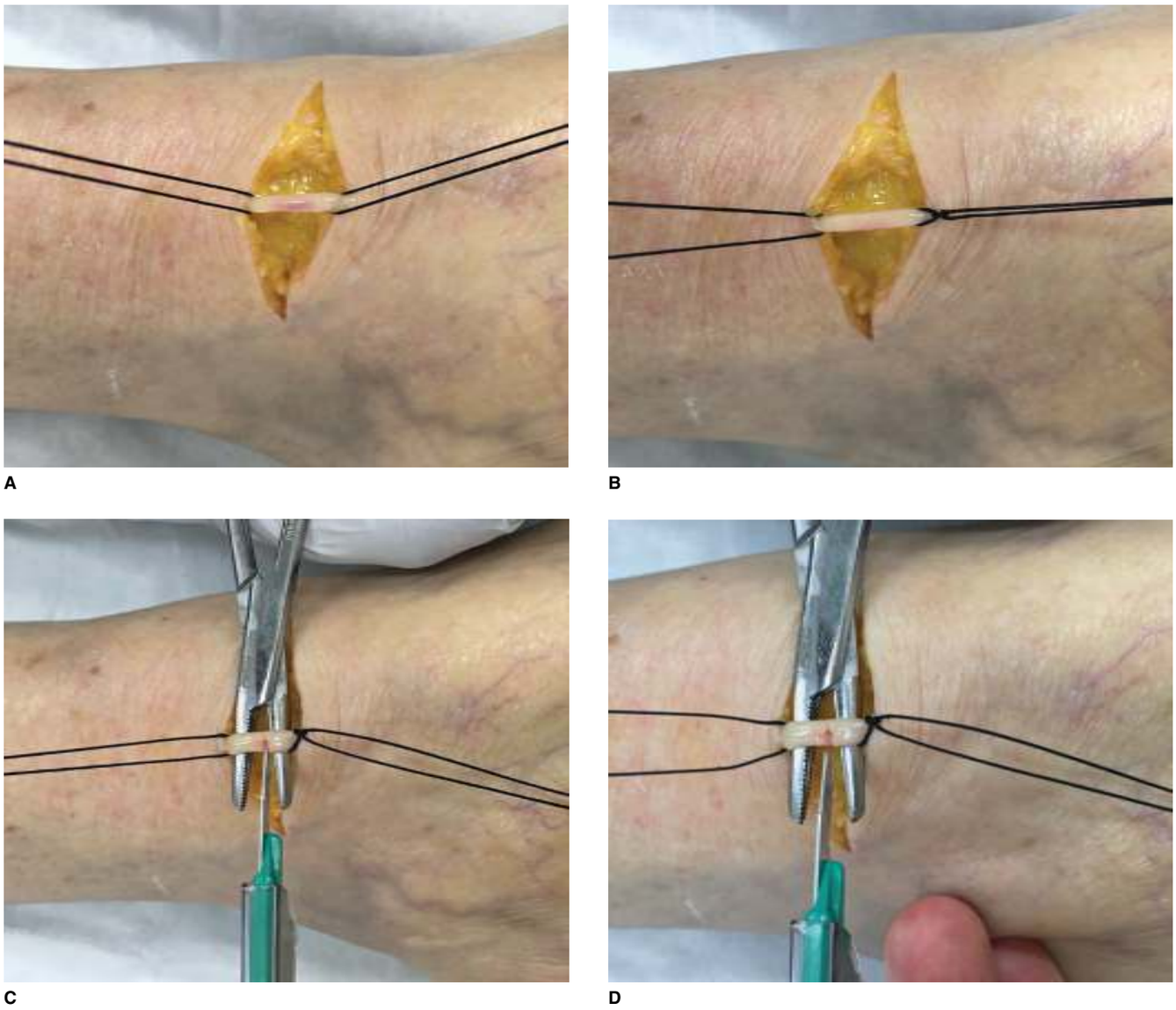


FIGURE 69-11. General access to the vein in the cadaver. ***A.*** The vein has been isolated. A silk suture has been placed proximally and distally around the vein. ***B.*** The distal suture is tied. ***C.*** An incision is being made through the vein with a #11 scalpel blade. ***D.*** An incision has been made through the vein.

SELDINGER TECHNIQUE

The traditional technique for peripheral venous cannulation can use the Seldinger method.[13,15] This technique will insert the catheter as if cannulating a central vein (Chapter 63). The required equipment can be found in a prepackaged central venous line access kit. This includes the guidewire, introducer sheath, dilator, and the venous catheter. This technique can accommodate a large caliber line, such as an 8 or 9 French introducer sheath. This technique may save 1 to 2 minutes on cannulation time by eliminating the ligature and tie off steps.

Isolate the chosen vein as described previously. Place a straight hemostat or a Kelly clamp under the midportion of the vein and open the jaws (**Figure 69-12*A***). Insert the catheter-over-the-needle into the vein (**Figure 69-12*A***). Stop advancing the catheter-over-the-needle when a flash of blood is seen in the needle hub. **Be cautious not to puncture the posterior wall of the vein.** Remove the straight clamp. Advance the catheter into the vein while securely holding the needle. Remove the needle. Insert the guidewire through the catheter. Remove the catheter by backing it out over the guidewire, which will remain in the vein. Place the dilator through the introducer sheath. Feed the dilator-introducer sheath unit over the guidewire (**Figure 69-12*B***). Advance the unit into the vein with a twisting motion while securely holding the guidewire (**Figure 69-12*C***). Continue to advance the unit until the hub of the introducer sheath is just above the vein (**Figure 69-12*D***). Remove the guidewire and dilator as a unit (**Figure 69-12*D***). Attach intravenous tubing to the hub of the introducer sheath and begin instilling fluids.

MODIFIED SELDINGER TECHNIQUE

An alternative and quicker method can be used to insert the introducer sheath into the vein (**Figure 69-13**). Isolate the chosen vein as described previously. Place a straight hemostat or a Kelly clamp under the midportion of the vein and open the jaws (**Figure 69-13*A***). Assemble the unit by placing the dilator through the sheath and insert the guidewire through the dilator (**Figure 69-13*B***). The guidewire should protrude 3 to 4 mm beyond the tip of the dilator. Make an incision in the lateral half of the vein (**Figure 69-13*C***). Hold the proximal

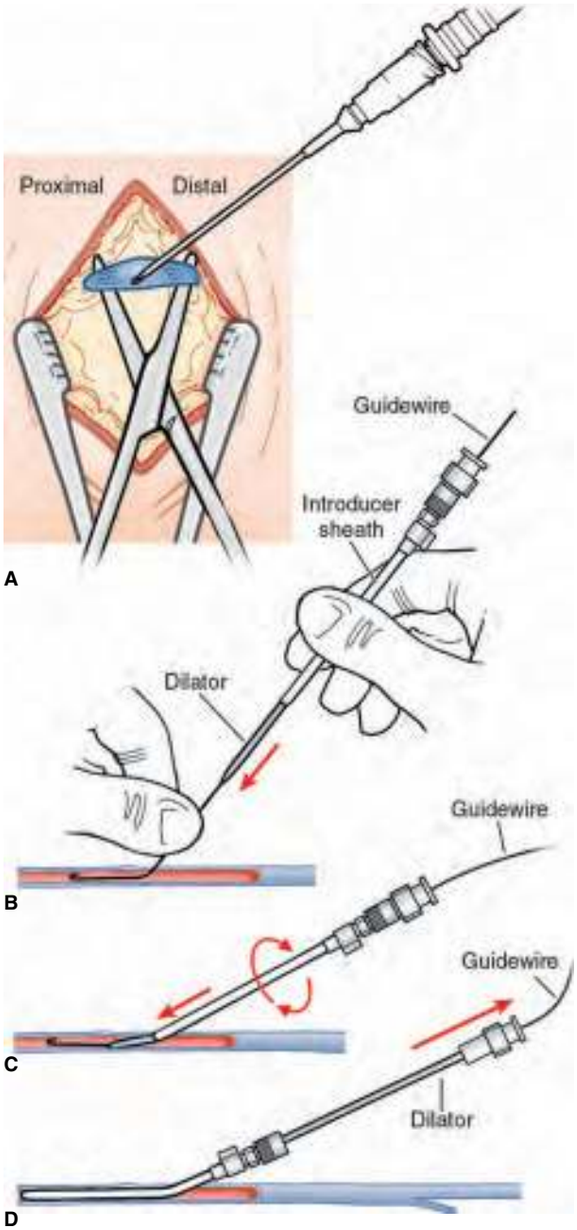


FIGURE 69-12. The Seldinger technique of venous cannulation. ***A.*** The vein has been isolated. The catheter-over-the-needle is inserted into the vein. ***B.*** A guidewire has been placed into the vein. The dilator and sheath are fed over the guidewire. ***C.*** The dilator and sheath are advanced into the vein with a twisting motion. ***D.*** The guidewire and dilator are removed as a unit.

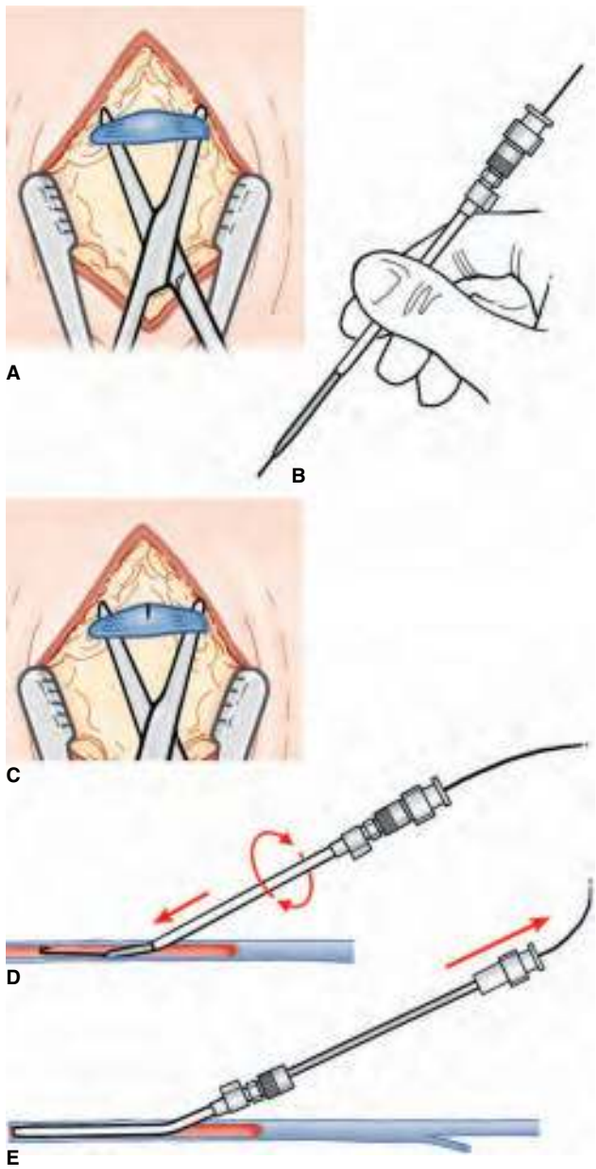


FIGURE 69-13. The modified Seldinger technique of venous cannulation. ***A.*** The vein has been isolated. ***B.*** The dilator, guidewire, and sheath are assembled as a unit. ***C.*** An incision has been made in the vein. ***D.*** The unit is inserted into the vein, guidewire first. A twisting motion will aid in its insertion. ***E.*** The guidewire and dilator are removed as a unit.

guidewire and sheath. Insert the distal guidewire into the vein. Continue to insert the entire unit with a twisting motion into the vein (**Figure 69-13*D***). Continue to advance the unit until the hub of the introducer sheath is just above the vein. Remove the guidewire and dilator as a unit (**Figure 69-13*E***). Attach intravenous tubing to the hub of the introducer sheath and begin instilling fluids.

SURGICAL TECHNIQUE USING INTRAVENOUS TUBING

Use an assistant to prepare the intravenous tubing. Attach sterile intravenous polyethylene tubing to a bag of sterile saline. Cut the angiocatheter attachment hub off the end of the tubing at a 45° angle. Some authors suggest using a feeding tube instead of intravenous tubing.[12] This is not recommended. Intravenous tubing is ubiquitously available. The rounded tip of the feeding tube may be more difficult to advance into the vein. The only advantage to using a feeding tube is that the rounded tip has less chance of puncturing the posterior wall of the vein.

Insert the angled edge of the intravenous tubing into the vein. Gently relax the tension on the proximal suture to allow the vein to open. Advance the intravenous tubing 2 to 3 cm into the vein (**Figure 69-14*A***). There is often considerable difficulty advancing the catheter. **Do not force the catheter through the vein as it is very delicate.** Troubleshoot by removing the catheter and make sure that the lumen of the vein has been cannulated. This is sometimes difficult to accomplish. If so, have an assistant control the proximal suture. Use a mosquito hemostat to grasp the cut edge of the vein and lift upward to expose the vein's lumen and insert the intravenous tubing (**Figure 69-14*B***). A mosquito hemostat may be too large to grasp the cut edge of small veins. Insert a vein

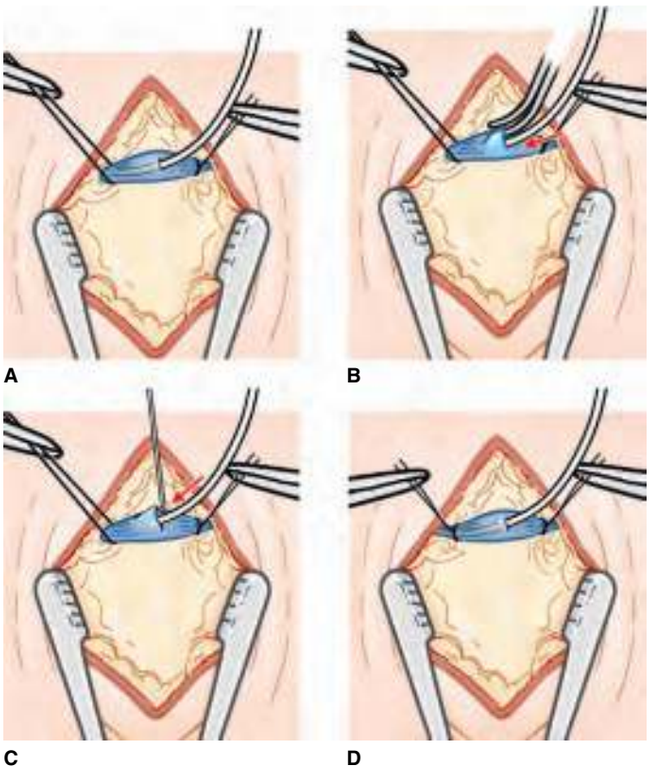


FIGURE 69-14. Venous cannulation using intravenous tubing. ***A.*** The tubing is inserted into the vein and advanced. ***B.*** A mosquito hemostat can be used to grasp the vein and hold it open while the tubing is inserted. ***C.*** For small veins, a vein pick or 18 gauge needle with the tip bent can be used to hold open the vein. ***D.*** The proximal suture is tied to secure the tubing.

pick or an 18 gauge needle with the tip bent into a 90° angle into the lumen of the vein (**Figure 69-14*C***). Lift upward to expose the vein's lumen and insert the intravenous tubing.

Palpate the posterior aspect of the vein after inserting the intravenous tubing for penetration of the catheter. **The catheter must be removed if it penetrates through the posterior wall of the vein.** Release the proximal suture and allow the intravenous fluid to flow into the vein if the tubing has not penetrated the posterior wall of the vein. Tie the proximal suture to secure the intravenous tubing within the vein if the fluid flow is unobstructed and the fluid is not extravasating into the surrounding tissues (**Figure 69-14*D***). **Do not tie the suture too tight to occlude the tubing.** Tying the suture too tightly may also result in a thrombosis.[11] If the tubing is within the lumen of the vein and the fluid is not flowing, the tubing may be against a venous valve. Gently advance the catheter 2 to 3 mm or withdraw it 2 to 3 mm and observe the fluid for flow.

An alternative method is available (**Figure 69-15**). Expose the vein as described previously. Grasp and elevate the distal skin edge with a hemostat. Make a stab incision with a #11 scalpel blade approximately 1 cm distal to the previously made skin incision (**Figure 69-15*A***). **Use caution not to cut the underlying vein.** Insert the intravenous tubing through the stab incision and pull it through the skin incision. Incise the vein and insert the tubing as described previously. This allows the tubing to be tunneled through the subcutaneous tissue before cannulating the vein (**Figure 69-15*B***).

INTRAVENOUS CATHETER TECHNIQUE

This technique involves the insertion of a standard, 16 to 18 gauge, intravenous catheter-over-the-needle into the vein (**Figures 69-16 and 69-17**). This technique is sometimes referred

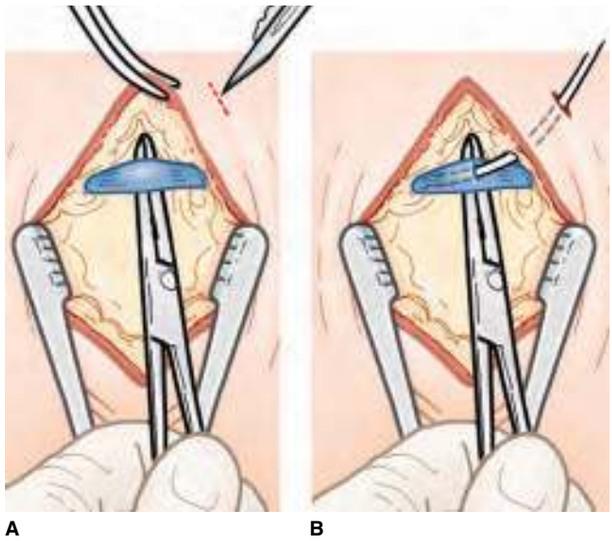


FIGURE 69-15. Alternative technique of venous cannulation with intravenous tubing. ***A.*** The distal skin edge is elevated. A #11 scalpel blade is used to make a stab incision in the skin and subcutaneous tissues. ***B.*** The tubing is fed through the stab incision and into the vein.

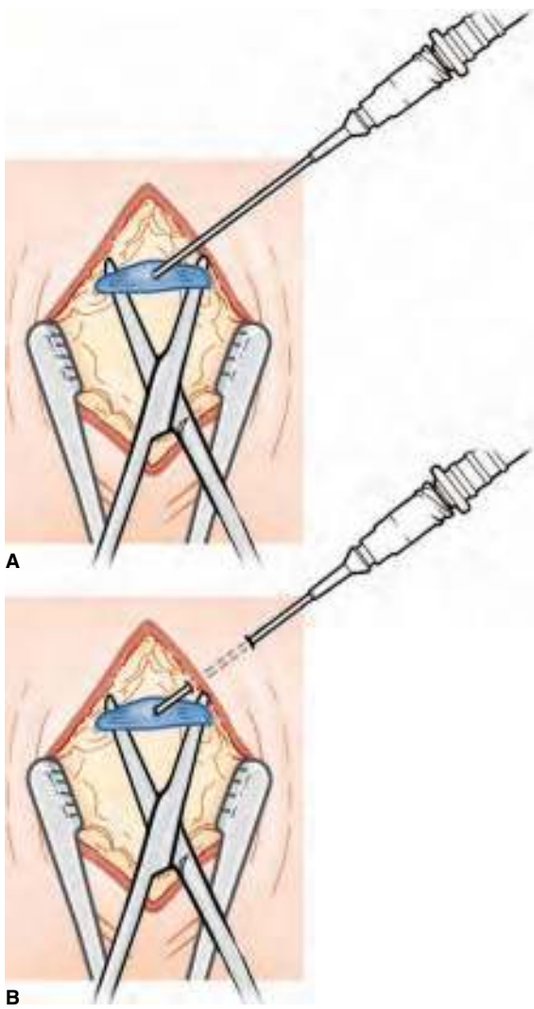
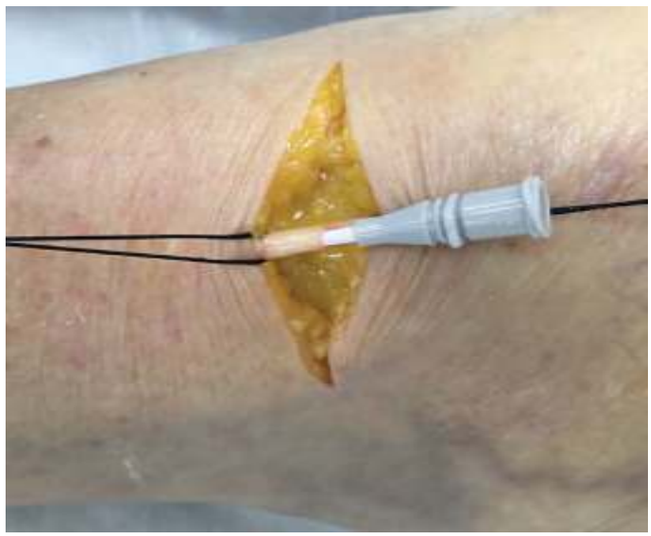


FIGURE 69-16. Artist illustration of insertion of the catheter-over-the-needle. ***A.*** Insertion through the incision and into the vein. ***B.*** Insertion through the skin and into the vein.

A



B

C

FIGURE 69-17. Insertion of the catheter-over-the-needle in a cadaver. ***A.*** Insertion through the incision and into the vein. ***B.*** The catheter has been advanced and the needle removed. ***C.*** Insertion through the skin and into the vein.

to as the mini-cutdown. The vein is cannulated under direct visualization through a skin incision. This technique will insert the catheter as if cannulating a peripheral vein (Chapters 59 and 61). This method is very quick, provides a more secure cannulation, and potentially decreases the chance of infection.

Isolate the chosen vein as described previously. Place a straight hemostat or a Kelly clamp under the midportion of the vein and open the jaws (**Figure 69-16*A***). Insert the catheter-over-the-needle into the incision and the vein under direct visualization (**Figures 69-16*A* and 69-17*A***). Stop advancing the unit when a flash of blood is seen in the needle hub. Advance the catheter into the vein while removing the hemostat. Advance the catheter until the hub is just above the vein (**Figure 69-17*B***). Remove the needle. Attach intravenous tubing to the catheter hub and begin instilling fluids. Gently tie the proximal suture to secure the catheter within the vein.

Alternatively, insert the catheter-over-the-needle through the skin, 1 cm distal to the distal skin edge until the tip is visualized in the incision (**Figures 69-16*B* and 69-17*C***). Insert the catheter-over-the-needle into the vein under direct visualization. Stop advancing the unit when a flash of blood is seen in the needle hub. Advance the catheter into the vein while removing the straight clamp. Advance the catheter until the hub is against the skin (**Figure 69-17*C***). Remove the needle. Attach intravenous tubing to the catheter hub and begin instilling fluids. The advantage of this method is that the catheter goes through the skin, which will stabilize the catheter and prevent it from becoming dislodged.

AFTERCARE

It is not necessary to close the skin incision immediate after the cutdown. Place a saline moistened gauze over the incision site. Wrap a sterile dressing around the extremity and the skin incision site. The skin incision must be closed after more definitive access has been obtained. If the intravenous tubing was inserted as above, the skin incision will be sutured closed and the tubing will exit the incision. Some feel this may allow access of bacteria through the wound and into the underlying vein.

Secure the cutdown more permanently if the patient survives the episode for which a venous cutdown was performed. Suture the wound closed with simple interrupted 4–0 nylon sutures (**Figures 69-18 and 69-19**). Place a suture through the skin, wrap it around the catheter, and tie it to secure the catheter to the skin (**Figure 69-18**). Apply antibacterial ointment to the incision, the sutures, and the site where the catheter exits the skin. Secure the intravenous tubing (**Figure 69-19*B***). The catheter can be looped around the toe, for a cutdown at the ankle, and secured with gauze wrap or an elastic wrap (**Figure 69-20*A***). The catheter can be secured by taping it to the skin (**Figures 69-19*B* and 69-20*B***). The limb can be immobilized on a board, after the catheter is secured at the ankle or elbow, for added security from inadvertent dislodgement of the catheter (**Figure 69-20*C***).

COMPLICATIONS

The complications of a peripheral venous cutdown include arterial injury, nerve injury, phlebitis, thromboembolism, wound dehiscence, and wound infection. The incidence of complications ranges from 2% to 15%.[16,17] The true complication rate is difficult to report given the high mortality rate in patients undergoing this procedure due to the primary problem (e.g., hypovolemia, sepsis, shock, or trauma). The knowledge of the local anatomy and the technical aspects of the various cutdown techniques may avoid arterial, venous, and nerve injury. The other complications may be reduced by removal of the catheter within 12 hours after placement.[17]

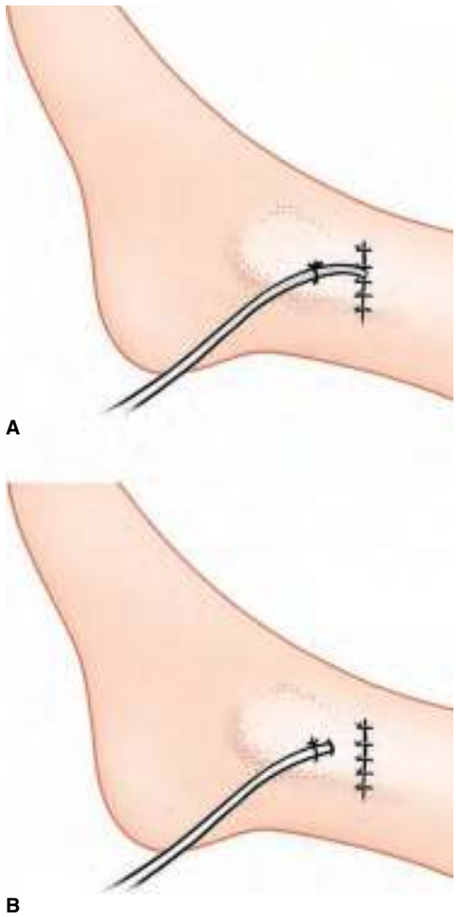


FIGURE 69-18. The skin incision is closed with interrupted 4–0 nylon sutures. The catheter exits the original incision (***A***) or a separate skin incision (***B***).

It can take time to insert the tubing or central line catheter into the vein.[18] The patient can deteriorate very quickly within this time. The cutdown procedure can be quicker if using a vein pick to open the cut vein, an 18 gauge needle prebent to 90° to open the cut vein, or a catheter-over-the-needle (i.e., mini-cutdown technique).

PHLEBITIS

It is generally agreed that phlebitis is more likely to occur in the lower extremity than the upper extremity although there is littledata to support this. Phlebitis, an inflammation of the vein, usually results from prolonged catheterization. It may be seen within hours of catheter placement to 18 days after the removal of the catheter.[17] In 1960, Bogen looked at 234 ankle cutdowns and found a 4% phlebitis rate.[19] He felt this was secondary to infection. The strength of the correlation was attributed to a previous unrelated study that found *Staphylococcus aureus* on almost all catheter tips in patients with phlebitis as opposed to catheter tips in patients without phlebitis. These cases of phlebitis all resolved without the use of antibiotics suggesting an inflammatory, rather than infectious, process to be the more likely etiology. Moran et al cultured 89 cutdown sites and observed that the pathogenic species causing infection were *S. aureus, Enterococcus*, and *Proteus*.[17] These organisms were cultured more frequently in patients who had cutdowns that were left in place for longer periods of time. They did not find a correlation between infection and phlebitis and postulated that phlebitis was due to irritation of the vein wall by the catheter.[17] **It is clear from these studies that early removal of the intravenous catheter within 12 hours of placement will significantly decrease the incidence of phlebitis.**

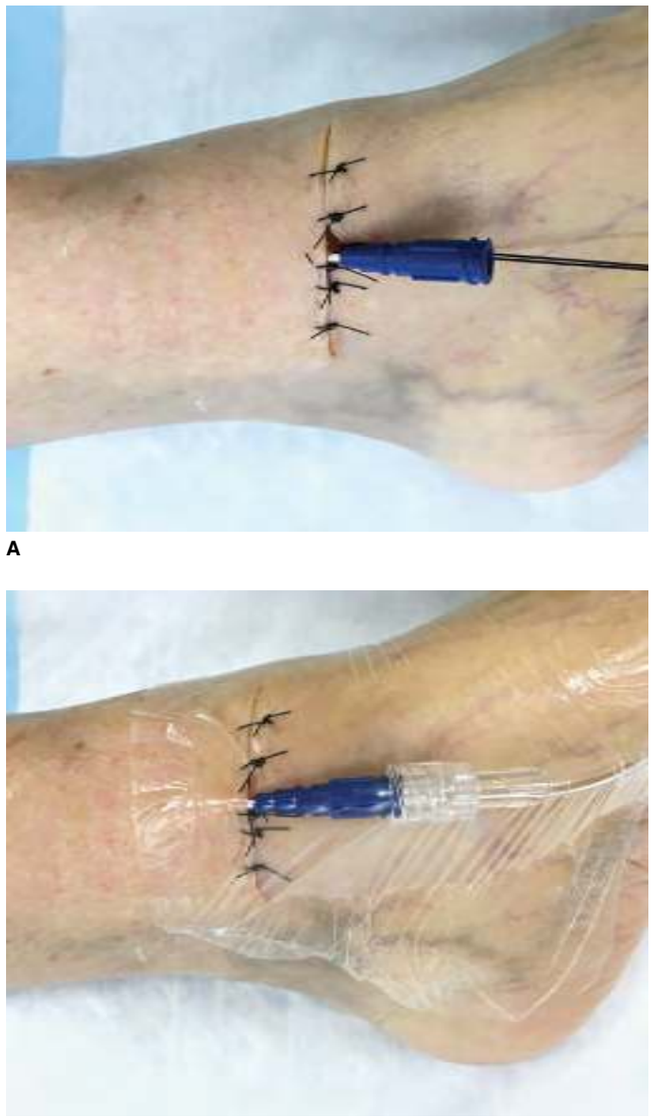


FIGURE 69-19. The skin incision is closed in a cadaver (***A***) and covered with a clear dressing (***B***).

INFECTION

It was not found that prophylactic antibiotics reduced infection rates.[17] The application of daily topical antibiotic ointment reduced positive local wound cultures from a rate of 78% to 18%. Collins et al studied polyethylene catheters and found a 2% bacteremia rate and a 1% death rate from *Pseudomonas* species in debilitated patients.[20] In 1988, Rhee et al found only one case of cellulitis in their study of 78 patients.[15] **It is clear from these studies that early removal of the intravenous catheter within 12 hours of placement will significantly decrease the incidence of infection and subsequent complications.** Sterile technique is encouraged with this procedure to minimize complications related to infection.

ASSOCIATED INJURIES

Injury to adjacent arteries, nerves, and veins can be avoided by a detailed understanding of the local anatomy and careful procedural

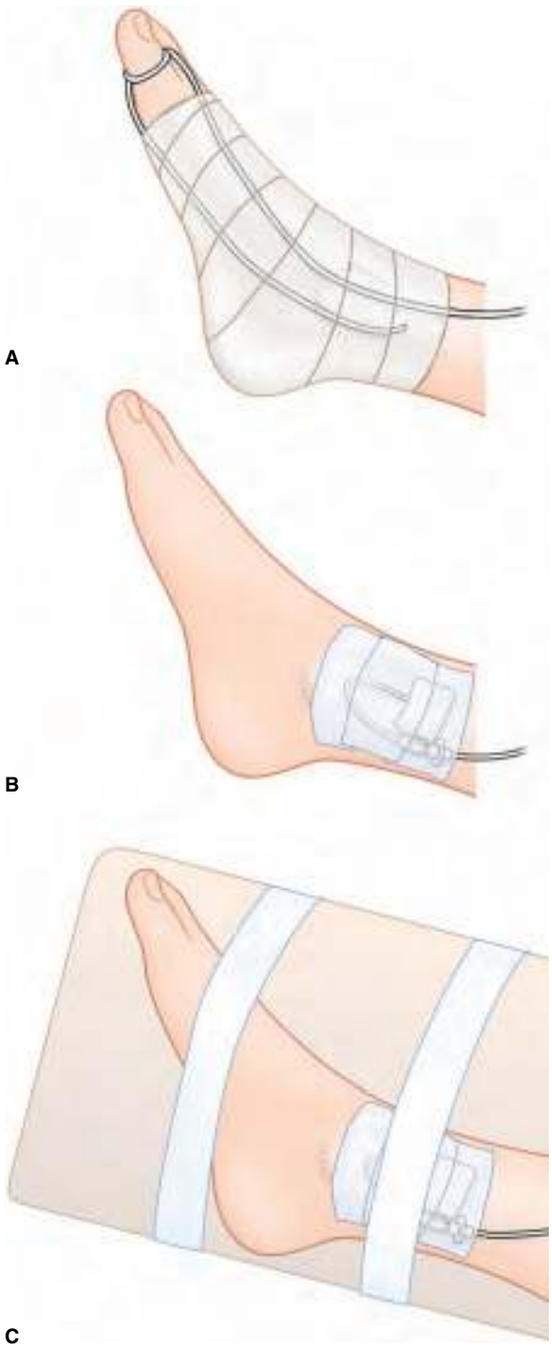


FIGURE 69-20. Securing the intravenous catheter. ***A.*** The catheter is looped around the great toe, for an ankle cutdown, and secured with gauze or elastic wrap. ***B.*** The catheter is secured with tape. ***C.*** The ankle or elbow can be secured to a board for additional security.

technique. Aggressive and forceful dissection without an understanding of the anatomy or the procedure will increase the incidence of complications. Injury to adjacent cutaneous nerves is unavoidable and inconsequential. Venous spasm, which causes nonuniform acceptance of the intravenous extension tubing, may also occur.[17]

SUMMARY

The peripheral venous cutdown is an excellent technique for rapid fluid or blood product infusion. This is usually performed when other methods of venous access are unavailable or have failed. It is a relatively simple procedure. If learned properly, it can be lifesaving in the critically ill or injured patient. It is imperative to understand the relevant local anatomy and identify the clinical landmarks before this procedure is performed. Strict adherence to sterile technique and the early removal of the catheter will decrease the rate of infection and other complications.

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70 Intraosseous Infusion

Beech S. Burns, James Heilman, and O. John Ma

INTRODUCTION

Obtaining peripheral vascular access in the critically ill patient may be difficult and time consuming. The vascular collapse that may accompany severe dehydration or a cardiac arrest can be profound and delay administration of essential therapies. Pediatric patients present a challenge due to the small size of their peripheral veins and the increased subcutaneous tissue. Administration of endotracheal medications may not provide rapid and reliable drug

absorption during a cardiorespiratory arrest.[1,2] Establishing vascular access quickly in these cases can be lifesaving.

Intraosseous (IO) access was first described in 1922 by Dr. Drinker. He referred to the medullary cavity as a "noncollapsible vein" that can be used for obtaining rapid vascular access. IO access for pediatric use was introduced in 1941, and the first IO blood transfusion was documented in 1942.[3] The IO route of venous access did not enjoy widespread use for several reasons. The equipment for performing an IO at the time was crude and did not improve until the 1970s. The saphenous venous cutdown technique was developed and gained popularity as an alternative method for obtaining vascular access when attempts at peripheral vein cannulation failed. Finally, the development of plastic, disposable, and single-use intravenous (IV) catheters revolutionized the technique of IV access and made this modality the preferred primary method.

IO access experienced a resurgence in popularity in the mid-1980s as its utility in the care of the critically ill patient was increasingly recognized.[4,5] IO access may be used to administer blood products, drugs, and fluids during cardiopulmonary resuscitation, a scenario where peripheral IV access may be particularly challenging. This procedure was more widely deployed in the care of pediatric patients due to the increased difficulty of obtaining vascular access in profoundly ill children. The IO placement time was slightly longer in pediatric cardiac arrest than peripheral venous access (3.0 versus 4.7 minutes), but the IO success rate was significantly higher (83% versus 17%).[6] IO infusion has been shown to be safe and effective in neonates and may be more rapidly obtainable than umbilical venous catheters.[7,8] The use of IO devices in pediatric patients has become widespread, associated with high success rates, and associated with a low incidence of complications.[7-9] IO access will cause metal artifact in most cases if obtaining a computed tomography (CT) scan of the area.[10]

IO access has been increasingly used in the resuscitation of adult patients when vascular access is unobtainable. It has become the first-line option for cardiac arrest patients.[11-14] IO access has become an invaluable procedure for prehospital providers and has repeatedly been shown to be safe and effective.[14-17] The newer commercially available powered devices make penetrating the adult bone cortex much less difficult. Several prehospital studies have demonstrated success rates for IO placement of ≥ 90%.[17,18] Emergency Medical Services systems have integrated IO devices into their vascular access protocols, and Emergency Departments routinely stock these devices.[17] A meta-analysis demonstrated overall success rates using a commercially available IO drill of about 90%, underscoring the fact that this modality can be used effectively and in all patients.[18]

ANATOMY AND PATHOPHYSIOLOGY

Long bones are composed of a dense outer cortex and inner soft, spongy (i.e., cancellous) bone (**Figure 70-1**). The nutrient artery supplies the bone with a rich vascular network. It pierces the cortex and divides into ascending and descending branches that further divide into arterioles and then capillaries. Venous drainage from the capillaries into the medullary venous sinusoids, located at the proximal and distal portions of the long bone, flows into the central venous channel located in the shaft of the long bone.[19]

The IO needle is inserted through the cortex and into the bone marrow (i.e., medullary) cavity of a long bone. Numerous anatomic sites can be used to access the medullary cavity. The most traditional site, which has been favored in pediatric patients, is the flat anteromedial surface of the proximal tibia (**Figure 70-2**). The distal tibia just above the medial malleolus is one of the preferred sites in adult patients (**Figure 70-3**). In the adult, it is easier to penetrate the cortex of the medial malleolus than the thicker cortex of the proximal tibia. Other sites for IO access include the flat anterior surface

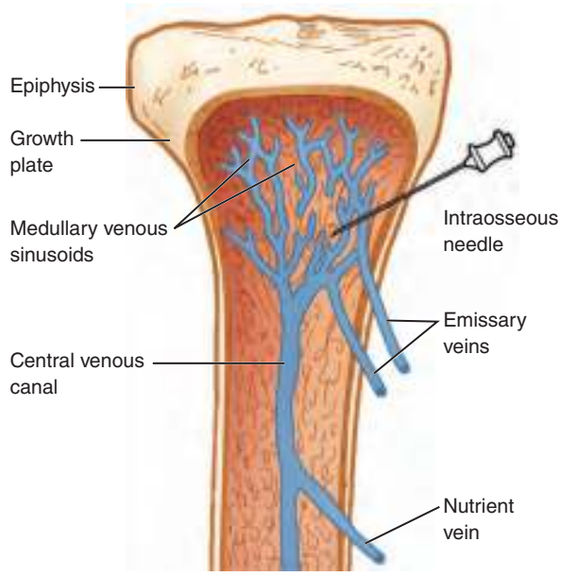


FIGURE 70-1. Venous anatomy of a long bone.

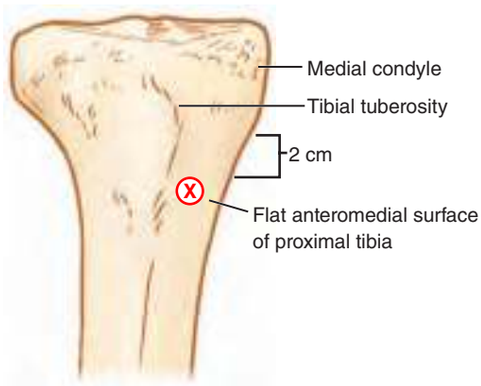


FIGURE 70-2. The proximal tibia is the traditional site used in pediatrics for IO access. The ⊗ represents the site of IO needle insertion.

FIGURE 70-3. The distal tibia is one of the preferred sites for IO access in adult patients. The ⊗ represents the site of IO needle insertion.

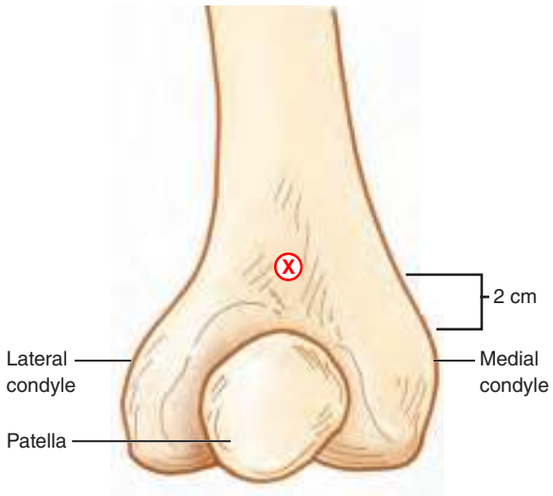


FIGURE 70-4. The distal femur is an alternative site for IO access. The ⊗ represents the site of IO needle insertion.

of the distal femur (**Figure 70-4**), the anterolateral surface of the proximal humerus (**Figure 70-5**), and the superior one-third of the sternum.[10,20-25]

Crystalloid infusion rates through an IO line vary depending on the bone accessed and the pressure of the fluid being administered. Under 300 mmHg of pressure, the sternum, proximal humerus, and proximal tibia have the following respective observed rates: 93, 70, and 30 mL/min.[26] The humerus provided greater flow rates than the tibia at all infusion pressures.[27] The flow rate of the proximal tibia was greater than the distal tibia at all infusion pressures.[28] Infusion rates approximate 10 to 20 mL/min using gravity.[26,29,30] Fluids and medications administered through an IO line are rapidly absorbed into the systemic circulation, and multiple studies have demonstrated similar effectiveness between IO and IV routes.[31,32] Succinylcholine has been effectively infused by the IO route for muscle paralysis prior to endotracheal intubation.[33] Sodium bicarbonate was shown to have superior buffering capacity when administered via an IO line than by peripheral IV.[34] It is unclear, however, whether

FIGURE 70-5. The proximal humerus is an alternative site for IO access in the adult. The ⊗ represents the site of IO needle insertion.

TABLE 70-1 Some of the Medications and Fluids Shown to Work Administered Through an IO Line*

Medications		
Adenosine	Dopamine	Morphine
Alteplase	Ephedrine	Naloxone
Amiodarone	Epinephrine	Norepinephrine
Anesthetics	Factor IX complex-human	Pancuronium
Antibiotics	Fentanyl	Phenobarbital
Antitoxins	HI-6 oxime	Phenytoin
Atracurium besylate	Heparin	Pralidoxime
Atropine	Human growth hormone	Propranolol
Calcium chloride	Hydroxocobalamin	Sodium bicarbonate
Calcium gluconate	Insulin	Succinylcholine
Contrast media	Isoproterenol	Tenecteplase
Dexamethasone	Ketamine	Thiopental
Diazepam	Levarterenol	Tranexamic acid
Diazoxide	Lidocaine	Vasopressin
Dicobalt edetate	Lipid emulsion	Vecuronium
Digoxin	Lorazepam	
Dobutamine	Mannitol	
Fluids		
Blood products	Lactated Ringer's solution	Sodium chloride solutions
Dextrose solutions	Packed red blood cells	
Iodinated contrast agents	Plasma	

*This does not mean that other medications will not work but were not tried.
Source: Compiled from references 10, 21, 30, 34, 35, 37, 39-56.

medications with extremely short half-lives (e.g., adenosine) are as effective when compared to administration via more central IV access.[35,36] Medications and fluids that may be administered by the IO route are listed in **Table 70-1**.[37-56] **The medication concentrations, dosages, and infusion rates through an IO line are the same as those through a peripheral IV line.**

INDICATIONS

The placement of an IO line is indicated when vascular access is rapidly required for the resuscitation of a patient and standard vascular access is unobtainable or delayed.[57,58] This includes neonates, infants, children, adolescents, and adults. It may be operationally useful to define a specific time frame or a specified number of peripheral IV attempts before proceeding to IO access. Situations that may require the placement of an IO line are cardiac arrest, extensive burns, sepsis, severe dehydration, shock, status epilepticus, trauma, or any condition that requires urgent administration of fluids, medications, or blood products.[38,59-64] Current guidelines recommend IO catheterization as the preferred second option if IV access is not quickly obtainable (e.g., 90 seconds or two attempts).[65-69]

IO access can provide blood samples for typing, crossmatching, and laboratory analysis.[70] Arterial blood gases, blood urea nitrogen (BUN), calcium, creatinine, electrolytes, and glucose from blood samples obtained through an IO needle are similar to samples taken via traditional routes.[70-73] Values are accurate for bedside point-of-care testing.[70,73] Laboratory values may be less accurate after 5 or more minutes of active resuscitation, if drugs have been infused through the IO site, or if fluids have been infused through the IO site.[71]

CONTRAINDICATIONS

Placement of an IO line is contraindicated in diseased (e.g., osteogenesis imperfecta) or osteoporotic bone. Avoid the placement of an IO line through areas of abscesses, burns, or cellulitis.[74] Fractures in the ipsilateral bone increase the risk of an extravasation-induced compartment syndrome and nonunion of the fractures.[75] Failed

placement of an IO line in the same bone is a relative contraindication. Do not use a bone that has had a previous orthopedic procedure, contains hardware, or may contain hardware (i.e., pins, plates, screws, and artificial joints). Do not insert an IO line if the patient is morbidly obese as the needle is too short to enter the medullary cavity or if the patient is so obese that the bony landmarks cannot be palpated. Other contraindications are an actual or possible compartment syndrome or vascular injury in an extremity.

There are contraindications specific to sternal IO access. Do not attempt sternal IO access in patients who weigh less than 50 kg, are small in stature, have a small sternum, have congenital sternal malformations, or have chest wall malformations. Do not consider the blunt trauma patient with a suspected sternal fracture or soft tissue injury over the sternum for sternal IO access. A history of a sternotomy or osteoporosis is a contraindication.

EQUIPMENT

- Sandbag or towels
- Povidone iodine or chlorhexidine solution
- Local anesthetic solution, 1% or 2% lidocaine
- Syringes, 5 to 60 mL
- Primed IV tubing with normal saline
- Tape
- Plastic protective cup
- Leg board for immobilization
- IO access device (**Table 70-2**)

IO needles are available in numerous sizes and styles (**Figure 70-6**). **Use only specifically designed IO needles for this procedure.** Spinal needles often bend and do not penetrate the cortex of the bone. Their long length causes increased resistance to fluid flow. Standard hypodermic needles (e.g., angiocatheters) often bend and do not penetrate the cortex of the bone. Spinal and standard hypodermic needles may break while being inserted and injure the Emergency Physician.

Available models of manually inserted IO devices (**Figure 70-6**) include the hand-driven threaded-needle SurFast and Cook Intraosseous needle (Cook Critical Care, Bloomington, IN), the Jamshidi bone marrow needle (Baxter Healthcare, Valencia, CA), and the MedSurg Industries Illinois sternal/iliac aspiration needle (MedSurg Industries, Rockville, MD). The typical unit consists of a detachable handle, an IO needle, an obturator, and a sleeve to prevent the needle from penetrating too deeply. The IO needles range in size from 12 to 20 gauge, with the majority used being 15 gauge. The IO needles available today are variations of the basic unit and include adjustable-length shafts to decrease the risk of penetrating too deeply, a variety of tips on the obturator (e.g., lancet, pencil point, and trocar), threaded versus nonthreaded shafts, needle sideports to increase flow rates, numerous lengths, and numerous handle types.

Powered and spring-loaded injection devices have evolved and are widely used. Three currently available devices are the EZ-IO Intraosseous Driver (Vidacare, San Antonio, TX), the Bone Injection Gun (BIG; Wais Medical, Kress USA Corporation), and the NIO (Vidacare, San Antonio, TX). The EZ-IO uses a battery-powered driver to insert the needle to the desired depth (**Figure 70-7**).[20] The BIG incorporates a loaded spring to inject the IO needle, and the desired depth of injection may be adjusted (**Figure 70-8**).[76] The BIG and EZ-IO are available in adult and pediatric needle sizes. The NIO is to be used in the humerus and proximal tibia of adults. The devices have numerous advantages when compared to the manual devices. They

TABLE 70-2 Some of the Devices Used for IO Access

Name	Manufacturer	Insertion location	Type of device(s)	Patient use
Bone Injection Gun	Waismed (www.waismed.com)	Humerus Proximal tibia	Spring driven	Pediatrics adults
Cook IO Needle Dieckman Modified IO Needle SurFast IO Needle	Cook Medical (www.cookmedical.com)	Distal femur Distal tibia Humerus Proximal tibia Sternum	Hand driven	Pediatrics adults
EZ-IO	Vidacare Corp. (www.vidacare.com)	Distal femur Distal tibia Humerus Proximal tibia	Battery-driven drill	Pediatrics adults
EZ-IO T.A.L.O.N.	Vidacare Corp. (www.vidacare.com)	Distal femur Distal tibia Humerus Proximal tibia Sternum	Hand driven	Pediatrics adults
FASTResponder IO infusion needles (Jamshidi, Illinois, etc.)	Pyng Medical (www.pyng.com) CareFusion (www.carefusion.com) Cardinal Health (www.cardinalhealth.com) Baxter Healthcare (www.baxter.com)	Sternum Distal femur Distal tibia Humerus Proximal tibia Sternum	Hand impact driven Hand driven	Adults Pediatrics adults
Monoject I-Type Sternal-Iliac Aspiration Needle	Covidien (www.medtronic.com)	Distal femur Distal tibia Humerus Proximal tibia Sternum	Hand driven	Pediatrics (except sternum) adults
NIO	Waismed (www.waismed.com)	Humerus Proximal tibia	Spring driven	Adults

FIGURE 70-6. IO infusion needles. ***A.*** The Cook Intraosseous infusion needle. ***B.*** The Illinois sternal/iliac aspiration needle. ***C.*** Another version of the Illinois sternal/iliac aspiration needle. ***D.*** The Jamshidi bone marrow needle.

are easier to use, are quicker at establishing IO access, require less or no force to insert the IO needle, and do not bend during insertion.

Sternal access devices include the Monoject I-Type Sternal-Iliac Aspiration Needle (Covidien, Mansfield, MA; **Figure 70-9*A***) and the First Access for Shock and Trauma (FASTResponder; Pyng Medical, Richmond, British Columbia, Canada; **Figure 70-9B**). The Monoject is a handheld IO device that is rarely used in the Emergency Department. The FASTResponder is a multiple-component kit to be used for sternal IO access in the adult patient. A special introducer limits the depth to which the needle can be inserted. This prevents injury to the underlying great vessels, heart, lung, and mediastinum. This system was evaluated in Special Forces military settings. It was found to be equally useful and safe for IO access.[76] Sternal IO access devices are used less often than the previously described powered devices due to possible interference with resuscitation attempts and their occasional incomplete removal, which necessitates surgical intervention.

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative if time permits. This procedure is often performed in emergencies. Informed consent can be waived when necessary. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

The primary site of choice for IO line placement is the proximal tibia. Alternate sites include the distal tibia, distal femur, proximal

A B

FIGURE 70-7. The EZ-IO. ***A.*** The driver, EZ-tubing, EZ-stabilizer, and three lengths of IO needles. ***B.*** The EZ-stabilizer close up. (Photos courtesy of Vidacare, San Antonio, TX.)

humerus, and the sternum. The proximal humerus allows more central access, while the proximal tibia is often accessible without disrupting airway management and cardiopulmonary resuscitation (CPR).[20]

Place the patient supine with the lower extremity supported behind the knee with a towel or sandbag. Identify by palpation the landmarks required to perform the procedure. Palpate the bony landmarks with the nondominant hand. The bony landmarks for the proximal tibia are the tibial tuberosity and the flat anteromedial surface of the proximal tibia. The site of IO needle placement is approximately 2 cm below the tuberosity on the flat anteromedial surface of the proximal tibia (**Figure 70-2**). The bony landmark for the distal tibia is the junction of the medial malleolus and the flat anteromedial surface of the distal tibia just posterior to the greater saphenous vein (**Figure 70-3**).[74] This is the preferred site in the adult patient. The bony landmarks for the distal femur approach are the medial and lateral condyles of the femur and the patella. Position the IO needle approximately 2 cm above these structures (**Figure 70-4**). This site is used less often in adults because of the abundance of muscle and soft tissue structures but may be an excellent option in infants due to the small size of the tibia. The bony landmark for the humerus is slightly anterior to the lateral midline of the arm at the greater tuberosity with care to avoid the bicipital groove (**Figure 70-5**). The arm should be adducted and internally rotated for optimal positioning.[20]

Prepare the patient. Clean any dirt and debris from the skin. Apply povidone iodine or chlorhexidine solution to the skin and allow it to dry if time permits. **This procedure is painful. The use**

A B

FIGURE 70-8. Spring-loaded IO devices. ***A.*** The BIG in adult and pediatric sizes. ***B.*** The NIO. (Photos courtesy of PerSys Medical, Houston, TX.)

A

B

FIGURE 70-9. Sternal IO devices. ***A.*** The Monoject I-Type Sternal-Iliac Aspiration Needle. (Photo courtesy of Covidien.) ***B.*** The Pyng FASTResponder. (Photo courtesy of Pyng Medical.)

of a local anesthetic solution in the conscious or semiconscious patient will reduce the pain associated with IO placement.[77] Infiltrate local anesthetic solution into the skin, subcutaneous tissues, and periosteum overlying the bone puncture site. Consider infusing 3 to 5 mL of 2% lidocaine (or 0.5 mg/kg in children, to a maximum of 3 to 5 mL) initially and at regular intervals to minimize patient discomfort associated with the IO infusion in the awake patient.[12]

TECHNIQUE

MANUALLY INSERTED IO DEVICES

Examine the IO needle to ensure that it appears to have been manufactured properly. Reidentify the landmarks with the nondominant hand. Stabilize the extremity with the nondominant hand (**Figure 70-10*A***). Grasp the IO needle firmly with the dominant hand. The handle of the IO needle should be firmly planted in the palm of the dominant hand. Insert the needle perpendicularly or slightly angulated at a 10° to 15° angle to the long axis of the bone (**Figure 70-10**). **The IO needle should always be directed away from the growth plate to avoid injuring it.** Direct the needle caudad in the proximal tibial approach and cephalad in the distal tibial and distal femoral approaches.

Advance the IO needle through the skin and subcutaneous tissue until the bone is contacted. Advance the IO needle through the bone. Use a twisting or rotary motion with the simultaneous application of downward pressure to cut through the cortex of the bone (**Figure 70-10*A***). **A significant reduction in the resistance to forward motion will be encountered when the cortex is penetrated and the needle enters the medullary canal.** This distance is rarely greater than 1 cm in most patients. An index finger may be placed 1 cm from the bevel of the IO needle prior to advancement. This will help prevent overpenetration into and through the cortex on the opposite side of the bone.[78] Alternatively, adjust the sleeve so that only 1 cm of the IO needle is exposed. **Stop advancing the IO needle when it enters the medullary canal.**

Remove the stylet when the medullary canal is entered (**Figure 70-10*B***). Attach a 5 or 10 mL syringe onto the hub of the IO needle (**Figure 70-10*C***). Aspirate blood from the medullary canal to confirm proper placement of the IO needle. Any samples obtained may be sent to the laboratory for subsequent analysis. The aspiration of more than 2 to 3 mL of blood may not be possible in cardiac arrest situations. Attach IV tubing to the hub of the IO needle and begin the infusion of fluids. Medications can be administered through the injection port of the IV tubing. Place a sterile dressing around the skin puncture site and apply pressure for 5 minutes.[79]

IO ACCESS USING THE EZ-IO

The EZ-IO is a reusable, nonsterile, battery-powered driver that uses sterile, disposable, single patient use IO needles. The IO needles are available in three lengths (i.e., 15, 25, and 45 mm) that are all 15 gauge (**Figure 70-7*A***). Choose the proper IO needle for the patient, their age, their body habitus, and the insertion site. Use a 45 mm needle in obese patients.[80] Assess the tissue depth by palpation. Choose an IO needle so that at least one black line on the IO needle shaft will be visible above the patient's skin when the tip is against the bone. The IO needle tip is designed to cut the bone and create a hole the same size as the needle.

Identify the IO insertion site and prepare for the procedure. Clean and prep the skin at the site and surrounding area. **The procedure requires aseptic technique.** Select the appropriate size IO needle. Open the cover of the case that holds the sterile IO needle. Insert the bit on the EZ-IO driver into the base of the IO needle (**Figure 70-11*A***). **Leave the cap on the IO needle until it is ready to be inserted.** Open the EZ-Connect tubing and flush it with a syringe containing 10 mL of sterile normal saline. Leave the syringe attached to the tubing after it is flushed.

Reidentify the landmark for the insertion site. Inject local anesthetic solution subcutaneously and down to the periosteum. Remove the cap from the IO needle. Insert the IO needle perpendicular or at a 90° angle to the skin (**Figure 70-11*B***). Advance the IO needle through the skin and subcutaneous tissues until the tip is against bone. **At least one black line on the needle shaft should be visible above the patient's skin.** Withdraw the needle and replace it with a longer IO needle if a black line is not visible on the needle shaft. Press the trigger to start the driver shaft and IO needle rotating. **Apply gentle and minimal downward pressure to advance the IO needle into the medullary canal. Stop advancing the driver when a loss of resistance is felt.**

Grasp and firmly hold the hub of the IO needle. Remove the driver (**Figure 70-11*C***). **Do not allow the IO needle to move while removing the driver.** Twist the stylet counterclockwise and remove

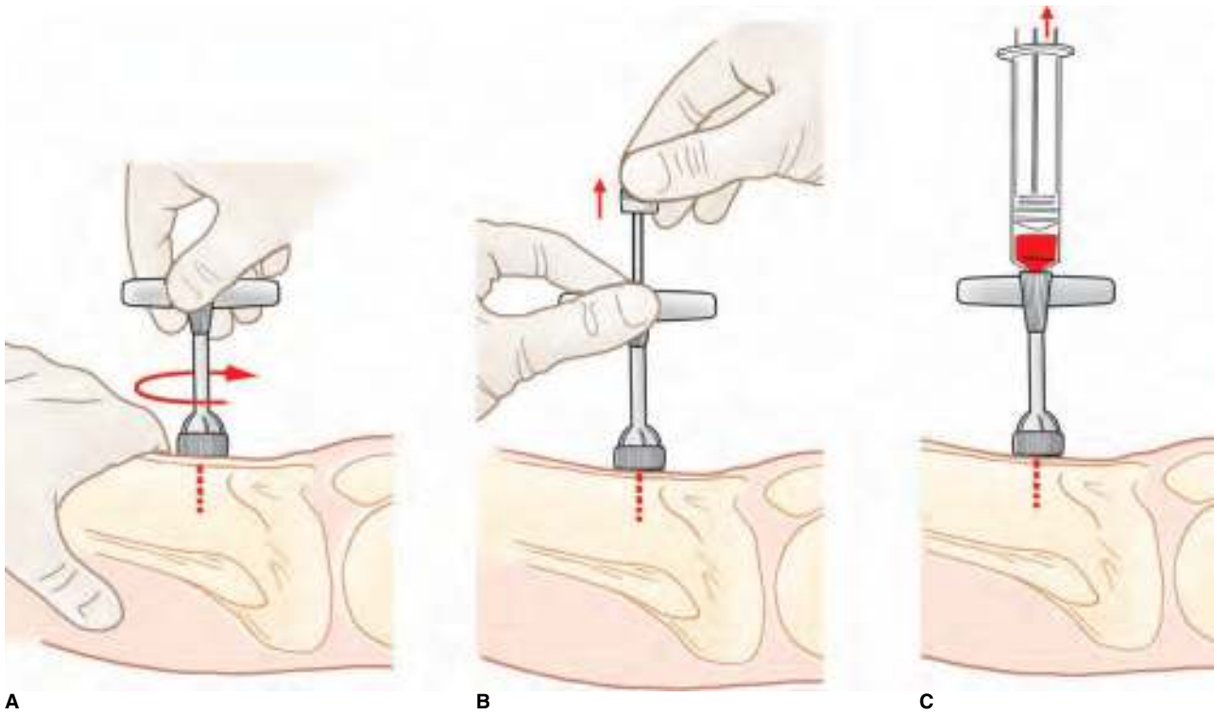


FIGURE 70-10. Placement of an IO line. ***A.*** The nondominant hand is used to support the extremity. The IO needle is inserted with a twisting motion to cut through the cortex of the bone. ***B.*** The handle and obturator are removed. ***C.*** A syringe is attached to the hub of the IO needle and bone marrow is aspirated.

it while securely holding the IO needle hub (**Figure 70-11*D***). Attach the EZ-IO stabilizer (**Figures 70-7*B* and 70-11*E***) followed by the primed EZ-Connect tubing. Aspirate bone marrow to confirm proper needle position. **Do not attach a syringe directly to the IO needle hub.** A syringe attached to the hub can result in a fracture of the hub. **Always attach the EZ-Connect tubing and then aspirate and inject through it and not directly through the IO needle hub.** Begin using the IO access.

IO ACCESS USING THE BIG

The BIG is an automatic, spring-loaded device that does not require a power source. It is available in two sizes: adult and pediatric. The insertion site is different for adults and children. Always start at the tibial tuberosity as the initial landmark. For adults, move 2 cm medially toward the inner leg and then 1 cm proximally toward the patient's head. For children, move 1 to 2 cm medially toward the inner leg and then 1 to 2 cm distally toward the patient's foot. Mark this final location as the insertion site. It may also be inserted in the proximal humerus in the adult patient.

Prepare for the procedure. **This procedure requires aseptic technique.** Clean and prep the skin at the insertion site and surrounding area. Open the package and remove the sterile BIG. Rotate the base of the unit to adjust the IO needle penetration depth to the pediatric patient's age: 0 to 3 years at 0.5 to 1.0 cm, 3 to 6 years at 1.0 to 1.5 cm, and 6 to 12 years at 1.5 cm (**Figure 70-12*A***). Use the adult BIG for patients over the age of 12 years. Reidentify the landmark for the insertion site. Inject local anesthetic solution subcutaneously and down to the periosteum.

Position the BIG at the insertion site with the base perpendicular or at 90° to the skin (**Figure 70-12*B***). Firmly grasp the BIG with the nondominant hand. Squeeze and pull out the red safety latch while firmly holding the BIG against the insertion site (**Figure 70-12*C***). **Do not discard the safety latch because it will be used later to secure the IO needle.** Grasp the upper part of the BIG with the dominant hand (**Figure 70-12*D***). Place two fingers under the wings and the palm on the top of the BIG (**Figure 70-12*D***). Continue to firmly hold the base against the patient's skin with the nondominant hand.

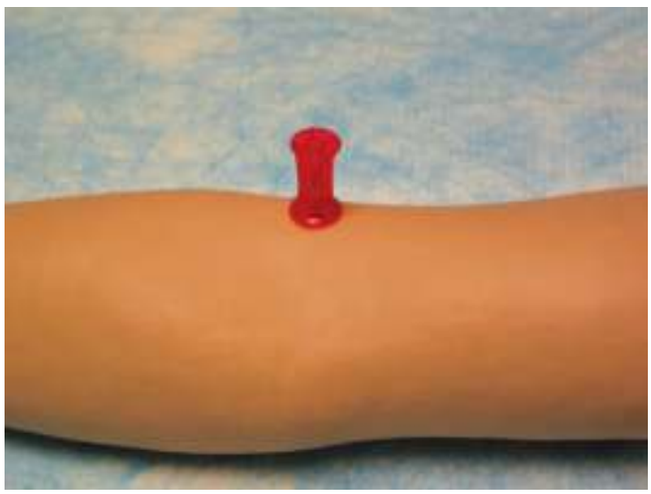
Insert the IO needle. Trigger the BIG by gently pressing down with the palm. The device will eject the spring-loaded IO needle into the bone. **The IO needle should be standing upright firmly in the bone and be stable.** If not, it has been placed into muscle or soft tissue. Gently lift and remove the BIG device without catching on the IO needle hub (**Figure 70-12*E***). Attach the safety latch onto the cannula between the patient's skin and the IO cannula hub (**Figure 70-12*F***). Secure the safety latch to the skin with tape (**Figure 70-12*F***). Gently remove the stylet-trocar, leaving the cannula in place (**Figure 70-12*G***). **If the stylet-trocar is "stuck" and not easily removable, do not pull or try to forcefully remove it.** The IO needle did not penetrate all the way into the medullary cavity and is embedded in the cortex. Insert the square end of the safety latch into the hub of the IO needle and gently twist to remove the IO needle. Repeat the procedure at another site. Gently attach a 5 or 10 mL syringe onto the IO cannula hub without advancing or withdrawing the cannula. Aspirate bone marrow to confirm proper placement (**Figure 70-12*H***). Remove the syringe while holding the hub of the cannula. Attach the IV tubing onto the hub of the cannula (**Figure 70-12*H***). Aspirate bone marrow to confirm proper placement (**Figure 70-12*H***). Flush the IV tubing and cannula with 5 to 10 mL of sterile saline. Begin using the IO access.

INSERTION OF THE NIO

The NIO is automatic and single use (**Figure 70-8*B***). It is spring loaded with a double safety mechanism to maximize caregiver and patient safety. The needle stabilizer fixates the needle firmly.

A

B



C

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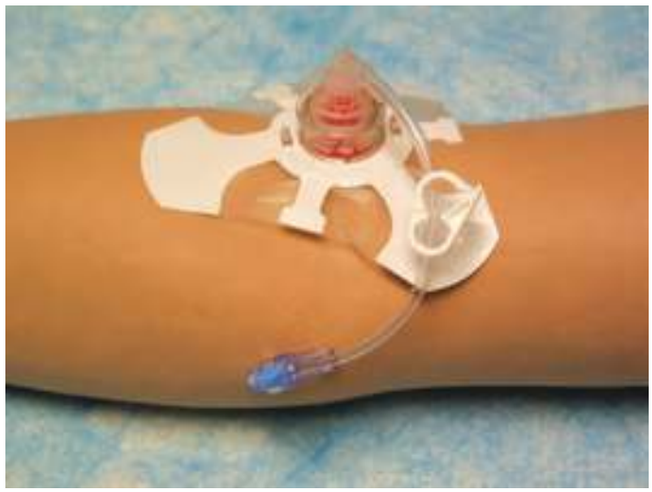

E

FIGURE 70-11. Using the EZ-IO. ***A.*** An IO needle has been applied onto the driver. ***B.*** Insert the IO needle at a 90° angle to the skin. ***C.*** The driver has been removed. ***D.*** The stylet is being removed. ***E.*** The EZ-stabilizer and tubing have been applied.

The device is for use in adult patients and not children. The recommended penetration depth is 2.5 cm. The primary site is the proximal tibia approximately 2 cm medially and 1 cm proximally to the tibial tuberosity. The humerus is an additional site. Adduct the patient's hand and locate the greater tubercle.

Open the pack and take out the NIO (**Figure 70-13*A***). Make sure that the NIO is free of all packaging parts. Choose the site for injection. Clean and prepare the skin. **This procedure requires aseptic technique.** Place the nondominant hand on the textured dots on the lower part of the NIO (**Figure 70-13*B***). Position the NIO at 90° to the skin at the injection site (**Figure 70-13*B***). Keep the nondominant hand at this position. Unlock the NIO. Rotate the cap 90° in either direction (**Figure 70-13*C***). Place the palm of the dominant hand over the cap. Place the fingers of the dominant hand under the wings of the NIO. Press the device against the patient's skin and maintain downward pressure. Pull the trigger wings upward while pressing down on the device to activate the device (**Figure 70-13*D***). Firmly hold the base of the needle stabilizer against the insertion site.

A

B

C

D

FIGURE 70-12. Using the BIG. ***A.*** Adjust the penetration depth. ***B.*** Apply the BIG at a 90° angle to the skin. ***C.*** Remove the safety latch. ***D.*** Grasping the BIG in preparation of triggering the unit. ***E.*** Remove the BIG. ***F.*** Attach and secure the safety latch. ***G.*** Pull the stylet-trocar from the IO cannula. ***H.*** Attach IV tubing onto the hub and aspirate bone marrow. (Photos courtesy of Vidacare, San Antonio, TX.)

E

F

G

H

FIGURE 70-12 (*continued*)

Gently pull the NIO up in a rotary motion (**Figure 70-13*E***). Hold the needle stabilizer and cannula in place. Remove the stylet by pulling it up in a twisting motion. The keyhole notch on the distal end of the NIO can be used to assist in removing the stylet (**Figure 70-13*F***). Attach a 10 mL syringe to the hub of the IO needle. Aspirate bone marrow to confirm the needle is within the medullary canal. Hold the hub of the IO needle and remove the syringe. Attach the IV tubing and begin the infusion (**Figure 70-13*G***).

The company recommends the NIO Fixation sticker to affix the NIO stabilizer (**Figure 70-13*G***). Cover the insertion site with a sterile occlusive dressing. Immobilize the patient's arm if the humeral head was used to prevent accidental removal of the device.

INSERTION OF THE FASTResponder

The FASTResponder is single-use device that is easy to insert (**Figure 70-14*A***). **The device is for use in patients age 12 and older and not children.** The primary and only site is the sternum. The depth control prevents overpenetration through the back of the sternum and into the mediastinum. The FASTResponder

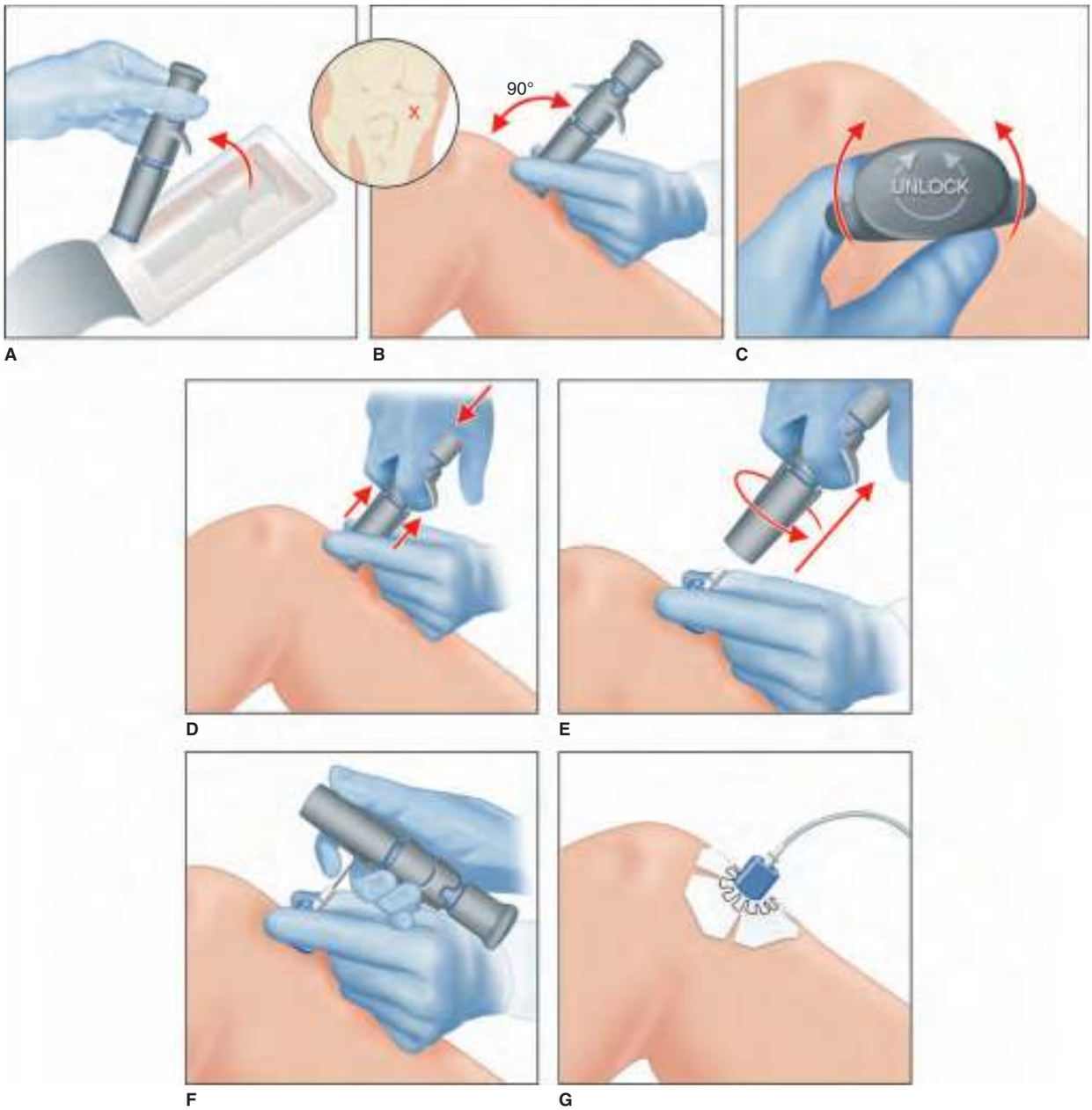

FIGURE 70-13. Using the NIO. ***A.*** The NIO is removed from the package. ***B.*** The NIO is grasped and held over the insertion site. ***C.*** The cap is turned 90° in either direction. ***D.*** The unit is compressed. ***E.*** The NIO is removed. ***F.*** The stylet is removed. ***G.*** The IV tubing is attached.

can be inserted and used with CPR due to its location on the manubrium of the sternum. The infusion tubing is mounted on the stylet inside the introducer. It is designed to flex with skin movement. The needles on the introducer do not enter the sternum.

Clean and prepare the skin over the upper sternum. **This procedure requires aseptic technique.** Stand above the patient's head. Open the pack and take out the FASTResponder (**Figure 70-14*B***). Remove the locking pin and adhesive liner covering the target foot of the FASTResponder (**Figure 70-14*C***). Align the notch on the target foot with the patient's sternal notch with the device at a 90° angle to the manubrium (**Figure 70-14*D***). Briefly pause CPR if this is occurring. Push down forcefully on the device to insert the infusion tube (**Figure 70-14*E***). Downward pressure pushes the infusion tube through the sternal cortex and into the marrow cavity. Firmly hold the target foot against the sternum. Pull the device upward leaving only the target foot and infusion tube (**Figure 70-14*F***). Remove the anti-buckle support from the infusion tube. Connect the IV tubing to the Luer lock of the infusion tubing (**Figure 70-14*G***). Attach the strain-relief hook of the infusion tubing to the target foot (**Figure 70-14*H***).

The following are optional steps. Aspirate to confirm the proper location. Remove the protected cover and attach the protective dome.

ASSESSMENT

Assess whether the IO needle is correctly positioned within the medullary cavity. First aspirate blood from the marrow cavity. This may not be possible because of poor circulation in patients with

a cardiac arrest. A second sign of correct placement is to assess whether the IO needle will stand erect without support. Finally, flush the IO line. The ability of the fluid to flow without inducing soft tissue swelling can also be used to confirm proper placement. Ultrasonic visualization of flow within the medullary cavity using color flow Doppler can confirm correct placement, although physical examination as described above may be the most reliable method.[81-83] Alternatively, a C-arm fluoroscopic imaging device may be used at the bedside to confirm accurate placement of the IO needle.[84] **A reassessment must be performed at regular intervals to ensure that extravasation does not occur.** Circumferential pressures of the involved extremity may be used for serial examination.[85]

AFTERCARE

Secure the IO needle. Secure the manually inserted IO needle by taping it in place. It may be easier to apply 4×4 gauze squares on two sides of the IO needle to support it and then tape the needle and gauze in place. Secure the EZ-IO and NIO needle in the same manner or use the EZ-stabilizer. Tape the safety latch of the BIG to the

A

B

C

D

FIGURE 70-14. Using the Pyng FASTResponder. ***A.*** The complete kit. ***B.*** The device in the packaging. ***C.*** Removal of the locking pin and attached target foot liner. ***D.*** Aligning the target foot to the sternal notch. ***E.*** Push down to insert the infusion tube. ***F.*** Removal of the device leaves the target foot and infusion tube. ***G.*** The IV tubing is attached to the infusion tubing. ***H.*** The strain-relief hook is attached to the infusion tubing. (Photo courtesy of Pyng Medical.)

E

F

G

H

FIGURE 70-14 (*continued*)

skin to secure it. Tape the IV tubing securely at several points. This will prevent traction on the tubing from pulling the IO needle out of the bone. Tape a plastic cup over the IO needle to avoid inadvertent disruption during patient resuscitation or positioning. Immobilize the extremity, if necessary, to help secure the IO line.

Remove the IO line once the resuscitation is complete and another form of secure vascular access has been obtained. Manually inserted IO devices can be removed by gently twisting them and pulling them straight out. **Do not rock the device.** To remove the EZ-IO, attach a 5 or 10 mL Luer lock syringe onto the IO needle hub. Rotate the syringe clockwise and pull it straight out. It may take several rotations of the syringe before the IO needle can be removed. **Do not rock the IO needle or syringe.** To remove the BIG cannula, insert the square end of the safety latch into the hub of the cannula and gently twist and pull to remove it. Remove the cannula and needle stabilizer of the NIO by twisting and pulling vertically. Remove the protective dome, if used, of the FASTResponder. Grasp the infusion tubing as close to the patient as possible with fingers or a clamp. **Pull the infusion tubing in a continuous motion until removed. Do not start and stop the pulling.** The infusion tube will slightly stretch when it is pulled. By twisting and pulling vertically, peel off the target foot. Bleeding at the insertion site can be controlled with a sterile pressure dressing followed by cleansing the skin and a simple bandage. Cover the insertion site with a sterile occlusive wound dressing.

What if advanced imaging (i.e., CT scans and magnetic resonance imaging [MRI] scans) is needed? Metal needles are problematic. They will produce an artifact if scanning the area of the IO. The metal IO is a contraindication to the use of MRI. Obtain a CT scan or delay the MRI scan until alternate access is obtained.

COMPLICATIONS

The most common complications of IO infusions are subcutaneous and subperiosteal extravasation of fluid.[5,86,87] Under ideal circumstances, the type of IO needle used should not affect extravasation rates.[88,89] Extravasation is usually due to under- or overpenetration of the cortex. There have been cases of tibial fracture due to overpenetration of the cortex, although this is extremely rare.[9,90-92] A compartment syndrome may occur when there is extravasation or when IO lines are placed in fractured bones.[93,94] Necrosis and sloughing of the skin at the insertion site of the IO needle are due to extravasation of fluid or medication.[88,94] Local hematomas can occur from penetrating muscle, superficial vessels, and the bones. Friction burns and abrasions can be avoided with proper training.[95]

Localized infections may occur after IO needle placement. Cellulitis or the formation of subcutaneous abscesses occurs in 0.7% of patients.[74] Osteomyelitis has been reported in less than 1% of patients

with IO needles.[96] Risk of osteomyelitis increases with prolonged IO use, administration of hypertonic fluids, and bacteremia.[96]

Injury to the growth plate is a commonly mentioned complication. The literature does not support this.[97,98] The rate of the IO infusion and the osmolarity of the infused fluid did not adversely affect the bone marrow or bone development in animal studies.[99] Air or fat embolism has been mentioned as a possible complication, although there exists no evidence that this is clinically significant.[100-102] Consider a fat embolism if the patient becomes hypoxic or experiences respiratory difficulty shortly after beginning an IO fluid infusion or bolus.

The flow rate of fluid through an IO line may be slower than that through a peripheral IV line depending on the site, gauge of the IO needle, and gauge of the peripheral IV. This may be due to a small marrow cavity, a fibrous marrow cavity, and/or the replacement of red marrow with yellow marrow. Fluid flow rates can be significantly increased by applying a pressure bag onto the IV fluid bag or using a level 1 infuser.[103] The placement of a second IO line may be required to further increase the amount of fluid that can be infused.

The complications specific to the EZ-IO include hub fracture after insertion and separation of the needle from the hub upon removal. Too much downward pressure upon IO needle insertion can result in it penetrating the back of the bone. The other general complications are described above.

The complications associated with the BIG or NIO are primarily from incorrect use. **Never remove the safety latch until the BIG is against the patient's skin over the insertion site.** Removal of the safety latch activates the device. The IO needle can be forcefully ejected and injure the patient or a health care provider if the BIG or NIO is squeezed after the safety latch is removed. **Never place a finger over the distal end. Always grasp the devices properly. Grasping it upside down can result in the IO needle penetrating the Emergency Physician's hand.** Incorrectly adjusting the penetration depth can result in the IO needle penetrating the back of the bone. The other general complications are described above.

There are complications associated with the FASTResponder. These are usually device related and minor. Flush the line if the infusion is slow or not flowing. If this does not resolve the flow problem, discontinue use of the sternal IO and try an alternative method. Extravasation at the insertion site should be minimal if at all. Significant extravasation requires the discontinuation of the sternal IO and an alternative method. Do not use the sternal IO if the infusion tubing is not secure. The company claims a 1 in 1,000,000 rate of sternal overpenetration.

SUMMARY

The placement of an IO line is an excellent option in the resuscitation of a patient when traditional vascular access techniques have failed. The procedure is technically straightforward and has been demonstrated to be successful in the hands of trained health care workers, including prehospital personnel. Complications have been related mostly to technical mistakes and can be avoided if care is taken to correctly identify landmarks, avoid the growth plate, regulate the depth of IO needle placement, and ensure early removal of the IO line.

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71 Umbilical Vessel Catheterization

Jeanne Noble

INTRODUCTION

Umbilical vessel catheterization was first described by Diamond in 1947 for an exchange transfusion in a neonate.[1] Umbilical vessel catheterization serves many important functions in the ill neonate and is a reliable method of obtaining rapid vascular access.[2] Umbilical vessel catheters may be used for fluid resuscitation, blood transfusion, medication administration, frequent blood sampling, and cardiovascular monitoring.[2-6] **The use of these catheters carries risk of morbidity and mortality.** Either the umbilical artery or vein may be used for vascular access. The umbilical arteries begin to constrict immediately after birth and can typically be cannulated during the first few days of life. The umbilical vein can be accessed up to 7 to 10 days after birth (i.e., when the umbilical stump dries up).[7]

Umbilical artery catheterization is more desirable than umbilical vein catheterization. The umbilical artery allows frequent arterial blood gas sampling and continuous blood pressure monitoring and administration of substances (e.g., blood products, fluids, and medications). **Umbilical artery catheterization is more difficult and time consuming to perform. Umbilical vein catheterization is the preferred procedure for the infant in shock and in need of rapid resuscitation.** Arterial access can be obtained later in the controlled environment of the Neonatal Intensive Care Unit (NICU). **Umbilical vessel catheterization can lead to serious complications and should be reserved for the patient in whom intraosseous or peripheral venous access cannot be rapidly secured.**[6,8]

ANATOMY AND PATHOPHYSIOLOGY

The fetal circulatory system differs from that of the neonate (**Figure 71-1**).[7] Oxygenated blood from the placenta travels via the umbilical vein, through the ductus venosus in the liver, to the inferior vena cava (IVC), and into the right atrium. Oxygenated blood from the IVC preferentially enters the left atrium through the foramen ovale. It then enters the left ventricle followed by the aorta. This oxygen-rich blood supplies the brain prior to mixing with the oxygen-poor blood coming through the

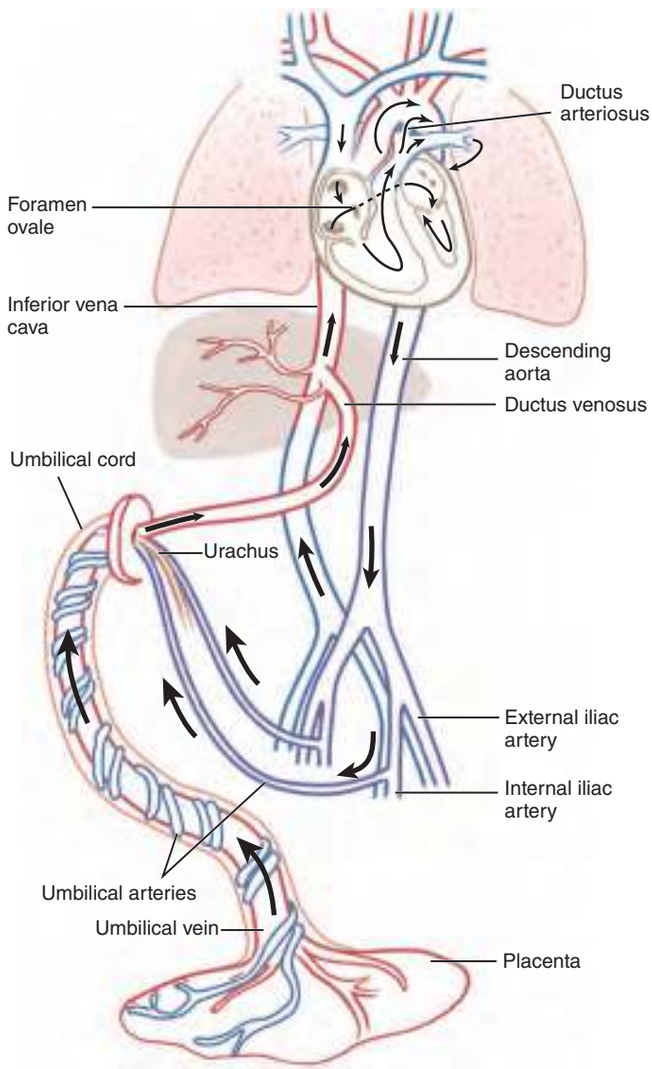


FIGURE 71-1. The fetal circulation.

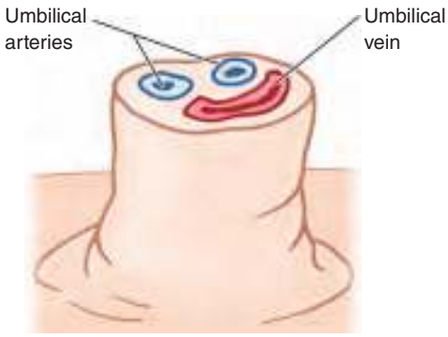


FIGURE 71-2. Anatomy of the umbilical cord.

ductus arteriosus. Deoxygenated blood from the superior vena cava (SVC) enters the right ventricle and is pumped to the pulmonary artery. It then passes through the ductus arteriosus to meet the oxygenated blood in the aorta.

Pulmonary vascular resistance decreases dramatically as the infant takes its first breath. The systemic vascular resistance increases when the umbilical cord is clamped. These changes in resistance cause the foramen ovale to close. The ductus arteriosus closes within 24 to 48 hours due to the release of prostaglandins and increased blood oxygen tension. The ductus venosus closes when the umbilical cord is clamped.

The umbilical vein and arteries can be easily differentiated by examination of a cross section of the umbilical cord (**Figure 71-2**). The umbilical vein is a single thin-walled vessel with a large lumen. It is usually flattened in one direction. There are two thick-walled umbilical arteries that are significantly smaller in diameter than the umbilical vein. Occasionally, only a single umbilical artery is present. Some describe the umbilical cord as a "happy face" with the two arteries as the eyes and the vein as the mouth.

INDICATIONS

Umbilical artery catheterization is indicated when frequent arterial blood gas determinations and continuous monitoring of blood pressure are required in the first few days of life.[5] An umbilical artery catheter (UAC) can be used for delivering blood, fluids, total parenteral nutrition, medications, and exchange transfusions.[3,4] It reduces the need for multiple venipunctures and heel prick capillary blood sampling in the critically ill neonate.[9] Neonates under 24 hours of age can usually be catheterized without much difficulty. A skilled Emergency Physician can sometimes perform this in neonates up to 7 days of age.[3]

Umbilical vein catheterization is easier to perform than umbilical artery catheterization. It is the preferred procedure for the neonate in shock needing rapid administration of intravenous fluids, blood, or medications.[1] The umbilical vein can be used to monitor central venous pressure and to perform invasive cardiac procedures.[10] The vein may be cannulated in neonates up to 10 days after birth.

CONTRAINDICATIONS

Umbilical vessel catheterization in the Emergency Department should be performed on severely ill neonates in whom intraosseous and peripheral vascular access is unlikely to be successful. The contraindications are similar for umbilical vein and umbilical artery catheterization. These include the presence of a gastroschisis, omphalocele, omphalitis, and peritonitis.[3,11] **Never insert an umbilical catheter if there are any signs of infection on or around the remnant of the umbilical cord.** Umbilical vessel catheterization is contraindicated if a neonate is older than 1 to 2 weeks of age. An alternate route of vascular access is required if the possibility of an abdominal abnormality exists, as manifested by a distended abdomen or visible defects.[3] Relative contraindications for placement of a UAC include decreased perfusion to the lower extremities, decreased perfusion to the buttocks, or suspicion of necrotizing enterocolitis.

EQUIPMENT

- Umbilical vein catheter, 5.0 French or 3.5 French
- Umbilical artery catheter, 5.0 French or 3.5 French
- Umbilical tape
- Povidone iodine or chlorhexidine solution
- Sterile gauze
- Adhesive tape
- Silk suture, 3–0 or 4–0
- Suture kit
- Smooth-curved iris forceps
- 2 small, smooth-curved hemostats
- #10 or #11 scalpel
- 3-way stopcock
- 10 mL syringe filled with normal saline
- Sterile drape
- Sterile gown and gloves
- Cap and face mask
- Radiant infant warmer
- Cardiac monitor
- Pulse oximeter

UACs and umbilical vein catheters (UVCs) are available in a variety of sizes. UVCs are available as single-lumen or double-lumen catheters. Catheters with side holes are only used for exchange transfusions. Use a 5.0 French umbilical catheter in neonates weighing more than 1500 gm and a 3.5 French catheter in neonates weighing less than 1500 gm.[7]

Umbilical vessel catheterization kits are commercially available from several manufacturers (**Figure 71-3**). The kits are sterile, disposable, and intended for single patient use. They contain all the equipment required for catheter insertion but do not always include the actual catheter. It is important to be familiar with the hospital's kit prior to use.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the parent and/or the patient's caregiver. Obtain an informed consent for the procedure and place this in the medical record. Give parents the option to leave the room or look away as the procedure can be disconcerting to some.

Umbilical vessel catheterization should be performed in the sick neonate when intraosseous and peripheral venous access is unlikely to be rapidly secured. All equipment should be readily available on a preprepared sterile tray. Place the tray with the instruments on a Mayo stand next to the neonate's bed. Place the neonate under a radiant warmer with a light source. Place the neonate on a cardiac monitor, continuous pulse oximeter, and supplemental oxygenation if needed. Place the neonate supine with the lower extremities in the frog-leg position (**Figure 71-4**). Place their arms and legs in soft restraints to prevent contamination of the sterile field (**Figure 71-4**).

A

B

C

FIGURE 71-3. Some of the commercially available kits for umbilical vessel catheterization.

This procedure requires strict sterile technique. The Emergency Physician performing the procedure should wear a cap, mask, sterile gloves, and sterile gown. Scrub the umbilical cord, umbilical cord clamp, and the neonate's abdomen with povidone iodine or chlorhexidine solution and allow it to dry. Place sterile drapes to form a sterile field around the umbilical area. **Ensure the neonate's head is exposed for observation.**

TECHNIQUES

PREPARATION OF THE UMBILICAL CORD

Sterile technique must be maintained throughout the entire procedure. Loosely tie a piece of umbilical tape around the base of the umbilical cord (**Figure 71-5**). A thin gauze packing strip can be used if umbilical tape is unavailable. Place purse-string sutures

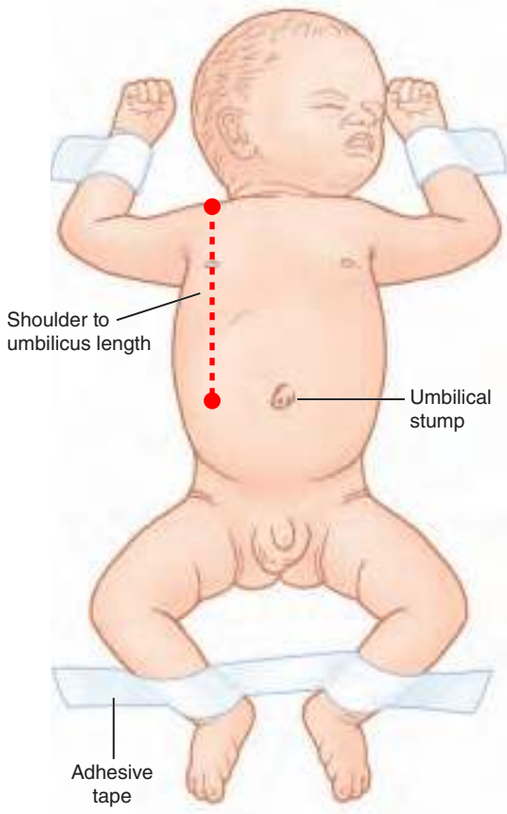


FIGURE 71-4. Positioning of the neonate.

with 3–0 or 4–0 silk in small bites around the base of the umbilical cord and just above the umbilical tape (**Figure 71-5**). **Avoid piercing the umbilical vessels or the skin of the abdominal wall.** Leave the suture untied and the ends uncut. This will be used later to secure the catheter. Gently grasp the umbilical cord. Use a #10 or #11 scalpel blade to cut the umbilical cord to expose a smooth, clean surface of the umbilical cord (**Figure 71-5**). **Do not cut the cord too close to the base in case it needs to be recut for a repeat attempt.** Tighten the umbilical tape if blood is oozing from the cut umbilical cord. **Blot the blood with gauze squares. Do not rub the gauze against the umbilical cord to prevent damaging the fragile umbilical cord.** Grasp the umbilical cord with forceps or fingers

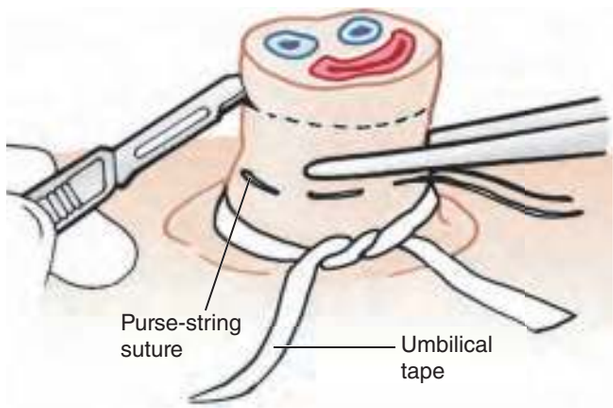


FIGURE 71-5. Preparing the umbilical cord stump. Umbilical tape has been placed at the base and tied loosely. A purse-string suture has been placed through the stump and just above the umbilical tape. The distal end of the umbilical cord is removed along the dotted line.

and identify the three vessels (i.e., two arteries and one vein). The arteries are smaller and round and have thick muscular walls. The vein is larger, has a thinner wall, and is less round than the arteries. The umbilical artery or vein may now be catheterized as described in the following sections.

UMBILICAL ARTERY CATHETERIZATION

Determine the desired position of the catheter tip (**Figure 71-6**). UACs have been placed in either a "high" or "low" position. The high position is associated with a lower occurrence of vascular complications, removal, and adverse sequelae.[5,6,12] It is the technique described here. **The catheter tip is placed between the sixth and tenth thoracic vertebrae.** This level corresponds to just above the diaphragm, between the ductus arteriosus and the origins of the mesenteric arteries.[3,13-15]

A variety of methods have been used to determine the insertional length of UACs.[16-19] Some of these include electrocardiographic changes during placement, echocardiography-guided insertion, and ultrasound-guided insertion.[20-24] These methods are hard to perform in clinical practice. Other methods for determining the appropriate depth of catheter insertion are the shoulder-to-umbilicus length and the neonate's birth weight referring to standardized graphs.[19]

Many centers use a calculation derived by Dunn on postmortem infants. The length of insertion to the high position is measured from the external shoulder to the umbilicus (**Figure 71-4**).[25] Measure the shoulder to umbilicus length by measuring the distance between the tip of the shoulder to the level perpendicular to the umbilicus (**Figure 71-4**). The catheter length to be inserted is determined using a nomogram using the shoulder-umbilical length plus the length of the umbilical stump (**Figure 71-7**). Three curves are available corresponding to the desired position of the UAC tip. The Dunn method always requires use of a standardized graph and may be impractical in emergency situations.

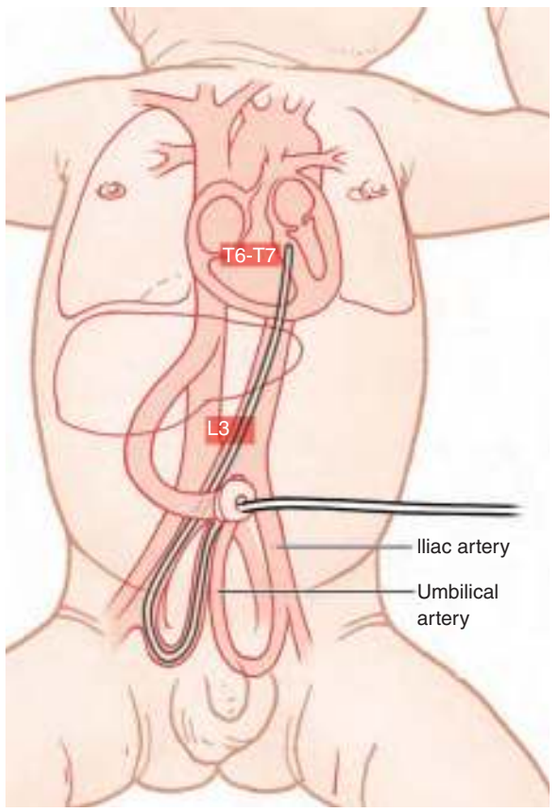


FIGURE 71-6. Positioning of the tip of the umbilical artery catheter. The low position is just above the aortic bifurcation between L3 and L5. The high position is above the diaphragm, between the ductus arteriosus and the mesenteric arteries and between T6 and T10. The shaded box represents the vertebral bodies in the midline.

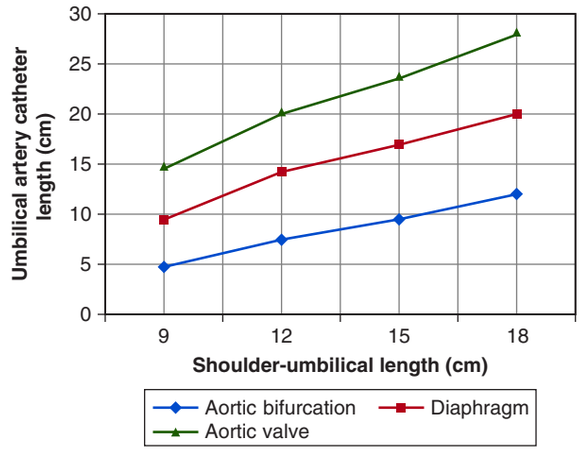


FIGURE 71-7. Graph to determine the correct length of catheter to insert into the umbilical artery. Using the shoulder-umbilical length and the desired position of the catheter tip, determine the length of catheter on the y-axis to be inserted into the artery. Add the length of the umbilical stump to the umbilical artery catheter length found on the y-axis of the graph to determine the corrected length of catheter to be inserted. (Adapted and modified from reference 13.)

The Wright equation has been shown to provide more accurate estimations of catheter insertion length in both full-term and low-birth-weight neonates.[26,27] It does not require the use of a graph. This formula is UAC insertion length (cm) = 4 × birth weight (kg) + 7 + the length of the umbilical stump (cm).

Fill the catheter with normal saline. Attach a three-way stopcock in the closed position to the hub of the catheter. Identify the arteries and choose one for catheterization. Grasp the distal portion of the umbilical cord with the two smooth-curved hemostats to stabilize the end of the umbilical cord (**Figure 71-8**). Instruct an assistant to hold the hemostats. **Do not apply traction to the hemostats to**

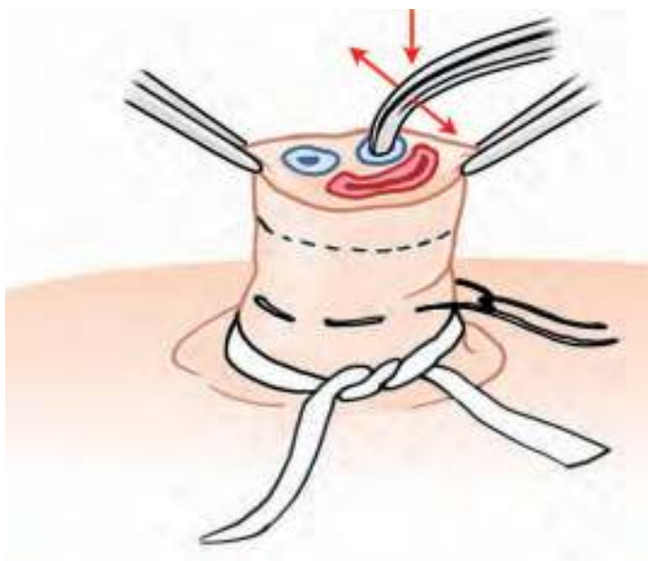


FIGURE 71-8. Preparation for catheter insertion. The upper edges of the umbilical cord stump are stabilized with two smooth-jaw curved hemostats. A smooth-jaw curved iris forceps is placed into one umbilical artery and allowed to open and dilate the artery.

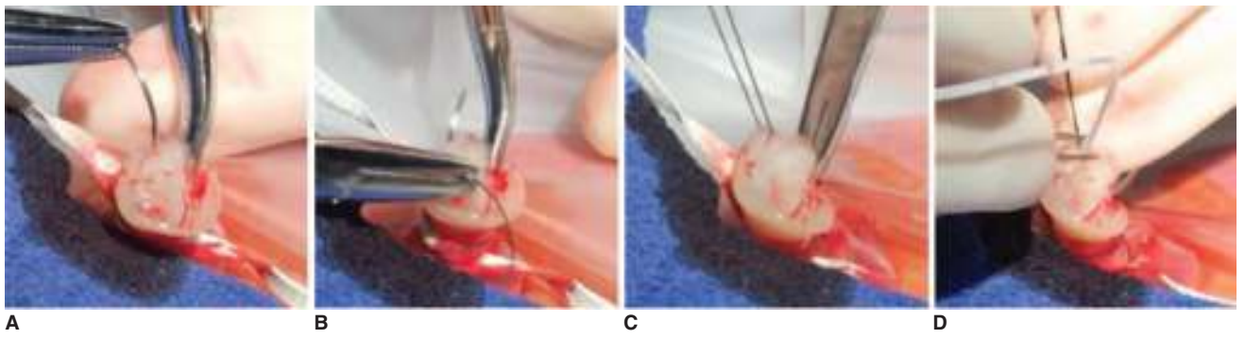


FIGURE 71-9. A new and novel technique for UAC placement. ***A.*** Insert suture needle directly into the umbilical artery lumen. ***B.*** Drive the needle through arterial sidewall and Wharton's jelly. ***C.*** Apply gentle upward traction to the suture. ***D.*** Insertion of umbilical catheter.

prevent injury to the umbilical cord. Insert the tips of the smooth-curved iris forceps approximately 0.5 cm into the lumen of the artery and gently allow the jaws of the forceps to open (**Figure 71-8**). This will dilate the arterial lumen. Repeatedly insert and open the tips of the curved iris forceps within the arterial lumen to a depth of 0.5 to 1.0 cm.[3] **Inadequate dilation is the most common cause of failure to catheterize the umbilical artery.**

A novel alternative technique has been developed.[23] Access the umbilical artery by inserting a 4–0 or 5–0 silk suture needle directly into the arterial lumen and out the sidewall of the umbilical stump approximately 3 to 4 mm below the opening of the arterial lumen (**Figures 71-9*A* and 71-9*B***). Apply gentle upward traction to the suture (**Figure 71-9*C***). This opens and stabilizes the lumen of the artery. Dilation of the lumen with iris forceps may be performed, if needed, as described above.

Grasp the umbilical catheter approximately 1.0 cm from the tip. Gently insert the tip of the catheter into the artery (**Figures 71-9*D* and 71-10**). Apply gentle pressure for about 30 seconds. The muscles of the artery may spasm and prevent the catheter from advancing. Remove the catheter if the spasm persists. Place 0.1 mL of a 2% lidocaine solution into the catheter tip.[3] Reinsert the catheter and flush the lidocaine into the artery at the level of the spasm. A gentle twisting motion may allow the catheter to advance if it still will not advance. Catheterize the other umbilical artery if this fails. Advance the catheter to the predetermined length. Increased resistance will be felt as the catheter goes through the base of the umbilical cord and navigates the curve of the umbilical artery into the femoral artery. Tighten the umbilical tape to temporarily secure the catheter.

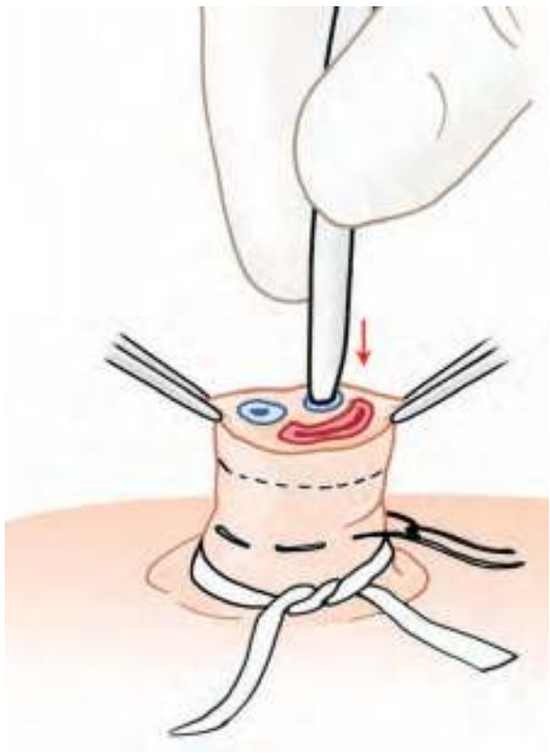


FIGURE 71-10. The catheter is inserted into the umbilical artery and advanced to the desired depth.

Obtain fluoroscopic, plain radiographic, or ultrasonic confirmation of placement prior to using the catheter. The catheter may pass into the aorta but then curve distally into the iliac or femoral artery. This can result in blanching, cyanosis, or necrosis of the lower extremity and/or the buttocks.[27,28] This occurs more frequently when a 3.5 French catheter is inserted in a large neonate. The larger 5 French catheter will usually advance cephalad in the aorta. The distally advanced catheter can be partially withdrawn, twisted 90°, and readvanced using real-time fluoroscopy or ultrasound. **Catheters provoking lower extremity cyanosis, blanching, or decreased distal perfusion must be removed even if placed correctly. Never advance a catheter that has been previously placed as it can introduce an infection.**

UMBILICAL VEIN CATHETERIZATION

Catheterizing the umbilical vein is much easier than the umbilical artery. The lumen is larger and easier to negotiate. This quality makes the umbilical vein more desirable for vascular access in emergency situations.[8,29]

It is acceptable to simply insert the UVC approximately 4 cm until blood is aspirated then advance the catheter an additional 1 to 2 cm during a resuscitation of the critically ill neonate. The desired position of the UVC tip can be more carefully determined in less emergent situations. It should be ideally located just beyond the ductus venosus at the junction of the IVC and the right atrium (**Figure 71-11**). Measure the shoulder-umbilical length (**Figure 71-4**). Determine the umbilical catheter length to be inserted into the vein using the shoulder-umbilical length and the desired position of the catheter tip (**Figure 71-12**). Add the length of the umbilical stump to the umbilical vein catheter length found on the *y*-axis of the graph to determine the corrected length of catheter to be inserted. Another method to calculate the depth of insertion is length (cm) = {[(3 × body weight [kg]) + 9] ÷ 2} + 1 cm.

Umbilical vein catheterization is like that of the artery with a few exceptions. Any visible clot should be removed from the vein prior to inserting a UVC. No dilation of the vein is required. **An emergency UVC is rapidly inserted 1 cm beyond where blood is first**

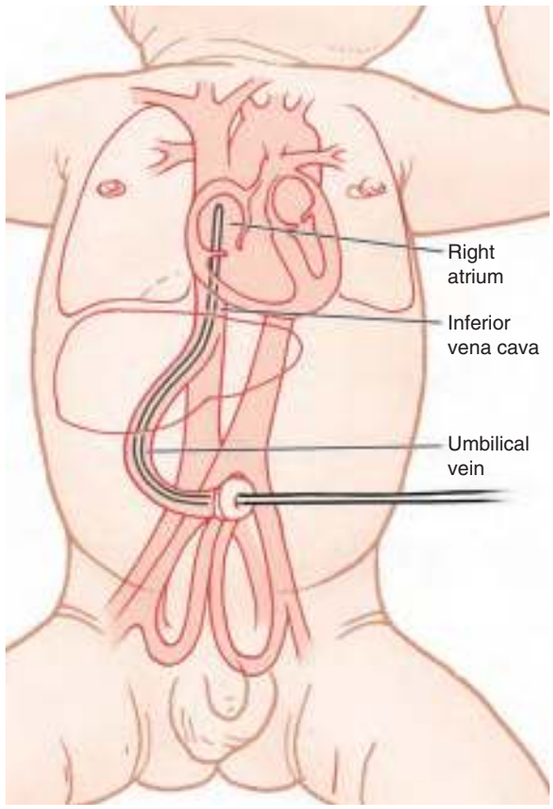


FIGURE 71-11. Positioning of the tip of the umbilical vein catheter. The tip should be above the diaphragm, at the junction of the inferior vena cava and the right atrium.

aspirated (i.e., approximately 5 cm beyond the umbilical stump) and may be used without waiting for radiographic confirmation of placement. It is imperative that the catheter not be inserted too deeply to prevent entry into the hepatic vessels via the ductus venosus as medications could potentially cause hepatocellular damage.[3,6,8,30,31] The catheter can be replaced with a more permanent line once the patient has been stabilized.[29] Place the nonemergent UVC with the tip above the diaphragm, at the junction of the IVC and right atrium as determined previously. Tighten the umbilical tape to temporarily secure the catheter. Obtain fluoroscopic, plain radiographic, or ultrasonic confirmation of placement prior to using the catheter.[23]

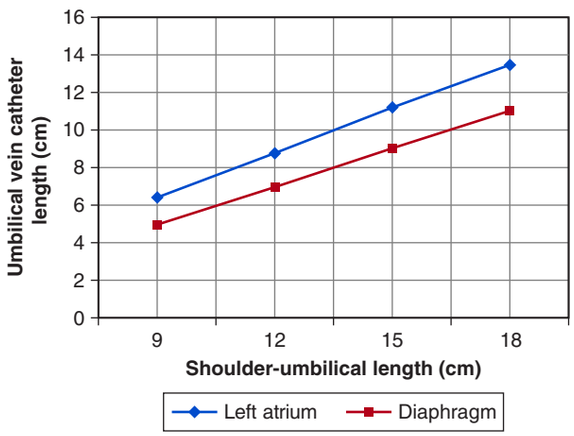


FIGURE 71-12. Graph to determine the correct length of catheter to insert into the umbilical vein. Using the shoulder-umbilical length and the desired position of the catheter tip, determine the length of catheter on the *y*-axis to be inserted into the vein. Add the length of the umbilical stump to the umbilical vein catheter length found on the *y*-axis of the graph to determine the corrected length of catheter to be inserted. (Adapted and modified from reference 13.)

UMBILICAL MULTIPLE-LUMEN CATHETERS

Reliable vascular access can be problematic in the sick neonate.[32] Insertion of a double-lumen or triple-lumen catheter into the umbilical vein can provide additional venous access for the administration of incompatible drugs (e.g., vasopressor agents, calcium solutions, or sodium bicarbonate solutions).[33,34] It is also useful for drugs requiring continuous infusions and leaves other ports for blood sampling, central venous pressure measurements, maintenance fluids, or medication administration.[34] Infuse all of these solutions into the proximal lumen(s) and allow measurements of central venous pressures from the distal port. Multilumen catheters can be placed directly by the same methods as a single-lumen catheter or using the Seldinger technique as with inserting a central venous line (Chapter 63). **The increased pliability of the multilumen catheter makes passage into the hepatic veins more likely.**

A wire exchange technique may be used to place a multiple-lumen catheter in neonates with an indwelling single-lumen catheter. This method decreases the probability of loss of vascular access during the exchange. It has the risk of the guidewire entering the heart and causing a cardiac dysrhythmia and/or myocardial perforation. This technique should only be performed by those familiar with the Seldinger technique in replacing an umbilical catheter.

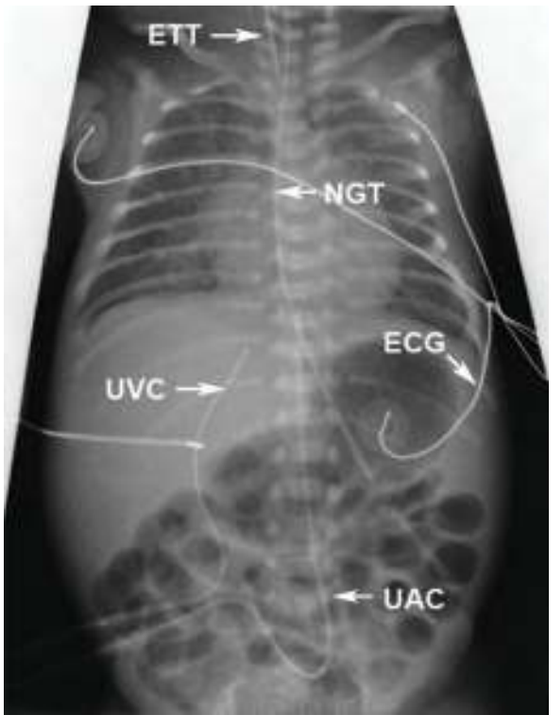
The catheter exchange over a guidewire requires strict sterile technique. Insert a guidewire through the umbilical catheter. Firmly hold the guidewire. Withdraw the catheter over the guidewire. **Never let go of the guidewire which must always remain within the vein.** Advance the multilumen catheter over the guidewire and into the vein while securely holding the guidewire. Continue to advance the multilumen catheter to its predetermined length. Remove the guidewire while maintaining the catheter in the umbilical vessel. Tighten the umbilical tape to temporarily secure the catheter. Obtain fluoroscopic, plain radiographic, or ultrasonic confirmation of placement prior to using the catheter.

ASSESSMENT

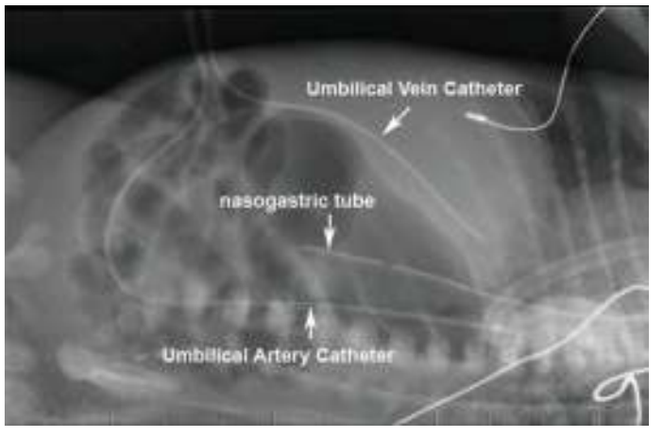
Confirm the proper placement of the catheter tip (**Figure 71-13**). The umbilical tape has been tightened to temporarily secure the catheter within the umbilical cord. **Do not infuse anything through the line until proper placement is confirmed in a nonemergent situation.** This can be accomplished with fluoroscopy, plain radiographs, or ultrasonography. If the catheter has been inserted too far, withdraw it and reevaluate the new catheter tip position. **If the catheter has not been inserted far enough, do not advance the catheter. Withdraw the catheter and restart the procedure with a new catheter.**

AFTERCARE

Secure the catheter more permanently after confirming that the catheter tip is in the desired location (**Figure 71-14**). Tighten and tie a knot in the previously placed purse-string suture. Wrap the loose ends of the suture around the catheter as it enters the umbilical vessel and secure it with several square knots (**Figure 71-14**). **Loosen and remove the umbilical tape to avoid umbilical cord necrosis.**[5] Secure the catheter to the abdominal wall with adhesive tape or a large transparent dressing (**Figure 71-14**). Antibiotic ointment may be applied to the junction of the umbilical cord and the catheter.

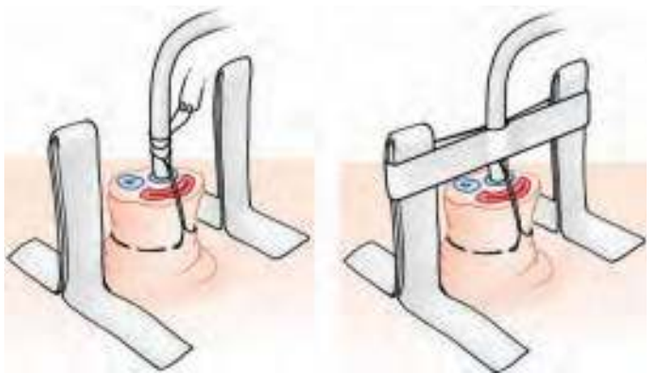


A



B

FIGURE 71-13. Plain radiographs to confirm the placement of an umbilical catheter. ***A.*** Anteroposterior view. ***B.*** Lateral view. ETT, endotracheal tube; ECG, electrocardiogram; NGT, nasogastric tube. (Used from http://wikiradiography.net.)



A B

FIGURE 71-14. Securing the umbilical vessel catheter. ***A.*** The purse-string suture is tied about the umbilical cord and the umbilical tape is removed. The ends of the suture are wrapped around the catheter, as it enters the umbilical vessel, and secured with square knots. ***B.*** Tape is applied to secure the catheter to the abdominal wall.

COMPLICATIONS

Significant morbidity can be associated with umbilical artery and vein catheterization.[35] Prevention of complications requires strict adherence to sterile technique, flushing of the catheter prior to insertion, gentle catheter manipulation during insertion, and accurate positioning of the catheter. It is essential that no air be allowed to enter the catheter. An air bubble can enter the central circulation, pass through the foramen ovale, lodge in an artery, and lead to end-organ damage (e.g., stroke, myocardial infarction, or death).[36,37]

Complication rates have been reported to be as high as 20% for venous catheters and 10% for arterial catheters.[38] A venous catheter placed in the portal system may lead to hepatic necrosis, hemorrhage, portal vein thrombus, portal hypertension, or pulmonary embolism.[4-6,30] Neonates are at higher risk for thromboembolic events because of their underdeveloped clotting mechanisms and smaller vessel diameters.[35] Phlebitis or nosocomial sepsis can ensue if strict aseptic technique is not followed.[38] The catheter can be transected or form a knot intravascularly.[39,40] Vessel and bowel perforation from forceful manipulation of the catheter, an air embolus from an unflushed catheter, false track formation, cardiac arrhythmias, damage to cardiac valves, and myocardial perforation have all been reported.[4,8,30,38,41] Necrotizing enterocolitis, biliary venous fistula, pericardial effusion, hypoglycemia from high positioning of a UAC, bladder rupture, congestive heart failure, hypertension, transection of an omphalocele, transection of the catheter, intravascular knots in the catheter, and Wharton's jelly embolus have also been reported.[41-48] A persistent urachus in the umbilical cord stump may be mistaken for an umbilical vein. Catheterization of the urachus will result in the flow of urine from the catheter. This is easily identified and corrected.

Malpositioning of the UVC has resulted in rare complications such as pneumopericardium, cardiac tamponade, pleural effusion, pulmonary hemorrhage, pulmonary edema, pulmonary infarction, pulmonary abscess, cardiac thrombosis, endocarditis, myocardial perforation, atrial flutter, hyponatremia, perforation of the peritoneum, perforation of a Meckel's diverticulum, perforated urachal remnant, ascites from intraperitoneal extravasation of total parenteral nutrition, esophageal varices, hepatic laceration, hepatic necrosis, hepatic abscess, hepatic calcifications or bladder calcifications with infusion of calcium gluconate, biliary fistula, perforation of the IVC, gastric outlet obstruction, or bladder rupture.[9,40,49-69]

Malpositioning of the UAC has rarely resulted in sciatic nerve damage, refractory hypoglycemia from infusion into the celiac axis, or spinal cord injury with paraplegia from infusion into the artery of Adamkiewicz.[3,27,38,70,71] Umbilical artery catheterization may be complicated by an abdominal aortic aneurysm, aortic coarctation, vasospasm, thrombosis, or embolism, causing ischemia of the lower extremities or intraabdominal organs.[2,6,30,38,72-75]

Vasospasm can occur within moments of catheter insertion or up to several hours following placement. The signs include progressive ischemia resulting in discoloration or mottling of the lower extremities and/or buttocks. Embolization of clots can cause loss of digits, hematuria, kidney failure, hypertension, necrotizing enterocolitis, bowel infarction, cyanosis or blanching of the skin (i.e., of the back, buttocks, or legs), or skin ulceration.

SUMMARY

Umbilical vessel catheterization is a readily available method of obtaining vascular access in the sick newborn. These vessels can be used for fluid resuscitation, blood transfusion, medication administration, frequent blood sampling, and cardiovascular monitoring. Umbilical access is reserved for infants in whom intraosseous and peripheral vascular access is likely to be unsuccessful. Serious complications can result from using the umbilical vessels.

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72 Arterial Puncture and Cannulation

Justin Grisham and Jennifer Stankus

INTRODUCTION

Arterial blood gas sampling is an essential component of the care of many Emergency Department patients. It provides key information regarding a patient's oxygenation and acid-base status. Arterial cannulation allows for continuous and accurate blood pressure monitoring and frequent blood gas sampling in the care of the critically ill patient. There are many issues associated with obtaining and maintaining arterial access. Placement of an arterial line occurs less commonly than venous access. It is important to know when to obtain arterial access, where to obtain arterial access, and how to obtain arterial access. The use of an arterial line provides a more accurate blood pressure than using a noninvasive cuff.

ANATOMY AND PATHOPHYSIOLOGY

Knowledge of the arterial anatomy is a key factor in the success of arterial puncture and cannulation. It is important to recognize that nerves and veins are in close proximity to the desired arteries in order to avoid complications. The anatomy and positioning for radial, brachial, femoral, and dorsalis pedis artery access are described below.

RADIAL ARTERY

The radial artery is the preferred site for arterial puncture and cannulation. One reason is the comparative ease of identifying the anatomic location of this artery. A second reason is the collateral nature of the arterial blood supply to the hand provided by the radial and ulnar arteries (**Figure 72-1**). The ulnar artery is not often used due to its smaller size. Terminal branches of these two arteries meet in the palm of the hand to form the deep and superficial palmar arterial arches (**Figure 72-1**).

The radial artery is just medial and proximal to the radial styloid process on the ventrolateral wrist (**Figure 72-1**). Dorsiflexing the wrist approximately 45° places the radial artery at its most superficial position to the skin and can aid in palpating the arterial pulse.[1] Another notable landmark is the flexor carpi radialis tendon immediately medial to the radial artery. The recommended point of needle or catheter insertion is at the proximal flexor crease of the wrist and directly above the radial artery pulse.

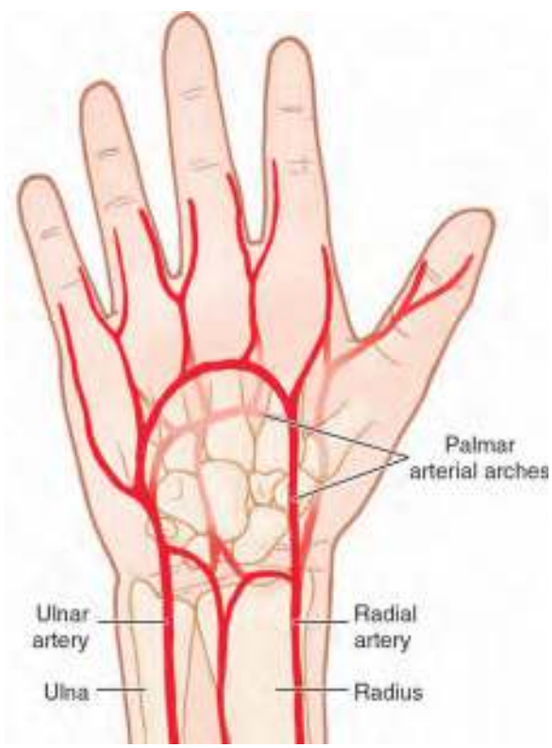


FIGURE 72-1. Anatomic location of the radial and ulnar arteries. Collateral circulation is provided by the superficial and deep palmar arches.

Perform an Allen test to assess the adequacy of the collateral circulation to the hand prior to radial artery puncture or cannulation (Figure 72-2).[2-4] Ask the patient to repeatedly close their hand tightly into a fist and open it to force blood out of the fingers while manually occluding the radial and ulnar arteries (**Figure 72-2*A***). Continue this process for 1 minute. Ask the patient to open their hand. The fingers will blanch and become pale due to the occlusion of the arterial inflow. Release the finger occluding the radial artery (**Figure 72-2*B***). Measure the time it takes for blushing of the palm to occur. It is considered normal if it is < 7 seconds, equivocal at 8 to 14 seconds, and abnormal if > 14 seconds.[2] Repeat the test with release of the ulnar artery compression to confirm arterial flow into the hand. **The purpose of an Allen test is to confirm arterial inflow from both the radial and ulnar arteries.**

An alternative method of evaluating the collateral circulation involves the use of a pulse oximeter with a visual pulse waveform display.[5] This is useful in the unconscious patient, uncooperative patient, or very young patient. Place the pulse oximeter sensor on the patient's thumb. Observe the visual display to confirm a waveform is present. Occlude the radial artery and examine the waveform on the monitor. Either an ulnar dominant system or a radial dominant system with adequate collateral circulation is likely if the waveform remains unchanged during radial artery occlusion.[5] Occlude the ulnar artery and examine the waveform on the monitor. The ulnar artery can supply the hand if the waveform is unchanged. An ultrasound (US) with Doppler can be used instead of pulse oximetry.[6]

The performance of an Allen test to confirm adequate collateral circulation to the hand is generally advocated before radial artery puncture or cannulation. There is concern that radial artery occlusion from an intraluminal clot or an external hematoma can result in hand ischemia if the ulnar artery cannot provide adequate collateral blood flow. Some authors have questioned the utility of performing an Allen test.[2,3,7,8] The Allen test is subjective, often improperly performed, and has poor sensitivity and specificity to predict complications.[9] The relative safety of radial artery cannulation without the Allen test has been demonstrated in a large case series of patients without major peripheral vascular disease.[7] **An abnormal Allen test may not preclude radial artery puncture or cannulation but may indicate a greater need for caution and alert the Emergency Physician to potential problems after the procedure.**[3,6] This can include arterial thrombosis, hand ischemia, and hand necrosis. **It is recommended, but not required, to cannulate another site if the Allen test is abnormal.**

FIGURE 72-2. The modified Allen test. ***A.*** The distal radial and ulnar arteries are occluded. ***B.*** The ulnar artery remains occluded while determining if the radial artery can supply adequate blood flow to the hand.

BRACHIAL ARTERY

The brachial artery courses along the medial side of the antecubital fossa just lateral to the median nerve (**Figure 72-3**). The brachial artery divides at approximately the level of the neck of the radius to become the ulnar and radial arteries. The brachial artery in the antecubital fossa is lateral to the medial epicondyle of the humerus and medial to the biceps brachii muscle. The brachial artery is more easily identified when the elbow is fully extended.

Palpate the medial epicondyle of the humerus. Move laterally until the medial edge of the biceps muscle is palpated. Palpate the brachial artery pulse just medial to the biceps muscle. The arterial pulsation is most easily identified at the level of the proximal flexor crease of the antecubital fossa. The preferred location for puncture or cannulation of the brachial artery is in the antecubital fossa and directly above the brachial artery pulse. Alternatively perform the procedure just proximal to the antecubital fossa. Keep the arm in extension while the cannula is in place. **The brachial artery does**

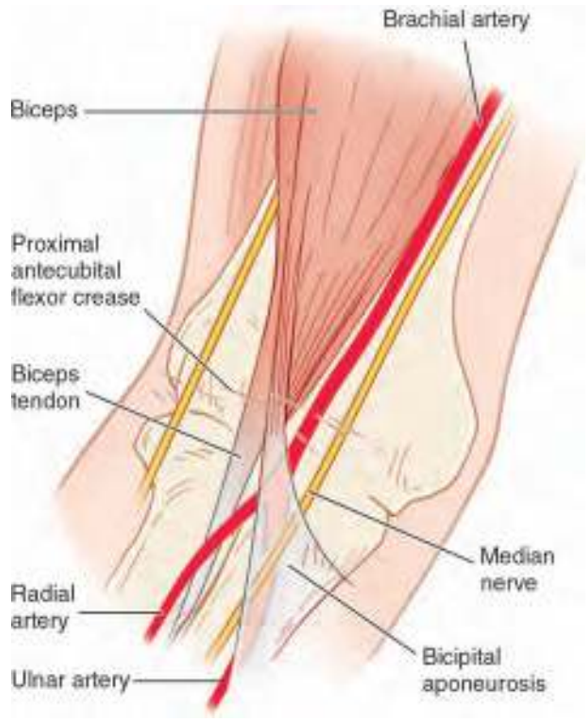


FIGURE 72-3. Anatomic location of the brachial artery. Note the median nerve running just medial to the artery and the biceps brachii muscle just lateral to the artery.

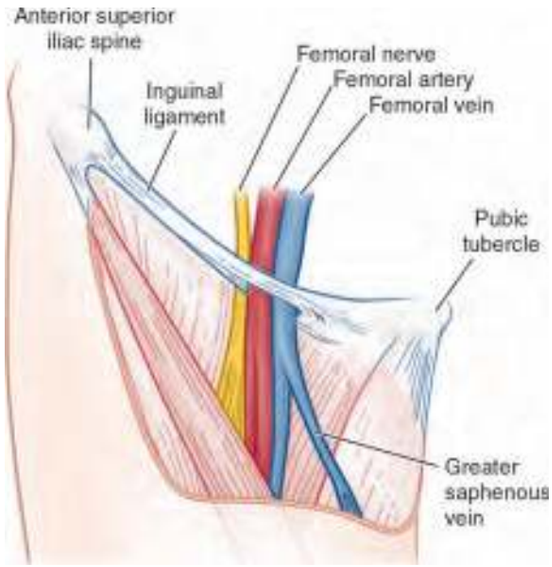


FIGURE 72-4. Anatomic location of the femoral artery. Note the proximity to the femoral nerve and vein.

not have the same degree of collateral circulation as the radial artery and care should be taken if this site is chosen.

FEMORAL ARTERY

The bony anatomic landmarks used to identify the femoral artery are the anterior superior iliac spine and the tubercle of the pubic symphysis. The artery lies approximately midway between these two points after it courses under the inguinal ligament to enter the thigh (**Figure 72-4**). The femoral nerve and vein run parallel and adjacent to the artery. The vein lies just medial to the artery and the nerve just lateral. The femoral artery is larger than other arteries commonly cannulated and lies significantly deeper than the radial or brachial arteries. This makes it necessary to use a longer cannula and may require a longer needle for a simple arterial puncture to be successful. Extension and slight abduction of the hip maximizes access to the femoral triangle, improves the ability to palpate the artery, and provides a maximal work area for the procedure.

DORSALIS PEDIS ARTERY

The dorsalis pedis artery is a continuation of the anterior tibial artery on the dorsal surface of the foot. Puncture and cannulation of the dorsalis pedis artery constitute a good second choice if the radial artery is unsuccessfully cannulated or unavailable.[10] The pulse is often easily palpable between the first and second metatarsal due to its superficial location. Its distal location does not interfere with other resuscitative efforts and is convenient for the patient. The risk of foot ischemia is minimal due to the abundant collateral circulation to the foot and ankle. There are no significant structures to injure adjacent to the dorsalis pedis artery. This artery may be absent in some people or have significant variability in its anatomic location. It may be difficult to identify the dorsalis pedis pulse in the hypotensive patient.

ALTERNATIVE ARTERIAL SITES

There are several alternative sites that are occasionally used for arterial puncture and cannulation. These include the superficial temporal artery, the axillary artery, and the ulnar artery. The superficial temporal artery is often used in neonates and young infants in the Intensive Care Unit. Cannulation of this artery is rarely performed in the Emergency Department because of its location and the ability to maintain access.

The axillary artery is a continuation of the subclavian artery after the first rib. The axillary artery crosses the teres major tendon to enter the arm as the brachial artery. The axillary artery can be deep to the skin surface. An advantage of this artery is the ease of locating the palpable pulse, even in the hypotensive patient.[11,12] An accurate blood pressure devoid of the effects of vasoconstriction can be obtained. US makes it easier to obtain arterial access and increases the success rate.[11,12]

The disadvantages of cannulating the axillary artery are significant.[13] The axilla is poorly accessible. The patient's arm must be abducted, externally rotated, and immobilized during and after the procedure. The course of the artery changes with arm position. The artery is contained within the axillary sheath along with the axillary vein and brachial plexus. **There is a significant risk of nerve injury if the needle penetrates the brachial plexus.** The risk of an arterial embolus is higher in central arteries when compared to peripheral arteries.

The ulnar artery and the radial artery are the terminal branches of the brachial artery. The ulnar artery is superficial at the wrist and the pulse is easily palpable. The ulnar artery is significantly smaller in most people when compared to the radial artery. This makes the procedure more technically difficult. An ulnar artery may be absent or extremely small in some patients. The ulnar artery lies adjacent to the ulnar nerve. This increases the possibility of nerve injury if the needle penetrates the ulnar nerve. It can be difficult to identify the artery in the patient with anasarca, obesity, peripheral edema, or peripheral vascular disease.

INDICATIONS

The principal indications for arterial blood sampling include the determination of the acid-base status, blood carbon dioxide (CO_2) content, and blood oxygen (O_2) content.[14,15] The need for arterial blood gas (ABG) sampling for O_2 determination has decreased substantially with pulse oximetry. Pulse oximetry may poorly reflect true oxygenation in the setting of hypothermia, severe hypotension, or severe hypoxia. Therefore, ABG analysis persists as the true measure of arterial oxygenation.[16] End-tidal CO_2 (E_TCO_2) monitoring has decreased the utilization of ABG samples for CO_2 measurement. E_TCO_2 measurements may grossly underestimate the true CO_2 contents of the blood in patients who have large dead space ventilation or low cardiac output.[17]

ABG samples are useful for accurate pH monitoring of patients with obstructive lung disease, other pulmonary disorders, and shock. Possibly the most important indication for ABG sampling in the critically ill patient is the determination of the patient's acid-base status. Venous sampling may occasionally suffice for monitoring the pH (e.g., diabetic ketoacidosis).[18] The measurement of venous blood pH is much less reliable as a surrogate for arterial pH in patients with shock and other critical illnesses.[19] Arterial blood samples are still used for the measurement of carboxyhemoglobin and methemoglobin level. Arterial and venous co-oximetry carboxyhemoglobin values are closely correlated and may be used to screen patients thought to have been exposed to carbon monoxide.[20]

The three principal indications for arterial catheter placement are the need for continuous monitoring of arterial blood pressure, the need for frequent ABG sampling, and the need for frequent blood sampling for laboratory analysis in the critically ill patient. Cycled oscillometric blood pressure measurement (i.e., an external blood pressure cuff) may be insufficient to gauge rapid

hemodynamic changes in critically ill hypertensive or hypotensive patients. External blood pressure cuffs may be ineffective in patients with severe burns or trauma. Continuous blood pressure monitoring facilitates titration of rapid-acting vasodilators and vasopressors. The accuracy of auscultated or oscillometric blood pressure readings under conditions of severe malperfusion or shock is suspect.[2,3] Centrally measured aortic pressure is the true gold standard.[2,3] Peripheral arterial pressure measured with an intraarterial catheter connected to a pressure transducer is the next best gauge given the impracticality of measurement of aortic pressure.

CONTRAINDICATIONS

Contraindications to arterial puncture and catheter placement relate primarily to abnormalities at the insertion sites. There are no absolute contraindications to arterial puncture or catheterization. Avoid skin and arteries that are already compromised by burns, infection, previous surgery in the area, severe dermatitis, severe peripheral vascular disease, or trauma.[2,14] Puncture or cannulation of synthetic vascular grafts is relatively contraindicated. **Do not puncture where the artery "should be" if the arterial pulsation cannot be palpated.** Attempts at cannulation of nonpalpable arteries are generally fruitless and sometimes hazardous. Arterial puncture or catheterization is relatively contraindicated in patients with bleeding diatheses. Consider whether arterial access is really needed in those who have received or may receive thrombolytic therapy.

EQUIPMENT

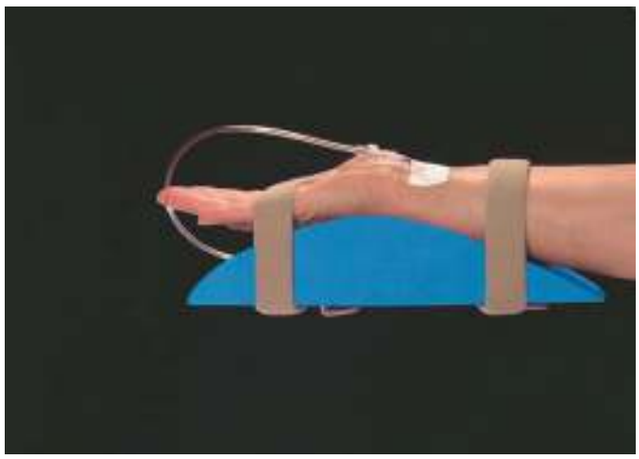
ARTERIAL PUNCTURE FOR SINGLE ABG SAMPLE

- Povidone iodine or chlorhexidine solution
- Dorsal wrist extensor splint or small rolled up towel (**Figures 72-5, 72-6, and 72-7**)
- 1% lidocaine, 1 mL in a tuberculin syringe
- 5 mL syringe for blood collection with cap retained
- Prepackaged blood gas syringe with lyophilized heparin pellet
- 20 to 22 gauge needle for arterial puncture
- 1 to 2 mL of heparin (1000 U/mL)
- Gauze pads
- Adhesive tape
- Specimen collection bag or cup with 2 to 3 inches of ice

ARTERIAL (BRACHIAL, RADIAL, OR DORSALIS PEDIS) CANNULATION

- Povidone iodine or chlorhexidine solution
- Dorsal wrist extensor splint or small rolled up towel (**Figures 72-5, 72-6, and 72-7**)
- 1% lidocaine, 1 mL in a tuberculin syringe
- 4 × 4 gauze squares
- Adhesive tape
- Nylon suture, 3–0 or 4–0
- Needle drivers
- 20 or 22 gauge, 1¾ inch polyurethane angiocatheter or catheter-over-the-needle
- 0.45 mm diameter, 5¼ inch spring wire guide compatible with above catheter
- Sterile dressing to cover cannula

A



B

C

FIGURE 72-5. Commercially available wrist splints used for radial arterial lines. ***A.*** Velcro wrist extension splint. ***B.*** Support A-Line Delux. ***C.*** MillSuss device. (Used with permission from reference 62.)

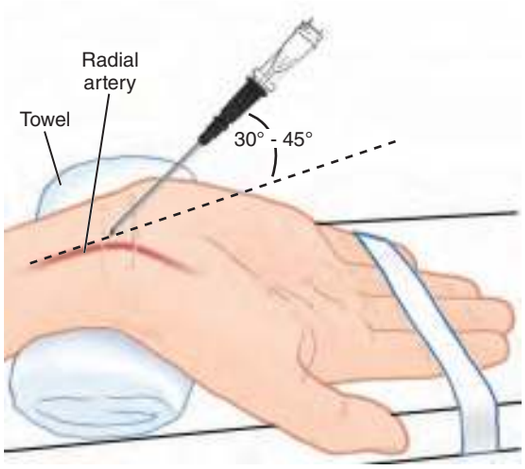


FIGURE 72-6. Correct positioning for radial artery puncture and cannulation. Dorsiflexing the wrist and supporting it with a small towel facilitate palpation of the artery and provide maximum working space. The needle or catheter-over-the-needle is aimed toward the oncoming blood flow at a 30° to 45° angle to the skin.

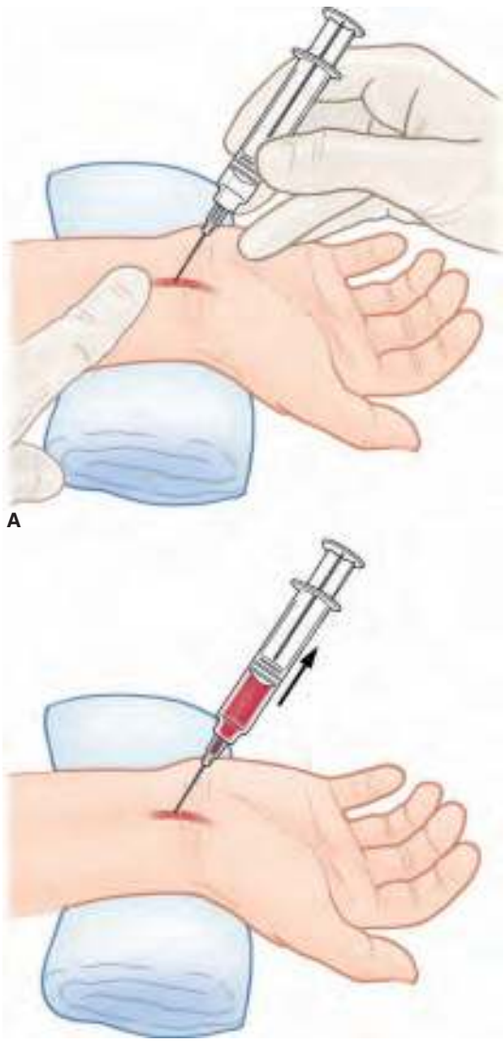


FIGURE 72-7. Radial artery puncture for an ABG sample. ***A.*** The pulse is palpated with the nondominant hand. The heparinized syringe is inserted at a 30° to 45° angle to the skin surface. ***B.*** Arterial blood fills the syringe.

FEMORAL ARTERY CANNULATION

- Povidone iodine or chlorhexidine solution
- 1% lidocaine, 3 mL in a syringe armed with a 25 gauge needle
- 4 × 4 gauze squares
- #11 scalpel blade
- Nylon suture, 3–0 or 4–0 and needle drivers
- Femoral artery needle, approximately 18 gauge and 3 inches long
- 4 French single lumen catheter, at least 15 cm in length
- 30 cm guidewire compatible with above needle and catheter
- Sterile dressing to cover cannula

ULTRASOUND GUIDANCE

- US machine
- 5 to 10 MHz linear array US transducer
- Sterile US gel
- Sterile US transducer cover

It is necessary to prepare the syringe using heparin solution if a syringe that has not been designed specifically for ABG sampling is used, it has not been pretreated with heparin, or it does not contain a lyophilized heparin pellet. Draw up 1 to 2 mL of heparin solution into the syringe and then expel the heparin, leaving only the heparin remaining in the dead space of the syringe and the needle. This amount of heparin is sufficient to prevent clotting of the sample.[14] It is important to remove all but the necessary amount of heparin as excess heparin has been found to falsely lower the PCO_2 measurement and may elevate the PO_2 measurement.[21]

Commercially produced, prepackaged kits are available for arterial puncture and arterial cannulation (**Figure 72-8**). These kits are complete and contain all the required equipment. Also available are

FIGURE 72-8. A commercially available arterial line kit. ***A.*** The kit. ***B.*** The catheter-needle-guidewire unit.

individually packaged heparinized ABG syringes and arterial line catheters in various sizes.

The catheter size used for the procedure will vary by patient age and the artery chosen to cannulate. Use a 22 or 24 gauge catheter for the radial or dorsalis pedis arteries and an 18 or 20 gauge for the femoral artery in neonates and infants. Use a 22 gauge catheter for the radial or dorsalis pedis arteries and a 16 to 20 gauge for the femoral artery in children up to 8 to 10 years of age. Use a 20 or 22 gauge catheter for the radial or dorsalis pedis arteries and a 14 to 20 gauge for the femoral artery in a larger child, adolescent, or adult.

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative. Obtain an informed consent unless the procedure is being performed emergently or the patient is unable to give consent. Document the informed consent or the reason why informed consent was not obtained in the medical record. Palpate the arterial pulse at the intended skin puncture site. Clean the skin of any dirt and debris. Cleanse the skin with chlorhexidine or povidone iodine solution and allow it to dry. There is some evidence that chlorhexidine is better than povidone iodine to prevent postprocedure infection.[22,23]

Studies that examined the use of a topical anesthetic agents have not shown a benefit that outweighs the delay required to perform the procedure in emergent situations.[24] The use of an injectable local anesthetic agent may greatly aid arterial puncture, aid in arterial cannulation, and significantly reduce patient discomfort.[25] Infiltrate 1 mL of local anesthetic solution subcutaneously over the brachial, dorsalis pedis, or radial arteries. Infiltrate 2 to 5 mL of local anesthetic solution subcutaneously and into the subcutaneous tissues over the femoral artery. **Aspirate before infiltrating the local anesthetic solution to prevent inadvertent intravascular injection.**

Descriptions of the anatomy for arterial puncture or cannulation sites have been described in detail earlier in this chapter. The preferred site for the initial attempt at arterial puncture or cannulation is the radial artery.[2] Begin distally where the pulse is most palpable near the proximal wrist flexor crease. Reattempt the procedure more proximally along this same artery if the first attempt is unsuccessful and the pulse is still palpable. The radial artery on the contralateral wrist is a satisfactory second site for attempted access. Other acceptable second-attempt sites of access include the brachial, dorsalis pedis, and femoral arteries. **An attempt at ipsilateral ulnar artery catheterization is not advisable as both limbs of the hand's circulation may be compromised.** The discussion below focuses on puncture or cannulation of the radial and femoral arteries as over 90% of arterial punctures or cannulations occur at these sites.[2] The use of other sites generally follows the techniques described for the radial artery except the regional anatomic differences. Other rarely used sites include the posterior tibial and superficial temporal arteries.

TECHNIQUES

ARTERIAL PUNCTURE FOR A SINGLE SAMPLE

Position the patient to maximize exposure of the skin surface overlying the chosen artery. Dorsiflex the wrist and support this position with a small towel rolled up under the dorsal wrist surface or a commercial device (**Figures 72-5, 72-6, and 72-7**).[26] The patient's hand may be secured to an arm board or other rigid object with tape to facilitate a secure puncture site that will remain in the most advantageous position. Ensure that the elbow or hip is extended fully for brachial and femoral artery puncture. These positions provide maximum exposure and working area for the procedure. Locate the chosen artery, prep the overlying skin, and infiltrate local anesthetic solution subcutaneously.

Reidentify the pulse by palpation with the nondominant hand using sterile technique (**Figure 72-7*A***). Grasp the heparinized syringe with the dominant hand. Withdraw the plunger of the syringe so that 1 to 3 mL of air space is available in the syringe. This will allow for easier assessment of arterial blood return. Insert the needle at a 30° to 45° angle to the skin and just above the arterial pulse (**Figure 72-6**). Advance the needle through the skin until blood enters the syringe (**Figure 72-7*B***). The blood flow will generally fill the syringe without necessitating the withdrawal of the plunger in a patient with a brisk pulse. Slowly withdraw the needle if no blood return occurs and watch for blood flow into the syringe. **If it is necessary to redirect the needle, it is imperative to first withdraw the needle until the tip is just below the skin surface before changing the angle to avoid lacerating the artery or adjacent structures.** Collect at least 1 mL of blood in a prepackaged syringe or 3 mL of blood if preparing your own syringe with heparin to minimize the possibility of error due to the presence of heparin.[14]

Withdraw the needle after the arterial sample has been collected. Apply pressure to the puncture site for 3 to 5 minutes followed by a bandage or gauze dressing. Carefully remove the needle from the syringe if not using a safety needle. Evacuate any air from the syringe and apply a cap on the syringe. Place the syringe on ice for immediate transportation to the laboratory. Recheck the skin puncture site in 5 to 10 minutes to assess for the formation of a hematoma and/or vascular compromise to the distal extremity.

RADIAL ARTERY CANNULATION: CATHETER-OVER-THE-NEEDLE TECHNIQUE

The most basic method is direct introduction of the catheter-over-the-needle (i.e., angiocatheter) like inserting an intravenous catheter (**Figures 72-6 and 72-9**). This method is as effective as the more specialized wire-guided catheter technique, except in situations where the pulse is weak, the pulse is absent, or in the hands of more experienced operators.[26,27]

Clean, prep, and identify the radial artery. Insert the catheter-over-the-needle at a 30° to 45° angle to the skin and directly over the arterial pulse (**Figures 72-6 and 72-9*A***). Advance the catheter-over-the-needle into the artery (**Figure 72-9*A***). Bright red blood in the hub of the needle indicates that the tip of the needle is within the artery. Advance the catheter-over-the-needle another 1 to 2 mm to ensure that the catheter tip is completely within the arterial lumen. Securely hold the hub of the needle. Advance the catheter over the needle until its hub is against the skin (**Figure 72-9*B***). Remove the needle and confirm pulsatile arterial flow from the hub of the catheter. **Free-flowing pulsatile blood confirms proper catheter placement within the artery.** Apply a stopcock or intravenous extension tubing to the hub of the catheter. Secure the catheter by suturing it to the skin or by using commercially available devices that do not require suturing. Apply a dressing to the skin puncture site. Using a self-adherent dressing that contain a chlorhexidine-impregnated patch may reduce catheter-related bloodstream infection.[28,29]

RADIAL ARTERY CANNULATION: SELDINGER-TYPE, SINGLE ARTERIAL WALL PUNCTURE

The second technique for arterial cannulation is a Seldinger-type technique (i.e., catheter-over-the-wire) using prepackaged commercially available kits. A commercially available one-piece catheter-over-the-needle kit is very popular (**Figures 72-8 and 72-10**). These wire-guided catheters have not been shown to increase successful cannulation or reduce the number of attempts except in situations

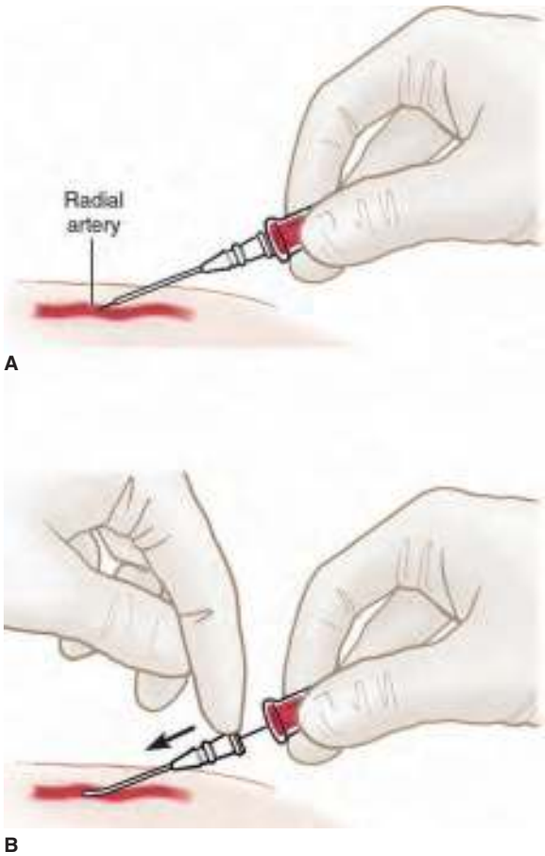


FIGURE 72-9. The catheter-over-the-needle technique for arterial cannulation. ***A.*** The unit is held at a 30° to 45° angle to the skin and advanced into the artery. ***B.*** The catheter is advanced over the needle and into the artery.

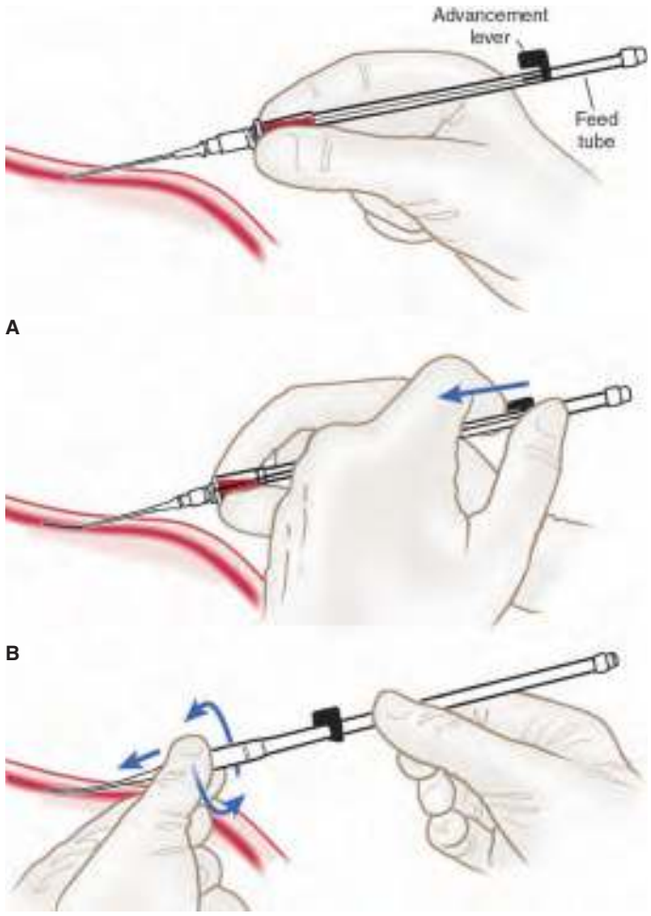


FIGURE 72-10. Catheter-over-the-needle technique using a commercially available one-piece unit. ***A.*** The catheter-over-the-needle is inserted into the artery. ***B.*** The guidewire is advanced through the needle and into the artery. ***C.*** The catheter is advanced over the guidewire into the artery with a twisting motion.

where a weak or absent pulse is present or with more experienced Physicians.[26,27] **The guidewire can be useful in salvaging an arterial line when a catheter-over-the-needle cannot be effectively advanced.**

Open the package, remove the unit, and remove the protective cover over the needle. Advance and retract the guidewire to confirm it moves smoothly and does not get caught on the needle (**Figure 61-15**). Retract the guidewire as far back as possible. Identify the arterial pulse with the nondominant hand. Insert the catheter-over-the-needle through the skin and into the artery using a slow and continuous forward motion (**Figure 72-10*A***). A flash of blood in the hub of the needle confirms successful entry through the arterial wall and into the vessel lumen. Stabilize the catheter-over-the-needle.

Advance the guidewire through the needle by pushing the actuating lever as far as possible toward the needle (**Figure 72-10*B***). **Immediately stop if resistance is encountered while advancing the guidewire. The guidewire may be within the artery wall or through the artery wall and into the perivascular tissue. Do not try to force the guidewire into the vessel. Do not retract the guidewire. Withdraw the entire unit and apply pressure to the puncture site to prevent a hematoma.** Obtain a new kit and repeat the procedure.

The guidewire is successfully within the arterial lumen once advanced. Firmly grasp the clear hub of the needle and advance the catheter over the guidewire and into the artery (**Figure 72-10*C***). **A rotating motion of the catheter is often helpful to advance it if difficulty is encountered.** Securely hold the catheter at the level of the skin. Firmly hold the catheter hub and remove the guidewire, needle, and feed tube assembly as a unit. Free-flowing pulsatile blood confirms proper catheter placement within the artery. Apply a stopcock or intravenous extension tubing to the hub of the catheter. Secure the catheter by suturing it to the skin or by using commercially available devices that do not require suturing. Apply a dressing to the skin puncture site.

A commercially available Seldinger-type catheter-over-the-needle kit is an alternative to the one-piece unit.[30] This is similar to the familiar central venous access kit (**Figure 63-9**). Open the package and review the equipment it contains. Place the finder needle on the syringe. Withdraw the plunger 1 cm to break the bead of the syringe. Identify the arterial pulse by palpation with the nondominant hand. Insert the needle through the skin and into the artery using a slow and continuous forward motion (**Figure 72-11*A***). A flash of blood in the syringe confirms the successful entry through the arterial wall and into the vessel lumen. Firmly hold and stabilize the needle. Remove the syringe. Advance the guidewire through the needle (**Figure 72-11*B***). **The guidewire should advance without resistance. Immediately stop if resistance is encountered while advancing the guidewire. The guidewire may be within the artery wall or through the artery wall and into the perivascular tissue. Do not try to force the guidewire into the vessel. Do not retract the guidewire to prevent shearing off a piece of the guidewire.**

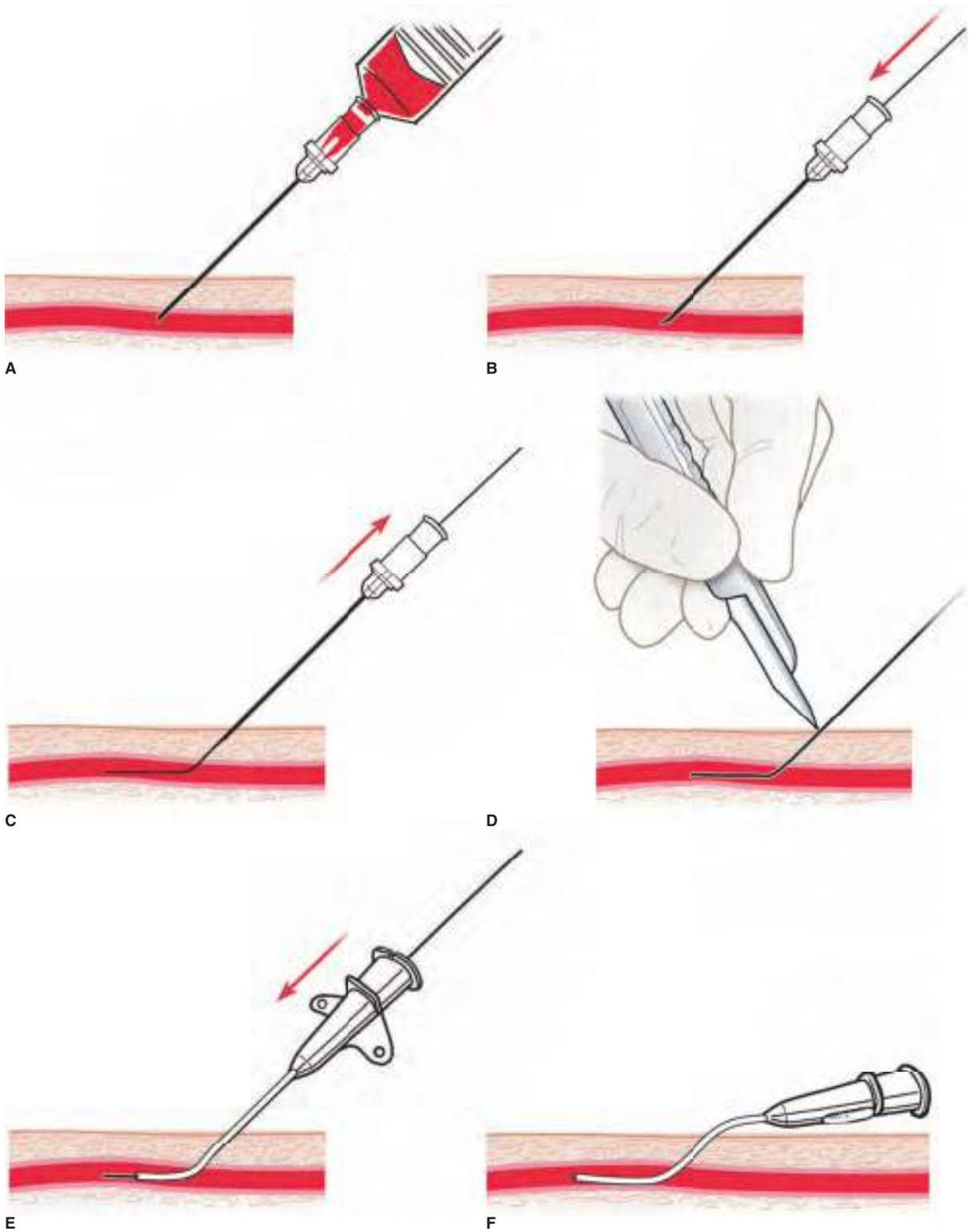


FIGURE 72-11. The Seldinger technique for arterial catheterization. ***A.*** The needle is inserted into the artery. ***B.*** The syringe has been removed from the needle. The guidewire is advanced through the needle and into the artery. ***C.*** The needle is removed while leaving the guidewire in place. ***D.*** The skin is punctured with a #11 scalpel blade to allow easy insertion of the catheter. ***E.*** The catheter is advanced over the guidewire and into the artery. ***F.*** The guidewire is removed.

Withdraw the entire unit and apply pressure to the puncture site to prevent a hematoma. Obtain a new kit and repeat the procedure.

Withdraw the needle while leaving the guidewire in place (**Figure 72-11*C***). **Do not readvance the needle as it can shear off a piece of the guidewire.** Make a 3 mm puncture wound next to the skin puncture site of the guidewire using a #11 scalpel blade to facilitate inserting the catheter (**Figure 72-11*D***). Thread the catheter over the guidewire. Advance the catheter over the guidewire until

the hub of the catheter is against the skin (**Figure 72-11*E***). **A rotating motion of the catheter is often helpful to advance it if difficulty is encountered.** Securely hold the catheter at the level of the skin. Remove the guidewire while firmly holding the catheter hub against the skin (**Figure 72-11*F***). Free-flowing pulsatile blood confirms proper catheter placement within the artery. Apply a stopcock or intravenous extension tubing to the hub of the catheter. Secure the catheter by suturing it to the skin. Apply a dressing to the site.

RADIAL ARTERY CANNULATION: SELDINGER-TYPE, DOUBLE ARTERIAL WALL PUNCTURE

While localization and needle puncture of the artery are identical in all three techniques, commonly encountered difficulties include threading of the catheter with the first technique and threading of the guidewire into the vessel lumen with the second technique. Blood may enter and rise through the needle if the needle orifice is merely at the vessel edge. The catheter may get hung up outside the lumen with the catheter-over-the-needle technique when trying to advance it.[31] The guidewire may not easily pass or may dissect into the vessel wall creating a false lumen when using the Seldinger-type single arterial wall puncture technique. Pulsatile flow is more easily confirmed prior to the passage of the guidewire. This third technique in which both walls of the radial artery are punctured may offer an advantage in passing the guidewire and catheter into the arterial lumen.

Assume a position out of the trajectory line of the catheter to avoid being sprayed with blood when using this technique. This method is a Seldinger-type technique that uses a guidewire separate from the catheter-over-the-needle. Locate the arterial pulse and insert the catheter-over-the-needle into the artery. Immediately and continuously advance the catheter-over-the-needle through the posterior wall as confirmed by no blood exiting the needle hub. Withdraw the needle while leaving the catheter in place through the artery. Flatten the angle of the catheter to the skin to less than 30°. Hold the tip of the guidewire poised at the hub of the catheter. Slowly withdraw the catheter with the nondominant hand. Promptly insert the guidewire through the catheter and into the artery when pulsatile blood flow is noted from the hub of the catheter. Advance the catheter over the guidewire and into the lumen of the artery. Withdraw the guidewire and apply a stopcock or intravenous extension tubing. Secure the catheter and apply a bandage or dressing.

This double-puncture technique for cannulation theoretically may be associated with greater vascular damage. Reserve this technique for instances in which the prior two arterial cannulation techniques have been unsuccessful. Investigators found no difference in complications when using this technique compared to other techniques.[32]

FEMORAL ARTERY CANNULATION

The femoral artery may be cannulated if the radial artery is inaccessible or attempts at cannulation are unsuccessful. A longer catheter, catheter-over-the-needle, and needle are required as the femoral artery is not superficial. Slightly abduct the patient's leg and identify the femoral artery pulse. Prep, drape, and anesthetize the skin and subcutaneous tissues. Maintain the fingers of the nondominant hand on the arterial pulse. Insert the needle or the catheter-over-the-needle at a 45° angle to the skin with the needle bevel pointing superiorly and into the femoral artery. The remainder of the technique is as described previously.

DORSALIS PEDIS ARTERY CANNULATION

The procedure for cannulation of the dorsalis pedis artery is the same as that described for the radial artery. Clean, prep, and plantar flex the foot. Do not plantar flex the foot more than 45° as this can stretch and occlude the artery. The needle angle of entry should be less than that used for the radial artery due to the superficial location of the artery. Insert it at 20° to 30° to the skin rather than 30° to 45°.

ULTRASOUND-GUIDED ARTERIAL CANNULATION

US can be used to guide arterial puncture or cannulation (**Figures 72-12 through 72-15**). It can be a rescue method if the palpation technique fails. It can be used initially to decrease the number of puncture attempts, to decrease the amount of time spent gaining access, and to decrease complications (e.g., arterial laceration and

A

B

FIGURE 72-12. Ultrasound-guided radial arterial puncture. ***A.*** Short axis image of the radial artery (*yellow circle*). ***B.*** Short axis image of the radial artery under compression. Note that it hardly compresses (*yellow circle*).

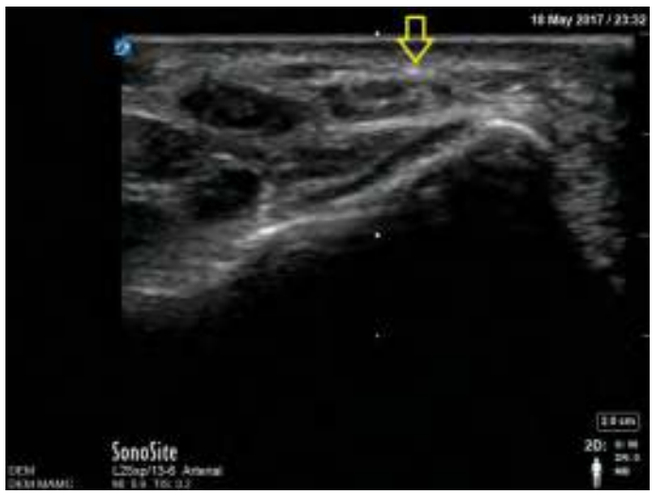


FIGURE 72-13. Ultrasound-guided radial arterial puncture. Short axis image of the radial artery with the needle approaching (*yellow arrow*).

FIGURE 72-15. Ultrasound-guided radial arterial puncture. Short axis image of the radial artery with a needle inside (*yellow circle*). Note the artifact from the needle.

hematomas).[11,12,33-41] The is some conflicting evidence that the use of US does not decrease the number of puncture attempts.[42,43] Most studies have focused on radial artery cannulation, but the technique for alternative sites is the same.[44] US as an adjunct to radial artery cannulation is considered best practice by some experts.[35]

Clean and prep the skin over the artery. Prepare the US transducer. Have an assistant apply US gel over the footprint of the transducer. Instruct the assistant to place the gel-coated US transducer in the sterile cover while you hold it. Have the assistant place sterile ultrasound gel over the target artery. Orient the transducer marker on the side of the transducer so it matches that on the screen. Identify the artery. It will appear as a round, thick-walled vessel that is not easily compressible and exhibits a pulsatile nature when compressed (**Figure 72-12*A***). Move the transducer proximally and distally over the artery to approximate its course, which is sometimes not perfectly parallel to the long axis of the extremity. Use Doppler flow to assist identifying arterial structures in low-flow or hypovolemic patients. Center the target artery on the screen. The center of the transducer is now directly over the vessel and can act as a guide to skin puncture. Hold the transducer with the nondominant hand or have an assistant hold the transducer.

Insert the catheter 1 to 2 cm distal to the transducer. Rock the needle in the subcutaneous tissue very slightly to create movement that is apparent on the screen. This will help to identify the location of the needle in relation to the artery. Slowly advance the needle once the tip of the needle has been identified (**Figure 72-13**). Move the transducer proximally and advance it until the needle tip is visible. This technique will help prevent advancing the needle beyond the view of the US and misidentifying the shaft of the needle as the tip. **The needle may not be apparent, but motion through the tissue can be used to infer the site of the needle tip.**

The needle will dimple the wall of the artery when it reaches the arterial wall. Advance the needle slowly while watching for the flash of blood in the needle hub. Continue to advance the needle into the artery (**Figure 72-14**). Use US to confirm placement of the needle and/or catheter within the artery (**Figure 72-15**). Proceed from this point with either the catheter-over-the-needle or the Seldinger-type technique to complete the procedure.

A

B

FIGURE 72-14. Ultrasound-guided radial arterial puncture. ***A.*** Long axis image of the radial artery between the yellow lines. ***B.*** Long axis image of the radial artery with the needle inserted (*yellow arrow*).

ALTERNATIVE TECHNIQUE

A cutdown technique may be performed to cannulate an artery. It is primarily used for the brachial, femoral, or radial arteries but may be used to access other peripheral arteries. This technique is rarely required with the availability of US to locate an artery. The technique is briefly described. Please refer to Chapter 69 for the complete details.

Clean, prep, anesthetize, and drape the skin overlying the chosen artery. Make a 1.5 to 2.0 cm transverse skin incision centered over the artery. **Do not cut into the subcutaneous tissue so that blood vessels, nerves, and tendons are not transected.** Spread the subcutaneous tissues parallel to the artery with a mosquito hemostat. Expose 1.0 cm of the length of the artery. Pass a silk suture under the proximal end of the exposed artery. Insert a catheter-over-the-needle through the skin just distal to the incision and advance it into the incision. Elevate the suture to control the artery and occlude distal blood flow. Advance the catheter-over-the-needle into the artery. Release the suture and advance the catheter into the artery. The remainder of the procedure is as described previously. Apply pressure over the incision site for 5 to 10 minutes to prevent the formation of a hematoma or seroma.

AFTERCARE

Apply direct pressure for 3 to 5 minutes to the skin puncture site after an arterial puncture or removal of an arterial catheter. Apply direct pressure for 10 minutes or more if the patient has a bleeding diathesis or has received thrombolytic therapy. Apply a bandage or gauze dressing to the skin puncture site. Reassess the skin puncture site in 15 minutes for continued bleeding or hematoma formation.

An arterial catheter must be secured to the skin to prevent inadvertent dislodgement, hematoma formation, and exsanguination. Sew the hub of the catheter to the skin using 3–0 or 4–0 nylon sutures. Apply a clear protective dressing (e.g., Tegaderm) over the site that will allow for monitoring of the cannulation site. Use a splinting device to secure the limb in the desired position for optimal monitoring if the catheter is in the wrist, arm, or foot (**Figure 72-5**).

Monitor the site regularly to assess for signs of arterial thrombosis, bleeding, catheter dislodgement, hematoma formation, or infection. Assess the extremity distal to the catheter for evidence of ischemia. Replace the dressing regularly in accordance with institutional guidelines.

Flush the arterial catheter with sterile saline solution. Heparinized saline was the standard arterial line flush solution in the past.[45] The use of heparinized saline has not been shown to improve functionality, prevent thrombotic complications, or increase the duration of catheter patency when compared to saline.[45] The use of heparin risks an adverse reaction or heparin-induced thrombocytopenia.

COMPLICATIONS

Arterial puncture and catheterization are generally safe procedures with an incidence of clinically significant complications of less than 5%.[2] The primary complications of arterial catheterization are arterial injury, arterial thrombosis, bleeding, and infection.[3,4,46-54] Secondary bacteremia and septic emboli may occur, but infection is usually limited to the site of catheterization. The risk of catheter site infection is linearly related to the length of time the catheter is in place.[3] The infection rate is similar for femoral and radial artery sites.[3,8,9,48,55] Nerve injury from direct puncture of the nerve has been reported.[56] Multiple cases of neuropathy have occurred from a hematoma formation and subsequent nerve compression.[21] Apply at least 3 minutes of direct pressure to radial, brachial, and dorsalis pedis puncture sites and 5 to 10 minutes of pressure to the femoral site to minimize hematoma formation after arterial puncture or arterial catheter removal.[56,57] Small hematomas are common after arterial puncture. Major bleeding is unusual and generally occurs only in the concealed retroperitoneum after femoral artery puncture or cannulation.[58] Angiographically demonstrable thrombosis is common in 25% to 40% of patients after prolonged arterial catheterization.[3] This rarely results in clinically significant morbidity. Secondary limb ischemia and necrosis requiring amputation occur in less than 1 in 2000 arterial catheterizations.[3,4,13,59] Other rare complications include the formation of an arteriovenous fistula or pseudoaneurysm.[38,39,60]

The complications associated with an arterial catheter can be limited.[13,61] Only insert an arterial catheter if it is truly needed. Always insert it using strict aseptic technique. Remove the catheter as soon as it is determined that it is no longer required to assist in the management of the patient. Frequently check the skin puncture site for signs of bleeding, a hematoma, and an infection. Frequently assess the distal extremity for signs of perfusion. Remove the catheter at the first sign of a complication. Place the catheter in the patient's nondominant hand, if known, to limit the impact of any complications.

SUMMARY

Arterial puncture and cannulation are quick, safe, and simple to perform. Arterial blood sampling may aid in determining the acid-base status and ventilator management of critically ill patients. An arterial catheter is appropriate in patients who need continuous monitoring of arterial blood pressure or frequent ABGs or in patients in whom indirect blood pressure monitoring is not possible. Avoid arterial puncture and cannulation in skin areas with evidence of burn, infection, severe dermatitis, severe peripheral vascular disease, or trauma. Palpation of the arterial pulse and correct anatomic positioning are necessary before these procedures are attempted. It is necessary to monitor the cannulated site for signs of bleeding, hematoma, infection, or thrombosis. Always apply direct pressure to the skin puncture site after an arterial puncture or removal of an arterial catheter.

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73 Extracorporeal Membrane Oxygenation

Jean W. Hoffman and Jordan R. H. Hoffman

INTRODUCTION

Extracorporeal life support (ECLS) has been used as a rescue therapy for patients presenting with acute cardiopulmonary failure. The use of ambulatory extracorporeal support parallels the growth and development of cardiopulmonary bypass during open heart surgery over the past 40 years. The first successful use of a cardiopulmonary bypass circuit occurred in the early 1950s.[1] It was not until 1971 that an extracorporeal membrane oxygenator was used for an extended period to support a patient with "shock-lung syndrome."[2] Portable extracorporeal membrane oxygenation (ECMO) was used in the late 1970s to rescue 39 patients with a variety of conditions (e.g., cardiac trauma, cardiogenic shock after myocardial infarction, drug overdose, and massive pulmonary embolus).[3] Portable cardiopulmonary support was largely abandoned due to poor outcomes after early reports.

Literature supporting the initiation of ECMO in adult medical patients comes from two modern trials examining outcomes in patients with acute respiratory distress syndrome (ARDS) and refractory cardiac arrest.[4,5] The literature supporting the use of ECMO remains sparse with only scattered case reports published over the past 10 years. Hesitancy to embrace ECMO as a rescue therapy in the adult surgical patient has been partially due to the need for therapeutic anticoagulation to maintain the bypass circuit. There are an increasing number of surgical patients being treated with "high-flow, no-heparin" ECLS to improve survival.[6]

Survival after initiation of ECMO is closely correlated with the indication for which ECLS was initiated.[7] The prognosis is related to baseline functional status, medical comorbidities, operative history, and the duration of ECLS.[8,9] These and other factors (e.g., body habitus, multiorgan failure, neurologic status, or terminal conditions) may influence outcomes prior to cannulation. **The Emergency Physician should anticipate the goals of therapy.** Will ECLS be used as a bridge to treatment (e.g., percutaneous intervention for coronary disease), as a bridge to recovery (e.g., severe ARDS), or as a bridge to transplant? These concepts are occasionally not mutually exclusive and are accomplished jointly. ECLS may be contraindicated if the underlying problem is not reversible with the help of ECMO.

Approach cannulation for ECLS in the Emergency Department cautiously. ECLS should only be performed by Emergency Physicians with experience in its initiation, maintenance, and discontinuation.[7] Multiple organizations offer training courses in ECMO cannulation and management. The Emergency Physician can sterilely place central venous and arterial lines while waiting for the Cardiothoracic Surgeon to arrive for cannulation (Chapters 63 and 72).[10,11] The Emergency Physician can initiate and perform ECMO for the patient in cardiac arrest.[10-13] It is up to individual hospitals to maintain up-to-date records for purposes of tracking outcomes and Emergency Physician credentialing.

Guidelines for the development of an ECMO program are beyond on the scope of this chapter but can be found through the Extracorporeal Life Support Organization (ELSO) website.[14] An ECMO program requires coordination between multiple departments and specialists so resource allocation occurs efficiently and justly. Successful ECMO programs can balance optimal care with the ability to provide advanced therapies to patients in need.

ANATOMY AND PATHOPHYSIOLOGY

CARDIAC FAILURE

Heart failure is the inability of the heart to propel blood at a rate equal to the metabolic needs generated by peripheral tissues. It can be classified by the presence or absence of a reduced ejection fraction, the ventricle affected (i.e., right heart failure, left heart failure, or biventricular failure), and the chronicity (i.e., acute, chronic, or acute-on-chronic).

The normal heart maintains several adaptive mechanisms to compensate for chronic decompensation. These mechanisms include enhanced contractility with activation of neurohumoral responses (e.g., the release of epinephrine and norepinephrine, activation of the renin-angiotensin-aldosterone system, or release of natriuretic peptides), compensatory structural changes (e.g., muscular hypertrophy), and rising preload (i.e., Frank-Starling mechanism).[15]

Acute heart failure is caused by conditions in which the heart is abruptly presented with a load that exceeds its capacity to function or filling is impaired. Chronic heart failure occurs because of a slowly developing intrinsic defect in myocardial contraction.[15] The indications for veno-arterial (V-A) ECMO cannulation in the Emergency Department are limited to acute or acute-on-chronic heart failure.

RESPIRATORY FAILURE

Respiratory failure is defined as an inability to exchange oxygen across the capillary and alveolar membranes, leading to hypoxemia. Respiratory failure can be classified by its chronicity (i.e., acute, chronic, or acute-on-chronic). It is broadly attributed to extrinsic compression, lower airway disease, neuromuscular disease, upper airway disease, or vascular occlusion. Indications for veno-venous (V-V) ECMO cannulation in the Emergency Department are limited to acute or acute-on-chronic respiratory failure.

ARDS remains the most well-studied indication for V-V ECMO. The conditions leading to the development of ARDS are numerous (**Table 73-1**).[15-37] The causes of ARDS share a similar final pathophysiologic pathway that culminates in activation of proinflammatory mediators, formation of a hyaline membrane impairing oxygen diffusion across the capillary-alveolar interface, pneumocyte destruction, and sloughed bronchoalveolar epithelium (**Figure 73-1**).[28]

TABLE 73-1 Conditions Leading to ARDS*

Acetylsalicylic acid	Lung irritant chemicals
Barbiturates	Lung irritant gasses
Burns	Lung irritants inhaled
Cardiopulmonary bypass	Methadone
Diffuse lung infections	Near-drowning
Miliary tuberculosis	Organic solvents
Mycoplasma	Oxygen toxicity
Pneumocystis	Pancreatitis
Viral	Paraquat
Disseminated intravascular coagulation	Poisoning
Drugs	Pulmonary contusion
Fat embolism associated with fractures	Sepsis
Gastric aspiration	Smoke
Heroin	Transfusion-associated lung injury
Hypothermia	Trauma
Hypothermia rewarming	Uremia
Ionizing radiation	

*Many of these are associated with the development of ARDS.

Source: References 15-37.

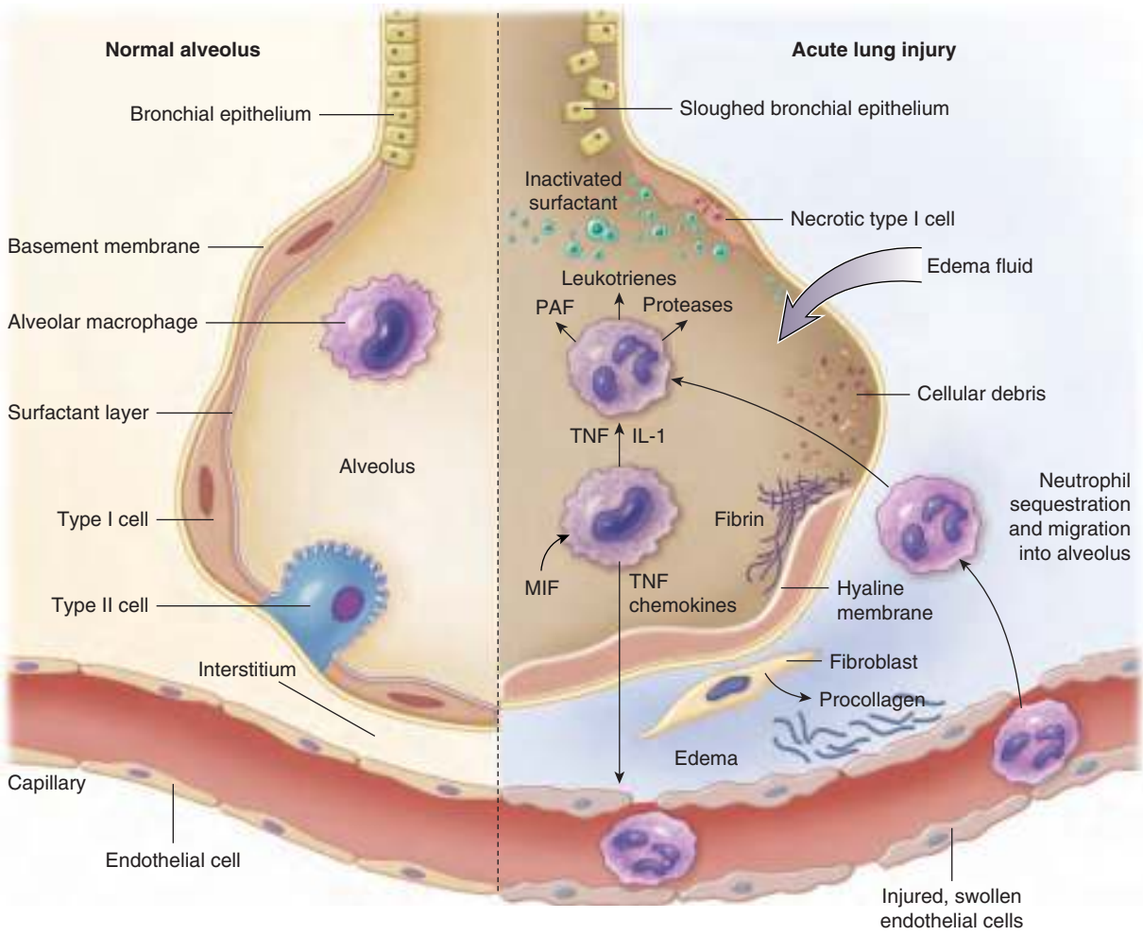


FIGURE 73-1. The normal alveolus (left) and the injured alveolus (right). IL-1, interleukin-1; MIF, migration inhibitory factor; PAF, platelet-activating factor; TNF, tumor necrosis factor. (Used with permission from reference 15.)

ECMO

ECMO circuits are composed of an oxygenator or artificial lung, a gas blender, a heat exchanger, cannula and tubing, monitoring equipment, and a blood pump (**Figure 73-2**). The gas blender mixes different concentrations of air, carbon dioxide, and oxygen and forces the mixture to the oxygenator. The oxygenator mixes the patient's blood in the ECMO circuit with the gas from the blender and oxygenates the blood.

Cannulation strategies can be peripheral or central. Peripheral cannulation requires placement of a drainage cannula in the femoral or jugular vein to remove blood from the body. Oxygenated blood can be returned via a return cannula to either an artery (e.g., V-A ECMO) or a vein (e.g., V-V ECMO). V-V ECMO relies on a working heart to deliver oxygenated blood to the patient's circulatory system. V-A ECMO bypasses the heart, and the ECMO circuit provides delivery of oxygenated blood to the patient's circulatory system. Central cannulation is the placement of drainage and return cannulas into a central artery or central vein. It requires the performance of a median sternotomy or thoracotomy. Additional cannulation strategies can include a combination of central and peripheral cannulation.

Blood flows of up to 8 L/min can be delivered under ideal circumstances using a centrifugal pump. Human physiology is described by Poiseuille's law. It dictates that flow within the ECMO circuit is a function of the radius of the cannulas chosen, length of the ECMO circuit tubing, viscosity of the blood, and pressure gradient generated by the centrifugal pump. **Make every effort to choose cannulas that will maximize flow. The flow is commonly the limiting factor to delivery of oxygenated blood via the return cannula.** The operator typically sets a desired pump rotation per minute (RPM) or speed that determines the blood flow within the circuit and to the body. Flow can be measured directly off the tubing for confirmation.

ECMO is designed for a longer duration of use compared to cardiopulmonary bypass. There is no reservoir in ECMO. All products must be given as a transfusion through a separate line. The physiologic monitoring of the ECMO circuit is complex and will be discussed throughout this chapter.

VENO-VENOUS ECMO

INDICATIONS

V-V ECMO is indicated in adults, children, and pregnant patients for acute respiratory failure or acute-on-chronic respiratory failure causing high airway pressures, refractory hypoxemia, and refractory hypercarbia (**Table 73-1 and Figure 73-2*B***).[15-37] The most widely accepted indication for V-V ECMO is ARDS refractory to maximal medical therapy. Determining which patients with ARDS should receive ECMO can be difficult. Trials have incorporated the lung injury score (i.e., the Murray score) to attempt to provide a uniform indication for consideration of ECMO.[38]

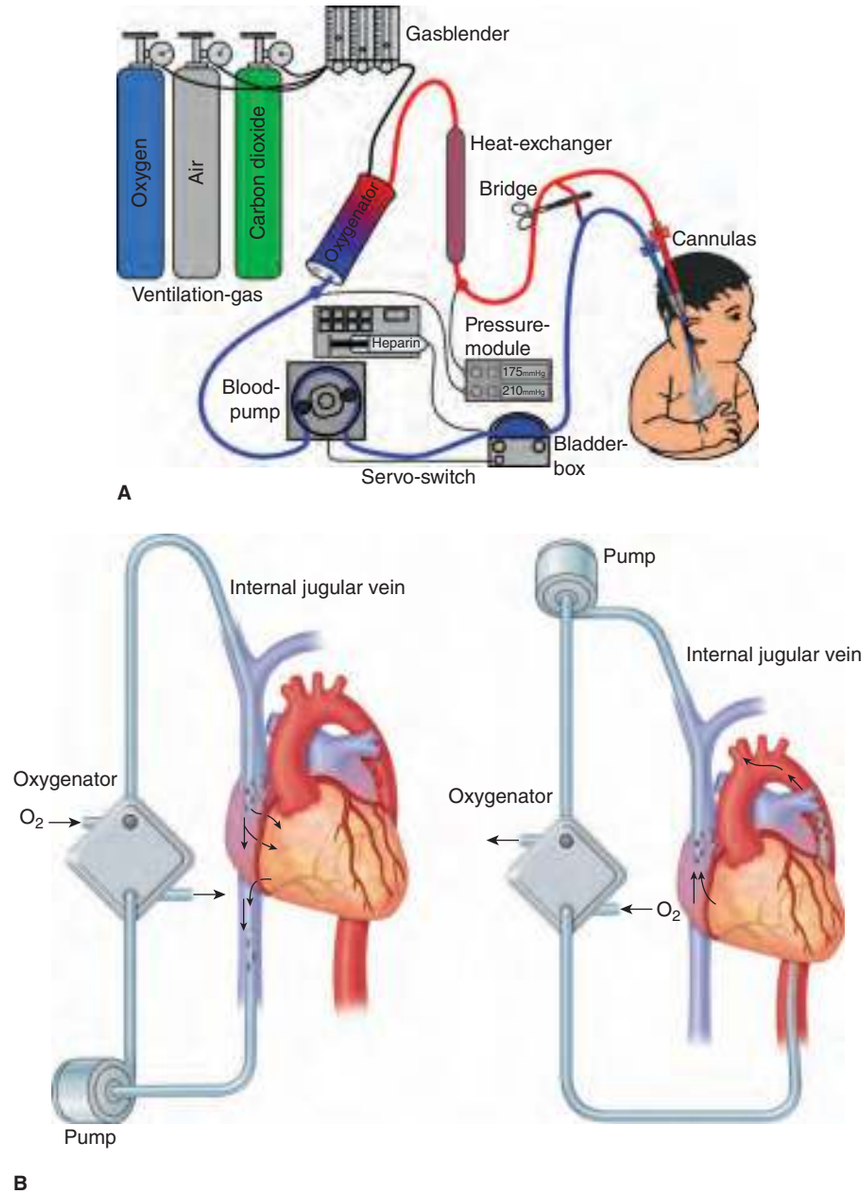


FIGURE 73-2. ECMO. ***A.*** An example of a circuit. (Used from Jurgen Schaub, https://commons.wikimedia.org.) ***B.*** Cannulation for V-V ECMO (left) and V-A ECMO (right). (Photo used with permission from reference 28.)

The lung injury score was designed to characterize the presence and extent of pulmonary damage in ARDS.[38] The score incorporates airway pressures, oxygenation, and radiographic findings (**Table 73-2**). Ventilation is not a component of this scoring system. Higher scores indicate higher ARDS-specific mortality. A lung injury score greater than 3 in the CESAR trial indicated a potential survival benefit with the addition of V-V ECMO compared to optimal medical management.[4] V-V ECMO should be considered when the patient's lung injury score is 2 (i.e., a mortality of > 50%) and is indicated when the lung injury score is ≥ 3 (i.e., a mortality of > 80%).[14]

Virtually any condition leading to ARDS represents a potential indication for V-V ECMO (**Table 73-1**). Patients with chronic respiratory failure can be candidates for V-V ECMO as a bridge to transplantation. Initiation of V-V ECMO in this population is outside the scope of the Emergency Department. It should only be performed at institutions after consultation with the lung transplant team.

CONTRAINDICATIONS

An absolute contraindication to V-V ECMO initiation is an underlying disease process that is progressive and irreversible. Examples include chronic severe pulmonary arterial hypertension with evidence of right heart dysfunction, permanent neurologic damage, severely depressed functional status, and terminal malignancy.

Relative contraindications differ between institutions but typically include age greater than 75 years old, certain hemoglobinopathies, inability to be anticoagulated, an innate or pharmacologically induced immunocompromised state, multisystem organ failure, and recoverable central nervous system (CNS) injury. ECMO can be safely managed without anticoagulation in the short term depending on institutional policy.[21] Obesity can present a technical challenge to ECMO cannulation. The initiation and maintenance of ECMO in obese patients is feasible and safe.[39]

TABLE 73-2 The Lung Injury Score (i.e., Murray Score)

Component	Characteristic	Lung injury value
Chest Radiography	No consolidation	0
	Alveolar consolidation in 1 quadrant	1
	Alveolar consolidation in 2 quadrants	2
	Alveolar consolidation in 3 quadrants	3
	Alveolar consolidation in 4 quadrants	4
Hypoxemia	$PaO_2/FIO_2 \geq 300$	0
	PaO_2/FIO_2 225–299	1
	PaO_2/FIO_2 175–224	2
	PaO_2/FIO_2 100–174	3
	$PaO_2/FIO_2 < 100$	4
Positive end-expiratory pressure (when ventilated)	$\geq 5\ cmH_2O$	0
	6–8 cmH_2O	1
	9–11 cmH_2O	2
	12–14 cmH_2O	3
	$\geq 15\ cmH_2O$	4
Respiratory system compliance score (when available)	$\geq 80\ mL/cmH_2O$	0
	60–79 mL/cmH_2O	1
	40–59 mL/cmH_2O	2
	20–39 mL/cmH_2O	3
	$\leq 19\ mL/cmH_2O$	4

The final value is obtained by adding the values and dividing by the number of components used.
No lung injury, score = 0.
Mild-to-moderate lung injury, score = 0.1–2.5.
Severe lung injury (ARDS), score > 2.5.
Source: Modified from reference 38.

TABLE 73-3 ECMO Supplies

General Supplies
Bedside sterile trash bag
Half drapes (2)
Sterile bowels (2)
Laparotomy sponges
Bandaging tape (1 roll)
Sterile gowns
Sterile gloves
Needle counter
Ring basin
Side drapes, two
Scalpel with handles (#10, #11, and #15)
Syringes (10, 20, and 30 mL)
Bulb syringe
Table cover
Top drape
Sterile towels (10)
Suction tubing (2)
Double spike line
Tubing (1/4 and 1/8 inch)
Monitor extension tubing (6 inch)
4-way stopcock (2)
Double male Luer lock (2)
Umbilical tape, 30 inch (2)
Y connectors, 3/8 inch (2)
Straight connectors, 3/8 inch with Luer lock (2)
Straight connectors, 3/8 inch without Luer lock (2)
Zip ties (10)
Kelly clamps (4)
Hemostatic forceps (8)
Towel clamps (8)
Bag decanter
Tegaderm, 4 × 4 inch (2)
Gauze, 4 × 4 inch (10)
Betadyne
Chloraprep stick, large (2)
Saline bags, 1000 mL (2)
Needles, 14 to 27 gauge
Local anesthesia solution
Heparinized saline, 1000 mL, 2 U/mL

Access Supplies and Dilators
Ultrasound machine
Linear transducer
Radiology C-arm
Sterile C-arm drape
Micropuncture kit
Dilators

Wires and Sheaths
Cope Mandril starter wire, 60 cm (2)
Guidewire, 180 cm (2)
Amplatz super stiff guidewire, 10 cm (2)
Hi-torque steelcore guidewire, 300 cm (2)
Access sheaths: 5, 6, and 7 French (2 each)
Flexor Check-Flo 10 French introducer sheath (2)
Super Sheath XL 14 French introducer catheter (2)
Percutaneous insertion kit with dilators (2)
Avalon insertion kit, 100 and 210 cm (2 each)

Cannulas
Avalon cannula, 23, 27, and 31 mm (2 each)
Femoral arterial cannula, 19–25 French (2 each)
Femoral venous cannula, 19–25 French (2 each)
Quickdraw venous cannula, 22 and 25 French (2 each)
Opsite arterial cannula, 16, 18, 20, and 22 French (2 each)

Suture and Hemostatic Agents
#1 Ethibond control release suture (8)
#0 silk ties, 30 inch (2 packs)
3–0 Prolene on RB-1 needle (6)
4–0 Prolene on C-1 needle (6)
5–0 Prolene on C-1 needle (6)
Disposable clip applier, medium and large (2 each)
Surgicel or another topical hematostatic

EQUIPMENT

Keep the required ECMO equipment in one area. It is beneficial to have multiple portable ECMO carts fully stocked and always available. Have the primed portable ECMO system with crystalloid solution ready prior to cannulation. The contents of a comprehensive and fully stocked ECMO cart can be found in **Table 73-3**.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Discuss the goals of the ECMO procedure and outcomes. Immediate complications include cardiac perforation, damage to vessels or nerves, ECMO discontinuation or termination, embolic events, failure to wean from ECMO, life-threatening hemorrhage (e.g., cannula site bleeding, gastrointestinal bleeding, or intracranial hemorrhage), limb ischemia, local or systemic infection, need for further cannulation procedures, and death. Discuss the need for tracheostomy placement as an anticipated outcome. Obtain an informed consent for the procedure, and place this in the medical record.

Prepare for the procedure. Evaluate the echocardiogram data, radiology imaging, and laboratory values prior to cannulation. These results may alter the proposed plan. Position the patient to maximize the exposure of the vessels to be cannulated. Perform a "time out" to confirm the patient and the proposed procedure. Tuck the patient's arms at their side unless additional arterial monitoring via the arm has not yet been established. Clean the skin at the site of cannulation of any dirt and debris. Apply chlorhexidine or povidone iodine solution and allow it to dry.

This procedure must be performed using strict sterile technique. Don full personal protective equipment (i.e., a hat, mask, eye protection, sterile gown, and sterile gloves) to protect from exposure to the patient's blood and to protect the patient from an iatrogenic infection. Apply sterile drapes to delineate a sterile field. Administer a heparin bolus before or after cannulation followed by a continuous heparin infusion unless contraindicated. Monitoring and goal parameters differ between institutions and are discussed below.

Prepare the ultrasound transducer. Have an assistant apply ultrasound gel over the footprint of the transducer. Instruct the assistant to place the gel-coated transducer in the sterile cover while you hold it. Have the assistant place sterile ultrasound gel on the patient or transducer.

TECHNIQUE

ECMO initiation can be divided into several phases including patient preparation, access, dilation, cannulation and connection, initiation, and follow-up care. The use of the right internal jugular and right femoral veins are preferred for venous cannulation. These vessels tend to travel along a straighter course with the superior and inferior vena cava (Chapter 63 and **Figure 63-1**). Review any available imaging for variant anatomy or in situ venous thrombus, which may complicate catheter placement. The following technique can be generalized to all peripheral venous access sites. The technique describes one site, but two are required. The second site is cannulated like the first. The technique of obtaining

venous access is briefly described below. Refer to Chapter 63 for the complete details.

ACCESS

Begin by preparing the access site as described above. Infiltrate the dermal and subcutaneous tissues with local anesthetic solution in the areas of cannulation. Connect the micropuncture needle to a syringe. Penetrate the anterior wall of the vein at a 45° angle under ultrasound guidance (Chapter 64). **Enter the vein anteriorly.** Entrance to the vein from the side or a double wall stick can complicate passage of large dilators and cause hemorrhage once anticoagulation is initiated. Advance the needle into the vein (**Figure 63-10*A***). The flow of blood indicates that the needle tip is within the vein. Hold and stabilize the needle. Carefully disconnect the syringe from the needle (**Figure 63-10*B***). It is not uncommon to have no blood return once the negative pressure of the syringe is removed.

Pass the guidewire from the micropuncture set through the needle and into the lumen of the vein (**Figures 63-10*B*** and **63-10*C***). The guidewire should pass smoothly. Accurate placement can be confirmed with ultrasound or fluoroscopy. Remove the needle while securely holding the guidewire. Make a small skin incision with a #11 scalpel blade next to the guidewire (**Figure 63-10*D***). Thread the dilator over the guidewire until its hub is against the skin (**Figure 63-10*E***). A twisting motion of the sheath may be needed to insert the sheath. Hold the hub of the dilator against the patient's skin. Remove the guidewire. Insert the 60 cm wire through the dilator. Hold the wire securely and remove the dilator.

Make a skin incision with a #11 scalpel blade large enough to allow passage of cannulas of up to 28 French. Place a size 5, 6, or 7 French access sheath over the wire while maintaining a hold on the wire (**Figure 63-10*F***). Insert the sheath until its hub is against the skin. A twisting motion of the sheath may be needed to insert the sheath. Remove the wire while securely holding the sheath (**Figure 63-10*G***). Apply a syringe of saline to the Luer lock of the sheath. Aspirate the sheath and then flush it to free the sheath of air bubbles (**Figure 63-13**). Secure the sheath to the skin with suture. The access sheath can act as a central venous line for fluid and medication delivery until ready to proceed with dilation.

DILATION

Insert the 180 cm stiff wire through the back the access sheath. Excessive manipulation of this wire may cause a breakdown of the seal between the sheath and wire, leading to bleeding onto the surgical field. Securely hold the wire. Remove the access sheath. Hold pressure above and below the skin puncture site with the nondominant hand while stabilizing the wire and occluding the vessel. **It is essential to always remain in control of the wire.** Unintentional loss of the wire can lead to loss of access, accidental vessel or cardiac perforation, or malposition of the final cannula.

Dilate the tract and vessel serially using vascular dilators (**Figures 73-3 and 73-4A**) over the wire until a dilator 2 French sizes smaller than the chosen ECMO cannula size is used. For example, dilate to a 20 French transitional dilator if a 22 French cannula is planned. The wire should move freely through each dilator indicating no kinking of the wire or vessel wall injury. There should be only minimal resistance with passage of the dilators. **Never force the dilator into a vessel. Maintain firm pressure over the vessel between dilations to ensure no hemorrhage or hematoma formation.** Remove the dilator and repeat the dilation of the tract with a smaller dilator if resistance is met. Check the skin incision to ensure that there is no intervening skin bridge and that the incision is of adequate length to accommodate larger dilators.

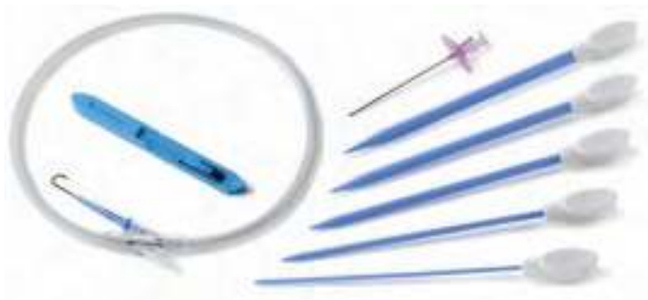
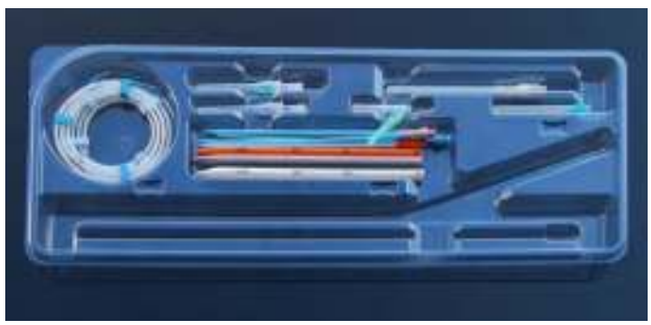


FIGURE 73-3. Examples of vascular dilators.

CANNULATION AND CONNECTION

This is the next step once the tract and vessel are successfully dilated. Apply and hold pressure with the nondominant hand as the last dilator is removed. Insert the stiff support catheter supplied with the ECMO cannula into the back of the ECMO cannula. Flush the entire system with saline. Apply the venous ECMO cannula over the wire. Advance the ECMO cannula over the wire. Insert it into the patient with constant forward pressure. Blood will escape through the multiple ECMO cannula side holes as the cannula is inserted. The bleeding will stop once the cannula is fully inserted. Flush the cannula free of air. Check the final position of the cannula with bedside ultrasound, fluoroscopy, or transesophageal echocardiography. Secure the cannula into place in at least three different sites along the length of the ECMO catheter with figure-of-eight sutures.

Connect the ECMO cannula to the ECMO system tubing with either straight or "Y" connecters depending on the chosen cannula



A

B

FIGURE 73-4. The equipment used for ECMO. ***A.*** A vascular access kit with dilators. ***B.*** Examples of the dual-lumen catheters. (Photos courtesy of OriGen Biomedical, Austin, TX.)

FIGURE 73-5. Examples of a straight connector and a Y connector.

configuration (**Figure 73-5**). The use of tubing connectors with Luer lock adapters allows removal of fine air bubbles through a syringe once the system has been connected. Apply zip ties to secure the tubing to the ECMO catheters and tubing connectors.

INITIATION

The two venous access sites have been cannulated and connected to the ECMO circuit. Have a nurse prepare vasopressors prior to ECMO initiation to neutralize large hemodynamic swings. Have fluid and blood products available in case of excess blood loss prior to or during the cannulation. It is helpful to have additional large-bore peripheral intravenous or central venous access for medication and fluid administration. Place an arterial cannula in the right radial artery for blood pressure monitoring if not inserted prior to ECMO cannulation (Chapter 72). Provide sedation and pain control during and immediately after ECMO cannulation and initiation. Initiate ECLS. The operator can titrate the pump speed to a desired flow while monitoring the patient's hemodynamics. Place the patient on ventilator settings to reduce the risk of barotrauma once hemodynamic parameters and oxygenation are confirmed to be acceptable (Chapter 36).

SPECIAL CIRCUMSTANCES

A dual-lumen ECMO cannula can be placed (**Figure 73-4*B***). It provides simultaneous venous drainage of deoxygenated blood and return of oxygenated blood via the right internal jugular vein. Dual-lumen catheters are designed to minimize recirculation of blood while allowing a patient to remain awake and ambulatory while on ECLS. Use fluoroscopy or transesophageal echocardiography for placement as the return port must be aimed at the tricuspid valve. The insertion technique is identical to the technique described above. The dual-lumen ECMO cannula has limited utility in the Emergency Department due to the precision required for insertion.

ASSESSMENT

Correct placement of venous ECMO cannulas is verified (**Figures 73-2*B* and 73-6**) with bedside ultrasound, transesophageal echocardiography, fluoroscopy, or radiography.[40-42] The distal end of the internal jugular venous cannula should rest at the junction of the superior vena cava and right atrium where it can deliver oxygenated blood to the right atrium. The distal end of the femoral venous cannula should lie in the inferior vena cava below the takeoff of the hepatic veins. Draw and analyze an arterial blood gas (ABG). The ABG goals include a pH of 7.2 or greater, a PaO_2 of 50 mmHg or greater, and a $PaCO_2$ of 50 mmHg or less. Arterial saturations may approach 80% to 85% depending on the amount of recirculation.[14] This is acceptable for systemic oxygen delivery assuming a normal cardiac output and hemoglobin level.[14]

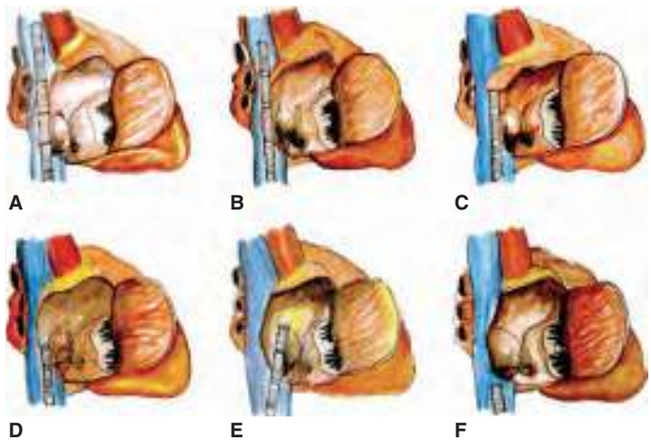


FIGURE 73-6. Drainage cannula positions. Optimal positioning is when the tip of the drainage cannula is in the superior vena cava as noted in parts A-C. ***A.*** ≥ 2 cm from the right atrium. ***B.*** ≤ 2 cm from the right atrium. ***C.*** In the right atrium ≤ 2 cm from the superior vena cava junction. ***D.*** In the right atrium ≥ 2 cm from the superior vena cava junction. ***E.*** Against the interatrial septum. ***F.*** In the inferior vena cava. (Used with permission from reference 40.)

AFTERCARE

Regular monitoring of patient data is essential while on ECMO. Normal hemodynamic values, laboratory data, and initial ECMO settings can be found in **Table 73-4**.[43] Titrate the oxygen by adjusting the ECMO flow. Flow is initially set to a maximum of 50 to 80 mL/kg/min and titrated down to maintain pulse oximeter saturations greater than 80% while on the ventilator. This should correlate with an oxygen delivery (DO_2) to consumption (VO_2) ratio of greater than 3 or a mixed venous oxygen saturation (SvO_2) that is 30% lower than a measured arterial oxygen saturation (SaO_2).

Clearance of carbon dioxide (CO_2) is controlled by the sweep gas flow. Blood flow to support oxygenation can cause excessive CO_2 removal if ECMO is initiated for hypoxemia. Titrate the sweep gas flow to maintain a $PaCO_2$ of 40 mmHg. Blood flow can be maintained at a low level while the sweep gas is titrated up if ECMO is initiated for hypercarbia (e.g., chronic obstructive pulmonary disease or asthma exacerbation).

Recirculation is a phenomenon where freshly oxygenated blood is being shunted from the return cannula to the drainage cannula due to their proximity or malposition. A continuous central venous

TABLE 73-4 Initial Settings for ECMO

Characteristic	Setting or goal
Arterial CO_2 tension	35–45 mmHg
Arterial O_2 saturation	V-V ECMO: 85–92%
	V-A ECMO: > 95%
Circuit flow	50–80 mL/kg/min
FIO_2	100%
Hematocrit	30–40%
Inlet pressure of centrifugal pump	> 100 mmHg
Mean arterial pressure	65–95 mmHg
Mixed venous O_2 saturation	> 65%
pH	7.35–7.45
Platelets	> 100,000
O_2 saturation of drainage cannula	> 65%
O_2 saturation of return cannula	100%
Sweep gas flow	50–80 mL/kg/min

Source: Modified from reference 43.

oxygen saturation ($ScvO_2$) is provided on most ECMO machines. This is a value measured from the blood in the return cannula as it exits the body. A mixed venous saturation greater than 65% to 70% is acceptable assuming an arterial saturation or hemoglobin saturation in the return cannula of near 100%. It may be an indication that significant recirculation is occurring if the $ScvO_2$ remains elevated. Correlate this finding with radiographs or ultrasounds of the cannula position. Adjust the cannula position accordingly.

Place the patient on "rest" ventilator settings once stabilized on ECMO. Set the ventilator in pressure control mode with a positive end-expiratory pressure of 5 to 15, an inspiratory pressure of 10, and a respiratory rate of 5. Adjust the fraction of inspired oxygen (FIO_2) to the lowest possible value while still maintaining pulse oximeter saturations greater than 80%. Rest ventilator settings vary by institution.

Provide deep sedation during and immediately after cannulation. Wean the sedation as the patient's condition improves so that they can breathe spontaneously and ambulate. The patient can assume control of their breathing while sedation is weaned because pressure control ventilation is time-triggered and pressure-triggered. Alternatively, the ventilator mode can be adjusted to provide only continuous positive airway pressure (CPAP) while the patient breathes spontaneously. **Make every effort to liberate a patient from the ventilator while on ECMO.** There may be situations where maintaining the patient on the ventilator in anticipation of ECMO weaning. Extubate the stable patient by ECMO day 5 or consider a tracheostomy.[14] A tracheostomy is an alternative if prolonged respiratory failure is expected.

Initiate all patients on anticoagulation using heparin unless contraindicated. Alternatives to heparin include argatroban and bivalirudin. There is limited data on therapeutic ranges and outcomes using alternative anticoagulation. Heparin can be titrated to achieve anti-Xa, activated partial thromboplastin time (aPTT), or activated clotting time (ACT) goal levels. Some institutions prefer a low-dose heparin protocol during V-V ECMO to reduce the rate of bleeding complications.

Monitor coagulation parameters (e.g., international normalized ratio [INR], partial thromboplastin time [PTT], and fibrinogen), hemoglobin levels, platelet levels, and serum free hemoglobin every 8 to 24 hours to assess the risk of bleeding, clotting, or hemolysis. Normal values will differ between institutions. Acceptable values are generally an INR less than 2, fibrinogen levels greater than 150 mg/dL, hemoglobin levels of 10 g/dL or greater, and platelet levels of 50,000/μL or greater. A serum free hemoglobin of greater than 40 mg/dL may indicate hemolysis. This test is unreliable when hyperbilirubinemia is present. Do not use lactate dehydrogenase as a marker for hemolysis. It is relatively nonspecific and may indicate excessive hemolysis, excessive clotting, or injury to other unrelated organs. Some institutions follow D-dimer levels as markers of clot burden within the membrane oxygenator.[44]

Daily assessments of each membrane oxygenator include visual inspection for clot burden. Clotting within the membrane oxygenator may present as white or red streaks that are typically located on the outskirts of the oxygenator. These should be comprehensively documented so that daily comparisons can be made between multiple teams. Additional oxygenator assessments should include preoxygenator and postoxygenator ABG measurements. ABG assessment should contain measurements of percent hemoglobin saturation, which can be used to calculate oxygen delivery (DO_2) and oxygen consumption (VO_2). Correlate flow measurement through drainage and return cannulas with the pump RPMs.

Complete a careful assessment of volume status at regular intervals. Observation of the ECMO circuit tubing may alert to hypovolemia within the ECMO circuit. Chugging and chatter are both terms used to describe the rhythmic shaking of the ECMO circuit tubing due to hypovolemia. Volume administration or decreasing pump flows will remedy this problem.

The process of weaning from extracorporeal support is beyond the scope of Emergency Medicine. Assess the ability to wean the patient daily. The primary indication that a patient is likely to wean successfully from ECMO is reversal of the underlying pathology. An example is ARDS. Expect improvement in radiographic findings, improvement in oxygenation and ABG values with minimal ECMO support, larger tidal volumes while breathing spontaneously, and normal hemodynamics without vasopressor support before weaning from ECMO. ECMO discontinuation is a complex process managed by a multidisciplinary team in an inpatient unit.

COMPLICATIONS

Patients are at risk for ECMO-related complications during initiation, maintenance, or discontinuation of extracorporeal support (**Table 73-5**).[43,45] The complications related to cannulation are like those for the placement of a central venous catheter (Chapter 63). The most common complications include arterial puncture and cannulation, bleeding, cardiac injury, limb ischemia, pneumothorax, and vascular injury. Mechanical complications, including pump thrombus, oxygenator failure, dislodged cannula, or accidental tubing disconnection, occur to lesser degrees.

Patients are at risk for gastrointestinal, intracranial, and catheter-related hemorrhage or thrombosis while ECLS is being maintained. Perform a head computed tomography scan for any acute change in the patient's mental status or neurologic examination. Check distal arterial pulses at regular intervals to assess for peripheral embolic events. Have a low threshold to check coagulation parameters, especially if the patient is experiencing bleeding. Mechanical circulation can cause an induced von Willebrand factor (vWF) deficiency. Consider a trial of cryoprecipitate, desmopressin, or antifibrinolytic amino acids if patients continue to bleed despite normal platelet levels and coagulation parameters, especially if concomitant renal failure is present.[46,47]

Infectious complications may occur. These include line-related sepsis, pneumonia, or a urinary tract infection. Some centers

TABLE 73-5 Complications of ECMO

Bleeding from cannulation site
Bleeding from gastrointestinal tract
Bleeding from surgical site
Bleeding from tracheostomy
Bleeding intracranially
Blood clots in hemofilter
Blood clots in oxygenator
Blood clots in pump
Blood clots in tubing
Cannulation-associated problems
Diffuse intravascular coagulation (DIC)
Encephalopathy
Hemolysis
Hyperbilirubinemia
Inability to wean
Infection
Intracranial bleeds
Limb ischemia
Multiple organ dysfunction
Oxygenator failure
Pump failure
Renal failure
Seizure
Stroke
Vascular access related problems

Source: Adapted from references 43 and 45.

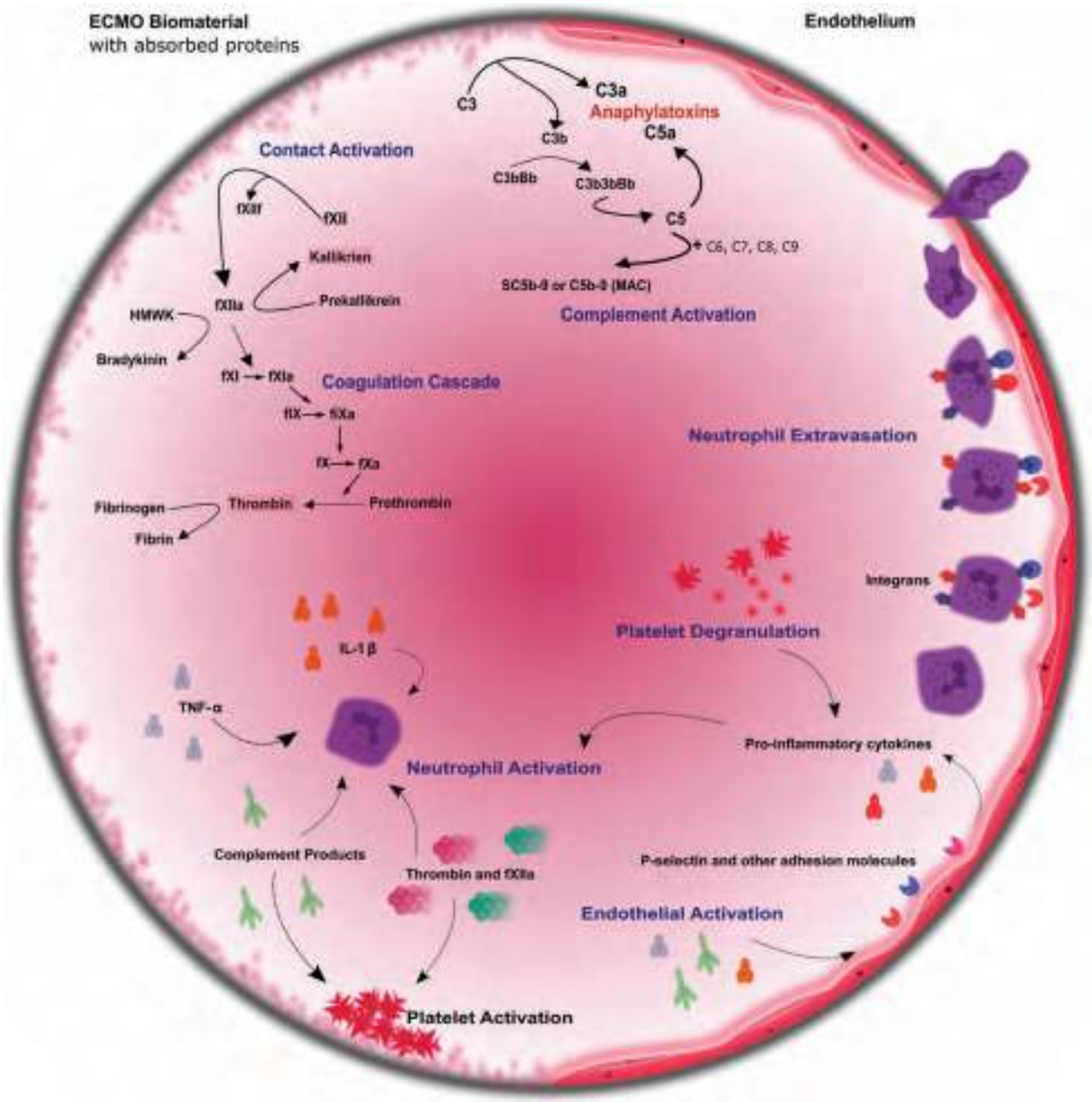


FIGURE 73-7. The inflammatory response to ECMO. (Used with permission from reference 49.)

advocate for routine prophylactic antibiotic administration while ECMO cannulas are in place. More data are needed before this becomes standard practice. Rule out an infection before attributing the symptoms to the ECMO circuit.

An infection needs to be determined versus inflammation (**Figure 73-7**).[48,49] Inflammation is a complication associated with ECMO. This response is similar to the systemic inflammatory response syndrome (SIRS) associated with an infection. The patient's blood in contact with the ECMO circuit results in activation of the complement system and other endogenous systems. This ultimately leads to neutrophil activation, tissue neutrophil infiltration, and organ damage.

ECMO can affect the pharmacokinetics of some medications.[50] Some medications are sequestered within the ECMO circuit. The volume of distribution is increased with the use of ECMO. This can reduce the concentration of medications by dilution. These can cause subtherapeutic medication levels in the patient's blood. A discussion of the individual drugs is beyond the scope of this chapter and usually managed in the Intensive Care Unit.

SUMMARY

V-V ECMO supports patients with a variety of conditions that lead to acute respiratory failure. Cannulation strategies employed primarily use a combination of femoral and internal jugular vein insertion sites. Understanding aftercare and complications will help the Emergency Physician troubleshoot most problems related to initiation and maintenance of ECMO.

VENO-ARTERIAL ECMO

INDICATIONS

V-A ECMO is indicated in adults, children, and pregnant patients for acute cardiac failure or acute-on-chronic cardiac failure leading to cardiogenic shock (**Figure 73-2*B* and Table 73-6**).[16,20,21,23,25,28,29,32-34,43,51-74] Acute cardiac failure is caused by conditions in which the heart is abruptly presented with a load that exceeds its capacity to function or when diastolic filling is impaired.

TABLE 73-6 Indications for V-A ECMO

Bridge to cardiac transplant
Bridge to ventricular assist device
Cardiogenic shock, ischemic
Cardiogenic shock, nonischemic
Cardiogenic shock from electrical storm
Cardiomyopathy
Congenital heart disease
Drug overdose
Extracorporeal cardiopulmonary resuscitation (ECPR)
Hyperkalemia
Hypokalemia
Hypothermia
Myocardial depression from sepsis
Myocardial infarction
Myocarditis
Near-drowning
Organ perfusion for donation after cardiac death
Pheochromocytoma circulatory collapse
Poisoning
Support for cardiac catheterization
Support after cardiac surgery
Support after cardiac transplantation
Support perioperatively
Witnessed cardiac arrest from ventricular fibrillation
Witnessed cardiac arrest from ventricular tachycardia

Source: Compiled from references 16, 20, 21, 23, 25, 28, 29, 32–34, 43, and 51–73.

A major advantage to temporary ECLS is the ability of the V-A ECMO circuit to partially offload the myocardium while it recovers. Hemodynamic values that may indicate circulatory support include a cardiac index (CI) below 2 L/min/m^2, a systolic blood pressure persistently below 90 mmHg despite maximal vasopressor or inotropic support with placement of an intraaortic balloon pump (IABP), or a lactic acidosis related to cardiogenic shock.[43]

Acute cardiac decompensation can be caused by ischemic or nonischemic insults to the myocardium. Nonischemic causes of acute cardiac failure include cardiomyopathy (i.e., idiopathic and viral), failure to wean from cardiac bypass (i.e., stunned myocardium), malignant dysrhythmias, pulmonary emboli, or toxic exposures.[24]

Cardiac arrest due to ischemia after myocardial infarction represents one of the primary indications for ECMO cannulation in the Emergency Department. This is sometimes referred to as extracorporeal cardiopulmonary resuscitation. Nonrandomized studies suggest that use of V-A ECMO in acute myocardial infarction complicated by cardiogenic shock improves in-hospital survival and reduces 30-day mortality when used in conjunction with revascularization.[75,76]

A bridge to transplantation or bridge to additional mechanical support (e.g., ventricular assist device) are unique indications not commonly encountered in the Emergency Department. A bridge to thrombectomy in pulmonary embolus may be considered in the Emergency Department. Additional indications for V-A ECMO are found in **Table 73-6**.

CONTRAINDICATIONS

Reversible acute heart failure usually requires temporary mechanical support until cardiac recovery occurs. Distinguishing between myocardial stunning and permanent myocardial injury present a major dilemma when considering a patient for temporary ECMO support.

V-A and V-V ECMO share many of the same contraindications. The ability to predict tissue recovery is variable in V-A ECMO, unlike in V-V ECMO. Acute cardiogenic shock and associated comorbid conditions may argue more heavily against ECMO initiation. An absolute contraindication to V-A ECMO initiation is an underlying disease process that is progressive and irreversible. Other absolute contraindications include permanent neurologic damage, severely depressed functional status, and terminal malignancy.

Acute severe pulmonary arterial hypertension with evidence of right heart dysfunction is not a contraindication to ECLS. V-A ECMO can provide some offloading of the myocardium. This is in contradistinction to V-V ECMO where pulmonary hypertension is a contraindication to cannulation.

The relative contraindications to V-A ECMO differ between institutions. Relative contraindications may exclude a patient from ECLS. These typically include age greater than 75 years old, certain hemoglobinopathies, immunocompromised states, inability to be anticoagulated, multisystem organ failure, and recoverable CNS injury. Obesity can present a technical challenge to ECMO cannulation. The initiation and maintenance of ECMO in obese patients are feasible and safe.[39] ECMO can be safely managed without anticoagulation in the short term depending on institution policy.[21]

EQUIPMENT

The equipment required is the same as that for V-V ECMO. The contents of a comprehensive, fully stocked ECMO cart should include supplies for both venous and arterial ECMO. An example of a typical ECMO supply cart can be found in **Table 73-3**. A perfusionist should be available to aid in cannulation and offer troubleshooting advice during initiation and maintenance of ECMO.

PATIENT PREPARATION

The consent, preprocedural data review, patient positioning, and patient preparation are the same as V-V ECMO. Prepare the equipment and ultrasound transducer as discussed previously. Additional risks include arterial dissection, extremity malperfusion, distal embolic event, or life-threatening hemorrhage. Systemically anticoagulate the patient before or after V-A ECMO cannulation. Goal monitoring values of anticoagulation will vary between institutions and should be reviewed prior to administration of anticoagulation.

TECHNIQUE

V-A ECMO initiation can be divided into several phases including patient preparation, access, dilation, cannulation and connection, initiation, and follow-up care. Review any available imaging for variant anatomy or in situ venous thrombus, which may complicate catheter placement. Multiple sites may be cannulated concomitantly, allowing ECMO to be initiated more expeditiously if multiple physicians with expertise in cannulation are present. Venous access for V-A ECMO is identical to that discussed for V-V ECMO.

Some physicians feel that percutaneous cannulation may lead to unnecessary complications due to improper placement. An open femoral artery exposure for cannulation may be performed if the cannulating physician is comfortable with this technique. **Percutaneous femoral artery cannulation remains the quickest and safest method for V-A ECMO cannulation in the Emergency Department and will be discussed below.**

ACCESS

Infiltrate with local anesthetic solution over the proposed access sites. Consider a percutaneous distal perfusion catheter. Place this catheter prior to placement of the larger femoral artery cannula. Do not delay the initiation of ECMO for this catheter. Placement of a perfusion catheter is discussed below under "Special Circumstances."

Locate the common femoral artery with an ultrasound linear transducer (Chapters 63 and 64). Use the micropuncture needle without an attached syringe to puncture the common femoral artery. The artery must be entered along the anterior wall using a front wall only technique to avoid unnecessary hemorrhage after anticoagulation. The access is the same as described for V-V ECMO.

DILATION

Percutaneous dilation of the subcutaneous tract and femoral artery is like that of the vein as described for V-V ECMO.

CANNULATION AND CONNECTION

Insertion of the catheter is like that of a venous catheter in V-V ECMO.

INITIATION

Connect the venous and arterial access sites to the ECMO circuit. Initiate A-V ECMO.

SPECIAL CIRCUMSTANCES

Placement of a percutaneous distal perfusion cannula has the theoretical advantage of avoiding limb malperfusion due to occlusion from the arterial ECMO cannula. This catheter is more easily placed prior to placing the much larger ECMO catheter. Place it into the superficial femoral artery under ultrasound guidance.

ALTERNATE TECHNIQUES

Additional access sites for V-A ECMO support include the axillary artery, central aorta, and innominate artery. These alternate cannulation sites require time-consuming dissections and are rarely performed outside the Operating Room. They are briefly mentioned for completeness. Axillary artery cannulation requires axillary artery exposure below the clavicle. An 8 mm polyethylene terephthalate (i.e., Dacron) graft is circumferentially sutured to a longitudinal opening made through the anterior artery wall once the artery is exposed. This graft is then tunneled laterally in the subcutaneous tissues to be connected to ECMO tubing over the right chest. Aortic and innominate artery cannulation requires the performance of a median sternotomy. The innominate artery is cannulated with a 10 mm Dacron graft sutured to a longitudinal opening in the anterior wall. This can then be tunneled to exit the subcutaneous tissue in the right upper quadrant where it is connected to the ECMO tubing. Aortic cannulation can be accomplished with a tunneled graft conduit or by direct arterial puncture. The chest can remain open and covered with a negative-pressure vacuum system or can be closed.

ASSESSMENT

Verify the correct placement of arterial and venous ECMO cannulas (**Figures 73-2*B* and 73-6**) with bedside ultrasound, transesophageal echocardiography, fluoroscopy, or radiography.[40-42] Arterial cannulas tend to be shorter in length than venous catheters (**Figure 73-8**). They will usually terminate in the common iliac artery or distal aorta. The distal end of the internal jugular venous cannula should rest at the junction of the superior vena cava and right atrium where it can deliver oxygenated blood to the right atrium. The distal end of the femoral venous cannula should lie in the inferior vena cava below the takeoff of the hepatic veins.

AFTERCARE

The aftercare of patients on V-A ECMO is like that of patients on V-V ECMO with minor differences. Key differences include the minimal contribution from recirculation, sedation, ventilator management, anticoagulation, and weaning. Patients on V-A ECMO are not at risk for recirculation given the nature of cannula placement. Take systemic, preoxygenator, and postoxygenator ABG measurements at least daily to assess DO_2, VO_2, and recovery of cardiac function.

FIGURE 73-8. The arterial catheters (red) are shorter than the venous catheters (blue). (Photo courtesy of Maquet Getinge Group, Hechingen, Germany.)

Sedation is maintained at a moderate level in patients on V-A ECMO. It may be difficult for patients to ambulate or participate in active recovery while on V-A ECMO due to the V-A catheter configuration. Ventilator settings can be minimized, but "lung rest" is not the primary goal in most cases of V-A ECMO. Discuss a tracheostomy with the patient's surrogate medical decision maker if prolonged intubation is expected.

The location of arterial ECMO cannulas puts the patient at high risk for embolization of blood clots to cerebral, coronary, peripheral, or visceral arteries. Most clinicians prefer anticoagulation strategies targeting higher ACT or aPTT goals. This lessens the risk of embolization but does not eliminate the problem. **Higher anticoagulation goals put patients at risk for hemorrhage.** Perform a thorough pulse and neurologic examination at regular intervals while a patient is on V-A ECMO. Any change should prompt an expeditious work-up.

Myocardial recovery is signaled by improvement in echocardiographic findings. V-V ECMO relies on radiographic improvement and laboratory values to indicate recovering lung function. The liberal use of cardiac echocardiography for V-A ECMO is indicated at least daily.

Stagnant deoxygenated blood ejected by a weak myocardium has little impact on systemic or cerebral oxygenation because the heart is bypassed during V-A ECMO. The heart's contractility intensifies and causes deoxygenated blood to be ejected more forcefully toward the head vessels and against the flow of the arterial return cannula in the femoral artery as cardiac function recovers.

The merging of mechanical and native circulation is a phenomenon known as a mixing cloud. A patient's heart could be sending deoxygenated blood into the cerebral circulation causing a neurologic catastrophe without monitoring. Causes of antegrade movement of the mixing cloud include strengthening of myocardial contraction or a reduction in retrograde flow from the arterial return cannula. The mixing cloud will move from the ascending aorta to the aortic arch as the heart recovers. The first vessel to receive deoxygenated blood from the mixing cloud will be the innominate artery. Arterial blood gasses drawn from an arterial cannula placed in the right arm will be the first indication that myocardial contractility is recovering.

A cardiac thrombus can result from stagnant ventricular blood. This is more common in a very weak heart. The stagnation of blood

may occur to varying degrees at any time during A-V ECMO. A thrombus may be found on echocardiography or suspected clinically. Treatment of a ventricular thrombus is aimed at prevention and includes regularly scheduled chest compressions, inotropic support, or placement of an IABP.

Assess the ability to wean from ECLS daily. The primary indication that a patient is likely to wean successfully from ECMO is reversal of the underlying pathology. Expect improvement in echocardiography findings if the underlying cause of the patient's initial instability was reversed. ECMO discontinuation is a complex process managed by a multidisciplinary team in an inpatient unit.

COMPLICATIONS

V-A ECMO–specific complications are discussed in the aftercare section above and include cerebral, visceral, and peripheral embolic events, "mixing cloud" causing cerebral hypoxemia, and cardiac thrombus due to stagnation of bypassed blood. A complete list of complications related to ECMO is found in **Table 73-5.**[43,45]

SUMMARY

V-A ECMO will provide cardiac and pulmonary support. Cannulation strategies employed in the Emergency Department primarily use a combination of femoral vein and femoral artery insertion sites. Understanding aftercare and complications will help the Emergency Physician troubleshoot most problems related to initiation and maintenance of ECMO.

SUMMARY

ECMO provides temporary cardiopulmonary support for patients presenting to the Emergency Department in extremis. Percutaneous cannulation can be achieved rapidly and is straightforward once arterial and venous access is established. A comprehensive understanding of the care for the ECMO patient will allow the Emergency Physician to successfully navigate many of the acute problems and complications related to ECMO initiation and maintenance.

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74 Catheter-Based Hemorrhage Control

Matthew Rosen and Andrew Grock

INTRODUCTION

Nine percent of the global annual mortality is from trauma.[1] Trauma is the third leading cause of death in the United States for all ages and the leading cause of death in patients 1 to 44 years of age.[1] Hemorrhage is the leading cause of potentially preventable death, is amenable to interventions, and prevention of hemorrhage can reduce morbidity and mortality.[2,3]

Management of the patient with massive hemorrhage should primarily focus on the basics of resuscitation (i.e., airway, breathing, circulation, obtaining large-bore intravenous [IV] access, and activating massive transfusion protocols). Stopping blood loss is critical to patient survival.[4-6] Direct compression at the source of bleeding is usually sufficient. Other options include proximal control with the use of embolization, surgical closure, or tourniquets.[2,4] Hemorrhage can occur from noncompressible or not readily accessible sites (e.g., gastrointestinal tract, intraabdominal, intrathoracic, pelvic, or

retroperitoneal).[7] Limited management options exist for hemodynamically unstable patients due to noncompressible hemorrhage. It may prove dangerous to transport these patients to the Endoscopy Suites, Interventional Radiology (IR), or the Operating Room for definitive hemorrhage control.

Aortic occlusion is one hemorrhage control method that temporarily stops noncompressible torso hemorrhage from injuries that receive blood supply from the subdiaphragmatic aorta. Aortic occlusion improves hemostasis, preserves coronary and cerebral perfusion, and can improve hemodynamics enough to allow for definitive hemorrhage control by embolization, endoscopy, or surgical repair.[8-10] Aortic occlusion may allow more time for induction of anesthesia, intubation, initiation of positive-pressure ventilation, and operative planning.[11-16]

Emergency Physicians achieved aortic occlusion traditionally through a thoracotomy (Chapter 54) and supradiaphragmatic clamping of the thoracic aorta (Chapter 58).[17,18] Resuscitative endovascular occlusion of the aorta (REBOA) is a less invasive method of aortic occlusion made possible by advancements in endovascular technology.[17-20] Japan has incorporated catheter-based hemorrhage control in the treatment of solid organ and pelvic injuries. The published data from Japan's use of REBOA are limited and largely retrospective. The conclusions based on the Japanese data differ on REBOA safety and effectiveness.[21-24] A systematic review from data obtained in the United Kingdom showed benefits with REBOA utilization in the prehospital setting.[25]

Success with REBOA has been shown in animal models of intraabdominal, pelvic, and uncontrolled junctional (i.e., buttocks, extremity trauma not amenable to tourniquet use, gluteal, groin, and perineum) hemorrhage.[9,13,26,27] Case reports and series support its use in hemorrhagic shock from nontraumatic etiologies of bleeding (e.g., during repair of abdominal aortic aneurysm, ectopic pregnancy, massive hemorrhage after hysterectomy, postpartum hemorrhage, and upper gastrointestinal bleeding).[16,28-32] Clinical studies in trauma patients have shown similar promise for hemorrhage control.[12,33] The largest prospective trial to evaluate aortic occlusion in the United States for trauma patients showed that REBOA resulted in hemodynamic improvement in 67% of patients and achieved hemodynamic stability for greater than 5 minutes in 47%.[34]

ANATOMY AND PATHOPHYSIOLOGY

Aortic occlusion results in an increase in the mean arterial pressure (MAP) proximal to the occluded aorta and preserves perfusion to the brain and heart.[8,10] Metabolic derangements associated with loss of blood flow distal to the balloon can occur. Elevated lactate and other cytokines may have significant effects on patient physiology, especially after balloon deflation.[10,23] Models are being developed that allow for partial or intermittent occlusion of the aorta. These models aim to obtain a goal MAP proximal to the aortic occlusion while allowing perfusion distally, which may mitigate some of the deleterious effects of prolonged aortic occlusion.[35,36]

The aorta is separated into three functional zones for the purposes of REBOA (Figure 74-1).[37] Aortic zone I is from just distal to the left subclavian artery to immediately proximal to the celiac artery. Aortic zone II is from the celiac artery to the lowest renal artery. Aortic zone III is from just below the lowest renal artery to the iliac bifurcation. Zone I occlusion provides hemostasis from injuries receiving their blood from the celiac plexus, inferior mesenteric artery, superior mesenteric artery, and pelvic vessels (**Figure 74-2*A***). Zone III occlusion provides hemostasis from bleeding pelvic vessels and lower extremity injuries (**Figure 74-2*B***). **Avoid zone II as it has been considered an area where REBOA should be avoided due to the possibility of not occluding bleeding vessels and providing no advantages over zone I occlusion.**[12]

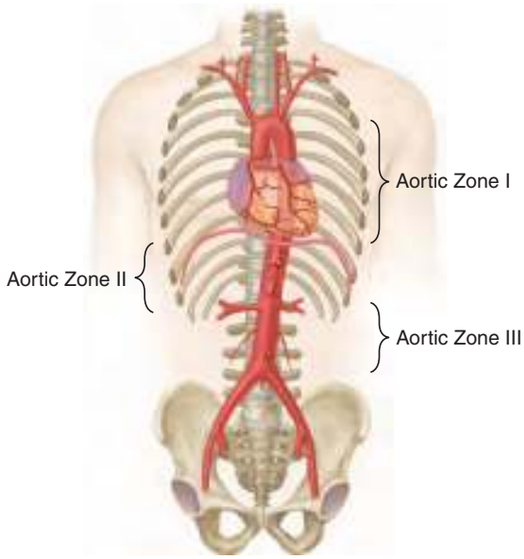


FIGURE 74-1. The functional zones of the aorta.

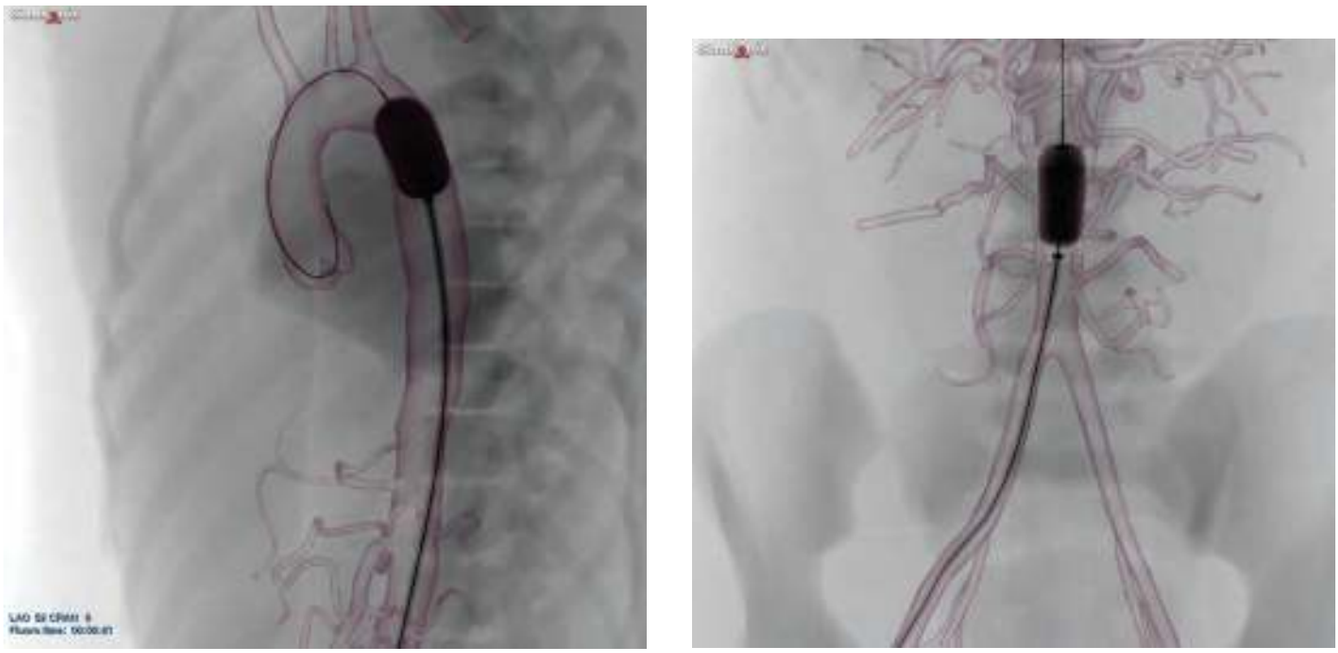
INDICATIONS

Currently, evidence-based indications for REBOA are poorly defined.[38] It can be performed for traumatic arrest from penetrating trauma to the abdomen, lower extremity, or pelvis with less than 15 minutes of cardiopulmonary resuscitation (CPR).[12,33,34,37,39] Traumatic arrest from blunt trauma with an organized rhythm on the electrocardiogram (ECG) or cardiac activity on focused assessment with sonography for trauma (FAST; Chapter 8) is another indication for REBOA.[12,33,34,37,39] Perform REBOA in cases of shock from blunt trauma with a positive FAST ultrasound (US) examination or pelvic fracture and without signs of thoracic aortic injury.[12,24-26,33,34,37,39,40] Shock from penetrating trauma to the abdomen, lower extremity, or pelvis is another indication for REBOA.[12,33,34,37,39] Patients with severe hemorrhage from nontraumatic sites are candidates for REBOA if the hemorrhage source receives its supply from the subdiaphragmatic aorta.[14-16,23,28,30-32] A proposed algorithm for the utility of REBOA in patients in hemorrhagic shock is shown in **Figure 74-3**.[19,33,37] REBOA is being proposed and studied for cardiac arrest unrelated to trauma or hemorrhage.[41]

REBOA may be advantageous for patients who are partial responders or nonresponders to resuscitation. It can be performed at the bedside in the Emergency Department without the risks associated with patient transport. Consider early femoral artery access in patients at high risk of developing hemorrhagic shock (Chapter 72). Obtaining arterial access is difficult in hypotensive patients or after cardiopulmonary arrest. A femoral arterial line is quickly converted to an appropriate sheath for the REBOA procedure. Perform REBOA in institutions where Interventional Radiologists, Surgeons, and staff are familiar with REBOA catheters including their inflation, deflation, and removal. Emergency Physicians in Japan perform REBOA frequently and proficiency as REBOA placement is a requirement for board certification.[42]

CONTRAINDICATIONS

Obtain chest radiography to examine the thoracic aorta prior to REBOA placement because the resulting increase in proximal MAP from REBOA could worsen a thoracic aortic injury. Use caution in patients with peripheral vascular disease or prior femoral artery procedures as their anatomy may be more challenging and increase the risk of complications.



A B

FIGURE 74-2. Examples of balloon catheter occlusion. ***A.*** Zone I. ***B.*** Zone III. (Photos courtesy of Simbionix, Israel.)

EQUIPMENT

There are several REBOA catheters, arterial access kits, and wires used for vascular procedures. The most commonly used current REBOA catheter is the Coda catheter (Cook Medical, Bloomington, IN) (**Figure 74-4**). There are radiopaque markers at the distal and proximal ends of the balloon. There are two ports, one for the balloon inflation/deflation and the other for guidewire placement. Performing REBOA with the Coda catheter or other catheters that require guidewires and larger femoral sheaths will be termed *traditional REBOA* in this chapter.[37] The ER-REBOA catheter (Prytime Medical, Lakewood, CO) does not require a long guidewire for catheter placement and uses a 7 French femoral sheath for catheter placement (**Figure 74-5**).[43] The orange sheath is used to straighten the nontraumatic P-tip for insertion into the sheath. The P-tip allows the ER-REBOA to be placed without a guidewire

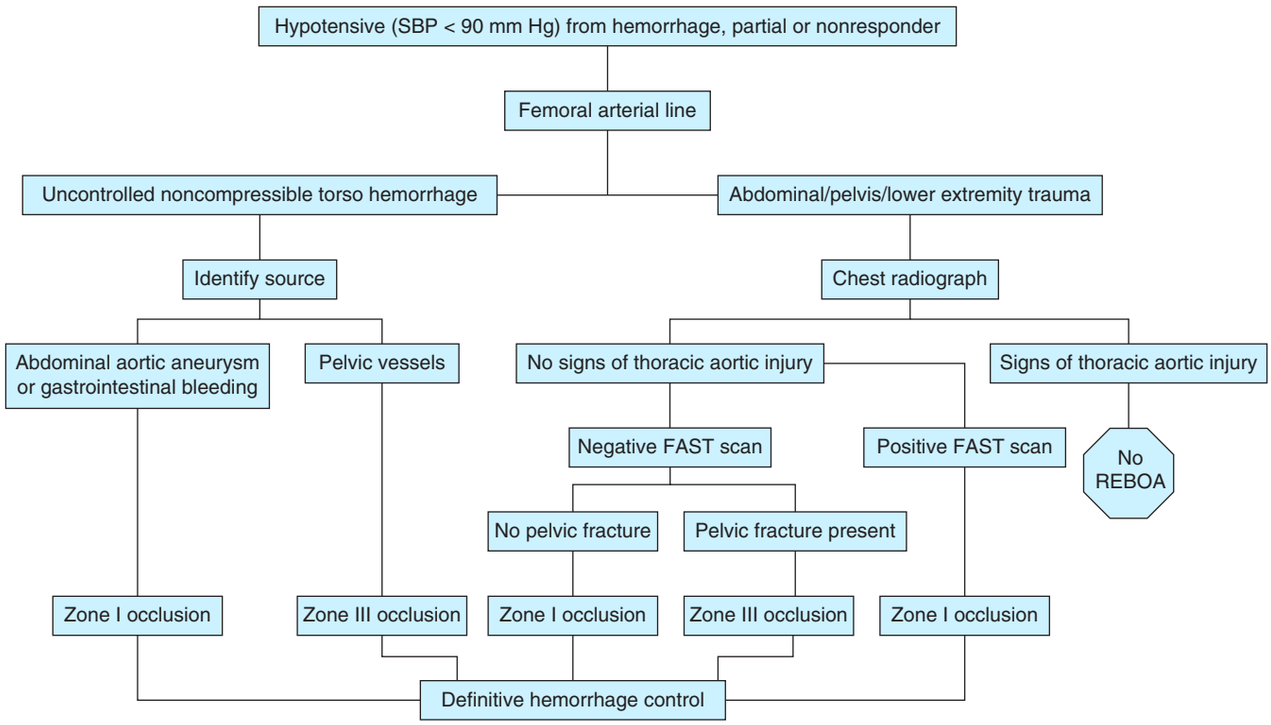
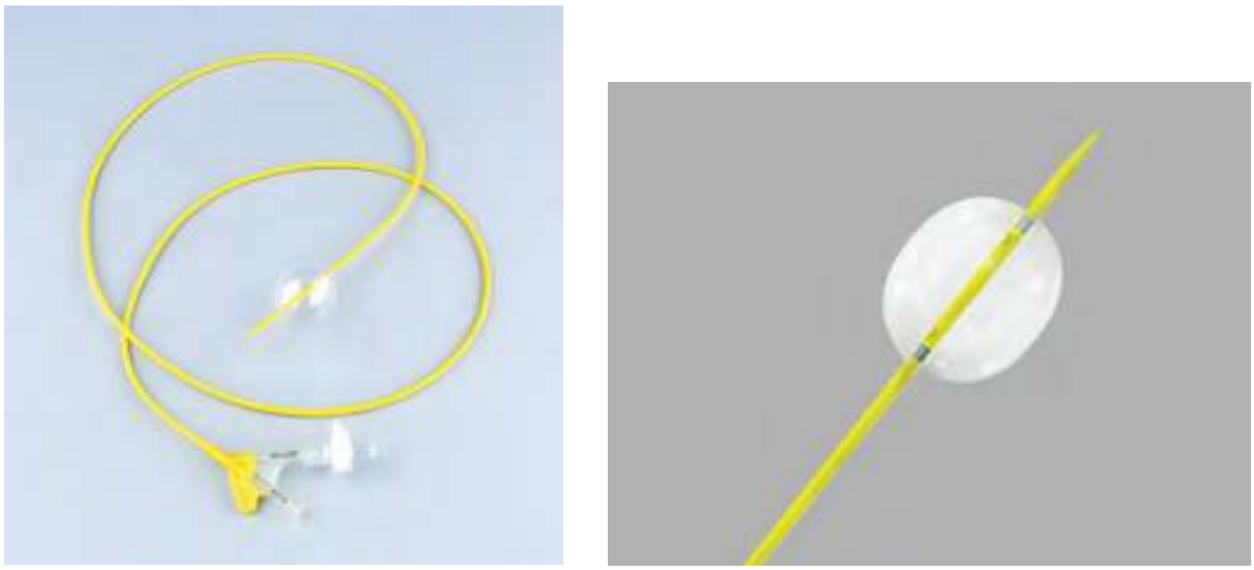
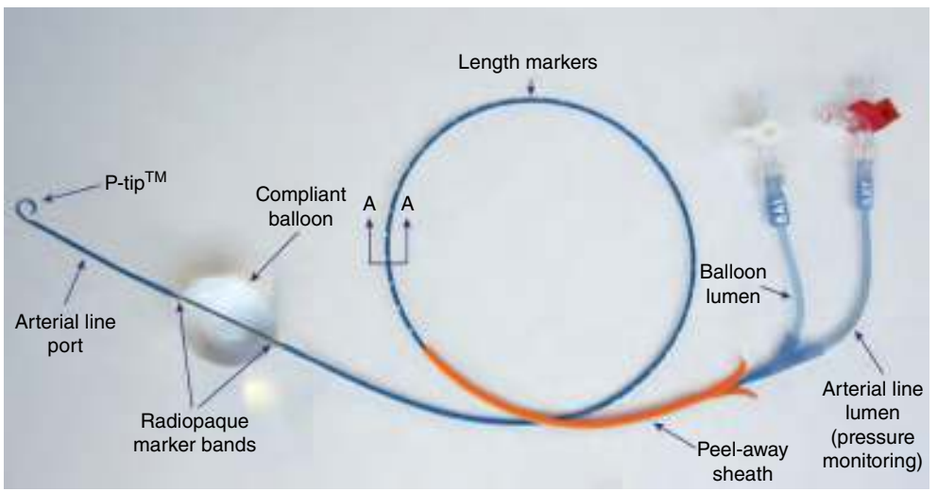


FIGURE 74-3. Algorithm for REBOA implementation in hemorrhagic shock. SBP, systolic blood pressure. (Adapted from existing algorithms.[33,37])



A B

FIGURE 74-4. Coda balloon catheter with inflated balloon. ***A.*** The catheter. ***B.*** The inflated balloon at the distal end. (Courtesy of Cook Medical, Bloomington, IN.)



A

B

C

FIGURE 74-5. ER-REBOA catheter with inflated balloon. ***A.*** The catheter with parts labeled. ***B.*** The catheter inserted into the port of the arterial sheath. ***C.*** An example of the catheter markings. (Photos courtesy of Prytime Medical, Boerne, TX.)

TABLE 74-1 Equipment Needed for Traditional REBOA

18 gauge arterial line set
US machine
Sterile US transducer cover
High-frequency linear transducer
Sterile drapes
Long working wires (J-wire, Rosen, Amplatz Super Stiff (0.035 inch, 260 cm)
Initial sheath and dilator (5 to 6 French, 8 to 15 cm in length)
Delivery, support sheath, and dilator (12 to 14 French, 45 to 60 cm in length)
Balloon occlusion catheter (Coda, Reliant, or Berenstein)
30 mL Luer lock syringes
3-way stopcock
100 mL injectable saline
Sterile basin
Scalpel, #11 or #15
Antibiotics

Other items to consider
Surgical set for femoral cutdown (Chapter 69)
Omnipaque contrast for balloon
Microbubble contrast agent for US identification

TABLE 74-3 Equipment Needed for ER-REBOA

18 gauge arterial line set
US machine
Sterile US transducer cover
High-frequency linear transducer
Sterile drapes
7 French introducer sheath with dilator
ER-REBOA catheter with included stopcocks
Delivery, support sheath, and dilator (12 to 14 French, 45 to 60 cm in length)
30 mL Luer lock syringes
3-way stopcock
100 mL injectable saline
Sterile basin
Scalpel, #11 or #15
Antibiotics

Other items to consider
Surgical set for femoral cutdown (Chapter 69)
Omnipaque contrast for balloon
Microbubble contrast agent for US identification

unlike other REBOA catheters. There are two ports, one for balloon inflation/deflation and a proximal arterial line port for monitoring pressures in the aorta proximal to the occlusion. The list of recommended supplies for performing traditional REBOA, a comparison of current REBOA catheters, and the equipment needed for using ER-REBOA are listed in **Tables 74-1 through 74-3**.

Fluoroscopy can aid in REBOA catheter placement by confirming central aortic wire placement, balloon catheter location, and balloon inflation.[44] Fluoroscopy may not be readily available in some institutions in emergent situations. Other methods to safely and accurately place REBOA catheters have been researched (e.g., external landmarks, morphometric roadmap equations to determine measurements from the femoral artery to major aortic branch points, and torso length).[45-47] US and contrast-enhanced US have shown promise in confirming wire placement and balloon positioning.[48,49]

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative if time and patient hemodynamics allow. Obtain an informed consent for the procedure, and place this in the medical record. Document the reason for not having a consent in the record if there is no time for it to occur. The patient should already be in the supine position. Place the patient on the cardiac monitor, noninvasive blood pressure cuff, pulse oximetry, and supplemental oxygen to monitor them during and after the procedure.

Prepare for the procedure. Identify the femoral arterial pulse by palpation or US. Clean the groin area of any blood, debris, and dirt. Apply chlorohexidine or povidone iodine to the area and let it dry. **Maintain sterility from this point forward.** Don full personal protective equipment (i.e., hat, mask, sterile gloves, and sterile gown). This protects the Emergency Physician from exposure to the patient's blood and the patient from iatrogenic infections. Apply full sterile drapes onto the patient. Instruct the nurse to administer prophylactic IV antibiotics.

Prepare the US transducer if using US with the procedure. Have an assistant apply US gel over the footprint of the transducer. Instruct the assistant to place the gel-coated US transducer in the sterile cover while you hold it. Have the assistant place sterile US gel over the transducer or the patient. Reidentify the femoral artery pulse. Apply a 25 gauge needle onto a 10 mL syringe. Fill the syringe with local anesthetic solution. Inject local anesthetic solution subcutaneously and over the femoral pulse.

TABLE 74-2 Commercially Available REBOA Catheters

Catheter name (manufacturer)	Diameter (mm)	Catheter size (French)	Recommended introducer sheath size (French)	Length (cm)
Coda	32 to 40	9	12	100
(Cook Medical)		9	12	120
		10	14	140
Reliant (Medtronic)	10 to 46	8	12	100
Berenstein (Boston Scientific)	11.5	6	6	80
ER-REBOA (Prytime Medical)	32	6	7	72

TECHNIQUES

New techniques and equipment are currently being developed.[20] The basic steps to performing REBOA include obtaining arterial access, positioning an appropriate catheter, and inflating the balloon. The above steps are all that is required of the Emergency Physician. Further steps in management will be described briefly as they take place after hemorrhage control with the patient in the care of the inpatient team. Two techniques for performing REBOA will be described, the traditional approach with catheters that require a guidewire for placement and aortic occlusion using the ER-REBOA catheter.

TRADITIONAL REBOA

Obtain femoral access with an introducer needle by using the anatomic landmarks (Chapter 72), a femoral cutdown (Chapter 69), or US guidance (Chapter 64). The approach using anatomic landmarks involves palpating for the femoral pulse and aiming 1 to 2 cm inferior to the inguinal ligament. Identifying the femoral artery may be challenging in hypotensive patients without palpable pulses. The Emergency Physician should have experience using a femoral artery cutdown if it is to be performed. **Observe several cardinal rules for the insertion of the catheter. Always occlude the open hub of a catheter, needle, or sheath to prevent an air embolism. Never let go of the guidewire to prevent its embolization into the circulation. Never apply excessive force to the guidewire on insertion or removal. Doing so may injure the vessel, break the guidewire, and/or embolize the guidewire.**

Attach the introducer needle to a 5 mL syringe containing 1 mL of sterile saline or local anesthetic solution. The specially designed introducer needle included with the catheter should be used, as it has a relatively thin wall and a larger internal diameter relative to its external diameter. It has a shorter bevel than a conventional hypodermic needle. It has a tapered hub to guide the guidewire into the needle proper.

If there is doubt about the exact location of the femoral artery, it may first be located with a small "finder" needle. Insert a 25 or 27 gauge needle attached to a 5 mL syringe through the skin puncture site previously chosen. Advance the needle at a 30° to 60° angle to the skin while applying negative pressure to the syringe. A flash of blood signifies that the tip of the needle is within the artery. Note the depth and location of the artery. Remove the finder needle. Alternatively, the finder needle may be left in place for reference.

Insert the introducer needle at a 30° to 60° angle. Inject a small amount of the fluid in the syringe to remove any skin plug that may block blood return once the artery has been penetrated. Apply negative pressure to the syringe by withdrawing the plunger. Advance the introducer needle into the artery (**Figure 63-10*A***). The artery's distance will vary depending on the patient's size and the target vessel's location. Stop advancing the introducer needle if the artery is not located within 3 to 5 cm of the skin. Withdraw the needle slowly while continuing to aspirate. The vessel will often have been completely traversed. If no blood is aspirated while withdrawing the needle, withdraw the introducer needle to the subcutaneous plane and redirect it. Avoid putting continuous pressure on the femoral artery pulse as even gentle pressure may collapse it.

Stabilize and hold the introducer needle perfectly still with the nondominant hand once blood returns in the syringe. The femoral artery has been entered if the blood is bright red and/or forces the plunger of the syringe back. Remove the syringe. **Blood should pulsatile flow freely from the hub of the needle.** Occlude the open hub of the introducer needle with the thumb of the nondominant hand while keeping the small finger of the hand in contact with the patient's skin. The Emergency Physician's proprioceptive reflexes will prevent movement of the introducer needle by maintaining contact with the patient's skin. Even a millimeter of movement may result in failure to stay within the lumen of the artery.

Prepare the guidewire (**Figure 63-11**). Grasp the guidewire and its sleeve with the dominant hand. The tip of the guidewire has a "J" shape when the sleeve is retracted (**Figure 63-11*A***). Slide the sleeve forward to straighten out the "J" of the guidewire (**Figure 63-11*B***). Insert the guidewire sleeve into the hub of the introducer needle (**Figures 63-10*B* and 63-11*C***). Advance the guidewire through the sleeve and into the introducer needle. **Never let go of the guidewire. One end of the guidewire must always be held to prevent its loss and embolization.**

Do not simply reverse the guidewire if the sleeve used to straighten the curved end of the guidewire is lost. The straight end of the guidewire can puncture the wall of the artery. Grasp the guidewire between the fourth and fifth fingers and the palm of the dominant hand (**Figure 63-12*A***). Apply gentle traction on the curved guidewire tip with the thumb and the second fingers to straighten the guidewire (**Figure 63-12*B***). The guidewire can then be inserted into the introducer needle hub without the use of the sleeve.

Advance the guidewire through the introducer needle and into the artery (**Figure 63-10*B***). The guidewire should advance easily into the artery. **Never force the guidewire.** Guidewire resistance may indicate that the introducer needle is not within the artery, is against the wall of a vessel, or is caught as the vessel bends. Slightly withdraw the guidewire, rotate it slightly, and readvance it. **The use of force will kink the guidewire and may cause it to damage the artery and adjoining tissues.** Advance the guidewire into the vessel. US can confirm the appropriate position of the guidewire in the femoral artery (**Figure 74-6*A***). Withdraw the introducer

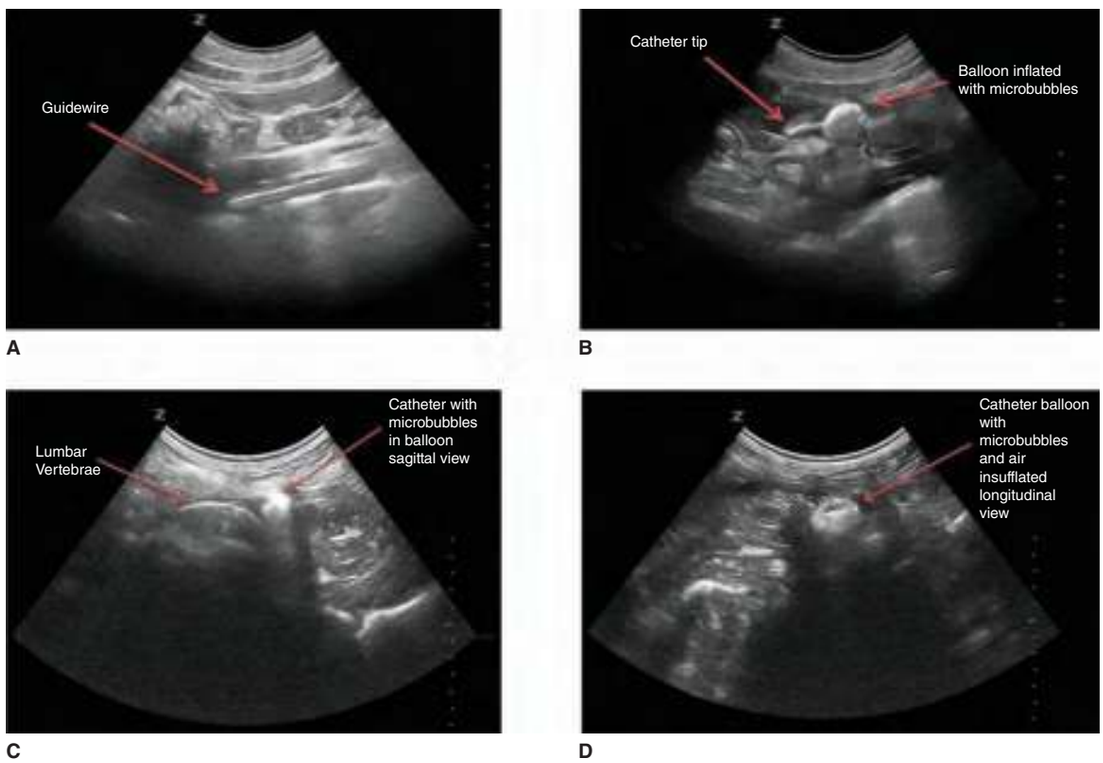


FIGURE 74-6. US images of the guidewire and catheter. ***A.*** Longitudinal view of the guidewire within aortic zone III. ***B.*** Longitudinal view of the Coda catheter tip and inflated balloon with microbubbles within aortic zone III. ***C.*** Sagittal view of the Coda catheter tip and inflated balloon with microbubbles within aortic zone III. ***D.*** Longitudinal view of contrast-enhanced US of balloon inflated with microbubbles and air within the aorta. (Used with permission from reference 48.)

needle and guidewire sheath while securely holding the guidewire (**Figure 63-10*C***). Grasp the guidewire with the nondominant hand as soon as the guidewire is visible between the tip of the introducer needle and the skin. Finish removing the needle over the guidewire.

Make a small incision in the skin adjacent to the guidewire using a #11 scalpel blade (**Figure 63-10*D***) to allow for passage of serial dilators. Place the dilator over the straight end of the guidewire (**Figure 63-10*E***). Advance the dilator over the guidewire, through the skin, and into the artery. A slight twisting motion of the dilator as it is advanced may aid in its insertion. Continue to advance the dilator until its hub is against the skin. **Do not release hold of the guidewire at any time.** Remove the dilator over the guidewire. Continue to apply the next size dilator, advance it, and then remove the dilator until the appropriate size is used.

Place the sheath tip over the guidewire. Advance the sheath over the guidewire and into the artery (**Figure 63-10*F***). **Do not release hold of the guidewire.** Gently rolling or twisting the sheath between the thumb and forefinger may aid in its advancement. Insert the sheath to the predetermined desired depth. Hold the sheath securely in place and remove the guidewire (**Figure 63-10*G***). **Occlude the open sheath lumen with a sterile-gloved finger to prevent an air embolism and excessive blood loss.** Ensure the stopcock on the sheath is in the off position or pointing toward the patient to avoid blood loss.

Determine the REBOA catheter length based on which zone of occlusion is indicated. Zone I occlusion requires a longer (i.e., 45 to 65 cm) sheath to maintain the balloon in appropriate position against the pulsatile forces in the proximal aorta. Zone III occlusion can be performed using the shorter introducer sheath (i.e., 10 to 25 cm) as the bifurcation of the aorta will firmly hold the inflated balloon in place.

The introducer sheath needs to be exchanged for the longer sheath to maintain the position of the inflated balloon for zone I occlusion. Advance a 0.035 inch wire until the floppy tip is in the distal aortic arch. Fluoroscopy is the best method to confirm proper placement. The distance from the femoral head to a point halfway between the xiphoid process and the medial clavicular head can be used as an estimated length of insertion if fluoroscopy is unavailable. The subxiphoid FAST view on US can be used to demonstrate central aortic wire placement.[49] Remove the short introducer sheath when the wire is placed. **Ensure the wire remains stationary by holding it against the patient as the sheath is removed.** The longer sheath is advanced over the wire to the desired location.

Apply the balloon catheter over the wire. Advance the balloon catheter through the sheath. Confirm appropriate balloon placement with fluoroscopy or US (**Figure 74-7*B***). If unavailable, the balloon can be advanced 5 cm longer than the sheath prior to balloon inflation. Inflate the balloon with a 30 to 60 mL syringe filled with half-saline and half-saline with iodinated contrast (**Figure 74-7*C***). Use fluoroscopy to adjust the balloon inflation so that the outer edges are parallel (**Figure 74-8**).[27,50] Other methods to determine adequate inflation include loss of distal pulses (e.g., especially the contralateral femoral pulse) and increased blood pressures proximal to the balloon measured with a proximal arterial line. US may be used to confirm catheter placement and balloon inflation with microbubble contrast (**Figures 74-6*B* through 74-6*D***).[48,51] Turn the three-way stopcock when the balloon inflation appears adequate. Turn the stopcock toward the balloon port or patient to maintain balloon inflation and occlusion. Note the volume instilled into the balloon and place this in the medical record.

Securing the balloon, sheath, and wire apparatus in the appropriate position is often best achieved by an assistant. There is the tendency of the REBOA catheter to migrate. Use an assistant who can identify and communicate position changes. This is preferable to suturing, taping, or using securing clips to secure the apparatus.

ER-REBOA

ER-REBOA requires only a 7 French introducer sheath, does not require a long guidewire, does not require fluoroscopy for placement, and comes with a proximal arterial line monitoring port (**Figure 74-5*A***). Obtaining femoral access is the same as the traditional REBOA procedure with the goal of inserting a 7 French introducer sheath.

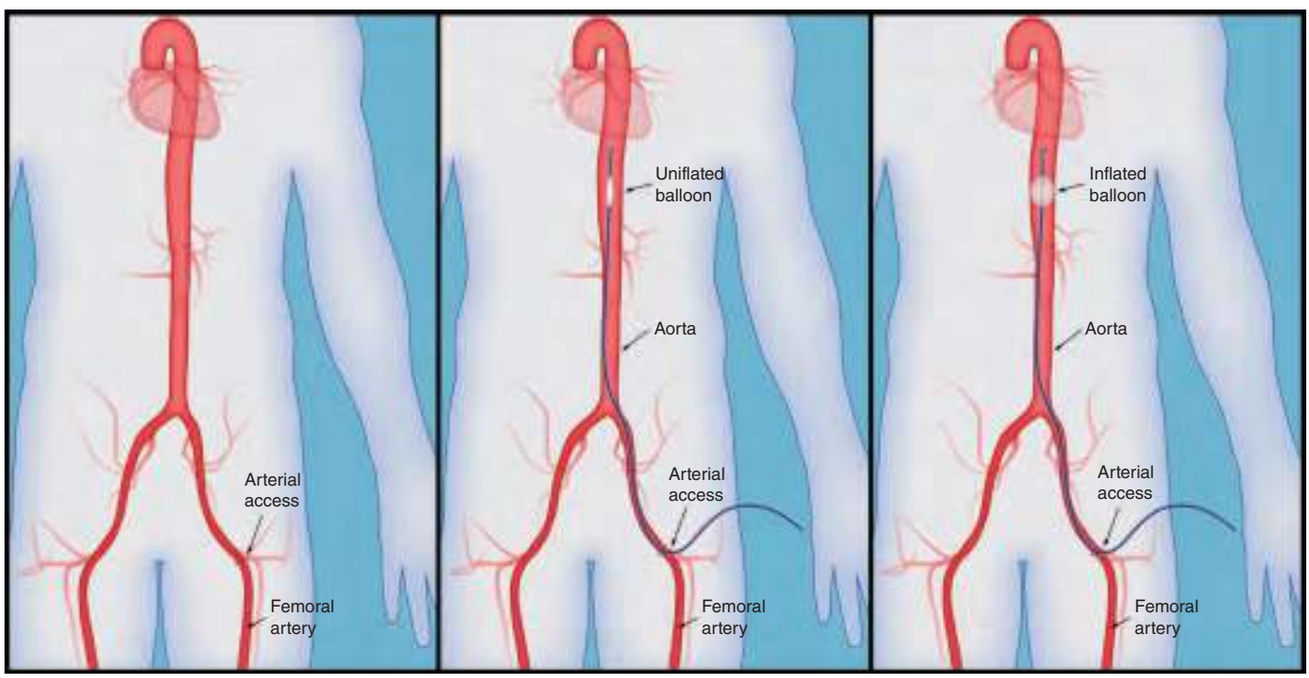
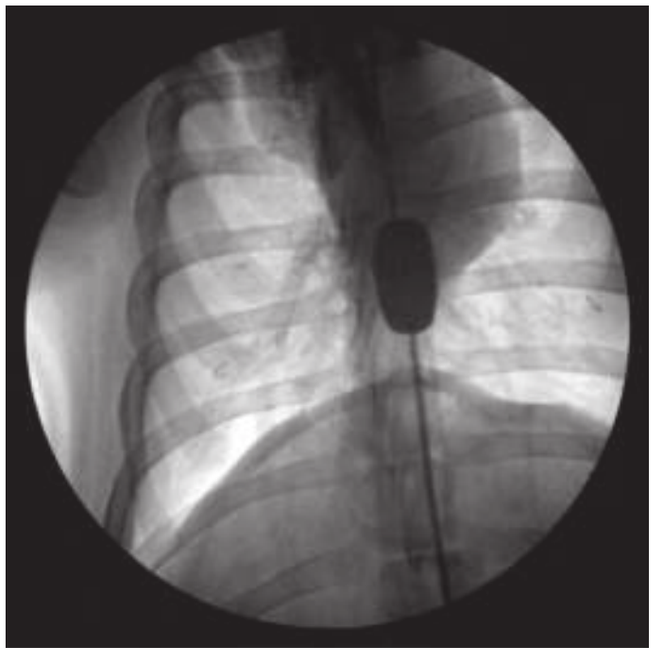


FIGURE 74-7. Insertion of the catheter. ***A.*** The arterial system. ***B.*** Insertion of the REBOA catheter. ***C.*** Inflation of the REBOA catheter balloon. (Courtesy of Prytime Medical, Boerne, TX.)



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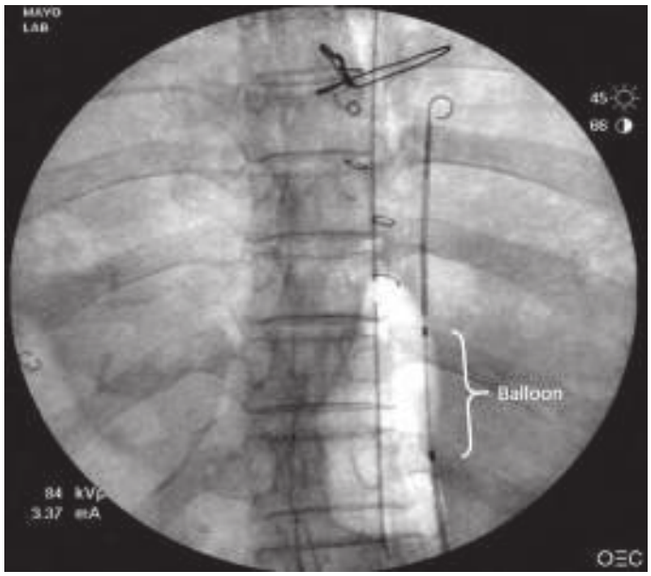

B

FIGURE 74-8. Inflated REBOA catheter in zone I. ***A.*** The catheter is conforming to the shape of the aorta. (Photo used with permission of reference 27.) ***B.*** The ER-REBOA balloon inflated with saline. (Photo used with permission of reference 50.)

Attach a 30 mL syringe filled with 24 mL of normal saline to the balloon port of the ER-REBOA catheter. An alternative is to attach a syringe containing a mixture of contrast and saline. Ensure the balloon functions and has no leaks by inflation and deflation. **Lock the stopcock after deflating the balloon to prevent an air embolism.** Attach a pressure transducer to the arterial line port of the catheter and zero the transducer (Chapter 72).

Measure to the zone of desired occlusion using external landmarks. Zone I is measured with the proximal edge of the balloon or closest portion to the catheter ports (**Figure 74-5**) at the edge of the xiphoid. Zone III is measured with the proximal edge at the umbilicus. A long introducer sheath for zone I occlusion is not needed as in the traditional approach.

Straighten the P-tip with the orange sheath. Insert the orange sheath into the femoral introducer sheath (**Figure 74-5*B***). Advance the ER-REBOA catheter a few centimeters into the introducer sheath. Pull the orange sheath back. Advance the ER-REBOA catheter to the predetermined depth. Catheter placement may be visualized using US or fluoroscopy to identify the catheter in the central aorta as is done with traditional REBOA.

Hold the ER-REBOA catheter in place. Inflate the balloon until moderate resistance is felt or the arterial line transducer shows an increase in blood pressure proximal to the balloon. It usually takes between 12 and 22 mL of saline depending on the size of the patient's aorta. **Instilling more than 24 mL can lead to device failure or aortic injury.** Turn the stopcock toward the balloon port or patient to maintain balloon inflation and occlusion. Note the volume instilled into the balloon and place this in the medical record.

Securing the balloon, sheath, and wire apparatus in the appropriate position is often best achieved by an assistant. There is the tendency of the ER-REBOA catheter to migrate. Use an assistant who can identify and communicate position changes. This is preferable to suturing, taping, or using securing clips to secure the apparatus.

PEDIATRIC CONSIDERATIONS

REBOA may eventually be an adjunct in the management of the pediatric patient with life-threatening hemorrhage.[52] Studies have yet to assess age-related vascular diameter changes, anatomic considerations, consistency of landmark identification, safety, and feasibility. It can be used in large adolescents like they are small stature adults.

AFTERCARE

The next step is definitive hemorrhage control with the balloon inflated. The remaining steps are beyond the scope of the Emergency Physician. It may be helpful to understand the basics of REBOA catheter removal. The assistant maintains the position of the balloon apparatus. The Surgeon deflates the balloon. Note the volume retrieved during deflation and compare this to the initial inflation volume. Deflation of the balloon in cases of prolonged inflation or incomplete resuscitation may lead to washout of metabolic byproducts, acidosis, and hypotension. Intermittent balloon deflation and inflation may be necessary until stability is maintained. Limit the amount of distal hypoperfusion from the REBOA catheter to minimize ischemic time and the consequences of inflammatory-mediated organ failure after REBOA.[23,53]

Remove the deflated balloon when REBOA is no longer necessary. Flush the sheath with 100 mL of heparinized saline made with 1000 units of heparin in 1 L of saline. Sheaths up to an 8 French can be removed safely with manual pressure or with a closure device to control bleeding from the arteriotomy site.[54] Catheters used for traditional REBOA are larger and will require femoral exposure and closure.

COMPLICATIONS

There are potential complications associated with performing a REBOA. Damage to the femoral vasculature during initial arterial access can occur. There are reports of external iliac artery injury and lower limb ischemia requiring lower limb amputation.[22] The long

wires used with the traditional REBOA have the potential to result in injury to cerebral vessels or coronary vessels or induce cardiac arrhythmias if inserted too far.[37] The inflated balloon can cause bowel ischemia and limb ischemia.[55] Other potential complications include vascular damage from balloon inflation in a nontarget vessel (e.g., iliac arteries and renal artery) or balloon overinflation. Paralysis secondary to spinal cord ischemia is a theoretical possibility. Thrombotic events can occur due to aorta occlusion, loss of distal blood flow, or the presence of a foreign body in the vascular system. Perform frequent neurovascular checks and angiographic imaging when indicated. Prolonged occlusion time may lead to severe lactic acidosis, metabolic derangements, and increased inflammatory markers. The combination of these effects may precipitate multisystem organ failure when the balloon is deflated and perfusion is restored.[22,39,53] The complications are decreased using smaller diameter 7 French sheaths.[56] In one trial of 46 patients, 4% developed a distal embolism and 2% developed pseudoaneurysms.[34] There were no amputations, extremity ischemia, fistulas, hematomas, infections requiring antibiotics, or cases of paralysis.[34]

SUMMARY

REBOA is a potentially lifesaving technique if performed in the appropriate patient. REBOA catheters can be placed to obtain aortic control in patients with hemorrhagic shock or may be placed prophylactically in patients at risk of developing hemorrhagic shock secondary to injuries with blood supplied by the subdiaphragmatic aorta. REBOA serves as a less morbid alternative to a thoracotomy for patients with life-threatening and noncompressible torso hemorrhage. The procedure can be broken down into the basic steps of obtaining arterial access, selecting a balloon occlusion catheter, placing the catheter, inflating the balloon, and eventually deflating and removing the catheter. The resuscitative environment must be conducive to the use of REBOA, and resources for timely definitive hemorrhage control must be available.

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SECTION 5

Gastrointestinal Procedures

75 Nasogastric Intubation

Alex Koo and Ryan Walsh

INTRODUCTION

Nasogastric (NG) intubation is a commonly performed procedure in the Emergency Department.[1] Its use as a conduit into the stomach was first popularized in the early twentieth century mainly through the efforts of Dr. Levin. Clinicians have since studied its use, proposed methods to improve the ease with which the NG tube is inserted, and determined ways to diminish the incidence of potentially lethal complications. A NG tube is often placed in patients who have a bowel obstruction, intoxication, intractable nausea and vomiting, significant trauma, or upper gastrointestinal (GI) bleeding or who are endotracheally intubated. The procedure is rapid, simple, and straightforward. The insertion of an NG tube is slowly decreasing.[2-7]

ANATOMY AND PATHOPHYSIOLOGY

The nasal cavity is lined by the very vascular nasal mucosa. The medial wall of the nasal cavity is composed of the septum. The lateral wall of the nasal cavity is covered by the turbinates or concha. The posterior nasal cavities are continuous with the nasopharynx that develops into the posterior oropharynx caudally (**Figure 75-1**). The oropharynx continues inferiorly as the esophagus that travels through the esophageal hiatus in the diaphragm and enters the stomach.[8]

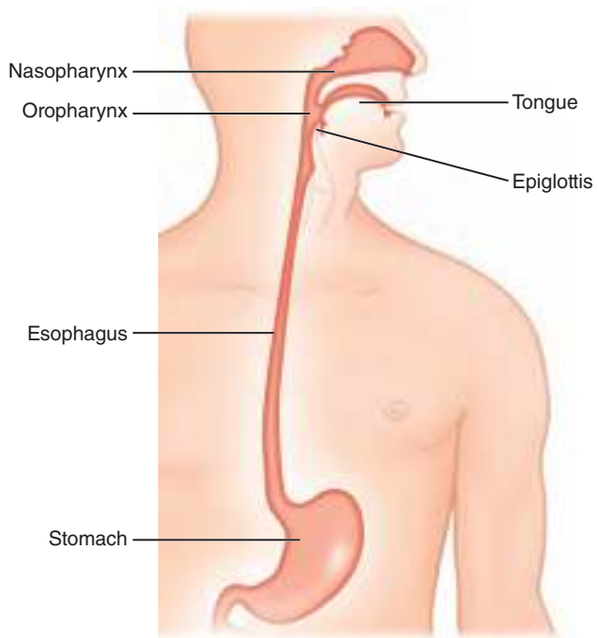

FIGURE 75-1. Basic anatomy of the path of the NG tube.

Pediatric nasopharyngeal anatomy differs from that of adults and can result in a more difficult NG tube insertion. The nostrils and nasal passages of children are quite small and limit the size of NG tube that may be passed. Children have relatively large tonsils and adenoids, which may hinder passage of the tube. The nasal tissues are often soft, easily injured, and may bleed as the NG tube is passed. The tongue is large by comparison with adults and may push into the oropharynx and impede passage of the NG tube.

INDICATIONS

NG intubation may be performed for diagnostic or therapeutic indications.[2] **The primary indication for NG intubation is gastric decompression.** This may help relieve a bowel obstruction, volvulus, or recurrent vomiting (e.g., pancreatitis). NG tubes are placed to decompress the stomach preoperatively, postintubation, or prior to a procedure (e.g., diagnostic peritoneal lavage or pericardiocentesis).

An NG tube may be placed for diagnostic purposes. A diagnostic indication for NG tube placement is to aspirate stomach contents to evaluate the presence, rapidity, and volume of an upper GI hemorrhage. The detection of an upper GI bleed may be unreliable via this technique.[4] Do not use the fecal Hemoccult card on gastric aspirates to test for occult blood in gastric aspirates. A Hemoccult card may be inaccurate due to gastric aspirate acidity. The Gastroccult card uses a developer that neutralizes gastric acid and renders it able to detect hemoglobin.[9,10] An NG tube may be inserted to instill air into the stomach to assess for an intraperitoneal perforation. Gastric fluid and contents may be aspirated for laboratory analysis. NG tubes may be placed to visualize the anatomy of the proximal GI tract on a radiograph. An NG tube demonstrates a diaphragmatic hernia or a blind esophageal pouch in children on chest radiography with coiling visualized superior to the diaphragm.

An NG tube can be used for administration of medications, oral contrast for diagnostic studies, nutrition, and gastric lavage. NG tubes are an effective and inexpensive means of hydration.[11-14]

CONTRAINDICATIONS

Absolute contraindications do not exist for NG tube placement. The relative contraindications are geared toward predicting which patients are more likely to experience complications and which patients are likely to have misplaced tubes. Avoid insertion of an NG tube unless necessary in the patient with midface trauma or a suspected basilar skull fracture. Intubation through the nasal cavity in these patients can result in the NG tube being misdirected through a perforation in the cribriform plate of the ethmoid bone and into the brain. Patients with facial trauma or basilar skull fractures are best served with orogastric tubes.[15]

Relative contraindications include coagulopathies, esophageal strictures, ingestions of alkaline substances, nasal obstruction, or recent nasal or gastric surgery. Patients with esophageal varices pose a relative contraindication. Placement of a semirigid tube into the esophagus or stomach has the potential to cause rupture of the varices and uncontrollable hemorrhage. NG placement is generally considered safe in these patients if done carefully.[16,17] Talk to

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the Surgeon before putting an NG tube in any patient with stomach surgery (i.e., recently or in the past). The tube can perforate the proximal pouch and may not decompress the stomach.

EQUIPMENT

- Topical anesthetic (e.g., benzocaine spray, cocaine, viscous lidocaine)
- Topical vasoconstrictor (e.g., phenylephrine, oxymetazoline, cocaine)
- 2% or 4% lidocaine for nebulization
- Glass of water with straw
- Emesis basin
- Water-based lubricant
- NG tube, various sizes
- 60 mL syringe
- Wall suction, set to low intermittent suction
- Suction tubing
- Benzoin
- 1 inch adhesive tape
- Tongue depressor

NG tubes are made of clear polypropylene. They are semirigid and single-use devices. The two most commonly used NG tubes are the Levin tube (**Figure 75-2*A***) and the Salem Sump tube (**Figure 75-2*B***). They both have multiple distal sideports for aspiration. The Levin tube is a single-lumen tube that is easy to insert (**Figure 75-2*A***). It is simple to use for the aspiration of gastric contents, the instillation of fluids and/or medications, and the application of low intermittent suction. The tube has a radiopaque stripe. The amount of suction is difficult to control with the Levin tube. The distal sideports can become occluded with the gastric mucosa and damage this tissue when the tube is attached to suction. The Salem Sump tube is a double-lumen, radiopaque tube (**Figure 75-2*B***). It has a smaller suction lumen than the Levin tube. The second lumen allows a constant inward airflow to prevent the sideports from becoming occluded by the gastric mucosa.

PATIENT PREPARATION

The most beneficial factor in the successful placement of a NG tube is a patient who is informed of the procedure and can cooperate with the instructions. Explain the risks, benefits, and complications associated with the procedure to the patient and/or their representative. Take the patient through each step prior to the start of the procedure to ensure maximal cooperation. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Place the patient seated upright. Examine the patient for nasal septal deviation or other anatomic abnormalities that may hinder the passage of the NG tube. Ask the patient to breathe through one nostril while the other nostril is occluded to determine which nostril is the most patent.[18] Drape the patient to protect them and the bedding from soilage if there is emesis. Have a glass of water and a straw within the patient's reach if not contraindicated. Have an emesis basin within reach of the patient.

Consider the use of anesthesia to make the procedure more tolerable (Chapter 205).[19,20] The application of topical lidocaine and phenylephrine to the nose and benzocaine spray to the throat resulted in significantly less pain and discomfort than the use of

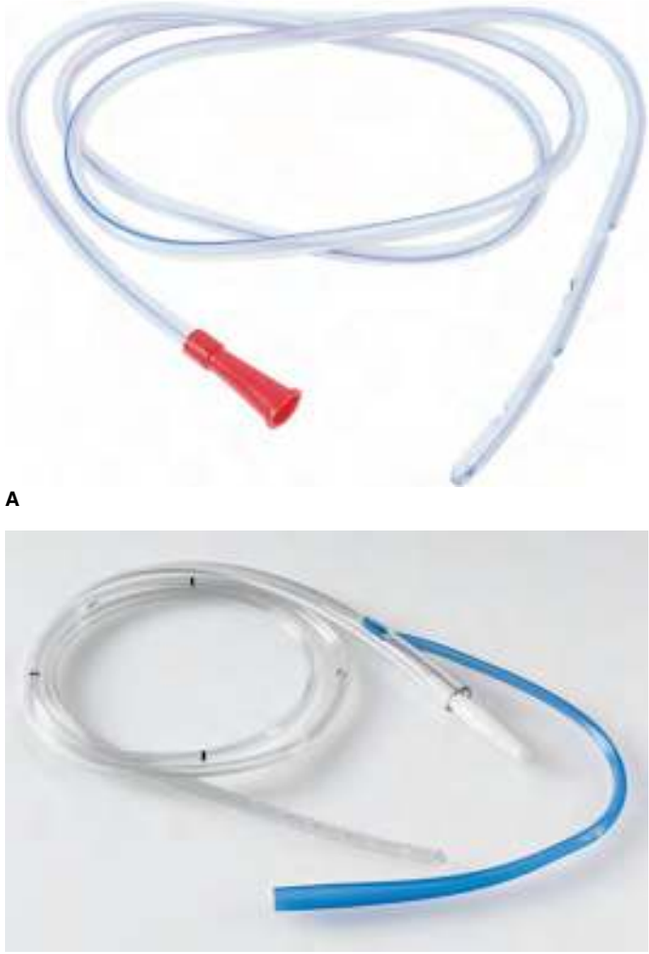

FIGURE 75-2. Common types of NG tubes placed in the Emergency Department. ***A.*** Levin tube. ***B.*** Salem Sump tube.

lubricant alone.[21] A sample protocol would include the instillation of 0.5% phenylephrine nasal spray followed by viscous lidocaine. Cetacaine spray can be used to anesthetize the nose and the throat.[18] Nebulized 4% lidocaine (e.g., 2.5 mL of saline containing 100 mg of lidocaine) via facemask can be used in adolescents and adults.[19] It has been shown to be superior to lidocaine spray as an anesthetic to reduce gagging and vomiting while increasing the success of NG tube placement.[22-24] The dose used in children has been 4 mg/kg of lidocaine for nebulization of a 2% or 4% lidocaine solution.[25] **Be cautious when calculating the proper weight-based doses and volume of solution for children.** Lidocaine can also be administered in the nasal cavity, nasopharynx, and oropharynx using a mucosal atomizer device (**Figure 29-1 and Table 29-1**). A small pilot study demonstrated that the administration of 2 mg of intravenous midazolam with cophenylcaine (i.e., lidocaine 5% with 0.5% phenylephrine) was more effective in controlling pain during NG tube insertion than cophenylcaine alone.[26] Lidocaine gel can be inserted in the nasal cavity.[27] Allow 3 to 5 minutes for these medications to take full effect before inserting the NG tube.

Choose a size of NG tube that is appropriate for the patient. A size 16 to 18 French is typically used for an adolescent or adult patient. A formula (e.g., [age in years + 16] ÷ 2) may be used to choose the proper size NG tube for children. Other less commonly used methods are available to determine the proper size NG tube in children.[28-30] This includes two times the endotracheal tube length

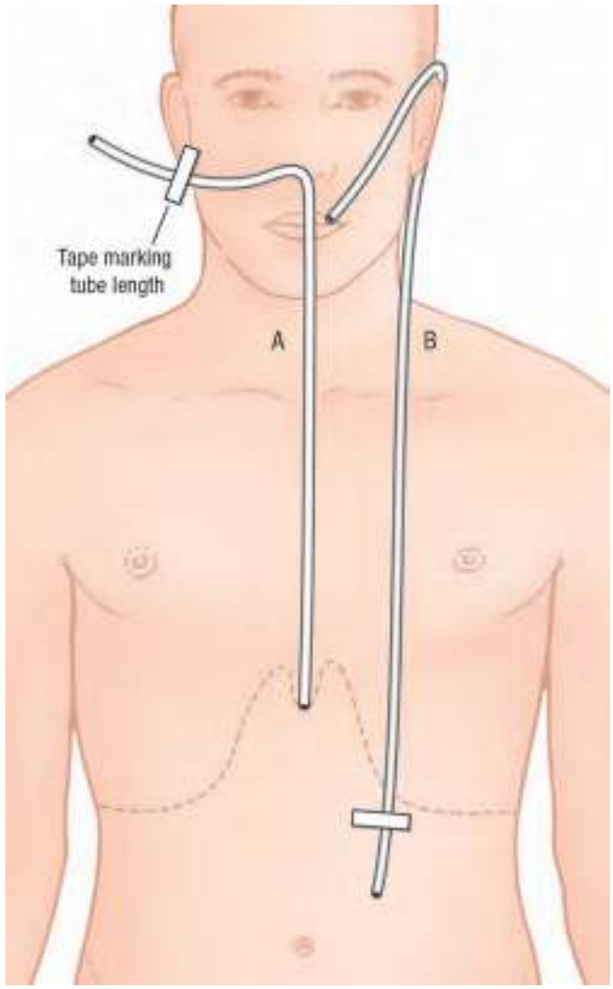

FIGURE 75-3. Determining the proper length of NG tube to insert. ***A.*** The length is determined by the distance from the xiphoid process to the tip of the nose to the earlobe. ***B.*** The length is determined by the distance from the tip of the nose or lip to around the left ear and to just below the left costal margin. A piece of tape should be used to mark the distance on the NG tube.

formulas. Typical sizes include 8 French for infants, 10 to 12 French for small children, and 12 to 14 French for older children.

Estimate the length of the NG tube to be inserted (**Figure 75-3**). Place the tip of the NG tube on the patient's xiphoid process and extend it to the tip of the nose and over the earlobe (**Figure 75-3*A***). Measure this distance and add 15 cm or 6 inches. Mark this distance with a piece of tape on the NG tube. Alternatively, place the tip of the NG tube on the tip of the nose or lip and extend it over the left ear and to just below the left costal margin (**Figure 75-3*B***). Mark this distance with a piece of tape.[18] Some use a formula in children of orogastric length = [3 × weight (kg) + 12 cm] or nasogastric length = [3 × weight (kg) + 13 cm].

TECHNIQUE

Administer the appropriate anesthetics, antiemetics, or anxiolytics.[31] Lubricate the first 4 inches or 10 cm of the NG tube with water-soluble lubricant. Position the patient. Place their neck in slight flexion. Gently introduce the NG tube along the floor of the nostril and under the inferior turbinate (**Figure 75-4*A***). Advance the NG tube parallel to the nasal floor until it reaches the nasopharynx as indicated by mild resistance (**Figure 75-4*A***). **Do not insert and advance the NG tube in an upward or lateral direction to prevent impingement and damage to the turbinates.** Instruct the patient to swallow. This may be assisted by having the patient sip water through a straw if not contraindicated. Continue to advance the NG tube (**Figure 75-4*B***). Advancement may be aided by rotating the NG tube medially. **Withdraw the NG tube if at any time significant resistance to advancement, respiratory distress, the inability to speak, or significant nasal hemorrhage occurs.**[18] Advance the NG tube until the distance previously measured with the tape is at the nostril (**Figure 75-5**). Verify proper placement (**Figures 75-6, 75-7, and 75-8**).

ALTERNATIVE TECHNIQUES

The NG tube may coil in the oropharynx, mouth, or hypopharynx (**Figure 75-6*C***). A larger bore NG tube may be used and may not coil. Cool the NG tube in cold tap water or ice water for 5 minutes to make the tube stiffer and then reinsert it. An alternative is to place the distal end of an NG tube into an oropharyngeal airway and then chill it in ice water (**Figure 75-9**). The oropharyngeal airway will place a curve in the chilled NG tube, which may allow it to pass easier. The chilled NG tube will retain this curve for 30 to 60 seconds until it rewarms (**Figure 75-9**). Another alternative is to turn the patient's head laterally if not contraindicated.[32]

One study attempted to improve the success rate of NG tube placement by providing external and medially directed pressure on the ipsilateral neck at the level of the thyrohyoid membrane.[33] This maneuver will collapse the piriform sinus and eliminate it as a potential site for impaction. This maneuver was successful for difficult NG intubation in 85% of patients.

There is a greater chance of tube misplacement in the intubated or unconscious patient due to difficulty with cooperativity and positioning. An option is to place several fingers through the patient's mouth and into the oropharynx. The fingers can be used to guide the NG tube against the posterior oropharyngeal wall and into the hypopharynx without it coiling. **Reserve the finger technique for intubated patients in whom the gag reflex is not a concern. Do not attempt this unless the patient is unconscious or paralyzed to prevent them from biting and injuring the fingers.** Use a tongue depressor to move the tongue to the floor of the mouth and make manipulation of the NG tube easier by creating more space in the mouth to maneuver.

Another technique to help pass an NG tube involves direct visualization using a direct or video laryngoscope and an airway forceps (e.g., Boedeker, Magill, or Tylke).[34,35] Insert the NG tube through the nose and advance it into the oropharynx or hypopharynx. Insert the laryngoscope blade and visualize the distal tip of the NG tube and the esophageal opening. Grasp the tip of the NG tube with the forceps and advance it into the esophagus. Continue to advance the NG tube until the tip is within the stomach. Feed a gum elastic bougie through the distal NG tube eyelet to give it more rigidity for initial insertion.[36] This technique can be used on the unconscious and intubated patient when the NG tube will not pass into the esophagus.

The NG tube may easily pass the hypopharynx but not pass completely through the esophagus and into the stomach. **The tip of the tube may be caught at the level of the cricopharyngeus muscle, behind the left mainstem bronchus, or at the lower esophageal sphincter.** Attempt to pass an NG tube that has been cooled for 3 to 5 minutes in cold tap water or ice water. Grasp the thyroid cartilage and lift it anterior and upward to open the esophagus and allow passage of the NG tube through the upper esophagus.

An orotracheally intubated patient requires an NG tube to decompress the stomach, which can be achieved via a modified Seldinger technique.[37] Remove the respiratory adapter from the proximal end of a second endotracheal tube. Liberally lubricate the endotracheal tube and insert it through the patient's mouth and into

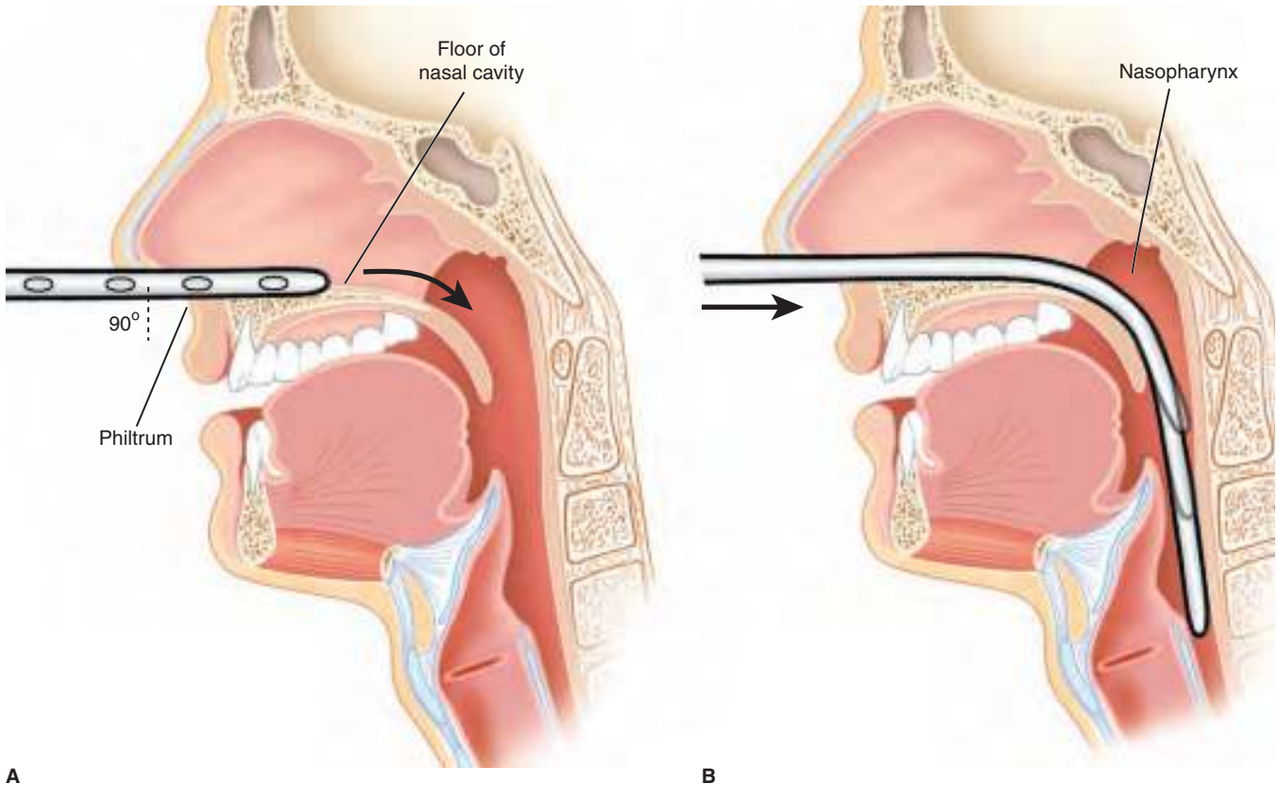

FIGURE 75-4. Insertion of the NG tube. ***A.*** The NG tube is inserted parallel to the floor of the nasal cavity and at a 90° angle to the philtrum. ***B.*** The NG tube is advanced along the floor of the nasal cavity, through the nasopharynx and oropharynx, and into the esophagus. It is further advanced until the tape mark is at the philtrum.

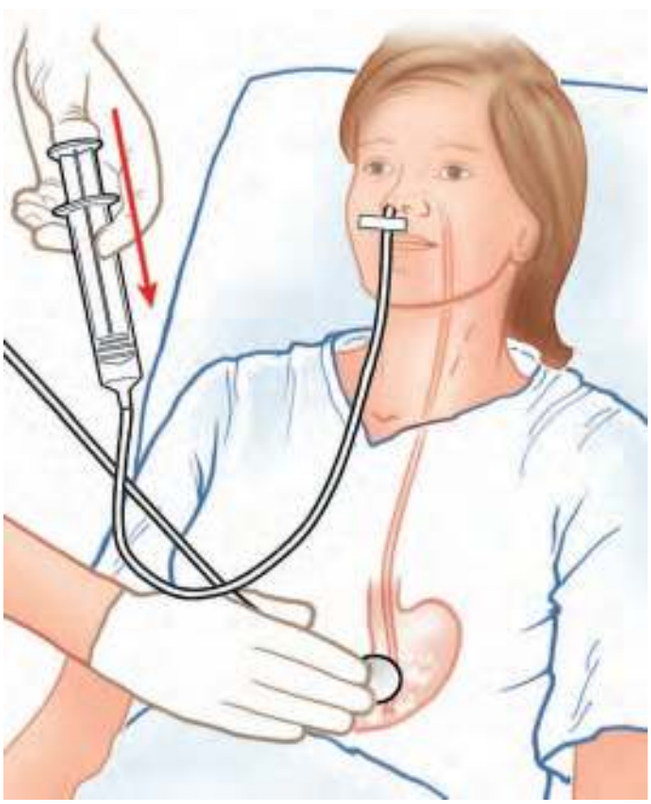

FIGURE 75-5. Proper placement of the NG tube. The tip of the tube resides within the stomach. To confirm proper placement, inject air through the NG tube while simultaneously auscultating over the stomach.

the esophagus. Insert a well-lubricated NG tube through the endotracheal tube and into the stomach. Confirm the proper position of the NG tube. Carefully withdraw the endotracheal tube over the NG tube. Reconfirm that the distal end of the NG tube remains within the stomach.

The risk for tube misplacement is greater in the intubated patient who is unable to assist with NG intubation. Ensure that the NG tube does not come out of the patient's mouth and that there are no changes in the patient's oxygen saturation when inserting the NG tube (**Figures 75-6*D* and 75-6*E***). It is very easy for the NG tube to pass by the cuff of an endotracheal tube without much resistance.[33] **It is paramount that correct placement of the NG tube is verified clinically and radiographically before it is used to instill any fluid or medication.**

ASSESSMENT

Verify proper placement by aspirating stomach contents, auscultating a rush of air over the stomach while 20 to 30 mL of air is insufflated using a syringe through the NG tube, and/or by radiographically demonstrating the tip of the NG tube in the stomach and below the diaphragm (**Figures 75-6, 75-7, and 75-8**).[38-40] **Infusion of substances through a misplaced NG tube could be disastrous.**

Verify the position of the NG tube with simple techniques.[38-40] The patient should be able to speak without respiratory distress immediately after placement of the NG tube. Observe the patient for complaints of drooling, dysphagia, fever, mediastinal air, neck pain, substernal chest pain, subcutaneous air, or trismus. These are signs of esophageal perforation or NG tube misplacement.[41,42] Air insufflated into the pleural space or the esophagus after misplacement of the NG tube can be just as easily heard over the upper abdomen.[11] Gastric contents should be able to be aspirated through the

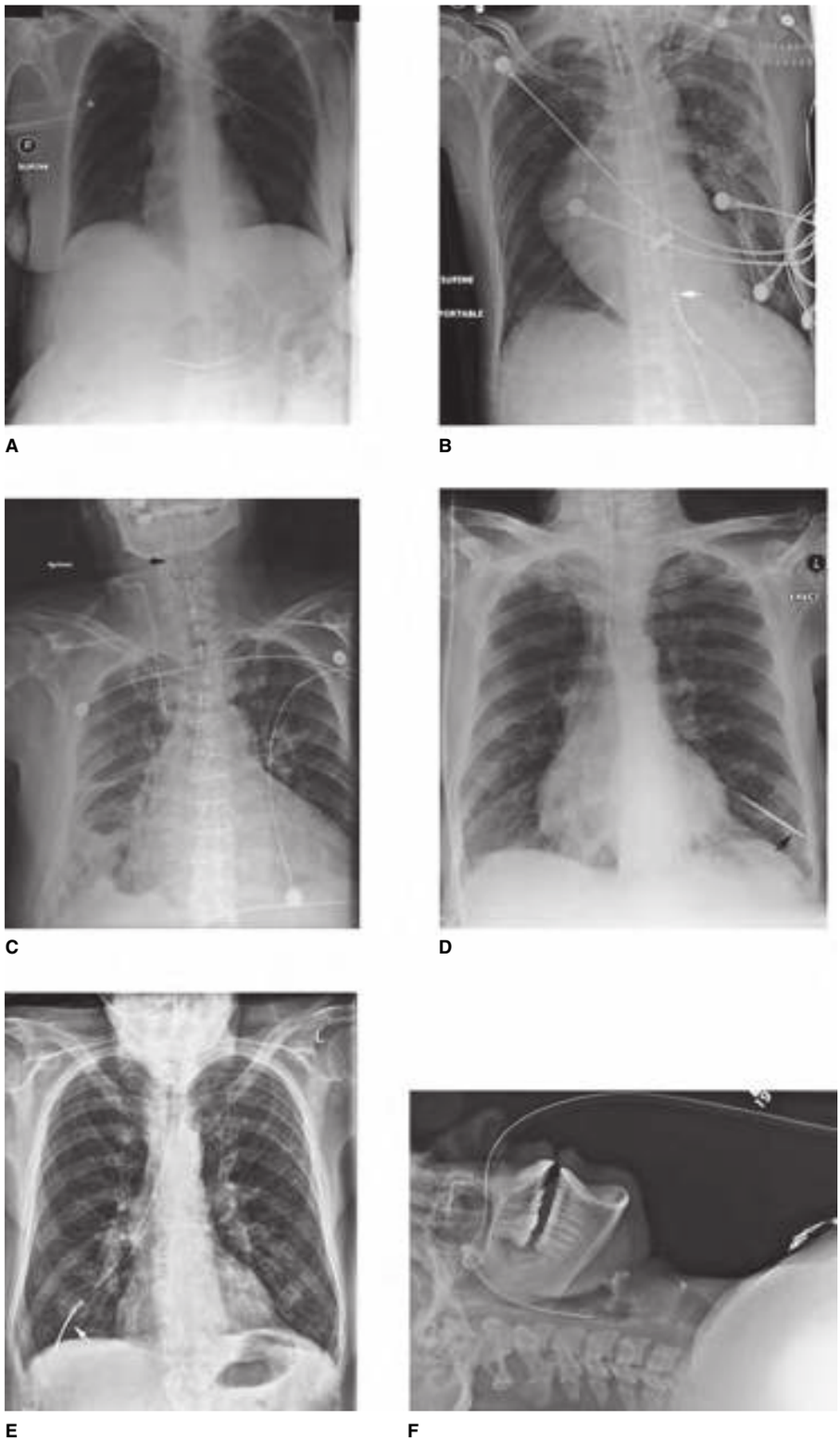

FIGURE 75-6. Radiographs of the inserted NG tube. ***A.*** The tip of the NG tube is in the stomach. ***B.*** The tip of the NG tube is in the esophagus after it curves in the stomach. ***C.*** The tip of the NG tube is coiled in the hypopharynx. ***D.*** The tip of the NG tube is in the left lower lobe of the lung. ***E.*** The tip of the NG tube is in the right lower lobe of the lung. ***F.*** The NG tube is knotted in the nasopharynx. (Photos used with permission from www.wikiradiography.net.)

NG tube. Testing the pH of the gastric contents can help predict the placement of the NG tube. The pH of the aspirated fluid will be ≥ 7 in 99% of patients if the NG tube is in the respiratory tree.[43] The pH of the aspirated fluid will be ≤ 5 in 70% of the patients if the NG tube was correctly placed in the stomach. The use of H_2 blockers or antacids makes the assessment of gastric pH difficult.

Radiography is the most reliable and should be strongly considered when the NG tube is to be used for medication administration

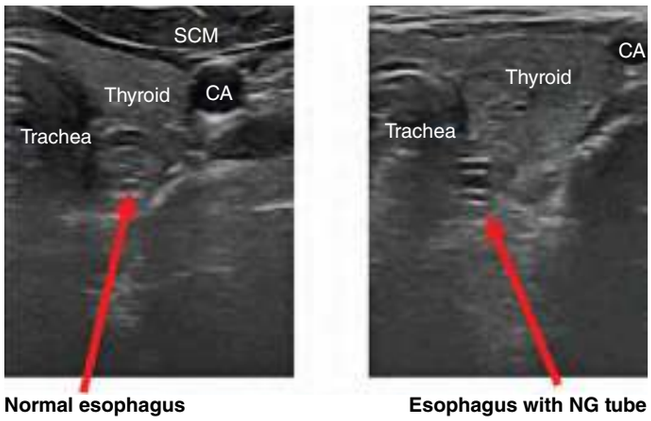

FIGURE 75-7. Ultrasound images of the esophagus without (left) and with (right) the NG tube. CA, carotid artery; SCM, sternocleidomastoid muscle. (Used with permission from reference 50.)

or alimentation.[42] Radiographic demonstration of the tube in the antral or fundal portion of the stomach is the preferred method of confirmation.[41,44,45] Make sure the NG tube bisects the carina, avoids the bronchial contours, and crosses the midline diaphragm, and that the tip is visible under the left hemidiaphragm.[41,45]

Multiple studies have shown the utility of ultrasonography to confirm NG tube placement in adults and children (**Figures 75-7 and 75-8**).[46-52] Use a high-frequency linear transducer. The endotracheal tube can be visualized as it passes through the esophagus below the level of the cricoid cartilage. Ultrasound can be used to verify the position of the NG tube after insertion. A chest radiograph should still be obtained if the ultrasound suggests the NG tube is not located in the stomach.[53]

FIGURE 75-9. An NG tube is inserted into an oropharyngeal airway before being chilled in ice water. The chilled NG tube will retain its curve for 30 to 60 seconds.

AFTERCARE

Secure the NG tube so it does not move.[54] Apply benzoin to the patient's nose. Apply tape to the nose and around the NG tube to secure it in place (**Figure 75-10**). Attach the NG tube to wall suction as needed.

NASOGASTRIC TUBE REMOVAL

Explain to the patient the procedure and what they will experience as the NG tube is removed. It is recommended for Emergency Physician to wear gloves, a mask with an eye shield, and a gown to

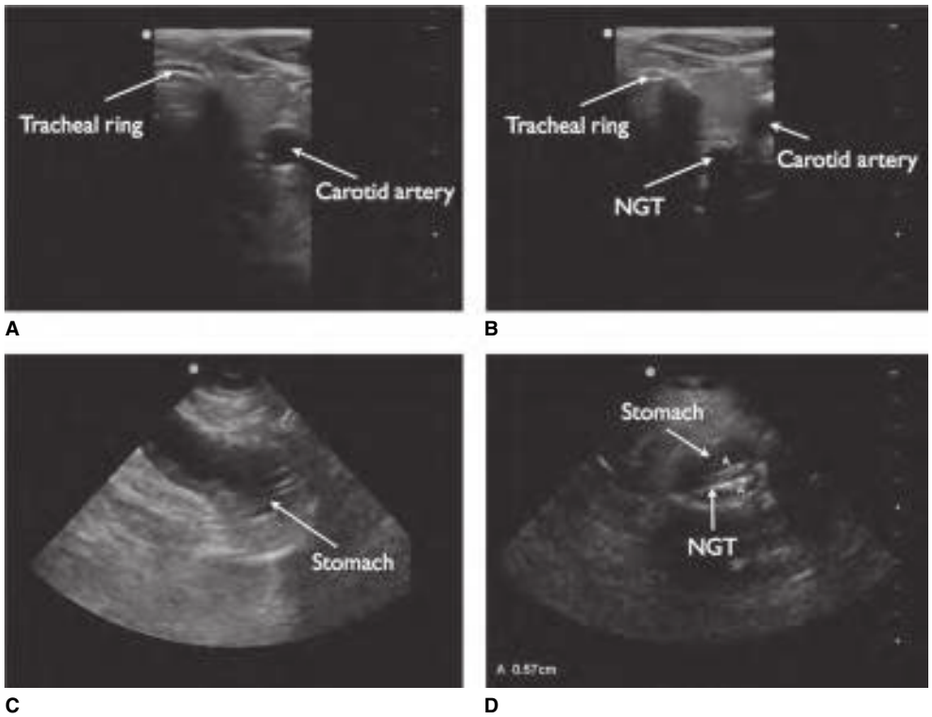

FIGURE 75-8. Ultrasound transverse images before and after an NG tube (NGT) insertion. ***A.*** The neck. ***B.*** The neck after placement of an NG tube. ***C.*** The epigastric area demonstrating the distended fluid-filled stomach. ***D.*** The epigastric area with the NG tube visible in the stomach. (Used with permission from reference 52.)

FIGURE 75-10. Securing the NG tube. Apply tincture of benzoin to the bridge of the nose. A piece of tape is attached to the nose with the distal end split into two and intertwined around the NG tube.

prevent being contaminated during the removal. Place the patient in the seated position. Place towels or pads over the patient's neck and chest. Have an emesis basin, tissues, and Yankauer suction immediately available. Disconnect the NG tube from suction. Fold over the proximal end of the NG tube and hold it tightly. Ask the patient to slightly flex their neck, breath in, and hold it. Place a drape or towel around the NG tube as it is exiting the patient's nose. Firmly squeeze the drape around the NG tube. Briskly withdraw the NG tube through the drape. The drape will contain all the secretions and bodily fluids to prevent them from splashing on the patient or the Emergency Physician. Discard the NG tube and the drape.

COMPLICATIONS

There are many complications associated with a NG tube (**Table 75-1**).[55] The most common complication of NG intubation is discomfort in the nasopharynx and oropharynx. Placement in the nares can result in epistaxis if the nasal mucosa is abraded, irritated, or ulcerated. These complications can be reduced or avoided with generous lubrication of the NG tube, the instillation of topical anesthetics, and the instillation of vasoconstrictors prior to inserting the NG tube. Sinusitis may occur from the NG tube obstructing the sinus ostia. These complications are usually of no clinical significance. Difficulty in insertion or removal can result in coiling in the hypopharynx (**Figure 75-6*C***), knotting around the endotracheal tube, or knotting of the distal end (**Figure 75-6*F***).[56,57]

TABLE 75-1 Some of the Complications Associated with the NG Tube

Breakage
Bronchial perforation
Bronchial placement
Discomfort
Double-backing of the tube
Empyema
Epistaxis
Erosion into adjacent structures
Esophageal perforation
Fistulas
Gastric perforation
Intracranial entrance
Kinks in the tube
Knotted tube
Lung perforation
Misplacement
Obstruction
Pneumothorax
Pulmonary hemorrhage
Rupture
Sinusitis

A serious consequence of NG intubation is misplacement into the respiratory tree (**Figures 75-6*D* and 75-6*E***).[58,59] **This is estimated to occur in up to 15% of cases.**[60] The incidence increases in frequency with the patient who has a diminished gag reflex or a decreased level of consciousness. **The presence of a cuffed endotracheal tube does not preclude passage into the respiratory tree.** The NG tube will pass the cuff of the endotracheal tube without significant resistance. Advancing the NG tube into the airway can result in a bronchopulmonary fistula, an empyema, a hydropneumothorax, perforation of a bronchus or the lung, a pneumothorax, or a pulmonary hemorrhage.[60] **These complications are increased if medication or alimentation is infused into the respiratory tree.**

A serious complication of NG tube placement is esophageal perforation.[61] This most often occurs in the posterior wall of the cervical portion of the esophagus and through the cricopharyngeus muscle. Risk factors for esophageal perforation include altered mental status, cardiomegaly, cervical osteophytes, esophageal abnormalities, multiple attempts, stiff NG tubes, and tracheal intubation.[42] Other risk factors for esophageal perforation include esophageal cancer or the ingestion of alkaline substances. Perforation often results in mediastinitis with a subsequent mortality rate of up to 30%.[42] Parenteral antibiotics, prompt recognition, and surgical repair can reduce the mortality rate to less than 10%. The use of softer and smaller NG tubes with generous lubrication can reduce the risk of esophageal perforation. The NG tube can rarely perforate the stomach in cases of connective tissue disease, gastric surgery, and peptic ulcer disease.[62]

Use caution when inserting an NG tube in patients who have suffered trauma to the face, neck, and/or skull, or if they have medical conditions affecting these areas.[42,63] Fractures of the base of the skull, cribriform plate, maxilla, nasal walls, orbital floors, and palate may allow a nasally inserted tube to exit the nasal cavity and cause further injury. The NG tube may exit traumatic or medical wounds and penetrate neighboring vascular structures.[63,64]

SUMMARY

NG intubation is commonly performed in the Emergency Department. It is primarily used to evacuate air and stomach contents in the bowel-obstructed, intubated, or poisoned patient. Placement is generally considered easy, although it can be uncomfortable. Studies suggest that generous lubrication, topical anesthetics, and topical vasoconstrictors can diminish the discomfort and reduce the chance of misplacement.

Placement of the NG tube into the airway or coiled in the esophagus can result in serious complications. Auscultation of air has been classically used to determine correct placement. Radiographic confirmation of gastric placement is considered the gold standard. Carefully observe the patient for signs of esophageal perforation.

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76 Activated Charcoal Administration

Jenny J. Lu

INTRODUCTION

The adsorptive capacity of charcoal has been documented since the time of Hippocrates and has been known for centuries. Two independent researchers were responsible for its wide acceptance in the early nineteenth century when each of them performed a demonstration of its effectiveness by ingesting lethal doses of strychnine and arsenic, respectively, followed by charcoal. Both of them survived. The twentieth century has seen charcoal come into wide medical use as further investigation showed its effectiveness at adsorbing a variety of compounds.[1] **Activated charcoal is currently the most commonly used mode of decontamination in the Emergency Department for poisoned patients.**[2]

ANATOMY AND PATHOPHYSIOLOGY

Charcoal is produced by the distillation of the pyrolysis products of vegetable matter or wood. It works by directly adsorbing toxicants via a variety of chemical binding properties and preventing substances in the gastrointestinal tract from being absorbed into the circulation. Enhancement of the adsorptive capability of charcoal is achieved by heating it to a temperature of 900°C and then subjecting it to a stream of oxidizing gas (e.g., carbon dioxide or steam). This process is termed "activation" and creates an internal pore structure that increases the surface area from 2 to 4 square meters per gram to greater than 2000 square meters per gram (**Figure 76-1**).[2] A typical dose of 50 gm of activated charcoal has the surface area of 10 football fields. **Activated charcoal is not absorbed from the intestinal lumen nor is it modified by the numerous enzymes that aid in the digestion of food.** It passes through the intestinal tract unchanged and is expelled as a sticky black substance.

Some activated charcoal preparations contain sorbitol. Sorbitol is used as a flavoring agent to make food, drinks, and medications more palatable. It is also used as a hyperosmotic laxative agent. It is poorly absorbed from the gastrointestinal tract and is converted into fructose by the liver. **Its limited absorption results in an increased volume of water being secreted into the intestine causing an increased intraluminal pressure that stimulates catharsis.**

Activated charcoal is usually used for decontamination, in single or multiple doses. **Multiple doses of activated charcoal are considered in exposures to drugs with sustained- or delayed-release preparations or in situations where there is evidence of ongoing absorption.** Multiple-dose activated charcoal can help enhance the elimination of certain compounds that exhibit enteroenteric or enterohepatic recirculation. A diffusion gradient is created in the intestine when multiple-dose activated charcoal is given for enhanced elimination due to the enormous adsorptive ability of charcoal to bind free toxicant. The intestinal mucosa functions as a semipermeable membrane, allowing absorbed drug or toxin to diffuse from the capillaries back into the lumen of the intestine. Activated charcoal can then adsorb and "trap" the toxin within the intestinal lumen for its subsequent excretion in the stool.[3,4] This mechanism essentially dialyzes the blood in the capillaries of the intestinal circulation, hence the term "gastrointestinal dialysis."

FIGURE 76-1. Activated charcoal.

Drugs and drug metabolites secreted into the bile may be absorbed and trapped by activated charcoal within the intestinal lumen. Activated charcoal may shorten the half-life by adsorbing these substances, although evidence for clinical benefit remains limited. Substances that may benefit from multiple-dose activated charcoal for enhanced elimination include phenobarbital, theophylline, dapsone, diazepam, amitriptyline, carbamazepine, phenytoin, quinine, salicylates, piroxicam, digoxin, doxepin, quinine, tricyclic antidepressants, and meprobamate.[3-5]

Activated charcoal has an excellent safety profile overall. It can be used in pregnancy, in lactation, and in the pediatric population. Its use is widely accepted by Emergency Physicians without good randomized controlled clinical studies demonstrating a mortality outcome benefit.[4] Studies supporting activated charcoal's efficacy include in vitro, animal, and volunteer studies. Activated charcoal has been found to be just as efficacious when compared with emetics or gastric lavage as a single modality for decontamination. A large randomized controlled trial examined gastric emptying procedures and compared ipecac-induced emesis, gastric lavage, and activated charcoal in patients with drug overdoses.[6] No significant differences were found in clinical severity, length of hospital stay, morbidity, or mortality among the treatment groups. These findings are similar to those found in other studies.[7,8]

Ipecac-induced emesis, activated charcoal, and gastric lavage were compared in volunteers 1 hour after the ingestion of ampicillin.[9] Activated charcoal was found to be superior in decreasing the absorption of blood ampicillin levels by 57%. Gastric lavage decreased absorption by 32%, and emesis decreased it by 38%. The authors concluded that activated charcoal without a gastric emptying procedure may be the preferred method of gastrointestinal decontamination. Limitations of the study included using subtoxic doses of ampicillin on volunteers and the small sample size.

INDICATIONS

Activated charcoal is currently considered the modality of choice for gastrointestinal decontamination.[10,11] It is generally indicated for ingestions considered to be serious or life-threatening and that are adsorbable to charcoal.

The optimal timing for administration of activated charcoal remains debatable and depends on the exposure and the individual case. It is generally agreed that the sooner after an ingestion the better.[12] There may be clinical scenarios in which delayed administration is appropriate, including exposures where there is evidence of delayed or ongoing absorption (e.g., salicylates). The American Academy of Clinical Toxicology has noted that there is insufficient evidence to support or exclude the use of activated charcoal more than 1 hour after a toxic ingestion.[12-14] One observational study found lower rates of liver injury in acetaminophen-poisoned patients presenting more than 4 hours after ingestion who were administered both activated charcoal and *N*-acetylcysteine.[15] The pros and cons of activated charcoal should be weighed on a case-by-case basis, regardless of time frame.[16-20]

CONTRAINDICATIONS

One absolute contraindication to the use of activated charcoal is in the patient with an unprotected airway. Patients with depressed levels of consciousness and those at risk for seizures from their toxic ingestions are at higher risk of aspiration. It is not recommended that patients be endotracheally intubated solely for the purpose of administering activated charcoal. Patients with absent bowel sounds, evidence of gastrointestinal obstruction, or recent gastrointestinal surgery should not be given activated charcoal due to the risk of bezoar formation or perforation and leakage of charcoal into the abdominal cavity.

Activated charcoal is not effective against xenobiotics such as acids and alkalis, alcohols, fluoride, heavy metals, inorganic salts, iron, lithium, or potassium. Its use is limited in liquid ingestions where absorption is rapid. It is unlikely to be helpful in hydrocarbon and pesticide ingestions. Administration of activated charcoal can render endoscopic visualization technically difficult. **Charcoal combined with cathartics is contraindicated in young children due to the risk of severe electrolyte imbalances and should be used with caution in renally impaired patients.**[21,22]

EQUIPMENT

Several charcoal preparations are available (**Figures 76-2 and 76-3**).[23] These include capsules, tablets, powder, oral suspensions, and suspensions containing sorbitol. Powder, premixed oral suspensions, and suspensions with sorbitol are the only preparations indicated for use in an acute poisoning. Powder is available in doses from 15 to 500 gm that must be mixed with water to make a slurry. The premixed suspension is available in strengths of 12.5 gm/60 mL to 50 gm/240 mL. Activated charcoal suspensions with sorbitol are available containing 25 to 50 gm of activated charcoal and 25 to 96 gm of sorbitol in a volume of 120 to 150 mL. Some Emergency Departments and hospitals mix sorbitol with the activated charcoal when it is required. The recommended dose is 1 to 2 gm/kg in adults, 1 to 2 mL of the 70% solution of sorbitol in adults, and 4.3 mg/kg of the 35% solution of sorbitol in children.[22]

PATIENT PREPARATION

The airway should be evaluated in any patient who has ingested a central nervous system depressant, tricyclic antidepressants, a sympathomimetic or other agent that may result in seizures, or any substances that can cause an altered mental status. Airway protection should be provided if clinically indicated prior to the administration of activated charcoal in patients who are comatose, drowsy, obtunded, unconscious, or have an absent or impaired gag reflex.

TECHNIQUE

Activated charcoal should ideally be administered as early as possible after an ingestion as its efficacy is reduced with the passage of time. Thoroughly mix the activated charcoal slurry prior to opening the container. The activated charcoal may settle at the bottom of the container. **An aqueous slurry should be used rather than tablets or powder.** A single dose is typically administered. Multiple doses may be considered if the toxicant is expected to be absorbed slowly (e.g., massive ingestions or sustained-release preparations) or if enhanced elimination of already absorbed substances (i.e., through enteroenteric or enterohepatic recirculation) is the objective. The ideal dose for the single or initial dose is 5 to 10 times the amount (in grams) of toxicant ingested. Standard practice is to administer 1 gm/kg (i.e., 50 to 100 gm to an adult or 10 to 25 gm to a child < 5 years).[23] Repeated doses may range from 15 to 30 gm every 2 to 4 hours. The optimal dosing is not known.[23] An alert patient typically tolerates drinking the slurry well. Nausea and vomiting can be alleviated with the administration of an antiemetic medication.

Sorbitol may be premixed with the activated charcoal. The sorbitol will decrease gastrointestinal transit time and may prevent bezoar formation. **Sorbitol-containing charcoal should be given only to adults and only with the first dose. Attention should be paid to verify whether or not the charcoal preparation contains sorbitol, especially where multiple doses are given.** Monitor hydration and electrolyte levels if multiple doses of sorbitol-containing activated charcoal are given.

A nasogastric tube (Chapter 75) can be placed to facilitate administration of the activated charcoal. **Correct placement of the nasogastric tube in the gastrointestinal tract is essential prior to activated charcoal administration.** Confirmation of gastric placement can be achieved with the auscultation of insufflated air through the nasogastric tube into the stomach over the epigastrium, the aspiration of stomach contents through the nasogastric tube, or radiographically demonstrating the nasogastric tube below the level of the diaphragm. Minimize gastric pressures caused by the overly aggressive administration, which can increase the risk of aspiration.[24]

COMPLICATIONS

The most feared complication of activated charcoal use is pulmonary aspiration. Activated charcoal can cause severe pneumonitis that can lead to respiratory failure, prolonged ventilatory support, and death. The activated charcoal can cause an empyema and bronchiolitis obliterans if aspirated. **Avoiding aspiration is a priority when giving activated charcoal.** Awake patients must have an intact gag reflex in order to protect against aspiration. Errant placement of a nasogastric tube into the trachea or bronchi and past the inflated cuff of a properly placed endotracheal tube can result in aspiration.[21] **The proper placement of a nasogastric tube must be confirmed prior to instilling activated charcoal.** Reports of life-threatening pulmonary complications as a result of aspiration have also been reported with the removal of the nasogastric tube subsequent to activated charcoal administration.[25] Aspiration of activated charcoal can occur in the intubated patient with an inflated endotracheal cuff and properly placed nasogastric tube.[26]

Activated charcoal containing sorbitol or given with magnesium decreases the risk of intestinal obstruction and constipation.

A

B

C

D

E

FIGURE 76-2. Several of the preparations of activated charcoal used in the Emergency Department.

Cathartics may induce other problems. Electrolyte disturbances can result despite the benefits of decreasing transit time through the gut and the concomitant decrease in toxicant absorption. Cathartic-induced hypernatremia, typically a complication seen in the very young, can lead to serious central nervous system damage, brain swelling, long-term morbidity, and death. Sorbitol dosing in children is not clearly established. The typical premixed preparations are not appropriate in the pediatric population. Hypermagnesemia is seen in the adult population when a magnesium-containing cathartic is given to a patient with gastrointestinal abnormalities or

FIGURE 76-3. Capsules of activated charcoal.

renal compromise. Dehydration is a problem encountered in the elderly and very young. Osmotic volume loss from the gastrointestinal tract can cause these fragile populations to decompensate.[21]

Intestinal obstruction is a rare complication but carries significant morbidity. Case reports of obstruction and perforation requiring a laparotomy have been reported. Charcoal bezoars are the usual culprits. Bezoars can form when intestinal motility is compromised, allowing continued absorption of water from the intestinal contents. Bonds form between charcoal particles once sufficient water has been absorbed and the mass continues to harden. Ingestions of anticholinergic substances, antiperistaltic co-ingestions, or medications administered during the hospital course have also been implicated. Use caution when giving these patients narcotic or anticholinergic medications. Patients receiving multiple-dose activated charcoal therapy or activated charcoal without cathartics are at greater risk. Constipation is a milder form of this complication and is uncommon and rarely of clinical significance.[21]

SUMMARY

Activated charcoal is currently the most commonly used modality of gastrointestinal decontamination in the acutely poisoned patient. This is despite limited evidence demonstrating improved clinical outcomes or decreased mortality rates. Administration of activated charcoal is relatively straightforward and well tolerated. The usual dose of activated charcoal is 50 to 100 gm in an adult and 1 gm/kg in a child. Preparations containing a cathartic (e.g., sorbitol) can be used with the first dose in the adult unless contraindicated. Cathartics and charcoal preparations containing sorbitol should not be used in children or those with renal impairment due to the risk of electrolyte disturbances. The most significant complications occur as a result of aspiration and can be minimized by paying close attention to the patients at risk (i.e., a decreased level of consciousness or a weakened gag reflex) and by checking placement of the nasogastric tube prior to administration of activated charcoal. The risks and benefits of multiple-dose activated charcoal for decontamination or for enhanced elimination must be carefully weighed. Activated charcoal may be judiciously administered in appropriately selected poisoned patients as long as there are no contraindications. The decision to use activated charcoal should be determined on a case-by-case basis by the treating Emergency Physician, with possible consultation from a poison control center or a Toxicologist.

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77 Gastric Lavage

Jenny J. Lu and Rosaura Fernandez

INTRODUCTION

Gastric lavage is a method of gastrointestinal decontamination, performed in the setting of an acute poisoning by ingestion, to decrease the absorption of substances in the stomach. This technique was first described in 1812 and has been used for nearly 200 years.[1] It was repopularized in the 1950s and 1960s and thrived during the heyday of the "tricyclic era" of the 1970s and 1980s. **The use of gastric lavage in the Emergency Department has decreased greatly in modern toxicology. Various sources continue to reiterate the potentially serious complications with this procedure. Trends toward evidence-based medicine and the growing body of experimental and clinical data point to the limited efficacy of gastric lavage.** Gastric lavage was performed in approximately 10.3% of all ED-treated poisoning cases between 1998 and 2003, a decrease from 18.7% during the period of 1993 through 1997.[2] The increasing use of other modalities for gut decontamination (e.g., activated charcoal) has further limited the role of gastric lavage.[1,3] The American Academy of Clinical Toxicology (AACT) states that "gastric lavage should not be performed routinely, if at all, for the treatment of poisoned patients."[4]

ANATOMY AND PATHOPHYSIOLOGY

The administration of activated charcoal is the current decontamination measure of choice. There remain very few indications for performing gastric lavage. These indications include a highly toxic or potentially lethal ingestion presenting acutely where no antidote exists, no antidote is available, or other usual therapies are ineffective. Gastric lavage has never been demonstrated to decrease mortality or improve the final outcome of the patient. The decision should be made with consideration to the specifics of the individual case.

The optimal timing to perform gastric lavage is controversial. **The efficacy of gastric lavage diminishes rapidly over time.** Any benefit of gastric lavage would likely be gained if performed promptly and within 1 hour of an oral ingestion.[3] Authors agree that sooner is better. The range of recovered ingestant is highly variable at each time point following an ingestion in volunteer and overdose studies. The trend for mean removal of ingestants is 90% recovery at 5 minutes postingestion, 45% recovery at 10 minutes, 30% recovery at 19 minutes, and as little as 8% recovery at 60 minutes.[3] Delayed gastric lavage should be considered only in a severe poisoning where delayed gastric emptying is suspected. Some toxicants or co-ingestants (e.g., anticholinergics or opioids) may cause delayed gastric emptying, whereas others may form masses or concretions in the stomach. Removal of a percentage of the ingested dose may theoretically lessen the severity of the poisoning in some cases, but these benefits remain unproven.

Nasogastric placement of a gastric lavage tube is not advised. The orogastric route should be used to avoid traumatic injury to the nasal mucosa, nasal turbinates, and nasal septum. **Nasogastric tubes should be limited to liquid overdoses.** Lavage fluid that does not return through the nasogastric tube will pass through the pyloric sphincter and could allow increased absorption of the toxicant.[5] Sustained-release tablets or capsules are particularly large in size and unlikely to be removed through a nasogastric tube or through the fenestrae of a 40 French orogastric lavage tube (**Figure 77-1**).

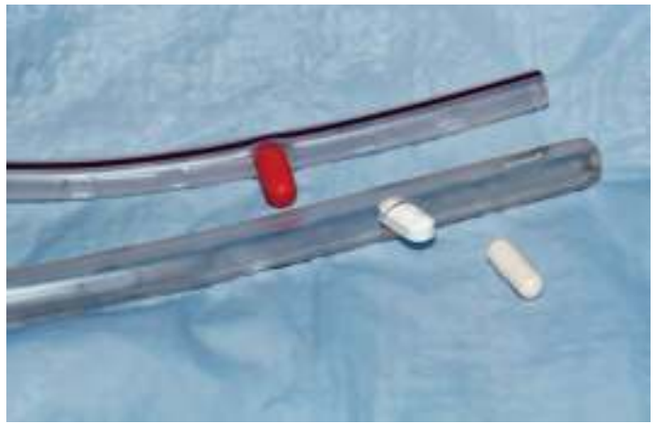

FIGURE 77-1. Orogastric lavage tubes demonstrating sideport size. A 36 French tube with a Stresstab 600 multivitamin in the sideport (*top*). A 40 French tube with a 450 mg sustained-release theophylline tablet in the side port (*middle*). A generic 250 mg ampicillin tablet (*bottom*).

Placement of gastric lavage tubes in the pediatric population is precarious (**Figure 77-2**).[6] A formula for the depth of insertion has been prospectively evaluated to ensure adequate placement in this population and is depicted graphically in **Figure 77-3**.[6,7] The size of the oropharyngeal aperture and esophagus are proportional to the

FIGURE 77-2. Malposition of an orogastric tube placed for gastric lavage in a pediatric patient. (Courtesy of Ann E. Klasner, MD, MPH.)

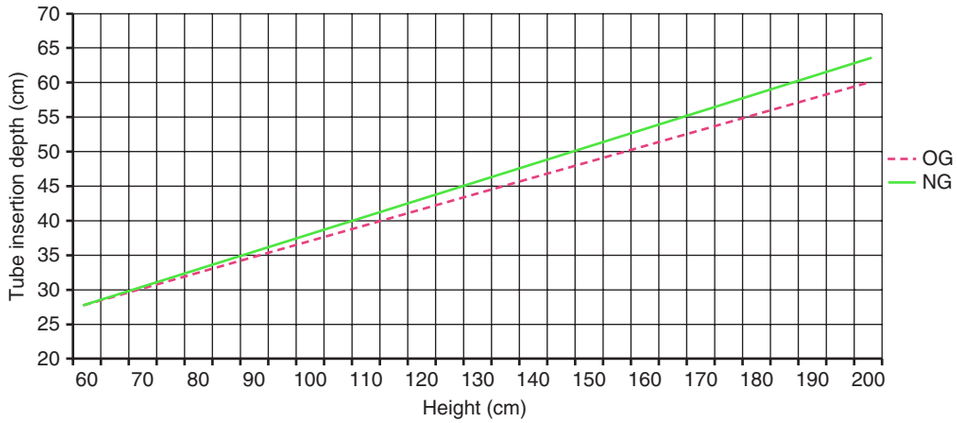

FIGURE 77-3. Estimated lavage tube insertion depth in children based on their height. The lengths for both nasogastric (NG) and orogastric (OG) tubes are represented on the graph. (Used with permission from reference 6.)

size of the individual. **Appropriate tube diameter is essential to minimizing complications.**

INDICATIONS

Gastric lavage may be considered for immediate stomach emptying within 1 hour for an ingestion determined to be potentially serious or life-threatening or where antidotal or other supportive modalities are unavailable or inadequate (**Table 77-1**).[4,8] Preference should first be given to other less invasive modalities of gastrointestinal decontamination (e.g., activated charcoal). Consideration should be given to the specific characteristics of the toxicant, the ingested dose, and the risks versus benefits of performing the procedure. It may be considered beyond 1 hour in patients with known or suspected delayed gastric motility (e.g., in the setting of anticholinergic or opioid ingestion) or where the evidence of a durable mass (e.g., concretion) of pill fragments is a concern.

TABLE 77-1 Indications for Performing a Gastric Lavage

Acute presentation (within 1 hour) and:

1. **Evident or high risk of morbidity or mortality**

Beta-blockers	Heterocyclic antidepressants
Calcium channel blockers	Iron
Chloroquine	Paraquat
Colchicine	Salicylates
Cyanide	Selenious acid
Heavy metals	

2. **Poor absorption by activated charcoal**
   - Heavy metals
   - Iron
   - Lithium
   - Toxic alcohols
3. **Evidence of formed concretion**
   - Enteric-coated preparations
   - Iron
   - Phenothiazines
   - Salicylates
4. **Ineffective or no antidotal therapy available**
   - Cellular poisons (e.g., colchicine)
   - Paraquat
   - Selenious acid

CONTRAINDICATIONS

The contraindications to performing a gastric lavage are summarized in **Table 77-2**. **An absolute contraindication to gastric lavage is in a patient with a depressed level of consciousness who cannot protect their airway.** Gastric lavage should not be performed in combative patients, patients at high risk of seizures, and in those who may be expected to deteriorate rapidly. **Intubating a patient solely for the purpose of gastric lavage is not recommended.** Gastric lavage is contraindicated in caustic ingestions. Local mucosal damage amplifies the risk for traumatic perforation. Gastric lavage should not be performed to retrieve large pills, large foreign bodies, or sharp foreign bodies. It is relatively contraindicated in hydrocarbon ingestions, especially where there is high pulmonary aspiration potential. Significantly abnormal upper airway or upper gastrointestinal anatomy (e.g., anomalies, strictures, or a fresh interposition graft) may restrict the use of gastric lavage.

Gastric lavage is contraindicated in the vomiting patient due to the risk of aspiration and perforation. Vomiting is common after many overdoses and may itself serve as a "natural" decontamination measure. Multiple episodes of emesis may clear the majority of a toxicant from the stomach and obviate the need for gastric lavage. Vomiting in the setting of caustic ingestions can cause further harm by reexposing the gastrointestinal mucosa to the caustic substance. Attempts at gastric intubation in the setting of an actively vomiting patient are likely to be met with minimal success and may cause injury.[9]

Gastric lavage is unlikely to change the outcome of a nontoxic or minimally toxic ingestion. The risk-benefit ratio is unacceptably high in such cases. The determination of whether or not to perform

TABLE 77-2 Contraindications to Performing a Gastric Lavage

- Abnormal or absent pharyngeal or upper gastrointestinal anatomy
- Active or substantial antecedent vomiting
- Caustic ingestion
- Coagulopathy
- Combative or uncooperative patient
- Decreased level of consciousness
- Diminished or absent airway reflexes
- Large pills
- Large or sharp foreign body
- Nontoxic or minimally toxic ingestion
- Significant aspiration risk (e.g., hydrocarbon ingestion)
- Seizures/high risk of seizures

a gastric lavage must be individualized. Consult a Medical Toxicologist or poison control center in questionable cases.

EQUIPMENT

- Pulse oximeter
- Cardiac monitor
- Noninvasive blood pressure monitor
- Protective clothing
- Bite block
- Oral airway
- Endotracheal tube and intubation equipment (Chapter 18)
- Endotracheal tube stabilizer
- Emesis basin
- Suction source, tubing, and Yankauer suction catheter
- Orogastric tube 36 to 50 French for adults or 24 to 34 French for children
- Large bulb suction device or 50 mL Toomey syringe
- Warm water or normal saline for adults
- Warm normal saline for children
- Water-soluble lubricant
- Activated charcoal (optional)
- Resuscitative equipment

All required instrumentation should be gathered at the bedside prior to initiating the procedure. A variety of orogastric lavage tubes are available. Most have a distal port and at least one sideport. A semirigid tube is preferable to soft rubber or polyvinyl chloride collapsible tubes. Several additional ports should be cut in the sides of the tube near the tip to maximize the return of pill fragments if the tube has only a single sideport. Examples of rigid tubing are shown in **Figures 77-1 and 77-4**. A larger tube diameter provides less flexibility and ensures the tube is less likely to kink, collapse, or curl back on itself. Larger-diameter tubes are more likely to facilitate retrieval of larger pill fragments. A 30 to 50 French lavage tube is preferred in an adult. A 30 to 34 French tube is adequate for an adolescent. Small children can generally accommodate 24 French tubes. Gastric lavage is generally contraindicated in neonates and infants.

There are numerous types of gastric lavage systems. Open systems are less expensive, messy, and time-consuming to use. A passive open system uses gravity to instill and drain the lavage fluid. An active open system uses a large syringe to inject and aspirate lavage fluid through the orogastric tube. The syringe must be removed from the orogastric tube to be filled with fresh lavage fluid, and used lavage fluid must be discarded after each lavage cycle. Closed systems are commercially available, single-patient use, self-contained, easy to use, and do not cause a mess (**Figure 77-4**). The Emergency Physician should be familiar with the type of equipment available at their institution.

Gastric lavage solution typically consists of tap water or normal saline. **Do not use tap water in small children as this can result in electrolyte abnormalities.** Specialized lavage solutions may be indicated if the ingested substance is fluoride, formaldehyde, iodine, iron, or oxalic acid. Fluoride ingestions may be lavaged with 15 to 30 gm/L of calcium gluconate to produce an insoluble calcium fluoride. Formaldehyde ingestions may be lavaged with 10 mg/L of ammonium acetate to produce the nontoxic substance methenamine. Iodine ingestions may be lavaged with 75 gm of cornstarch in 1 L of water. Iron ingestions may be lavaged with 2% sodium bicarbonate (e.g., 50 mEq in 150 mL of normal saline) to produce the insoluble ferrous carbonate. Oxalic acid ingestions may be lavaged with 15 to 30 gm/L of calcium gluconate to form the insoluble calcium oxalate. **These specialized lavage solutions should be used only in consultation with a Medical Toxicologist or poison control center.**

FIGURE 77-4. Example of a closed lavage pump system. (Courtesy of Kimberly-Clark Corporation.)

PATIENT PREPARATION

Explain the indications, details of the procedure, risks and benefits, and alternatives with the patient and/or their representative. Informed consent should be obtained when possible.

Place the patient in the left lateral decubitus position and in 15° to 20° of Trendelenburg. This position is intended to maximize gastric emptying. The supine and lateral decubitus positions are associated with a higher risk of pulmonary aspiration in comatose and mechanically ventilated patients.[10-13] Most gastric lavages can be performed safely and effectively with the conscious patient placed in the semi-upright position. The use of a topical anesthetic spray into the oropharynx may decrease the patient's gag reflex and allow easier passage of the orogastric tube. This can also increase the risk of aspiration. It is recommended that a cardiac monitor, noninvasive blood pressure cuff, and pulse oximeter be placed on the patient prior to performing the lavage.

TECHNIQUE

Measure the length of the orogastric lavage tube to be inserted (**Figure 77-5**). The length should be marked with a permanent marker or a piece of surgical tape. Liberally lubricate the tip of the lavage tube. Place a bite block into the patient's mouth if they are conscious. A bite block or oral airway may preclude biting of the tube by an uncooperative or stuporous patient.[14]

Gently insert the lavage tube into the patient's mouth and direct it into the hypopharynx. Flexion of the patient's neck may facilitate

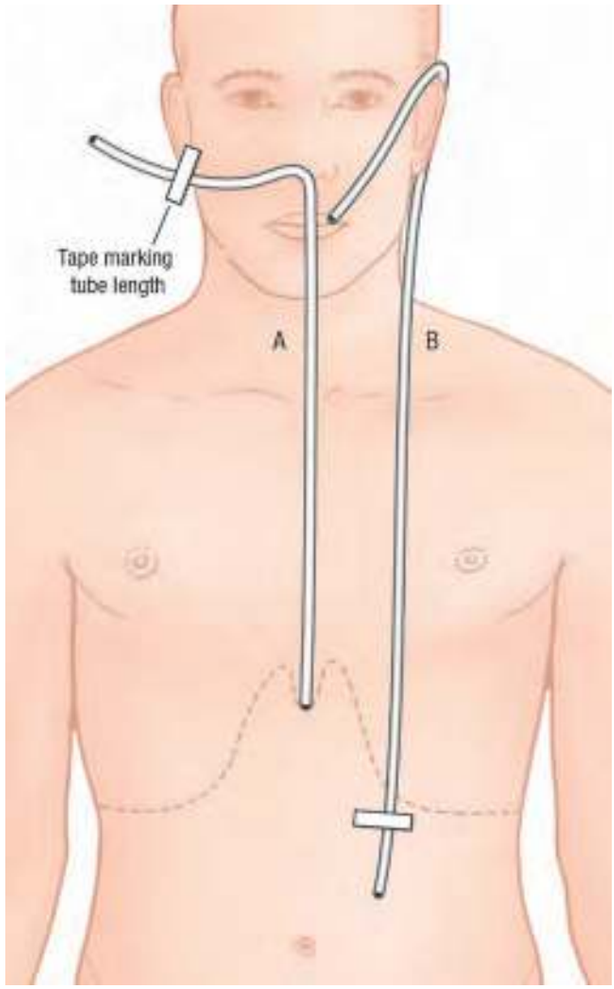

FIGURE 77-5. Determining the proper length of orogastric tube to insert. ***A.*** The length is determined by the distance from the xiphoid process to the tip of the nose to the earlobe. ***B.*** The length is determined by the distance from the tip of the nose or lip, around the left ear, and to just below the left costal margin. A piece of tape is used to mark the distance on the orogastric tube.

passage of the tube into the esophagus and avoid endotracheal insertion. Conscious and cooperative patients may be asked to swallow water through a straw or their saliva to facilitate passage of the tube. Stridor, cough, or cyanosis indicates that the lavage tube is in the airway and should prompt removal of the tube. Significant resistance is occasionally met in the hypopharynx. Apply gentle pressure to the tube while instructing the patient to swallow allows passage through the upper esophageal sphincter. **The tube should not be forced, as misplacement may damage the larynx or perforate the pyriform sinus.** Slowly advance the tube to the premeasured depth.

Confirmation of intragastric tube placement must precede instillation of any fluid through the tube. Proper placement should be confirmed by aspiration of gastric contents, auscultation of insufflated air over the epigastrium, and/or radiography. Gastric irrigation should be preceded by aspiration of available gastric contents. The initial aspirate may be sent for toxicologic assay.

Perform the gastric lavage. Instill normal saline or tap water through the lavage tube. The lavage fluid should ideally be warmed to body temperature. This is often not practical, and room temperature lavage fluid is satisfactory. Instill aliquots of 10 to 15 mL/kg to a maximum of 300 mL of the irrigant solution. Instillation of larger volumes may result in vomiting, pulmonary aspiration, and the passage of gastric contents past the pyloric sphincter where subsequent absorption may occur.[2] Lavage aliquots may be instilled by placing a funnel in the free end of the lavage tube and allowing gravity instillation, or they may be infused with a Toomey syringe. Remove the lavage fluid after a brief (i.e., 1 to 2 minutes) equilibration period by either gravity drainage into an emesis basin or aspiration (e.g., a syringe or suction bulb). Repeat the lavage process until 2 to 3 L of irrigant has been used in the adult or the lavage fluid is free of particulate matter and pill fragments. Alternatively, closed systems are available for both instilling and suctioning lavage fluids through a common tube (**Figure 77-4**).

Activated charcoal (Chapter 76) may be administered, if desired, through the lavage tube before it is removed. A dose of 1 gm/kg of body weight is typically given initially, either in a premixed slurry or diluted in normal saline or tap water (e.g., 30 gm charcoal per 240 mL of diluent).

Remove the lavage tube. It is recommended for the Emergency Physician to wear gloves, a mask with an eye shield, and a gown to prevent being contaminated during the removal. Place towels or pads over the patient's neck and chest. Have an emesis basin, tissues, and Yankauer suction immediately available. Disconnect the lavage tube from its proximal attachment. Fold over the proximal end of the lavage tube and hold it tightly. Ask the patient to slightly flex their neck, breath in, and hold it. Place a drape or towel around the lavage tube as it is exiting the patient's mouth. Firmly squeeze the drape around the lavage tube. Briskly withdraw the lavage tube through the drape. The drape will contain all the secretions and bodily fluids and prevent them from splashing on the patient or the Emergency Physician. Discard the lavage tube and the drape.

Further gastric access, when needed, should be provided by the subsequent placement of a smaller-bore nasogastric tube.

ALTERNATIVE TECHNIQUES

An alternative sequence could be considered because of concern over toxicants passing or being pushed through the pyloric valve during the gastric lavage procedure. Administer activated charcoal after the initial aspiration of gastric contents through the lavage tube. Perform the gastric lavage anticipating that a significant amount of activated charcoal will be removed along with the ingestant. Infuse a second dose of activated charcoal through the lavage tube before it is removed. The rationale here is an attempt to make charcoal available to adsorb toxicant from any gastric contents pushed into the small bowel during the lavage procedure.[1] It is reasonable to consider even though no data support this technique.

ASSESSMENT

Continuously monitor the patient and reassess them throughout the procedure. Strict adherence to the procedures noted will help minimize the risk of complications. Prompt removal of the lavage tube at the end of the procedure will help decrease the risk of delayed complications.

AFTERCARE

Most patients requiring gastric lavage for an overdose or poisoning usually require inpatient monitoring for their poisoning. A situation wherein a patient requires gastric lavage and is then sent home immediately is unlikely. An observation period of 6 to 8 hours for the immediate complications of gastric lavage and the effects of the ingested substance is appropriate.

COMPLICATIONS

The complications associated with gastric lavage can be minimized by careful patient selection and technique (**Table 77-3**).[9] Placement of the lavage tube can result in mucosal injury, bleeding, esophageal

TABLE 77-3 Complications Associated with Gastric Lavage

Cardiac dysrhythmias
Electrolyte abnormalities
Empyema
Esophageal tear or perforation
Gastric perforation
Hypothermia
Laryngospasm
Nasal, oral, or pharyngeal injury
Pneumothorax
Pulmonary aspiration
Pyriform sinus perforation
Tracheal placement
Tube impaction

perforation, gastric perforation, or endotracheal placement. Monitor the patient for cardiac arrhythmias, hypoxemia, and tachycardia.[1,8] No more than 10 mL/kg or 300 mL aliquots of lavage fluid should be used to prevent vomiting, aspiration, or pushing of gastric contents into the small bowel. The impaction of a lavage tube may prevent its removal.[1,9,15] **Do not use force to remove the lavage tube, as this may injure or rupture the stomach or esophagus.** Evaluate the tube using fluoroscopy or plain radiographs. A lavage tube that is kinked or knotted will require endoscopically aided or surgical removal.

Gastric lavage with large volumes of cold fluid can result in hypothermia. Warmed lavage fluid should be used if available, although this is somewhat controversial. Warm lavage fluid may dissolve more of the toxicant and allow rapid access of gastric contents past the pylorus to be absorbed into the systemic circulation. Electrolyte abnormalities may result, especially in children, if the lavage fluid is hypotonic (i.e., tap water). **The use of normal saline for gastric lavage is recommended.**

It is usually unknown if pills and/or pill fragments remain within the patient's stomach when they present to the Emergency Department. The routine use of CT scans to identify pills and/or pill fragments in the stomach cannot be recommended. Two case reports used CT scans to determine that pills and/or pill fragments remained in the stomach, and subsequently, gastric lavage was performed.[16,17] There is no evidence that the expense and radiation exposure prevents any morbidity or mortality or provide any benefit to the patient.

SUMMARY

Gastric lavage is an invasive decontamination procedure to empty the stomach following a toxic ingestion. Its use has significantly decreased. There are rare indications in which gastric lavage could be considered. Maximal efficacy of the procedure would be obtained if performed within 1 hour of an acute ingestion. Gastric lavage is not indicated in the setting of a nontoxic ingestion, minimally toxic ingestion, or where antidotes exist and are readily available. Complications can be minimized if the procedure is performed cautiously. Consultation with a Medical Toxicologist or poison control center is prudent and can provide valuable information regarding the indications, contraindications, complications, and techniques associated with gastric lavage.

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78 Whole Bowel Irrigation

Henry D. Swoboda and Steven E. Aks

INTRODUCTION

Whole bowel irrigation is the infusion of polyethylene glycol electrolyte lavage solution into the stomach at flow rates higher than are otherwise commonly used. This technique can be used to decontaminate the gastrointestinal tract after an acute toxic ingestion or overdose. **The goal is to give a large volume of a balanced electrolyte solution rapidly to flush the bowel without creating electrolyte disturbances while removing the ingestant.** Most of the literature supporting its use is in the form of case reports or case series, with cohort studies indicating benefit that is of unclear clinical significance and does not support broad use of the therapy.[1] Demonstrated benefits are small.[2] The indications for whole bowel irrigation are mostly theoretical and will be refined as more extensive data becomes available.[3] The role of whole bowel irrigation remains limited.

ANATOMY AND PATHOPHYSIOLOGY

Current methods of gastrointestinal decontamination (i.e., activated charcoal, emesis, and gastric lavage) focus primarily on decontaminating the stomach. Absorption of most toxicants occurs principally

TABLE 78-1 Conditions in Which Whole Bowel Irrigation Can Be Considered

1. Acute, life-threatening, or serious ingestion and hemodynamically stable
2. Any of the following:
   - Concretions
   - Delayed, enteric-coated, or sustained-release preparation
   - Heavy metals
   - Ingested packets of illicit drug
   - Iron
   - Lithium
   - Massive ingestions
   - Toxin or toxicant poorly adsorbed by activated charcoal

in the proximal small bowel. Sustained-release or delayed-release preparations continue to liberate the drug during intestinal transit that is then available for absorption throughout the bowel. Infusion of polyethylene glycol electrolyte lavage solution decreases the enteric transit time, attenuating the contact time of a toxicant with the gastrointestinal mucosa.[4] This reduces absorption of the drug or toxin throughout the gastrointestinal tract. The high molecular weight PEG 3350 polyethylene glycol electrolyte lavage solution (PEG-ELS) is specifically designed to prevent electrolyte and fluid shifts.

INDICATIONS

Whole bowel irrigation may be indicated for acute ingestions where severe or potentially fatal toxicity is anticipated (Table 78-1). Use other decontamination methods (e.g., activated charcoal) if they are known to be effective rather than whole bowel irrigation. PEG-ELS has been shown to decrease the effectiveness of activated charcoal in some poisonings. An initial dose of activated charcoal may increase the effectiveness of whole bowel irrigation.[5,6] Whole bowel irrigation may be indicated in situations where activated charcoal is known to be ineffective. Whole bowel irrigation has been safely used to decrease bioavailability of ingested iron, lithium, and heavy metals, and it may decrease the poisoning severity associated with lithium overdose.[7-12] Whole bowel irrigation has been proposed to be effective in flushing the gastrointestinal tract free of toxicant before absorption of sustained-release preparations.[13] Whole bowel irrigation may speed gastrointestinal transit of ingested packets or vials of illicit drugs ingested by "body packers" who purposely ingest carefully prepared drugs as a means of internal concealment for transportation.[14] The role of whole bowel irrigation in "body stuffers" who hastily ingest drugs to avoid legal prosecution has been questioned.[15] Additional settings may be envisioned where whole bowel irrigation might be useful. There are no data to support broader indications.

CONTRAINDICATIONS

There are few contraindications to performing whole bowel irrigation (**Table 78-2**). **Do not use it in the patient with a potentially compromised or unprotected airway.** Whole bowel irrigation could result in pulmonary aspiration.[16,17] Significant vomiting will hinder the ability to perform whole bowel irrigation. Do not perform whole bowel irrigation in the hypotensive or hemodynamically unstable patient.[16] Other contraindications include abnormal upper airway or upper gastrointestinal anatomy (e.g., anomalies, fresh interposition graft, or strictures). Ingestion of toxic substances that markedly slow gastrointestinal motility (e.g., anticholinergics or opioids) may cause an ileus, diminishing the ability to perform whole bowel irrigation effectively. The administration of polyethylene glycol in a patient with a bowel obstruction will result in vomiting with the potential for aspiration. Do not use polyethylene glycol solution with an actual or suspected perforated bowel. The risk of whole bowel irrigation is quite small, but the procedure is not without effort, expense, and risk of complications.[18] The risk-benefit ratio is high if whole bowel irrigation is used to treat a nontoxic ingestion.

TABLE 78-2 Contraindications to Performing Whole Bowel Irrigation

- Bowel obstruction or perforation
- Gastrointestinal bleed
- Gastrointestinal tract is not intact
- Hypotension or hemodynamic instability
- Ileus
- Indications to go to the Operating Room
- Intractable vomiting
- Nontoxic ingestion
- Potentially compromised or unprotected airway
- Significant gastrointestinal bleeding
- Signs of leakage of cocaine packets

EQUIPMENT

- Nasogastric or enteral feeding tube, size 10 to 12 French
- Water-soluble lubricant
- Nasal decongestant and anesthetic
- Enteral feeding reservoir and tubing
- Polyethylene glycol electrolyte lavage solution (packets or reconstituted)
- Emesis basin
- Stethoscope
- Toomey syringe
- Intravenous extension tubing

The equipment required for whole bowel irrigation is readily available in most Emergency Departments. A small-bore, 10 to 12 French, nasogastric tube is sufficient for an adult or adolescent. An enteral feeding tube can be substituted if a sufficient flow rate can be ensured. A smaller tube is required in infants or very small children. Infants will tolerate an 8 French tube, and a 10 to 12 French tube is adequate beyond the first year.[19,20]

Standard enteral feeding reservoirs and tubing are typically available as packaged kits. An enteral feeding or an enema bag with enteral feed tubing may be substituted. Enteral feeding pumps are not useful as the flow rate through the pump is typically inadequate to perform whole bowel irrigation effectively. Alternatively, an empty 1 L intravenous fluid bag with intravenous extension tubing can be used for the procedure.

Atomizer devices are usually not available in the Emergency Department. Many devices are available commercially. These are disposable devices that are single patient use (**Figure 29-1 and Table 29-1**). A popular device is the MAD line of mucosal atomizer devices (LMA North America, San Diego, CA). They make multiple devices for all topical needs.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Informed consent can be assumed in the case of suicidal ingestions. Inspect the nasal passages and oropharynx to rule out any anatomic abnormalities or obstruction that would preclude the passage of a nasogastric tube.

Place the patient in the upright or semi-upright position (**Figure 78-1**).[20] The supine and lateral decubitus positions are associated with a higher risk of pulmonary aspiration in comatose and mechanically ventilated patients.[21-24] It can be assumed

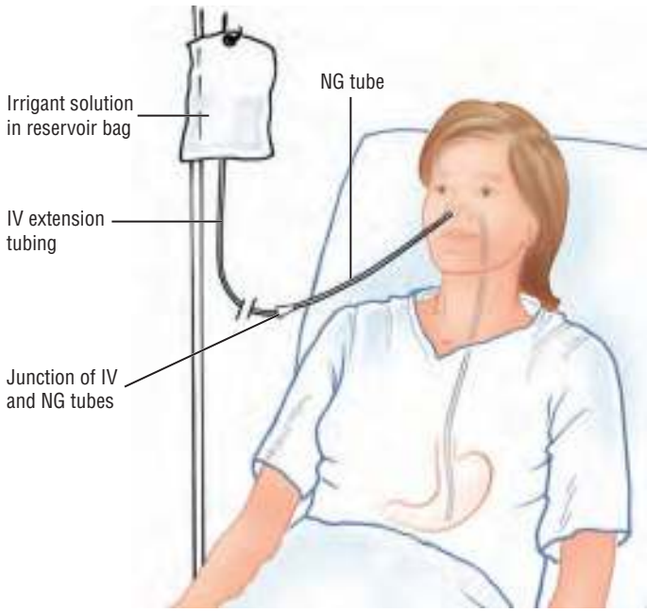

FIGURE 78-1. Setup required to perform whole bowel irrigation. IV, intravenous; NG, nasogastric.

that this positioning risk is similarly increased in patients undergoing whole bowel irrigation.

It is recommended that a cardiac monitor, noninvasive blood pressure cuff, and pulse oximeter be placed on the patient prior to performing whole bowel irrigation. This equipment may also be required based on the patient's physiologic status, the nature of the ingestant or toxicant, and/or other underlying problems.

Choose the smallest diameter nasogastric or feeding tube that will allow adequate flow of polyethylene glycol electrolyte lavage solution into the stomach. Place all necessary equipment at the bedside and assembled prior to beginning the procedure.

Place a cuffed endotracheal tube prior to performing whole bowel irrigation in patients who are comatose, drowsy, obtunded, unconscious, or have an absent or impaired gag reflex. Strongly consider endotracheal intubation in any patient who has ingested a central nervous system depressant, tricyclic antidepressants, a sympathomimetic or other agent that may result in seizures, or any substances that can cause an altered mental status.

TECHNIQUE

Insert a nasogastric tube (Chapter 75). Measure the length of the nasogastric tube to be inserted. Place the tip of the nasogastric tube over the xiphoid process. Extend the tube over the anterior chest, lateral neck, behind the angle of the mandible, and to the tip of the nose. Mark this length with a permanent marker or a piece of surgical tape. Liberally lubricate the tip of the nasogastric tube. The application of a nasal anesthetic and decongestant is optional but can facilitate the passage of the nasogastric tube.

Gently insert the nasogastric tube into the naris. Advance the tube posteriorly along the nasal floor into the nasopharynx and through to the hypopharynx. Flexion of the neck may facilitate passage of the tube into the esophagus and avoid endotracheal insertion. The conscious and cooperative patient may be asked to swallow water through a straw or to swallow their saliva to facilitate passage of the tube. **Coughing, cyanosis, or stridor may indicate endotracheal passage and should prompt removal of the tube.** Gentle pressure and patient swallowing allow passage through the upper esophageal sphincter if significant resistance is met in the hypopharynx. **Do not force the nasogastric tube as misplacement may damage the larynx or perforate the pyriform sinus.** Advance the nasogastric tube to the marked level. Confirm proper placement by the aspiration of gastric contents, by auscultation of insufflated air over the epigastrium using a 50 mL syringe, and/or by radiography. **Confirmation of intragastric placement must precede administration of any fluids through the nasogastric tube. A radiograph must be obtained prior to using the nasogastric tube if any question exists regarding its position.**

Administer 1 gm/kg in children or 50 to 100 gm in adults of activated charcoal via the nasogastric tube prior to administering the PEG-ELS if the suspected toxicant is adsorbed by activated charcoal. Instill the PEG-ELS through a setup such as that shown in **Figure 78-1**. Flow rates are dependent on the size of the patient. Begin instillation at an initial rate of 25 to 30 mL of PEG-ELS per kilogram per hour. Adults may tolerate more than 3 L of solution per hour. The rate may be adjusted somewhat to accommodate patient tolerance (e.g., abdominal distension or vomiting). Continue irrigation until the patient passes the ingestant in the stool or until clear liquid rectal effluent is passed. Liquid stools will continue to be passed after discontinuing whole bowel irrigation. **Stop the irrigation if the patient vomits, develops an ileus, or if gastrointestinal perforation is suspected.**

ALTERNATIVE TECHNIQUES

Some Emergency Physicians may attempt whole bowel irrigation by offering the patient PEG-ELS to drink. This is rarely successful. Experience shows that whole bowel irrigation is ineffectively performed if a nasogastric tube is not placed. Even the most cooperative patient is unlikely to drink the solution at the required administration rate.

ASSESSMENT

Patient assessment must be continuous throughout the process of whole bowel irrigation. Mild abdominal distention, gassiness, and mild abdominal discomfort are common side effects and do not require the discontinuation of the infusion. Providers must be vigilant in monitoring the patient's bowel sounds. Hold the irrigation for 30 to 90 minutes and have the patient reassessed if bowel sounds cease or significant abdominal distension is noted. Resume the irrigation at a reduced rate if bowel sounds return, if the clinical status improves, and if the patient then tolerates the infusion. Significant electrolyte or osmotic shifts do not occur solely from whole bowel irrigation. Electrolytes may be monitored if otherwise indicated for the type of ingestion or for overall patient status.

Of great concern is vomiting with the risk of aspiration. **Maintain the patient's posture in the upright sitting or the semi-upright position to facilitate passage of irrigant solution into the small bowel and to protect against aspiration.** Discontinue whole bowel irrigation if the patent develops altered mental status or hemodynamic instability.

AFTERCARE

Patients who undergo whole bowel irrigation must all be admitted to the hospital for ongoing assessment of the intervention and the underlying ingestion of overdose. Intensive Care Unit or step-down monitoring will be required to ensure adequacy of the intervention and to monitor for complications of the ingestion or overdose. Delayed complications from whole bowel irrigation are unlikely once the procedure is completed.

COMPLICATIONS

Documented complications from whole bowel irrigation include pulmonary aspiration of the irrigant solution and/or ingestant.[16,17] This is especially concerning in the patient with an unprotected and

potentially compromised airway. An ileus and intestinal distension have been documented in a hypotensive patient receiving whole bowel irrigation.[17]

Osmotic and electrolyte abnormalities will not occur with the standard PEG-ELS solutions (e.g., Colyte, GoLYTELY, NuLYTELY). Complications of nasogastric tube placement are well described and can occur (Chapter 75). These are unlikely if proper technique is employed and can be minimized by use of the smallest effective diameter nasogastric tube.

SUMMARY

Whole bowel irrigation is a technique performed to speed gastrointestinal transit and decontaminate the gut after an acute toxic ingestion. Available reports suggest that whole bowel irrigation can decrease bioavailability of toxicants by two thirds in volunteers.[3,6,15] It may be useful where activated charcoal is not expected to adequately bind ingestants (e.g., iron, lithium). Efficacy is still undefined in the setting of sustained-release or delayed-release preparations. Whole bowel irrigation is not the method of choice when more effective methods of gastrointestinal decontamination are possible (e.g., repetitive-dose activated charcoal for sustained-release theophylline). Whole bowel irrigation's role in managing the toxic ingestion remains limited.

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79 Video Capsule Endoscopy for Gastrointestinal Bleeding

Eric S. Levy, Christopher J. Haines, and Grant Wei

INTRODUCTION

Gastrointestinal (GI) bleeding is a very common and potentially life-threatening pathology encountered by the Emergency Physician. It accounts for more than 400,000 hospital admissions each year, with mortality rates as high as 15%.[1] Expedient recognition, diagnosis, resuscitation, and treatment of the underlying etiology are essential in preventing morbidity and mortality. **The initial resuscitation to achieve hemostasis is paramount in the Emergency Department and often overshadows the search for the underlying etiology. The origin of the bleeding directs the algorithm for further management after stabilization.**

Since its introduction and approval by the U.S. Food and Drug Administration (FDA) approximately 15 years ago, video capsule endoscopy has been increasingly used. A pill-sized camera is swallowed, and real-time images of the GI tract are visualized. There have been more than two million video capsule endoscopies performed worldwide and thousands of research papers on its various aspects.[2] Video capsule endoscopy has shown significant promise and is quickly becoming one of the mainstays of medical management when compared to traditional methods of diagnosis and risk stratification.

This chapter introduces the use of video capsule endoscopy, a relatively novel method for the rapid diagnosis and risk stratification of acute GI bleeds. Video capsule endoscopy can be used in the Emergency Department. An Emergency Physician must be familiar with its potential complications and current limitations.

ANATOMY AND PATHOPHYSIOLOGY

The human GI system is divided into three main components derived from their embryological origins (i.e., the foregut, midgut, and hindgut). The foregut gives rise to the abdominal esophagus, the stomach, the proximal half of the duodenum, the liver, the gallbladder, and the pancreas. The midgut gives rise to the distal half of

the duodenum, the jejunum, the ileum, the cecum, the appendix, the ascending colon, and the proximal two-thirds of the transverse colon. The hindgut gives rise to the distal third of the transverse colon, the descending colon, the sigmoid colon, the rectum, and the proximal anal canal. Each of these regions is supplied by a specific branch of the aorta. It should be noted that the branches of these arteries frequently anastomose with each other and provide alternative routes of arterial supply.

The celiac trunk supplies the foregut derivatives. The celiac trunk divides into three branches. The common hepatic artery supplies the liver, gallbladder, stomach, duodenum, and pancreatic head and neck. The left gastric artery supplies the stomach and the esophagus. The splenic artery supplies the spleen, stomach, and the rest of the pancreas.

The superior mesenteric artery supplies the midgut derivatives. The superior mesenteric artery divides into many branches. The inferior pancreaticoduodenal artery supplies the head of the pancreas and the duodenum. The jejunal and ileal branches give rise to 15 to 18 intestinal branches. The middle colic artery supplies the transverse colon. The right colic artery supplies the ascending colon. The ileocolic artery supplies the ileum, cecum, appendix, and proximal ascending colon.

The inferior mesenteric artery supplies the hindgut derivatives. The inferior mesenteric artery divides into many branches. The left colic artery supplies the distal transverse colon and the descending colon. The sigmoid arteries supply the sigmoid colon. The superior rectal artery supplies the distal sigmoid colon and proximal rectum.

The venous drainage of the GI tract primarily flows through the hepatic portal system, which drains through the liver and its sinusoids into the hepatic vein and inferior vena cava. The portal vein is the main venous drainage system and is formed by the combination of the superior mesenteric vein and the splenic veins. The superior mesenteric vein drains portions of the foregut and all of the midgut. The splenic vein drains the spleen, pancreas, portions of the foregut, and all the hindgut derivatives via the inferior mesenteric vein. Similar to the arterial supply, the portal system has many anastomoses that prevent total occlusion should one of the major veins become obstructed. The most important of these anastomoses are in the lower esophagus (i.e., varices), the rectum and anal canal (i.e., hemorrhoids), and the paraumbilical region of the skin (i.e., caput medusae).[3]

Acute GI bleeding is clinically categorized into two general locations, upper and lower, that are separated by the ligament of Treitz. This ligament is a suspensory ligament classically attached to the distal segments of the duodenum or the duodenojejunal flexure, although considerable anatomic variation exists.[4] Bleeding in the upper portion of the GI tract is defined by any lesion proximal to the ligament of Treitz, while a lower GI bleed occurs in any point distal to the ligament of Treitz. The designation of upper versus lower remains essential as the pathophysiology greatly varies, the secondary diagnostic modalities differ, and the therapeutic modalities differ. This chapter focuses primarily on upper GI bleeding, as this is where video capsule endoscopy has been shown to be most useful in the Emergency Department.

Upper bleeding sources account for up to 75% of cases of GI hemorrhage.[5] Upper GI bleeding is further classified into variceal and nonvariceal (**Table 79-1**).[6] **The most common source of nonvariceal bleeding is peptic ulcer disease, which accounts for 50% to 60% of cases.**[7] The etiologies include infection with the *Helicobacter pylori* bacterium, the use of nonsteroidal anti-inflammatory drugs (NSAIDs), physiologic stress, and/or excess gastric acid.[7] The *H. pylori* bacterium disrupts the superficial gastric mucosa and the cellular structure of the mucous membrane, causing increased susceptibility to acid damage. NSAIDs both worsen and predispose to ulceration of the GI tract by inhibition of prostaglandins, specifically cyclooxygenase-1 (COX-1), which plays a role in maintaining the integrity of the mucosal lining.[8,9] The following conditions have been shown to significantly correlate with the increased incidence of gastrin formation and peptic ulcer formation: physiologic stress, chronic ingestion of corrosive substances (e.g., caffeine, alcohol, and tobacco), coagulopathies, mechanical ventilation for more than 48 hours, traumatic brain injuries, traumatic spinal cord injuries, burn injuries, and glucocorticoid therapy.[10] Excess gastric acid as the sole cause of a peptic ulcer is rare but is seen in patients with diseases like Zollinger-Ellison syndrome in which there is increased gastrin production.

TABLE 79-1 The Causes of Upper GI Bleeding

Nonvariceal bleeding	Variceal bleeding*
Esophageal	Duodenal varices
Esophageal cancer	Esophageal varices
Esophageal ulcers	Gastric varices
Esophagitis	
Mallory-Weiss tear	
Gastric	
Dieulafoy's lesions	
Gastric antral vascular ectasia	
Gastric cancer	
Gastric ulcer	
Gastritis	
Duodenal	
Duodenal ulcer	
Hematobilia	
Hemosuccus pancreaticus	
Superior mesenteric artery syndrome	
Vascular fistulae	
Vascular malformations	

*Note: All of the above sources of variceal bleeding are caused by portal hypertension, the most common cause of which is liver cirrhosis. Other causes include focal nodular hyperplasia, portal vein thrombosis, schistosomiasis, and idiopathic.
Source: Adapted from reference 6.

Varices, or tortuous veins, originate from the portal system and account for 10% to 30% of all upper GI bleeds and anywhere from 50% to 90% of bleeds in cirrhotic patients.[7] Bleeding variceal veins have a higher mortality rate than other sources, reaching greater than 20% mortality after 6 weeks despite advancements in medical management.[7] Variceal bleeding can be divided into three main etiologies, all of which can cause bleeding from either the esophagus or the stomach. Prehepatic is when blood flow to the liver is obstructed. Posthepatic is when there is obstructed flow from the liver to the heart. Intrahepatic is the case with cirrhosis or other liver disease affecting hepatic sinusoidal blood flow.

Normal portal venous pressure ranges from 3 to 6 mmHg.[3] Portal venous pressure increases to compensate for decreased blood flow and leads to pressures over 12 mmHg. The excessive pressure can cause the variceal veins to rupture and hemorrhage due to poor vascular wall strength. **It is imperative to distinguish variceal bleeding from other etiologies when the undifferentiated patient enters the Emergency Department due to the high mortality rate of variceal hemorrhages.** While rapidly recognizing and diagnosing the underlying etiology of an upper GI bleed is paramount in the grand scheme of medical management, it should be emphasized that timing is critical. **Priority should initially be given to the initial resuscitation.**

MANAGEMENT OF GI BLEEDING

Recent clinical guidelines have divided the timetable of upper GI bleeding management into the following four stages.[11,12] The first stage is resuscitation, risk assessment, and pre-endoscopy

management. This includes the primary survey, hemodynamic stabilization, airway management, clinical scoring systems for risk stratification, the use of a nasogastric tube, capsule endoscopy or other means to determine the bleeding etiology, the use of initial antibiotics if a variceal bleed is suspected, the use of pre-endoscopic erythromycin to increase endoscopy diagnostic yield, and a proton pump inhibitor to increase the gastric pH to stabilize the clot. Second is endoscopic management within 24 hours of the patient's arrival. It is the direct visualization and stabilization of the lesion with the use of bipolar electrocoagulation, banding, sclerosant clips, or a heater probe. Third is the pharmacological management beyond the initial resuscitation measures. This involves longer term management such as continuous proton pump inhibitor infusions, treatment of suspected *H. pylori* infection, and the use of adjunctive therapies (e.g., albumin infusions, octreotide, vasopressin, or nitroglycerin). Last is the nonendoscopic and nonpharmacological management. This includes surveillance for the possibility of rebleeding and alternative management if bleeding remains refractory (e.g., interventional radiology or surgery).

The intricacies of each stage are beyond the scope of this chapter. **It is important to place video capsule endoscopy's role into the general context of emergency clinical management so that it can be used appropriately for both risk stratification and lesion detection.**

RISK STRATIFICATION

Aside from video capsule endoscopy, there are a couple of methods for initial risk stratification of the patient with an upper GI bleed. These include a thorough history and physical examination followed by clinical scoring systems (e.g., the Glasgow-Blatchford scale and the Rockall score).[12] Both of these can interconnect with laboratory tests and hemodynamic status to paint a clinical picture that would set the stage for further management beyond the endoscopic evaluation.

The history and physical examination are the foremost keys to differentiating etiologies and risk stratifying the upper GI bleed with or without the assistance of video capsule endoscopy. The history should be focused primarily on determining the patient's risk factors for hemorrhage. This includes a thorough review of the medication list for anticoagulants, NSAIDs, or steroids. The pertinent medical history is also important to obtain, especially a history of liver disease or other comorbidities that may predispose to complications with management. Examples are heart or lung disease predisposing to subsequent hypoxemia, cancers or leukemias and their treatments that hinder coagulation, or dementia or hepatic encephalopathy, which increases the risk of aspiration.[13] Systemic signs and symptoms such as chest pain, shortness of breath, dizziness, syncope, decreased urine output, and altered mental status indicate decreased perfusion, suggest active or impending decompensation and the potential for shock, and require close monitoring of vital signs.[13] Other relevant history includes previous abdominal or aortic surgeries, social history, presence and color of hematemesis, duration of symptoms, presence of dark tarry stools, presence of melena, and presence of hematochezia. For example, several episodes of nonbloody emesis followed by hematemesis make a Mallory-Weiss tear more likely, versus a sudden onset of copious hematemesis, which is more characteristic of a variceal bleed. The objective appearance of the blood may help determine if there is a significant risk of hemorrhage.[13,14] For example, brown stool occultly positive is less likely to be a high hemorrhage risk or have hemodynamic significance compared to coffee-ground emesis and melena.

Clinical scoring systems are summarized in **Table 79-2**.[15,16] They have been proposed for use in risk stratification and the need for urgent versus emergent endoscopy in nonvariceal bleeds.

TABLE 79-2 The Glasgow-Blatchford and Rockall Bleeding Scores

Glasgow-Blatchford score	Rockall score
• Scores ≥ 6 are associated with a > 50% risk of requiring intervention	• Scores < 3 have a good prognosis • Scores > 8 are associated with a high risk of mortality
Blood urea nitrogen 6.5–8.0 = 2 points 8.0–10.0 = 3 points 10.0–25.0 = 4 points > 25.0 = 6 points	**Age** < 60 = 0 points 60–79 = 1 point ≥ 80 = 2 points
Hemoglobin (men) 12.0–12.9 = 1 point 10.0–11.9 = 3 points < 10.0 = 6 points	**Shock** No signs of shock = 0 points Pulse > 100, blood pressure > 100 systolic = 1 point Systolic blood pressure < 100 = 2 points
Hemoglobin (women) 10.0–11.9 = 1 point < 10.0 = 6 points	**Comorbidities** None = 0 points CHF or CAD = 2 points Renal or liver failure = 3 points Metastatic cancer = 3 points
Systolic blood pressure 100–109 = 1 point 90–99 = 2 points < 90 = 3 points	**Diagnosis** Mallory-Weiss tear = 0 points Nonmalignancy diagnoses = 1 point GI malignancy = 2 points
Other markers Pulse > 100 (per minute) = 1 point Presentation with melena = 1 point Presentation with syncope = 2 points History of hepatic disease = 2 points Cardiac failure = 2 points	**Evidence of bleeding** None = 0 points Present = 2 points

CAD, coronary artery disease; CHF, congestive heart failure.
Source: Adapted from references 15 and 16.

The Glasgow-Blatchford scale aims to predict which patients will eventually require intervention in the form of blood transfusion, endoscopy, or surgery. The Rockall score predicts the probability of rebleeding and mortality. The Glasgow-Blatchford scale was found to be far superior in its respective goal compared to the Rockall score in terms of negative predictive value since none of the patients with a score of 0 on the Glasgow-Blatchford scale required endoscopy.[17-20] Although a score of 0 on the Glasgow-Blatchford scale is highly indicative of a low-risk patient, any score above 0 is not as absolute, and therefore, the score generally requires supplementation with further testing.[17-20]

Video capsule endoscopy has shown exciting promise in terms of risk stratification compared to clinical scoring systems.[21,22] Video capsule endoscopy correctly identified 100% of the high- and low-risk patients on subsequent endoscopy. This confirmed the findings of a similar earlier pilot study, which predicted 100% of the high- and low-risk patients.[22]

EARLY DETECTION OF BLEEDING ETIOLOGY

The use of a nasogastric tube (Chapter 75) with gastric lavage is currently one of the most commonly used techniques for the detection of upper GI bleeding. The benefit is three-fold. It removes potential aspirate in the case of frequent emesis. The removal of stomach contents may enhance visualization of the anatomy when an endoscopy is later performed.[13] It also shows if a bleed is present and its characteristics (i.e., color, amount, and consistency).

A nasogastric lavage is somewhat invasive and often poorly tolerated. There are some significant limitations and potential complications. Studies have been done that have debunked some of the proposed benefits. There was a statistically significant correlation between a bloody lavage and high-risk lesions on subsequent endoscopy, with an odds ratio of 4.82 when compared to clear aspirate

and 2.8 when compared to coffee-ground aspirate.[23] However, the negative predictive value of clear aspirate was found to be inconsistent, with as many as 15% of patients having high-risk lesions on endoscopy. A study compared the use of erythromycin alone versus nasogastric lavage plus erythromycin as a means to prepare patients for subsequent endoscopy.[24] There was no significant difference in visualization or other outcomes between the two treatment groups. Erythromycin was also better tolerated and less invasive than nasogastric lavage. Based on this study, it is not necessary to rely on nasogastric lavage to improve visualization during endoscopy. Nasogastric lavage has not shown any significant improvement in the patient outcomes of mortality, hospital length of stay, or the need for subsequent transfusions.[25] The study concluded that nasogastric lavage is an invasive procedure that appears to have unclear clinical benefit and should not routinely be performed.

Video capsule endoscopy is much less invasive, better tolerated, and has been shown to have more consistent results than nasogastric aspirate and lavage in relatively small studies.[26] Nasogastric aspirate only identified 33% of bleeding lesions. Video capsule endoscopy identified 83% of bleeding lesions. Of the 17% of bleeding lesions missed, approximately one-third of missed bleeds were due to battery death before reaching the duodenum. This study also found that there was no significant difference between the identification of peptic or inflammatory lesions between capsule endoscopy and upper endoscopy, although upper endoscopy remains the gold standard.

The results from video capsule endoscopy are easy to interpret and do not require specialist training.[27,28] Emergency Physician versus Gastroenterologist reads of video capsule endoscopy images from 25 subjects with upper GI bleeds found a 96% agreement between the two groups.[27] Additionally, video capsule endoscopy in these patients had an 88% sensitivity and 64% specificity for detection of fresh blood when compared to endoscopy. Another study by the same group further supported this result.[28] They compared Emergency Physicians and Gastroenterologists after a brief 10-minute training session, finding a sensitivity of 94% and a specificity of 87%.

Video capsule endoscopy is a cost-effective method for early low-risk bleeding detection and risk stratification when compared to the Glasgow-Blatchford scale (and all further testing it required), nasogastric lavage, and an admit-all strategy using standard decision analysis software.[29] Video capsule endoscopy had an average cost of $5,691 compared to $8,159 for the nasogastric tube, $10,695 for risk stratification with subsequent tests, and $22,584 for the admit-all strategy.

INDICATIONS

Video capsule endoscopy should be used within 6 hours after the patient's arrival in the Emergency Department with upper GI bleeding. Other indications not used in the Emergency Department include the evaluation of celiac disease, Crohn's disease, iron deficient anemia, NSAID enteropathy, obscure bleeding, polyposis, portal hypertension, tumors, and unknown persistent abdominal pain.[30,31]

CONTRAINDICATIONS

Video capsule endoscopy is generally considered to be safe and well-tolerated.[32] **The presence of any signs of possible or impending airway compromise is an absolute contraindication to the use of video capsule endoscopy. Protection of the airway and initial resuscitation from blood loss always takes precedence over all further diagnostic studies.** In patients with altered mental status, dementia, or swallowing difficulties, aspiration of the capsule can occur. The inability to swallow for any reason is a contraindication to the procedure. Known dysphagia, esophageal strictures, esophageal webs, or disorders that may potentially hinder the passage of the capsule through the digestive tract (e.g., diverticula, fistulas, gastroparesis, small bowel obstruction, small bowel stenosis, and small bowel strictures) are other contraindications to video capsule endoscopy.[32] Use caution with a history of prior abdominal surgery because patients can have adhesions or small bowel narrowing. Magnetic resonance imaging cannot be performed until the capsule is evacuated from the GI tract.

It has also been proposed that patients with cardiac pacemakers or defibrillators should not be considered candidates for video capsule endoscopy due to potential radiofrequency interference.[32,33] This recommendation is found on the FDA video capsule package insert. However, multiple studies have shown that video capsule endoscopy can be used safely with pacemakers and defibrillators, and therefore, these are a relative contraindications.[33-38] Consult a Gastroenterologist before performing video capsule endoscopy on these patients.

Pregnancy is considered a relative contraindication to the use of video capsule endoscopy. This is due to the increased risk of impaction requiring surgical intervention and thus potential harm to the fetus.[32] There have been case reports of video capsule endoscopy being successfully used in pregnant women.[39,40] The exact risk to the fetus is currently unclear. Consult a Gastroenterologist before performing video capsule endoscopy on these patients.

EQUIPMENT

- Wireless video capsule (**Table 79-3, Figures 79-1 and 79-2**)
- Software for the video capsule
- Sensing system (**Figure 79-3**)
- Sensing pads
- Data recorder with a battery pack (**Figure 79-4**)

TABLE 79-3 Some of the Different Types and Characteristics of Video Capsules

Video capsule name	CapsoCam	EndoCapsule	MiroCam	OMOM	PillCam Colon2	PillCam ESO2	PillCam SB3
Manufacturer	CapsoVision	Olympus Japan	Intromedic Company	Jinshan Science & Technology	Given Imaging	Given Imaging	Given Imaging
Dimensions (mm)	11 × 31	11 × 26	10.8 × 24.5	13 × 27.9	11.6 × 31.5	11.4 × 26.4	11.4 × 26.2
Field of view	360°	145°	170°	140°	172°	169°	156°
Frames per second	Up to 20	2–3	3	2	4–35	18	2–6
Imaging heads	4	1	1	1	2	2	1
Operating time	15 hours	8+ hours	12 hours	6–8+ hours	10 hours	30 minutes	8+ hours
Resolution	212 × 896	512 × 512	320 × 320	640 × 480	320 × 320	320 × 320	340 × 340
Transmission	USB	RF	EFP	RF	RF	RF	RF
Weight (gm)		3.50	3.25–4.70	6.00	2.90	2.90	3.00
FDA approved	No	Yes	Yes	No	Yes	Yes	Yes

EFP, electric field propagation; FDA, U.S. Food and Drug Administration; RF, radiofrequency; USB, universal serial bus.
Source: Manufacturers' websites and modified from references 30 and 42.

FIGURE 79-1. Examples of video endoscopy cameras. ***A.*** The PillCam. (Photo courtesy of Given Imaging.) ***B.*** The MicroCam. (Photo courtesy of Intromedic Company.) ***C.*** EndoCapsule. (Photo courtesy of Olympus Japan.)

- Compatible handheld sensing system that allows real-time image viewing
- Computer capable of running proprietary software for image review and interpretation

There are many video capsules on the market (**Table 79-3**). They differ in regard to their battery life, field of view, dimensions, image requisition, and optics.[41,42] Most video capsules move passively through the GI system. Research is well under way for external magnet-guided and self-propelled capsules (**Figure 79-5**).[43] The capsule used by most of the current studies is the PillCam ESO2 (Given Imaging, Yokne'am Illit, Israel). The PillCam measures 11.4 mm by

FIGURE 79-3. The PillCam sensing belt. (Photo courtesy of Given Imaging.)

26.4 mm, approximately the size of a large multivitamin pill, and weighs approximately 3 gm.[27,44] Each video capsule has similar components including a disposable plastic exterior with a complementary high-resolution charge-coupled device image capture system or metal oxide semiconductor, white light-emitting illumination sources, transmitter, a short focal length capability, and an internal battery.[45] The video capsule can detect lesions as small as 0.1 mm in diameter. On each end of the capsule, the PillCam ESO2 has an optical dome capable of capturing approximately 18 frames per second, has a wider angle view of approximately 169°, and has more advanced optics (i.e., three lenses) than most other small and large bowel capsules.[44] The battery life of this capsule is significantly lower than other capsules, lasting only for approximately 30 minutes. The data from the lenses is transmitted via ultra-high-frequency band radio telemetry through the sensory pads and belt to the data recorder, which then transmits the date to the computer or handheld device.[45] Some of the software marks images to be reviewed that contain blood.

PATIENT PREPARATION

The patient should ideally be fasting for at least 2 hours prior to a video capsule endoscopy procedure.[46] Conditions in the Emergency Department are often less than ideal, and few, if any, patients are able to be prepped appropriately. The studies evaluating the success of video capsule endoscopy discussed earlier took this aspect into account, and all results were in patients who lacked the ideal preparation. **The main preparation required prior to this procedure is initial stabilization of airway, breathing, and circulation as well as assessing for and ruling out any significant contraindications.**

The patient and/or their representative must be informed of the procedure, including its risks and benefits. Obtain an informed consent for the video capsule endoscopy procedure. No anesthesia or sedation is required. Obtain plain radiographs of the abdomen to make sure there is no bowel obstruction prior to the procedure.

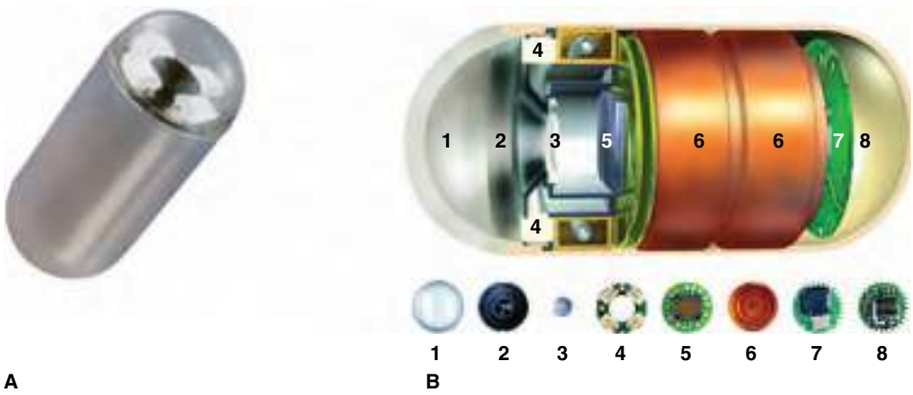

FIGURE 79-2. Schematic of a video capsule. ***A.*** External view. ***B.*** Schematic cross-section and elements (1 – optical dome, 2 – lens holder, 3 – short focal aspheric lens, 4 – white LEDs, 5 – CMOS imager, 6 – batteries, 7 – ASIC transmitter, 8 – antenna.) (Used with permission from reference 40.)

FIGURE 79-4. The PillCam data recorder. (Photo courtesy of Given Imaging.)

Attach the sensing pads to the patient over the midline of the upper sternum, over the midline of the lower sternum, and in the left upper quadrant of the abdomen over the anatomical location of the stomach (**Figure 79-6**).[27] Attach the wiring to the data recorder either around the patient's waist or over the patient's shoulder.[27]

TECHNIQUE

Turn on the recorders. Activate (i.e., turn on) the video capsule when the procedure is ready to begin. Instruct the patient to drink approximately 100 mL of water while standing followed by the ingestion of the activated capsule in the supine position with a 10 mL sip of water through a syringe or straw.[45] The ingestion protocol is then followed. This involves 2 minutes of recording with the patient supine, followed by 2 minutes with the head of the bed raised to 30° degrees, an additional minute at 60°, and finally, 15 minutes in the upright position to maximize data capture time.[45] Alternatively, the capsule can be ingested with a sip of water in the prone position. After 2 minutes of recording in this position, place the patient in an upright seated position while the rest of the images are recorded.[22] If the capsule remains in the stomach for more than 15 minutes, the patient can be placed in the right lateral decubitus position to facilitate passage into the duodenum.[22] The images are then viewed on either the computer or handheld device.

ASSESSMENT

Review the images on either the computer or handheld device (**Figure 79-7**).[47] Look for bleeding sites (active or inactive), masses, mucosal damage, petechia, red spots, tumors, ulcerations, or any other abnormalities.

AFTERCARE

Minimal aftercare is required. After the 30 minute of recording, the capsule automatically deactivates and is passed through the intestine via peristalsis, leading to the eventual natural evacuation.[36]

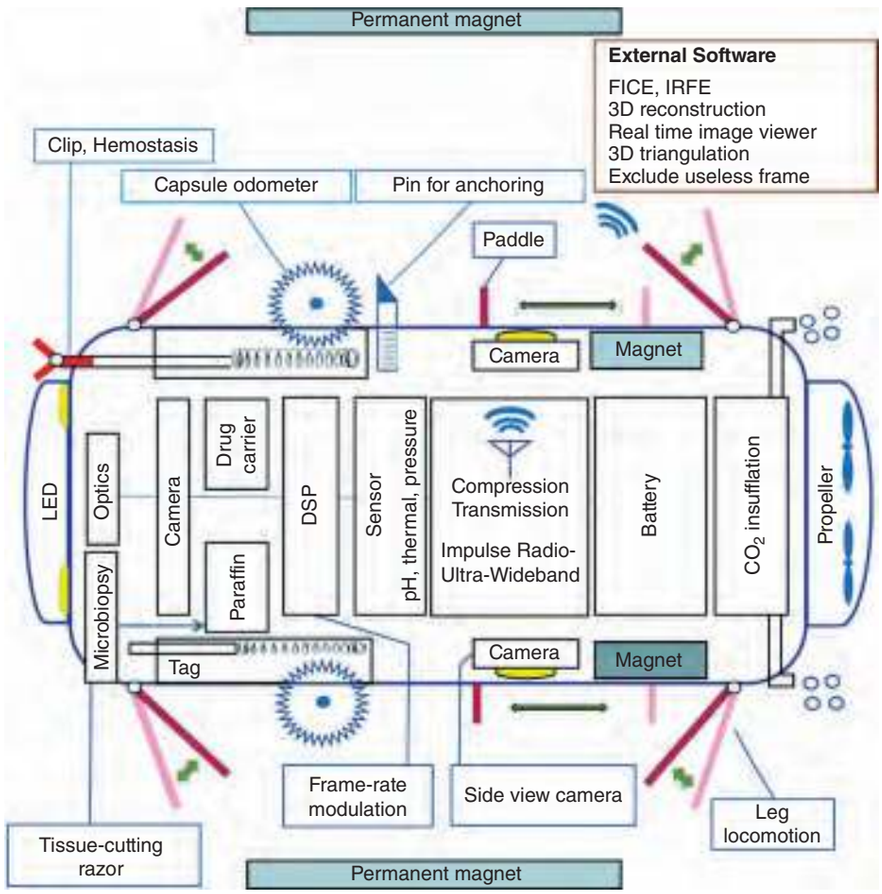

FIGURE 79-5. Schematic of a magnetically guided video endoscope camera. (Used with permission from reference 42.)

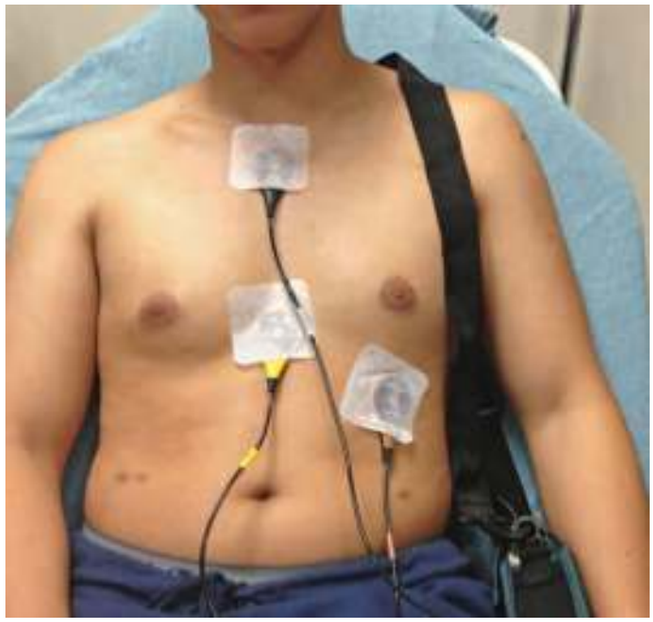

FIGURE 79-6. Sensing pad placement. (Photo used with permission from reference 27.)

However, postprocedural follow-up is often recommended to ensure appropriate passage of the capsule and to address any potential complications.

COMPLICATIONS

Video capsule endoscopy is usually well tolerated with a very low complication rate. However, as with any procedure, complications do exist. **Video capsule aspiration leading to airway compromise with or without obstructive pneumonitis is always a possibility and must be carefully monitored during ingestion.**[48] Bronchoscopy will be required for removal of the video capsule.[48,49] The risk of aspiration can be decreased by ruling out significant contraindications and proper patient positioning during the video capsule ingestion.

The most common postingestion complication is capsule retention. This occurs in approximately 1.4% of cases and is the stem from which a majority of other potential complications can occur.[48] The reasons for retention are extensive but mainly involve undiagnosed inflammatory bowel disease (most common), gastroparesis, neoplasms, NSAID-induced enteropathy, postabdominal radiation, postsurgical stenosis, small bowel strictures, tuberculosis, or bowel ischemia.[50] While the majority of these patients remain asymptomatic, a very low percentage develop subsequent complications.[48] In a retrospective study of over 1000 video capsule endoscopies, none of the patients had significant symptoms as a result of capsule retention.[51]

Several steps can be taken to minimize retention risk. This includes an adequate initial history for risk factors, motility agents, anti-inflammatories, or colonoscopy preparation fluids to help promote spontaneous passage, nasogastric tube insertion for bowel decompression, and close monitoring with serial abdominal plain films to ensure progression.[50-53] If conservative measures do not lead to spontaneous passage, either endoscopic or surgical removal should be considered depending on the retained location.

There have been a few documented case reports of rare complications from capsule retention, the most serious of which is bowel perforation requiring immediate surgical intervention, although preexisting inflammatory bowel disease seems to be the most likely underlying culprit.[54,55] An even rarer complication is the development of acute appendicitis from capsule retention, which is managed by appendectomy.[56,57]

LIMITATIONS OF VIDEO CAPSULE ENDOSCOPY

Although the technology behind capsule endoscopy is exponentially advancing, current models still have several limitations, the most noticeable of which is the operator's inability to control the

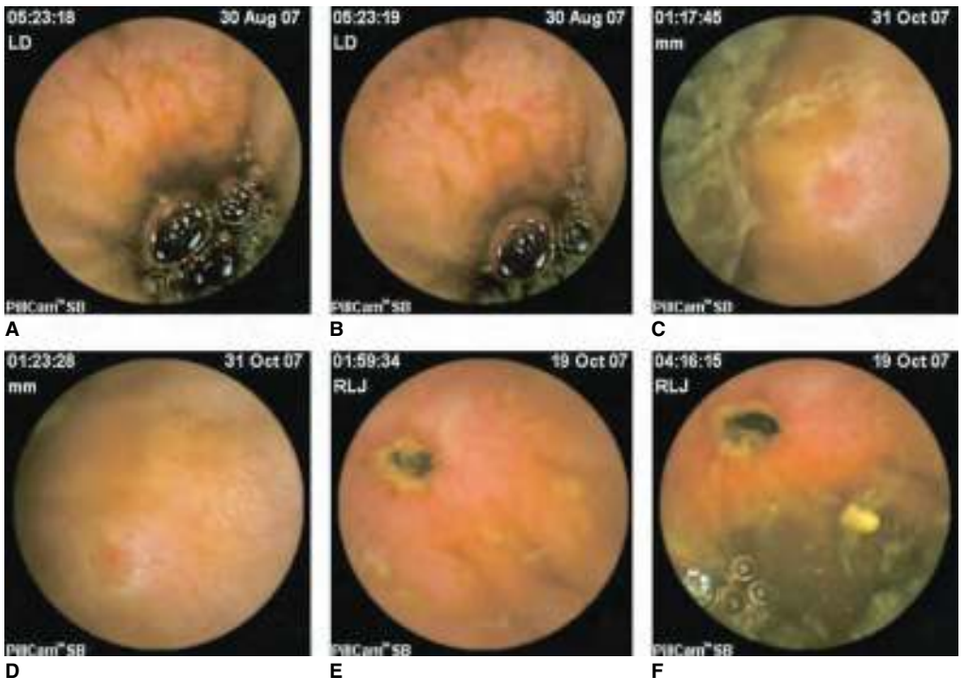

FIGURE 79-7. Examples of the PillCam images. ***A.*** Petechiae and red spots. ***B.*** Petechiae and red spots. ***C.*** Mucosal erosion. ***D.*** Mucosal erosion. ***E.*** Ulceration. ***F.*** Ulceration. (Used with permission from reference 47.)

video capsule maneuvers throughout the GI tract.[2] As a result, the quality and location of the images obtained are at the mercy of capsule visibility and the angle of the capsule, both of which are difficult to predict. This can be somewhat alleviated by proper patient positioning during ingestion but remains a potential obstacle. The esophageal capsule models used in the emergent setting have a typical battery life of only 30 minutes. This may limit the capsule's ability to reach the duodenum prior to loss of battery power.[32] Unlike upper endoscopy, current capsule models remain solely diagnostic without any therapeutic ability. The video capsule cannot immediately address a high-risk bleeding lesion if detected. Despite these limitations, video capsules are continuously improving and may one day show significant improvement in these areas.

SUMMARY

Upper GI bleeding remains a high-stakes pathology with which the well-trained Emergency Physician must be proficient. The approach to each of these patients must remain systematic, with priority given to initial resuscitation before any further intervention is considered. After the initial resuscitation, video capsule endoscopy has emerged as one of the most promising diagnostic tools for risk stratification as well as detection of upper GI bleeding etiology. Despite its current limitations, it has taken significant strides toward acceptance in the Emergency Department.

Video capsule endoscopy should always be considered in the context of the overall clinical picture. Further studies with much greater sample sizes are still warranted to confirm the initial findings and compare this treatment with alternative methods. Video capsule endoscopy should be used in conjunction with the other established methods in comprehensive clinical decision making.

Each year, the technology for video capsule endoscopy progresses. This has the potential of making current limitations ephemeral. Video capsule endoscopy could become one of the mainstays in the medical management of upper GI bleeding in the Emergency Department.

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80 Esophageal Foreign Body Removal

Bashar M. Attar

INTRODUCTION

Most foreign bodies (i.e., 90%) that are ingested enter the gastrointestinal tract, while 10% enter the tracheobronchial tree.[1] Approximately 1500 people die annually in the United States from ingested foreign bodies in the upper gastrointestinal tract.[2] Most objects (i.e., 80% to 90%) usually pass spontaneously, but approximately 10% to 20% must be removed endoscopically.[3] Approximately 1% require surgical removal.[4] Approximately 80% of esophageal foreign bodies occur in children, followed by edentulous adults, prisoners, and psychiatric patients.[5] Recurrent episodes of foreign body ingestion occur in 5% to 10% of patients, especially prisoners and psychiatric patients.[1]

The presentations are best divided into accidental and deliberate ingestions.[1,2,4-7] The accidental ingestion patient is usually cooperative and has a single foreign body. The deliberate ingestion patient is often uncooperative, the foreign bodies are multiple, and the foreign bodies are often unusual. It is important to identify such individuals at their initial presentation since foreign body removal is usually performed under conscious sedation or general anesthesia.

The patient's history is the most important part of the diagnostic evaluation.[4,8] The identity of the object ingested is usually known to the patient. **Persistent odynophagia, dysphagia, or foreign body sensation may indicate the presence of an esophageal foreign body despite negative radiographic results. A high index of suspicion must be maintained in younger children and mentally retarded adults.**[9-11] Patients with a history of eosinophilic esophagitis have a higher incidence of food impaction and higher risk of perforation associated with interventions.[12]

The physical examination is most likely negative unless complications are present. Note any stridor, wheezing, signs of consolidation, and the absence of breath sounds. Subcutaneous emphysema in the neck or chest indicates perforation of the esophagus or the stomach. **The most common sites for a foreign body to get trapped are where the esophagus is narrow (i.e., at the cricopharyngeus muscle, where the aortic arch crosses the esophagus, and at the gastroesophageal junction).**

Radiographic evaluation is often helpful in the evaluation of an esophageal foreign body (**Figure 80-1**).[4-7,13-16] Obtain plain radiographs of the neck and chest in the posteroanterior and lateral positions. Evaluate the radiographs for the presence of a foreign body in all planes. Air in the subcutaneous tissues, mediastinum, and/or beneath the diaphragm is indicative of a perforation. Barium studies are undesirable in patients with a food bolus impaction and obscure endoscopic visualization. Esophagrams performed using a minimal amount of thin barium may be necessary in situations where the foreign body is made of wood, thin metals, aluminum can top, and plastics. **Meglumine diatrizoate (Gastrografin) is contraindicated in food bolus impactions because it is highly hypertonic and can lead to severe chemical pneumonitis if aspirated into the lungs.**[6] Toothpicks, wood, and fish bones may not be seen on radiographs. Food or meat bolus impaction may not be evident radiographically unless it contains bony tissue. **Failure to locate an object on radiologic examination should not rule out its presence.** Computed tomography may be useful, especially in cases where the foreign body could not be detected as it may have become embedded in or penetrated the esophageal wall.[16,17]

The lateral soft tissue of the neck can be particularly helpful in evaluating for an esophageal foreign body.[13,14] The radiograph can be obtained at the bedside in ill patients, is inexpensive and quick to obtain, and may show soft signs of an esophageal foreign body (**Figure 80-1**). A single air column is normally seen (**Figure 80-1*A***). A double air column represents air in the esophagus and the airway (**Figures 80-1*B* and 80-1*C***). An air-fluid level may be seen in the esophagus.

Endoscopy is important for both the diagnosis and possible removal of an esophageal foreign body.[7,18-20] Extraction with the flexible endoscope is successful in 84% to 98% of cases with no associated complications.[21,22] Consider an Otorhinolaryngology consultation if foreign bodies are at or above the cricopharyngeus level. Success is more likely and complications are minimized with proper patient preparation.

INDICATIONS

Esophageal foreign body extraction is required in a minority of patients. Most foreign bodies will pass spontaneously into the stomach. The indications for removal depend on the type of foreign body

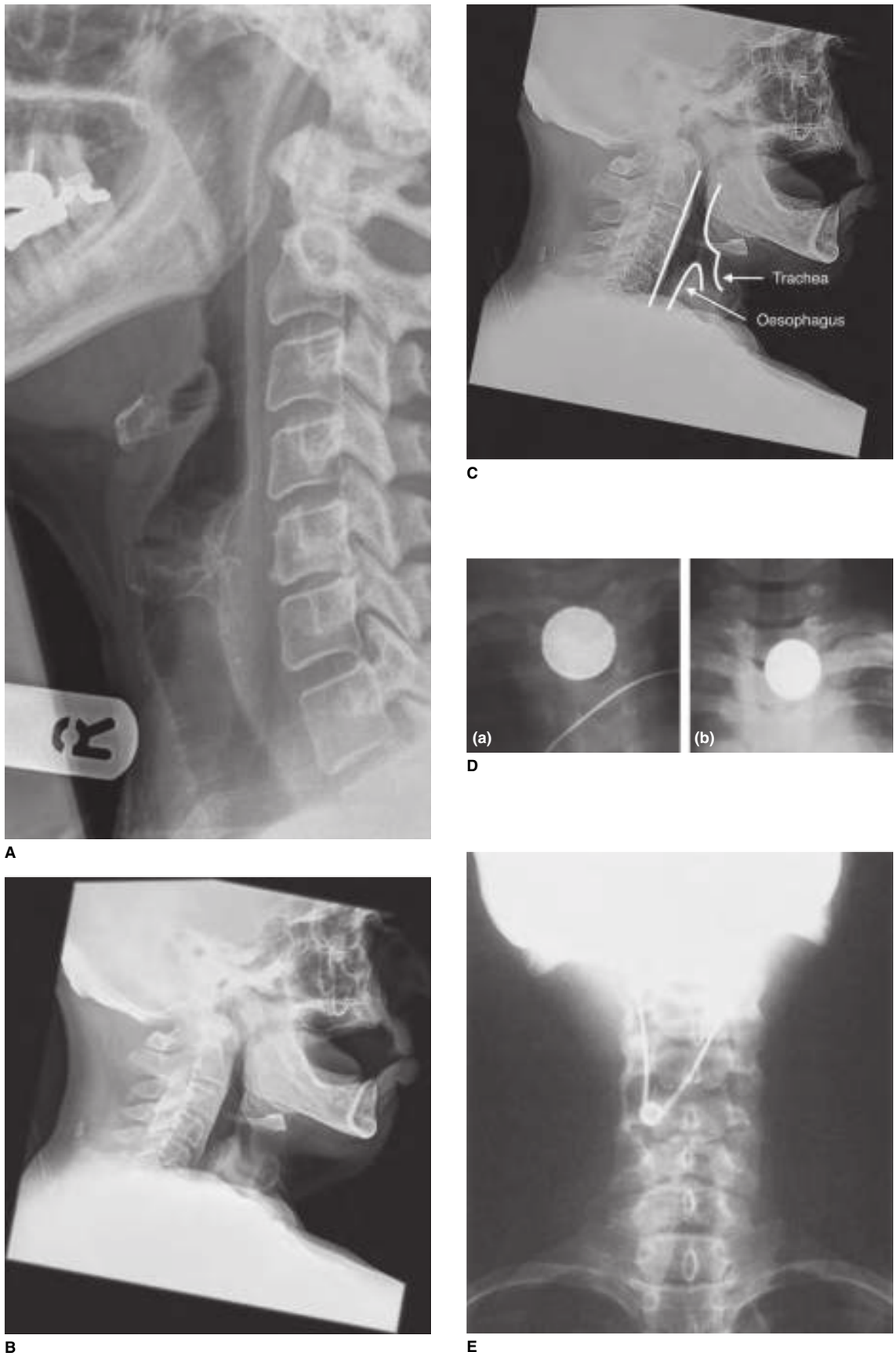

FIGURE 80-1. Lateral soft tissue radiographs of the neck. ***A.*** Normal. ***B.*** Impacted food bolus 30 cm from the incisors. ***C.*** Outline of the double air column. ***D.*** A button battery (A) versus a coin (B). ***E.*** Safety pin. (Photos used with permission of reference 13 and 14.)

and whether it impacts in the esophagus. Remove sharp objects impacted above the cricopharyngeus under direct vision using a laryngoscope to elevate the soft tissues and forceps (i.e., Boedecker, Magill, or Tylke) to grasp it.

Meat boluses are commonly impacted in the esophagus. Patients will swallow large pieces that may or may not be well chewed. **Impaction of a meat bolus, or another foreign body, at or just below the cricopharyngeus muscle with tracheal compression and resultant respiratory obstruction is a true emergency.** The Heimlich maneuver may be lifesaving in this situation.[23] **Immediately intubate the patient orotracheally or perform a cricothyroidotomy if the Heimlich maneuver is unsuccessful.** Early removal of a meat bolus impaction is recommended, even when the bolus is in the distal third of the esophagus. Delays in extraction allow the food to soften and make extraction more difficult. The administration of glucagon intravenously may lead to esophageal relaxation and facilitate spontaneous passage of the food bolus to the stomach. It must be endoscopically removed if glucagon fails.

Blunt and round objects may become impacted in the esophagus. **Earlier removal is necessary if a blunt object is impacted higher in the esophagus with associated sialorrhea and the potential for pulmonary aspiration.** Esophageal obstruction at lower levels requires prompt, but not emergent, treatment. Most rounded objects in the lower third of the esophagus will often pass spontaneously into the stomach. A 12 hour period of observation is permissible in this situation.[24] It is common for sharp, pointed, and elongated objects to become impacted in the esophagus. Toothpicks, open safety pins, nails, and chicken bones are associated with up to a 35% incidence of esophageal perforation and should be removed.[25,26] Toothpicks should be removed promptly from the esophagus or stomach even if they are not impacted because they are prone to penetration of the gastrointestinal wall. Toothpicks may migrate into surrounding structures and lead to serious complications.[25]

Numerous other objects can become impacted in the esophagus. Elongated, narrow foreign bodies such as stiff wires are prone to penetration and perforation of the esophageal wall. They should be removed even if they passed through the esophagus and into the stomach. These objects may become trapped by the retroperitoneally fixed angles of the duodenum and eventually result in perforation. Plastic bag clips, although not sharp and pointed, should be removed before they pass from the esophagus into the stomach and through the pylorus. They have claws that can attach to the small bowel mucosa, leading to ulceration, stricture formation, and bleeding.[27] Toxic foreign bodies (e.g., button batteries) that become impacted in the esophagus should be removed promptly to prevent perforation and systemic toxicity.

CONTRAINDICATIONS

There are no absolute contraindications to the removal of an esophageal foreign body. Treat life-threatening and limb-threatening injuries prior to esophageal foreign body removal. Evaluate and support the patient's airway, breathing, and circulation prior to removing the foreign body.

EQUIPMENT

- Flexible upper gastrointestinal endoscope
- Rigid gastroscope
- Through-the-scope balloon
- Steigmann-Goff friction-fit adaptor of the esophageal variceal rubber banding ligating kit
- Glucagon
- 44 French Maloney rubber dilator
- Lubricant gel
- Endoscopic overtube, for use with sharp or pointed foreign bodies
- Soft latex protector hood for the flexible scope
- Rat-tooth or alligator forceps
- Retrieval nets or Dormia baskets
- Polypectomy snares
- Laryngoscope
- Laryngoscope blades, Miller and Macintosh of various sizes
- Airway forceps (i.e., Boedecker, Magill, or Tylke)
- Curved clamp
- Medications for procedural sedation (Chapter 159)
- Cardiac monitor
- Pulse oximetry
- Supplemental oxygen
- Foley catheters, 14 to 16 French
- Topical anesthetic spray
- Water-soluble contrast material
- 5 mL syringe
- Fluoroscopy machine, optional

PATIENT PREPARATION

Obtain a duplicate sample of the ingested foreign body if possible. Manipulate the duplicate object with available foreign body forceps and snares. Determine which instrument is best suited to grasping the foreign body. Instruments that are most useful include alligator and rat-toothed forceps, endoscopic overtubes, polypectomy snares, and Dormia baskets.

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure, and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

The timing of the endoscopic intervention is important (**Table 80-1**). It depends on the clinical condition of the patient, the nature of the

TABLE 80-1 Timing of Endoscopy for Foreign Bodies in the Esophagus

Nonurgent endoscopy
Coin in the esophagus of an asymptomatic patient
Coins and batteries in the stomach of an asymptomatic patient
Foreign body with a diameter of > 2.5 cm in the stomach
Foreign body longer than 3 cm in infants and young children
Foreign body longer than 5 cm in older children, adolescents, and adults
Urgent endoscopy
Absorptive foreign bodies
Batteries in stomach if battery is > 20 mm diameter
Esophageal food impaction not causing a complete obstruction
Esophageal foreign bodies that are not sharp
Foreign body longer than 6 cm in adolescents and adults
Multiple magnets
Sharp foreign body in the stomach if asymptomatic
Emergent endoscopy
Battery in the esophagus
Battery in the stomach if symptomatic
Complete esophageal obstruction
Magnets in the esophagus
Magnets in the stomach if symptomatic
Sharp foreign body in the esophagus
Sharp foreign body in the stomach if symptomatic

foreign body, the risk of aspiration, or the risk of additional complications (e.g., obstruction or perforation). Patients who have difficulty managing their secretions or who ingested sharp-pointed objects or disk batteries that localized to the esophagus should undergo emergent endoscopy. Patients who ingested blunt foreign bodies that are in the esophagus without complete esophageal obstruction will require urgent endoscopy. Urgent endoscopic removal is required for magnets, objects < 6 cm in length at or above the proximal duodenum, and sharp or pointed objects in the stomach or duodenum. Objects with a diameter > 2.5 cm in the stomach will require nonurgent endoscopy. Coins located in the esophagus may be monitored for 12 to 24 hours prior to endoscopic removal if the patient is free of symptoms.[28]

The management of ingested foreign body requires assessment of the patient's airway and ventilation status. Most esophageal foreign bodies may be removed under procedural sedation. Airway protection may require endotracheal intubation and general anesthesia, especially when the foreign body is difficult to remove or located in the proximal esophagus. Endoscopic overtubes facilitate the frequent passage of the scope and protect the airways. Endoscopic overtubes protect the esophageal mucosa from injury upon the removal of sharp objects.[4,28]

Apply monitors for electrocardiogram, blood pressure, and pulse oximetry along with an intravenous line for the administration of drugs. Administer supplemental oxygen by nasal cannula or "blow-by." The patient may be in a sitting, semirecumbent, supine, or left semilateral position. The sitting position will displace redundant pharyngeal tissue anteriorly and open the pharyngeal space by gravity in morbidly obese patients. Maneuvers performed by an assistant (e.g., the jaw thrust or pulling the tongue forward with a cotton swab) can help to move the pharyngeal soft issues anteriorly and allow increased maneuverability.

Instrumentation may cause the patient to produce copious secretions. This makes removal of the foreign body extremely difficult. Consider administering 0.3 to 0.4 mg of glycopyrrolate. Administer this potent antisialagogue intravenously at least 10 minutes before or intramuscularly 30 minutes before the foreign body is removed. Atropine is often more readily available in the Emergency Department than glycopyrrolate. It can be administered in a dose of 0.5 mg intravenously at least 10 minutes before foreign body removal. The only disadvantages of atropine are that it crosses the blood-brain barrier and can cause central nervous system effects, whereas glycopyrrolate does not. Refer to Chapter 12 for a more complete discussion of these two antisialagogues.

Sedation can be extremely beneficial in gaining the patient's cooperation during the foreign body removal. **Sedative drugs, if used at all, should be judiciously titrated to the desired effect with continual assessment of the patient's level of consciousness while avoiding respiratory depression.** Ketamine given in small doses (e.g., 0.5 to 1 mg/kg) and titrating to the desired level of sedation has the advantage of producing minimal respiratory depression and may be preferable to opioids in some cases. Refer to Chapter 12 for a more complete discussion of the pharmacologic adjuncts to intubation. **Avoid sedation if the patient has a tenuous airway, labored respirations, or a distended abdomen or is vomiting.**

It remains an unresolved controversy whether to abolish the laryngeal reflexes of a patient considered to have a "full stomach." The Emergency Physician must weigh the risk of abolishing the laryngeal reflexes and rendering the patient potentially vulnerable to aspiration versus leaving the laryngeal reflexes intact. Instrumentation in patients with an intact gag reflex can induce vomiting.

The oral cavity, oropharynx, and supraglottic larynx can be anesthetized. Place a tongue blade on the patient's tongue and apply topical anesthetic spray (**Figure 28-10**). Benzocaine or lidocaine spray will usually provide effective anesthesia for the posterior pharynx within 30 seconds. They can be administered from a commercially available spray container or using an atomizer device (**Figure 28-3 and Table 28-3**). There are reports of methemoglobinemia from the overzealous use of benzocaine. Limit its use to several short sprays.

Alternatively, the patient can swish 2% viscous lidocaine in their mouth for several minutes to provide effective anesthesia. One of the most effective methods of blocking the glossopharyngeal nerve is the "lollypop method." Create a lollypop by soaking sterile gauze in lidocaine ointment and taping it to a tongue depressor. Place the lidocaine lollypop into the patient's posterior mouth while setting up for the fiberoptic foreign body removal. Advance the lollypop 1 cm every 2 minutes until the patient's posterior pharynx is completely anesthetized.

Place a suction catheter into the oropharynx. This will clear the airway of secretions and blood that can impair the visual image. It will also determine the adequacy of the topical anesthesia for preventing coughing and gagging.

TECHNIQUES

GENERAL PRINCIPLES

The flexible upper gastrointestinal endoscope (Figure 80-2*A*) is similar to a flexible bronchoscope (Chapter 28). It is inserted under direct visualization to avoid inadvertently striking the foreign body and further impacting it or causing it to penetrate the esophageal wall. Blunt foreign bodies (e.g., button batteries and coins) can be securely grasped with forceps or a snare (**Figure 80-3**). **A firm grasp on the foreign body is required before withdrawal is attempted.** The foreign body may otherwise become dislodged as it is withdrawn through points of anatomic narrowing. This can result in aspiration of the foreign body. An overtube should be used if multiple insertions and withdrawals of the endoscope are needed (**Figure 80-2*B***). Pointed foreign bodies should be withdrawn with the point trailing to avoid perforating any structures (**Figure 80-4**). Grasp objects with sharp edges (e.g., razor blades) and extract them through an overtube to prevent secondary injury (**Figure 80-5**). Elongated foreign bodies such as wires or pens should be grasped with a snare close to the cephalad end of the object so it can align itself with the long axis of the esophagus during withdrawal. Foreign bodies that penetrate the mucosa can be safely extracted with the endoscope if frank perforation or vascular penetration has not occurred.

FOOD IMPACTIONS

ENDOSCOPY

Food impactions are more likely to occur in the distal esophagus. There is no need for barium studies because it will obscure visualization during endoscopy if the patient is symptomatic. **Endoscopic intervention should be carried out immediately to prevent aspiration if the patient is salivating and unable to handle oral secretions. The impacted food bolus should not remain in the esophagus for more than 12 hours. The risk of complications increases significantly the longer the foreign body is in the esophagus.**

Underlying esophageal disease is found in 65% to 97% of adults presenting with an esophageal food impaction.[21,29,30] **Endoscopic removal is the procedure of choice if a food bolus does not pass spontaneously or after an unsuccessful trial of gas-forming agents, glucagon, nifedipine, or nitroglycerine (Figure 80-6).** Remove the entire bolus slowly under direct visualization. Snugly pull the grasping instrument with the food bolus against the tip

A

B

FIGURE 80-2. The flexible upper gastroscope. ***A.*** The scope. (Used from www.commons.wikimedia.org.) ***B.*** An example of an overtube.

of the endoscope when the endoscope is just below the cricopharyngeus muscle. Extend the patient's head and quickly remove the endoscope.[31]

A piecemeal approach can be accomplished with several passages of the endoscope through an overtube if the food bolus is soft.[32] The overtube will facilitate reinsertion of the endoscope. Insert a 44 French Maloney rubber dilator into the esophagus and proximal to the foreign body. Pass the overtube, lubricated internally and externally, over the Maloney dilator. Remove the Maloney dilator. Introduce the flexible endoscope through the overtube.

Another method is the push technique. It has been useful in dealing with an impacted food bolus.[22] A small-caliber flexible endoscope may be used to bypass the food bolus and evaluate the area distal to the obstruction. If the endoscope passes into the stomach successfully, pull it back until it is just proximal to the food bolus. Use the endoscope to gently push the food bolus into the stomach. It is preferable to push from the right side of the food bolus rather than straight through the middle. The gastroesophageal junction usually takes a left turn as it enters a hiatal hernia. The presence of a bone spicule should always be considered, whether the meat bolus is being extracted or pushed into the stomach.

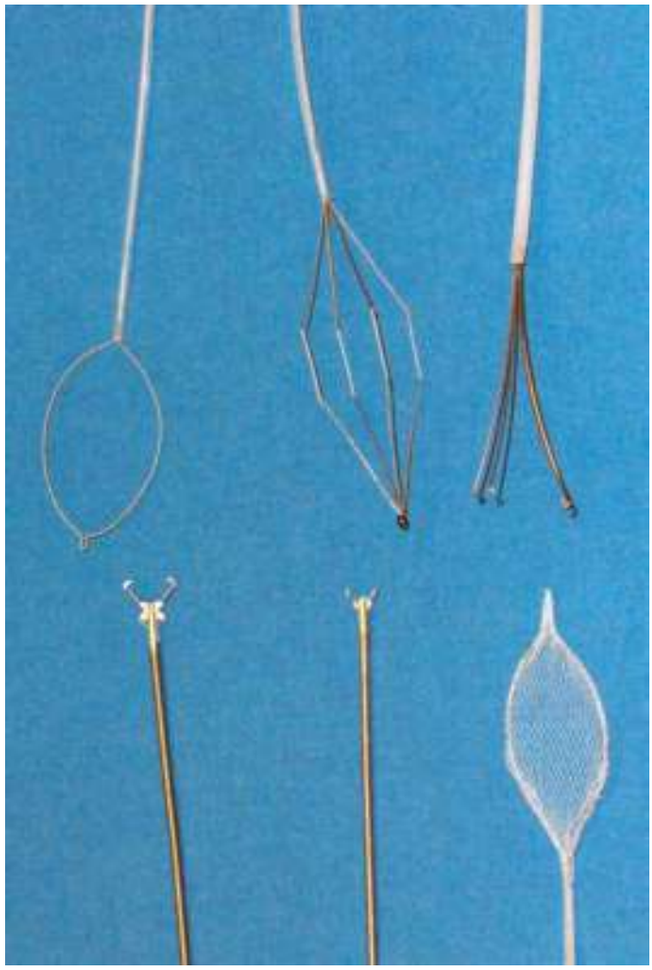

FIGURE 80-3. Endoscopic foreign body instruments. Top row: snare, basket, and multiarm grasper. Bottom row: grasper, a traumatic grasper, and Roth net. (Photo used with permission from Callahan CJ: Endoscopic foreign body removal. *Today's Veterinary Practice* 2015;5(6):77-83, www.veteriankey.com.)

A newer technique is accomplished by attaching the Steigmann-Goff friction-fit adaptor of the esophageal variceal rubber banding ligating kit to the tip of the endoscope.[33] The tip of the endoscope is

FIGURE 80-4. Endoscopic removal of a sharp foreign body. The pointy end is trailing for the removal so it does not perforate soft tissues. (Photo courtesy of Olympus Endoscopy.)

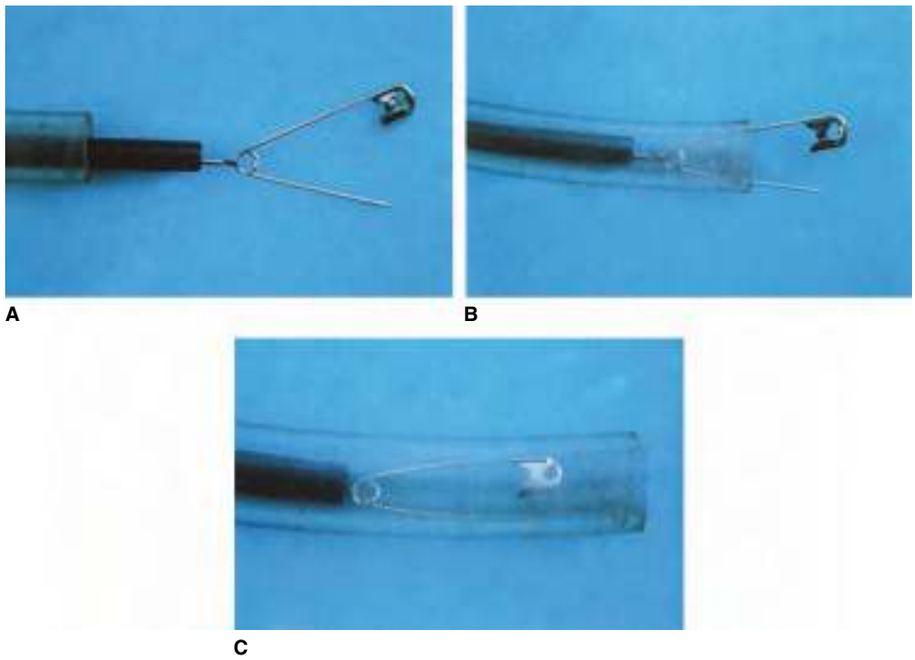

FIGURE 80-5. Endoscopic removal of a sharp foreign body through an overtube. ***A.*** Grasping the foreign body. ***B.*** Pulling the foreign body into the overtube. ***C.*** Removal of the foreign body. (Photo used with permission from www.veteriankey.com.)

replaced with a screw-on drum from the variceal ligation kit. Place an esophageal overtube proximal to the food bolus. Insert the endoscope through the overtube.[34-37] A Roth retrieval net may be passed through the endoscope to retrieve food from the esophagus to avoid the risk of dropping the food in the trachea.[38,39]

A last resort approach is the use of an Nd:YAG laser to burn an opening in the center of an impacted meat bolus. This method is expensive and carries a high risk of complications.[40] Consider rigid endoscopy under general anesthesia if a food impaction of the esophagus cannot successfully be removed using the flexible endoscope.[41]

GAS-FORMING AGENTS

Gas-forming agents can be used to relieve a distal esophageal food impaction. They are occasionally used to relieve a food impaction in the proximal and middle thirds of the esophagus. These agents produce carbon dioxide gas that distends the esophagus, relaxes the lower esophageal sphincter, and "pushes" the food bolus through the gastroesophageal junction with the aid of esophageal peristalsis. Gas-forming agents may be used in conjunction with glucagon, nifedipine, or nitroglycerine to help relieve the impacted food bolus. Complications associated with gas-forming agents include aspiration, vomiting, forceful vomiting, and esophageal perforation due to distention and/or vomiting. Many physicians do not use these agents due to the risk of perforation.

FIGURE 80-6. Endoscopic removal of a food bolus. (Photo courtesy of Olympus Endoscopy.)

Three classes of gas-forming agents have been used. Commonly used are commercially available agents that are used by Radiologists for upper gastrointestinal contrast studies. A mixture of tartaric acid (1.5 to 3.0 gm in 15 mL H_2O) immediately followed by sodium bicarbonate (1.5 to 3.0 gm in 15 mL H_2O) has been successfully used. Another agent is carbonated soda pop.

GLUCAGON

A trial of intravenous glucagon before endoscopic therapy is a reasonable approach.[42,43] It may disimpact a food bolus in the distal esophagus and allow it to pass into the stomach. Glucagon relaxes the smooth muscle of the lower esophagus and decreases lower-esophageal sphincter tone. Glucagon relaxes the esophageal smooth muscle within 1 minute of intravenous injection, and its effects last approximately 20 to 25 minutes. Glucagon has no effect on the proximal third of the esophagus that is composed of skeletal muscle. It has a minimal effect on the middle third of the esophagus that is composed of both skeletal and smooth muscle. Glucagon has an overall success rate of ≤ 50%. Glucagon has been also combined with gas-forming agents to enhance esophageal clearance.

The dose of glucagon is 0.03 to 0.1 mg/kg intravenously with a maximum dose of 1 mg in children and 2 mg in adults. It should be administered over 1 to 2 minutes. **It is recommended to give a test dose (e.g., one-tenth of the full dose) and observe the patient for 5 minutes for signs of hypersensitivity or hypotension before giving the full dose.** Have the patient take one to two sips of water after the administration of glucagon to stimulate lower esophageal

peristalsis. Administer a second dose of glucagon if the food bolus impaction is not relieved within 10 to 20 minutes.

Glucagon is a relatively safe medication.[42,43] It should not be administered to patients with known hypersensitivity to glucagon, esophageal fibrosis, esophageal rings, esophageal strictures, insulinomas, pheochromocytomas, sharp or irregular foreign bodies, or Zollinger-Ellison syndrome. Exogenous glucagon stimulates the release of catecholamines. It can stimulate a pheochromocytoma to release catecholamines, resulting in marked hypertension and tachycardia. The hypertension can be controlled using 5 to 10 mg of intravenous phentolamine. Glucagon's hyperglycemic effect can cause an insulinoma to release insulin and cause subsequent hypoglycemia.

Common complications associated with glucagon include nausea, vomiting, transient hyperglycemia, allergic reactions, tachycardia, and hypertension. Glucagon is a polypeptide hormone synthesized in nonpathogenic *Escherichia coli* that have been genetically altered. This is the basis of the allergic or hypersensitivity reactions. A transient rise in blood pressure and heart rate is often seen after administration of glucagon. Patients taking beta-blockers may be more susceptible to transient hypertension and tachycardia. These side effects are short-lived as the half-life of glucagon is 8 to 18 minutes.

NIFEDIPINE

Nifedipine is a calcium channel blocker that decreases lower esophageal sphincter tone. It is sometimes administered to allow an impacted food bolus to pass into the stomach. It should not be administered if the patient has an allergy to calcium channel blockers, has hypotension, or has ingested a sharp or irregular-shaped foreign body. With a single dose, nifedipine has few side effects. These are usually minimal (e.g., dizziness, flushing, headache, hypotension, lightheadedness, muscle cramps, nausea, nervousness, and palpitations) and do not preclude its use.

The most significant effect of nifedipine is hypotension that may last 6 to 8 hours. Some patients will have a significant hypotensive response to nifedipine, and there is no way to predict which patients will be affected. For these reasons, many physicians will not use nifedipine in the elderly or in patients with a history of cardiac disease, coronary artery disease, stroke, or who are concurrently taking antihypertensive medications.

The typical dose is 10 mg of oral nifedipine. The medication may be chewed and then held in the mouth and subsequently swallowed. Alternatively, open the capsule, place the medicine sublingually, and have the patient hold it in their mouth and then swallow the dissolved nifedipine. Attempt another technique if the food bolus does not pass with one dose of nifedipine.

NITROGLYCERINE

Sublingual nitroglycerine (i.e., 0.3, 0.4, or 0.5 mg) relaxes vascular smooth muscle and the smooth muscle contained within the middle and distal thirds of the esophagus. The use of sublingual nitroglycerine may allow the esophagus to dilate enough so that a food bolus can pass into the stomach.

Nitroglycerine should not be administered if the patient is hypotensive or has ingested a sharp or irregular-shaped foreign body. It should also not be administered if the patient is taking prescription medications for erectile dysfunction (e.g., vardenafil [Levitra], tadalafil [Cialis], or sildenafil [Viagra]). The combination of these medications with nitroglycerine can result in life-threatening hypotension. The onset of action is within 1 to 3 minutes, with a maximum effect by 4 to 5 minutes. The major side effect of nitroglycerine is hypotension, but that is short-lived. Administer one pill sublingually and allow 4 to 5 minutes for an effect. The dose may be repeated a second time. Attempt another technique if the food bolus does not pass after two doses of nitroglycerine.

PAPAIN

Papain is a proteolytic enzyme that has been used to dissolve an impacted food bolus. It is available in markets as meat tenderizer and in health food stores as a digestive supplement. It will dissolve the esophageal mucosa and continue to work its way through the esophageal wall and into the mediastinum if it does not first dissolve the food bolus. The use of papain to dissolve an impacted food bolus may be associated with a fatal esophageal perforation and, if aspirated, hemorrhagic pulmonary edema. **Papain should never be used to dissolve an impacted food bolus.**

SUMATRIPTAN

Sumatriptan is a 5-HT_1 agonist that reduces fasting fundic tone, prolongs fundic relaxation, and delays gastric emptying.[44] It increases the number of esophageal motor waves. This may be useful in cases of distal esophageal food boluses and coins, although no formal studies have been conducted.

SHARP AND POINTED FOREIGN BODIES

Removal of sharp and pointed objects requires extreme caution due to potential life-threatening complications, higher morbidity, and higher mortality. **An experienced Endoscopist should manage these cases.** It may be safer in some cases to consider surgical intervention. Toothpicks and bones are the most common foreign bodies requiring surgical removal.[45-47] Nails, needles, razor blades, safety pins, and dental prostheses may be removed endoscopically.[45-47] It is important to remember that "advancing points puncture while trailing points do not."[48] Objects longer than 5 cm and wider than 2 cm require removal as they will rarely pass through the pylorus.[49] Intravenous glucagon in doses of 0.4 to 0.6 mg in adults may be used to facilitate extraction from the stomach and duodenum.

Alligator forceps or snares are needed to grasp the object. Consider the use of a plastic overtube (**Figure 80-2*B***) for the removal of any sharp object.[34,50] The overtube should be at least 60 cm long to remove a sharp object from the stomach. This will limit objects for endoscopic removal to those smaller than 11 to 15 mm in diameter that fit within the overtube.[51] A soft latex protector hood may be used for the removal of large objects.

Razor blade ingestions may be managed with the flexible esophagoscope in adults. An alligator forceps, a snare, and an overtube will be needed. A razor blade that has passed the pylorus will often traverse through the intestinal tract without difficulty.

Safety pins and toothpicks pose additional risks due to their sharp ends that may perforate the esophagus. Push an open safety pin with the open end proximal into the stomach with the flexible endoscope. Turn the safety pin and grasp the hinged end to pull it out (**Figure 80-5**). A closed safety pin in the stomach will often pass without difficulty. Grasp a toothpick with an alligator forceps or snare very close to the tip so that the longitudinal axis of the toothpick is parallel to the scope as it is withdrawn into the overtube.[31]

Numerous other sharp objects are often encountered in the esophagus. Pens, pencils, thermometers, and wires are extracted in a fashion similar to a toothpick with a snare grasping the end of the object.[29] Glass may be withdrawn similarly or by using an end-hood attachment.[50,52]

Attempts should be made to remove all sharp and pointed foreign bodies before they pass from the stomach. Approximately 15% to 35% of sharp or pointed foreign bodies will cause intestinal perforation, especially in the area of the ileocecal valve.[53,54]

DISK OR BUTTON BATTERIES

Most disk or button batteries ingested (96%) are small and 7.9 to 11.6 mm in diameter.[55,56] Batteries less than 15 mm in diameter almost never lodge in the esophagus. Only 3% of button batteries are larger than 20 mm, but the batteries are responsible for severe esophageal injuries.[56-61] Guidelines for the evaluation and treatment of button battery ingestions are available.[7,62]

Button batteries cause injury by multiple methods.[57,63-68] Their electrical discharge causes hydrolysis and the creation of hydroxide ions in tissue, which causes alkali burns. Leakage of the high pH contents can result in alkali burns. Their physical presence can cause direct-pressure necrosis. Some button batteries contain mercuric oxide. Mercury toxicity can result if the mercuric oxide leaks from the batteries.

Most of these batteries contain manganese dioxide, silver oxide, mercuric oxide, zinc air, mercuric oxide, or lithium. Obtain anteroposterior and lateral abdominal and chest radiographs to distinguish between coins and button batteries (**Figures 80-1*D* and 80-7**). A double density shadow is suggestive of batteries. The coin has a much sharper edge. **Do not mistake a battery and a coin.**[69]

A button or disk battery lodged in the esophagus is a true emergency, and immediate removal is indicated to avoid the rapid corrosive action of the alkaline substance on the mucosa and subsequent complications.[57,70-73] Endotracheal intubation is usually necessary to protect the airway prior to endoscopic removal. The battery is removed from the esophagus under direct viewing using a through-the-scope balloon. A biopsy forceps may be needed to free the edge of the battery prior to removal. Alternatively, the battery may be pushed to the stomach and then removed using a basket, net, or polypectomy snare. **Do not use a Foley catheter or a magnet to remove a button battery without the aid of endotracheal intubation and general anesthesia due to the possibility of the button battery falling into the airway.**

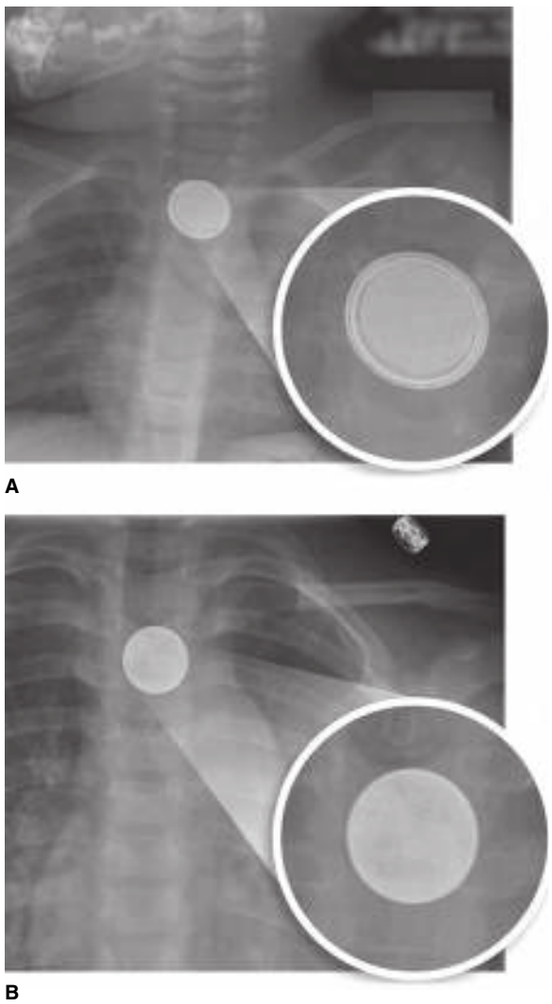

FIGURE 80-7. Radiograph of a chest with a foreign body in the esophagus. ***A.*** Button battery. ***B.*** Coin. (Photo used with permission from reference 57.)

Admit the patient to the Intensive Care Unit and monitor them for signs of perforation and sepsis if they suffered severe esophageal injury when evaluated by endoscopy after removal of the button battery.[65] Bronchoscopy may be performed to evaluate the extent of injury if it is localized to the anterior wall of the esophagus.

A button battery in the stomach generally need not be removed unless the patient is symptomatic with abdominal pain, tenderness, or gastrointestinal bleeding. Asymptomatic patients with button batteries less than 15 mm in diameter in their stomachs need follow-up abdominal radiographs every 24 hours to document forward progress until it is expelled. Endoscopically remove the battery if it is larger in size and has not passed within 48 hours in a child less than 6 years old.[55,74] Patients may be placed on H_2 blockers and/or proton pump inhibitors to decrease the acid in the stomach and decrease the battery reaction. Obtain serum and urine mercury levels and monitor them if mercury poisoning is anticipated.

MAGNETS

Magnets are commonly found in homes and are easily accessible to children. They are contained in appliances, jewelry, and toys. The ingestion of a single magnet is usually not problematic. **The ingestion of multiple magnets is considered an emergency requiring removal.**[71,75] The magnets can move separately through the gastrointestinal tract. They then have the potential to attract each other and trap bowel between them. This can result in pressure necrosis, fistula formation, and perforation.[75]

Perform plain radiographs to determine the number and the location of the magnets. Treat a single magnet ingestion as any other small, nonsharp, foreign body ingestion. Multiple magnets require removal. Remove them endoscopically if they are located within the esophagus and/or stomach. Consult a Surgeon for urgent removal if they have passed the pylorus versus careful inpatient monitoring by the Surgeon and frequent radiographs to localize the magnets.

NARCOTIC PACKETS

Body packing may be used as a method of an internal concealment of illegal drugs using latex condoms or plastic balloons. Endoscopic removal is contraindicated because any rupture or leakage of the packets will be fatal. Consult a Surgeon for removal in the Operating Room.[28]

FOLEY CATHETER TECHNIQUE

A Foley catheter has been successfully used to remove recently ingested, radiographically opaque, smooth and blunt foreign bodies from the esophagus.[76] This technique is inexpensive, has a high success rate, does not require hospitalization, and avoids the complications associated with endotracheal intubation and general anesthesia. Coins are the foreign bodies primarily removed with a Foley catheter. The technique has been used to remove button batteries, food boluses, and other smooth foreign bodies.

This technique cannot be used on all patients with an esophageal foreign body. **Do not use this technique in patients who are confused or uncooperative. Endotracheally intubate patients with an altered mental status, airway compromise, or potential airway compromise prior to Foley catheter removal of the foreign body.** Sharp or irregular-shaped objects can lacerate or perforate the esophagus upon removal. Known or suspected esophageal perforation is a contraindication to this technique. Patients with complete esophageal obstruction, as demonstrated by an esophageal air-fluid level on radiographs, are not candidates. Esophageal fibrosis, esophageal tumors, anatomic anomalies, or a history of prior esophageal surgery are also contraindications.

The equipment required for the technique is minimal. This includes topical anesthetic spray, a bite block, and a size 12 to 16 French Foley catheter with a 5 to 10 mL balloon. The technique may be performed ideally in a fluoroscopy suite or blindly in the Emergency Department. A water-soluble contrast agent is required if using fluoroscopy. **The most dangerous and immediate complication of this technique is airway obstruction. Airway and emergency equipment must be available if this technique is to be performed.**

Explain the procedure to the patient, including the sensations they will experience. The use of a topical anesthetic spray for the oropharynx is beneficial but optional. Its use may increase the risk of aspiration. The use of physical restraints (Chapter 232), procedural sedation (Chapter 159), and/or intubation (Chapter 18) may be required on a case-by-case basis. Preinflate the Foley catheter balloon with 5 to 10 mL of water-soluble contrast material. Inspect the integrity of the balloon. Withdraw the contrast material back into the syringe to deflate the balloon. The small amount of contrast material left in the balloon will facilitate identification under fluoroscopy. Place the patient prone in 10° to 20° of Trendelenburg or in the left lateral decubitus position in 10° to 20° of Trendelenburg. The fluoroscopy technique and then the blind technique are described in the following paragraphs.

Insert the Foley catheter. Some physicians insert a bite block and place the Foley catheter through the mouth (**Figure 80-8**). Others

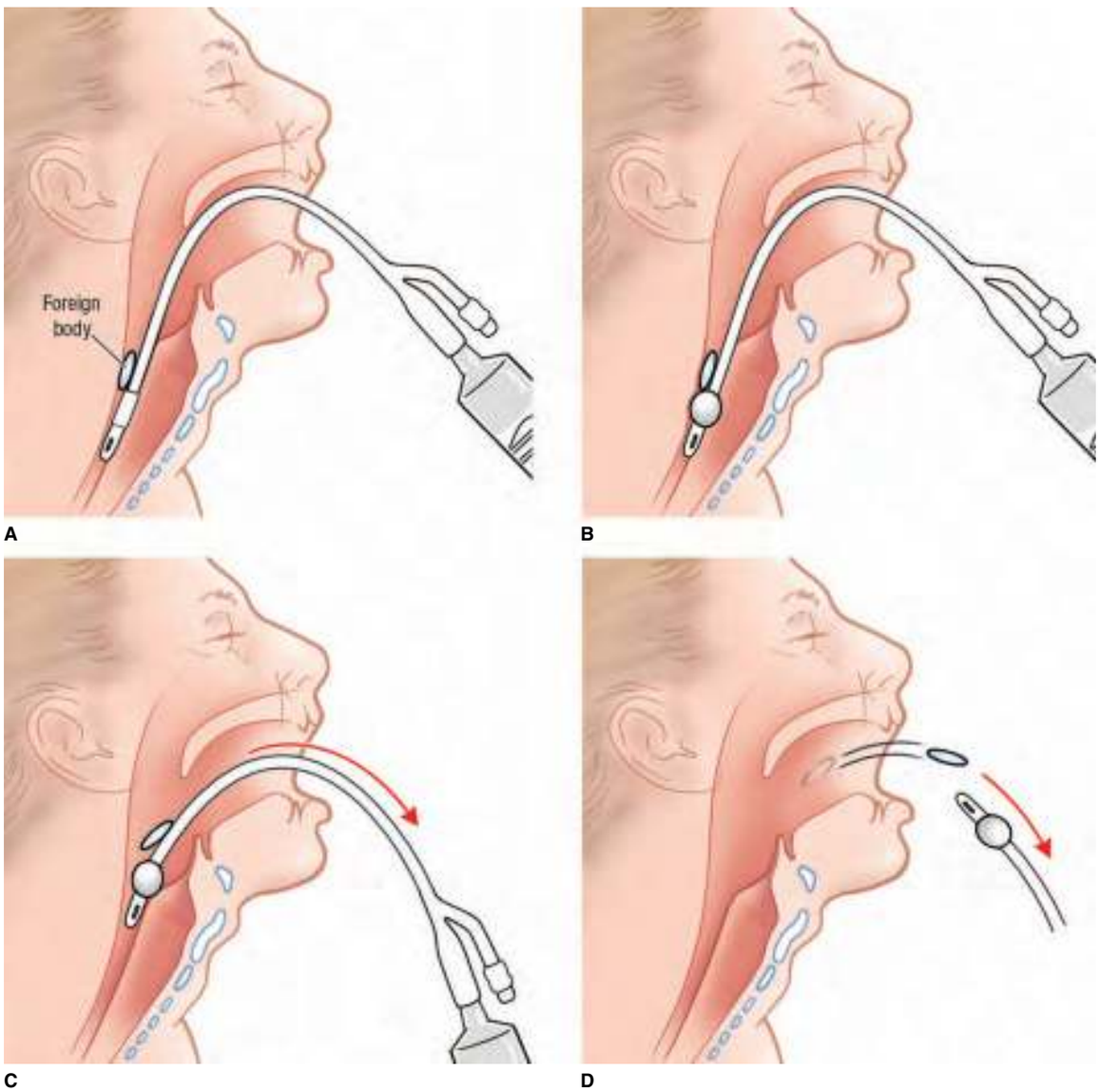

FIGURE 80-8. The Foley catheter technique to remove an esophageal foreign body. This illustration demonstrates the oral route for catheter insertion. The nasal route may also be used. ***A.*** The catheter is inserted and advanced until the balloon is just distal to the foreign body. ***B.*** The balloon is inflated. ***C.*** The catheter is withdrawn and pulls the foreign body into the hypopharynx. ***D.*** Completely withdrawing the catheter will pull the foreign body into and out of the mouth. Steps ***C*** and ***D*** are performed in one smooth motion.

use the nasal route. The oral route avoids the potential problem of lodging the foreign body in the nasopharynx with subsequent aspiration. Advance the Foley catheter under fluoroscopy until the balloon is just distal to the foreign body (**Figure 80-8*A***). Slowly inflate the balloon with 5 mL of contrast material (**Figure 80-8*B***). Stop inflating the balloon if the patient complains of pain. Deflate the balloon and reposition the catheter before reinflating. Withdraw the catheter with moderate and steady traction until it exits the mouth (**Figures 80-8*C* and 80-8*D***). **Stop withdrawing the catheter if resistance is met to prevent an esophageal tear or perforation.** The balloon may occasionally slide past the foreign body as the catheter is being withdrawn. Reinsert the catheter and inflate the balloon with 7 to 8 mL of contrast material and then withdraw the catheter. **Do not overinflate the balloon as it can rupture the esophagus. Do not attempt this technique more than twice.** The balloon will pull the foreign body ahead of it into the hypopharynx and then the mouth. Tell the patient to spit out the foreign body or grasp it (e.g., fingers, forceps, or a hemostat).

This technique may also be used "blindly" if fluoroscopy is not available. Estimate the distance on the radiographs from the mouth or nose to the foreign body. Place the Foley catheter over the radiograph with the balloon just distal to the foreign body. Mark the distance with tape on the catheter as it exits the mouth or nose. Insert the catheter into the patient until the tape is positioned at their mouth or nose. Use saline rather than contrast material to inflate the balloon. Remove the catheter and foreign body as above. Obtain repeat radiographs if the foreign body is not expelled with the catheter as it may have been pushed into the stomach. The remainder of the technique is the same as described for removal under fluoroscopy.

Complications with this technique are uncommon but do occur. This technique may not be able to remove the foreign body. Insertion of the catheter can cause laryngospasm or vomiting. The Foley catheter may enter the airway and result in coughing and laryngospasm. The esophagus may be lacerated or perforated if the foreign body is large, completely impacted, sharp, irregular, or has been in place for over 12 to 24 hours. Overinflation of the balloon can rupture the esophagus. Removal through the nose may result in epistaxis or impaction of the foreign body in the nasopharynx or nasal cavity. The most feared complication is complete or partial airway obstruction if the foreign body falls into the larynx.

BOUGIENAGE

The use of an esophageal bougie to push an esophageal foreign body into the stomach has been used in children.[77-81] The technique has been successfully used in asymptomatic children who have radiographs documenting a coin in the esophagus and no history of esophageal disease and when less than 24 hours have passed from the time of ingestion. The advantages of this technique are that it is quick, is simple to perform, does not require sedation, does not require intravenous access, decreases length of stay, and saves money when compared to endoscopy.[77,78,82]

Apply a topical anesthetic spray to the child's oropharynx. Select an appropriate size esophageal bougie. Physically restrain the patient while they are sitting upright or standing on the bed. Place the tip of the esophageal bougie at the mouth and run the rest of it to the earlobe and to just below the left costal margin. Place a piece of tape on the esophageal bougie 3 to 4 cm below the costal margin. Insert the esophageal bougie through the patient's mouth and advance it in one smooth motion until the tape is at the mouth. Remove the esophageal bougie. Obtain a repeat radiograph to confirm that the coin is now in the stomach.

Complications can occur and are avoided with proper patient selection. Esophageal perforation may occur if the patient has known esophageal disease, prior esophageal surgery or manipulation, a sharp foreign body, or an irregular-shaped foreign body. A foreign body present for more than 24 hours can cause pressure necrosis of the esophagus and increase the risk of perforation.

OROGASTRIC TUBE MAGNET

The orogastric tube magnet (OGTM) is an orogastric tube that has a magnet sealed within the distal end. It may be used to retrieve smooth metallic foreign bodies from the esophagus and stomach under fluoroscopy. Place the patient in the lateral decubitus position. Apply a topical anesthetic spray to the oropharynx. Insert the OGTM through the mouth. Advance the OGTM under fluoroscopy into the esophagus and directed toward the foreign body until it makes contact. Withdraw the OGTM and the foreign body out the mouth. This is a rarely used technique.

LARYNGOSCOPIC REMOVAL

Hypopharyngeal and upper esophageal foreign bodies may be removed with the laryngoscope.[83-85] The Emergency Physician is already familiar with the equipment. The curve of the forceps (e.g., Boedecker, Magill, or Tylke) allows the Emergency Physician to simultaneously view the forceps tip and the hypopharynx. Select the type and size of the forceps based on the availability of different forceps and sizes, the foreign body, and Emergency Physician experience using the forceps. The foreign body must not be embedded. Consider endotracheal intubation before foreign body removal. Have endotracheal intubation supplies and suction readily available.

Use a direct or video laryngoscope. Slowly insert the laryngoscope with the left hand until the epiglottis is observed. Gently insert the forceps and advance it to the foreign body. Open the jaws of the forceps. Move the forceps forward and grasp the foreign body. Gently withdraw the forceps and remove it with the foreign body.

ALTERNATIVE TECHNIQUES

Numerous other methods for the removal of esophageal foreign bodies have been tried and reported in the literature.[83,86-90] Surgical removal is rarely indicated except when complications (e.g., perforation or vascular penetration) have occurred. **Do not blindly push food boluses into the stomach.** This is hazardous because many patients have underlying esophageal disease. **Blind maneuvers can result in esophageal perforation.**

PEDIATRIC CONSIDERATIONS

Children less than 4 years of age are predisposed to occasionally have an esophageal foreign body. They explore objects with their mouth, have a high curiosity level, lack molars to chew food, and have poor motor and sensory coordination. Common esophageal foreign bodies include balls, button batteries, buttons, candies, coins, gumballs, jacks, marbles, partially chewed food, and pen caps. **A high index of suspicion must be maintained as a history of an ingestion may not be obtained.**[9-11,58-62]

Children may present asymptomatically or with cough, drooling, dysphagia, respiratory distress, unwillingness to eat, or vomiting.[65] An asymptomatic child with an esophageal coin or round object, except button batteries, can be admitted to the hospital and watched for 24 hours to see if the object will spontaneously pass into the stomach.[91] Remove the object with a esophageal bougie, a Foley catheter, or an endoscope if the child is symptomatic, becomes symptomatic, or if the object does not pass within 24 hours.[92] Foreign bodies in a mainstem bronchus require removal by an Otolaryngologist in the Operating Room with a rigid bronchoscope.[92]

Turning the child upside down or performing the Heimlich maneuver may move the foreign body into the trachea or larynx and cause a complete airway obstruction.

Approximately 25% to 30% of esophageal coins in children pass spontaneously without complications.[86] Treatment of these patients may reasonably include a period of observation of 8 to 16 hours, especially in older children with coins in the distal esophagus. A "combination" technique using fluoroscopy and endoscopic forceps, called the "penny-pincher," has been described to remove coins in children.[87] In this technique, grasping endoscopic forceps are covered by a soft rubber catheter and fluoroscopically guided into place. The forceps firmly hold the coin while the catheter protects the oropharynx and aligns the device with the coin. When the tip of the catheter is close to the upper edge of the coin, the retracted radiopaque prongs of the forceps are deployed. The coin edge is grasped and the coin is extracted.

It is important not to attempt these techniques or bougienage with patients with known anomalies of the gastrointestinal tract.[92] These anomalies may be anatomic, functional, or postsurgical. They can lead to the coin again becoming lodged, this time in a position that would require a more invasive intervention than endoscopy.[88]

ASSESSMENT

Observe the patient until the effects of the sedation, if used, have resolved per hospital policy. Give the patient a trial of liquids to swallow (i.e., drink) prior to discharge. The patient may be safely discharged after they tolerate oral fluids, are awake and oriented, and ambulate without difficulty. The patient should be driven home by another person if sedation was used to extract the foreign body.

Admission is required in a few instances. The inability to tolerate oral fluids is a contraindication to discharge. Admit patients with foreign bodies that are not retrievable in the Emergency Department for observation, further endoscopy, or operative removal. Any patient with evidence of esophageal perforation should be admitted, observed, and evaluated by a Surgeon.

Examine the esophagus endoscopically after removal of most foreign bodies. Consult a Gastroenterologist on anyone with an esophageal foreign body. Note the presence of any lacerations, perforations, or erosions that may require repair. Any strictures will require dilation in the future.

AFTERCARE

Instruct the patient to only ingest liquids for the first 12 to 18 hours. Begin a soft diet after this trial period of liquids and advanced to a general diet over 24 hours. Instruct the patient to take small bites of food and completely chew it before swallowing. They should immediately return to the Emergency Department if they experience abdominal pain, chest pain, dysphagia, hematemesis, hemoptysis, melena, or odynophagia, or if they have any concerns. All patients discharged from the Emergency Department should follow-up with a Gastroenterologist in 24 to 48 hours.[72,93] Some recommend a second endoscopic follow-up in 3 to 6 weeks for reevaluation of the esophagus. This is up to the consultant.

COMPLICATIONS

Esophageal perforation can occur due to the foreign body or the extraction procedure.[17,94-96] The patient may present with fever, tachycardia, shortness of breath, chest pain, abdominal pain, and crepitation in the neck. Perform an immediate chest radiograph and/or a radiographic contrast study if the extraction of the foreign body has been difficult.[1,2,4] An aortoesophageal fistula can form due to sharp foreign bodies in the esophagus that erode through the esophageal wall. This should be considered when the patient presents with dysphagia and significant hematemesis.[22,50] A latency period between the ingestion of the foreign body and hematemesis is usually 1 to 3 weeks, although it can occur years later.[29] Tracheoesophageal fistulas can occur more than a year after ingestion of the foreign body.[4]

Other life-threatening cardiopulmonary emergencies include mediastinitis, lung abscess, pericarditis, cardiac tamponade, pneumothorax, and pneumomediastinum. These complications mostly result from a delay in recognizing an esophageal perforation.[21,22,29] The remaining complications are discussed with each of the individual techniques of foreign body removal.

SUMMARY

Foreign body ingestion and food bolus impaction in the upper gastrointestinal tract are relatively common problems seen in the Emergency Department. Several studies have shown that 80% to 90% of foreign bodies in the gastrointestinal tract will pass spontaneously. Approximately 10% to 20% will require nonoperative intervention, while 1% will require surgical removal.[4,88] The most common presenting symptoms are chest or pharyngeal pain, odynophagia, dysphagia, foreign body sensation, and sialorrhea.[89] No foreign body could be found in almost half of patients despite performing emergent upper endoscopy.[90] The urgency for the endoscopic examination depends on the potential risk of aspiration or perforation, the type and size of the foreign body, the degree of obstruction, and the inability to manage secretions.[22] The presence of dysphagia and the immediate onset of symptoms would increase the probability of a positive foreign body finding on endoscopy.

Numerous techniques may be used to remove an esophageal foreign body. The technique of choice depends on the level of patient cooperation, the type of foreign body, the time since ingestion, physician experience and comfort, Gastroenterology consultation, and presenting symptoms. Complications can be minimized by the proper selection of the removal technique and the appropriate patient for the technique.

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81 Balloon Tamponade of Gastrointestinal Bleeding

Bashar M. Attar

INTRODUCTION

Gastroesophageal varices are among the most dangerous complications associated with cirrhosis. They are present in 50% to 60% of cirrhotic patients.[1] Approximately 30% of patients will experience an episode of variceal hemorrhage within 2 years of the diagnosis of varices.[1] The major factors that determine the risk of bleeding are variceal size and the degree of liver dysfunction.[1-3] Variceal bleeding stops spontaneously in 20% to 30% of cases.[1-4] It recurs in 70% of patients within 1 year of the initial episode.[1-4] Mortality is as high as 50% in the first year.[5] Variceal bleeding accounts for almost one-third of deaths in cirrhotic patients. Variceal hemorrhage has a poor prognosis if it is associated with coexisting or subsequent complications (e.g., rebleeding, infection, hepatic dysfunction, and portal pressure ≥ 12 mmHg).[6,7] Somatostatin and its analogues cause splanchnic vasoconstriction, leading to reduced portal pressure and portal blood flow, whereas venodilators reduce portal pressure by reducing resistance to portal flow.[7,8]

Doctors Sengstaken and Blakemore developed the concept of balloon tamponade to control bleeding esophageal and gastric varices in 1950. They developed a triple-lumen and double-balloon system that bears their names. **The Sengstaken-Blakemore (SB) tube is used as a temporizing measure to stop variceal bleeding until more definitive means are available (Figures 81-1 and 81-2).** The SB tube was designed for the control of esophageal varices and as a diagnostic aid to determine the source of hemorrhage into the stomach. There are variants of the SB tube (e.g., the Minnesota tube and the Linton-Nachlas tube). The Minnesota tube is a quadruple-lumen, double-balloon system (**Figure 81-3**).[9] The Linton-Nachlas tube was designed with only an esophageal balloon (**Figure 81-4**). It controls bleeding for esophageal varices. These tubes are rarely used today due to the significant complications and the widespread availability of endoscopy and its therapeutic interventions.

Removal of the balloon after its initial control of the bleeding results in a 50% rebleeding rate. The tubes are associated with serious complications (e.g., esophageal ulceration and perforation).[10] Emergency Physicians should become familiar with the SB and Minnesota tubes. They can be potentially lifesaving in an emergent setting to achieve bleeding control when endoscopy is contraindicated, fails, or is not immediately available.[11-13]

ANATOMY AND PATHOPHYSIOLOGY

The venous anatomy of the abdominal cavity and thorax is shown in **Figure 81-5**. The veins of the torso are devoid of valves. There are extensive connections (i.e., anastomoses) between the veins. This extensive network of veins allows blood multiple ways to return to the heart. Many of the veins are small. They can enlarge to accommodate the extra flow of blood.

Cirrhosis results in portal venous hypertension and a decrease in blood flow through the portal system. Collateral circulation develops so that the blood in the portal vein can find an alternative route to the inferior vena cava. Large collateral systems include the esophageal, gastric, paraumbilical, and rectal veins. The left gastric and esophageal veins form one of the larger collateral circulation channels due to the pressure generated from the portal venous system and the large volume of blood flow through them.

The collateral veins distend from the pressure and large volume of blood flow, which results in weakening of the walls of the vein. Ulceration and rupture of these veins can result in large amounts of blood entering the esophagus and stomach. Patients may present with bright red blood per rectum, hematemesis, hemorrhagic shock, hypotension, or complications associated with hypotension and hemorrhage (e.g., cerebrovascular accidents and myocardial infarction).

The inflated balloons will control most bleeding. The esophageal balloon exerts lateral pressure to tamponade esophageal varices. The gastric balloon exerts pressure on the gastric cardia to tamponade varices. Both balloons are inflated and meet to compress the gastric cardia against the diaphragm and block the upward flow of collateral blood from feeding the esophageal varices.

INDICATIONS

Consider balloon tamponade in patients with acute bleeding from esophageal and/or gastric varices if medical therapy (e.g., somatostatin, octreotide, vasopressin) or emergent endoscopic therapy

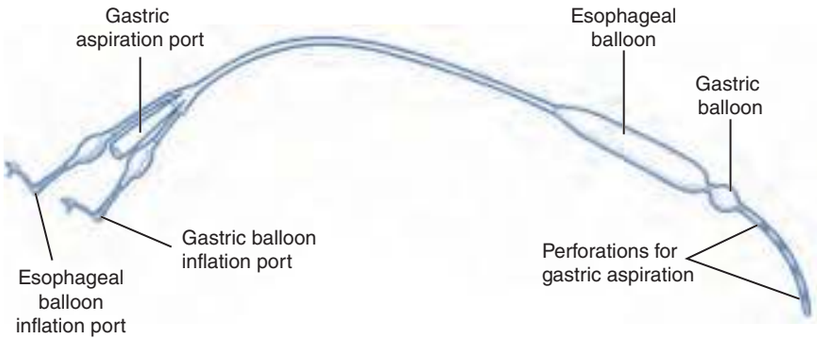


FIGURE 81-1. Schematic illustration of the Sengstaken-Blakemore tube.

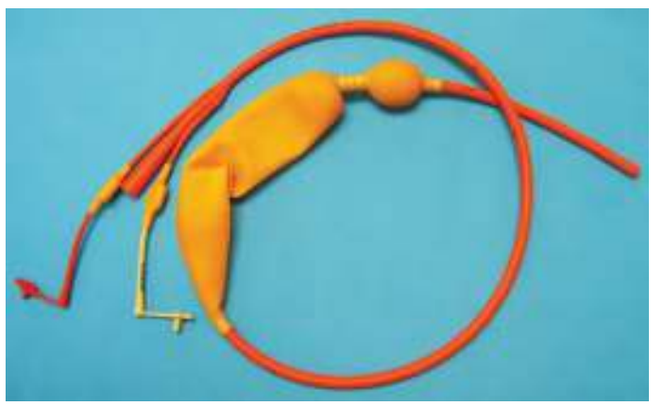
(i.e., banding or sclerotherapy) is not available, contraindicated, or unsuccessful.

CONTRAINDICATIONS

Absolute contraindications to balloon tamponade of variceal bleeding include a history of esophageal stricture, a history of recent surgery involving the gastroesophageal junction, or if the hemorrhage has terminated based on nasogastric lavage and aspiration.

There are numerous relative contraindications to balloon tamponade of variceal bleeding. Do not perform the procedure if the equipment required is defective or missing components. Untrained support staff make the procedure and aftercare more difficult. Significant active medical problems (e.g., respiratory failure, congestive heart failure, and cardiac arrhythmias) preclude the use of balloon devices. Incomplete gastric lavage leaving particulates in the stomach can cause retching and elevated intraabdominal pressure. The balloons will not properly position and may perforate the esophagus if the patient has a hiatal hernia. Esophageal ulcerations preclude the use of the esophageal balloon, but the gastric balloon may be used. The device is not helpful if a variceal source of bleeding cannot be demonstrated by examination, history, and/or nasogastric aspiration.

Secure the airway of patients with altered mental status, confusion, diminished gag reflexes, or hypoxemia or who are uncooperative by endotracheal intubation prior to this procedure. Recurrent bleeding after the initial successful tamponade should be followed by endoscopic or operative intervention.



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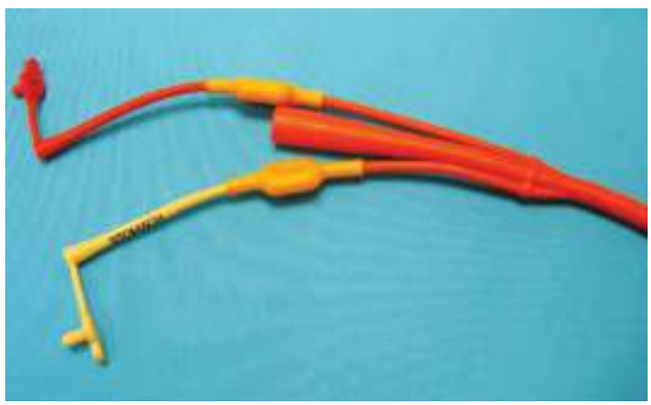

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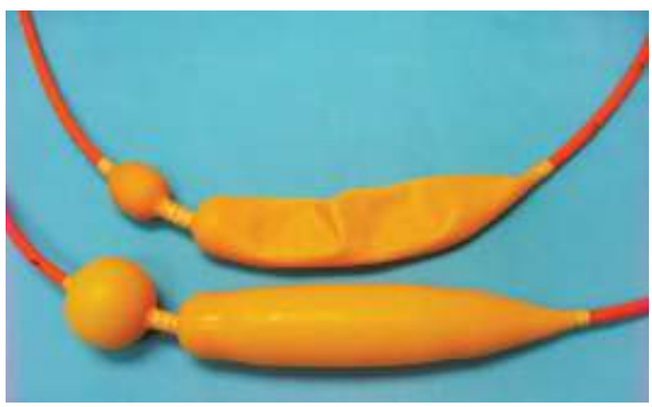
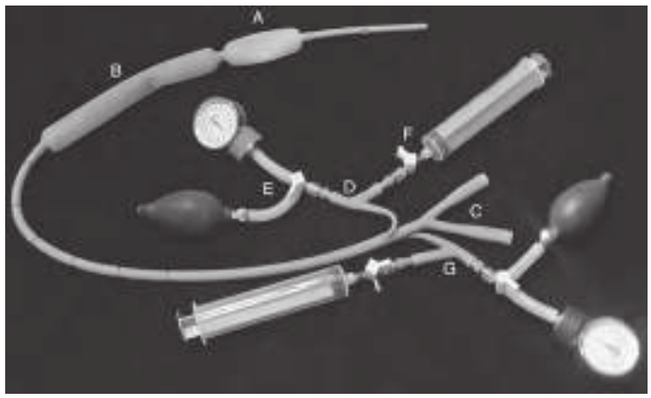


C

D

FIGURE 81-2. The Sengstaken-Blakemore tube. ***A.*** Overall view of the SB tube. ***B.*** The proximal ports. ***C.*** The esophageal and gastric balloons in the inflated and deflated states. ***D.*** The distal end.



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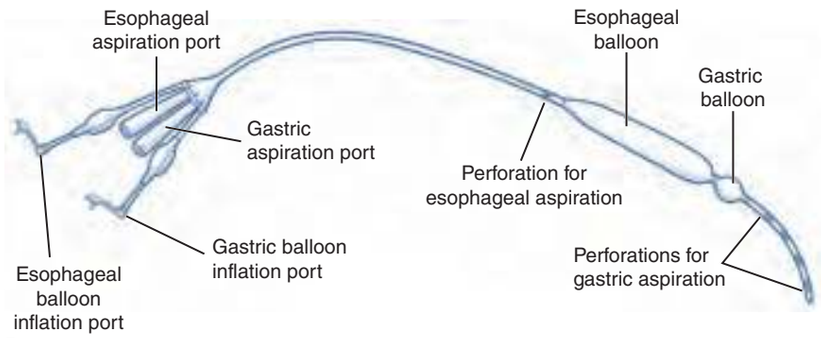


B

FIGURE 81-3. The Minnesota tube. ***A.*** Photo. Gastric balloon (A), esophageal balloon (B), gastric and esophageal suction ports (C), gastric balloon control branch point (D), gastric balloon control port with Christmas tree connector and three-way stopcock (E and F), repeat of gastric balloon set-up on the esophageal balloon side (G). (Photo used with permission from reference 9.) ***B.*** Schematic illustration.

EQUIPMENT

- SB tube or Minnesota tube
- Topical anesthetic spray
- Tongue blades
- Lidocaine or water-soluble jelly
- 60 mL syringe
- Catheter tips for the syringe
- Two wall-suction setups with plastic connectors and suction tubing
- Adhesive tape
- Rubber shod clamps, hemostats, or plugs
- Scissors

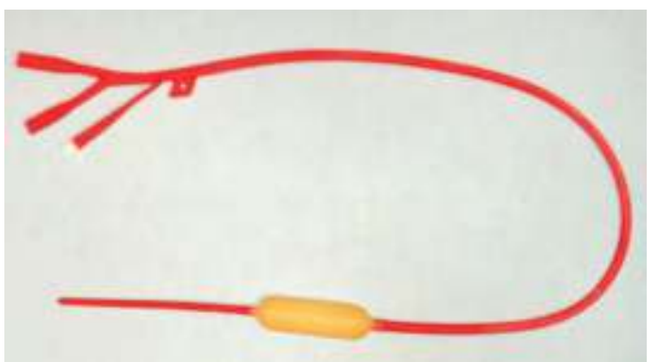


FIGURE 81-4. The Linton-Nichlas tube.

- Y-adapter or three-way stopcock
- Intravenous extension tubing
- Pressure bulb
- Mercury manometer or handheld manometer
- Bite block
- Nasogastric tube
- Football helmet, catcher's mask, or endotracheal tube holder

The SB tube is a triple-lumen and double-balloon system (**Figures 81-1 and 81-2**). It is available in a variety of sizes, including pediatric sizes. The proximal end contains three ports. A syringe attaches to the esophageal balloon inflation port to inflate the esophageal balloon. A syringe attaches to the gastric balloon inflation port to inflate the gastric balloon. The gastric aspiration port may be used to aspirate gastric contents, instill fluid into the stomach, or lavage the stomach. The ports may be opened and closed by the application of rubber shod clamps, hemostats, or plugs. The tube is made of a soft rubber and is extremely flexible. The proximal or esophageal balloon is elongated. The distal or gastric balloon is round. The maximum volume to inflate each balloon is specific to the manufacturer. Numerous perforations in the distal end of the tube allow for gastric aspiration and lavage.

The Minnesota tube is a quadruple-lumen and double-balloon system (**Figure 81-3**). It is similar to the SB tube with the exception of an additional port and lumen. The esophageal aspiration port allows saliva and esophageal secretions to be aspirated from a perforation just above the esophageal balloon. The only advantage of this tube over the SB tube is that the SB tube requires a nasogastric tube to be passed nasally or orally into the esophagus to aspirate secretions.

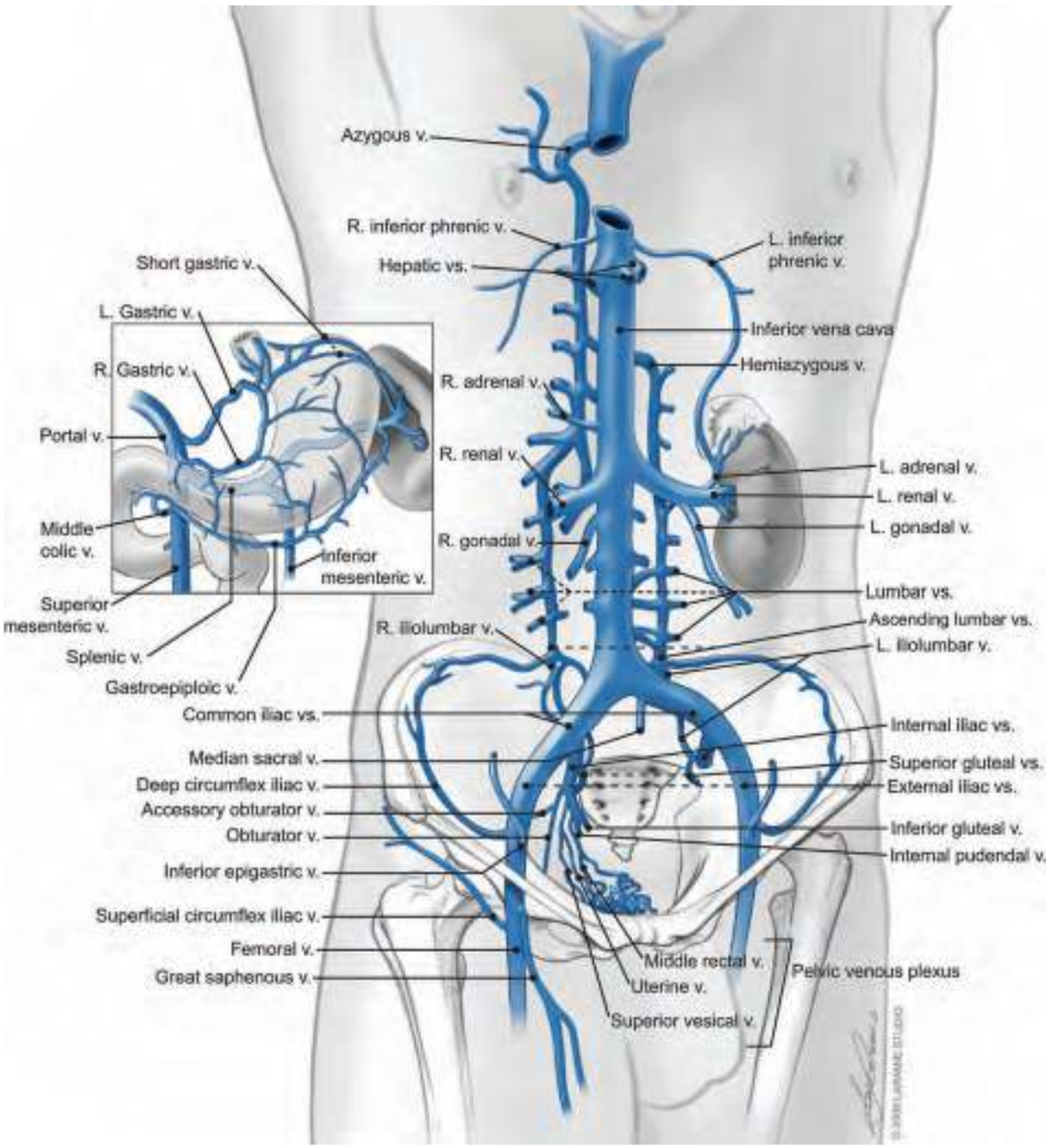
The Linton-Nachlas tube is a triple-lumen and single-balloon system (**Figure 81-4**). It is similar to the SB tube with the exception of one balloon. The esophageal aspiration port allows saliva and esophageal secretions to be aspirated from a perforation just above the esophageal balloon. The gastric aspiration port allows gastric secretions to be aspirated from below the balloon.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure, and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

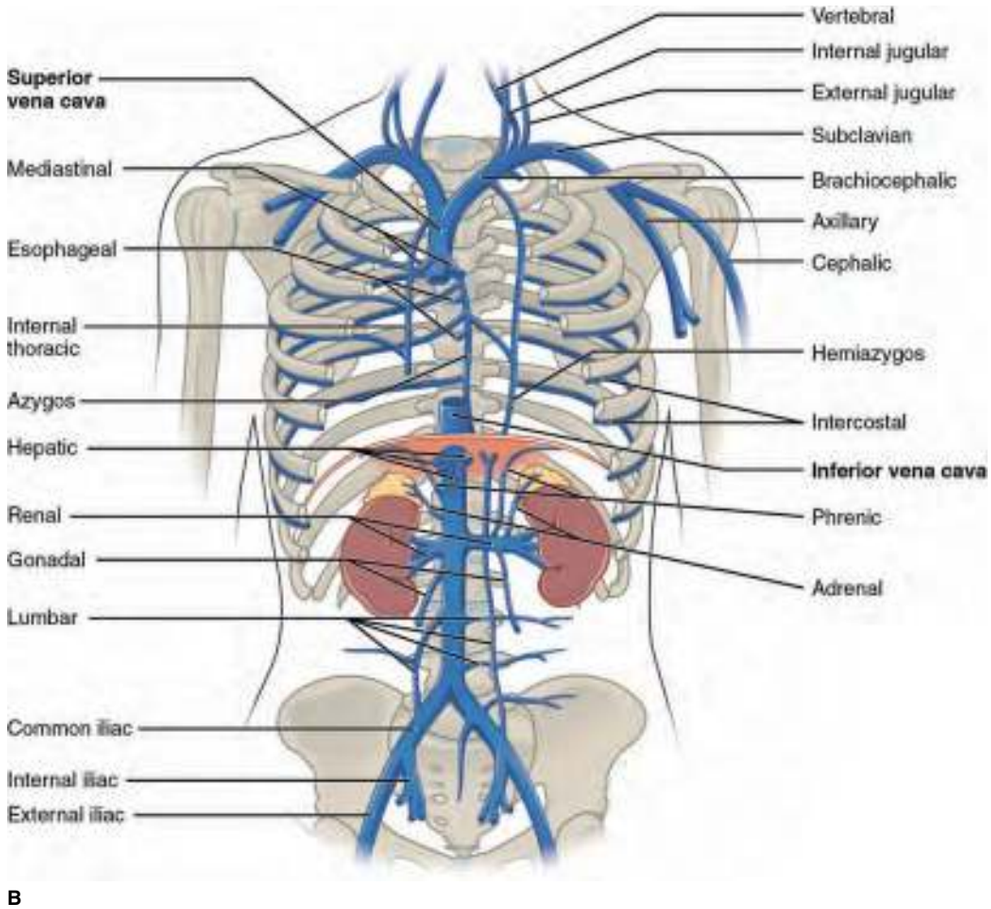
Thoroughly assess the patient's airway and breathing. **Have a low threshold to protect the airway by endotracheal intubation (Chapter 18).** Endotracheal intubation is required before performing the procedure if the patient has altered mental status, airway compromise, or the potential for airway compromise. Patients are often ill and require intubation for other reasons.[14] The procedure and the tube are not well tolerated, and intubation makes it easier. Endotracheal intubation reduces the risk of aspiration.

Establish intravenous access with at least two large-bore catheters. Apply cardiac monitoring, continuous pulse oximetry, and supplemental oxygen. The patient may require the judicious use



A

FIGURE 81-5. The veins of the torso. (Photos used with permission from Duty B, Daneshmand S: Venous resection in urological surgery. *J Urol* 2008; 180(6):2338-2342.)



B

FIGURE 81-5. (*continued*)

of intravenous sedatives (Chapter 159) and soft restraints (Chapter 232) during the insertion and inflation of the tube. Resuscitate the patient to stabilize them hemodynamically.

What is the ideal position? Place the patient sitting upright to semi-upright by elevating the head of the bed at least 45°. The procedure can be performed with the patient in the left lateral decubitus position if they cannot sit upright. The procedure can be performed with the patient supine if they are endotracheally intubated.

Apply topical anesthetic spray to the nostrils and pharynx. Insert a nasogastric tube (Chapter 75) or Ewald tube (Chapter 77). Aspirate the stomach contents. Evacuate the stomach with tap-water lavage through the nasogastric tube or Ewald tube. Remove the nasogastric or Ewald tube.

Prepare the equipment. Flush the aspiration ports with air to ensure their patency. Inflate the balloons with the maximum recommended volume of air and check for leaks. It is advisable to inflate the balloons under water to look for small leaks. Connect the mercury manometer to the balloon ports and inflate the balloons to the recommended pressures and check for leaks. Completely deflate the balloons. Record the manometric pressure of each balloon when it is deflated. Insert the plastic plugs in the balloon inflation ports or loosely clamp each port with a hemostat or rubber shod clamp.[15-17] Lubricate the SB tube with a water-soluble lubricant. Place the SB tube on a table. Position a nasogastric tube next to the SB tube so that the tip of the nasogastric tube is just above the esophageal balloon (**Figure 81-6**). Place a piece of tape on both tubes to mark a common point, proximal to the 50 cm mark of the SB tube, that will be outside of the patient. The nasogastric tube will be inserted after the SB tube is placed. Alternatively, position the tubes as above and tape both tubes together at two or three sites. This allows the nasogastric tube to be inserted simultaneously with the SB tube.

The SB tube may be inserted through the nose or through the mouth. The nasal route is more difficult to use and may be associated with a higher rate of complications. Many physicians recommend the nasal route in the awake patient. The oral route is the preferred route of insertion by some if the patient is intubated. Apply topical anesthetic spray into the nasal cavity and oropharynx if the SB tube will be placed nasogastrically. Apply topical anesthetic spray into the oropharynx if the SB tube will be placed orally. Place a bite block in the patient's mouth if the SB tube will be placed orally to prevent the patient from biting the SB tube.

TECHNIQUES

SB TUBE

Insert the lubricated SB tube until the 50 cm mark is located just outside the nares, or outside the teeth if inserted through the mouth. Flush the gastric aspiration port with air while auscultating over the epigastrium (**Figure 81-7*A***). A rush of air should be heard to ensure that the distal end of the SB tube is properly positioned within the stomach. If possible, confirm the position of the SB tube with portable plain radiographs or fluoroscopy (**Figure 81-7*B***).[14] **It is imperative to know that the gastric balloon is within the stomach before it is inflated.**[18] Apply suction to the gastric aspiration port.

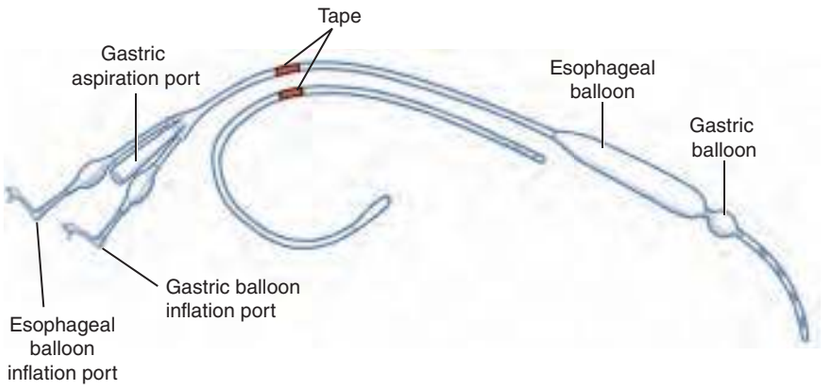
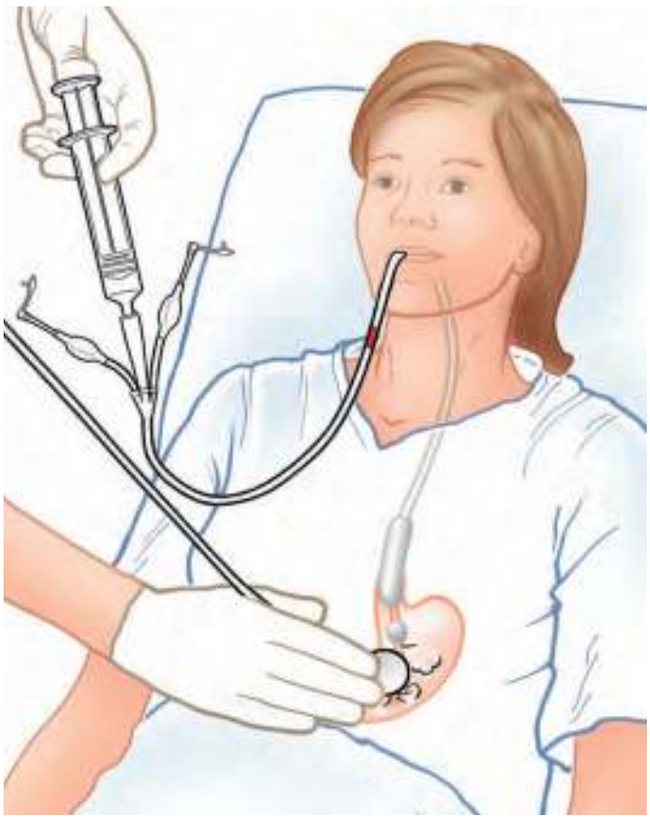


FIGURE 81-6. Preparing the nasogastric tube. Place the nasogastric tube alongside of the SB tube with the tip just above the esophageal balloon. Place tape on both tubes to mark a common point proximal to the 50 cm mark on the SB tube.

Remove the rubber shod clamp and plastic plug from the gastric balloon inflation port. Connect the Y-adapter with a handheld or mercury manometer and a pressure bulb or a 50 mL syringe with a catheter tip to the gastric balloon inflation port (**Figure 81-8**). Measure the uninflated intragastric balloon's pressure. **If the intragastric balloon pressure after intubation is 15 mmHg greater than that prior to the intubation, deflate the balloon as it may be located within the esophagus**. Inflate the gastric balloon in increments with 50 to 100 mL boluses of air (**Figure 81-9**). **Deflate the balloon immediately if the patient experiences chest pain. This signifies that the gastric balloon is in the esophagus**. Clamp the gastric balloon inflation port when the gastric balloon is inflated initially with 200 to 250 mL of air. Pull the SB tube back gently until resistance is felt as the gastric balloon lodges against the gastroesophageal junction (**Figure 81-10**).

Apply slight tension to the SB tube to occlude the veins at the gastroesophageal junction. **This tension must be maintained by one of several methods.** Fix the upper end of the SB tube as it exits from the mouth or nose to the crossbar of a football helmet or a catcher's mask (**Figure 81-11**). Apply over-the-bed traction with a 1 pound weight (**Figure 81-12**). Fix the upper end of the SB tube, if it emerges from the nostril, by a cuff of sponge rubber held in place by an adhesive tape band. The tube can be secured to a cervical collar (**Figure 81-13**).[19] Insert the nasogastric tube until the tape is at the level of the tape on the SB tube (**Figure 81-14**).

Connect the gastric aspiration port and the nasogastric tube to the suction source (**Figure 81-15**). Place the gastric aspiration port and the nasogastric tube on low intermittent suction. Unclamp the esophageal balloon port clamp. Attach the Y tubing, manometer,



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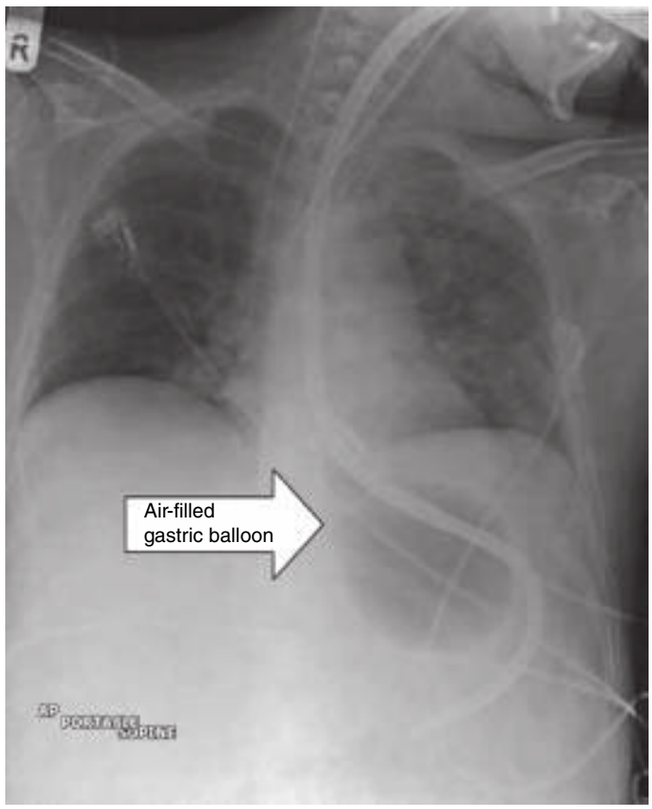


B

FIGURE 81-7. The SB tube has been inserted until the 50 cm mark is just outside the teeth. ***A.*** Inject 50 mL of air while auscultating over the epigastric area. A rush of air should be heard if the distal end of the SB tube is within the stomach. ***B.*** Chest radiograph of the subdiaphragmatic placement of the gastric balloon. (Photo used with permission from reference 14.)

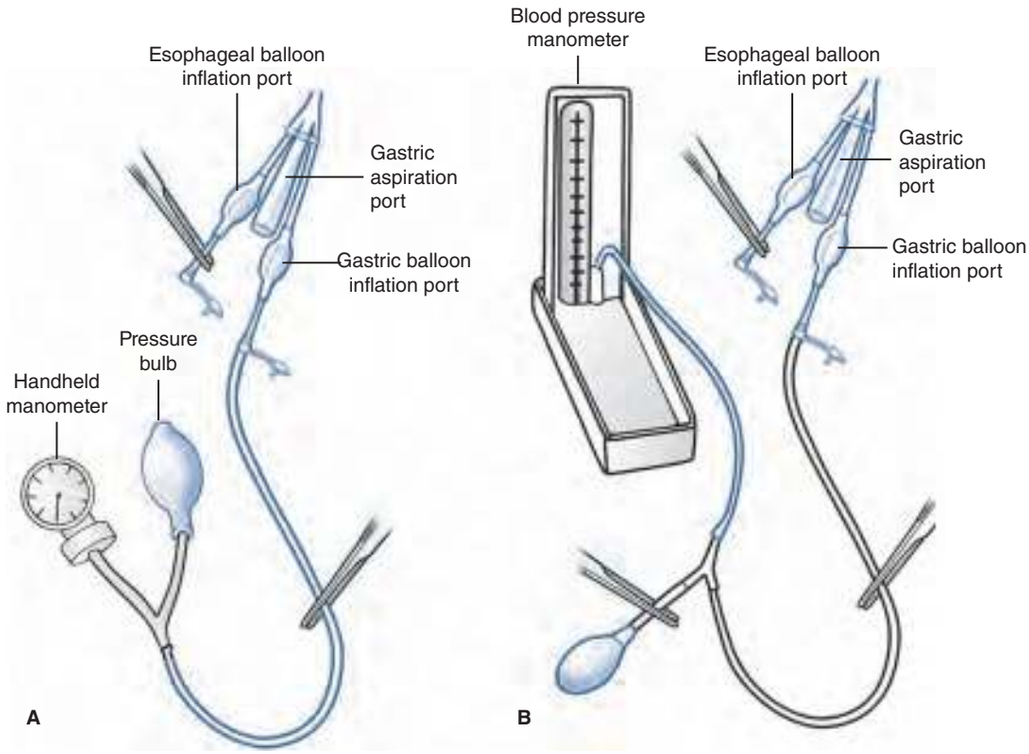


FIGURE 81-8. Connecting the handheld manometer (*A*) or mercury manometer (*B*) to the gastric aspiration port. A 50 mL syringe may be used if a handheld pressure bulb is not available.

and pressure bulb (or 50 mL syringe) to the esophageal balloon port (**Figure 81-15**). Inflate the esophageal balloon to a pressure of 25 mmHg if bleeding continues through the gastric aspiration port or the nasogastric tube. If bleeding continues to persist, increase the esophageal balloon pressure in 5 mmHg increments until 45 mmHg is achieved or the bleeding stops. Double clamp the SB tube to prevent accidental deflation of the balloons. The continued bleeding despite maintaining the esophageal balloon pressure at 45 mmHg

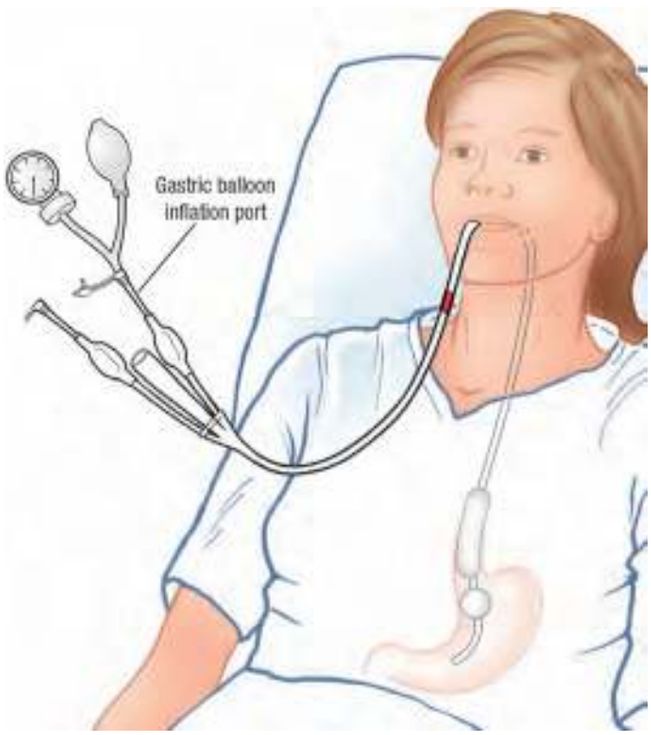


FIGURE 81-9. The gastric balloon is inflated with 50 to 100 mL increments of air to a volume of 250 to 300 mL.

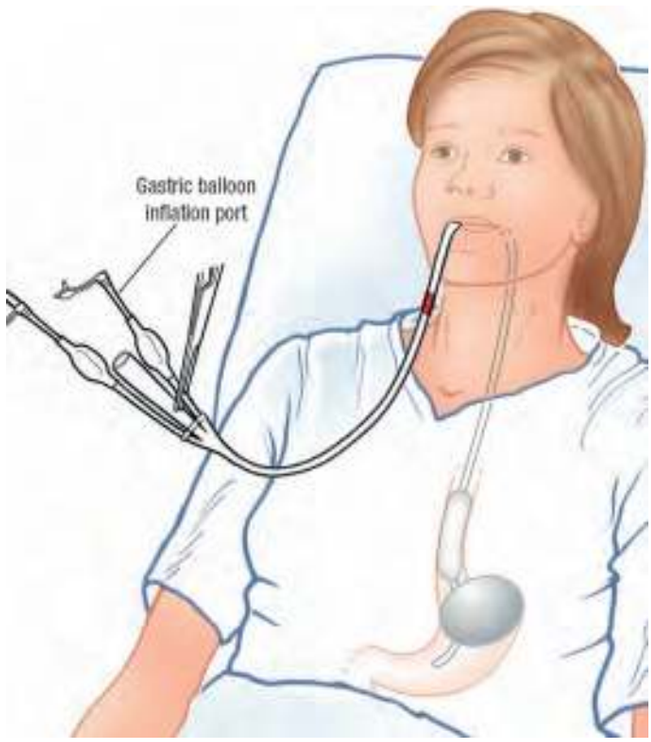


FIGURE 81-10. Tension is applied to the SB tube to lodge the gastric balloon against the gastroesophageal junction.

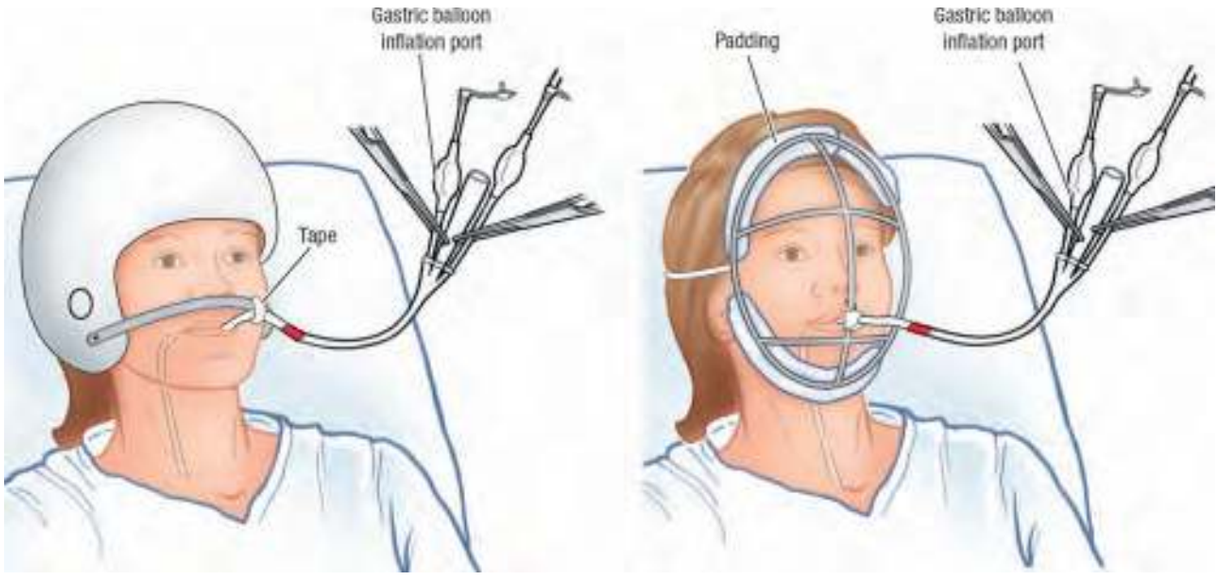


FIGURE 81-11. The SB tube is commonly secured to the faceguard of a football helmet (*A*) or to a catcher's mask (*B*).

is suggestive of a gastric wall varix. Check that the tube is snugged up firmly and taped securely. Fill the gastric balloon with more air gradually up to a total volume of 300 mL of air.

MINNESOTA TUBE

The above technique is also applicable to the four-lumen esophagogastric tamponade Minnesota tube. This tube is a modification of the triple-lumen SB tube that incorporates a separate esophageal suction port to the existing gastric suction port.[20,21] The design of the Minnesota tube may help prevent aspiration of esophageal contents.[22] Always check the manufacturer characteristics regarding the maximum inflation volume of both the gastric and esophageal balloons, as these are dependent upon the tube manufacturers.

LINTON-NACHLAS TUBE

The insertion of the Linton-Nachlas tube is similar to the SB tube. The only difference is that it only has the esophageal balloon and inflation port. It does not have a gastric balloon or gastric inflation port. It only stops esophageal bleeding. This tube is rarely stocked in the Emergency Department.

ALTERNATIVE TECHNIQUE

An effective method for placement of the SB tube is endoscopically.[23,24] This method will avoid the need to obtain radiologic confirmation of the gastric balloon within the stomach.[23] Delay in treatment while waiting for a radiologic confirmation may put patients at risk, as they are almost always in critical condition and require immediate attention.[4-6] If endoscopic intervention is not successful, pass an SB tube and confirm the position of the gastric balloon under direct

FIGURE 81-12. A pulley-traction device.

FIGURE 81-13. The tube is secured to a cervical collar. (Photo used with permission from reference 19.)

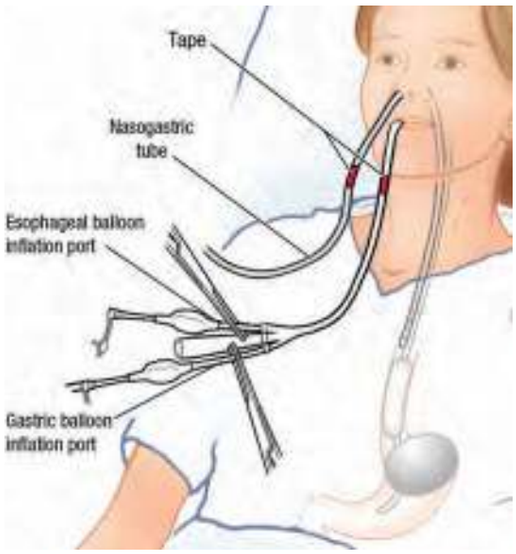


FIGURE 81-14. The nasogastric tube is inserted until the tape mark lines up with the tape mark on the SB tube. The football helmet/catcher's mask is omitted from this illustration for the sake of clarity.

visualization through the endoscope. The balloons are inflated and secured in the usual fashion.[23]

ASSESSMENT

Check the pressure in the balloons periodically with the mercury manometer or keep the manometer attached for constant monitoring. Obtain a portable radiograph to confirm proper placement of the SB tube and the inflated balloons. The patient may be reclined, but always maintain at least 6 to 10 inches of head elevation on the bed to prevent aspiration in the awake patient. Tape a scissors to the head of the bed for quick access in case the balloon requires emergent deflation (**Figure 81-16**).

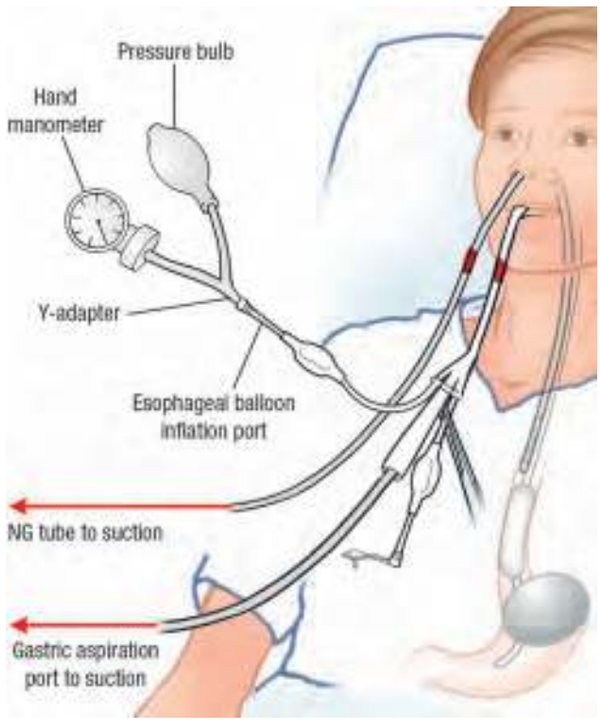


FIGURE 81-15. The esophageal balloon is inflated after the gastric aspiration port and the nasogastric (NG) tube have been attached to a suction source. The football helmet/catcher's mask is omitted from this illustration for the sake of clarity.

FIGURE 81-16. Emergent removal of the SB tube. Cut the SB tube between the ports and the patient to deflate both balloons rapidly.

AFTERCARE

If the bleeding is controlled, reduce the esophageal balloon pressure by 5 mmHg every 3 hours until 25 mmHg is reached without bleeding from the nasogastric tube in the esophagus or the gastric aspiration port. Deflate the esophageal balloon for 5 minutes every 6 hours to avoid esophageal pressure necrosis. Give the patient nothing by mouth. Oral medications, if required, may be administered through the gastric aspiration port. Check the tension on the SB tube every 3 hours and adjust it as necessary. Verify patency of the SB and nasogastric tubes by checking the gastric and esophageal return regularly and periodically flushing both lumens.

Monitor the patient continuously for signs of chest pain, respiratory distress, and aspiration. **Migration of the esophageal balloon into the hypopharynx of an awake patient will result in respiratory distress. This situation is a true emergency, and the tube should be removed immediately**. Cut the SB tube between the ports and the patient with a scissors (**Figure 81-16**). The balloons will immediately deflate and allow the removal of the SB tube.

The esophageal balloon should not remain inflated for more than 24 hours to avoid mucosal necrosis. The SB tube is usually left in place with the gastric or gastric and esophageal balloons inflated for 24 hours if variceal bleeding is controlled. If there is no bleeding after 24 hours, deflate the esophageal balloon and leave the gastric balloon inflated for an additional 24 hours. The SB tube may be left in place for an additional 24 hours after both balloons are deflated. If variceal hemorrhage recurs, reinflate the appropriate balloons while alternative therapy to control bleeding is sought. Patients who rebleed have a higher mortality rate. Consider other therapeutic interventions (e.g., rubber banding, sclerotherapy, transjugular intrahepatic portocaval shunt, or surgery). Remove the SB

tube if hemostasis persists for 24 hours after deflation of both balloons. Control of esophageal variceal bleeding can be achieved by balloon tamponade in 50% to 94% of patients.[25-29] Rebleeding occurs in 38% of patients.

COMPLICATIONS

Major complications occur in up to 15% of patients.[25-29] Lethal complications have been described in up to 6.5% of patients.[25-29] **Complications associated with the SB tube are often life-threatening**. The airway may become occluded due to proximal migration of the esophageal balloon into the hypopharynx of the awake patient from traction on the SB tube.[30] The patient will begin choking and gagging if not intubated. **Cut the SB tube distal to the ports to immediately deflate the balloons and allow the SB tube to be removed.** Pulmonary aspiration and subsequent pneumonia may occur during SB tube insertion. Consider airway protection by endotracheal intubation in all patients prior to insertion of the SB tube.[25]

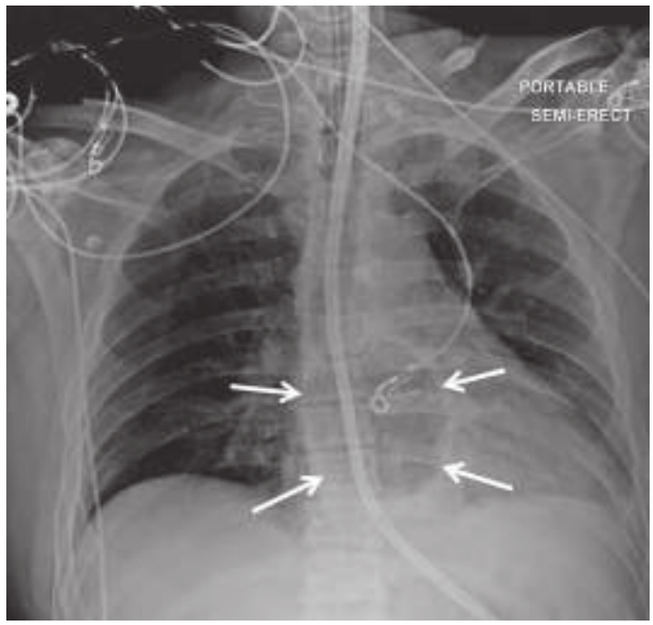
The SB tube may still become malpositioned and end up in the airway despite endotracheal intubation.[18,31] Inflation of the balloons in the airway can result in respiratory distress, respiratory tract obstruction, tracheal or bronchial rupture, or tracheoesophageal fistulas from pressure necrosis. **It is essential to confirm proper placement prior to inflating the balloons.** A video laryngoscope can be used to place the tube into the esophagus and not the trachea.[32]

Excessive balloon pressure or prolonged balloon inflation may lead to pressure necrosis and ulcerations of the esophagus, gastroesophageal junction, and/or stomach. Periodic deflation of the esophageal balloon every 6 hours will help prevent this. Rupture or lacerations of the esophagus, stomach, or small intestine may occur.[26] Esophageal rupture can result in mediastinitis, abscess formation, and sepsis. These can be prevented by adhering to proper balloon inflation techniques with pressure monitoring. Cardiac arrhythmias and pulmonary edema can occur and require continuous monitoring in the setting of an Intensive Care Unit. Distension of the stomach can cause inferolateral ST-segment elevation on the electrocardiogram.[33] The balloon can cause extracardiac compression of the heart.[34] Pulmonary edema is often due to pressure from the esophageal balloon on mediastinal structures.

Numerous other complications are associated with the use of the SB or Minnesota tube. Inflation of the gastric balloon in the esophagus can cause a ruptured esophagus (**Figure 81-17**).[35-37] The tube can break above the balloons and require endoscopy to retrieve the balloons.[38] Unintentional deflation of the balloons can be prevented by the application of rubber shod clamps or hemostats to the balloon inflation ports after the balloons are inflated. Cut the SB tube distal to the ports if the balloons will not deflate. The patient may become agitated from the discomfort of the tube, migration of the tube into the hypopharynx resulting in hypoxemia and asphyxiation, or as chest and back pain is experienced from a misplaced or overdistended balloon. Hiccoughs are due to pressure on the diaphragm by the balloons. Excessive traction on the tube can result in epistaxis or pressure necrosis of the lips, nose, or tongue.[39] The helmet can cause pressure necrosis of the face.[40] Use air and not a liquid to inflate the balloons. Liquid in the balloons causes them to be heavy, increases the risk of pressure necrosis, and makes them hard to deflate.

SUMMARY

Balloon tamponade of variceal bleeding is an uncommonly performed procedure in the Emergency Department. The SB tube plays an important role in the temporary control of hemorrhage from esophageal or gastric varices.[27-29] It is used in cases of variceal bleeding, usually documented by endoscopy, that continues despite aggressive medical management (e.g., lavage, correction of blood clotting abnormalities, intravenous somatostatin or vasopressin infusion, rubber banding, and emergent sclerotherapy).[23,41-43] The SB tube can be placed if these methods are contraindicated or unavailable. Sustained bleeding control occurs in only 40% to 50% of patients.[44] Balloon tamponade has been shown to be as effective as intravenous vasopressin in controlling esophageal variceal bleeding. Only 25% of patients with ascites, jaundice, and encephalopathy achieve lasting hemostasis with balloon tamponade. Long-term efficacy in terms of rebleeding depends in part on the patient's



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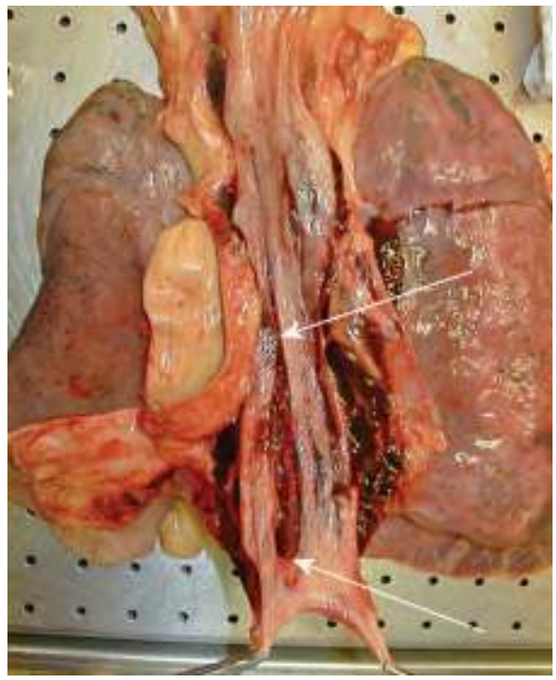

B

FIGURE 81-17. The inflation of the gastric balloon (*arrows*) in the esophagus. ***A.*** Chest radiograph. (Photo used with permission from reference 37.) ***B.*** Autopsy photo of an esophageal rupture between the arrows. (Photo used with permission from reference 35.)

underlying liver disease.[23] Maintain a low threshold to intubate the patient endotracheally to prevent aspiration, protect the airway, and prevent airway occlusion from migration of the esophageal balloon.

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82 Gastrostomy Tube Complications and Replacement

Erika Flores Uribe and Erick Eiting

INTRODUCTION

The gastrostomy tube (G-tube) is a commonly used device that provides prolonged enteral support in patients who are unable to obtain sufficient nutrition orally. Indications for enteral feeding tube placement include prolonged neurogenic or mechanical dysphagia, prolonged mechanical ventilation, and poor intake. Relatively common complications related to enterostomy tubes encountered in the Emergency Department include tube dislodgement, tube occlusion, leakage around the tube, and skin changes (e.g., hypergranulation, ulceration, erythema, and infection).[1,2] Some less common problems include buried bumper syndrome, peritonitis, gastric outlet obstruction, gastro-colo-cutaneous fistula, and stomal herniation.[1-5] Some of the complications may require replacing G-tubes.[6] Simplified techniques for their placement and improved materials have made gastrostomies common in the outpatient setting. Emergency Physicians fill a valuable role in solving G-tube problems as many

patients present to Emergency Departments with various G-tube complaints. This chapter reviews the methods and materials used in gastrostomies and the approaches to replacing displaced or malfunctioning G-tubes.

ANATOMY AND PATHOPHYSIOLOGY

Familiarity with the basic techniques used to create gastrostomies and the characteristics of common G-tubes is helpful in solving problems with their function and replacement.

PLACEMENT OF G-TUBES

The choice of access route (i.e., gastrostomy, gastrojejunostomy, or jejunostomy) and the choice of placement technique (i.e., surgical, endoscopic, or radiologic) often depend on individual patient issues and the treating physician (e.g., specialty, experience, and preference). Feeding tubes have been surgically placed in patients for more than a century. Three main procedures that remain in use today are the Stamm (described in 1894), the Witzel (described in 1891), and the Dupage and Janeway (described in 1913) (**Figure 82-1**).[7-12] They all require a laparotomy under general anesthesia and provide long-term access to the stomach for feedings or decompression while attempting to minimize the potential for gastric leakage.

Each of the techniques attempts to create a leakproof interface between the stomach, the feeding tube, and the anterior abdominal wall. The Stamm gastrostomy secures the stomach to a G-tube using a double purse-string suture to invaginate the stomach about the feeding tube (**Figure 82-1*A***). The Witzel technique places the G-tube through a seromuscular tunnel in the stomach wall (**Figure 82-1*B***). The Janeway technique creates a formal tunnel from a gastric flap to envelop the G-tube and form a gastrocutaneous stoma (**Figure 82-1*C***). **All three techniques involve suturing the stomach wall to the undersurface of the abdominal wall.** The stomach wall remains attached to the abdominal wall, and chance of intraperitoneal contamination is decreased if a surgically placed G-tube is accidentally dislodged in the early postoperative period. These surgical gastrostomies are considered long-term or semipermanent stomas.

Modern endoscopic techniques have provided a less invasive option for the placement of percutaneous feeding tubes (i.e., G-tubes, percutaneous endoscopic gastrostomies, or PEGs).[13] Distinct advantages of endoscopically placed G-tubes over surgically placed tubes include relative ease of placement, avoidance of general anesthesia, smaller incisions, and a lower morbidity rate.[8,13,14] The disadvantages of endoscopic techniques include the inability to place tubes in patients with thick abdominal walls, a history of multiple previous operations, the presence of abdominal wall hernias, and an increased risk of injuring overlying viscera during the "blind" placement of the PEG tube. Another disadvantage is the unique danger of accidental dislodgement of a newly placed PEG tube. If a new PEG tube is dislodged, the stomach wall gastrotomy can pull away from the abdominal wall, allowing the escape of gastric secretions and feedings with resultant peritonitis.

Popular endoscopic techniques in use today require two persons—an Endoscopist and an operator at skin level. An endoscope is first advanced into the stomach (**Figure 82-2*A***). The stomach is then insufflated with air to displace adjacent or overlying loops of bowel and appose the stomach wall to the anterior abdominal wall (**Figure 82-2*B***). The Endoscopist then visualizes the light of the endoscope as it transilluminates the abdominal wall. They then pierce the anterior abdominal wall with a hollow-bore needle. (**Figure 82-2*C***). The Endoscopist confirms placement into the gastric lumen (**Figure 82-2*C***).

The G-tube is then placed. A guidewire is fed into the stomach through the needle, grasped with an endoscopic snare, and pulled out of the patient's mouth (**Figure 82-2*C***). The "Ponsky pull" technique uses this guidewire as a means for pulling the G-tube into position through the patient's mouth (**Figure 82-3*A***). The guidewire is secured around a G-tube and pulled retrograde from the mouth to the exit site on the anterior abdominal wall. The "Ponsky push" technique involves pushing the G-tube down and over the guidewire toward the gastrostomy site (**Figure 82-3*B***).[15] The "Russell poke" eliminates the need to direct the G-tube through the mouth.[16] A dilator is used under direct visualization to enlarge the skin puncture site. An introducer and peel-away sheath are then used to place the G-tube directly through the anterior abdominal wall (**Figure 82-3*C***). **Regardless of which technique is used to establish the gastrostomy, the stomach is not sutured to the abdominal wall**. A fibrous tract eventually forms between the anterior abdominal wall and the stomach. **The maturation of this tract is an important consideration in assessing G-tubes.**

Endoscopy-guided percutaneous gastrostomy (PEG) tubes are commonly placed in patients in whom oral intake is contraindicated.[17] A PEG tube may not be preferred in mechanically ventilated or critically ill patients due to risk of aspiration. Administration of the nutrients directly into the jejunum through a percutaneously placed jejunostomy tube is recommended to bypass the stomach and decrease the risk for aspiration.[18] Other indications for direct administration of nutrients into the jejunum include malfunction of the swallowing mechanism, gastric outlet obstruction, gastroparesis, pancreatitis, and the presence of esophageal fistulas or enteric foregut leaks.[18] A meta-analysis found that radiologically guided gastrostomy compared favorably with surgical and endoscopic gastrostomy, with similar or improved success and complication rates.[19]

Radiologic percutaneous gastric fixation is only required for tubes that are pushed through the abdominal wall (i.e., percutaneous radiological G-tubes or percutaneous radiological gastrojejunostomy

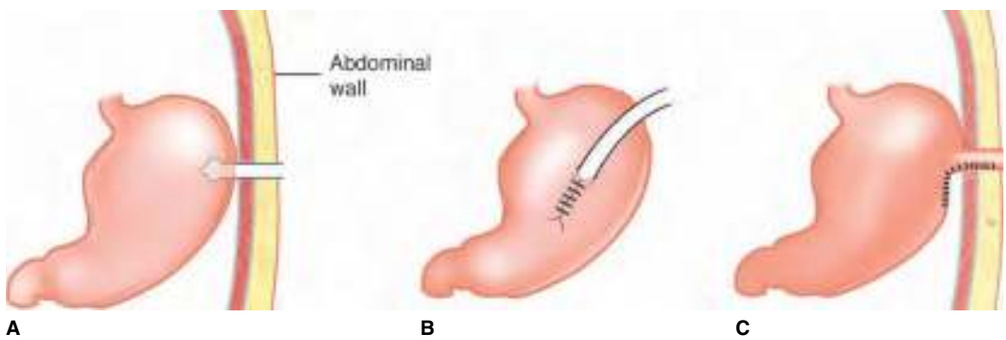

FIGURE 82-1. Surgical gastrostomies. ***A.*** The Stamm technique. ***B.*** The Witzel technique. ***C.*** The Janeway technique.

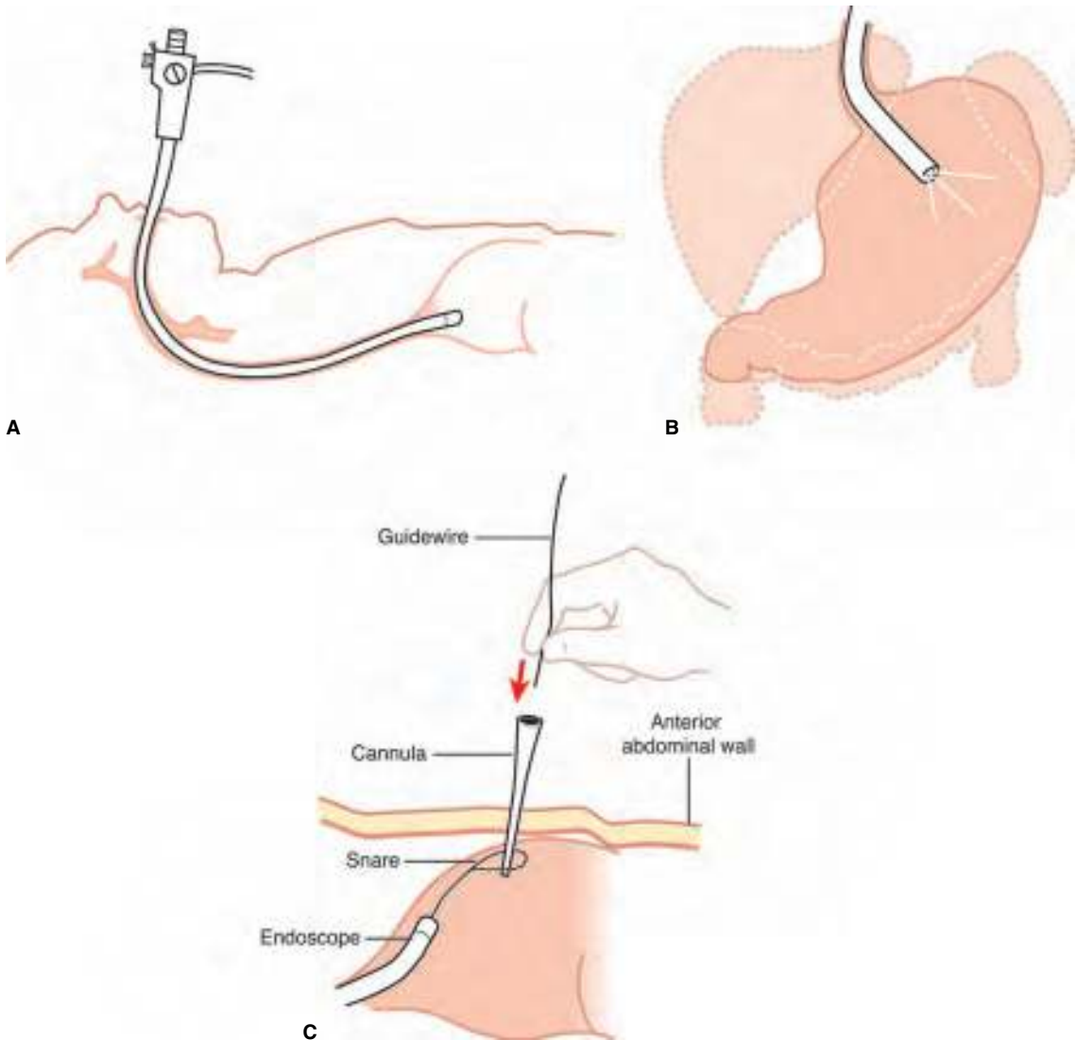

FIGURE 82-2. Endoscopic G-tube placement. ***A.*** The endoscope is inserted. ***B.*** The stomach is inflated with air and transilluminated. ***C.*** The anterior abdominal wall is pierced with a cannula or needle under endoscopic guidance. A suture or guidewire is then introduced into the gastric lumen and grasped with a snare.

[GJ] tubes). There are two kinds of anchor devices made of short metal bars attached to a surgical suture: the Cope suture anchor (Cook, Bloomington, IN) and the T-fastener (Boston Scientific, Natick, MA). Anchors are inserted into the stomach using a gastropexy set. Multiple anchors are required for 12 French or larger tubes. Tract dilatation with fascial dilators exerts a large amount of force. The performance of gastropexy is controversial but standard.

TYPES OF G-TUBES

There are many types of G-tubes available. A basic understanding of G-tubes is all that is needed to diagnose and treat most problems. Familiarity with the characteristics of G-tubes is helpful in assessing tube function and determining appropriate replacements.

Not all G-tubes can be removed safely in the Emergency Department. Standard de Pezzer and mushroom catheters modified with rings or bolsters may require endoscopy for removal. GJ-tubes will need advanced replacement techniques to appropriately place the jejunal part of the tube. Contact the provider who inserted the tube to determine if removal can be completed safely in the Emergency Department when in doubt. **The visible portion of the G-tube outside the skin may or may not indicate what type of internal stabilization exists.**

The G-tube is simply a conduit for enteral feedings. The essential features of the G-tube include the tube, an internal bolster, an external fixation (i.e., bolster or retention) device, and the ports (**Figure 82-4**). The main body of the G-tube is made of silicone or polyurethane. It is designed to minimize tissue reactions and optimize patient comfort. G-tubes come in a variety of sizes ranging from 12 to 24 French. Some are reinforced with an inner steel wire. Many come with external identification marks to denote caliber, commercial brand name, centimeter markings to aid in positioning, and radiopaque lines to aid in radiographic identification (**Figure 82-4**).

The internal bolster fixes the G-tube within the lumen of the stomach and creates a seal to discourage leaking (**Figure 82-4**). Commercially available PEG tubes come with a variety of choices for internal bolsters including balloons, crossbars, T-bars, flanges, round disks, three-leaf retainers, soft domes, and others (**Figure 82-5**). The most basic tubes use a balloon as an internal bolster. Surgical gastrostomies commonly use Foley balloon catheters, mushroom-tip de Pezzer catheters, or Malecot catheters. Some internal bolsters are deformable and allow removal with gentle traction on the external tube. Others are not intended to give way with traction and require more invasive techniques for removal. **The nature of the internal bolster will determine if a G-tube can be removed in the Emergency Department or requires endoscopic removal.**

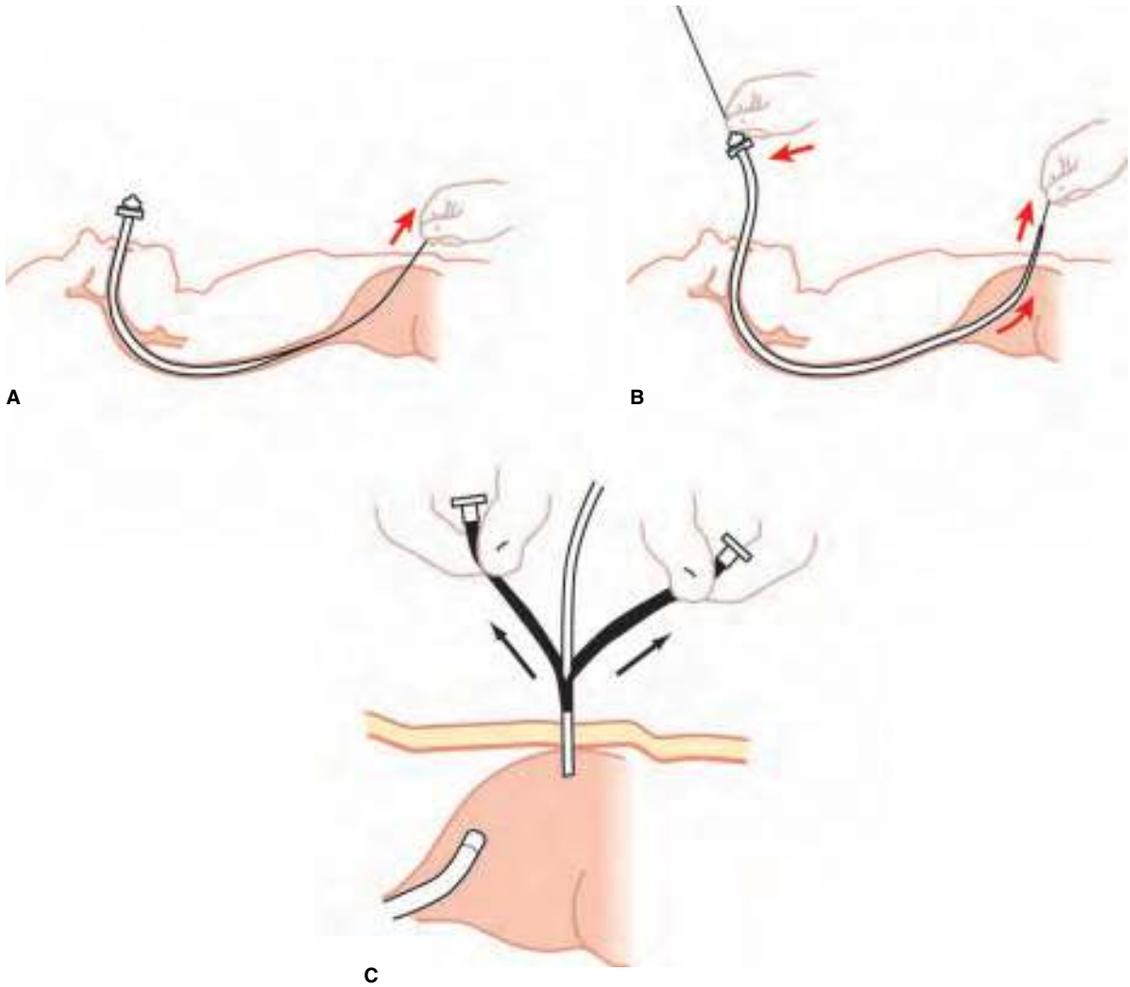

FIGURE 82-3. Endoscopic G-tube placement. ***A.*** The "Ponsky pull." A suture is attached to a modified G-tube and pulled in a retrograde direction through the anterior abdominal wall. ***B.*** The "Ponsky push." A guidewire serves as a trolley for the G-tube to be pushed over. ***C.*** The "Russell poke." The G-tube is inserted through a peel-away sheath.

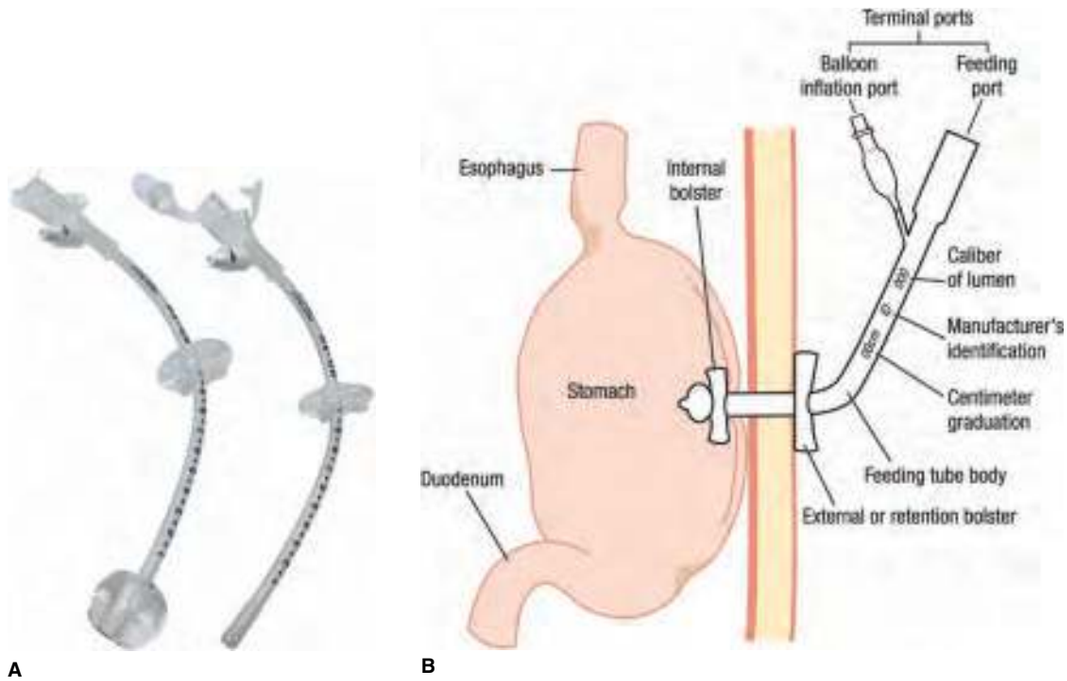

FIGURE 82-4. The basic gastrostomy or percutaneous endoscopic gastrostomy (PEG) tube. ***A.*** Photograph of a typical tube. ***B.*** Schematic illustration.

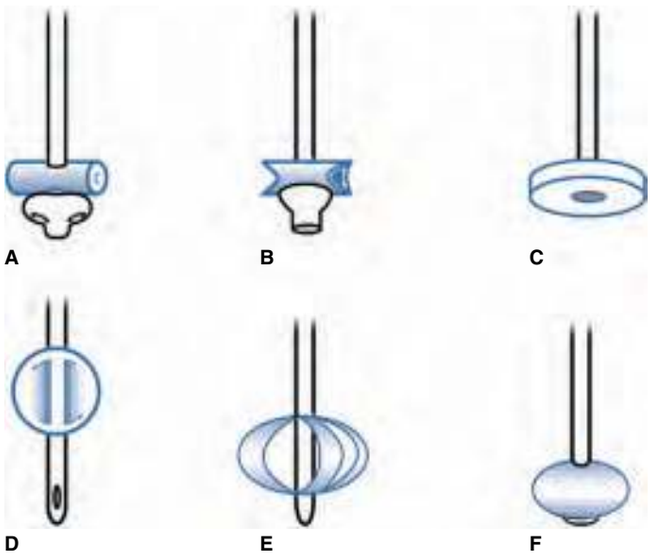

FIGURE 82-5. Examples of internal bolsters used in PEGs. ***A.*** Mushroom tip with de Pezzer flange. ***B.*** Crossbar. ***C.*** Round disk. ***D.*** Balloon tip. ***E.*** Malecot. ***F.*** Soft dome.

External bolsters are devices at the skin exit site that secure the G-tube to the abdominal wall and prevent its inward migration (**Figure 82-4**). Surgical gastrostomies frequently rely on only a silk suture. PEGs typically use a T-bar or retention disk. The external bolster has little impact on the function of the G-tube or the technique used to place it. The external bolster may cause the G-tube to kink, fracture, or clog if it is too tight. An external bolster can pull the internal bolster too tightly toward itself and cause stomach wall and/or abdominal wall necrosis. The G-tube may migrate inward with peristalsis and cause a bowel obstruction if the external bolster is too loose. Inappropriate care of the external bolster may lead to problems that contribute to the need for replacement, although it does not affect the ability to remove the G-tube. Identification and correction of problems with the external bolster can prevent unnecessary damage to the G-tube.

The external end of the G-tube contains the port(s) to access the lumen(s). Many tubes have a Y port, with one port for enteral feedings and a pop-off valve port to access a balloon (**Figure 82-4**). Some tubes have multiple ports to access each separate lumen that is designated for a specific purpose (e.g., medication administration or feeding). Others have a suction port for gastric suctioning and a port for distal feeding.

Some manufacturers now supply skin-level devices to improve patient comfort and acceptance. These are also referred to as low-profile systems or buttons. These tubes combine the external access port and the external bolster to provide a more cosmetically appealing look without a dangling tube.

INDICATIONS

There is no need for routine removal or replacement of G-tubes. The most common indication for G-tube replacement is accidental removal. G-tubes occasionally require replacement because they wear out, kink, or fracture. They should be replaced if the lumen becomes clogged with precipitate and cannot be unclogged. Most of these problems can be avoided with diligent care of the G-tube. Feedings should never be forced, and this is a common error that can weaken the tube. The feeding tube should be flushed with water after each use. Medications should never be mixed with enteral feeding solutions. Proper care of the G-tube should prevent premature loss. **Family members and health care providers should receive detailed instructions regarding G-tube management to avoid common problems.**

CONTRAINDICATIONS

A damaged, malfunctioning, or displaced G-tube should be replaced as soon as possible, with a few exceptions. **The existing tube should be left in place if the tract is immature. Premature removal of a tube in an immature tract, particularly if it is endoscopically placed, can lead to gastric spillage and peritonitis.** The exact time for a tract to mature depends on the procedure used and the patient's nutritional status. A conservative approach is to consider any tract less than 4 weeks old to be immature. The specialist who performed the original procedure should be consulted prior to any manipulation of the immature tract. G-tube removal begins with deflating the balloon, if one exists. Then, while providing traction on the tube, press a flat, gloved hand against the abdominal wall for countertraction. The tube should slide out with minimal resistance. **Abort the removal if significant resistant is felt as an internal ring or bolster that requires endoscopic removal may exist.**

The G-tube and tract should be left alone and a Surgeon consulted if a patient has peritonitis at the time of presentation. If the patient has acute abdominal pain after manipulation of a gastrostomy site, feedings should be withheld until an investigation determines the source of the problem. **Do not manipulate or change the G-tube if the patient has pain at the skin entry site or with movement of the tube.** This may signify an underlying abscess, infection, or intraabdominal pathology.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Commercial replacement G-tube kits
- Foley catheter of a similar caliber as the original tube
- 20 mL syringe with saline to fill the Foley balloon
- Water-soluble lubricant
- Toomey syringe or bulb syringe to aspirate gastric contents
- Some form of external bolster (see text below)
- Adapter to cap off Foley catheter or attach it to feeding assembly
- Gloves

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. This is important in case the track is lost, the G-tube cannot be placed into the stomach, or complications result. Teach the parent or caregiver to replace a displaced tube to keep the stoma open.

G-tube replacement can usually be accomplished with little preparation or anesthesia. Place the patient supine. Clean the skin surrounding the entry site of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. Anesthesia should not be necessary, as the gastrostomy site should not be tender. **Significant pain at the site may signify an infection, abscess, or intraabdominal pathology. The G-tube should not be manipulated if anything more than minor discomfort occurs.** Small children or anxious patients may benefit from mild sedation.

TECHNIQUES

The technique for replacing a G-tube will depend on the original procedure used to place the tube, the maturity of the tract, and the nature of the internal bolster. Obtain as much information as

possible about the age and nature of the existing tube. The following discussion reviews the procedure for replacing G-tubes in mature tracts, factors to consider prior to removing an existing but dysfunctional tube, and techniques to use in fashioning replacement systems.

REPLACING A DISLODGED G-TUBE

Every effort should be made to replace a dislodged G-tube as soon as possible. Gastrostomy sites begin to close as soon as the tube is removed. The tract will close within hours to days depending on its age, maturation, and size of the tube. An original tube in good condition can simply be reinserted to stent the tract until a permanent replacement is found. A commercially available replacement that is compatible with the original tube may be used. Foley catheters are simple to use, are widely available, and function well as temporary replacements.[14,20-22]

Select a tube of similar caliber to the patient's G-tube. A Foley catheter is most commonly used. A decision regarding the choice of external fixation should be made and the tube adapted appropriately prior to its insertion (see discussion below). Lubricate the replacement tube liberally. Gently insert the replacement tube through the tract. **Do not advance the tube against any significant resistance. Advancement against more than mild resistance can result in complications.** Aspirate gastric contents to confirm proper placement. Inflate the Foley catheter balloon with saline. Pull the Foley catheter snug to lodge the balloon immediately behind the anterior abdominal wall. **The entire process should be relatively painless and should not require dissection or force.** Inject the feeding port with water-soluble contrast and obtain a plain radiograph to confirm proper placement.[23]

A few types of feeding tubes merit special attention. Most gastrostomies have a short and direct route to the stomach. The Witzel uses a more circuitous tunnel and a smaller tube. The tract may be difficult to maneuver. Consult a Surgeon when problems replacing a surgically placed G-tube arise.

Not all feeding tubes terminate in the stomach. The feeding tube sometimes enters the stomach, travels through the pylorus and duodenum, and terminates in the proximal jejunum. The tract may be stented with a replacement G-tube if a patient is known to have a jejunostomy (i.e., percutaneous endoscopic jejunostomy or PEJ). More information should be obtained prior to resuming feedings.

Some catheters are not intended to be replaceable. Small needle jejunostomies may use 5 to 7 French catheters. There is little one can do to correct a catheter that has become disrupted or occluded. Consult with the patient's Primary Care Physician to discuss the options for management and follow-up.

PLACING AN EXTERNAL BOLSTER

After the successful insertion of a replacement tube, it needs to be secured in place.[24] The tube should be kept 90° relative to the skin and fixed in some way to prevent migration into the distal bowel. A short-term solution is to dress the site with 4×4 gauze squares. Build the gauze up along the exit site to create a pyramid-shaped dressing several inches high. A strip of urethane foam can be used to bolster the tube (**Figure 82-6*A***).[25] A 3 cm section of latex tubing can be wrapped about the base of the G-tube and secured with a plastic cable tie (**Figure 82-6*B***).[26] A modification to a Foley catheter uses a retention disk and a plastic ring from a nasogastric tube to secure the Foley catheter (**Figure 82-6*C***).[22]

Another option is to secure and bolster the tube with a suture (**Figure 82-7**). Some prefer to inject local anesthetic solution subcutaneously to minimize the discomfort of placing the single suture. Others believe placing the one suture is equal to the pain of injection

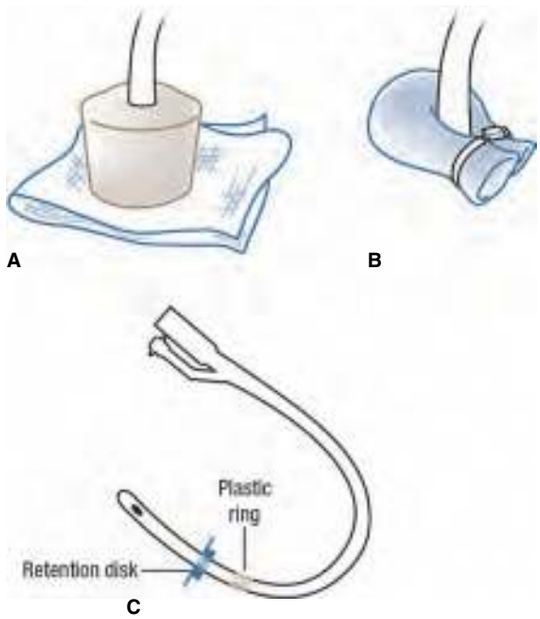

FIGURE 82-6. Types of external bolsters. ***A.*** Urethane foam dressing. ***B.*** Latex tubing wrapped about the base of the tube and secured with a plastic cable tie. ***C.*** A Foley catheter modified with a retention disk and plastic ring.

of the local anesthetic agent and do not use it. This decision should be decided on a case-by-case basis after a discussion with the patient and/or their representative. Place a suture using a large bite of tissue near the skin exit site. This serves as a retention suture. Leave an open loop of 1 to 2 cm between the skin and the knot to avoid unnecessary traction on the skin. Wrap the ends of the suture up the G-tube in a laddered fashion and tie them securely. This method allows some room for the G-tube to move while preventing inward migration as the patient changes position.

These techniques are adequate for short-term use. A more permanent form of external fixation is desirable. External bolsters have

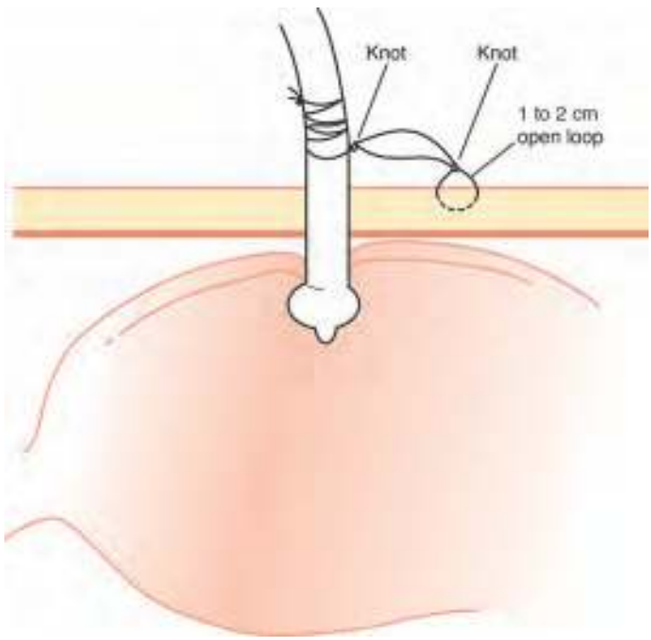

FIGURE 82-7. Simple external fixation using a silk suture laddered up the G-tube.

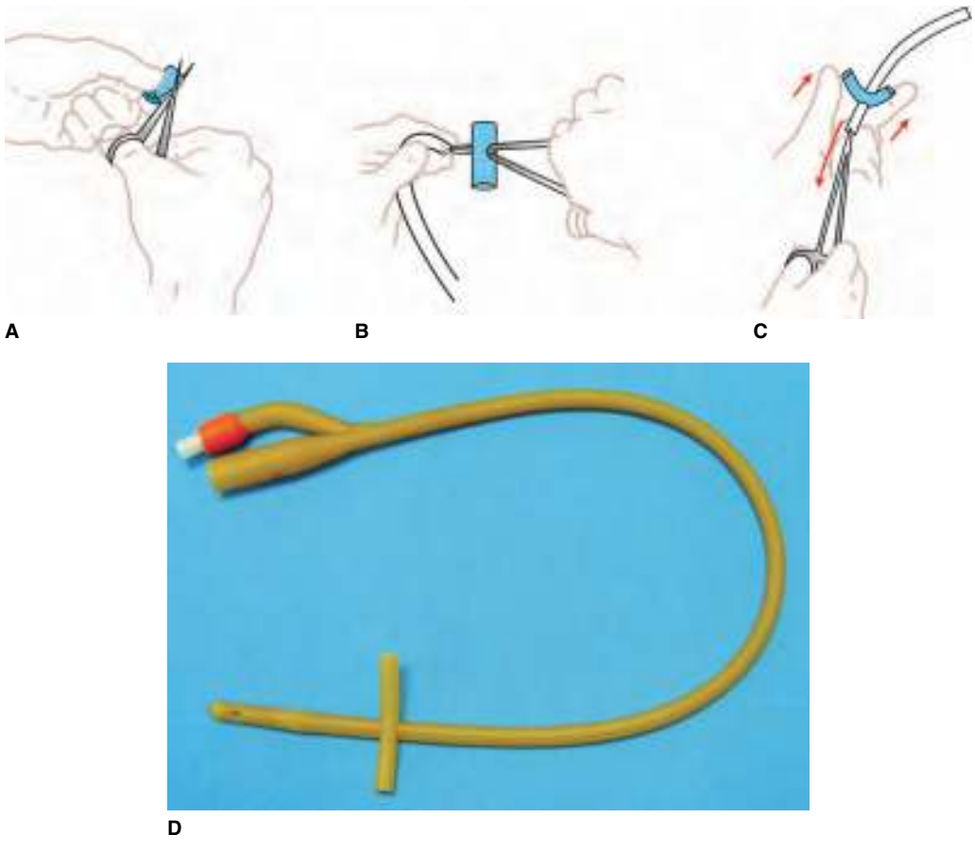

FIGURE 82-8. Fashioning an external bolster from a latex tube and a Foley catheter. ***A.*** The 3 cm piece of latex tube is folded and cut. ***B.*** A hemostat is inserted through the latex T-bar and grasps the distal end of the Foley catheter. ***C.*** The latex tube T-bar is advanced onto the catheter and pulled into position. ***D.*** The modified Foley catheter with a latex T-bar.

been described that can be fashioned from materials available in most health care facilities. A T-bar external bolster can be made by cutting a 3 cm piece of tubing from a latex or silicone Foley catheter or feeding tube (**Figure 82-8**).[20] Fold the piece of tubing in half. Make two diamond-shaped cuts placed on the sides of the fold and opposite each other (**Figure 82-8*A***). Insert a hemostat through the holes and gently grasp the replacement G-tube (**Figure 82-8*B***). **Do not grasp and damage the balloon.** Slide the latex T-bar along the G-tube (**Figures 82-8*C* and 82-8*D***). The latex T-bar must be snug enough to prevent migration but not so snug as to compress the G-tube lumen and disrupt function. Position the latex T-bar so that it is about 0.5 to 1.0 cm from the skin surface after placement of the G-tube.

There are several commercially available products designed specifically for replacement gastrostomies. They are convenient, but disadvantages do exist. They are expensive, may not be compatible with the original tube, and may not be on hand when needed.

Confirm and secure the clamp end of the replacement G-tube or fit it with an appropriate feeding adapter. Some tubes use the replaceable Lopez valve (**Figure 82-9**). Other G-tubes have the valve built in.

NONFUNCTIONING G-TUBES

A patient may present with a clogged, leaking, cracked, or fractured G-tube. **Numerous factors should be considered prior to removing any existing tube.** Is the tract mature? Is replacement necessary, or can other measures remedy the problem? What is the type of internal bolster, and can it be removed by external traction? **The most important factor is the age and maturity of the tract. Premature removal of the G-tube from a fresh tract may lead to peritoneal contamination with gastric contents and peritonitis. A new gastrostomy site should not be manipulated without consultation with the specialist who placed the original tube. A malfunctioning tube may have to remain in place to stent a fresh tract while alternative methods of nutritional support are provided to the patient.**

The underlying problem with a nonfunctioning G-tube should be investigated prior to its removal and replacement. A fractured tube or a tube with a ruptured balloon will need replacement. A kinked tube may only need revision of the external bolster. A clogged tube should first be irrigated with warm tap water, saline, or a carbonated beverage to open the lumen.[27,28] **Do not force the irrigation fluid into the G-tube. It may rupture and injure the patient.** A variety of other options may be tried to open a clogged G-tube. These include the instillation of enzymes to break up or dislodge the clog with an endoscopic snare, biopsy forceps, or Fogarty

FIGURE 82-9. The Lopez valve.

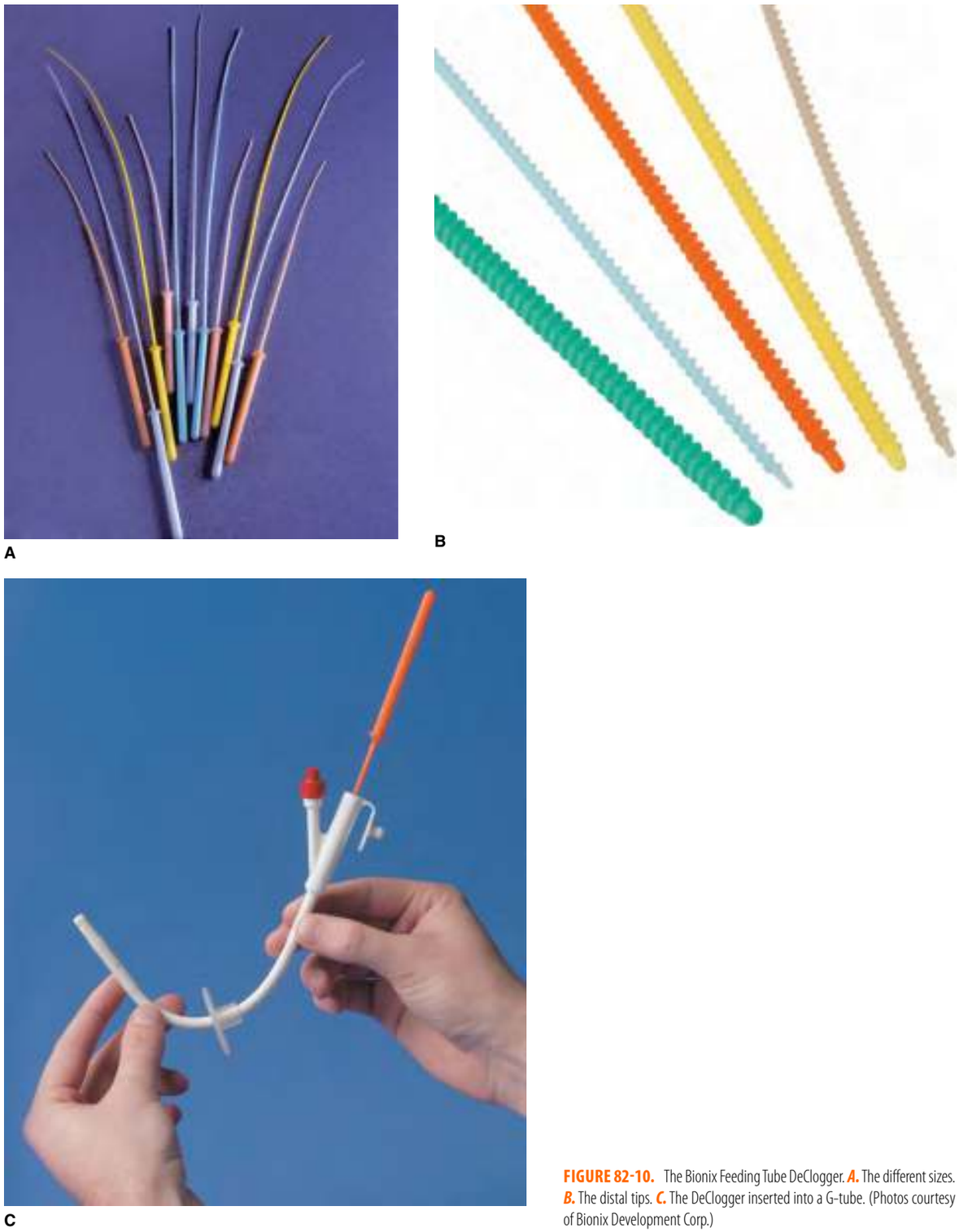

FIGURE 82-10. The Bionix Feeding Tube DeClogger. ***A.*** The different sizes. ***B.*** The distal tips. ***C.*** The DeClogger inserted into a G-tube. (Photos courtesy of Bionix Development Corp.)

catheter.[27-30] The latter methods are usually performed by a consultant and not the Emergency Physician.

A simple, quick, inexpensive, and easy to use device has been developed to declog a G-tube (Feeding Tube DeClogger, Bionix Development Corp., Toledo, OH). These are flexible plastic wands with a basic screw thread (**Figure 82-10**). They come in lengths for gastrostomy and jejunostomy tubes and in various sizes (12 to 24 French). Insert the DeClogger into the feeding port (**Figure 82-11**). Slowly advance it while rotating clockwise. Advance the DeClogger until it is fully inserted. Slowly remove the DeClogger from the feeding port. Aspirate and insufflate air to confirm the G-tube patency. This device may prevent the time and expense of replacing a clogged feeding tube. The patient and/or their caretaker can be taught how to use this device at home, which may prevent a trip to an office or Emergency Department.

The decision to replace a G-tube requires the Emergency Physician to obtain information regarding the type of tube in place.

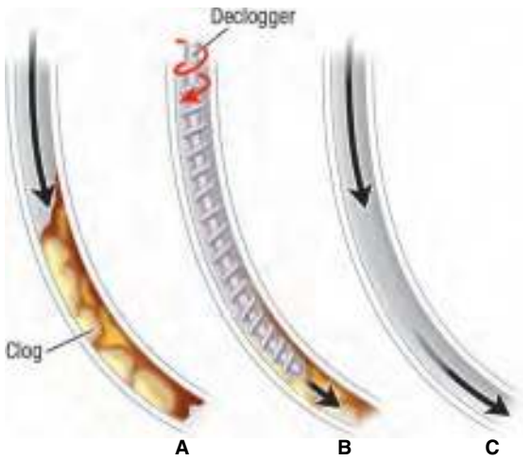

FIGURE 82-11. Using the Bionix Feeding Tube DeClogger. ***A.*** The clogged G-tube. ***B.*** The DeClogger is slowly advanced through the clog while being rotated clockwise. ***C.*** The clog has been removed and free flow reestablished.

Many are removable by gentle external traction. **Never apply more than gentle traction when removing a G-tube.** Simply deflating the internal bolster balloon will allow the G-tube to be removed. Other bolsters such as soft domes and T-bars may deform easily with gentle constant traction. **If gentle traction is not sufficient to remove the G-tube, either the internal bolster is not intended to be removed externally or it may have become embedded within the gastric wall.** Consult the physician who placed the tube if difficulty occurs when trying to remove a G-tube. The most likely problems and alternate solutions are discussed below.

THE BALLOON DOES NOT DEFLATE

The balloon may not deflate if the balloon inflation port is clogged or damaged. There are four options to remedy this situation. Simply cutting the G-tube may allow the balloon to deflate on its own. Cut the G-tube close to the ports. Maintain a firm hold, manually or with a hemostat, so that the cut tube does not migrate inside the patient and require endoscopic removal. The G-tube can be pulled taut to the skin and a needle advanced into the tract to puncture the balloon.[31-33] A guidewire may be advanced through the balloon port to puncture and deflate the balloon.[34] The balloon may have to be ruptured internally with an endoscopic snare.

THE G-TUBE DOES NOT WITHDRAW WITH EASE

Simply cutting the feeding tube at the skin level and pushing the remaining tube through the tract and into the stomach is not universally recommended. This allows the internal components to pass through the patient's gastrointestinal tract. Retained G-tube components have been known to cause bowel obstructions and perforations.[35] Most G-tubes pass without incident.[35] This procedure works for many types of G-tubes. It is not clear that this is a safe option for balloon bolsters that have not been deflated. **This option should be used only if a Primary Care Provider agrees with that choice and is available to follow the patient until the contents have passed.** Radiopaque components can be followed by plain radiographs at 24 to 48 hour intervals. The gastrostomy "hardware" may have to be retrieved endoscopically or surgically if it fails to pass within 2 to 3 weeks or the patient experiences obstructive symptoms. This option is not recommended for small children under the age of 6 years or weighing less than 20 kg.[36] These patients have a greater risk for complications.[36]

REMOVAL OF THE G-TUBE FROM A FRESH OR IMMATURE TRACT

The disruption of a fresh tract raises the possibility of gastric spillage and peritonitis. All enteral feedings should be discontinued and the patient observed for the development of peritonitis. Replacement options have been described. The tract may be allowed to close spontaneously and a replacement PEG placed in 7 to 10 days if the patient remains well.[37] Endoscopy can attempt to reintroduce a replacement tube under direct visualization through the original tract.[38] A Surgeon should be consulted and laparotomy considered if peritonitis develops. The Surgeon may choose to place a surgical gastrostomy. **Attempts to replace a removed tube from a fresh or immature tract should not be made in the Emergency Department.**

THE TRACT HAS CLOSED

A gastrostomy tract that is not stented will begin to close within hours.[39] A lost tract may require a repeat procedure. This problem requires consultation with an Endoscopist. Dilation of a closing gastrostomy site has been described using filiform catheters and followers or urethral sounds adapted from Urology.[40] **This procedure should be performed with caution as it can result in numerous complications.** Interventional Radiologists can often replace dislodged tubes using special techniques.

EARLY BALLOON RUPTURE OF AN INTACT G-TUBE IN AN IMMATURE TRACT

The tract is stented but there is the risk of gastric leakage around a deflated balloon. Consider replacement using a guidewire to exchange the G-tube.[41] This technique is performed using endoscopic guidance to snare the guidewire and withdraw the original G-tube.

ASSESSMENT

The replacement G-tube should be placed to allow gravity drainage, or the stomach contents should be aspirated. Use of the G-tube can be resumed if there is free flow of gastric contents. Its position should be confirmed radiographically if there was any difficulty with placement of the G-tube or if the return is equivocal. A small amount of water-soluble radiopaque contrast should be administered through the G-tube and a plain radiograph obtained. Normal G-tube placement will show intraluminal contrast. **Any extravasation of contrast is abnormal and requires enteral feedings to be withheld and a Surgeon to be consulted.** The patient will require hospitalization for parenteral antibiotics, observation, and bowel rest until the tract heals. Some physicians elect to evaluate all replaced G-tubes radiographically prior to their use. Doing so is theoretically harmless to the patient and causes no complications. This process cannot be routinely recommended as it is time-consuming and expensive.[42,43]

AFTERCARE

Routine maintenance can resume after successful replacement of the G-tube. Any factors that contributed to the malfunction should be addressed to prevent a recurrence. **The patient and/or their caregivers should be taught the proper care and maintenance of a G-tube.[27,44]** The patient should follow up with their primary physician in 24 to 48 hours for evaluation, removal of the temporary tube, and placement of a G-tube. Instruct the patient to immediately return to the Emergency Department if they develop a fever, abdominal pain, nausea, or vomiting.

COMPLICATIONS

A variety of complications may accompany the initial insertion of a G-tube (**Table 82-1**).[8,12,45,46] Replacement at a mature site should be relatively free of problems. Excessive force during replacement can disrupt the tract. A misdirected tube may end in a blind pouch or the peritoneal cavity if the stomach separates from the anterior abdominal wall. Installation of enteral feedings intraperitoneally will cause a chemical peritonitis. This should be suspected if there is poor return from the replaced G-tube, difficulty installing feedings, or the patient develops pain or fever after the procedure. Peritonitis is preventable if proper positioning is confirmed prior to using the replacement tube.[47]

A replacement G-tube must be sufficiently secured externally so that the effect of peristalsis does not carry it distally.[24] This is particularly true of balloon-tipped tubes. A G-tube that migrates past the pylorus can cause bowel obstructions and perforations.[8,12] This can be avoided by carefully securing the replacement G-tube with an external device or suturing it securely to the skin.

Always advance the Foley catheter into the stomach before carefully and slowly inflating the balloon. Inflation of the balloon within the gastrocutaneous tract can result in hemorrhage, pain, and rupture of the tract. Over-insertion of the catheter can cause the balloon to enter the esophagus, duodenum, or gastroesophageal junction. These structures can rupture when the balloon is inflated. These complications can be avoided by inserting the Foley catheter 8 to 10 cm, slowly inflating the balloon, and not inflating the balloon to its maximal volume.

A mature gastrostomy tract begins to close as soon as the tube is removed. The tract narrows without the presence of the G-tube to keep it patent. Replace the G-tube as soon as possible. **Never force a tube through the tract.** This can result in bleeding, gastric perforation, intraperitoneal penetration, pain, and the formation of a false passage. **Pass a smaller-size tube if necessary to reestablish the tract and maintain its patency.**

An indwelling or replacement G-tube may result in a gastric outlet obstruction. The patient usually presents with distention of the stomach and vomiting. Immediately and gently pull back on the G-tube until it is snug against the abdominal wall. Secure the G-tube with an external bolster. Observe the patient for resolution of their symptoms and any complications.

The skin exit site may become edematous, erythematous, and tender. A simple cellulitis should be managed with oral antibiotics and local wound care. Failure to resolve with antibiotics alone may suggest the presence of an abscess, and further imaging or drainage may be required. A dermatitis can result from leakage of gastric contents around the G-tube. Ensure that the internal bolster is secured against the anterior abdominal wall. If the leakage persists, replace the G-tube with a larger one that occludes the tract. Local wound care is all that is necessary once the problem with the G-tube is corrected. Hypersensitivity to the adhesive, cleansing solutions, or the G-tube itself may occasionally develop. The use of different materials and topical corticosteroids will correct this problem. A yeast infection (e.g., *Candida albicans*) appears erythematous and moist with satellite lesions. Topical antifungal creams and local wound care will alleviate the yeast infection. Granulation tissue around the stoma can be eliminated by coagulation with silver nitrate sticks. The patient should follow up with their primary physician in 24 to 48 hours for a reevaluation of all these clinical entities.

TABLE 82-1 Troubleshooting Enterostomy Tubes

Problem	Possible cause(s)	Action(s)
Vomiting	Balloon obstruction	Check tube length
	Balloon malposition	Pull back tube
		Check placement KUB without contrast
Diarrhea	Fistula	Contrast study for fistula evaluation
Tube comes out in ≤ 4 weeks	< 2 weeks	Call Interventional Radiology
		Do not replace
Tube comes out in ≥ 4 weeks	> 4–6 weeks	Place urinary catheter in stoma
		Confirm placement with contrast study
Tube clogged	Review medication list for possible causes: ciprofloxacin suspension, lactulose, Kayexalate, tube feeds, etc.	Flush with warm water
		Flush with carbonated beverage
		Consider pancrelipase
Tube leaking	Check connection joints	Seal with tape
	Look for holes in tube	Replace tube
Pain or resistance with feedings	Buried bumper syndrome	Contrast study
	Peristomal leak leading to erosion of internal bolster	Tube replacement
	Cellulitis	Start antibiotics
Erythema at tube site	Increased mobility	Secure tube
	Skin sensitivity to tape	Change tape
	Leakage of gastric acid	Add acid-blocking agent
	Granulation tissue	Cauterize with silver nitrate
	Cellulitis	Topical steroids
		Start antibiotics
Abdominal pain and fever	Peritonitis	Contrast imaging
		IV ABx, IVF
		Cessation of TF
		Surgical consult
		Nasogastric suction
Respiratory distress	Aspiration	Specialty consult
	Slipped Nissen	Fundoplication
	*Pediatric patient who has not had antireflux surgery	Gastrojejunostomy

ABx, antibiotics; IV, intravenous; IVF, intravenous fluids; KUB, kidneys, ureter, bladder; TF, tube feedings.
*XXXX.

PEDIATRIC CONSIDERATIONS

Pediatric patients face many of the same complications as adult patients with G-tubes.[47-50] There are some special considerations with G-tube placement and tube sizing that require some caution in pediatric patients. There are important questions to ask the caregiver when a feeding tube is dislodged or not functioning properly or there is concern about the site (e.g., bleeding, leaking, redness). These include the timing of initial placement of the tube, size of the tube, type of tube (e.g., button, PEG, mushroom tip, GJ), and how long it has been dislodged (if applicable).[46] The integrity of the tract between stoma and stomach is usually well formed by 4 to 6 weeks postoperatively. Note the length of the tube at the level of the skin. This might be an indicator of inward or outward movement of the tube. Note the balloon-filling instructions on the tube itself. The G-tube should note how many milliliters would fill the balloon. A small child may require 3 mL of air, while a larger child may require 5 mL of air to fill the G-tube balloon.[46,49,51]

Dislodgement of G-tubes is the leading reason for presentation to the Emergency Department. It accounts for approximately 60% of all Emergency Department visits related to G-tubes. Clogging of the feeding tube can occur. This is much more common when feeding via GJ-tubes versus G-tubes. The smaller caliber and extended length of GJ-tubes can cause increased obstruction of the lumen. GJ-tubes have a proximal port entering the stomach and a more distal port that feeds directly into the jejunum. Medications and formulas can cause sludging within the lumen of G-tubes and GJ-tubes. The tube should be flushed with saline after these are administered. Some children have GJ-tubes placed as an alternative for fundoplication

to prevent aspiration pneumonia. A child with a G-tube who has not had antireflux surgery (e.g., Nissen fundoplication) is at risk for aspiration pneumonia. Aspiration should be considered as a cause of respiratory distress.[33]

Migration of a G-tube can occur and lead to intestinal obstruction. The average length of the traditional G-tube or GJ-tube that protrudes from the stoma at the skin level is between 4 and 6 cm. This may be shorter for very small children and infants. Check the centimeter markings on the outside of the tube to provide evidence that a tube has migrated inward. Pulling back on a G-tube until the balloon is against the abdominal wall can relieve obstruction due to migration. G-tubes with a length of external tubing are initially placed then switched to a button-type tube that sits flatter to the skin. Button-type tubes have a decreased risk of dislodgement from pulling or migration distally into the gastrointestinal tract than traditional G-tubes.[18,19,52-55]

G-TUBE REPLACEMENT

Replace the tube with one the same size as the one that was dislodged. Have a smaller one available in case the stoma has closed. Sometimes the original-sized tube does not fit into a narrowed stoma. Start with a smaller-sized Foley or replacement tube and dilate the tract using progressively larger tubes or a set of Hegar dilators until the original tube size can be used. Replacement tubes can be a simple Foley catheter, G-tube, or button G-tube. Do not force the replacement tubes to avoid creating a false tract.[56] Clinical confirmation of correct placement can be checked by suctioning gastric contents, checking the pH with litmus paper, or dye studies with a small amount of radiocontrast material (e.g., 5 to 10 mL) followed with plain films. The tube is appropriately positioned on the radiograph if the outline of the stomach, or jejunum if there is a jejunal tube, is clearly seen.[56] The use of radiologic dye studies to confirm placement is variable among institutions, and this routine practice may not be necessary if one can clinically prove the tube is properly placed.

GASTROJEJUNAL TUBE REPLACEMENT

The patient may not know what type of tube they have. G-tubes typically have two ports, and GJ-tubes have three ports. Medications requiring an acidic environment for ideal absorption are usually given via the gastric port, whereas continuous drip feeds are given via the jejunal port. Bolus feeding into the jejunum is not feasible. The osmotic load leads to diarrhea. Parents or caregivers will be able to tell you whether they drip or bolus feed.

Contact an Interventional Radiologist to replace the tube under fluoroscopy or transfer the patient to an appropriate center for tube placement for GJ-tube dislodgment. Place a small catheter in the stoma to temporarily maintain patency until a more definitive feeding tube can be placed.[52]

CORRECTING LEAKAGE AT THE STOMA SITE

It is not recommended when there is leakage at the stoma site to replace the tube with a larger one and expand the stoma.[6] The correct probe position and balloon inflation should be verified by pulling the tube until the balloon is snug against the internal stomach wall.[57,58] A new tube can then be positioned through the same site when the leakage persists for several days. The G-tube can be removed in consultation with the specialist who placed the tube and allow partial stoma closure.[42]

Irritation from gastric content leakage can sometimes be mitigated by ensuring that the tube is directly against the anterior stomach wall. This can be done by first ensuring that the balloon is inflated, gently pulling the G-tube portion outward until resistance is met, and then pushing the external retention disc down toward the skin. This helps to seal any defects in the space between the stoma and the part of the G-tube within the stomach lumen to prevent further leakage of stomach contents. Place a gauze pad between the disc and the abdominal wall skin. The application of topical sucralfate, magnesium hydroxide, or menthol/zinc oxide may be used to relieve the irritation of acidic stomach contents on the skin.[59]

SUMMARY

G-tubes result in a simple fibrous tract connecting a feeding tube to the stomach. A mature tract can be safely and easily manipulated. Problems with an immature tract should prompt consultation. Percutaneous radiologically placed tubes are ultimately replaced by Interventional Radiology given its indication of placement and anatomical location. Techniques for the basic maintenance and repair of G-tubes are straightforward. Patients with dislodged G-tubes that have been replaced with Foley catheters, G-tubes, or buttons can be discharged home to follow-up with the service that placed the tube if the patient does not require admission. Familiarity with the procedures and equipment used to establish gastrostomies will help the Emergency Physician solve common G-tube problems and intervene in an appropriate and timely manner.

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83 Paracentesis

Alex Koo and Ryan Walsh

INTRODUCTION

Ascites is an abnormal accumulation of fluid in the abdominal cavity (**Figure 83-1**). It has important implications diagnostically, prognostically, and therapeutically. Cirrhosis of the liver is usually related to alcoholism and accounts for 75% of cases of ascites.[1,2] Malignancy accounts for an additional 10% to 12% and cardiac failure for another 5%. The remaining cases have a variety of etiologies. The physical examination is not very reliable when it comes to detecting ascites, making paracentesis and ultrasound (US) important clinical tools.[3] US-guided paracentesis has two key benefits. It facilitates

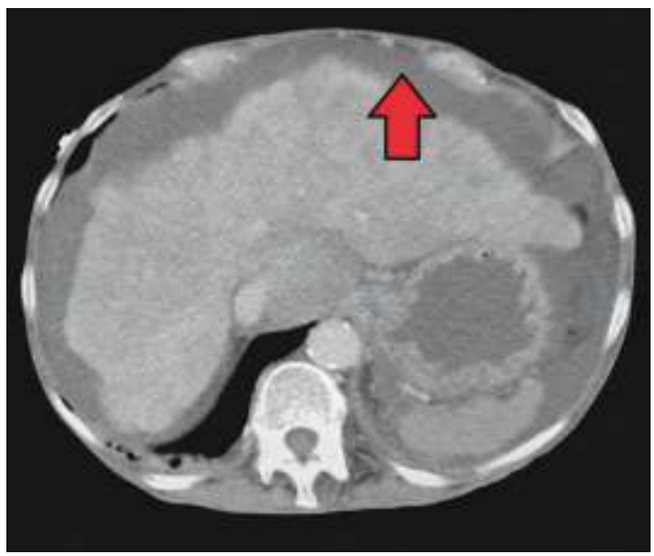


FIGURE 83-1. Computed tomography image of the abdomen with ascites (*arrow*). (Used from www.commons.wikimedia.org.)

performance of the procedure and identifies patients in whom the procedure is not warranted or could potentially be harmful.[4]

Peritoneal aspiration of ascitic fluid or paracentesis was first described in the early twentieth century.[5] Paracentesis fell out of favor in the 1950s with the introduction of diuretics and the fear of procedure-related complications and was replaced by medical management. Large-bore needles were being used at that time, and complication rates were significant. Clinical studies published in the late 1980s demonstrated that performing a paracentesis was a safe procedure.[6,7] A paracentesis is now common in Emergency Departments.

Paracentesis is an important diagnostic tool for patients with new-onset ascites to determine its etiology and in patients with long-standing ascites to detect the presence of an infection. Spontaneous bacterial peritonitis (SBP) can be a very subtle disease. Infection occurs in as many as 27% of cirrhosis patients admitted for evaluation of symptoms associated with their ascites.[8] It is well known that some patients with SBP are asymptomatic.[9] **This makes peritoneal fluid aspiration, analysis, and cultures imperative.**[10] Large volumes of ascitic fluid can be removed therapeutically to improve a patient's respiratory status and comfort level from the pressure of tense ascites. This often occurs in patients with end-stage liver disease and some cases of malignancy. Malignant ascites may occur with carcinomas (e.g., breast, colon, ovary, pancreas, stomach, and testes), lymphomas, and sarcomas.

ANATOMY AND PATHOPHYSIOLOGY

The abdominal cavity is partially lined by a serous membrane known as the peritoneum. The abdominal cavity is protected from the environment by the abdominal wall musculature, fat, and skin. The peritoneum serves as protection for its encased organs, secretes nutrients, and secretes proteins. Intraperitoneal organs include the stomach, first portion of the duodenum, jejunum, ileum, appendix, transverse colon, sigmoid colon, part of the rectum, liver, spleen, and the tail of the pancreas. The right and left rectus muscles are nourished by the epigastric vessels. The epigastric vessels meet in the midline at the avascular linea alba. The umbilicus is located along the lower portion of the linea alba. The layers of the anterior abdominal wall structures vary above and below the level of the anterior superior iliac spine (**Figure 83-2**).

The anatomy of the abdominal cavity is consistent among individuals. The liver sits in the upper right quadrant. Studies demonstrate that estimation of size through physical examination is inaccurate, even with expert evaluation.[11-13] The consistency of the liver may confer information on cirrhosis or masses.[14] The spleen is normally contained in the upper left quadrant. It can extend into the left lower quadrant of the abdomen when enlarged. The physical examination is not sensitive but specific for splenomegaly when the spleen is palpable. The diagnosis of hepatomegaly or splenomegaly can be aided with the use of US.[15] The intestines occupy most of the abdominal cavity and are not rigidly adherent, allowing them to move about in the abdominal cavity. The bladder sits inferior to the abdominal cavity. The bladder can enter the abdomen when it is distended with urine.

There is a potential space intraperitoneally that is considered abnormal when there is fluid present. Ascitic fluid can be found anywhere in the peritoneal cavity. The location of the ascitic fluid depends primarily on the amount of fluid present and the patient's position. Fluid follows the law of gravity and collects in the most dependent areas. Small amounts of fluid generally accumulate first in the hepatorenal recess (i.e., Morrison's pouch) while the patient is lying supine. Large amounts of fluid can be found bathing the intestines (**Figure 83-3**). There can be distinct pockets of fluid in areas

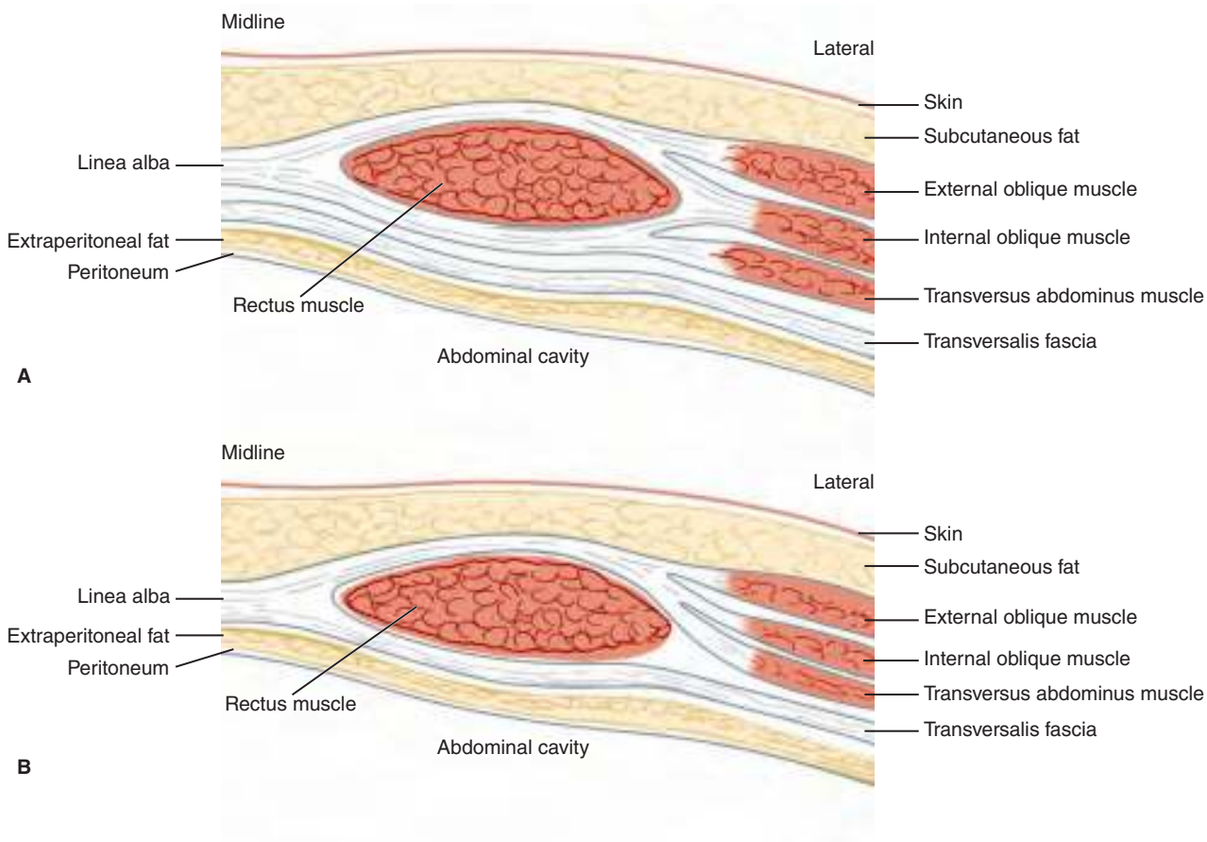


FIGURE 83-2. The layers of the anterior abdominal wall vary above (*A*) and below (*B*) the level of the anterior superior iliac spine.

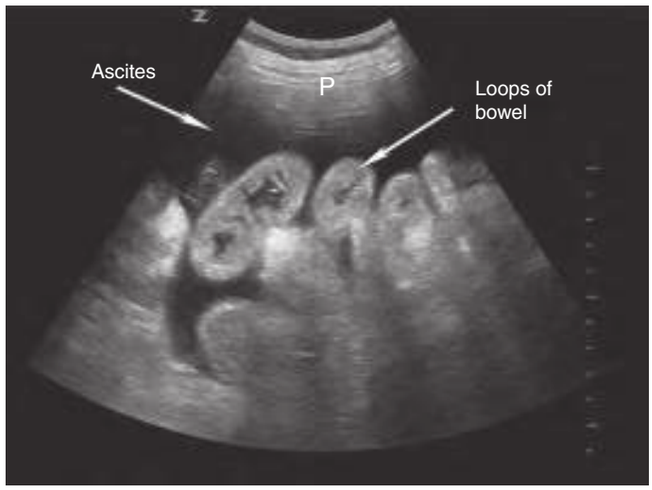


FIGURE 83-3. US of the abdomen demonstrating loops of intestine floating in ascitic fluid. (Used with permission from reference 23.)

of intestine adhesions or scarring. These localized areas of fluid are typically found in patients who have infections, previous surgery, or trauma.

Paracentesis is easily performed blindly. **US can provide several benefits.** US is diagnostically useful to ensure there is enough fluid to perform a paracentesis.[11] It will identify and help avoid solid organs, masses, and vascular structures. US is sensitive for hepatomegaly and splenomegaly.[14,15] US can identify areas where the intestine is adherent to the abdominal wall from adhesions and puncture should not occur. US can measure the soft tissue thickness from the skin to the peritoneum, locate the thinnest entry point, and determine the needle length required to perform the procedure in obese patients. US can be used to position the patient so that the maximum amount of fluid collects at the sampling site.

INDICATIONS

A paracentesis can be performed for diagnostic or therapeutic purposes. A paracentesis or "abdominal tap" is warranted in a patient with new-onset ascites to establish the etiology of the fluid. A patient with a history of ascites may need the procedure if they have associated signs and symptoms suggestive of infection (e.g., abdominal pain, dyspnea, fever, encephalopathy, peripheral leukocytosis, or renal impairment).[16] A paracentesis can be performed therapeutically if medical management with diuretics has not been successful. A paracentesis may be therapeutic in patients with cardiorespiratory or gastrointestinal manifestations secondary to tense ascites. It is most commonly performed when an intraperitoneal infection is suspected. **Clinical guidelines recommend that patients with new-onset ascites admitted to the hospital undergo a paracentesis regardless of whether they have symptoms of SBP.**[16,17] Paracentesis has been used to aid in the diagnosis of intestine perforation, hemoperitoneum due to trauma, and ruptured ectopic pregnancy. More accurate diagnostic procedures should be used rather than a paracentesis for these conditions if they are available. Paracentesis is used to manage some patients with hepatorenal syndrome in conjunction with the consultant.[18]

CONTRAINDICATIONS

There are no absolute contraindications to performing a paracentesis. The relative contraindications include abdominal wall cellulitis, coagulopathy, current intestine obstruction, history of abdominal surgery, pregnancy, or thrombocytopenia. Pregnancy is listed because the gravid uterus may fill the space where the procedure is normally performed. **A paracentesis should be performed superior to the uterine fundus. Avoid sites of previous surgical incisions.** Adhesions may fix the intestine wall to the abdominal wall and increase the possibility of perforation.

Many patients who are subjected to a paracentesis have underlying liver disease and resultant coagulopathies. Historically, some have advocated patients with thrombocytopenia or an abnormal international normalized ratio (INR) receive platelet transfusions or factor replacement prior to performing a paracentesis. This practice is controversial, and there are no controlled data to support these contentions.[19,20]

One large retrospective study of 4729 paracenteses performed in patients with coagulopathies and/or thrombocytopenias found very low rates of severe hemorrhage (0.19%), defined by hemodynamic instability or a decrease in hemoglobin of > 1.5 gm/dL, and death (0.016%).[21] Bleeding was unrelated to the elevation in INR and the level of thrombocytopenia. Patients with prophylactic transfusion of platelets or plasma had similar outcomes to those without prophylactic transfusion.

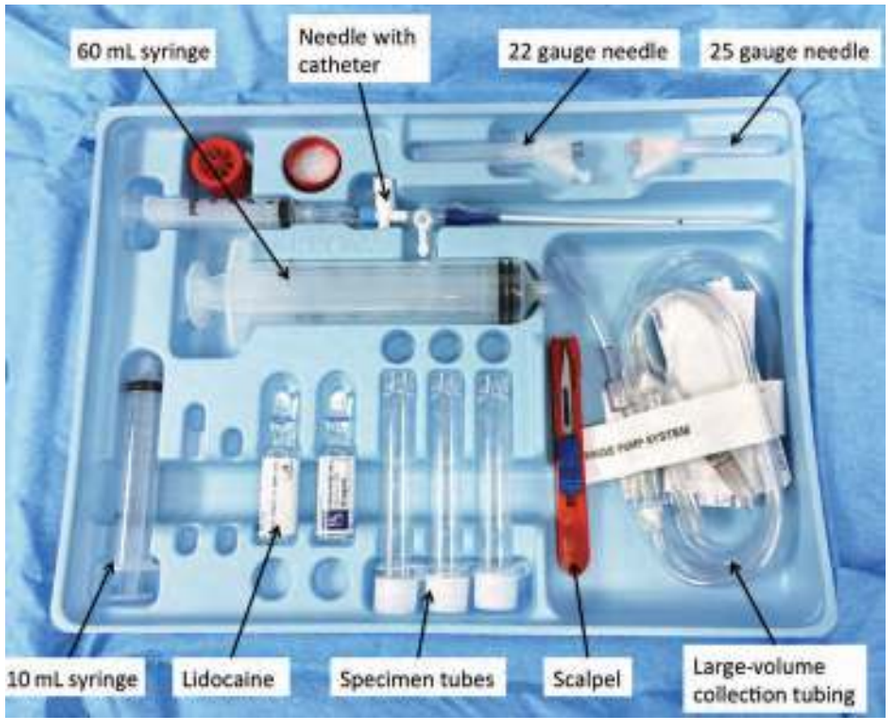
EQUIPMENT

- Protective eyewear
- Sterile gloves and gown
- Cap and mask
- Povidone iodine or chlorhexidine solution
- Sterile 4×4 gauze
- Sterile drape
- Local anesthetic solution with epinephrine
- 25 or 27 gauge needle to anesthetize the skin
- Large syringe(s) for fluid collection (20 to 60 mL)
- Intravenous tubing or blood collection tubing if vacuum bottles are being used
- Collection bottles (vacuum) or collection bag
- Adhesive dressing
- Three-way stopcock[22]
- Blood collection tubes for white blood cell count, electrolytes, albumin, pH, etc.
- Blood culture bottles (aerobic and anaerobic)
- Sterile specimen container for cytology (optional)
- US machine
- US transducer, 3.5 to 5 MHz curvilinear and 8 to 12 MHz linear
- Sterile US gel
- Sterile US transducer cover

CHOICE OF NEEDLE OPTIONS

- 18 to 22 gauge needle, 3½ inch needle or spinal needle
- Seldinger-type guidewire kit
- Catheter-through-the-needle
- Catheter-over-the-needle (i.e., angiocatheter), consider Caldwell needle

Commercially available paracentesis kits are available (**Figure 83-4**). These contain all the required equipment except the collection bottles. They include the sterile drapes and local anesthetic solution. These are convenient and relatively inexpensive.



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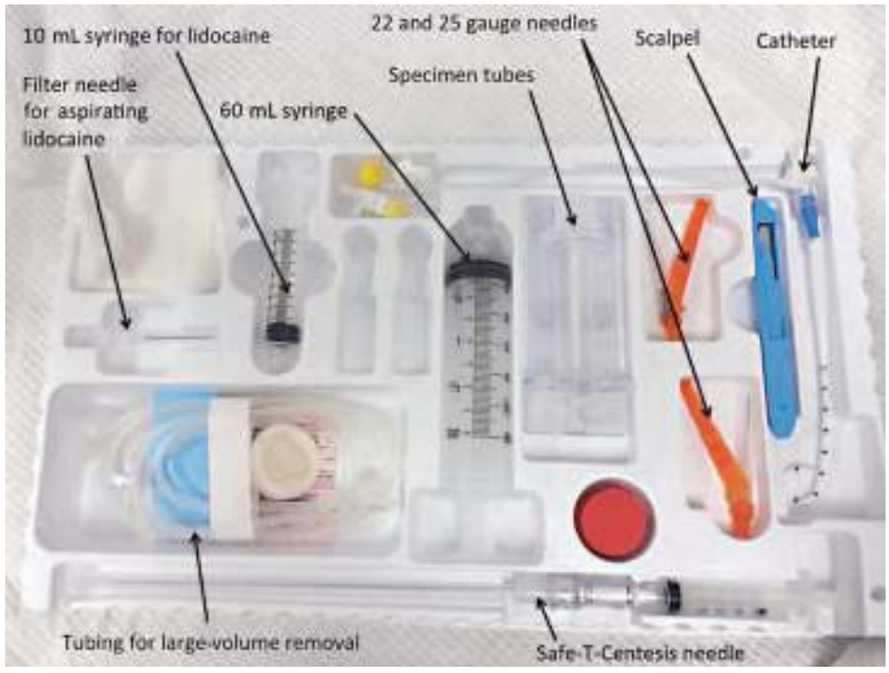

B

FIGURE 83-4. Commercially available paracentesis kits. ***A.*** The Arrow kit (Teleflex Inc., Morrisville, NC). ***B.*** The Safety-Centesis kit (Becton Dickinson, Franklin Lakes, NJ). (Photos courtesy of Jason Williams, MD, and www.proceduralist.org.)

A general-purpose curvilinear or phased-array US transducer provides the best combination of penetration and field of view into the abdomen. A linear transducer can be used for children, small adults, and thin patients. Use the 3.5 to 5 MHz curvilinear transducer to locate the ascites if it is small.[23] Use the 8 to 12 MHz high-frequency linear transducer for the procedure.[23]

Some patients have indwelling subcutaneous ports. These patients have ports for frequent access to the abdomen. This includes patients receiving antibiotics, chemotherapeutics, and frequent paracenteses. Refer to Chapter 66 for accessing these devices.

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative. Obtain an informed consent for the procedure. The patient's bladder should be empty. Use a US machine to check

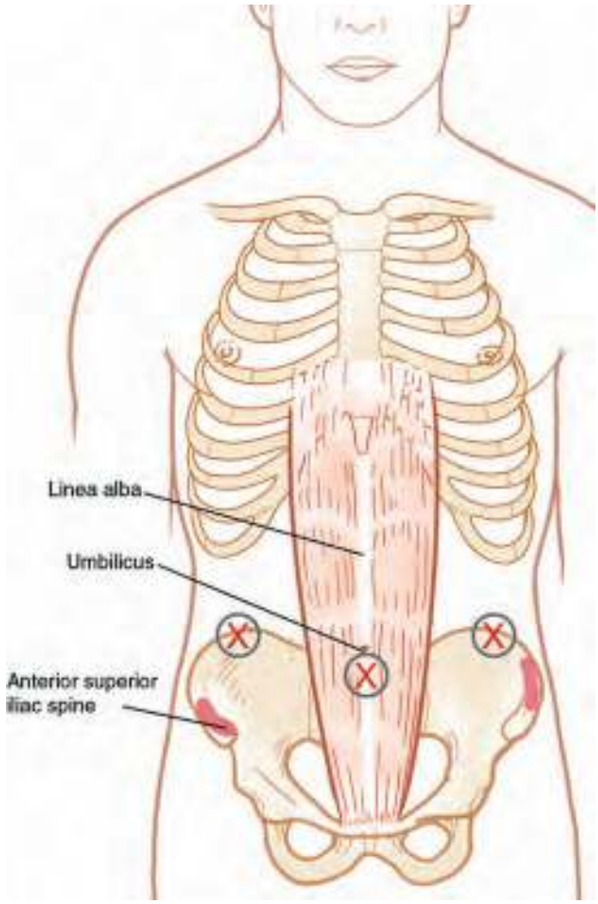

FIGURE 83-5. Needle insertion sites to perform a paracentesis (indicated by a red ⊗). The preferred site is in the midline and 2 cm below the umbilicus. Alternative sites are just medial and 4 to 5 cm above the anterior superior iliac spines.

for a full bladder or place a Foley catheter to decompress the bladder if the patient is unable to urinate voluntarily. Placement of a nasogastric tube can be considered, although it is not routine. It prevents an iatrogenic perforation if the stomach is dilated or if a concomitant intestine obstruction is present. It is recommended if the blind technique is performed to avoid a perforation.

There are two recommended areas of entry for the paracentesis needle (**Figure 83-5**). The first site is in the midline and 2 cm below the umbilicus through the avascular linea alba. Alternatively, the region 4 to 5 cm superior and just medial to the anterior superior iliac spine in one of the lower quadrants may be used. **This location should be lateral to the rectus abdominis muscle to avoid injury to the inferior epigastric artery, which runs vertically along the muscle sheath.** Some Emergency Physicians choose the right lower quadrant to avoid the sigmoid colon and spleen. Others choose the left lower quadrant to avoid the cecum and liver. Remember to exercise caution in the regions of caput medusa, prominent veins, veins, scarring, over an area of inflamed or infected skin to minimize complications. US-assisted paracentesis is recommended if a US machine is readily available. US can assist in identifying the ideal fluid pocket and hopefully avoid a "dry tap."[4]

Place the patient sitting 45° upright for a midline approach. This positioning can be used for the right or left lower quadrant approach if there is ample ascites. Lying in the right lateral decubitus position for a right lower quadrant approach or lying in the left lateral decubitus position for a left lower quadrant approach increases dependency of the ascites to a desirable quadrant while displacing the intestine superiorly. Another position one might consider is having the patient assume a hand-knee or "crawling" position. This position is awkward for the Emergency Physician performing the procedure. Remember that the fluid will pool in dependent areas and the intestine will float on top of it, barring any adhesions or masses.

Prepare the patient. Clean the skin around the chosen puncture site of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. Apply sterile drapes to delineate a sterile field. Don a cap, mask, sterile gloves, and sterile gown. **Perform the paracentesis using strict sterile technique.** Inject 2 to 5 mL of local anesthetic solution subcutaneously and along the needle insertion tract. Allow 3 to 5 minutes for the local anesthetic to take effect.

TECHNIQUES

Z-TRACK TECHNIQUE

A "Z-track" is used to decrease the possibility of an ascitic fluid leak, especially in patients with tense ascites (**Figure 83-6**). This is the preferred method for inserting the needle. This technique should be followed when using the other techniques described below. Apply traction on the skin 2 cm cephalad or caudad to the needle insertion site so that the skin is pulled taut when the needle enters the peritoneum (**Figures 83-6*A* and 83-6*B***). The skin will return to its normal position when the tension is released and seals off the pathway of the paracentesis needle.

Apply an 18 to 22 gauge, 3½ inch needle or spinal needle to a 60 mL syringe. Slowly insert and advance the needle perpendicular to the skin (**Figure 83-6*A***) or at 45° to the skin and aimed caudally (**Figure 83-6*B***). Apply negative pressure to the syringe as it is being advanced. A loss of resistance will be felt as the needle enters the peritoneal cavity. Stop advancing the needle when ascitic fluid enters the syringe. Continue to aspirate until the syringe is one-half to three-fourths filled with fluid. Depending on the size of syringe, consider using a three-way stopcock so syringes can be removed as they are filled.

The omentum, a loop of intestine, peritoneal fat, or other tissue may be occluding the needle tip if ascitic fluid suddenly stops flowing into the syringe. Release the plunger of the syringe. Reattempt to aspirate. Inject 1 to 2 mL of ascitic fluid back into the peritoneal cavity if fluid still will not flow and then reattempt to aspirate. Reposition the needle if ascitic fluid still does not flow into the syringe. **Never reposition the needle while the sharp tip is within the peritoneal cavity. The needle can lacerate a blood vessel, the intestine, or the omentum.** Withdraw the needle to the dermis, reposition it, and then readvance it into the peritoneal cavity.

The Caldwell needle appears to be superior to a conventional angiocatheter needle when it comes to problems with fluid return. The Caldwell needle's unique design with fenestrations on the side is believed to allow for continued flow despite occlusion of the needle tip.[24,25]

Note the color and clarity of the ascitic fluid. Aspirate 30 to 50 mL if the procedure is being performed for diagnostic purposes. Withdraw the needle after obtaining the ascitic fluid. Immediately place the fluid into the appropriate collection tubes and culture bottles.[26] Hold the needle or catheter securely and remove the syringe if the reason for the paracentesis is therapeutic and there is a large collection of fluid that must be drained. An assistant can place the sample into laboratory containers. Connect the needle or catheter to intravenous tubing. Connect the other end of the intravenous tubing to a suction bottle or bag to drain off the desired amount of ascitic fluid. Remove the needle or catheter once the procedure has been completed. Removal results in the formation of the Z-track so that ascitic fluid will not leak from the skin (**Figure 83-6*C***). Apply a

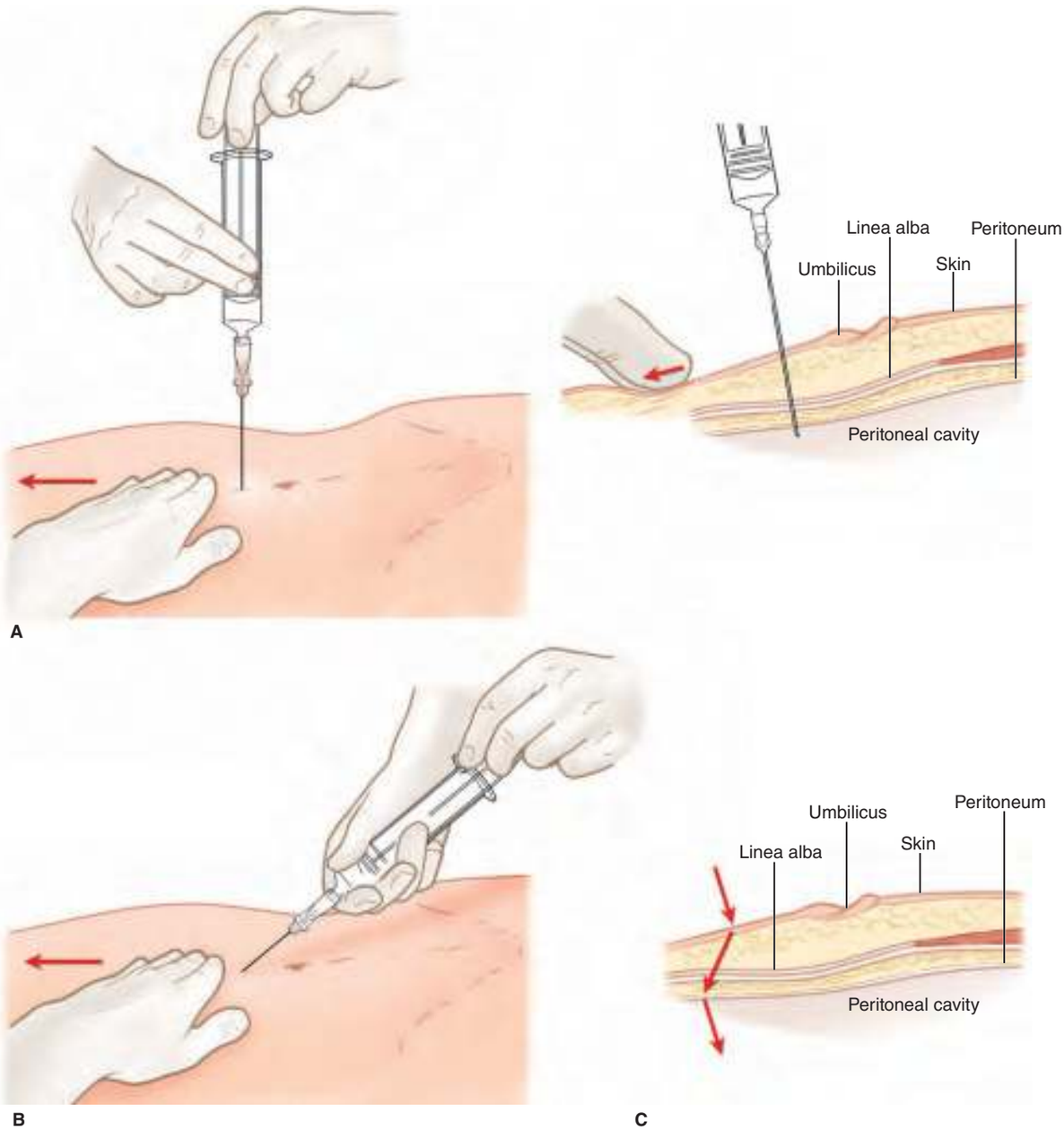

FIGURE 83-6. The Z-track. ***A.*** The needle is inserted perpendicular to the skin while the skin is pulled taut. ***B.*** Alternatively, the needle can be inserted at 45° to the skin and aimed caudally. ***C.*** The resultant Z-track (*arrows*).

bandage to the skin puncture site. Consider a compression dressing with an occlusive bandage if fluid is oozing from the puncture site.

SELDINGER TECHNIQUE

This technique allows for the placement of a catheter over a wire (**Figure 83-7**). The wire used must be longer than the catheter. The needle used to insert the wire can be short and a smaller gauge than the catheter. Materials needed for catheter insertion are commercially available in a prefabricated kit. The Seldinger technique is most commonly used for central venous catheter insertion and familiar to the Emergency Physician.

Choose the puncture site and prepare the patient for the procedure. Insert the thin-walled introducer needle in a Z-track manner while applying negative pressure to the syringe (**Figure 83-7*A***). The introducer needle has a tapered hub on the proximal end to guide the wire into the needle lumen. **Avoid using a standard hypodermic needle as it does not allow for the passage of the guidewire.** A flash of fluid in the needle hub signifies that the tip of the needle is within the peritoneal cavity (**Figure 83-7*A***). Advance the needle an additional 2 to 3 mm. Hold the needle in place securely and remove the syringe.

Occlude the needle hub with a sterile gloved finger. This will prevent air from entering and ascitic fluid from exiting. Insert the guidewire through the hub of the needle (**Figure 83-7*B***). Advance the guidewire to the desired depth and ensure it is at least several centimeters beyond the beveled end of the needle. **Always have at least one hand holding the guidewire to prevent it from slipping completely into the peritoneal cavity.** Hold the guidewire securely in place. Remove the needle over the guidewire (**Figure 83-7*C***).

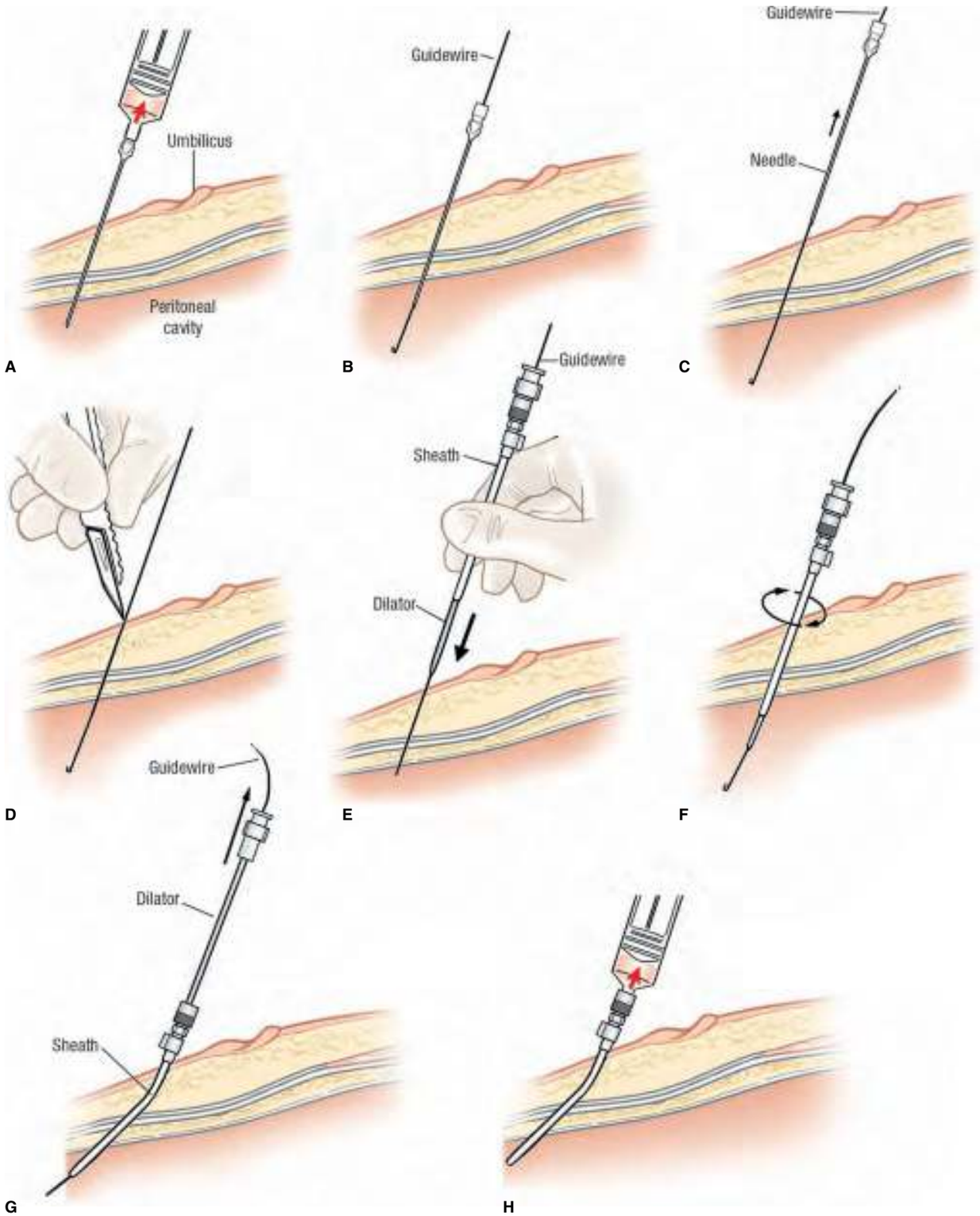

FIGURE 83-7. The Seldinger technique. ***A.*** The needle is advanced into the peritoneal cavity. ***B.*** The syringe is removed and a guidewire is inserted through the needle and into the peritoneal cavity. ***C.*** The needle is removed, leaving the guidewire in place. ***D.*** An incision is made where the guidewire enters the skin. ***E.*** The dilator and sheath are advanced as a unit over the guidewire. ***F.*** The dilator and sheath are advanced into the peritoneal cavity with a twisting motion. ***G.*** The guidewire and dilator are removed as a unit, leaving the sheath in place. ***H.*** A syringe is attached to the sheath. The aspiration of ascitic fluid confirms proper intraperitoneal placement of the sheath.

Make a small nick in the skin adjacent to the guidewire with the #11 scalpel blade included in the kit (**Figure 83-7*D***). **Direct the sharp edge of the scalpel blade away from the guidewire to avoid nicking the guidewire.**

Place the dilator through the sheath or catheter to form a unit. Advance the dilator and sheath unit over the guidewire (**Figure 83-7*E***). Continue to advance the dilator and sheath unit over the guidewire and into the peritoneal cavity (**Figure 83-7*F***). A twisting motion may aid in its advancement through the skin and into the peritoneal cavity (**Figure 83-7*F***). Advance the unit until the hub of the sheath is against the skin. Hold the hub of the sheath securely. Remove the guidewire and dilator as a unit (**Figure 83-7*G***). Attach a syringe to the hub of the sheath (**Figure 83-7*H***).

Aspirate fluid from the sheath to confirm intraperitoneal placement (**Figure 83-7*H***). Hold the hub of the sheath securely and remove the syringe. Pass the syringe to an assistant to place the sample into laboratory containers. Connect the hub of the sheath to intravenous tubing. Connect the other end of the intravenous tubing to a suction bottle or bag to drain the desired amount of fluid. An alternative is to attach a three-way stopcock to the distal end of the intravenous tubing.[22] A syringe may then be attached to the stopcock to withdraw laboratory samples or "pump out" the ascitic fluid into a nonsterile container. Remove the sheath once the procedure is completed. Apply a bandage to the skin puncture site. This technique seems complicated at first glance. It is easy to learn and can be performed in a few minutes by an experienced Emergency Physician.

CATHETER-THROUGH-THE-NEEDLE TECHNIQUE

This system is used for central venous access outside the United States. Select a catheter size that is appropriate for the patient and the site of entry. Packaged with each catheter are a needle and a needle guard. The needle will have an inner diameter that is slightly larger than the outer diameter of the catheter. The needle guard has a beveled channel in which the needle can reside. The needle guard hinges closed over the needle to hold it securely and prevent the needle from shearing the catheter.

Choose the puncture site and prepare the patient for the procedure. Place the needle on a tuberculin syringe. Insert the needle through the skin in a Z-track manner and into the peritoneal cavity while applying negative pressure to the syringe (**Figure 83-8*A***). A flash of fluid in the syringe confirms that the tip of the needle is within the peritoneal cavity. Advance the needle an additional 2 to 3 mm to ensure that the tip of the needle is completely within the peritoneal cavity. Securely grasp and hold the needle with the nondominant hand. Remove the syringe with the dominant hand. Immediately place the nondominant thumb over the needle hub to prevent air from entering and fluid from exiting.

Insert the catheter through the hub of the needle (**Figure 83-8*B***). Advance the catheter through the needle until the desired length of catheter is within the peritoneal cavity. **Remove the catheter and needle as a unit if the catheter will not advance. Never withdraw the catheter through the needle. The sharp bevel of the needle may cut the catheter as it is being withdrawn and result in a catheter embolism in the peritoneal cavity.**

Withdraw the needle over the catheter (**Figure 83-8*C***). **Do not allow the catheter to be withdrawn through the needle.** Continue to withdraw the needle until the tip is completely outside the skin. Apply the needle guard over the needle (**Figure 83-8*D***). Attach a syringe onto the hub of the catheter. Aspirate fluid from the catheter to confirm intraperitoneal placement. Hold the hub of the catheter securely and remove the syringe. Pass the syringe to an assistant to place the sample into laboratory containers. Connect the catheter to intravenous tubing. Connect the other end of the tubing to a suction bottle or bag to drain the desired amount of fluid. An alternative is to attach a three-way stopcock to the distal end of the tubing.[22] A syringe may then be attached to the stopcock to withdraw laboratory samples or "pump out" the ascitic fluid into a nonsterile container. Remove the catheter once the procedure is completed. Apply a bandage to the skin puncture site.

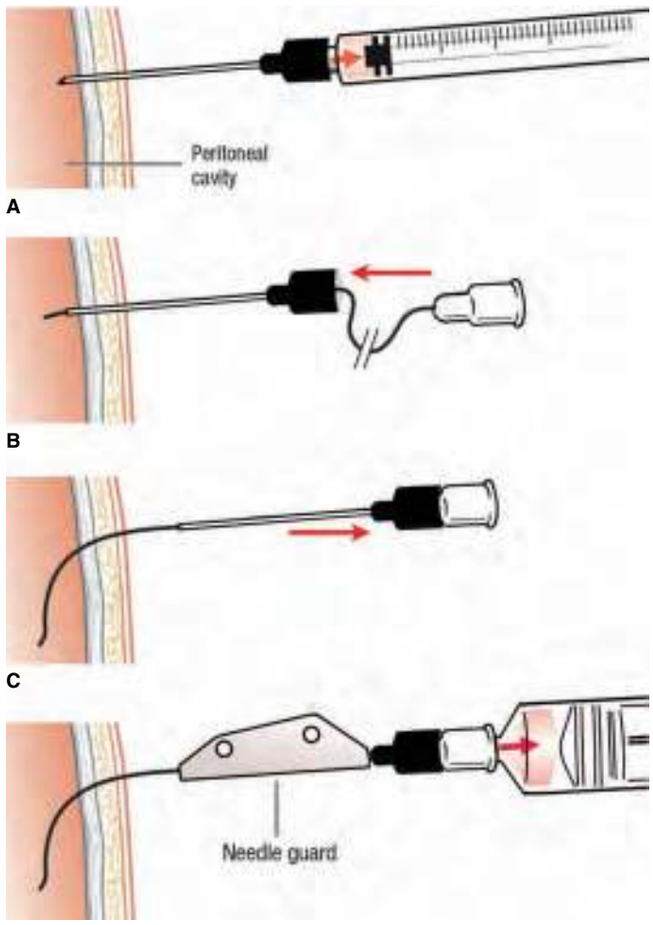

FIGURE 83-8. The catheter-through-the-needle technique. ***A.*** The needle is advanced into the peritoneal cavity while maintaining negative pressure on the syringe. ***B.*** The syringe has been removed. The catheter is inserted through the needle. ***C.*** The needle is withdrawn and the catheter is left within the peritoneal cavity. ***D.*** A syringe is attached to the catheter and the needle guard applied. The aspiration of ascitic fluid confirms proper intraperitoneal placement of the catheter.

The main disadvantage of this technique is the possibility of the needle tip shearing off the catheter and resulting in a catheter embolism. **This can be prevented by not withdrawing the catheter through the needle and applying the needle guard immediately after the needle is withdrawn from the skin.** Another disadvantage is that the contaminated needle must be handled to some extent, creating a potential risk for needle-stick injuries.

CATHETER-OVER-THE-NEEDLE TECHNIQUE

The catheter-over-the-needle systems are most commonly used for peripheral venous access. They are inexpensive, come in a variety of diameters and lengths, and are widely available. The catheter fits closely over a hypodermic needle. The needle and the catheter are advanced as a unit into the peritoneal cavity. Versions designed to minimize accidental needle–stick injuries are available, and their use is encouraged (**Figure 59-15**). Placement of these catheters is

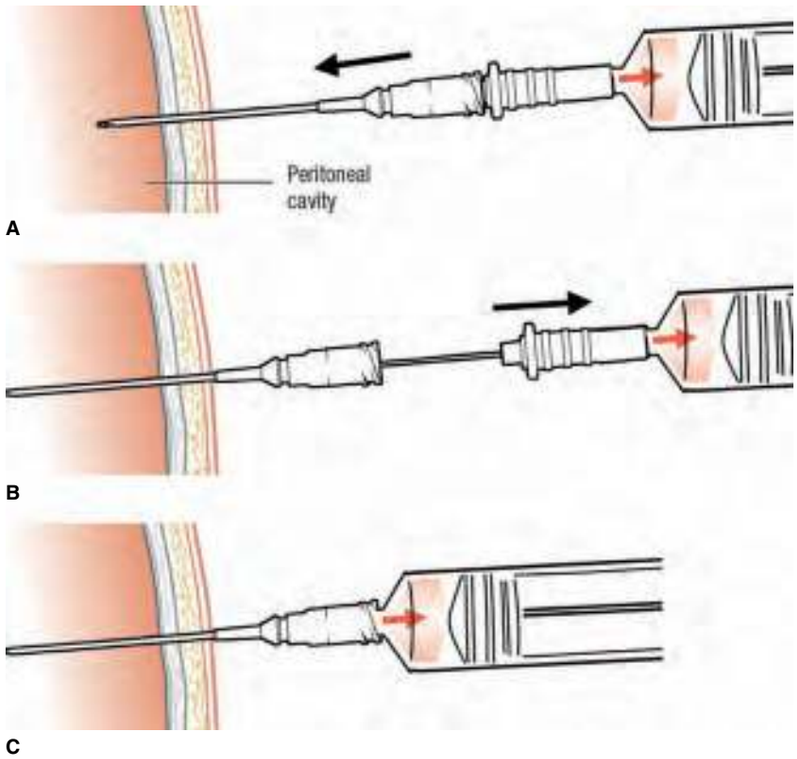

FIGURE 83-9. The catheter-over-the-needle technique. ***A.*** The catheter-over-the-needle is inserted into the peritoneal cavity while maintaining negative pressure on the syringe. ***B.*** The needle and syringe are removed. ***C.*** A syringe is attached to the catheter. The aspiration of ascitic fluid confirms proper intraperitoneal placement of the catheter.

usually quick and simple. Consider using a Caldwell needle with fenestrations on the side to help minimize problems with the flow of fluid.

Choose the puncture site and prepare the patient for the procedure. Insert the catheter-over-the-needle through the skin in a Z-track manner and into the peritoneal cavity (**Figure 83-9*A***). A flash of fluid in the hub of the needle confirms that the tip of the needle is within the peritoneal cavity. Advance the catheter-over-the-needle an additional 2 to 3 mm to ensure that the catheter is within the peritoneal cavity. Hold the hub of the needle securely. Advance the catheter over the needle until its hub is against the skin (**Figure 83-9*B***). Withdraw the needle and syringe as a unit. Attach a syringe onto the hub of the catheter (**Figure 83-9*C***). Aspirate fluid from the catheter to confirm intraperitoneal placement. Hold the hub of the catheter securely and remove the syringe. Pass the syringe to an assistant to place the sample into laboratory containers. Connect the catheter to intravenous tubing. Connect the other end of the tubing to a suction bottle or bag to drain the desired amount of fluid. An alternative is to attach a three-way stopcock to the distal end of the tubing.[22] A syringe may then be attached to the stopcock to withdraw laboratory samples or "pump out" the ascitic fluid into a nonsterile container. Remove the catheter once the procedure is completed. Apply a bandage to the skin puncture site.

ULTRASOUND-ASSISTED AND -GUIDED PARACENTESIS

The use of US increases the success rates for performing a paracentesis and decreases complications.[27] US may be used to assist or guide the paracentesis. A static technique is used to identify the skin puncture site and ascitic fluid location. The remainder of the procedure is "blind" using one of the above described techniques. Place the US transducer back on the abdomen to locate the needle and determine if it is off target or not long enough to reach the peritoneal cavity if ascites is not aspirated.

A dynamic technique is used occasionally, and the needle is inserted using real-time US. Position the patient. Clean and prep the skin. Scan initially with a 3.5 to 5 MHz transducer longitudinally and transversely to survey each potential needle entry site (**Figure 83-10**).[23] Ascites appears anechoic (black) and outlines the loops of intestine (**Figure 83-11**). The intestine may be seen undulating in the ascites due to intestinal peristalsis. Without ascites, the individual loops of intestine cannot be visualized. Scan with the high-frequency 8 to 12 MHz transducer to provide improved resolution, improved identification of the inferior epigastric vessels, and better needle visualization during the procedure. Identify the inferior epigastric artery and vein in the abdominal wall (**Figure 83-12**).[28]

FIGURE 83-10. The patient is positioned and being surveyed for the location of ascitic fluid.

FIGURE 83-11. The presence of anechoic free fluid (FF) allows the individual intestine loops to be discerned.

Note the amount of fluid and the presence of any structure that might make a site undesirable. Reposition the patient if no site seems suitable and repeat the US. Note the depth from the skin surface to the fluid. **Choose a site with the most fluid at the least depth.** Position the US transducer so that the target is in the middle of the US view screen. The entry site will now be under the center of the transducer. Mark the skin, wipe off the US gel, and repeat the prep. Prepare the US transducer. Have an assistant apply US gel over the footprint of the transducer. Instruct the assistant to place the gel-coated US transducer in the sterile cover while you hold it. Have the assistant place sterile US gel over the abdomen. Reidentify the ascites and proceed with the procedure (**Figure 83-13**).

Do not mistake a cystic fluid collection or the bladder for ascites. Ascites will outline individual intestine loops and appears in many places around the abdomen. Fluid in a cyst or the bladder has rounded borders and is localized. Loculated ascites will occasionally mimic a cyst but will still outline the loops of intestine.

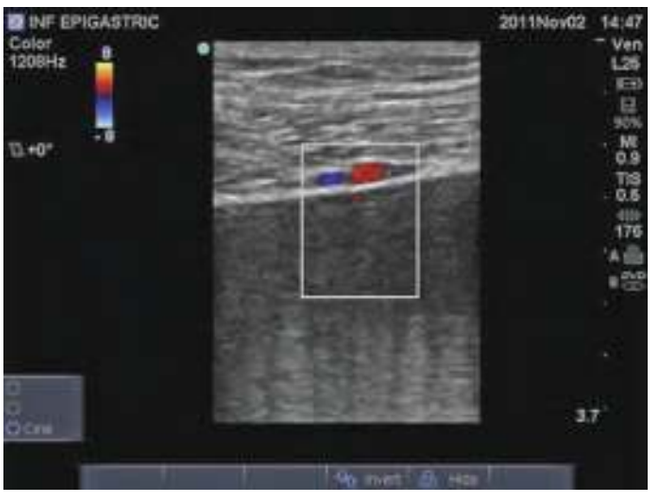

FIGURE 83-12. Abdominal US image identifying the inferior epigastric vessels in the abdominal wall. The artery is red and the vein is blue. Hypoechoic ascites can be seen in the abdomen. (Used with permission from reference 28.)

FIGURE 83-13. The needle (*arrows*) is inserted through the abdominal wall and into the free fluid.

ALTERNATIVE TECHNIQUES

Use wall suction to safely and quickly remove a large volume of fluid (**Figure 83-14**).[29-31] The use of disposable plastic containers instead of bottles saves cost per patient.[29] This eliminates the need to find

FIGURE 83-14. Paracentesis set up using plastic vacuum containers mounted on a stand. The same set-up can stand on a bedside table. Note the paracentesis catheter (*arrow*) attached to the tubing. (Used with permission from reference 29.)

vacuum bottles, which are sometimes in short supply. There are many advantages to using plastic containers attached to wall suction (e.g., no shattering or exploding, no changing between bottles, and constant suction). Wall suction is often lower than the initial pressure in bottles and using a syringe attached to a stopcock.[29] The wall suction pressure can be adjusted where the bottles cannot be adjusted. The drainage is constant with wall suction because the pressure does not change. Bottle pressure decreases as the bottle fills with fluid, and the ascites comes out slower as the bottle becomes filled. Using plastic containers and wall suction may shorten the procedure time.

AFTERCARE

Bandage the skin puncture site once the procedure is completed. The site will occasionally ooze fluid in patients with tense ascites. Instruct these patients to change their dressings regularly. Occlusive tape can be helpful in preventing the fluid from leaking onto the patient's clothing and bedding. Sometimes a simple mattress or figure-of-eight suture is necessary to control the drainage. Emergency Physicians have used a tissue adhesive (e.g., Dermabond, Indermil, or Histoacryl) to help control a fluid leak.[32] The patient may be discharged home if the purpose of the paracentesis was to relieve tense ascites, no signs or symptoms of SBP exist, and the fluid analysis is unremarkable. Instruct the patient verbally and in writing to immediately return to the Emergency Department if they develop abdominal distension, abdominal pain, fever, nausea, or vomiting.

The patient will require hospitalization and intravenous antibiotics if SBP is suspected or diagnosed. Administer empiric antibiotics that cover gram-negative enterics (of which *Escherichia coli* is the most likely) and streptococcal species (including *Enterococcus*) in the Emergency Department. A third-generation cephalosporin (e.g., cefotaxime) covers the most common causative agents.[16] Add ampicillin if *Enterococcus* is suspected. Check previous culture results for antibiotic sensitivities if the patient has a history of SBP.

COMPLICATIONS

Complications from abdominal paracentesis are infrequent. Known complications include abdominal wall hematoma, intestine perforation, hemoperitoneum, infection, persistent ascitic fluid leak, shearing of the peritoneal catheter, and systemic hemodynamic compromise.

Bleeding is a potential complication of paracentesis.[33] The administration of prophylactic blood products (e.g., fresh frozen plasma) is not warranted.[21,34,35] Spontaneous hemoperitoneum secondary to mesenteric variceal bleeding has been reported to occur in patients receiving large-volume (e.g., > 4000 mL) paracentesis.[36] Patients who developed this complication have had advanced cirrhosis with refractory ascites, hemorrhagic shock without evidence of gastrointestinal bleeding, and previous large-volume paracentesis. Mortality from this complication is exceedingly high. An additional vascular complication, an inferior epigastric artery pseudoaneurysm or injury, has been described.[37] Embolization may be warranted if there is large-volume hemorrhage.[38]

Perforation of the intestine during paracentesis is rare. Most of these injuries are self-sealing and develop no further problems. Generalized peritonitis and abdominal wall abscesses have been reported and are exceedingly rare. Do not move the needle when it is within the peritoneal cavity to avoid lacerating the intestine wall.

Ascitic fluid may continue to leak from the site of paracentesis. Apply skin closure strips to the skin puncture site to stop leaks. This does not always work. A simple suture at the site may correct the problem. Evaluate patients with persistent leaks for peritonitis.

Rapid removal of significant amounts of ascitic fluid has been found to cause hemodynamic compromise.[39,40] Initial removal of large amounts of ascites causes improvement in circulatory function, likely related to both mechanical (e.g., improved cardiac venous return) and neurohumoral factors. Total paracentesis in cirrhotic patients may cause delayed (i.e., over 12 to 24 hours postprocedure) effective hypovolemia by accentuation of baseline arteriolar vasodilation through neurohumoral mechanisms.[41] The literature suggests that some of the postparacentesis circulatory dysfunction may be avoided by pretreating patients with an intravenous colloid (e.g., albumin).[19,39,42] This is not a universally accepted practice, and colloid replacement is likely unnecessary for patients with ≤ 5 L removed. A typical starting dose of albumin, if choosing to administer it, is 6 to 8 gm of albumin for every liter of ascites removed.[19,43]

TABLE 83-1 Laboratory Tests for Ascitic Fluid

Recommended	Optional	Unusual
Albumin	Amylase	Acid-fast smear and culture
Cell count	Glucose	Cytology
Cultures	Grams stain	Triglycerides
pH	Lactate dehydrogenase	
	Total protein	

ASCITIC FLUID ANALYSIS

Normal ascitic fluid should appear clear with a yellow color. Increased turbidity may suggest infection, elevated triglyceride levels, or other particulate matter. Sanguineous fluid is present in patients with malignancy, intraperitoneal bleeding from the intraabdominal organs (i.e., iatrogenically or spontaneously introduced), or tuberculous peritonitis. The appearance of the ascitic fluid is not sensitive for detecting SBP and cannot be used as a screening tool.[44-47]

The specific analytical tests ordered on ascitic fluid should reflect the Emergency Physician's clinical suspicion (**Table 83-1**). Simple analysis of fluid with an albumin concentration, cell count and differential, and routine cultures are all that is necessary in patients with uncomplicated cirrhosis. These initial tests can be supplemented depending on clinical suspicion. An ascitic fluid pH < 7.35 and a blood-ascitic fluid pH gradient ≥ 0.10 can aid in the diagnosis of SBP.[8] Amylase, bilirubin, glucose, lactate dehydrogenase, total protein, and triglycerides are not helpful except in select circumstances and are not warranted on a routine basis. The serum-ascites albumin gradient is approximately 97% accurate in indicating portal hypertension.[6,48] The gradient is calculated by subtracting the ascitic fluid albumin concentration from the simultaneously measured serum albumin concentration. A gradient of ≥ 1.1 gm/dL suggests portal hypertension as the etiology of the ascites (**Table 83-2**). A gradient of < 1.1 gm/dL suggests that the patient does not have

TABLE 83-2 Classification of Ascites by the Serum to Ascites Albumin Concentration Gradient

High gradient (≥ 1.1 gm/dL)	Low gradient (< 1.1 gm/dL)
Alcoholic hepatitis	Nephrotic syndrome
Budd-Chiari syndrome	Pancreatic ascites
Cardiac ascites	Peritoneal carcinomatosis
Cirrhosis	Serositis in connective tissue disease
Fatty liver of pregnancy	Tuberculous peritonitis
Fulminant hepatic failure	
Massive liver metastases	
Mixed ascites	
Myxedema	
Portal vein thrombosis	
Venoocclusive disease	

portal hypertension and that the ascites has some other etiology (**Table 83-2**). A dipstick for glucose, pH, and protein can be used to differentiate an exudate from a transudate.[49] This method uses a hard to remember equation and requires the use of a calculator (k = 0.012protein – 0.012glucose – 3.329pH + 23.498).

Suspect peritoneal carcinomatosis and order cytology in patients with a history of breast cancer, colon cancer, gastric cancer, pancreatic cancer, or the suspicion of undiagnosed malignancy and ascites. Smear and cultures for mycobacteria are not sensitive. They should still be ordered when the suspicion for tuberculous peritonitis is high (e.g., patients who have immigrated from endemic areas or have an immunocompromised status).[50]

Patients with uncomplicated ascites secondary to cirrhosis should have an ascitic fluid white blood cell (WBC) count < 500 cells/mm^3. The cells should be predominantly lymphocytes and mesothelial cells without clinical evidence of peritonitis. A polymorphonuclear (PMN) cell or neutrophil count of > 250 cells/mm^3 confirms SBP.[51] The PMN count is obtained by multiplying the total WBC count by the percentage of PMNs in the differential. Subtract one neutrophil per 250 red cells/mm^3 to adjust for significant blood in a specimen (i.e., RBC > 10,000 cells/mm^3) from a traumatic tap.[16] A Gram stain is not sensitive for SBP secondary to the low concentration of bacteria in ascites. Patients receiving continuous peritoneal dialysis can have SBP diagnosed with a WBC > 100 cells/mm^3.[51] **Culture all potentially infected ascitic fluid by directly inoculating blood culture bottles at the bedside.[25,26]**

SUMMARY

Paracentesis is a safe procedure that is common in the practice of Emergency Medicine. There are few relative contraindications to its performance. It is most commonly performed diagnostically to detect SBP. It can be performed therapeutically for symptomatic relief in patients with tense ascites. Proper technique with assistance of US can increase the chances of a successful paracentesis while decreasing the chances of complications.

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84 Diagnostic Peritoneal Lavage

Leann Mainis and Rene Ramirez

INTRODUCTION

The diagnostic peritoneal lavage (DPL) was first described in 1965 by Dr. Root who developed a method for sampling the peritoneal cavity to more rapidly determine the presence of hemoperitoneum after trauma.[1] The initial physical examination can be misleading in up to 45% of blunt trauma patients, and DPL can be useful in diagnosing intraabdominal injury in a timely fashion.[2-4] DPL is performed less frequently due to the use of focused abdominal sonography for trauma (FAST) bedside ultrasound (US) scanning and helical computed tomography (CT).[5,6] DPL is the only invasive test of the three and remains a highly sensitive test for mesenteric and hollow viscus injuries.[4,7,8] The main disadvantages of DPL are that it gives no information about the specific organ injured and a positive DPL requires an invasive procedure in the Operating Room versus conservative management and observation. The overall use of DPL is declining with the progression of advanced CT scanning, repeat/serial FAST exams, and the increased availability of these modalities.[3,5,9]

Dr. Root's description of the DPL represented an improvement upon the use of paracentesis to identify a hemoperitoneum as described by Salomon in 1906.[10] His initial description of a DPL used a trocar placed into the peritoneal cavity to instill fluid. The fluid was visually inspected upon removal and the patient then underwent a laparotomy if it appeared bloody.

DPL has undergone several modifications since its initial description. The trocar technique was initially abandoned in favor of the open technique, which later fell to the Seldinger or closed technique.[11,12] A novel method that combines the use of diagnostic laparoscopy and DPL has been termed laparoscopic diagnostic peritoneal lavage (L-DPL).[13] This procedure combines the visual advantages of laparoscopy with the sensitivity and specificity of a DPL for the diagnosis of significant penetrating intraabdominal injury. While the DPL was first described for blunt abdominal trauma, it has found an indication in the patient with penetrating abdominal trauma.[2] Initial attempts to quantify the effluent based on its appearance have been replaced by the red blood cell (RBC) count, the white blood cell (WBC) count, and the measurement of various enzymes.[14-16] The debate is still present in the literature as to which criterion best determines the need for a laparotomy.

ANATOMY AND PATHOPHYSIOLOGY

The gross anatomy of the abdomen is well known to the Emergency Physician and is important to review when preparing for a DPL. The abdominal cavity is lined by the peritoneum and is protected from the environment by the abdominal wall musculature, fat, and skin. The right and left rectus muscles, which are nourished by the epigastric vessels, meet in the midline at the avascular linea alba. The umbilicus is located along the lower portion of the linea alba. The layers of the anterior abdominal wall structures vary above and below the level of the anterior superior iliac spine (**Figure 84-1**).

The DPL is always performed in the anterior midline of the abdomen (Figure 84-2). The linea alba is an avascular location through which the peritoneal cavity may be entered using either an open technique or a closed Seldinger type technique. This midline location minimizes the number of false-positive lavages that occur due to bleeding from the abdominal wall muscles or blood vessels. This also allows the Surgeon to perform a midline laparotomy, if necessary, through the lavage site and avoid the formation of an avascular skin bridge.

Most DPLs may be safely performed 1 to 2 cm below the umbilicus (**Figures 84-2*A* and 84-2*B***). This location allows the DPL catheter to be directed into the pelvis, minimizes the occurrence of inadvertent vascular injury, and increases the likelihood that fluid will be sampled from a dependent portion of the abdomen. It is more difficult to retrieve fluid if the DPL is performed above the umbilicus due to interference from the omentum. All closed and semi-open DPLs, as well as many open DPLs, are performed below the umbilicus.

The resultant retroperitoneal hematoma in patients with a pelvic fracture may extend anteriorly to the level of the linea semilunaris. **Perform the DPL in patients with a pelvic fracture using an open technique above the umbilicus (Figure 84-2*C*).** This will avoid a false-positive DPL and the inadvertent decompression of the hematoma.[17]

Another special situation occurs in the pregnant patient. **Perform the DPL using an open technique superior to the uterine fundus to minimize the chance of injuring the gravid uterus.**[11] It may be performed below the umbilicus if the patient is in an early stage of pregnancy.

It may be necessary to change the location of the DPL site in a patient with previous abdominal surgery. These patients must be individualized based on the location of their scar. The DPL should be performed supraumbilically if the patient has a lower abdominal scar and infraumbilically if the patient has an upper abdominal scar.[1,14] **Perform the DPL using an open technique to avoid any adhesions and minimize complications.**[18]

INDICATIONS

A DPL is indicated in any patient with suspected abdominal trauma, blunt or penetrating, who does not have an obvious indication for a laparotomy and in whom serial physical examinations are not practical. It can be performed quickly, will reliably exclude significant intraabdominal trauma, and will allow the diagnosis and treatment of associated injuries. It does not require transfer of the patient out of the monitored environment of the Emergency Department as does a CT scan. DPL does not require the sophisticated equipment or US training that is required to perform a FAST exam.

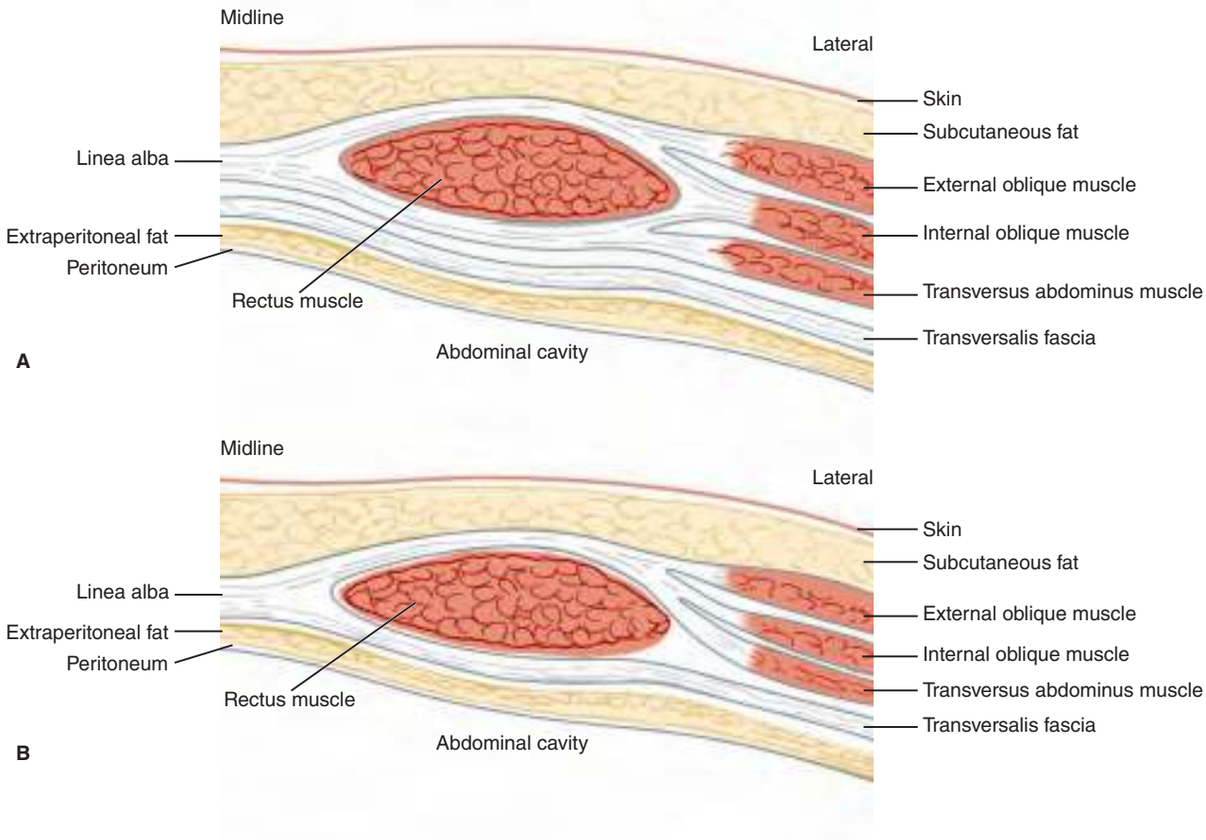

FIGURE 84-1. The layers of the anterior abdominal wall vary above (*A*) and below (*B*) the level of the anterior superior iliac spine.

A DPL is especially useful in patients with an unreliable or equivocal abdominal examination and those who will be unavailable for serial physical examinations. The patient with an altered mental status (e.g., alcohol, drugs, head injury) or abnormal sensation due to a spinal cord injury is considered to have an unreliable physical examination.[11,16,19] Patients may have an equivocal examination due to tenderness from surrounding fractures of the ribs, spine, or pelvis.[16,18] They may have tenderness over the wound or an area of injury that is difficult to distinguish from peritonitis. The third group of patients who may benefit are those who undergo surgery for another injury (e.g., neurosurgical or orthopedic).[19] These patients are unavailable for serial examinations while in the Operating Room. Their examination may be altered postoperatively due to the analgesics that they receive.

DPL may be useful when the patient presents in shock with other potential sources of hemorrhage (e.g., intrathoracic or retroperitoneal bleeding). It may confirm or rule out the abdomen as the source of the patient's bleeding and shock.[11] **An injury confined to the retroperitoneum will not result in a positive DPL.**[20] DPL can be used in critically ill patients with possible mesenteric ischemia and decrease operative procedures.[21] A DPL has been used to stage gastric cancer.[22]

CONTRAINDICATIONS

The only absolute contraindication to performing a DPL is if the patient has an obvious indication for a laparotomy. Patients who present following abdominal trauma with blood per orifice, evisceration, peritonitis, pneumoperitoneum on chest radiography, retained stabbing implements, or shock have obvious indications for a laparotomy. Preexisting coagulopathy is a relative contraindication due to increased risk of bleeding.

There are several relative contraindications to performing a closed DPL. Patients with a pelvic fracture may have a large retroperitoneal hematoma that extends anteriorly to the linea semilunares. The DPL may be performed using the open technique above the umbilicus to avoid decompression of the hematoma.[17] Pregnant patients should not have a closed DPL performed because of the risk of uterine injury. It is safe to perform an open DPL above the uterine fundus.[11] Patients with evidence of previous abdominal surgery may have visceral adhesions to the abdominal wall that make intraabdominal injury more likely to result when a closed DPL is performed.[1,14] An open technique may be performed in a location away from the scar.[18] Compartmentalization of the abdomen may have occurred due to intraperitoneal adhesions. This makes a false-negative DPL result more likely. Perform the DPL using the open technique in patients who are unable to have a Foley catheter placed due to urethral injury or stricture. This minimizes the chance of inadvertent injury to the bladder. Morbid obesity represents a difficulty in technically performing the DPL when the abdominal wall is thicker than the 2.5 inch long locator needle. It is useful to perform the DPL in these patients using a semi-open technique.[23]

EQUIPMENT

CLOSED TECHNIQUE

- Povidone iodine or chlorhexidine solution
- Face mask
- Sterile gloves

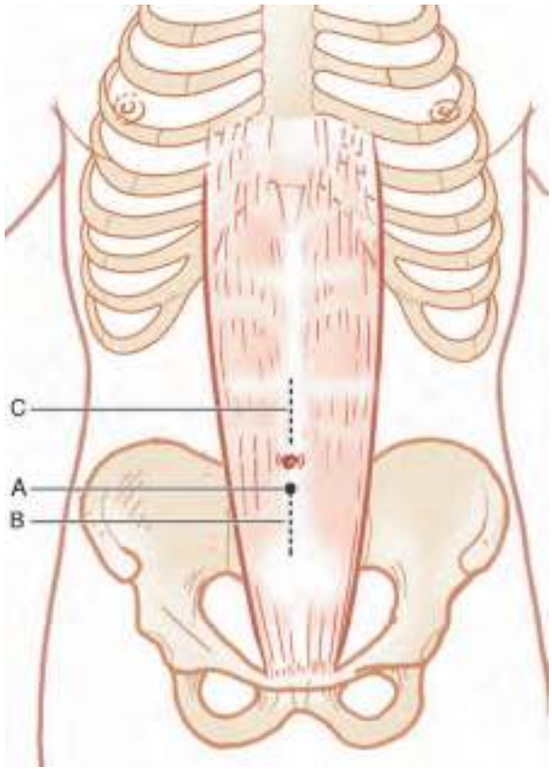

FIGURE 84-2. The preferred sites to perform a DPL. ***A.*** The midline and approximately 1 to 2 cm below the umbilicus is the location for a closed DPL. ***B.*** A midline incision beginning 1 to 2 cm below the umbilicus and extending inferiorly for 5 to 6 cm is the location for semi-open and select open DPLs. ***C.*** A midline incision beginning 2 cm above the umbilicus and extending superiorly for 5 to 6 cm is the location for most open DPLs.

- Sterile gown
- 4×4 gauze squares
- Local anesthetic solution
- Purple top blood collection tube
- 5 mL syringes
- 18 gauge needles
- 25 gauge needles
- Nasogastric tube
- Foley catheter
- 1 L of intravenous fluid to infuse 0.9% NaCl or lactated Ringer's solution
- Commercial peritoneal lavage kit (e.g., Arrow AK-09000)

SEMI-OPEN OR OPEN TECHNIQUE

- All items listed above
- Razor
- #10 scalpel blade on a handle
- Abdominal skin retractors, Weitlaner or skin rakes
- 2 tissue forceps
- 2 Allis clamps
- 4 hemostats
- Needle driver
- Suture for vessel ligation (e.g., 4–0 Vicryl, 4–0 Dexon, or 4–0 chromic)

FIGURE 84-3. The equipment required to perform a closed DPL. Note that lavage fluid is not included.

- Suture for fascial closure (e.g., 0 Vicryl, 0 Dexon, 0 Maxon, or 0 Prolene)
- 4–0 nylon suture or skin stapler for skin closure
- Suture scissors

A commercially available, disposable, and single-patient use peritoneal lavage kit is available from numerous manufacturers. The kit includes all the material required to perform a closed DPL except lavage fluid and the personal protective equipment for the Emergency Physician (**Figure 84-3**). An example is the Arrow kit (Arrow International, Reading, PA). It contains 10% povidone iodine swabs, gauze squares, a fenestrated drape, intravenous (IV) fluid administration tubing, 1% lidocaine, 5 mL syringes, 22 and 25 gauge needles, an 18 gauge × 2.5 inch introducer needle, an 0.89 mm × 45 cm J-tipped guidewire, a lavage catheter, and a #11 blade scalpel.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. This should include the possible complications and alternative options (e.g., observation or serial FAST exams). Obtain an informed consent from the patient or from the family if the patient is unable to consent due to age or mental status. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. **The consent process should not delay the performance of the DPL in an unstable patient.**

Place the patient in the supine position. A distended stomach or bladder may be inadvertently perforated during the procedure and require decompression prior to performing a DPL (**Figure 84-4**). Decompress the stomach using a nasogastric or orogastric tube (Chapter 75).[19,24] Decompress the bladder using a Foley catheter once a urethral injury has been ruled out (Chapters 173 and 176).[11,12,19,24]

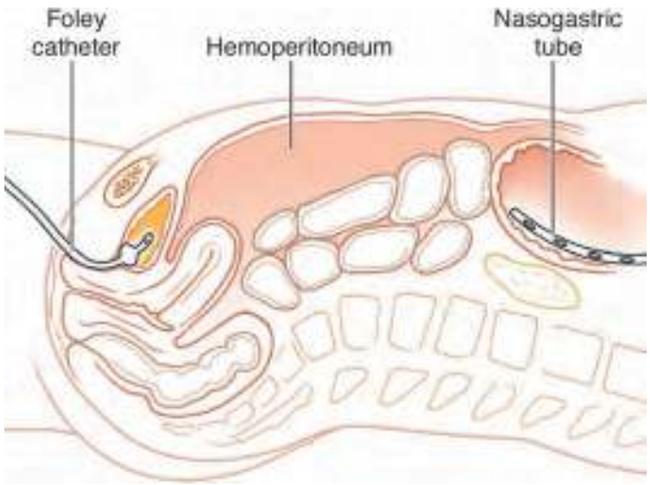

FIGURE 84-4. Midsagittal section through the abdomen and pelvis demonstrating decompression of the stomach with a nasogastric tube and decompression of the bladder with a Foley catheter.

Prepare the abdomen.[19] Shave the area surrounding the incision site if an open or semi-open DPL is to be performed. Clean the skin of any dirt, debris, and blood. Apply povidone iodine or chlorhexidine solution and allow it to dry. Apply sterile drapes to delineate a sterile field. Each health care provider involved in the procedure should don a hat, face mask, sterile gown, and sterile gloves.[19]

TECHNIQUES

A DPL is performed one of three different ways: closed, open, or semi-open.[25,26] The closed technique relies on percutaneous needle access to the peritoneal cavity followed by the insertion of a catheter using the Seldinger technique. The open technique uses a vertical infraumbilical incision and direct visualization of the peritoneal cavity before inserting the catheter. The semi-open technique follows the same principles of the open technique except that the midline fascia is penetrated with a needle and the catheter is advanced using the Seldinger technique. There is no difference in overall outcomes or rates of injury to visceral contents between the three techniques.[27-30]

PERCUTANEOUS (CLOSED) TECHNIQUE

The closed technique is the preferred method unless contraindications exist (**Figure 84-5**). The closed DPL can be performed more rapidly than the open technique.[27,30] There is no difference in the amount of fluid retrieved from the abdomen, the diagnostic accuracy, or the complication rate between the closed and open techniques.[27]

The closed DPL is performed in the midline and approximately 1 to 2 cm below the umbilicus (**Figure 84-2*A***).[24] Prepare the patient as described previously. Infiltrate the skin, subcutaneous tissue, and fascia with local anesthetic solution. Place the 2.5 inch introducer needle onto a 5 mL syringe. Insert the needle in the midline and 1 to 2 cm below the umbilicus. Advance the needle at a 45° angle toward the pelvis (**Figure 84-5*A***). Apply negative pressure to the syringe as it is advanced. Three "pops" may be felt as the needle penetrates the skin, fascia, and peritoneum. Slowly advance the needle after the third pop another 2 to 3 mm while maintaining negative pressure on the syringe. **A flash of blood will be seen in the syringe if the patient has a massive hemoperitoneum. This is considered a positive tap and concludes the procedure.**

Continue the procedure if the tap is negative. Stabilize the introducer needle with one hand at the level of the abdominal wall and remove the syringe. **Do not allow the introducer needle to move as it may lacerate the intraabdominal organs.** Insert the guidewire through the introducer needle until 7 to 10 cm of the guidewire remains outside of the hub of the needle (**Figure 84-5*B***). The guidewire should advance through the needle easily with only minimal resistance. **Difficulty in advancing the guidewire or the patient complaining of pain requires removal of the guidewire and introducer needle as a unit. Never withdraw the guidewire through the introducer needle.** The tip of the introducer needle can shear off the guidewire resulting in a piece of the guidewire being left in the peritoneal cavity and necessitating an operative procedure. Reinsert the introducer needle and restart the procedure.

Stabilize the guidewire and withdraw the introducer needle over the guidewire (**Figure 84-5*C***). Make a small incision in the skin and subcutaneous tissue alongside the guidewire with a #11 scalpel blade to facilitate passage of the lavage catheter (**Figure 84-5*D***). **Make the incision no more than 5 mm in length.**

Place the lavage catheter over the guidewire (**Figure 84-5*E***). Advance the lavage catheter into the peritoneal cavity (**Figure 84-5*F***). It may be helpful to gently twist the catheter as it passes through the fascia to aid in inserting it into the peritoneal cavity. Advance the catheter until its hub is against the abdominal wall in the adult patient. Securely hold the hub of the lavage catheter. Remove the guidewire (**Figure 84-5*G***). Attach a 5 to 10 mL syringe to the hub of the lavage catheter. Apply negative pressure to the syringe. Note if any blood is aspirated. **The aspiration of a small amount of blood is not considered to be a positive tap and the procedure should continue.**

The presence of gross blood upon entering the peritoneal cavity with the introducer needle (i.e., positive tap) is considered an indication for laparotomy. It represents blood throughout the peritoneum. Confusion exists when blood is withdrawn through the lavage catheter (i.e., peritoneal aspirate). It is considered a positive aspirate and concludes the procedure if 10 mL of blood, gross bile, stool, or food is aspirated initially by the lavage catheter.[31] The patient should be prepped for a laparotomy. The DPL catheter has been directed into the most dependent portion of the peritoneal cavity (**Figure 84-6**). **A small amount of blood withdrawn (i.e., less than 10 mL) through the catheter may represent the only blood present within the abdomen and is not an indication for laparotomy.[19]**

Continue the procedure if the aspirate is negative. Pass the proximal end of the IV tubing to a nonsterile assistant to insert into a bag of IV fluid (e.g., 0.9% NaCl or Ringer's lactate) and prime the tubing. Warmed IV fluid is preferable to room temperature fluid if available. Attach the distal end of the IV tubing to the lavage catheter (**Figure 84-7*A***). Open the clamp on the IV tubing and allow the lavage fluid to flow freely into the peritoneal cavity (**Figure 84-7*A***). The lavage catheter may not be within the peritoneal cavity if the lavage fluid does not flow quickly but seems to drip in slowly. Reassess the catheter position. If necessary, remove and reinsert the lavage catheter. Infuse 1 L in the adult patient and 10 to 20 mL/kg to a maximum of 1 L in the pediatric patient.[12,18] Stabilize the catheter with one hand during the infusion of the lavage fluid.

Place the fluid bag on the floor after the lavage fluid has been instilled to allow the fluid to flow out from the peritoneal cavity (**Figure 84-7*B***). **There should be a steady rate of flow of the lavage fluid from the patient's abdomen into the bag.** Diminution of flow usually results from the omentum blocking the side holes of the lavage catheter. Firm palpation of the patient's abdomen may increase the flow rate if it slows. The lavage catheter may need to be withdrawn slightly and reinserted. These maneuvers may help to dislodge the omentum. If manipulation of the catheter does not improve flow, a second liter of fluid may be infused in an adult and an additional 10 mL/kg in children. The threshold for a positive

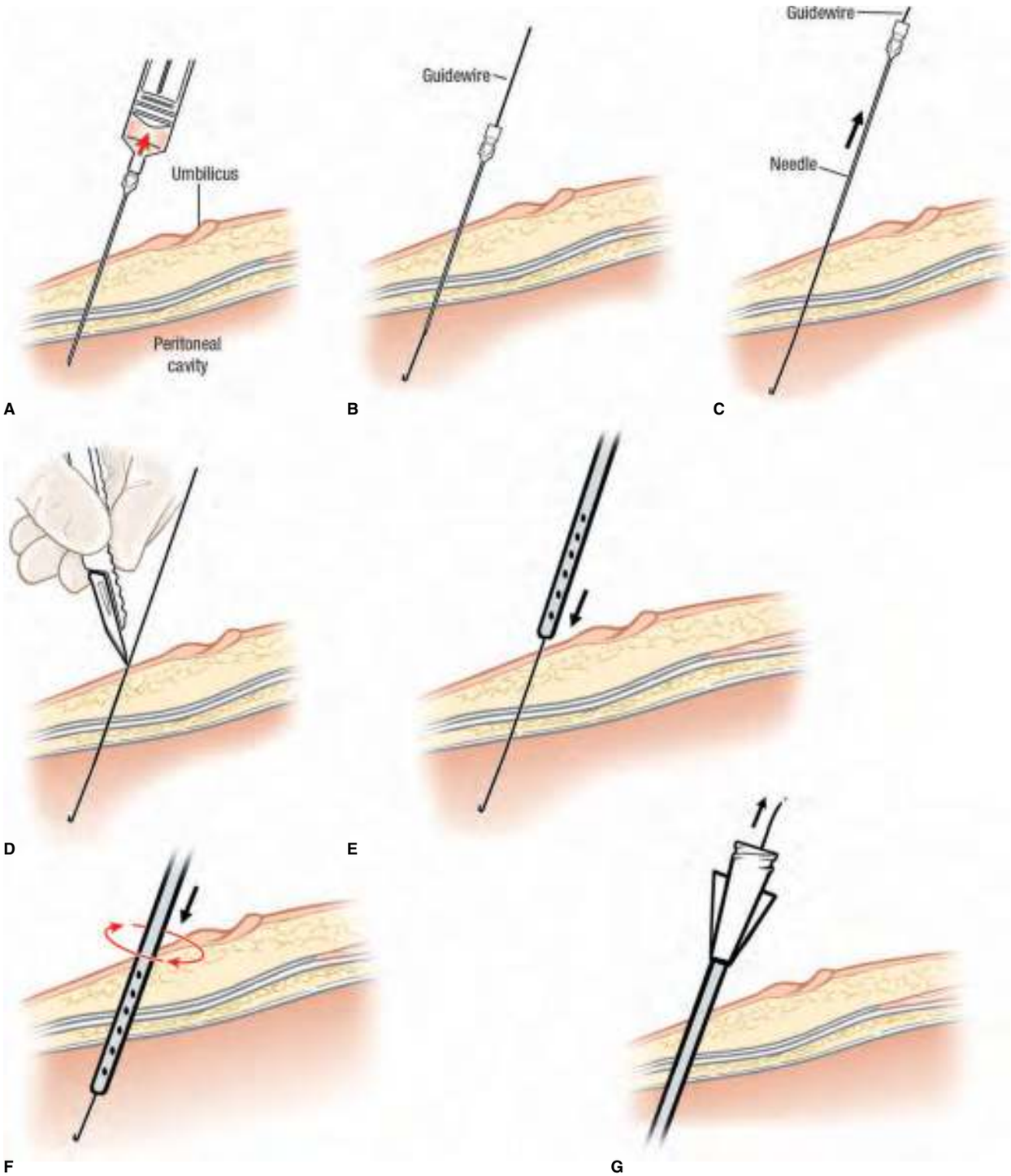

FIGURE 84-5. The percutaneous or closed DPL technique. ***A.*** The introducer needle is advanced caudally and at a 45° angle to the skin of the abdominal wall while negative pressure is applied to the syringe. ***B.*** The syringe has been removed and the guidewire is inserted through the introducer needle. ***C.*** The introducer needle is withdrawn over the guidewire, leaving the guidewire in place. ***D.*** A small nick is made in the skin and subcutaneous tissue adjacent to the guidewire using a #11 scalpel blade. ***E.*** The lavage catheter is placed over the guidewire. ***F.*** The lavage catheter is advanced over the guidewire and into the peritoneal cavity. A twisting motion may aid in the advancement of the catheter. ***G.*** The guidewire is removed, leaving the lavage catheter in place.

DPL must be halved (i.e., down from 100,000 to 50,000 RBC/mm^3 in blunt trauma) if it becomes necessary to infuse additional fluid as twice the fluid is infused diluting the RBC concentration.

At least 300 to 350 mL of lavage fluid should be returned from the peritoneal cavity to result in a reliable cell count.[9,30,32,33] This fluid is referred to as the effluent. Place a new lavage catheter if less than 300 to 350 mL of effluent is obtained after the manipulations described above.[9,12] Remove the existing lavage catheter while maintaining the sterility of the distal end of the IV tubing. Place a second lavage catheter 1 cm inferior to the site of the first lavage catheter. Reconnect the IV tubing and place the IV bag to gravity drainage to obtain the lavage effluent.

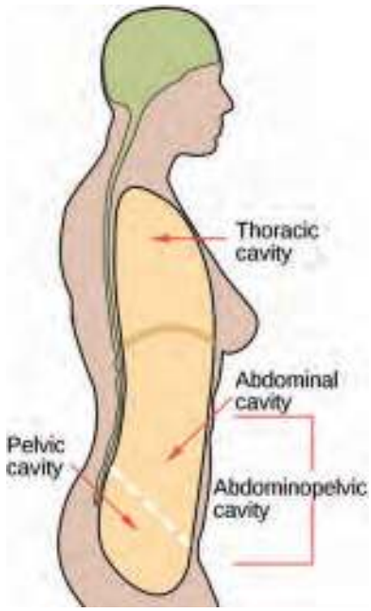

FIGURE 84-6. Midsagittal view of the body cavities. (Modified from http://en.wikipedia.org/wiki/laparotomy.)

Remove the lavage catheter after obtaining as much effluent as possible from the peritoneal cavity. Place a small gauze dressing over the skin puncture site and remove the drapes. Transfer a small aliquot of the lavage effluent to a purple top blood collection tube. Transport the tube to the laboratory for a cell count. The remainder of the lavage effluent may be discarded or reserved for subsequent testing.

OPEN TECHNIQUE

Prepare the patient as described previously. Infiltrate the skin, subcutaneous tissue, and fascia with a local anesthetic solution in the area where the incision is to be made. Begin the incision 2 cm below the umbilicus and extend it 5 to 6 cm inferiorly in patients who have an upper abdominal scar (**Figure 84-2*B***). Begin the incision 2 cm above the umbilicus and extend it 5 to 6 cm superiorly in patients with a pelvic fracture or lower abdominal scar (**Figure 84-2*C***). Make the incision in the midline above the uterine fundus in patients who are pregnant.

Incise the skin and subcutaneous tissue longitudinally 5 to 6 cm in the midline (**Figure 84-8*A***). The incision may need to be longer in an obese patient. Clamp and ligate any small vessels that are bleeding with absorbable suture prior to incising the fascia. This will minimize the incidence of false-positive lavage results. Retract the skin and subcutaneous tissues with a Weitlaner retractor or skin rakes to aid in viewing the fascia (**Figure 84-8*B***). The fascial midline may be identified by the interdigitation of its fibers (**Figure 84-8*B***). Carefully incise the fascia longitudinally along the length of the previous skin incision (**Figure 84-8*C***). Identify the peritoneum. Any preperitoneal fat that is present can be gently moved aside by an assistant using either a forceps or gauze. Grasp and elevate the peritoneum using two Allis clamps (**Figure 84-8*D***). **Use extreme care to ensure that no bowel is included in the clamps.**

Make a small (i.e., <5 mm) incision in the peritoneum. Insert the lavage catheter through the incision and directed caudally into the pelvis (**Figure 84-8*E***).[11] The remainder of the procedure is as described above in the percutaneous (i.e., closed) technique. It may be necessary to gently retract the peritoneum upward with the Allis clamps to prevent leakage of the lavage fluid around the catheter.

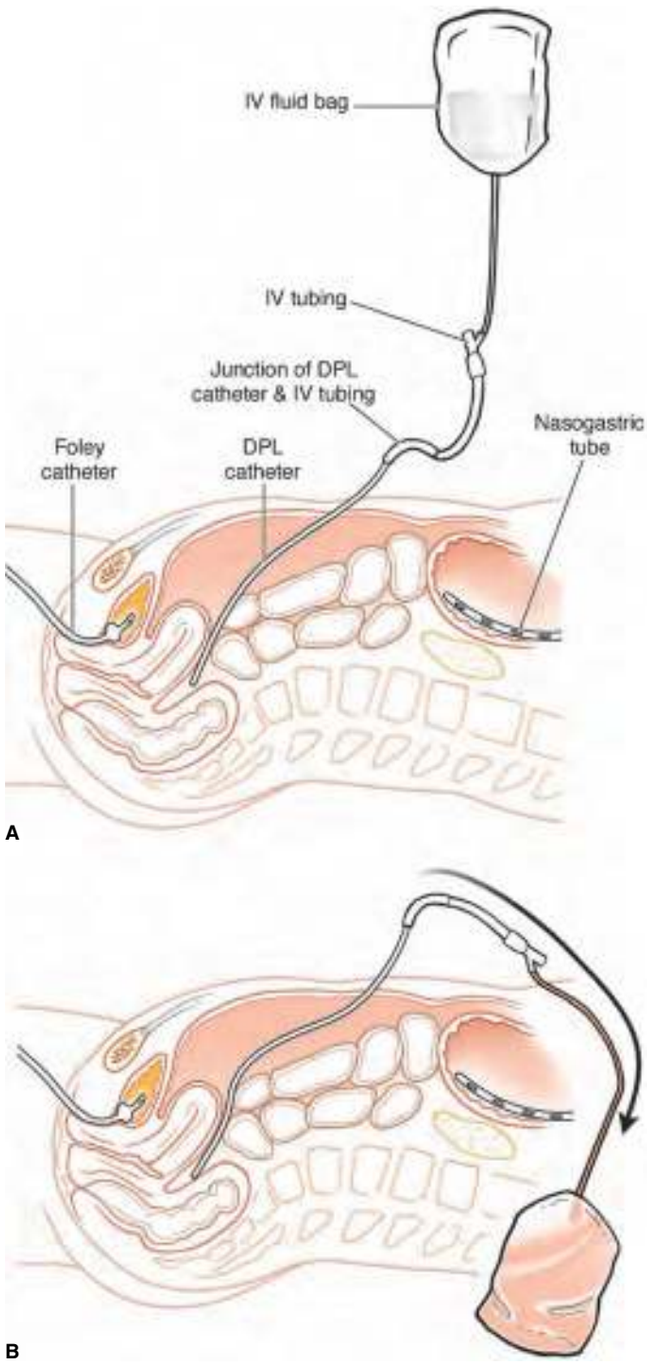

FIGURE 84-7. The instillation and removal of lavage fluid. ***A.*** The fluid bag is attached to the lavage catheter using IV tubing. The fluid bag is then suspended and allowed to infuse into the peritoneal cavity. ***B.*** The fluid bag is placed on the floor to allow the lavage fluid to exit the peritoneal cavity and flow back into the bag.

Remove the lavage catheter and Allis clamps after obtaining as much effluent as possible from the peritoneal cavity. It is not necessary to suture the peritoneum. Close the fascia with #0 Maxon, Prolene, Vicryl, or Dexon suture in a running fashion. Inspect the edges of the incision for any bleeding blood vessels. Obtain hemostasis of the subcutaneous tissue by ligating small vessels with absorbable suture. Close the skin with interrupted 4–0 nylon sutures or skin staples. Place a gauze dressing over the incision site. Remove the drapes.

SEMI-OPEN TECHNIQUE

This technique is used primarily for patients in whom there is no contraindication to the performance of a closed lavage and their abdominal wall is thicker (e.g., > 2.5 inches) than the length of the introducer needle. The procedure often begins as a closed technique and is converted to a semi-open technique once it is realized that the introducer needle is not long enough to enter the peritoneal cavity. It is a modification of both the closed and open techniques.[23]

Prepare the patient and begin the procedure as if performing the open technique. Make the midline incision and retract the tissues (**Figures 84-8*A* and 84-8*B***). Identify the interdigitations of the fascia in the midline. Place the introducer needle on a 5 mL syringe. Insert the introducer needle through the midline and at a 45° angle directed toward the pelvis (**Figure 84-5*A***). Apply negative pressure to the syringe while advancing the needle. A pop will be felt as the introducer needle penetrates the peritoneum. The remainder of the procedure is as described above under the percutaneous or closed technique.

Remove the lavage catheter after obtaining as much lavage fluid as possible from the peritoneal cavity. Inspect the edges of the incision for any bleeding blood vessels. Obtain hemostasis of the subcutaneous tissue by ligating small vessels with absorbable suture. Close the skin using interrupted 4–0 nylon sutures or skin staples. Place a gauze dressing over the skin incision site. Remove the drapes.

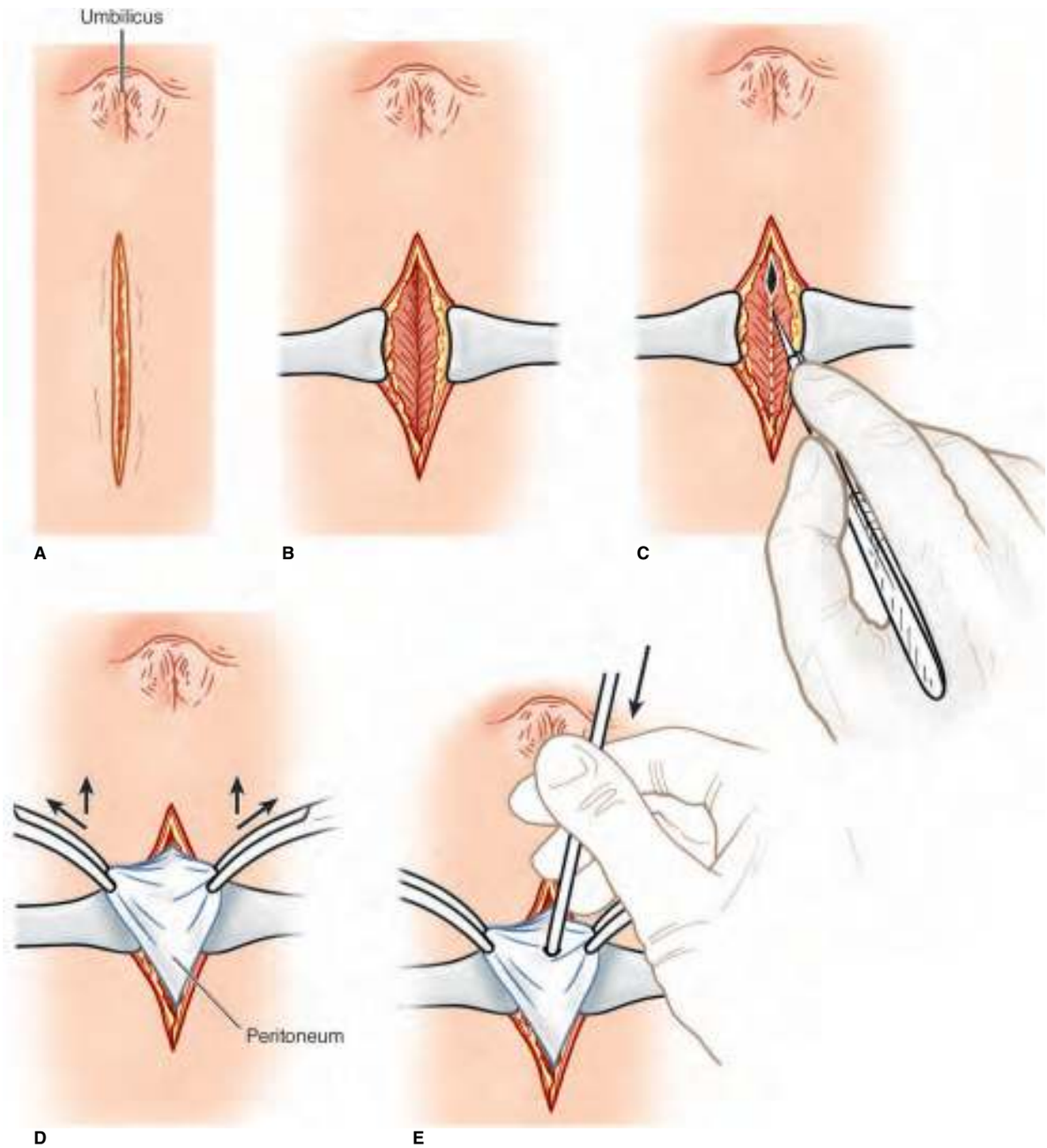

FIGURE 84-8. The open DPL. ***A.*** An incision is made in the skin and subcutaneous tissues. ***B.*** The skin and subcutaneous tissues are retracted. Note the interdigitation of the fibers of the fascia in the midline. ***C.*** An incision is made in the fascia. ***D.*** The peritoneum is grasped and elevated with Allis clamps. ***E.*** The lavage catheter is inserted through a small incision in the peritoneum.

LAPAROSCOPIC DPL (L-DPL)

The technique of L-DPL should be considered in hemodynamically stable patients with penetrating lower thoracic or abdominal stab wounds.[13] It is especially applicable for Trauma Surgeons with experience in basic laparoscopic techniques. The procedure is used to obtain conclusive evidence of significant intraabdominal injury, confirm peritoneal penetration, control intraabdominal bleeding, and repair lacerations (e.g., diaphragm and abdominal wall). The combination of laparoscopy and DPL adds to the sensitivity and specificity of DPL and avoids under- or oversensitivity, which has limited the use of DPL in hemodynamically stable trauma patients with suspicious or proven peritoneal penetration.

ANALYSIS OF PERITONEAL LAVAGE FLUID

There are several laboratory evaluations that can be performed on the lavage effluent to determine whether the patient requires a laparotomy. Gross evidence of injury exists when there is a positive peritoneal tap or aspirate, bile-stained peritoneal fluid is seen in the effluent, or enteric contents are seen in the effluent.[14,25] The most commonly used laboratory determination is that of the erythrocyte or RBC count. Other determinations include the leukocyte count and the presence of amylase in the lavage effluent.

Peritoneal lavage was first described as using visual inspection of fluid for the presence of blood.[1,7,14] This has been subsequently shown to be very inaccurate, even when performed by experienced physicians.[34] A much better method of quantification of the presence of blood uses the counting of erythrocytes manually or with an automated cell counter.

A positive DPL in an adult classically requires one of the following results: blood on the initial tap, 10 mL of gross blood on aspiration, greater than 500/mm^3 WBCs, greater than 100,000/mm^3 RBCs, or the presence of enteric contents or food matter.[9] The RBC count threshold can be adjusted from 100,000 RBC/mm^3 in patients who have sustained blunt trauma or an anterior abdominal stab wound to a lower count of 10,000 RBC/mm^3 in patients who may have suffered penetration of the abdominal cavity. Both counts appear to be very sensitive, specific, and accurate at diagnosing injury or penetration.[35]

In blunt abdominal trauma, 100,000 RBCs/mm^3 is the most commonly used threshold for a laparotomy.[11,16,25] This corresponds roughly to the presence of 20 mL of blood in the peritoneal cavity. Using the count of 100,000 RBCs/mm^3 to determine the presence of a hemoperitoneum provides a sensitivity of 77% to 97% and a specificity of 93% to 99.6%.[9,16] The false-positive results that occur are usually the result of bleeding from the lavage site. False-negative DPLs usually result in red blood cell counts in the mid-10,000s, which some authors refer to as indeterminate.[11] The injuries that are missed by a count of 100,000 RBCs/mm^3 are injuries to hollow viscera and the diaphragm, organs that may not bleed enough to result in a positive lavage.

Penetrating trauma has no universally agreed upon threshold for a laparotomy. Penetration of the peritoneal cavity is felt to be an indication for laparotomy in all gunshot wounds and most stab wounds to the abdomen. Peritoneal penetration by a gunshot wound results in a significant intraperitoneal injury in 98% of cases.[36] DPL to prove abdominal penetration is useful in cases of potentially tangential gunshot wounds.[20,37] Penetration into the peritoneal cavity from a thoracoabdominal wound results in a diaphragmatic injury that should be repaired.[24,38] Penetration through the retroperitoneum, from the back or flank, will cause a significant injury in 73% of cases.[20,32] Penetration of the peritoneal cavity may not bleed enough to result in a 100,000 RBCs/mm^3 count. Most authors believe that a lower RBC count should be used.

The RBC count varies from 1,000 to 50,000 RBCs/mm^3 as a threshold for laparotomy in penetrating trauma.[16,23,30,38-40] Higher cell counts are more likely to result in a greater false-negative rate.[16,19,24] Lower cell counts are more likely to result in a negative laparotomy and an increased false-positive rate.[16,17,24] A threshold of 10,000 to 25,000 RBCs/mm^3 provides the most acceptable balance between false-negative and false-positive results.[24,37,39,41] This threshold results in a sensitivity of 88% to 99%, a specificity of 97% to 98%, and an accuracy of 95% to 98%.[19,24,37]

A special case of penetrating abdominal trauma exists when there is a stab wound to the anterior abdomen. Only two-thirds of these wounds will penetrate the peritoneum. Less than 50% of those that penetrate will cause an injury that requires repair.[20] Using a lower threshold for DPL to determine penetration will result in a greater than 50% negative laparotomy rate. Wounds to the anterior abdomen are lavaged with the standard RBC count of 100,000 RBCs/mm^3 to determine injury, and not penetration, as the threshold for an operation.[42]

Many laboratories will routinely report the WBC count on the lavage effluent. The presence of over 500 WBCs/mm^3 is often quoted as a standard indication for a laparotomy.[25] The presence of an elevated lavage WBC count by itself has poor predictive value in the trauma patient.[32] There is commonly an associated elevation in the RBC count that will trigger a laparotomy. A recently described "cell count ratio" may help to diagnose the presence of a hollow viscus injury by comparing the WBC/RBC ratio in the lavage effluent to the WBC/RBC ratio in the blood.[43,44] There is a high likelihood of hollow viscus injury if the lavage ratio is greater than that of blood. This ratio is not universally accepted.

While the quantification of RBCs and occasionally WBCs in the DPL effluent is the most common test, other laboratory tests may be used in indeterminate cases. The most commonly used is the determination of amylase.[2,11,14] Amylase may be elevated in the presence of an injury to the gastrointestinal tract. The amylase level is neither sensitive nor specific.[15]

AFTERCARE

Observe the patient for up to 24 hours after performance of a negative DPL. The Foley catheter and nasogastric tube may be removed if they are no longer needed for other indications. Reexamine the patient periodically during this observation period for the development of peritonitis that may result from a false-negative lavage or a complication of the lavage procedure. The performance of the lavage should not alter the patient's examination, although there may be localized wound tenderness if the lavage was performed using the open or semi-open technique.[1] Prescribe analgesics as needed after the procedure. Keep the patient NPO during the initial portion of their observation. They can be fed near the end of the 24 hours to verify they tolerate oral intake prior to discharge.

Discharge instructions for the patient should instruct them to return for the development of a fever, nausea, vomiting, increasing abdominal or wound pain, or wound drainage. The patient should be seen in 7 to 10 days for removal of the skin sutures or staples after undergoing an open or semi-open DPL. No evidence exists supporting the use of prophylactic antibiotics.

COMPLICATIONS

The complication rate for DPL is relatively low. It varies between 0.6% to 2.3%.[18,45] There is no difference in complication rates between the three techniques.[27-30] The complications may be classified as wound-related or puncture-related.

Wound-related complications occur primarily with open or semi-open lavage techniques. Inadequate hemostasis during performance

of the DPL may result in bleeding and a hematoma formation at the wound site.[11,18,45] There is a risk of wound infection at the lavage site.[11] This infection is usually due to skin flora and may be treated simply by opening the wound and performing twice-daily wet-to-dry dressing changes.

Puncture-related complications may occur after any DPL technique. Virtually any organ within the abdominal cavity may be punctured by the introducer needle, the guidewire, or the lavage catheter. Puncture of the bladder and stomach may be avoided by placing a Foley catheter and nasogastric tube, respectively, prior to performing the DPL.[19,24] A punctured bladder is usually noted by obtaining urine during syringe aspiration or by lavage fluid exiting through the Foley catheter. Removing the lavage catheter and continuing Foley catheter drainage for 24 to 48 hours will treat this complication.[14] Puncture of the small bowel, colon, and their mesenteries may occur.[11,18,19,41,45] Puncture of blood vessels (e.g., ranging from mesenteric vessels to iliac vessels) has been described.[1] These latter complications will usually result in a positive DPL based on bleeding or return of enteric contents and are repaired at laparotomy.

Some advocate using the abdominal wound to insert the lavage catheter and not making a fresh entry site.[46] **This is not recommended.** This can result in false-positive results from blood in the wound tract, it can increase the potential for infection, the wound may be too far away that the catheter cannot reach into the pelvic recesses, and the lavage catheter could track through injured organs.

The lavage fluid may be instilled into the abdominal wall or the retroperitoneum on occasion.[1,19] This may result in a false-positive lavage, a false-negative lavage, or more commonly an inadequate lavage fluid return. This complication requires no specific treatment other than recognition. The body will reabsorb the fluid over time.

SUMMARY

The diagnostic peritoneal lavage is a well-described procedure for determining the need for a laparotomy after trauma. It has undergone several modifications since its initial description over 50 years ago. The procedure may be performed using a closed, semi-open, or open technique depending on the patient's history and associated injuries. All three techniques are safe, accurate, and easily performed. Several criteria may indicate a positive result, and knowledge of these criteria is important in the evaluation of the DPL effluent.

It is important that a DPL be performed after informed consent (if feasible) and using strict sterile technique because this is an invasive procedure. There is a small risk of complications and missed injuries necessitating the close observation of the patient with a negative DPL.

The DPL remains one of the most useful tests in the patient with abdominal trauma.[26] The DPL remains the test of choice in the patient with penetrating abdominal trauma and in select patients with blunt abdominal trauma despite advances in imaging technology.

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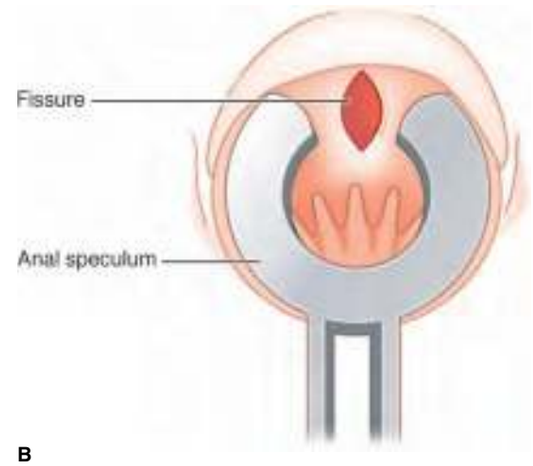
A



B

FIGURE 85-1. The anal fissure. ***A.*** Picture of an anal fissure. (Used with permission of Thieme Medical Publishers.) ***B.*** Illustration of an anal fissure.

85 Anal Fissure Management

Eric F. Reichman

INTRODUCTION

An anal fissure, or fissure-in-ano, is one of the most common anal disorders seen by physicians. It is a linear tear or crack that extends into the anoderm from the mucocutaneous junction to the dentate line (**Figure 85-1**). An anal fissure usually results from the passage of hard stool that traumatizes and tears the anoderm. Frequent bowel movements with diarrhea can cause similar "cracks" that eventually result in fissures. Anal fissures are often seen in infants but primarily are a condition of young and middle-aged adults.[1-3] It is the most common cause of acute-onset painful rectal bleeding in adults and in the first year of life. Thus, some form of "trauma" results in anal sphincter spasm that causes ischemia.[4,5]

A fissure may be acute or chronic, occur at any age, and affect both genders equally. It is the most common cause of rectal bleeding in infants. Fissures are typically a few millimeters long and occur primarily (85% to 90%) in the posterior midline.[5] The remaining 10% to 15% are found in the anterior area.[5-8] There is a slight gender difference, with 1% to 7% of anal fissures found anteriorly in men and up to 12% anteriorly in women.[9] Atypical locations (e.g., lateral) account for 1% and suggest the presence of an underlying disease such as anal cancer, Crohn's disease, HIV, leukemia, previous anal surgery, syphilis, tuberculosis, and other infections.[5,10]

ANATOMY AND PATHOPHYSIOLOGY

The anal canal begins at the level of the anorectal ring and extends distally for 4 cm to the anal verge. The internal anal sphincter and external anal sphincter muscles surround the anal canal. The internal anal sphincter muscle is a continuation of the involuntary layer of circular smooth muscle of the rectum that begins at the anorectal ring. It is approximately 2.5 to 4.0 cm long and 2 to 3 mm thick. The internal anal sphincter is not under voluntary control and is continuously contracted to prevent unplanned stooling. It is contracted at rest so that the lower margin can be palpated 1 to 2 cm below the dentate line in the intersphincteric groove. The internal anal sphincter muscle supplies up to 60% of the resting tone of the anus.[11] The external anal sphincter muscle is an elliptical cylinder of voluntary striated muscle tethered to the coccyx and surrounding the anal canal. It merges proximally with the puborectalis and levator ani muscle. It is voluntarily controlled. Columnar epithelium lines the upper anal canal while the lower anal canal is lined by squamous epithelium. The transitional zone lies between the two different types of mucosa. The anoderm is a thin layer of stratified squamous epithelium around the anus, distal to the dentate or pectinate line, that lacks sweat glands and hair follicles. This area is richly endowed with cutaneous sensory nerve endings.

It is hypothesized that anal fissures usually occur in the posterior midline secondary to a decreased vascular supply causing ischemia or a decreased number of external sphincter muscle fibers predisposing the posterior area to a weakness.[12] Constipation or a hard bowel movement causes a tear in the anoderm that causes pain. The tear often results in a vicious cycle of injury by exposing the underlying internal sphincter muscle. This results in spasms of the muscle which cause severe pain, results in further separation of the fissure edges, and possible further tearing during subsequent stool passage. A delay in wound healing may result and can ultimately lead to a chronic anal fissure.

Ischemia may contribute to the cycle of injury. The posterior midline, being the primary location for anal fissures, has less than half the blood flow compared to other quadrants in the anal canal.[13,14] Individuals with chronic anal fissures have higher anal pressures than controls or patients with other colorectal disorders due to increased internal anal sphincter tone which results in blood flow reduction as well.[15] These factors lead to the rationale for the use of topical nitroglycerin in treating anal fissures.

A tight anal sphincter is another hypothesis for the etiology of a fissure. Anal manometry in patients with fissures reveals an elevation of the resting anal canal pressures and infrequent spontaneous relaxation of the internal anal sphincter muscle.[15] These high pressures can impede blood flow. The anodermal blood supply passes through the internal anal sphincter muscle and increased resting pressure may result in an ischemic ulcer or fissure.

An acute anal fissure has the appearance of a clean longitudinal tear in the anoderm with minimal inflammation (**Figure 85-1**). A chronic anal fissure is deeper, and exposed internal anal sphincter muscle fibers may be seen at its base. A skin tag or sentinel pile is usually seen externally and a hypertrophied anal papilla may be found at its upper aspect. Patients with a chronic anal fissure may also complain of anal discharge, pruritus, or "a lump." Many people complaining to their physicians of bright red blood per rectum and anal pain think they are suffering from "hemorrhoids." A good history and physical examination can confirm the diagnosis of an anal fissure. These patients will typically describe a sharp, burning, or shearing pain with defecation that lasts for a few moments up to an hour afterward. Some people will complain of a chronic ache that is exacerbated with a bowel movement. The prolonged pain is usually attributed to internal anal sphincter muscle spasm. Bright red blood is usually seen on the stool or toilet paper. Occasionally, a few drops of blood will fall into the toilet bowl.

Patients can usually describe the initial event that triggered the fissure as either an episode of constipation with a hard bowel movement or diarrhea. Most patients with a painful anal fissure will not tolerate a digital rectal examination or anoscopy. A close inspection of the anus can be performed after reassuring the patient and gently pulling the buttocks apart. An anal fissure with a sentinel pile, an abscess, or a thrombosed external hemorrhoid may also be seen. The application of anesthetic jelly may be required to examine the patient with an acute, painful anal fissure. Palpating the anal fissure with a gloved finger or cotton swab will reproduce the patient's pain.

INDICATIONS

Anal fissures are extremely painful and uncomfortable for the patient. They can cause poor job attendance or performance. All anal fissures should be treated when they are identified. Treatment relieves anal sphincter spasm and alleviates the ischemia to allow healing. The anal sphincter spasm can be relieved in the Emergency Department by injection, mechanical, neurotoxic, pharmacologic, or surgical methods. **Initial therapy is conservative, followed by topical medications, injection therapy, and then surgery.** Surgical treatment is warranted for patients for whom nonoperative therapy fails, who have "chronic" anal fissures, or who experience severe anal pain.

CONTRAINDICATIONS

A patient with perianal Crohn's disease or ulcerative colitis is a relative contraindication to performing a lateral internal sphincterotomy. Medical management is advocated to initially treat these fissures associated with inflammatory bowel disease followed by the judicious use of an internal sphincterotomy.[16] A patient who has had multiple fistulotomies in the past or a sphincteroplasty should have their anal sphincter evaluated by anal ultrasound or manometry. The patient may have marginal sphincter function, and an internal sphincterotomy can render them completely incontinent. A sphincterotomy should be performed only by a Colorectal Surgeon in these difficult patients.

EQUIPMENT

ANAL ANESTHESIA

- Povidone iodine or chlorhexidine solution
- 10 mL syringe
- 27 gauge needle, 2 inches long
- Local anesthetic solution with epinephrine
- 4×4 gauze squares
- Sodium bicarbonate solution

LATERAL INTERNAL SPHINCTEROTOMY

- Anal speculum
- #15 scalpel blade on a handle
- Metzenbaum scissors
- Forceps
- 4–0 chromic gut sutures

MISCELLANEOUS (DEPENDING ON TECHNIQUE)

- Topical nitroglycerine (0.2% to 2.0%)
- Topical nifedipine (0.2% to 2.0%)

FIGURE 85-2. The prone jackknife position. Note that the lower abdomen is not touching the edge of the table.

- Topical captopril (0.5%)
- Topical diltiazem (2%)
- Botulinum toxin
- #11 scalpel blade

DRESSING

- 4×4 gauze squares
- Gelfoam
- 2 inch adhesive tape

PATIENT PREPARATION

Explain to the patient and/or their representative the risks, benefits, complications, and options for treatment. The following discussion applies to the injection or surgical treatment of an anal fissure. Explain the postoperative care if a surgical technique is to be performed. Obtain an informed consent for the procedure. Undress the patient from the waist down. Place the patient prone or in the prone jackknife position (**Figure 85-2**). Tape the buttocks apart and to the procedure table to gain better exposure of the anus (**Figure 85-3**). Clean any dirt and debris from around the anus. Apply povidone iodine or chlorhexidine solution and allow it to dry. Place drapes to delineate a sterile field.

Perform an anal block. Mix 10 mL of local anesthetic solution with epinephrine in a syringe with 1 mL of sodium bicarbonate. This will decrease the burning sensation upon injection of the anesthetic solution. Inject the anesthetic solution into the subcutaneous tissue circumferentially around the anus, under the fissure, and laterally to anesthetize the pudendal nerves. The use of procedural sedation (Chapter 159) is recommended but not required.

TECHNIQUES

Begin the examination with a slow, gentle separation of the buttocks. This usually provides adequate visualization of the clean slit-like, acute anal fissure. The chronic fissure will appear similar to a deep ulcer with skin tags and enlarged anal papillae. Chronic fissures can be confused for external hemorrhoids when accompanied by a sentinel pile, which is a swollen external tag of skin at the base of the fissure. Test for adequate anesthesia. Inspect the lateral areas for an avascular area, usually on the right side between the right posterior

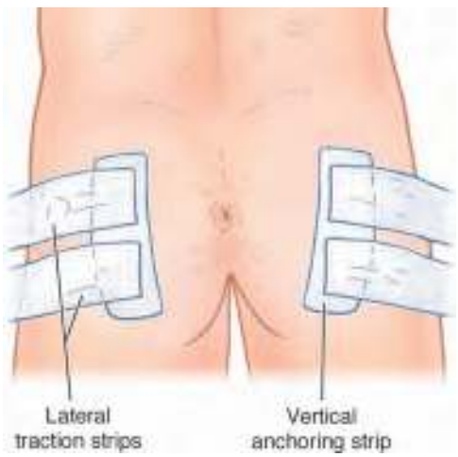


FIGURE 85-3. Taping the buttocks open in the prone patient allows for unobstructed access. The lateral traction strips are taped to the examination table or gurney.

FIGURE 85-4. The single-use disposable anal speculum. (Photo courtesy of OBP Medical.)

and right anterior hemorrhoid complexes. Palpate the intersphincteric groove in the avascular area. It is a palpable depression between the caudal ends of the internal and external anal sphincter muscles.

Anoscopy can be performed for enhanced visualization when necessary. Most patients have too much pain and involuntary sphincter spasm, which precludes a digital or anoscopic exam. Some require an exam under anesthesia. A proctoscopic exam may assist for ruling out secondary causes.

OBP Medical (Lawrence, MA) has recently developed a single-use, latex-free, nonsterile (clean), and disposable anal speculum (**Figure 85-4**). It is fully assembled with a built in LED light source and ready for use. Just pull the tab from the anal speculum to activate the light source for approximately 20 minutes. The advantages include no cleaning of specula, no worrying about contamination, no charger to plug into an outlet or find, no external light source, and no cords.

Atypical appearances of anal fissures, that might be eccentrically located, should prompt alternative diagnoses such as inflammatory bowel disease, ulcerative colitis, and Crohn's disease. Sexually transmitted infections such as chlamydia, gonorrhea, herpes, syphilis, and HIV, as well as tuberculosis, anal neoplasms, and sickle cell disease can present with anal fissures. If rectal bleeding is a prominent finding, endoscopy plus sigmoidoscopy or colonoscopy should be performed.

Management of an anal fissure begins conservatively.[4,17] If unsuccessful, the next steps include topical preparations and injection therapy. A surgical sphincterotomy is the procedure of choice in refractory cases and has a 95% success rate.[18]

FOUR-FINGER ANAL STRETCH

In the past, a four-finger anal stretch technique was performed. This involved putting the index and long fingers of both hands into the patient's anal canal and pulling the anal canal forcibly open. This would often cause an uncontrolled tear in the internal anal sphincter muscle. Although patients had relief of their symptoms, 40% developed a recurrence of the fissure, and a significant proportion had some level of incontinence. **This technique is no longer recommended and should never be performed**. It is included only for the sake of completeness.

CONSERVATIVE MANAGEMENT

Most remedies for an anal fissure aim to alleviate internal sphincter hypertonia and anal pain. A trial of conservative treatment is employed for acute fissures and chronic fissures with mild to

moderate symptoms. This consists of bulk fiber supplements, stool softeners, a high-fiber diet, increased oral intake of water, sitz baths, heat, and topical anesthetics.[4,19,20] The large, soft, bulky stools that result gently dilate the anal sphincter. Bulking agents and mild laxatives are 87% effective when used for 3 weeks.[5] The combination can reduce recurrences if they are continued.[5] The topical anesthetics and sitz baths in warm water after bowel movements will cleanse the area, alleviate the anal pain, and relieve internal anal sphincter muscle spasm. Topical anesthetics such as lidocaine gel may be soothing but are not more effective than fiber and sitz baths alone.[21] Oral pain medications and muscle relaxants such as diazepam may be helpful. Suppositories are not recommended because they ascend to the rectal ampulla and do not effectively treat the problem within the anal canal. If an initial trial of conservative therapy for 4 weeks fails, the patient can undergo pharmacological therapy, injection therapy, or operative treatment.

A relatively new product is LEVORAG Emulgel (THD SpA, Italy) and which is available by prescription. It consists of a mixture of many ingredients in a 3.5 mL single-use tube with a built-in applicator. It acts like an emollient, lubricant, film-forming gel, and protectant that soothes when used twice a day for 3 weeks. Emulgel contains no anesthetics, cortisone, dyes, fragrances, or heavy metals. A pack or carton consists of 20 single-dose tubes.

TOPICAL PHARMACOLOGICAL TREATMENT

Nitric oxide has been identified as a chemical messenger mediating relaxation of the internal anal sphincter muscle. Patients treated with 0.2% glyceryl trinitrate ointment 2 to 3 times a day for 6 weeks to their lower anal canal (near the fissure) daily exhibited relief of their anal pain, reduced maximal anal resting pressure, and increased anodermal blood flow as measured by laser Doppler flowmetry.[22] Topical nitroglycerin increases blood flow to the site, which is thought to result in less internal anal sphincter pressure, less pain, and a resultant improvement in healing rates in acute and chronic anal fissures.[22,23] After 8 weeks, 68% of patients treated with glyceryl trinitrate had healed their fissures. Some studies have shown no significant difference in healing rates when compared to placebo.[24] However, up to 75% of patients will have an adverse reaction, mainly a headache unresponsive to mild pain relievers or develop a tolerance to the nitrate.[25] Up to 50% of patients will have a recurrence of an anal fissure after the initial anal fissure has healed with nitroglycerin treatment.[26,27] The overall efficacy of nitroglycerine ranges from 47% to 86%.

Oral nifedipine, topical nifedipine (0.2% to 2.0%), oral diltiazem, topical diltiazem (2%), topical captopril (0.5%), and bethanechol have also been studied for the treatment of anal fissures.[5,28-38] They were all found to be comparable to topical nitroglycerin and with fewer side effects. The US Food and Drug Administration approved 0.4% nitroglycerine ointment (Rectiv) for the treatment of anal fissures in 2012.[18,39] The cost is approximately $50.00 for a 30 gm tube.

Side effects such as headache, primary, may occur 10 minutes after application of topical nitroglycerin and typically last less than 30 minutes. Orthostatic hypotension and syncope have also been described in patients.[18] Use of a glove or finger cot during the application of topical pharmacologic agents can prevent some systemic absorption and side effects. Patients taking phosphodiesterase type 5 (PDE5) inhibitors for erectile dysfunction (i.e., Cialis, Levitra, or Viagra) should not receive nitroglycerin-containing medications within 24 hours of their use due to the potential for life-threatening hypotension.[18,39] After 24 hours, a lower concentration of nitroglycerin (e.g., 0.2% to 0.4% glyceryl trinitrate) is recommended compared to standard 2% ointments.

Topical nifedipine gel in a concentration of 0.2% used every 12 hours for 3 weeks has been successfully used to decrease anal sphincter pressures and help heal fissures.[40] No systemic side effects or significant anorectal bleeding was observed in 141 patients treated with topical nifedipine.[40]

BOTULINUM TOXIN

Botulin toxin is another pharmacological approach to the treatment of a chronic anal fissure. Botulinum toxin inhibits the release of acetylcholine from presynaptic nerve endings and has been shown to be a beneficial treatment for chronic anal fissures.[5,41] Injection close to each side of the fissure improves healing rates.[41] Inject 20 units of botulinum toxin A (Botox; Allergan, Irvine, CA) or 0.4 mL (50 U/mL) into the internal anal sphincter muscle on either side of the fissure using a 27 gauge needle. This technique has few side effects. Injection of high-dose (100 units) botulinum toxin A has also been used successfully.[4] This can alleviate anal pain, decrease anal sphincter pressure, and promote healing.[42] Unfortunately, this can result in some form of temporary incontinence for 2 to 6 months.[18,41,43] The cost associated with the one-time injection is approximately $400.00. If the underlying pathophysiology that led to the anal fissure is not addressed and corrected, a high rate of recurrence exists.[4,17,44] The next step if botulinum toxin A injection fails is the sphincterotomy.

LATERAL INTERNAL SPHINCTEROTOMY

The mainstay of operative therapy is a lateral internal sphincterotomy. This technique is curative in over 95% of patients.[18,45] This procedure can result in a transient incontinence of flatus and stool in most patients.[46] Unfortunately, approximately 15% of patients will be left with some form of permanent minor incontinence of flatus, stool smearing, and stool.[27] The technique divides the internal anal sphincter muscle between the dentate line and the anal verge. A meta-analysis of 2727 patients undergoing operative techniques for anal fissures revealed no significant difference between open versus closed lateral internal sphincterotomy for the persistence of fissure or incontinence.[47] However, significant differences were found when anal stretch was compared to all forms of sphincterotomy. The recurrence of an anal fissure after a sphincterotomy can often be cured by a re-do sphincterotomy.[48]

A sphincterotomy requires some form of anesthesia. This is usually accomplished by an anal block injection. In some cases, usually due to anxiety and/or pain, the patient will require procedural sedation (chapter 159) or brief general anesthesia.

CLOSED LATERAL INTERNAL SPHINCTEROTOMY

The closed lateral internal sphincterotomy technique is preferred by some physicians (**Figure 85-5**). Its advantage is that a smaller wound is created. Unfortunately, this is a blind procedure and can result in injury to the patient and the physician.

Prepare the patient as described previously. **Place a well-lubricated anal speculum into the anal canal.** Open the speculum to provide a slight stretch to the anal sphincter muscles. Expose and view the left or right posterolateral quadrant of the anal canal. Place a gloved finger into the anal canal and palpate the internal aspect of the internal anal sphincter muscle. **Perform the remainder of this procedure while carefully palpating the course of the scalpel blade with the gloved finger in the anus.**

Insert a #11 scalpel blade horizontally through the skin and into the intersphincteric groove (**Figure 85-5*A***). Advance the scalpel blade into the plane between the internal and external anal sphincter muscles and up to the level of the dentate line (**Figure 85-5*A***). Direct the scalpel blade medially by turning it 90° (**Figure 85-5*B***). Slowly divide the full thickness of the internal anal sphincter muscle while withdrawing the scalpel blade. **Do not cut the anoderm**.

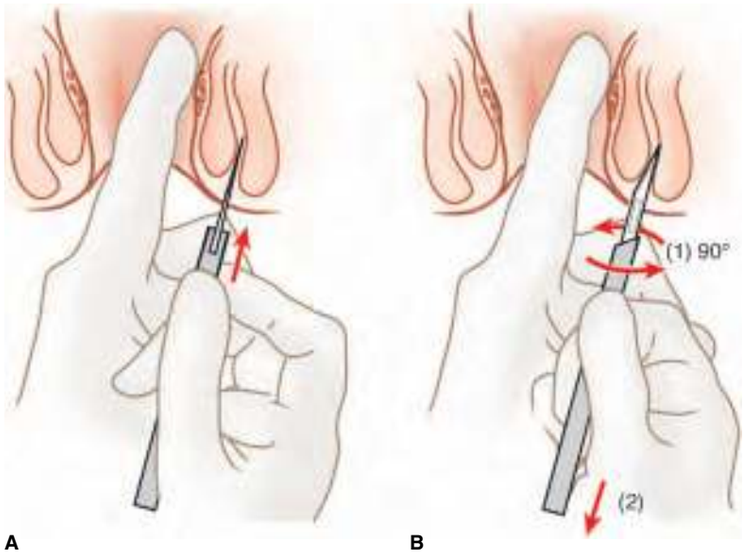


FIGURE 85-5. The closed lateral internal sphincterotomy. ***A.*** The #11 scalpel blade is inserted horizontally between the internal and external anal sphincter muscles. ***B.*** The scalpel blade is turned 90° (***1***) then withdrawn (***2***) to transect the internal anal sphincter muscle.

Withdraw the scalpel from the anal canal. Remove the anal speculum. Close the incision site with one or two size 4–0 chromic gut interrupted sutures. Pack the anal canal with 4×4 gauze squares for 10 to 15 minutes to aid hemostasis and prevent the formation of a hematoma.

OPEN LATERAL INTERNAL SPHINCTEROTOMY

The open technique provides a clear exposure to the anatomy of the region. This is especially important for those less experienced with the procedure. It avoids the potential for injury to the physician when compared to the closed technique.

Prepare the patient as described previously. **Place a well-lubricated anal speculum into the anal canal.** Open the speculum to provide a slight stretch to the anal sphincters and view the fissure (**Figure 85-6*A***). Rotate the speculum 90° to expose and view the left or right posterolateral quadrant of the anal canal (**Figure 85-6*B***). Place a gloved finger into the anal canal and palpate the internal aspect of the internal anal sphincter muscle.

Make a 1 cm longitudinal incision through the skin and subcutaneous tissue, between the dentate line and the anal verge (**Figure 85-6*B***). This will center the edge of the internal anal sphincter muscle in the middle of the incision. Slide a scissors submucosally along the white internal anal sphincter muscle until the tips are at the level of the fissure, but not beyond the dentate line. Spread the arms of the scissors once to open the jaws of the scissors. Repeat this process on the other (deep) side of the internal sphincter muscle. Grasp and elevate the internal anal sphincter muscle with a forceps. Use a scissors to make a cut in the internal sphincter muscle the same length as the length of the anal fissure (**Figure 85-6*C***).[49] **Do not completely transect the internal anal sphincter muscle. Preserve at least one-third of the proximal internal sphincter muscle intact.**[49]

Pack the anal canal with 4×4 gauze squares to apply pressure to the area for 10 to 15 minutes to aid in hemostasis and to prevent

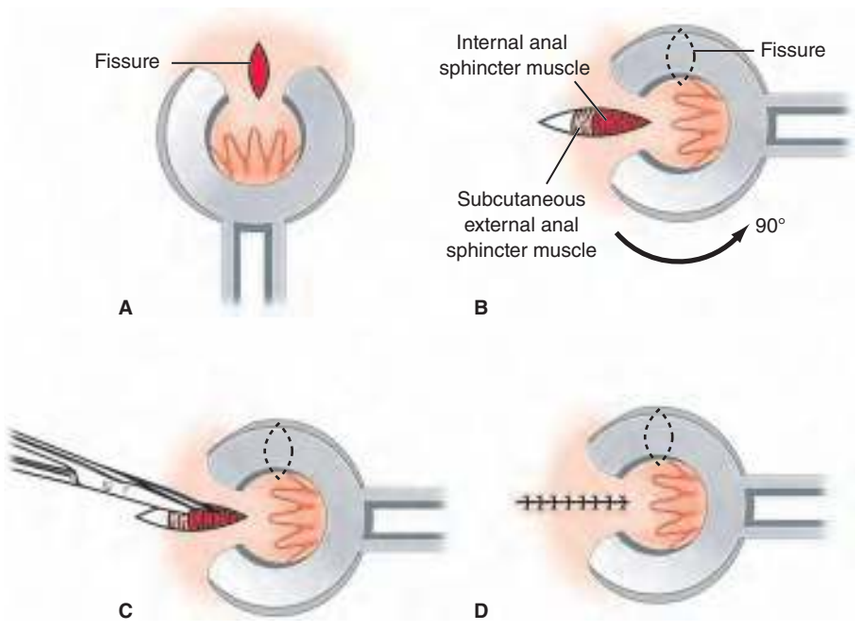


FIGURE 85-6. The open lateral internal sphincterotomy. ***A.*** The anoscope is inserted so that the anal fissure is visible. ***B.*** The anoscope has been rotated 90°. An incision has been made through the anoderm and subcutaneous tissue to expose the underlying anal sphincter muscles. ***C.*** The internal anal sphincter muscle is partially transected with a scissors. ***D.*** Closure of the incision.

the formation of a hematoma. Suture the incision closed with size 4–0 interrupted chromic gut (**Figure 85-6*D***). Place a small piece of Gelfoam over the wound. Remove the anal speculum. Dress the incision site with 4×4 gauze squares.

ALTERNATIVE TECHNIQUES

There are several alternatives used to cure an anal fissure. Injection of a sclerosing agent in the office has had good results.[50] There is not enough information at this time to recommend the injection of a sclerosing agent in the Emergency Department. The combination of a fissurectomy with the injection of botulinum toxin A for a chronic anal fissure is as effective as a sphincterotomy.[51] Reserved for a failed sphincterotomy is either a fissurectomy with primary closure or a fissurectomy with a flap repair (e.g., V-Y flap or island flap).[4,48]

AFTERCARE

Instruct the patient to remove the dressing in 12 to 24 hours or before a bowel movement. Warm sitz baths four times a day will keep the area clean and alleviate any pain. Prescribe a high-fiber diet with oral stool softener supplements to keep the stools soft and bulky. Oral analgesics such as acetaminophen or nonsteroidal anti-inflammatory medications with supplementary narcotic analgesics will often ease the immediate and postoperative pain. The patient should follow up in 1 to 2 weeks for reevaluation. The patient should immediately return to the Emergency Department if a fever, severe pain, or bleeding from the incision site develops.

COMPLICATIONS

Many complications have been associated with a lateral sphincterotomy. Itching, burning, bleeding, delayed wound healing, and constipation are minor problems. The patient may complain of mucus drainage or fecal soiling during the healing phase. Bulking agents or a high-fiber diet may help decrease the drainage. Recurrent fissures occur in about 8% of patients, 66% of which heal with conservative treatment. Up to 45% of patients may experience some degree of incontinence, but only 3% of patients may have their life affected.[52] Incontinence can persist.[27] A fecal impaction, abscess, or hemorrhage can become significant. Enemas are useful for the constipated patient. An abscess should be incised and drained, preferably in the Operating Room for adequate anesthesia to completely explore the wound and debride any necrotic tissue. A subcutaneous fistula can develop if the anoderm is violated during the sphincterotomy and not recognized. This is easily taken care of by doing a fistulotomy of this superficial skin bridge. The physician may be injured while performing the closed technique. This technique should be reserved for those with experience and a patient who is sedated to decrease the chances of injury.

SUMMARY

There is no perfect treatment for an anal fissure. Anal pain with bleeding due to an anal fissure is initially treated conservatively with a high-fiber diet, stool softeners, and warm sitz baths. Most patients will respond to these measures. A few will fail conservative therapy and need pharmacological or operative therapy. A sphincterotomy remains the best curative procedure, but is also the most invasive. The goal of all the different regimens is to decrease anal pain, reduce anal sphincter spasm, and heal the fissure.

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86 External Hemorrhoid Management

Katherine Holmes, Michael Balkin, and Hao Wang

INTRODUCTION

Thrombosed external hemorrhoids consistently cause patient pain and embarrassment. The etiology is still unclear. The mainstay of treatment is excision. **It is important to remember that excision is performed to alleviate or palliate pain and shorten the course of the disease.** The natural history of an untreated thrombosed external hemorrhoid is to rupture and spontaneously evacuate the clot or to resorb the clot over time. Therefore, treatment should give the maximum amount of pain relief with the least chance of complications. To make this decision, it is important to take a thorough history from the patient and include questions about the time course, severity, improvement or deterioration, and what has worked or failed in the past. **It is also important to perform a physical examination to rule out prolapsed grade IV internal hemorrhoids, perianal abscesses, and other perianal masses.**

ANATOMY AND PATHOPHYSIOLOGY

External hemorrhoids fall into three main groups: left lateral, right anterior, and right posterior (**Figure 86-1**).[1] They are covered with anoderm and visible on the outside of the anal canal. They are composed of a venous plexus mixed with connective tissue. External hemorrhoids drain into the middle and inferior rectal veins that terminate into the internal iliac and femoral veins, respectively. **External hemorrhoids do not prolapse like internal hemorrhoids. They engorge and thrombose to cause significant pain and discomfort. It will not benefit the patient to try to reduce an external hemorrhoid since their normal location is mostly outside the anal canal and reduction will not remove the clot. External hemorrhoids are never covered with mucosa.** The overlying skin may appear to look shiny, swollen, gangrenous, or like an orange peel mimicking the look of mucosa.

It is important to differentiate internal versus external hemorrhoids. Internal hemorrhoids originate above the dentate line, lack sensation, and are covered with rectal mucosa. Prolapsed internal hemorrhoids are painless unless they become gangrenous, infected, strangulated, or thrombosed. External hemorrhoids originate below the dentate line, have sensory innervation, and are covered with squamous epithelium that matches the surrounding skin.

The patient usually complains of a history of sudden onset of pain and swelling. The exact cause of thrombosed external hemorrhoids is unknown.[2] Some studies have shown correlations between thrombosed external hemorrhoids and constipation, excess straining during defecation, and strenuous exercise (e.g., bicycling, jogging, or weight lifting). Since this disease process affects younger individuals more frequently, it seems that intense physical activity plays a more prominent role than does constipation or straining to defecate.

External hemorrhoids can be diagnosed when a patient complains of sudden onset of pain, swelling, and usually no bleeding.

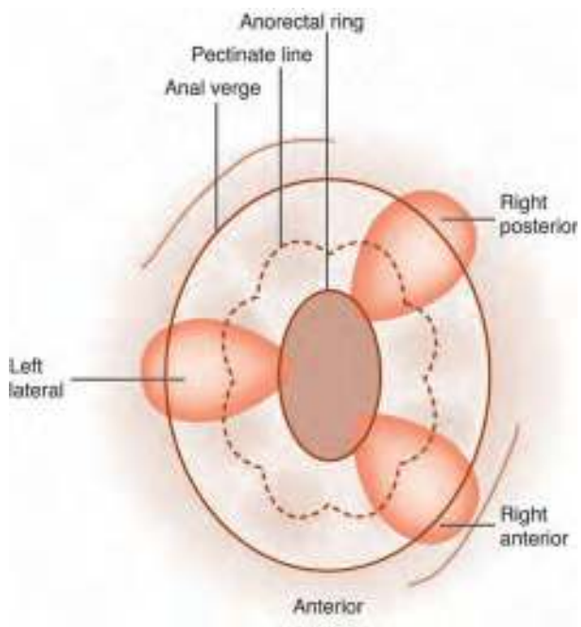

FIGURE 86-1. The position of the three main groups of external hemorrhoids.

A

B

C

D

FIGURE 86-2. Thrombosed external hemorrhoid management. ***A.*** The thrombosed external hemorrhoid. ***B.*** Injection of local anesthetic. ***C.*** The incision is made. ***D.*** Expelling the blood clot. (Used with permission from reference 3.)

The physical examination will reveal a tensely swollen area covered with anoderm. The swelling will be visible by gently spreading the buttocks and inspecting the area near the anal canal. They have a bluish coloration, especially in patients with little skin pigmentation, and almost no redness (**Figure 86-2*A***). The swelling is abrupt and looks like placing a marble under a sheet and tucking in the edges. **This differentiates the appearance of a thrombosed external hemorrhoid from the appearance of an abscess that would have erythema and gently sloping sides from edema.**

INDICATIONS

The primary indication for the excision of a thrombosed external hemorrhoid is pain.[3-8] The excision should occur as soon as possible from the onset of pain and is usually not indicated after the fourth day when the clot is already being absorbed and the risks associated with surgery may outweigh the benefits.[9] Improving pain suggests that medical management is the more appropriate method of care as thrombosed external hemorrhoids will spontaneously resolve in

a few weeks with no complications. Medical management includes bulk-forming fiber supplements (e.g., Citrucel [methylcellulose], Fibercon [polycarbophil], Metamucil [psyllium], and Benefiber [wheat dextrin]), sitz baths, stool softeners (e.g., Colace or docusate sodium), topical corticosteroids (e.g., Proctofoam HC, Proctosol HC, Preparation H, or Anusol HC), 0.2% glyceryl trinitrate (GTN), nifedipine, and over-the-counter hemorrhoidal astringents containing witch hazel (e.g., Preparation H pads and Tucks pads).[1,5,6,10] It is important to note that thromboses lasting for less than 72 hours should be evaluated for excision.[3,4,7] Surgery significantly reduces the disease time course and severity. There is a small subgroup of patients who do not seem to improve with medical management and will require excision late in the course of the disease. Surgical excision has a lower frequency of recurrence and a longer time interval to recurrence than conservative treatment.[11] The thrombosis to be excised should involve 1 or 2 hemorrhoids at most.

CONTRAINDICATIONS

There are several contraindications to the Emergency Department incision and drainage of a thrombosed external hemorrhoid.[12] Grade IV internal hemorrhoids associated with thrombosed external hemorrhoids, circumferential hemorrhoids, or very large thrombosed external hemorrhoids should be managed by a Surgeon in the Operating Room using electrocautery or suture ligation to control hemorrhage and provide anesthesia. Patients taking anticoagulants require meticulous care and possible reversal of the coagulopathy. An allergy to local anesthetic agents will require a trip to the Operating Room to excise the hemorrhoid. **Painless masses are never thrombosed external hemorrhoids and require evaluation by a Surgeon.** Draining external hemorrhoids should be followed-up in 24 hours by a Surgeon. Patients who are unable to cooperate with the procedure may require procedural sedation (Chapter 159) or general anesthesia. A Surgeon should manage patients who have thrombosed external hemorrhoids and also have inflammatory bowel disease, anorectal fissures, anal fistulas, cirrhosis, perianal infections, portal hypertension, rectal prolapse, anorectal tumors, or who are immunocompromised. Patients with external hemorrhoids that are not thrombosed usually require no treatment. They may need medical care and appropriate referral for their anal pain. Discuss the management of pregnant patients with an Obstetrician and Colorectal Surgeon before incising a thrombosed hemorrhoid.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Local anesthetic solution with epinephrine
- 5 mL syringe
- 25 gauge needle
- 18 gauge needle
- #11 scalpel blade on a handle
- 4×4 gauze squares
- 2–0 absorbable sutures (e.g., Vicryl, Dexon, chromic gut, or plain gut)
- 3–0 absorbable sutures (e.g., Vicryl, Dexon, chromic gut, or plain gut)
- Small dissecting scissors
- Small grasping forceps
- 2 inch adhesive tape
- Tincture of benzoin
- Silver nitrate applicator sticks

FIGURE 86-3. The prone jackknife position. Note that the lower abdomen is not touching the edge of the table.

- Moisture-resistant drapes
- Post-hemorrhoidectomy pack or 18 gauge Foley catheter

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. It is also important to explain what to expect after the procedure. Obtain an informed consent for the procedure. This procedure may cause the patient significant anxiety and embarrassment. A thoughtful and gentle approach is recommended.

It is crucial to position the patient so that the anus is clearly visible and the Emergency Physician can work with both hands.[13] Place the patient in the prone or prone jackknife position with their hips flexed (**Figure 86-3**). Tape the buttocks to the procedure table (**Figure 86-4**). Apply tincture of benzoin to the buttocks and allow it to dry. Place strips of 2 inch adhesive tape on the left and

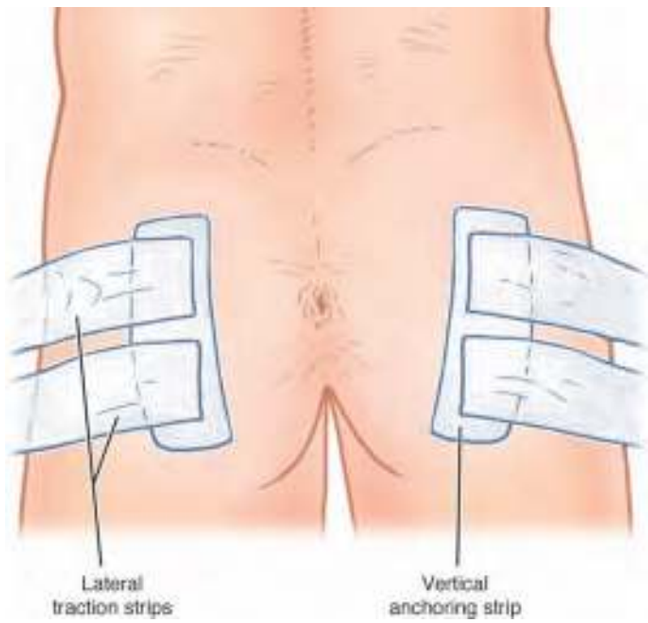

FIGURE 86-4. Taping the buttocks open in the prone patient allows for unobstructed access. The lateral traction strips are taped to the examination table or gurney.

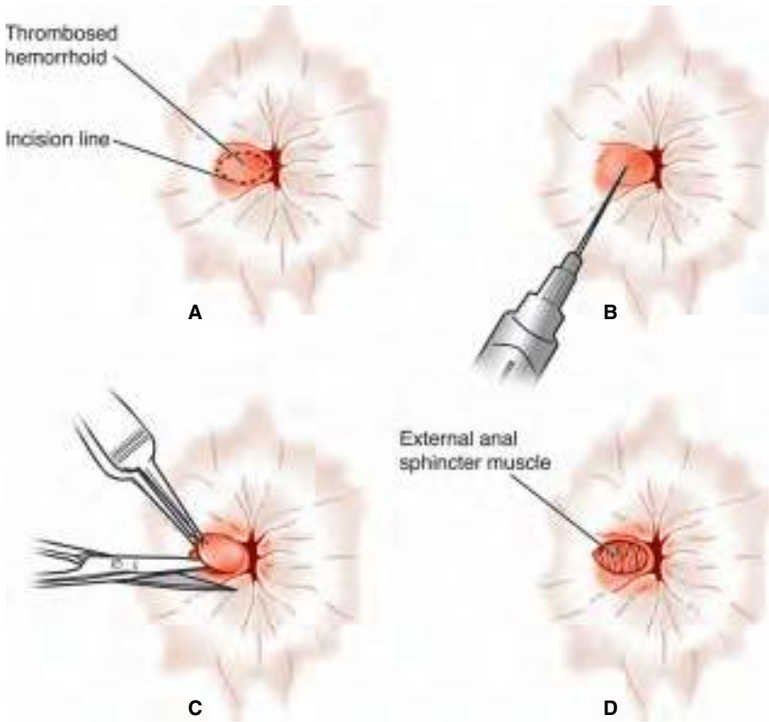

FIGURE 86-5. Excision of the thrombosed external hemorrhoid. ***A.*** The dotted line represents the incision lines to remove the skin and underlying thrombosis. ***B.*** Injection of local anesthetic solution. ***C.*** The skin incision has already been performed. The skin and underlying thrombosis are dissected free with a scissors. ***D.*** The ellipse of skin and the underlying thrombosis have been removed. The fibers of the underlying external anal sphincter muscle are visible.

right cheeks of the buttocks and perpendicular to the anus. Apply lateral and slightly cranial traction with adhesive tape to get the proper exposure. Attach the distal ends of the tape to the procedure table or stretcher to maintain exposure during the procedure. This positioning works best in the Operating Room when the patient is under anesthesia and unlikely to move. Explain to the patient that they must relax and refrain from squeezing their buttocks shut. It is very difficult to stop and reposition the patient to place a suture for hemorrhage control if the patient is poorly positioned and there is difficulty with hemorrhage in the middle of the procedure. Apply drapes to protect the patient and clothing from spills. Clean the anus and surrounding area of any dirt, debris, and stool. Apply povidone iodine or chlorhexidine solution and allow it to dry.

The patient usually is in significant pain. The injection of local anesthetic solution can be excruciating (**Figures 86-2*B* and 86-5*B***). Consider the use of ice packs to the area for 5 to 10 minutes before injection, intravenous analgesics, sedatives, or procedural sedation (Chapter 159). This will be greatly appreciated by the patient.

TECHNIQUE

Determine the area of the incision. **The best pain relief will be achieved if the thrombosis is excised rather than incised.**[14] If the hemorrhoid is very large, one-third or greater of the anal circumference, it is best to excise the middle third of the hemorrhoid leaving as much anoderm as possible to prevent the wound healing with a stricture. Excision can be achieved with two radial incisions starting near the center of the anus and enclosing an ellipse of skin that will be removed with the thrombosis (**Figure 86-5*A***).

Inject local anesthetic solution containing epinephrine starting laterally and injecting medially to and beyond the thrombosed hemorrhoid (**Figures 86-2*B* and 86-5*B***).[9] The injection should include both lines of the planned incision and, if possible, the area medial to the thrombosis. The local anesthetic agent should also cross the midline anteriorly and posteriorly to include nerve fibers crossing over from the opposite side of the anus. Allow 5 minutes for the local anesthetic to take effect. The degree of local anesthesia can be checked by pinprick or by pinching the thrombosed hemorrhoid with forceps.

Make the incisions with a #11 scalpel blade when satisfactory local anesthesia has been achieved (**Figure 86-2*C***). Dissect the ellipse of skin and the underlying clot from lateral to medial with a scissors (**Figure 86-5*C***). **Do not cut the anal sphincter at the base of the wound (Figure 86-5*D*). It is important to remove the entire clot since the purpose of the excision is only to palliate the patient's pain (Figures 86-2*D* and 86-5*D*).** Small clots in or between the sphincter muscles may still cause considerable pain. These can be grasped with fine forceps and removed.

Examine the wound carefully for hemostasis. Localized areas of bleeding can often be controlled with the application of silver nitrate to cauterize the wound. It is the editor's personal belief that there is less discomfort if silver nitrate is used for hemostasis than if one is forced to use suture ligature. A 3–0 absorbable suture can be used in a figure-of-eight formation over the area of continued bleeding. Plain catgut is preferred because the suture will dissolve very quickly. A suture that remains in place for several weeks in this area can sometimes be very uncomfortable for the patient. Dress the wound with three or four gauze squares folded in half and one piece of tape across the buttocks to hold the dressing in place.

AFTERCARE

The patient should leave the dressing in place until the next day or the next bowel movement. It is best to remove the dressing in a sitz bath and replace it with dry 4×4 gauze placed between the buttocks to collect any moisture. Tape is usually not necessary. Encourage the patient to take three or four sitz baths per day and after every bowel movement. Instruct the patient to use warm water and bathe for 20 minutes each time. The sitz bath serves two functions. It keeps the wound clean and helps relax the internal anal sphincter muscle spasm to help relieve the pain. An alternative to wiping after bowel movements and sitz baths is to shower. The shower will gently wash

away any material and not hurt like wiping. Continue the sitz baths and dressing changes until the wound is healed.

Fiber supplements and stool softeners should be continued for at least 6 weeks.[1] Prescribe 1 tablespoon of psyllium products (e.g., Metamucil) with water twice a day to soften and bulk the stool. The goal is to achieve an atraumatic stool that gently dilates the anus as it passes. If the patient is unable to tolerate psyllium, 100 mg of docusate orally once or twice a day can be used to soften the stool but will not provide the bulk.

The patient should feel much less pain after the thrombosed hemorrhoid is excised. The remaining pain can frequently be controlled with the sitz baths. Some form of oral analgesia is required. Acetaminophen or ibuprofen is usually adequate. The use of codeine or opiates has a pronounced constipating effect that could result in painful bowel movements. Do not prescribe narcotic analgesics for more than 24 hours. The patient should return to the Emergency Department if the pain is not improved, if bleeding continues, or if they develop a fever. The wound must be watched for any signs of infection.

COMPLICATIONS

The rate of complications for excision of thrombosed external hemorrhoids is not well reported.[15-18] Reported complication rates for more major anal surgery show bleeding occurs in 1.5% to 4.0% of patients and infection occurs in 2% of patients.[11,15,16] This is a very low rate of infection considering the persistent fecal contamination at the anus. We estimate the complication rate for the excision of a thrombosed external hemorrhoid would be even less.

Any post-hemorrhoidectomy bleeding that is minimal can be managed by the application of local pressure. Moderate to severe bleeding will require surgical cauterization or the insertion of a commercially available post-hemorrhoidectomy pack.[19] This is an accordion-like pack that is inserted through the anoscope and into the anal canal. Pulling the two strings of the pack accordions the pack down into the anal canal to tamponade the bleeding. A Foley catheter may be substituted if the packs are not available. These patients require intravenous analgesics, intravenous sedation, and hospitalization.

The treatment for a patient with an infection in the perianal area who has not had surgery is to open the abscess and place the patient on sitz baths. Infection is very unlikely since the wound is already open from the excision procedure and the patient is taking sitz baths. Broad-spectrum antibiotics for aerobic and anaerobic bacteria should be given to any patient with a postprocedural infection and the wound examined under general anesthesia to rule out any underlying pathology.

Long-term theoretical complications include stricture and incontinence. These are exceedingly rare and can be prevented by not removing too much anoderm and not injuring the underlying external anal sphincter muscle.

The use of a linear incision should be avoided.[20] The stretched skin will close and create a pocket in which a hematoma or abscess can form. Removal of clots through a linear incision is often difficult, inadequate, and may lead to a higher incidence of recurrence. **Always make an elliptical incision.**[20]

SUMMARY

External hemorrhoids may thrombose and cause the patient considerable pain, discomfort, anxiety, and embarrassment. The natural history of this disease is for the clot to drain or resorb without significant long-term morbidity. Excision of the thrombosed external hemorrhoid will provide considerable relief if the patient presents acutely and within 2 to 3 days from the onset of symptoms. It is important to achieve good hemostasis and not damage the underlying external anal sphincter muscle or remove too much anoderm to avoid problems with continence or stricture formation. An elliptical excision is far superior to a linear incision as more clot can be expressed through the linear incision with fewer surgical complications. Fiber supplements and sitz baths should be prescribed rather than surgical excision if the patient presents later in the course of the disease.

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87 Prolapsed Rectum Reduction

Jamil D. Bayram and Eric F. Reichman

INTRODUCTION

Rectal prolapse is an uncommon condition (**Figure 87-1**). It was first described in the Bible (2 Chronicles 21). "You yourself will be very ill with a lingering disease of the bowels, until the disease causes your bowels to come out. . . . After all this, the Lord afflicted Jehoram with an incurable disease of the bowels. In the course of time, at the end of the second year, his bowels came out because of the disease,

FIGURE 87-1. A patient with a rectal prolapse. (Used with permission from Knoop KJ, et al (eds): *The Atlas of Emergency Medicine*, 3rd ed. New York: McGraw-Hill, 2010. Photo contributor: Alan B. Storrow, MD.)

and he died in great pain." The pathophysiology of a rectal prolapse has been evolving since 1543 when Vesalius described the detailed anatomy of the anorectum. Today, three types of rectal prolapse are recognized, and they represent three stages of a continuum.[1]

Rectal prolapse usually affects people at the extremes of age.[2] It is most common in the very young and the elderly. The condition usually manifests itself in children within the first 4 years of life, with the highest incidence occurring in the first year.[3] The gender incidence in children is equal but slightly weighted toward males.[1] The peak incidence in the elderly is approximately between 60 and 70 years of age. It affects primarily elderly women, with a 6:1 ratio of females to males.[4]

ANATOMY AND PATHOPHYSIOLOGY

A rectal prolapse is classified into one of three stages (**Figure 87-2**). An internal prolapse is the prolapse of the upper rectum and sigmoid colon into the rectal ampulla (**Figure 87-2*A***). It is also known as a hidden or occult prolapse. This type of prolapse does not emerge through the anus. Mucosal prolapse is more common in children. It results from the loose attachment of the mucosa to the submucosal layers and an associated weakness of the anal sphincter. A mucosal prolapse is diagnosed by the presence of radial folds and the absence of muscular wall.[4]

If the condition progresses, it leads to the protrusion of part or all layers of the rectum through the anal orifice. If only the mucosa is prolapsed, it is classified as an incomplete prolapse (**Figures 87-2*B* and 87-2*C***). Synonyms include mucosal prolapse and partial prolapse. A complete rectal prolapse occurs when all bowel layers, including the muscular wall, are involved (**Figures 87-2 *D* and 87-2*E***). This condition is also known as a procidentia. The complete rectal prolapse is more common in the elderly. It results from generalized weakening of the pelvic floor and anal sphincter muscles. A complete rectal prolapse is characterized by the presence of concentric folds. A double-thickness muscular wall will be felt upon palpation.[5]

Numerous risk factors are associated with a rectal prolapse.[5-13] These include malnutrition, chronic constipation, excessive straining, and diarrheal disorders such as amoebiasis, giardiasis, and other parasitic infections. Rectal prolapse in children is often idiopathic. However, there is an association with paraplegia, meningomyelocele, and pinworms. Anatomic variations such as a vertical course of the

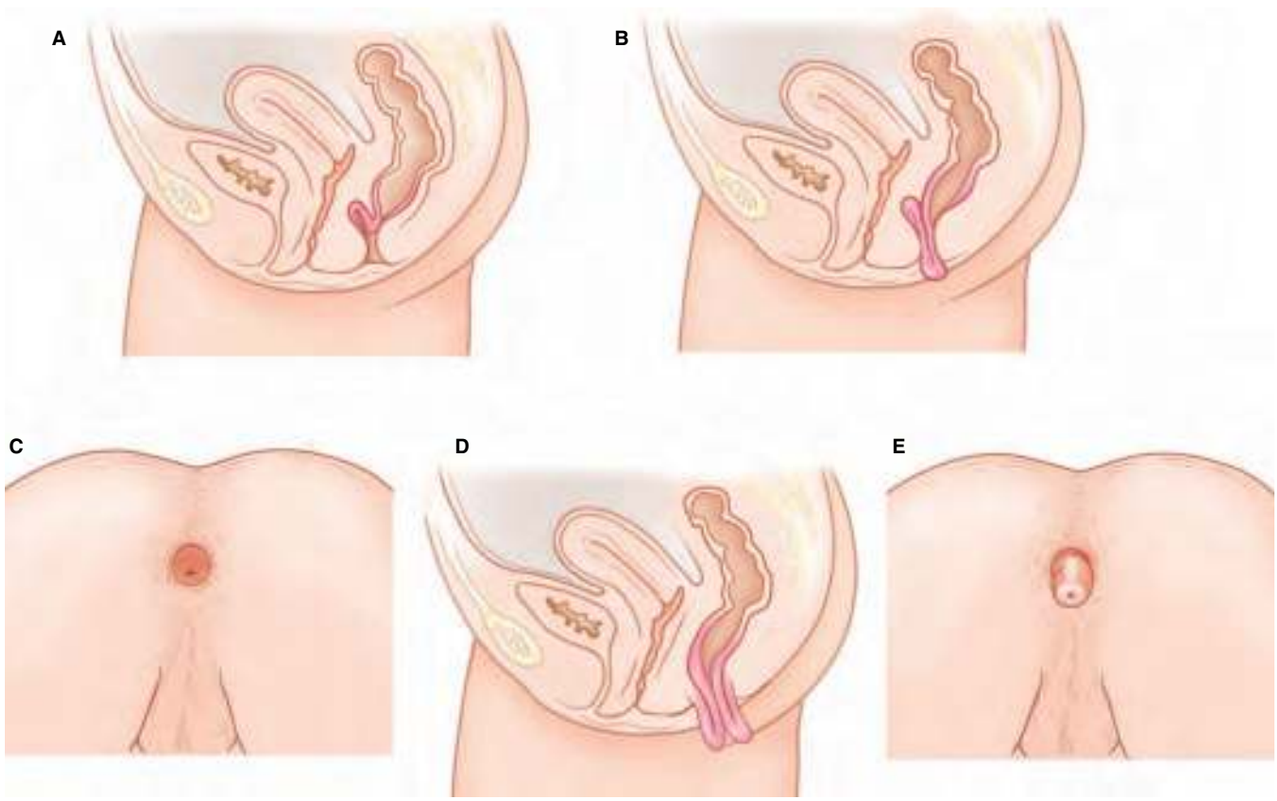

FIGURE 87-2. Types of rectal prolapse. ***A.*** Midsagittal view of the internal, hidden, or occult prolapse. ***B.*** Midsagittal view of the incomplete, mucosal, or partial prolapse. ***C.*** Posterior view of the incomplete, mucosal, or partial prolapse. ***D.*** Midsagittal view of the complete prolapse or procidentia. ***E.*** Posterior view of the complete prolapse or procidentia.

rectum, flat sacrum and coccyx, and lack of levator ani support can also result in a rectal prolapse. Children placed on adult toilet seats for prolonged periods of time may develop a rectal prolapse. One of the most serious risk factors for a rectal prolapse in children is cystic fibrosis.[7] Patients with cystic fibrosis have an 18% incidence of rectal prolapse. Children with no apparent cause for a rectal prolapse should be considered for a sweat chloride test. In the elderly, rectal prolapse is associated with collagen vascular diseases, malignancy, pelvic floor weakness, mental retardation, organic brain syndrome, stroke, chronic psychiatric conditions, and chronic neurologic conditions (e.g., tabes dorsalis, cauda equina, and multiple sclerosis).[6,12] It is important to note that patients with a rectal prolapse often present with no apparent causes. The physician should maintain a high index of suspicion for the risk factors mentioned above.

The diagnosis can be difficult in the early stages when the prolapse remains in the upper canal (i.e., internal or hidden prolapse). The patient may complain of anorectal pain, back pain, discomfort during defecation, difficulty initiating a bowel movement, feeling of incomplete evacuation, tenesmus, pelvic fullness or pain, bloody discharge, or mucoid discharge. At this stage, asking the patient to strain may provoke the prolapse.

The diagnosis becomes easier in a partial or complete prolapse because it protrudes through the anus (**Figures 87-2*C* and 87-2*E***). These patients may complain of an anal mass when they sit, stand, or walk.[1,5] It is usually the parents of young children who note the anal mass during defecation, although this may also be the complaint of an adolescent or adult.[2] The Emergency Physician should consider the mass described by the parents to be a rectal prolapse if the physical examination is negative.[3] Spontaneous reduction will often occur in this age group. It might be noted as an incidental finding on physical examination.

The differential diagnosis of a rectal prolapse includes anal warts, hemorrhoids, intussusception, prolapsed rectal polyp, a prolapsed rectal tumor, or a small bowel evisceration.[14-18] **Mistaking an intussusception for a rectal prolapse may result in significant morbidity and mortality.**[19-22] **Differentiating features of an intussusception include the ability to pass the finger between the prolapsed bowel and the anal sphincter (Figure 87-3). This is in contrast to patients with a rectal prolapse in which the protruding mucosa is continuous with the perianal skin and the examiner's finger will not pass that junction (Figure 87-2*D*).**[1,3,8] Patients with an intussusception usually appear ill, whereas those with a rectal prolapse appear well.[3] Prolapsing hemorrhoids are more often seen in adolescents and adults, are usually purple in color, have deep grooves between the areas of prolapsing tissue, and lack radial or concentric folds. A prolapsed polyp or tumor is plum-colored, does not involve the entire anal circumference, is movable, and is usually palpable as a small growth on a stalk.[3,9]

FIGURE 87-3. An intussusception. Note the junction where the finger can be passed (*arrows*).

INDICATIONS

Reduction should be attempted on all patients with a visible rectal prolapse as soon as possible to avoid vascular compromise of the bowel. It is easier to reduce before edema occurs from prolonged prolapse. Early reduction may avoid complications and stretch damage to the pelvic floor ligaments, the pelvic floor muscles, and the anal sphincter muscles.

CONTRAINDICATIONS

There are few absolute contraindications to the reduction of a rectal prolapse. Gangrene, necrosis, and ulceration of the mucosa are signs of vascular compromise or ischemia and require an emergent consultation by a General Surgeon or Colorectal Surgeon. **Do not reduce ischemic tissue as it may precipitate peritonitis or cause a perforation of the rectum. If the Surgeon's arrival is delayed, prompt gentle reduction should be attempted only after discussions with the Surgeon. Do not reduce an intussusception**. Consult a Surgeon for further evaluation and management of an intussusception.

EQUIPMENT

- Gloves
- Water-soluble lubricant
- 4×4 gauze squares
- 2 inch wide adhesive tape
- Benzoin spray or swabs
- Sedatives as necessary

PATIENT PREPARATION

Explain the reduction procedure to the patient and/or their representative. Reduction is most likely successful in a relaxed and nonstraining patient. Sedation may sometimes be required in adults. Sedation is more often needed in pediatric patients. Children tend to be more anxious, crying, fighting, or straining, all of which will increase the intraabdominal pressure and make reduction more difficult. The sedation may be administered intramuscularly, intravenously, orally, or subcutaneously.

Position the patient.[9,10] Place the child in the prone knee-chest position on the parent's lap or on the examination table (**Figure 87-4**).

FIGURE 87-4. The prone knee-chest position for children.

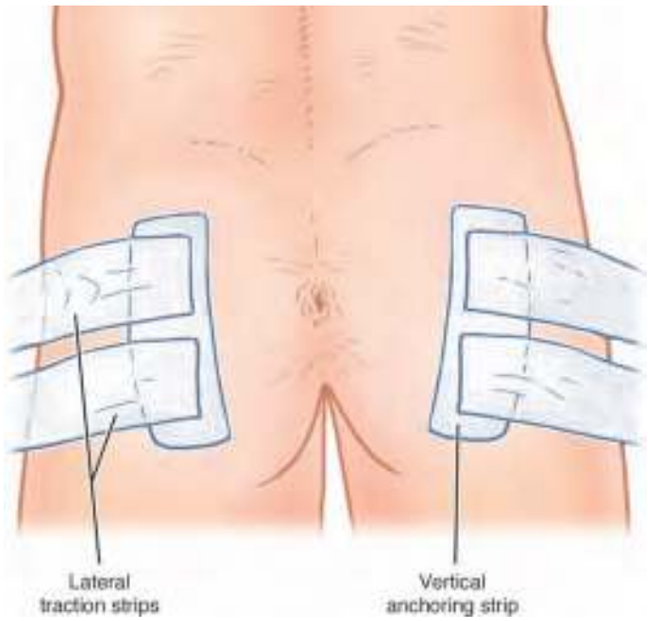

FIGURE 87-5. Taping the buttocks open in the prone patient allows for unobstructed access. The lateral traction strips are taped to the gurney or examination table.

Place the adult patient in the prone position on an examination table. Large buttocks or tense buttocks may interfere with the reduction of a prolapsed rectum. In these cases, apply benzoin to the buttocks and allow it to dry. Tape the buttocks open for better access (**Figure 87-5**). Alternatively, in both age groups, the patient can be placed in the lateral decubitus position.

TECHNIQUE

Position the patient. Liberally apply water-soluble lubricant onto the prolapsed rectum. Apply gauze squares onto the prolapsed tissue at the 3 o'clock and 9 o'clock positions (**Figure 87-6**). The bowel wall is quite slippery after lubrication, and the gauze will improve the grip on the mucosa. Place both thumbs near the bowel lumen with the hands stabilized on the buttocks (**Figure 87-6*A***). Apply steady, gentle thumb pressure to gently roll the prolapsed rectum back through the anus. Alternatively, the index and the middle fingers can be used to reduce the prolapsed rectum (**Figure 87-6*B***). **Regardless of the method used, constant and steady pressure must be applied to the prolapse.** The reduction often requires patience as it may take up to 15 minutes to reduce a prolapsed rectum.

The rectum will become edematous, and swelling will be noted if the rectal mucosa has been prolapsed for a prolonged period of time. Wrap a gauze square or an elastic wrap around the prolapsed rectum and apply gentle manual compression for 3 to 5 minutes before attempting the reduction.[23] If the first effort is unsuccessful without sedation, a second attempt at reduction after administering sedation is appropriate. If the prolapsed rectum will not reduce (i.e., incarcerated), consult a General Surgeon or Colorectal Surgeon for reduction under general anesthesia and possible surgical repair.

ALTERNATIVE TECHNIQUES

A novel method to help reduce a prolapsed rectum uses simple table sugar.[24-26] The longer the rectum is prolapsed, the more edematous it becomes and the more difficult it is to reduce. Apply a liberal amount of sugar onto the prolapsed rectum and cover it with several layers of gauze. Allow the patient to relax for 15 to 30 minutes. The hyperosmotic sugar draws fluid out from the edematous prolapsed rectum. Gently wipe off the sugar with saline- or water-moistened gauze. Attempt manual reduction as described above.

ASSESSMENT

Perform a digital rectal examination to ensure that the reduction is complete.[9] If not, apply pressure with the examination finger to completely reduce the prolapse. The digital rectal examination should be followed by anoscopy in the Emergency Department to look for any gross pathology. Refer to Chapter 88 for the complete details of anoscopy.

AFTERCARE

The application of a bulky pressure dressing will prevent an acute recurrence of the prolapse. Apply petrolatum gauze over the anus. Apply several gauze squares over the petrolatum gauze and into the gluteal cleft. Tape the buttocks together. **The patient and the family must be informed that reduction might be temporary and the prolapse could recur.** Training cooperative parents to reduce the prolapse is warranted in cases of recurrent rectal prolapse in the pediatric age group. Be sure to send them home with gauze squares, gloves, and lubricant.

The underlying cause of the rectal prolapse should be treated.[27] A prophylactic regimen of laxatives and stool softeners should be started if the patient is constipated. Instruct the patient on proper eating habits including fruits, vegetables, and roughage. In cases of diarrhea, treatment should target the underlying causes. Seating

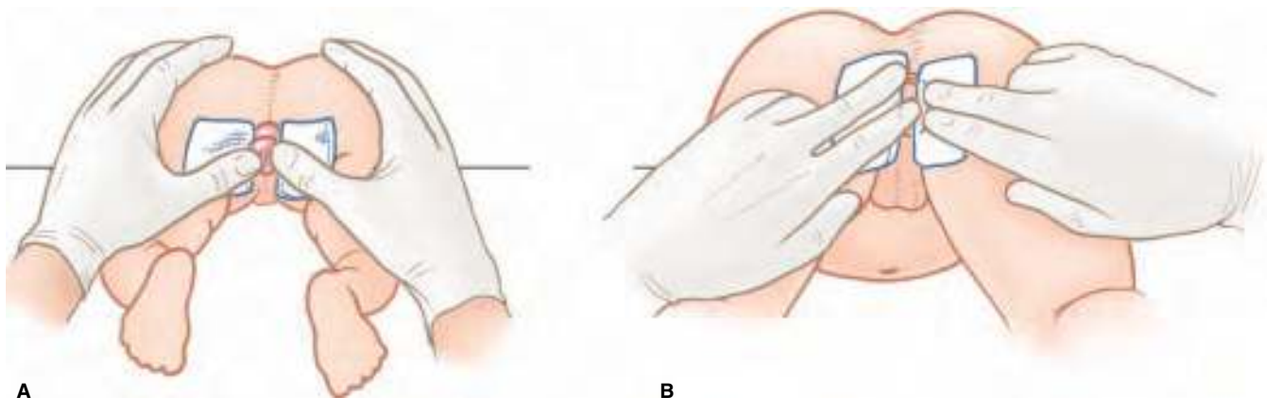

FIGURE 87-6. Rectal prolapse reduction techniques. ***A.*** Thumb method. ***B.*** Finger method.

children on a child's potty-chair or on an adult toilet seat with a small hole may prevent future episodes of rectal prolapse. Discourage excessive squatting and straining.[4]

All patients who have undergone successful reduction should be referred to a Colorectal Surgeon for further evaluation.[28-31] Children should be followed up to rule out serious etiologies such as cystic fibrosis. As the child grows, the supporting tissue around the rectum develops. Therefore, rectal prolapse in this age group is usually self-limited and surgery is rarely required. Adults should be referred for proctosigmoidoscopy to rule out a tumor. Conservative management in the elderly is rarely successful, and most patients eventually require surgical repair. Early referral can avoid complications and stretch damage to the pelvic floor ligaments, pelvic floor muscles, and anal sphincter muscles.

COMPLICATIONS

The complications of the procedure are often minimal. The reduction itself may lead to minimal mucosal bleeding that is self-limited. The patient may experience a slight discomfort in the anus for up to 24 hours after the reduction. This can be managed with oral acetaminophen or nonsteroidal anti-inflammatory drugs.

The inability to reduce a rectal prolapse is an indication for surgical consultation in the Emergency Department. An incarcerated rectal prolapse can lead to vascular compromise. Signs of vascular compromise include mucosal gangrene, necrosis, and ulceration. These patients require admission to the hospital with an emergent surgical consultation, even if the reduction is felt to be successful, due to the risk of reducing ischemic bowel that could perforate. The rupture of an incarcerated rectal prolapse with small bowel herniation through the tear has very rarely been reported during attempted reduction.[11] Fecal and urinary incontinence may also occur as a result of a long-standing prolapse. This is due to the entrapment and stretching of the pudendal or perineal nerve resulting in neurovascular dysfunction and not a complication of the reduction procedure.

SUMMARY

A rectal prolapse is an uncommon condition affecting the very young and the elderly. Reduction can usually be performed in the Emergency Department. It is important to differentiate a prolapsed rectum from an intussusception and from external hemorrhoids. The reduction procedure is quick and simple. The application of constant, firm, and gentle pressure to the rectum in a relaxed and nonstraining patient will reduce most rectal prolapses. All patients with a prolapsed rectum should be referred for further evaluation to rule out underlying pathologic causes for the prolapse.

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88 Anoscopy

Charles Huggins, Claudia Kim, and Hao Wang

INTRODUCTION

Examination of the anal canal is important to evaluate several common patient complaints relating to the anus including bleeding, itching, and pain.[1,2] It is possible to examine parts of this area with flexible instruments or a rigid rectosigmoidoscope. The only method that will give a consistent clear view of the anal canal is anoscopy.[3] To properly perform anoscopy, it is necessary to thoroughly understand the anatomy of the region, be aware of the possible causes of

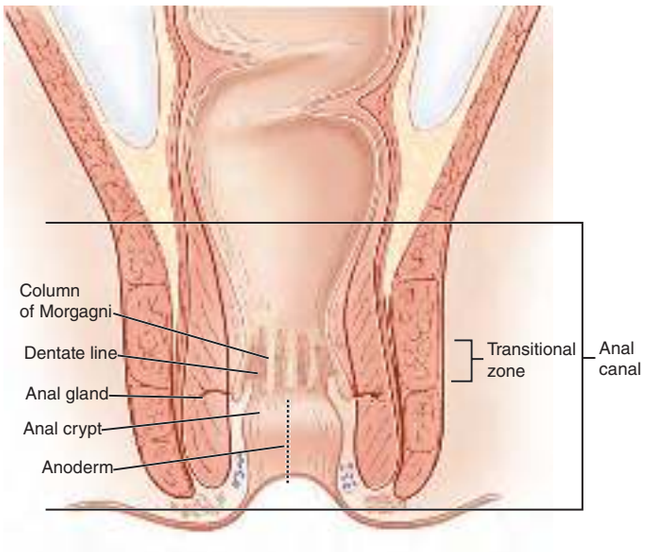

FIGURE 88-1. The topical anatomy of the anal canal.

the symptoms evaluated, use the appropriate equipment, and position the patient correctly.

ANATOMY AND PATHOPHYSIOLOGY

It is necessary to understand the anatomy of the anal canal in order to evaluate the patient's signs and symptoms properly.[4] The anal canal is completely extraperitoneal and is approximately 2.5 to 4 cm long. The anatomy can be divided into topical anatomy and major supporting structures.[5] The topical anatomy is depicted in **Figure 88-1**.

Perineal skin covers the perineum, is fully innervated, and includes both hair follicles and apocrine glands. It can be grossly distinguished from the anoderm surrounding the anal canal by the visible hair. The anoderm is specialized squamous epithelium that lines the majority of the anal canal. It is fully innervated but does not have apocrine glands or hair follicles. This epithelium is very thin and elastic, and if it is destroyed by surgery or infection, stricture formation during healing may occur.

Looking into the anal canal, the anoderm can be seen to end in an irregular line called the dentate line (**Figure 88-1**). This is a demarcation of anoderm to transition zone mucosa. The anal canal proximal to the dentate line is lined with columnar epithelium. It has no cutaneous sensation and is insensitive to cutting. This allows minor therapeutic procedures (e.g., banding or suture ligation) to be done without an anesthetic agent. It is also the reason internal hemorrhoids usually do not cause pain. The transition zone continues proximally for a variable length of 6 to 12 mm before it becomes the rectal mucosa (**Figure 88-1**). The junction of the transitional zone with the rectal mucosa is not visible to the naked eye. The rectal mucosa decreases in diameter in the area of the transitional zone. The mucosa appears to be bunched together in columns called the columns of Morgagni at the level of the dentate line (**Figures 88-1 and 88-2**). Crypts are formed between the columns as the transitional zone becomes the dentate line. Under the anoderm in the crypts are multiple anal glands. Blockage of the anal glands by foreign material leads to infection. Blockage or primary infection of the glands causes the majority of abscesses that arise around the anus. The crypts are areas to look for foreign bodies (e.g., fish or chicken bones).

External hemorrhoids are located in the left-lateral, right-posterior, and right-anterior portions of the distal anal canal and are covered with anoderm. Their normal position is below the dentate line and they can be examined by gently spreading the buttocks. The internal hemorrhoids also are located at the left-lateral, right-posterior, and right-anterior positions. They are normally located above the dentate line and are covered with transitional epithelium and rectal mucosa. They can best be examined with an anoscope if they are not prolapsed. It is possible to see them with a retroflexed

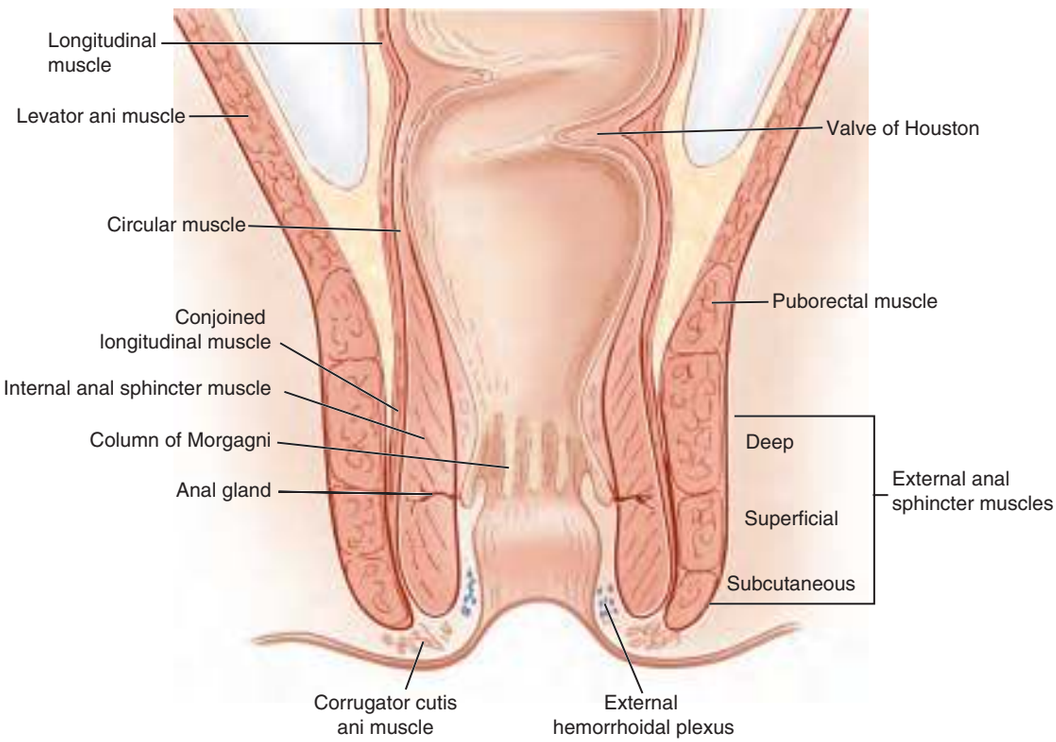

FIGURE 88-2. The major supporting structures of the anal canal.

sigmoidoscope or colonoscope, but the increased air pressure tends to flatten them out giving a false impression of their size.

The anoscope is used to examine the closed portion of the anal canal. It dilates the anal sphincter and allows one to examine the underlying canal through an opening cut out from the side of the anoscope called a fenestration. It is important to understand the anatomy and functions of the anal sphincter musculature to properly perform this examination (**Figure 88-2**). Immediately below the anoderm is the internal anal sphincter muscle. It is circumferential and consists of a thickening and rounding of the continuation of circular smooth muscle from the rectum. This muscle is not under conscious control. The first response of the internal anal sphincter muscle to a rectal examination or anoscopy is contraction. **It is necessary to pause and use slow gentle dilatation to prevent patient discomfort and complete the examination.** The axis of the anal canal will normally follow an imaginary line drawn from the patient's anus to their umbilicus. Surrounding the internal anal sphincter muscle is the external anal sphincter muscle. This is comprised of three external sphincters of striated muscle that are more loop-like than circumferential. Deep to these muscles and at the top of the anal canal is the puborectalis muscle. This forms a sling to pull the anus anterior. It is connected to both the anal sphincters and the levator ani group of muscles. The puborectalis muscle can be felt as a funnel-shaped structure during the digital rectal examination.

INDICATIONS

Anoscopy is indicated for the evaluation of most anal symptoms.[1-10] There are numerous common complaints and conditions where an anoscope, in conjunction with visualization and a digital rectal examination, would be used for evaluation or therapy.[6-8] Anoscopy can be used diagnostically to evaluate rectal bleeding, itching, discharge, fecal impaction, anal pain, pain with defecation, perirectal infections, fistulas, foreign bodies, internal hemorrhoids, anal masses, and abnormal digital rectal examinations. It can also be used for the evaluation of uncomplicated anal trauma, inflammatory bowel disease, and sexual abuse. Anoscopy can be used therapeutically to open the anus and allow the application of medications, procedures to be performed, or observation of anal pathology management (e.g., anal fissures, anal fistulas, intraanal condylomata, and hemorrhoids).

CONTRAINDICATIONS

Most contraindications to anoscopy are relative. Anoscopy should not be performed in unwilling patients, patients with unstable medical conditions, or patients who are immunosuppressed. The amount of discomfort a patient will undergo relates to their tolerance. Minor discomfort associated with topical skin excoriations can be treated with 2% lidocaine jelly used as a lubricant and the examination can then continue. Moderate pain can be managed with the application of procedural sedation (Chapter 159). Severe pain associated with anal fissures or anal abscesses is best managed in the Operating Room under general anesthesia.

Strictures can occur from postsurgical changes, inflammatory bowel disease, chronic diarrhea, and other disease processes. Anoscopy should not be performed if the patient has anal strictures, a partially imperforate anus, a completely imperforate anus, or prolapsed thrombosed internal hemorrhoids. The Emergency Physician should determine if the anoscope will pass through the anus during the visual examination and the digital rectal examination. **Never insert the anoscope if resistance is encountered. The anoscope should not be used to dilate the anus**.

Anoscopy is contraindicated if major genitourinary trauma is sustained or in patients with an acute abdomen. It is also contraindicated if any recent surgical procedure has been performed on the anus. The exception is for the physician who performed the surgical procedure. Anoscopy should be performed with caution in patients with prosthetics or valvular heart diseases requiring prophylactic antibiotics and those with coagulopathy or taking anticoagulants if biopsy is considered.

EQUIPMENT

- Water-soluble lubricant
- Lidocaine jelly, can be used as lubricant for patients with pain
- Anoscope or Vernon-David rectal speculum (**Figure 88-3**)
- Drapes
- Examination table, preferably a proctoscopy table
- Nonsterile examination gloves
- 4×4 gauze squares
- Large cotton-tipped applicators
- 4–0 chromic gut suture
- Suction, optional
- Bright directional light source
- Tincture of benzoin
- 2 inch adhesive tape

There are many different types of anoscopes available (**Figure 88-3**). The type of instrument chosen is largely the preference of the examining physician. The Vernon-David type anoscope is often preferred as it has a wide fenestration and a diameter that is not too large for most patients while allowing the best view (**Figure 88-3*A***). The Ives (**Figure 88-3*B***) or Hirschman (**Figure 88-3*C***) anoscope may be used if a larger diameter instrument is required. There are disposable plastic anoscopes that are available and used in many Emergency Departments (**Figures 88-3*D*, 88-3*E*, and 88-3*F***). Some of the metal reusable and plastic disposable anoscopes allow for the attachment of a fiberoptic light source. Many of these anoscopes are convenient but do not allow the same view as a Vernon-David anoscope. The AnoSpec (OPB Medical, Lawrence, MA) is disposable plastic with a built-in light source (**Figure 88-3*F***). **Some physicians use a glass test tube as a substitute for an anoscope. Test tubes should not be used as they can break and cause serious complications.**

The anoscope is a two-piece device (**Figure 88-4**). It ranges from 7 to 25 cm in length. It can be manufactured from clear plastic, opaque plastic, or metal. The anoscope may have a handle that allows the operator to control its movements. The proximal end is funnel-like and tapers into the cylindrical shaft that is approximately 2.5 cm in diameter. The distal end of the anoscope is tapered on one side and known as the fenestration. The fenestration allows the mucosa to be viewed within the anoscope. It is also where procedures are performed through the anoscope. Some anoscopes have a site proximally to attach a light source. The obturator is smooth tipped, fits within the anoscope, and occludes the anoscope. Its rounded, distal end protrudes from the anoscope. The obturator is used each time the anoscope is inserted to prevent trauma to the anal mucosa. It is removed after the anoscope is inserted to allow viewing through the anoscope.

PATIENT PREPARATION

The area being examined is within the anal canal and requires no special preparation to view correctly. The patient should be given an opportunity to voluntarily evacuate their bowels prior to the

FIGURE 88-3. Some of the types of anoscopes available. ***A.*** Vernon-David anoscope. ***B.*** Ives anoscope. ***C.*** Hirschman anoscope. ***D.*** A disposable anoscope. ***E.*** Sani-Scope 2 anoscope. ***F.*** Various sizes of the AnoSpec. (Photo courtesy of OPB Medical.)

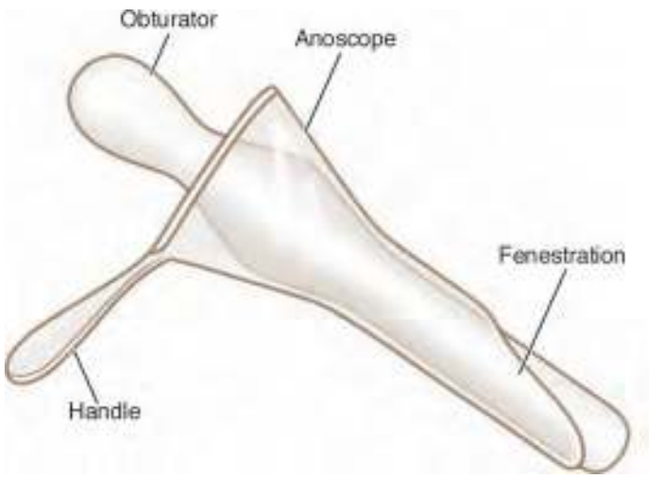

FIGURE 88-4. An example of an anoscope.

examination. It is necessary to have the patient remove their clothes from the waist down and provide barriers to protect the patient, the patient's clothing, and the surrounding area from spills. It is usually wise to avoid enema preparations as liquid stool is much more difficult to contain than solid stool.

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. Explain to the patient what to expect. Discuss the order of the examination, the importance of relaxing, the reason for multiple insertions and withdrawals of the anoscope, and what you are looking for or expect to find. Obtain an informed consent for anoscopy and any procedures that may be performed.

Anoscopy can be performed with the patient in one of many positions.[10] The position that allows the best observation in most patients is the knee-chest or prone position (**Figure 88-5**). This allows the buttocks to be to either side and places the anus at the proper angle. Examination of the anus in this position is easiest on a proctoscopy table but can be performed on an examination table or gurney. The lateral decubitus position with the knees drawn up and the buttocks

FIGURE 88-5. The knee-chest position.

protruding partly off the table is acceptable for limited diagnostic examinations. Large buttocks may prevent the examination. Apply tincture of benzoin to the buttocks and allow it to dry. Apply 2 inch adhesive tape to the buttocks and tape them to the examination table to spread the buttocks apart (**Figure 86-4**). Place a Mayo stand or bedside examination table next to the patient's buttocks.

Carefully inspect the entire perineum and anal verge. Many types of pathology such as fissures, fistulas, hemorrhoids, condylomata, and dermatologic conditions can be seen at this time. **A digital rectal examination with a well-lubricated gloved finger prior to anoscopy is mandatory**. The digital rectal examination has many advantages.[1] It allows one to find pathology that is better palpated than viewed. It gives the examiner the size and angle of the anal canal. It will allow the examiner to identify whether the patient has tenderness that would preclude anoscopy. Any strictures will be identified and allow the examiner to prevent the anoscope from advancing through these strictures and lacerating the tissues. It may identify pathology, so that the examiner can focus anoscopy in a specific area. It prelubricates the anal canal to make insertion of the anoscope easier. Lidocaine jelly can be used as a lubricant and as an anesthetic if the patient has pain from excoriation. The use of intravenous analgesics, intravenous sedatives, procedural sedation, or general anesthesia may be occasionally required if the patient has significant pain.

TECHNIQUE

The technique of anoscopy is rather simple.[9] Look closely at the anoscope and be sure it is intact and correctly assembled. Liberally lubricate the obturator and anoscope. Ensure that the obturator is easily removed and replaced from within the anoscope. Grasp the anoscope with the dominant hand and the obturator secured in place by the thumb of that hand (**Figure 88-6*A***).

Use the nondominant hand to spread the anus (**Figure 88-6*A***). Place the obturator at the center of the anus. **Slowly insert the anoscope and allow time for the internal anal sphincter muscle to relax.** Direct the tip of the obturator toward the patient's umbilicus (**Figure 88-6*A***). Insert the anoscope to its fullest depth to start the examination with the area of fenestration pointed toward the area you wish to view first (**Figures 88-6*B* and 88-6*C***). Slowly remove the obturator and place it on a Mayo stand or a bedside table where it can easily be retrieved. Adjust the directional light for the best illumination.

The usual starting place for the examination is the posterior midline (**Figures 88-6*B* and 88-6*C***). Slowly withdraw the anoscope under direct observation to evaluate the entire depth of the anal canal exposed by the fenestration. Remove any fecal material with a cotton-tipped applicator. Note the appearance of the dentate line, the mucosa, and the anoderm. Note the presence and location of any blood, hemorrhoids, masses, mucus, purulence, or other abnormalities. A cotton-tipped applicator can be used to gently move the mucosa to view between the columns if the anoscope does not fully dilate the columns of mucosa. Continue to withdraw the anoscope as the mucosa is viewed.

Replace the obturator when the anoscope is fully removed from the patient. Rotate the anoscope 30° to 40° and reinsert it into the anal canal. It often takes four or five repeated insertions to evaluate the entire circumference of the anal canal. Some physicians prefer to replace the obturator and rotate the anoscope while it is still within the anal canal. This method is discouraged as it may pinch the tissue

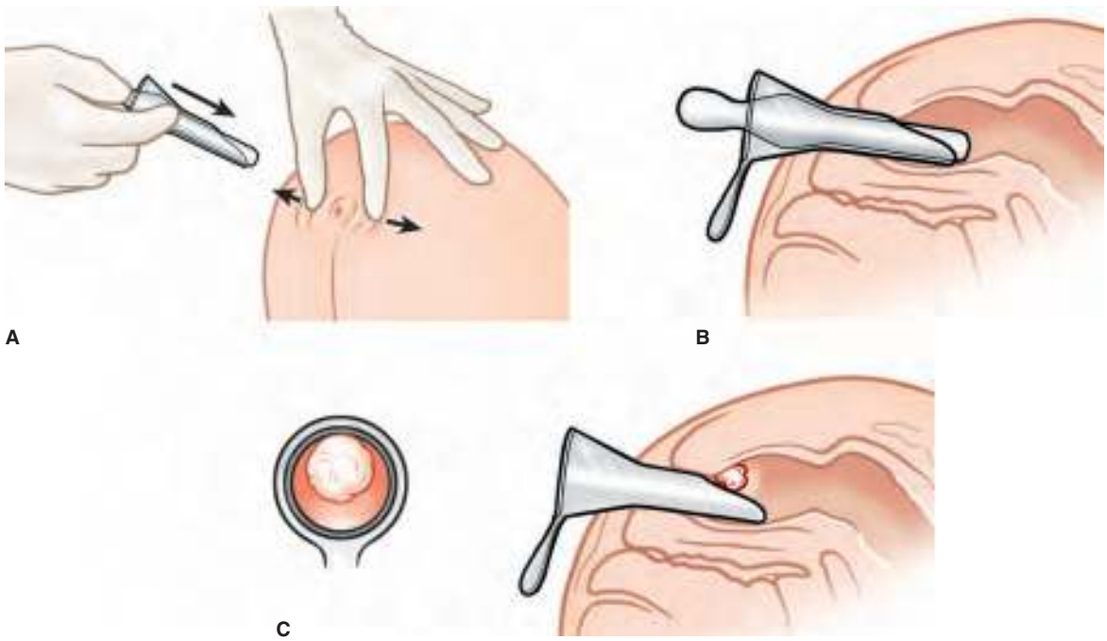

FIGURE 88-6. Placement of the anoscope. ***A.*** The nondominant hand is used to spread the anus. The anoscope is inserted at an angle and aimed toward the umbilicus. Note that the dominant thumb is used to secure the obturator within the anoscope. The anoscope is completely inserted with the fenestration pointed toward the posterior midline (***B***) or the area of interest (***C***).

of the anus between the obturator and the viewing tube and does not allow the examiner to see the entire depth of the anal canal.

It is helpful to ask the patient to bear down when examining the left-lateral, right-posterior, and right-anterior sections of the anal canal when evaluating the internal hemorrhoids. It is often possible to reproduce the prolapsing of internal hemorrhoids to evaluate the grade of the hemorrhoids. Bearing down is particularly important if bleeding is the symptom. It is possible to identify the area of bleeding.

PEDIATRIC CONSIDERATIONS

Anoscopy may be performed in children for the same indications as an adult. An anoscope of 8 to 10 cm in length and 1 cm in diameter is appropriate for a neonate and young infant. An anoscope of over 12 cm in length and 1.5 cm in diameter is appropriate for an older infant and child. An adult anoscope is appropriate for an older child and adolescent. Disposable plastic laryngoscopes with a built-in light source (AnoSpec, OPB Medical, Lawrence, MA) are available in many Emergency Departments (**Figure 88-3*F***). Position the young child supine with their buttocks at the edge of the examination table. Have an assistant grasp, abduct, and flex the child's thighs so they touch the abdomen without compressing the abdominal wall. The remainder of the procedure is as described above.

COMPLICATIONS

Examination of the anal canal with an anoscope should have minimal or no complications. It is possible to cause abrasions or lacerate the very thin anoderm. This is normally avoidable with adequate lubrication, the use of the obturator when inserting the anoscope, and a gentle technique. Minimal bleeding from mucosal irritation is common and self-limited. Dislodgement of a clot may result in hemorrhoidal bleeding that can be controlled with direct pressure, packing the anal canal with gauze squares, or a 4–0 chromic gut figure-of-eight suture. The most common complication is pain. This is avoidable by allowing enough time for the anus to relax while gently inserting the anoscope. The presence of an anal fissure may preclude the examination. The use of a topical anesthetic will usually allow the examination to proceed.

SUMMARY

Anoscopy is a commonly performed procedure in the Emergency Department. The anal canal is a cylindrical structure surrounded by sensitive anoderm and contracting anal sphincter muscles. The best way to examine this area is with a side-viewing fenestrated anoscope. Anoscopy allows direct viewing of the anal canal to best evaluate anal and perianal complaints. It will give the most information with minimal discomfort when properly performed. Always perform a digital rectal examination prior to anoscopy.

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89 Rigid Rectosigmoidoscopy

Hao Wang, Nicole Remish, and Nestor Zenarosa

INTRODUCTION

Rigid rectosigmoidoscopy, although still widely used, has largely been replaced by the flexible sigmoidoscope for routine elective screening and diagnostic workups due to less patient comfort, relatively low diagnostic yield, and difficulty in performing associated procedures.[1] However, the rigid rectosigmoidoscope is superior to the flexible sigmoidoscope in measuring distances accurately, examining an unprepared patient, and working within the bowel lumen (e.g., when removing foreign bodies). The larger lumen of the rigid rectosigmoidoscope allows for a larger biopsy where pathology is in question. The cost associated with this examination is less than that for flexible sigmoidoscopy. The rigid rectosigmoidoscope can be purchased in a disposable model that performs well. It is important for the Emergency Physician who evaluates and treats problems related to the colon, rectum, and anus to be familiar with rigid rectosigmoidoscopy.

ANATOMY AND PATHOPHYSIOLOGY

The detailed anatomy of the anal canal is covered in Chapter 88. The gross anatomy of the colon is reviewed in **Figure 89-1*A***. It is important to be aware of the large folds that impinge on the lumen of the colon called the valves of Houston (**Figure 89-1*B***). These folds must be gently flattened to advance the rigid rectosigmoidoscope and clearly see the proximal side of the valve when looking for pathology. It is also necessary to understand the three-dimensional path followed by the distal colon, rectum, and anus. The direction to follow will be toward the patient's umbilicus for 3 to 5 cm initially. The anus then turns posteriorly as it becomes the rectum and follows the curve of the sacrum. The rectosigmoid junction is reached at 10 to 15 cm where the lumen sharply angulates anteriorly and to the left. Because the scope is rigid and straight, it is necessary to angle the tip of the rigid rectosigmoidoscope toward the lumen of the bowel and then gently flatten the haustra or move the patient's colon so that the lumen is in a straight line.

INDICATIONS

Many of the indications for rigid rectosigmoidoscopy are the same as those for performing flexible sigmoidoscopy. The rigid scope, however, is superior when the bowel is not properly prepared, if a bigger biopsy is needed, or if a larger instrument needs to be passed to the last 25 cm of the colon.

The rigid rectosigmoidoscope may be used to evaluate the rectum and sigmoid colon in the office or the Emergency Department. It can be used diagnostically to evaluate symptoms such as rectal bleeding, constipation, and diarrhea. Rectal bleeding can be evaluated in the unprepared patient. Rigid rectosigmoidoscopy is particularly helpful to determine if stool is mixed with blood when evaluating hematochezia and for determining whether colonoscopy is indicated. It provides diagnostic value when performing a biopsy or cytology of

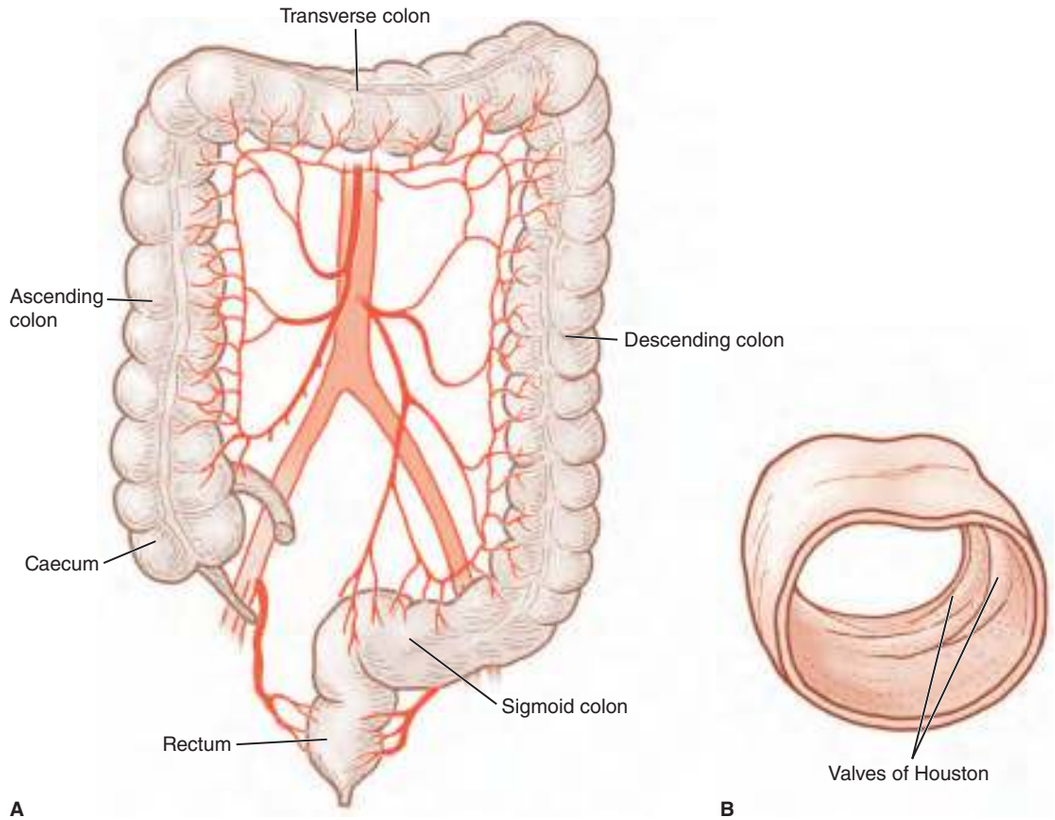


FIGURE 89-1. Anatomy of the colon. ***A.*** The gross anatomy. ***B.*** Cross-section through the colon demonstrating the valves of Houston.

lesions within reach of the instrument. It can be used to accurately measure the distance to rectal lesions from the anus prior to surgery. It is also used for surveillance of colon or rectal cancer after a subtotal colectomy. As neoadjuvant treatments for rectal cancers significantly differ depending on the position and location of the tumor, rigid rectosigmoidoscopy can be used for identifying tumor burden locations. Current specific treatment is based on measurement standards.[2] The rigid rectosigmoidoscope is superior for traumatic injuries. The rectum is seldom prepared in the evaluation of a traumatic injury and flexible instruments are unable to clear solid or thick stool. Rigid sigmoidoscopy has also been shown to be superior to digital rectal examination alone in identifying penetrating rectal trauma. Rigid sigmoidoscopy had a sensitivity of 78% in diagnosing trauma compared to a sensitivity of 51% with digital rectal examination.[3] Rigid rectosigmoidoscopy can also be used therapeutically. Foreign bodies in the rectum or sigmoid colon can be removed with grasping forceps through the scope. The rigid rectosigmoidoscope allows for larger biopsy specimens. Electrocoagulation of bleeding points or electrofulguration of small lesions can be performed during this procedure. A sigmoid volvulus can be decompressed using the rigid rectosigmoidoscope to pass a tube to splint the volvulus. It is much easier to suction out a large volume of obstructed material in the colon and leave a large tube with the rigid rectosigmoidoscope.

CONTRAINDICATIONS

Absolute contraindications are the same as those for anoscopy, which is reviewed in Chapter 88. Relative contraindications include an uncooperative patient, severe anal pain that may require general anesthesia, recent surgical anastomosis in the distal 25 cm of the colon and rectum, severe stenosis of the anus or rectum, and peritonitis. Rigid rectosigmoidoscopy should be performed with great caution in patients with severe coagulopathy or patients requiring prophylactic antibiotics (e.g., neutropenia, endocarditis, valvular heart disease, or prosthetics) since the risk of bacteremia is high. This procedure should not be performed by anyone unfamiliar with the equipment and technique as significant complications can occur.

EQUIPMENT

- Phosphate soda enemas
- Rigid rectosigmoidoscope with obturator, air insufflator, eyepiece, and light source
- Suction catheter
- Suction machine or wall suction
- Biopsy forceps
- Long cotton-tipped applicator with a silver nitrate applicator on the opposite end
- Proctoscopy table or examination table
- Protective drape with exam fenestration
- 4×4 gauze squares
- Water-soluble lubricant
- Exam gloves
- Impermeable gown
- Instrument stand
- Anoscope should be available to examine the anal canal if indicated

The rigid rectosigmoidoscope and other required instruments are available in preassembled sterile trays. The tray usually contains the

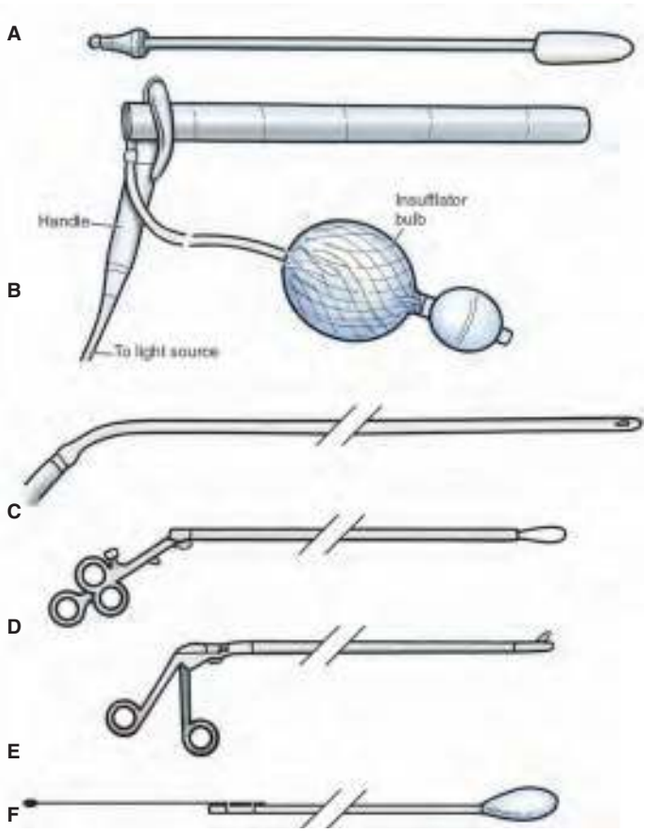


FIGURE 89-2. The instruments required to perform rigid rectosigmoidoscopy. ***A.*** Obturator. ***B.*** Rigid rectosigmoidoscope. ***C.*** Suction catheter. ***D.*** Polypectomy snare. ***E.*** Biopsy forceps. ***F.*** Cotton-tipped applicator with silver nitrate matchstick taped to the opposite side.

rigid scope, an obturator, a suction catheter, polypectomy snare, and biopsy forceps (**Figure 89-2**). These trays are available from the Operating Room, hospital central supply, or the surgical clinics. The trays may contain disposable single-use instruments or multiuse instruments depending on the institution. The disposable equipment eliminates the risk of cross-contamination.[4] The remainder of the equipment must be gathered from around the Emergency Department.

The rigid rectosigmoidoscope is a simple instrument (**Figures 89-2*A* and 89-2*B***). The shaft is approximately 30 to 40 cm in length and has 1 cm increments marked on the outside. Attached to the proximal end are an eyepiece, a handle, and an inflation port. The eyepiece swings to open and close over the proximal shaft of the scope. The handle is used to direct and move the scope. Inside the handle is a fiberoptic light source that transmits into the shaft. The insufflator bulb and tubing attach to the inflation port. These are used to insufflate air through the scope and into the colon. The obturator fits within the shaft of the scope. The distal end of the obturator is smooth, occludes the shaft of the scope, and projects 1 to 2 cm distal to the scope.

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. It is also important to explain to the patient what to expect. Discuss the order of examination, the importance of relaxing, the reason for multiple insertions and withdrawals of the rigid rectosigmoidoscope, and what you expect to find. It is important to explain the reason for insufflating air and how this may produce discomfort. Explain that the procedure should not require procedural sedation and that the patient should inform the Emergency Physician of any discomfort. Assure the patient that the complaint of pain will at least temporarily stop the examination. Perforations are unlikely with a relaxed and cooperative patient. Obtain a signed informed consent for this procedure.

While it is possible to perform an examination with rigid rectosigmoidoscopy on an unprepared rectum, more information about the mucosa will be obtained if the bowel has been prepared. Two 4-ounce phosphate soda enemas given at 2 hours and 1 hour before the examination will provide adequate preparation in most patients. The use of high-volume polyethylene glycol (PEG) preparations, saline, or mannitol should be reserved for colonoscopy. There is no need to clean the entire colon. The preparations used for colonoscopy are expensive, involve too much preparation, and cause patient discomfort for the extent of the examination. The judicious use of intravenous sedation may be required in some patients who are anxious.

It is important to check the equipment prior to insertion into the patient. Open the tray and place the instruments and supplies on the Mayo stand. The eyepiece on the proximal end of the scope should open easily, close easily, and seal against the rigid rectosigmoidoscope. Open the eyepiece and insert the obturator completely within the rigid rectosigmoidoscope. The handle, including the light source, must be firmly attached. Turn on the light source. The light must be seen coming from the end of the scope. The bulb of the insufflator must pump air into the rigid rectosigmoidoscope and should reinflate after the bulb is released. Liberally lubricate the distal 5 cm of the rigid rectosigmoidoscope and the obturator. Reinsert the obturator into the rigid rectosigmoidoscope.

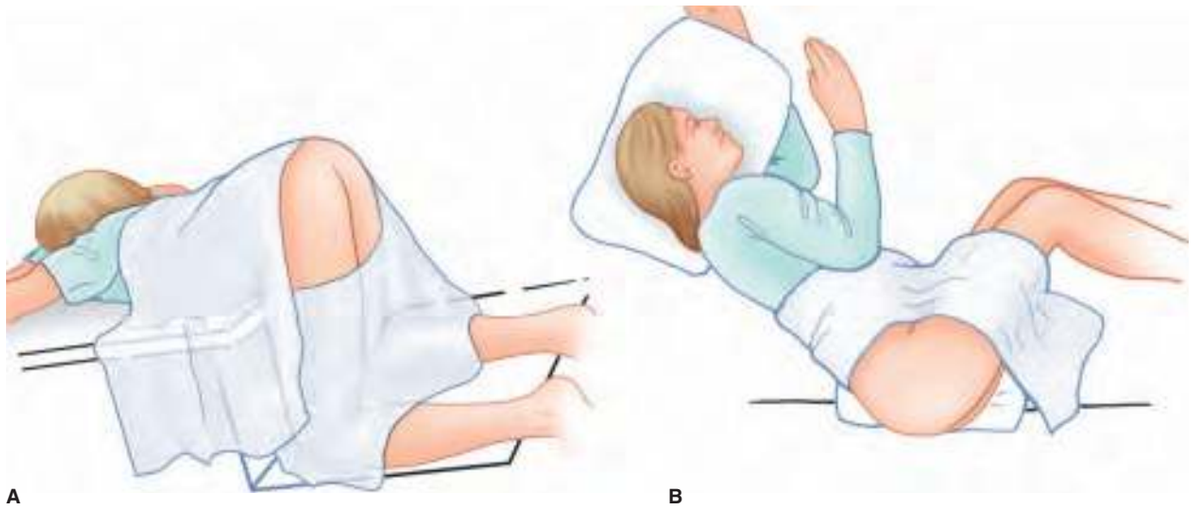
Disrobe the patient from the waist down. Place the patient in the prone jackknife (i.e., knee-to-chest) position on a proctoscopy table if one is available (**Figure 89-3*A***). Examination in this position is relatively easy and less air inflation is required during the procedure, thus reducing patient discomfort. If the patient is unable to assume this position, or only a routine examination table or stretcher is available, place the patient in the left lateral decubitus or Sims position (**Figure 89-3*B***). Examination in this position requires some experience and is not recommended for novices. The Emergency Physician should be prepared with an impermeable gown and one glove on the left hand and two on the right hand (if right handed). It is helpful to have someone assist with the remainder of this procedure. Place the fenestrated drape over the patient so that the buttocks are completely exposed (**Figure 89-3**). Place a Mayo stand or bedside procedure table within reach of the patient's buttocks.

As in anoscopy, it is important to carefully inspect the entire perineum and anal verge prior to the examination. Many types of pathology such as fissures, fistulas, hemorrhoids, condylomata, and dermatologic conditions may be seen at this time.

Digital rectal examination with a well-lubricated, gloved finger is mandatory prior to rigid rectosigmoidoscopy. This step allows the examiner to find pathology that is better palpated than viewed and allows greater focus on a specific area. It will also allow the examiner to evaluate the size and angle of the anal canal, assess for patient sensitivity or tenderness prior to rectosigmoidoscopy, and prelubricate the anal canal making insertion of the rigid rectosigmoidoscope easier. Use 2% lidocaine jelly as a lubricant and to provide some local anesthesia if the patient has pain from excoriation. This examination is usually performed with the right hand. Remove the extra glove from the dominant hand before picking up the rigid sigmoidoscope. This prevents the contaminated glove from holding the scope near the Emergency Physician's face.

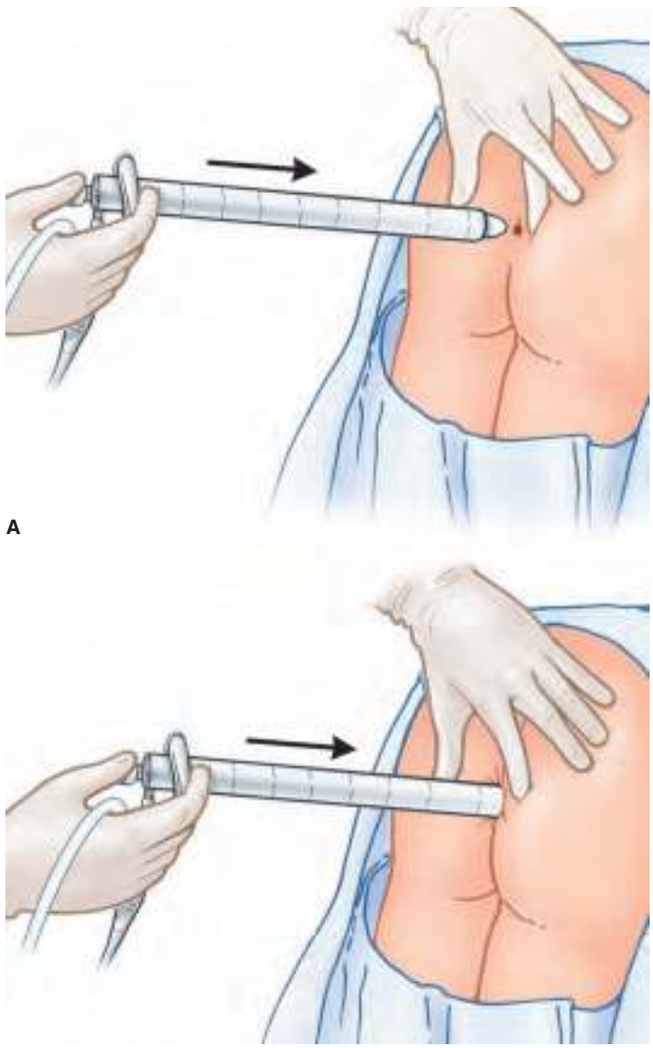
TECHNIQUE

Stand to the left side of or directly behind the patient. Place the left, or nondominant, hand on the patient's buttocks (**Figure 89-4*A***). Grasp the handle of the rigid rectosigmoidoscope with the right,



A B

FIGURE 89-3. Patient positioning. ***A.*** The prone jackknife position on a proctoscopy table. ***B.*** The left lateral decubitus position on an examination table with the buttocks extended over the edge of the table. Note that the drape is placed so that the buttocks are completely exposed.



A

B

FIGURE 89-4. Insertion of the rigid rectosigmoidoscope. ***A.*** The left (nondominant) hand is placed on the buttocks and used to spread the buttocks. ***B.*** The right (dominant) hand is used to insert and advance the scope while the thumb keeps the obturator properly seated.

or dominant, hand. Place the right thumb on the obturator to keep it properly seated (**Figure 89-4*B***). Spread the buttocks with the left hand (**Figure 89-4**). **Gently and slowly insert the rigid rectosigmoidoscope into the anus and aimed toward the patient's umbilicus.** Advance the scope to the 5 cm mark.

Support the rigid rectosigmoidoscope at the anus with the left hand (**Figure 89-5*A***). This is necessary so that if the patient moves, the instrument will move with the patient. Remove the obturator and place it on the Mayo stand. Close the eyepiece and insufflate air into the rectum.

One must control the insertion and direction of the rigid rectosigmoidoscope with the right hand while stabilizing it with the left hand (Figure 89-5*A*). Continue to slowly advance the rigid rectosigmoidoscope under direct viewing while simultaneously insufflating air to distend the walls of the colon (**Figure 89-5*A***). The anal canal is approximately 4 to 5 cm in length (**Figure 89-6**). The rectum will be seen to turn somewhat posteriorly and follow the hollow of the sacrum (**Figure 89-5*B***). Moving the right hand and the rigid rectosigmoidoscope anteriorly will direct the tip posteriorly (**Figure 89-6*B***). Use the left hand as a fulcrum to help maneuver the distal tip of the rigid rectosigmoidoscope to follow the lumen of the rectum (**Figure 89-6**). The sigmoid colon can also be identified by the presence of transverse folds that are lacking in the rectum. The rectosigmoid junction is approximately 16 cm from the anus and can be seen when the lumen turns anteriorly and to the right (**Figure 89-6*C***). It is necessary to use the rigid rectosigmoidoscope to gently straighten the colon while completely inserting the instrument to a depth of 25 cm. **It is important to view the bowel lumen at all times. Never advance the instrument blindly as this may cause a perforation.**

Maintain communication with the patient, inform them of what is happening, and ensure patient comfort throughout the procedure. Open the eyepiece if it becomes clouded with moisture and wipe it with dry gauze so the view is kept clear. **Do not keep your face near the eyepiece when opening the eyepiece.** The insufflated air may forcefully expel stool and secretions.[5] The bowel must be reinflated when the eyepiece is reclosed.

After the rigid rectosigmoidoscope is advanced to 25 cm, or as far as the patient will allow, slowly remove it with a circular motion (**Figure 89-7**). View the mucosa while removing the rigid rectosigmoidoscope in a circular motion. This flattens out the haustra and valves of Houston so that the entire mucosal surface can be viewed.

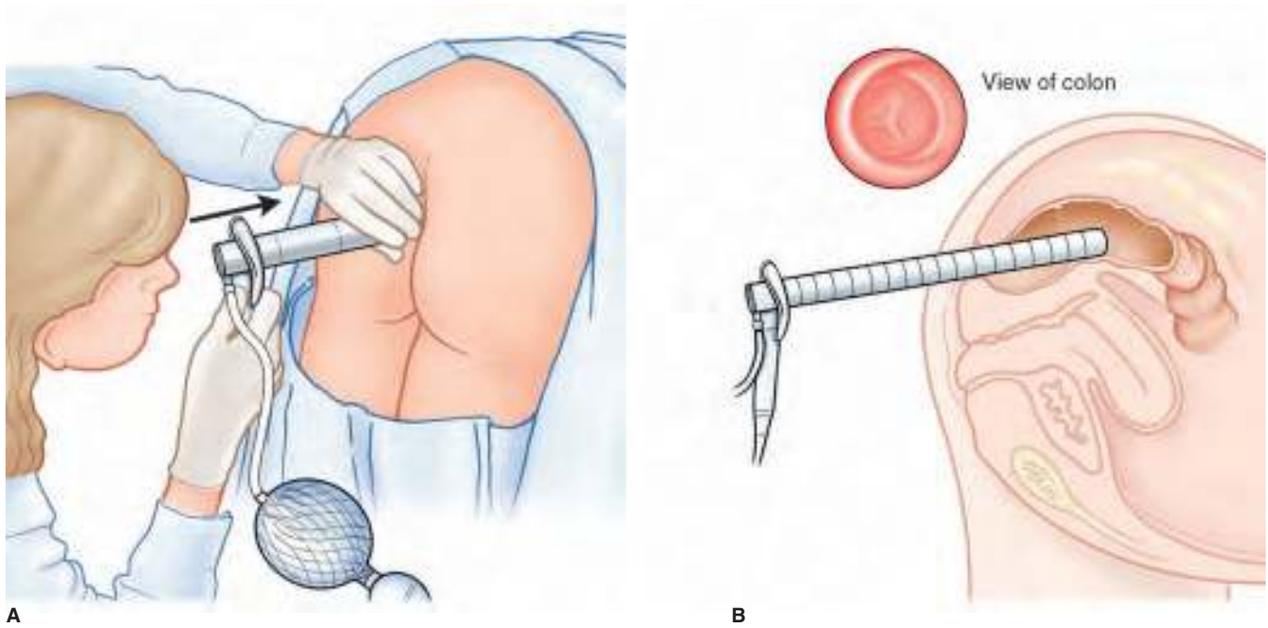


FIGURE 89-5. Advancement of the rigid rectosigmoidoscope. ***A.*** The nondominant hand stabilizes the scope while the dominant hand advances it under direct visualization. ***B.*** The rectum begins as the anal canal turns posteriorly toward the sacrum.

Attempt to keep at least 50% of the colonic lumen in view at all times. Open the eyepiece just prior to completely removing the rigid rectosigmoidoscope to release as much of the insufflated air as possible. This will make the patient more comfortable.

The viewing window, which seals in the air, must be opened if it is necessary to biopsy or to grasp something. This will result in the collapse of the rectum and loss of view. It is important to place the tip of the rigid rectosigmoidoscope over the area in question so it will stay in the examiner's view. The obturator should be reinserted if it is necessary to reinsert or readvance the rigid rectosigmoidoscope.

ASSESSMENT

Completely and carefully inspect the colonic mucosa. Note the presence, location (i.e., anterior, posterior, left, right), and depth of any bleeding, diverticula, fistulas, hemorrhoids, lesions, masses,

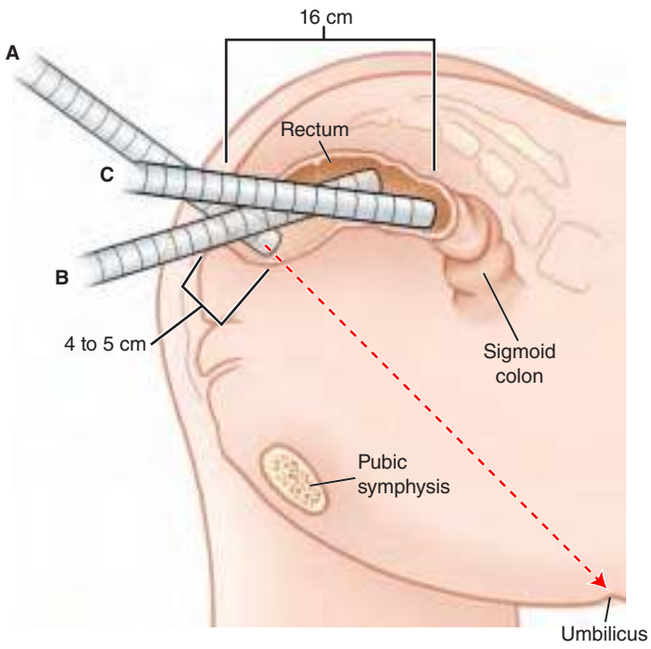


FIGURE 89-6. Insertion and advancement of the rigid rectosigmoidoscope. ***A.*** The scope is inserted and aimed toward the umbilicus. ***B.*** Moving the handle anteriorly will direct the tip of the scope posteriorly. ***C.*** The rectosigmoid junction is located approximately 16 cm from the anus, where the rectal lumen turns anteriorly and to the right.

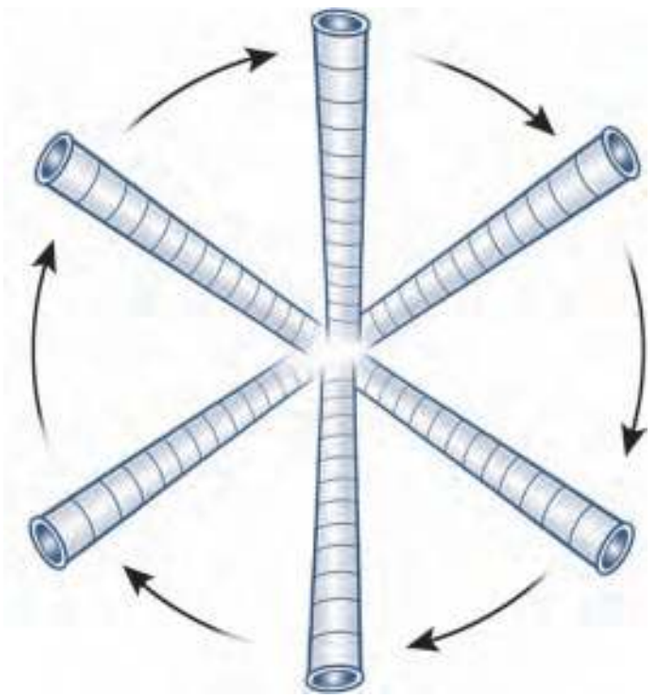


FIGURE 89-7. The rigid rectosigmoidoscope is rotated in a circular motion while simultaneously withdrawing the scope and visualizing the mucosa.

mucosal irregularities, and/or polyps. Document any of these findings in the medical record.

AFTERCARE

The patient may be discharged home after the procedure if there are no complications and there is no other reason to admit them to the hospital. They may experience mild discomfort, flatus, and spotting of blood in the stool for several hours. Instruct the patient to immediately return to the Emergency Department if they develop a fever, abdominal pain, nausea, vomiting, bright red blood per rectum, or if they have any concerns. Follow-up should be arranged with a Family Practitioner, Internist, Gastroenterologist, or Surgeon depending on the findings of the examination.

COMPLICATIONS

The primary complication of rigid rectosigmoidoscopy is perforation of the rectum or sigmoid colon. Air insufflation may cause an existing perforation (e.g., one associated with diverticulitis) to burst and is the reason to not perform this examination if the patient has existing peritonitis. Perforation can occur with forceful insertion of the instrument without viewing the colonic lumen or if the patient moves suddenly and the scope is not supported properly. Perforation may also occur with instrumentation in related procedures (e.g., a biopsy). The risk of perforation greatly decreases when performed with proper technique, maintaining visualization of the colonic lumen while advancing the scope, and responding appropriately to the patient's complaints of pain.

Minor complications may occur. Trauma to the mucosa from the rigid rectosigmoidoscope or instrumentation from related procedures (e.g., a biopsy) can result in minor bleeding that is usually self-limited. Brisk bleeding can be controlled by the judicious use of a silver nitrate matchstick. Bacteremia may occur in up to 10% of patients. Administer antibiotic prophylaxis for high-risk patients prior to the procedure. Mild abdominal discomfort and flatus can last a few hours.

The light source can fail suddenly. This causes problems for both the Emergency Physician and the patient. There is no need to discontinue or postpone the rigid rectosigmoidoscopy. Attach a penlight to the rigid rectosigmoidoscope and continue the examination.[6]

SUMMARY

Rigid rectosigmoidoscopy is inexpensive, easy to perform, and useful in visual examination of bowels with poor preparation and/or where wider access is needed. These characteristics make the instrument useful in many situations. The proper training and understanding of the anatomy of the region is imperative. This examination is well tolerated by the patient and is useful in diagnosing and treating many colonic and rectal problems.

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90 Rectal Foreign Body Extraction

Chad Holmes, Jon Wolfshohl, and Hao Wang

INTRODUCTION

Foreign bodies within the rectum are the result of an ingestion or placed into the anus.[1] Fortunately, the majority of items ingested orally that pass the pylorus and ileocecal valve also pass the anal sphincter. The most frequent types of items found in the anus from ingestion are undigested fish or chicken bones.[2] Foreign bodies that are placed into the rectum are placed iatrogenically (e.g., enema tips and thermometers), inserted by the patient in an attempt to remove impacted stool, inserted by the patient or their partner as a form of sexual gratification, forcibly placed in the anus during a rape, or placed in the rectum as a means of concealment (i.e., body packing and body stuffing).[2-4]

The items placed into the rectum from the anus seem to be limitless and represent all shapes and sizes imaginable.[5] This makes their removal more difficult. **It is important to attempt to identify the characteristics of the foreign body to devise the safest method of removal.** As an example, consider the typical electric lightbulb. The glue that attaches the metal base to the glass loosens with moisture and time. Pulling off the metal base exposes a thin, sharp glass edge. The glass globe is thin and breaks very easily. If the glass breaks, it may take a long time to remove the fragments and cause considerable damage to the surrounding rectal mucosa or the examining finger. **The idea is to remove the foreign body without causing further damage to the rectum or the anal sphincter muscle.** The more knowledge the Emergency Physician has about the foreign body and how it was inserted, the more likely it is that it will be removed safely.[6,7]

ANATOMY AND PATHOPHYSIOLOGY

The significant anatomy of the anal canal is discussed in Chapter 88. An important anatomic consideration in removing rectal foreign bodies include the axis of the lumen of the anus. The anus is pointed toward the patient's umbilicus, while the curve of the sacrum forms a posterior arc. If the length of the foreign body is longer than the curve of the sacrum (e.g., a long vibrator or dildo), the sacral promontory causes the distal end of the foreign body to be directed toward the tip of the sacrum or coccyx. When the object is being removed by bringing the distal end anteriorly, the middle portion may push anteriorly (i.e., into the prostate, uterus, or bladder) and cause considerable discomfort.

The important physiologic considerations include the anal sphincter muscles, edema, and the creation of a vacuum. The anal sphincter is a complex group of muscles. The external anal sphincter muscle is made up of voluntary muscle fibers. The internal anal sphincter muscle is made of smooth muscle fibers. The reflex response to dilatation of the rectum is contraction of the external anal sphincter muscle. The normal tone of the anal sphincter comes from the internal anal sphincter and can go into spasm with manipulation.

Therefore, it is very important to try to remove foreign bodies with slow and steady traction. Maintain constant pressure and wait for the sphincter muscle to fatigue. The technique is not too dissimilar to the methods used to relocate a shoulder. The slow and constant traction will also help with the edema that forms around rectal foreign bodies that have remained in the rectum for a prolonged period.

It is important to consider the formation of a vacuum that may result from traction on a large and/or smooth foreign body (e.g., a bottle or lightbulb). It is sometimes necessary to place one or more soft catheters above the foreign body so that air may get around the object as it is removed. This will prevent the formation of a vacuum and allow the foreign body to be removed. Large Foley catheters with the balloon

inflated can be used for air insertion to prevent the vacuum and traction on it can bring the object further down into the rectum.

INDICATIONS

The indication to remove a foreign body from the rectum is the identification of a foreign body in the rectum. The majority of patients presenting with a rectal foreign body have attempted to remove the object prior to arrival. Common efforts to remove these objects at home include expulsion by bowel movement or oral laxatives.[8] It is impossible to estimate the number of rectal foreign bodies that are removed at home. It is estimated that very few objects inserted through the anus will pass spontaneously. Delaying extraction is detrimental for the vast majority of rectal foreign bodies. Sharp objects may already have begun to perforate the rectum. Large foreign bodies will continue to cause irritation and edema, making removal more difficult with the passage of time. An algorithm for rectal foreign body removal is provided in **Figure 90-1**.

CONTRAINDICATIONS

There are a few absolute contraindications to removing a rectal foreign body in the Emergency Department. **The removal of a rectal foreign body that has not perforated is not an emergency. Time**

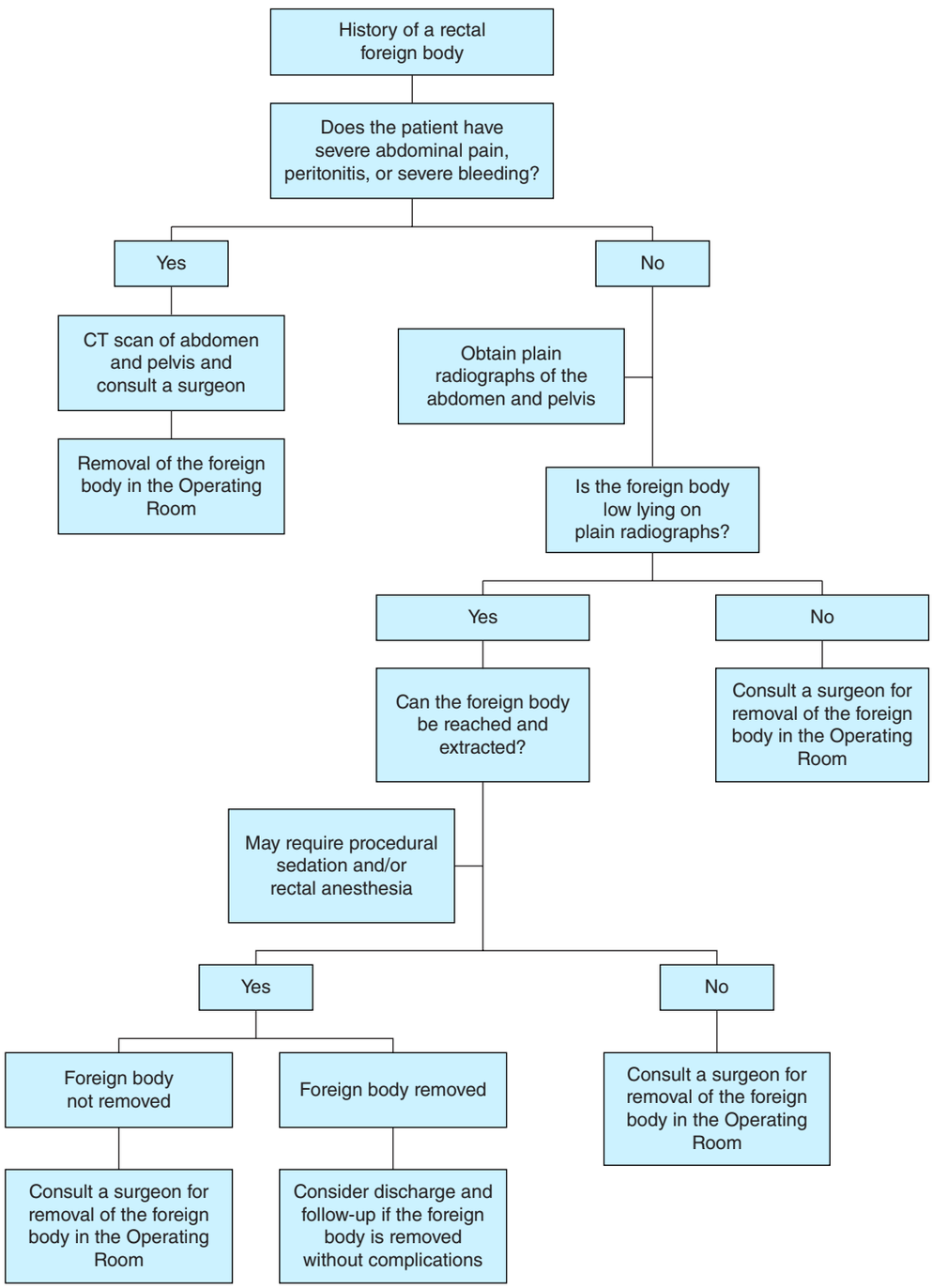

FIGURE 90-1. An algorithm for the management of a rectal foreign body. CT, computed tomography.

should be taken to plan the extraction and achieve adequate anesthesia and relaxation of the anal sphincter muscles, especially if the object is difficult to retrieve. The patient's general condition must be considered. Patients with peritonitis require operative removal and exploration.[9] The time taken to remove the object is wasted and identification of the perforation is easier in the Operating Room with the item in place. Patients with lower abdominal pain and fever may have a perforation in the extraperitoneal space that can be confirmed with a water-soluble contrast enema. Foreign bodies that are large, irregularly shaped, or have sharp edges should be extracted in the Operating Room. **A complete assessment of all the patient's injuries is mandatory in patients who have been assaulted.** It is usually better to leave the object in place to help identify any associated injuries. The foreign body should be removed in the Operating Room if it is not palpable, it is not visible upon dilating the anus, or removal of the object may cause injury to the patient. Packets containing illicit drugs should be extracted with a rigid rectosigmoidoscope or in the Operating Room. Rupture of the packets can result in significant morbidity and mortality.

EQUIPMENT

- Local anesthetic solution with epinephrine (e.g., 1% lidocaine or 0.5% bupivacaine)
- 25 gauge needle, 2 inches long
- 10 mL syringe
- Povidone iodine or chlorhexidine solution
- Anoscope
- Ring forceps
- Tenaculum
- Park retractor
- Hill-Ferguson retractor
- Large spoons
- Foley catheters
- Endotracheal tubes
- Endoscopic snare
- Rigid rectosigmoidoscope
- Vacuum extractor, optional

PATIENT PREPARATION

The patient must undergo a complete history and physical examination.[3,10,11] It is important to ascertain the overall health of the patient in case it is necessary to go to the Operating Room to extract the foreign body. **Attempt to identify patients with peritonitis or rectal perforations because they need to go to Operating Room immediately**.[9] Biplane upright plain radiographs are useful to determine the number, shape, and location of foreign bodies as well as the presence of free air under the diaphragm. It is necessary to look at both the anteroposterior view as well as the lateral view to completely appreciate the object in three dimensions.[1] It is important to inform the patient in advance that while 90% of the objects can be removed from below, some may require general anesthesia or even an operation and a temporary colostomy. Explain the risks, benefits, and complications of the local anesthesia and the extraction procedure to the patient and/or their representative. Obtain an informed consent for the extraction of the foreign body and the method of anesthesia.

Ascertain the type and number of objects in the rectum.[12] It is possible to remove one foreign body and miss others that the Emergency Physician was not aware were present. It is common to find that the patient is unsure of this part of the history. Plain radiographs can be helpful.[8,13] It is also important to identify the technique of insertion. **Patients with objects that were inserted forcefully should undergo a trauma-oriented workup, and sexual abuse must be considered.** Determine the length of time since insertion. Edema formation will be significant if the object has been present more than 24 hours. This will make it more likely that an anesthetic will be needed or that it will be necessary to use catheters to break the vacuum that forms during the extraction.

The parts of the physical examination that are most helpful are the abdominal and rectal examinations. The abdominal examination should focus on the presence or absence of tenderness, peritonitis, and the palpation of a mass. Some objects are long enough to be palpated in the left lower quadrant. It may be useful to apply pressure on the lower portion of the abdomen to help remove the object. The rectal examination should include a careful external examination looking for evidence of tissue bleeding or other forms of trauma. The digital portion of the examination can roughly evaluate sphincter tone and squeeze pressure.

The remainder of the examination should consist of careful palpation of the foreign body to determine its location, tensile strength, texture, mobility, and to identify possible areas to grasp. Examples would include the open end of a bottle or the narrow end of a lightbulb. Determining if the object is hard, soft rubber, or plastic will help to determine the tool best suited to grasp the object. One contraindication to performing the digital rectal examination would be determining, on radiographs or by history, whether the object is sharp (e.g., a knife blade) or consists of broken glass**. It is recommended to postpone the digital rectal examination, especially in prisoners and psychiatric patients, until radiographs rule out a sharp foreign body.** If the examiner is not able to palpate the foreign body, a rigid rectosigmoidoscope should be used to identify and remove the object. The technique of rigid rectosigmoidoscopy is described in Chapter 89.

ANESTHESIA

The second general consideration is the patient's comfort level during the procedure. Several methods can be used to relax the patient and reduce anal sphincter spasms.[12] The use of intravenous or procedural sedation (Chapter 159) will relax the patient and relieve much of the discomfort associated with the procedure. It may be necessary to inject the anus with local anesthetic solution if the patient experiences pain or if slow steady traction will not overcome the tone of the anal sphincter.[14,15] Anesthesia of the sphincter muscles will provide for patient comfort and allow dilation of the sphincter muscles. Place the patient in the lithotomy position (**Figure 90-2*A***). Wipe away any dirt, debris, and fecal material from the perianal skin and surrounding area. Apply povidone iodine or chlorhexidine solution and allow it to dry. Inject local anesthetic solution subcutaneously and circumferentially around the anus (**Figure 90-2*A***). Inject 1 to 1.5 mL of local anesthetic solution into the anal sphincter muscles at the 12, 3, 6, and 9 o'clock positions (**Figure 90-2*B***).

An alternative is a pudendal nerve block. Determine if the ischial spines are palpable through the rectum. Insert the needle through the skin and toward the ischial spine. Use the finger inside the rectum to palpate and guide the needle to the ischial spine. Inject 3 to 5 mL of local anesthetic solution just medial to the ischial spine. Repeat this procedure on the other side. **The main disadvantage of the pudendal nerve block is that it is a blind procedure and has the potential for a needle-stick.** It will be necessary to take the patient to the Operating Room for regional or general anesthesia if procedural sedation or a pudendal nerve block is not successful.

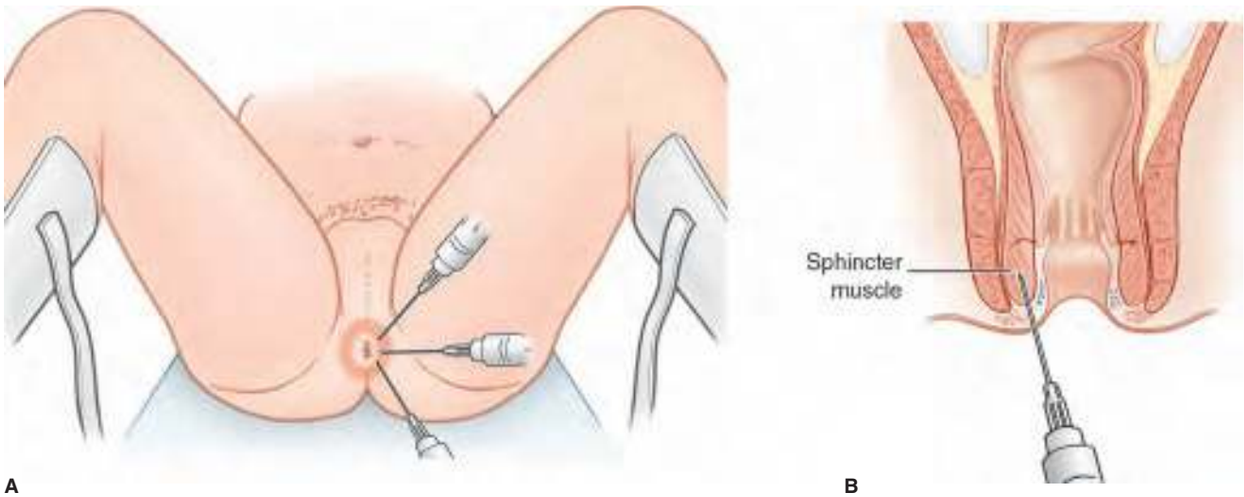

FIGURE 90-2. Anesthesia of the anal region. ***A.*** Local anesthetic solution is infiltrated subcutaneously and circumferentially around the anus. ***B.*** Injection of local anesthetic solution into the anal sphincter muscles.

The patient may remain in the lithotomy position for the extraction of the foreign body (**Figure 90-3*A***). Other positions include the modified Lloyd Davies position (**Figure 90-3*B***) and the Sims position (**Figure 90-3*C***). The choice of positions is physician dependent and limited by patient comfort.

TECHNIQUES

It is important to understand that the types of foreign bodies found in the rectum are variable. It is impossible to give exact instructions on how to remove them. There are some general considerations to remember. The first is to try to visualize the foreign body. Gentle suprapubic pressure will often push the foreign body into the distal rectum. The distal end of the foreign body may get caught along the curve of the sacrum. Place a finger in the rectum to redirect the orientation of the foreign body. The second consideration is to grasp the object. Sometimes an object is low-lying and may be grasped with gloved fingers.[12] The object can then be removed by gentle continuous traction. Low-lying objects may be soft enough to be grasped with an instrument. **It is necessary to do this under direct vision so that the rectum will not be damaged in the attempt to grasp the foreign body**. Hard, low-lying objects made of metal or plastic may be grasped with a tenaculum.

Insert two well-lubricated fingers into the anesthetized anus and gently dilate the anal sphincter muscles. The anus can be maintained in the open position by the insertion of a Park retractor (**Figure 90-4*A***) or a Hill-Ferguson retractor (**Figure 90-4*B***). An anoscope or vaginal speculum may be used if these retractors are not readily available in the Emergency Department. View the distal end of the foreign body. **Never insert instruments unless the foreign body is visualized. Blind insertion of instruments may push the foreign body more proximally or perforate the rectum. Never grasp a foreign body blindly. This can cause injury to the rectum upon removal of the instrument if the rectal mucosa is entrapped between the instrument and the foreign body**.

DIGITAL EXTRACTION TECHNIQUE

Foreign bodies within reach may be grasped with the fingers and extracted. This often proves very difficult as the foreign body will be coated with lubricant, mucus, and/or stool. Instruct the patient to bear down as if having a bowel movement. The intraabdominal pressure generated in this way may expel the foreign body. The foreign body may become entrapped against the sacrum. Insert a finger into the rectum to dislodge the foreign body from against the sacrum as the patient continues to bear down. Attempt one of the techniques described below if digital extraction is ineffective.

FOLEY CATHETER TECHNIQUE

A Foley catheter has been successfully used to aid in the extraction of rectal foreign bodies.[2,16-20] This is especially helpful if the foreign body is large, made of glass, an inverted bottle or can, or has been retained for more than 12 to 24 hours. Pulling these types of foreign bodies can result in the formation of a vacuum proximally and inhibit their extraction. It is sometimes necessary to place one or more soft catheters above the foreign body so that air may get around the object as it is removed to prevent the formation of a vacuum.

Liberally lubricate a Foley catheter. Insert the Foley catheter between the foreign body and the rectal mucosa. Advance the catheter until the balloon is proximal to the foreign body (**Figure 90-5*A***). Fluoroscopy, if available, can be used to confirm the location of the balloon and guide the removal of the foreign body.[21] Inflate the balloon with 30 mL of saline or water. Inject air with a syringe through the Foley catheter to break the vacuum proximal to the foreign body. Remove the syringe so that air can move freely through the Foley catheter and prevent a vacuum from becoming reestablished. Apply constant, gentle, and steady traction to the Foley catheter. This will prevent the foreign body from migrating proximally. It may also move the foreign body distally so that it can be grasped and extracted with fingers or an instrument. The use of two to four circumferentially placed Foley catheters may apply evenly distributed traction to extract the foreign body more easily.

ENDOTRACHEAL TUBE TECHNIQUE

An endotracheal tube has been used in place of a Foley catheter by some physicians (**Figure 90-5*B***). The advantage of using an endotracheal tube is that it is relatively stiff and can apply more traction than a Foley catheter to help remove the foreign body. Unfortunately, there are also disadvantages. The endotracheal tube is larger and less flexible than a Foley catheter. This may result in difficulty advancing it past the foreign body. The larger and stiffer endotracheal tube can more easily lacerate the rectal mucosa and perforate

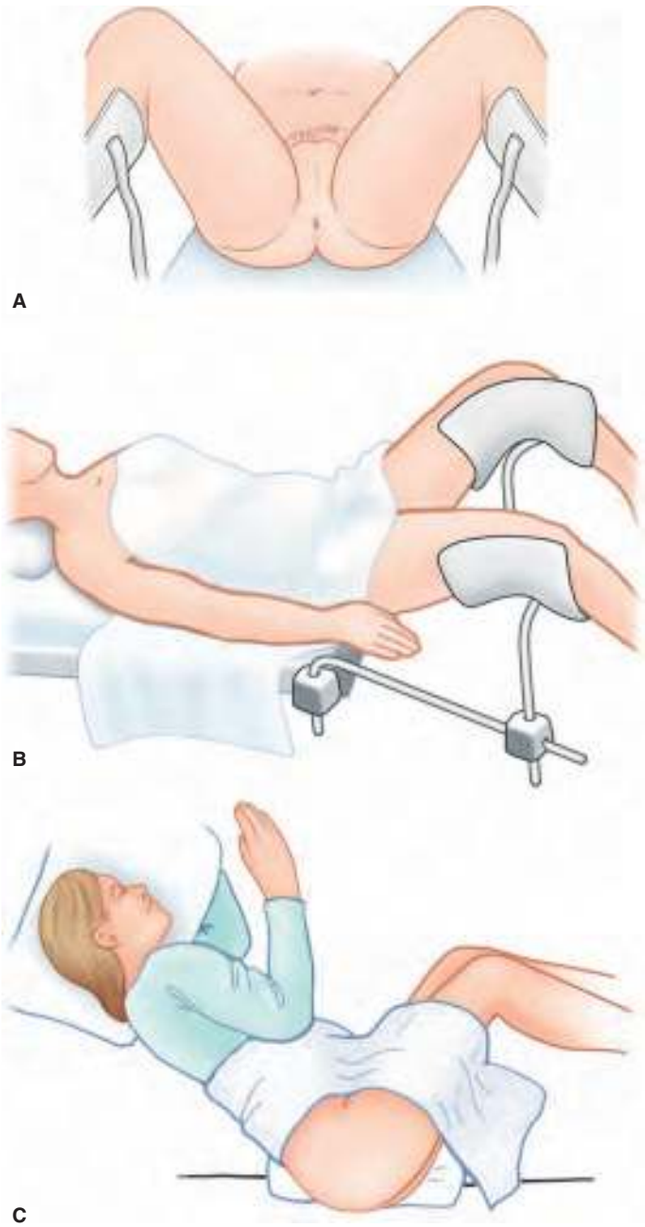

FIGURE 90-3. Patient positioning for removal of rectal foreign bodies. ***A.*** Lithotomy position. ***B.*** Modified Lloyd Davies position. ***C.*** Sims or lateral decubitus position.

the rectum. **For these reasons, the use of an endotracheal tube cannot be recommended.**

SPOON TECHNIQUE

The removal of smooth, round, fragile, or glass foreign bodies can be problematic. This can include lightbulbs, balls, fruits, and vegetables. These foreign bodies can be extracted with a pair of large spoons. Liberally lubricate the posterior surface of a pair of spoons. Insert a Foley catheter to eliminate the vacuum proximal to the foreign body. Insert the spoons until they are cupping the foreign body (**Figure 90-5*C***). Grasp and gently squeeze the handles of the spoons so that they hold the foreign body. Withdraw the spoons and the foreign body.

A more readily available alternative to large spoons are obstetric forceps.[22] These have also been successfully used to extract a rectal foreign body. **The use of obstetric forceps cannot be recommended due to their large size, unfamiliarity to most Emergency Physicians, and the potential to perforate the rectum**.

RING FORCEPS OR TENACULUM TECHNIQUE

Rectal foreign bodies that are not fragile may be grasped with a ring forceps or tenaculum and then extracted.[23,24] Insert a Foley catheter to eliminate the vacuum proximal to the foreign body. Firmly grasp the foreign body with a ring forceps or a tenaculum (**Figure 90-5*D***). A tenaculum has teeth on its distal tips. These may be advantageous as the teeth can grip and firmly hold the foreign body to make the extraction easier. **This must be performed under direct visualization to ensure that the rectal mucosa is not entrapped between the instrument and the foreign body**. Apply gentle and firm traction to extract the foreign body.

VACUUM EXTRACTION TECHNIQUE

A vacuum dart or obstetrical vacuum extractor may be used to extract a rectal foreign body.[17,25,26] Emergency Physicians are usually not trained in the proper use of a vacuum extraction device. It may be beneficial to have an Obstetrician, Gynecologist, Family Practitioner with obstetric experience, or a Nurse Midwife to assist with this technique.

Insert a Foley catheter to eliminate the vacuum proximal to the foreign body. Place the vacuum cup of the device onto the foreign body. **Ensure that none of the rectal mucosa is entrapped between the rim of the suction cup and the foreign body**. Apply the suction to seal the cup against the foreign body. **Recheck to ensure that none of the rectal mucosa has become entrapped.** Apply steady traction to extract the foreign body.

MISCELLANEOUS TECHNIQUES

Numerous other techniques have been devised to remove a rectal foreign body.[27-31] These include the use of proctoscopes and snares, Magill forceps (**Figure 18-16*A***), Boedeker forceps (**Figure 18-16*B***), Tylke forceps (**Figure 18-16*C***), de Pezzer catheters, cyanoacrylate glue, clamps covered with rubber tubing, a tonsil snare, Sengstaken-Blakemore tubes, and plaster of Paris. Cyanoacrylate glue can be used to attach a handle to the foreign body. The foreign body can be extracted by withdrawing the handle after the glue has dried. A Sengstaken-Blakemore tube can be inserted into a foreign body with a small opening (e.g., glass bottle, soda can). Inflate the balloons and apply traction to extract the foreign body. Plaster of Paris can be used to fill a hollow object and allowed to harden around a tongue blade (**Figure 90-5*E***). This is analogous to making a popsicle on a stick. The major disadvantage of using plaster of Paris is that it generates heat as it hardens. This heat may damage the rectal mucosa or shatter a glass bottle. The technique of rectal foreign body extraction is limited only by one's level of experience, comfort with the procedure, and available resources.[32]

ASSESSMENT

Examine the rectum with a rigid rectosigmoidoscope to assess the mucosa for tears or perforations after the foreign body is extracted. Some physicians believe that this examination is not necessary if the patient is asymptomatic, the foreign body is smooth, the foreign body was extracted atraumatically, and no complications arose from the extraction procedure. These patients may be discharged with follow-up as an outpatient for rectosigmoidoscopy. The patient should remain in the hospital for observation and consultation with a General or Colorectal Surgeon if there is damage to the rectal or anal mucosa. The patient should remain NPO and be prepared for

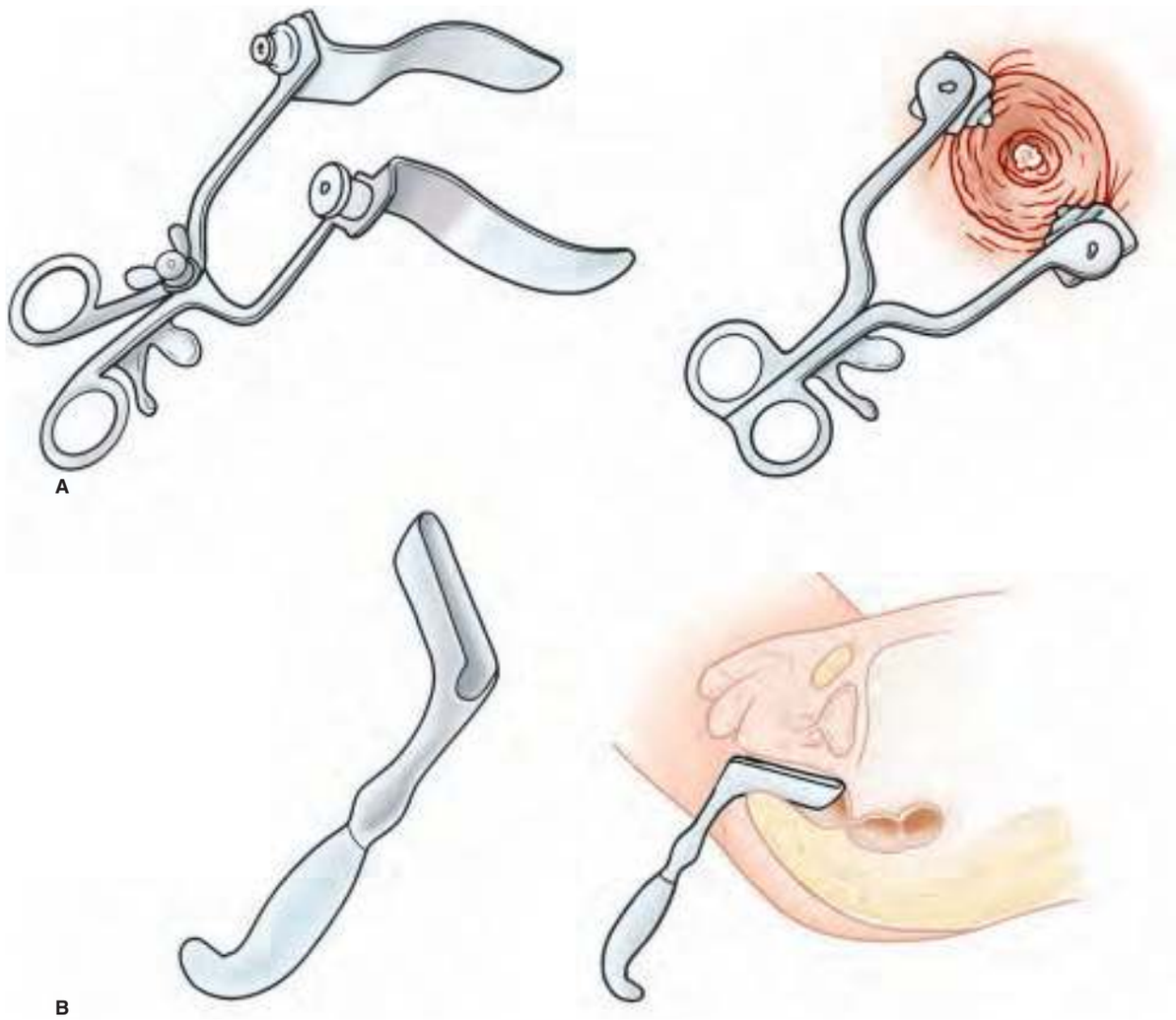

FIGURE 90-4. Retractors placed in the anus allow for better exposure and easier extraction of the foreign body. ***A.*** The Park retractor. ***B.*** The Hill-Ferguson retractor.

possible surgery. Other indications for admissions include abdominal pain, significant bleeding, or the suspicion of a bowel or rectal perforation. Broad-spectrum intravenous antibiotics should be administered if a perforation is suspected.

Perform a digital rectal examination to document the presence and quality of anal sphincter tone after the procedure. This should be delayed until the effects of any general, local, or regional anesthesia have dissipated. Rectal tone that is decreased or diminished from the preprocedural digital rectal examination requires the consultation of a General or Colorectal Surgeon.

AFTERCARE

Patients may be discharged home after the extraction procedure if they are asymptomatic, have normal rectal tone, and have no complications demonstrated on rigid rectosigmoidoscopy. They should be instructed to return to the Emergency Department immediately if they develop abdominal pain, pelvic pain, bright red blood per rectum, or a fever.

COMPLICATIONS

The major complications include rectal bleeding, bowel or rectal perforation, and damage to the anal sphincter.[2] **Since the patient may very well have presented with these complications, it is important to document them or their absence on the preprocedural examination**. Rectal bleeding is common after a difficult extraction. It is important to rule out a perforation. This can be performed with the rigid rectosigmoidoscope after the extraction.[32] Most perforations occur at the rectosigmoid junction, approximately 15 to 16 cm from the anus. Perforation above the peritoneal reflection will result in peritonitis and free air noted under the diaphragm on upright plain radiographs. Perforation below the peritoneal reflection may take several days to manifest pelvic pain, signs of a pelvic abscess, or sepsis. The patient may still be discharged if there is minor rectal bleeding with no evidence of perforation or significant mucosal damage. However, large amounts of bleeding or significant mucosal damage require observation and consultation with a General or Colorectal Surgeon. A Gastrografin enema without the use of the balloon may be used to identify a perforation if there is significant concern. Patients with perforation of an unprepared rectum require surgical intervention and broad-spectrum intravenous antibiotics.[33] Any evidence of acute sphincter damage requires a surgical evaluation for possible debridement. The majority of these lesions are observed, allowed to heal secondarily, and then repaired surgically.

SUMMARY

The majority of rectal foreign bodies can undergo transanal extraction. Removal of rectal foreign bodies should include an appropriate history and physical; biplane abdominal radiographs;

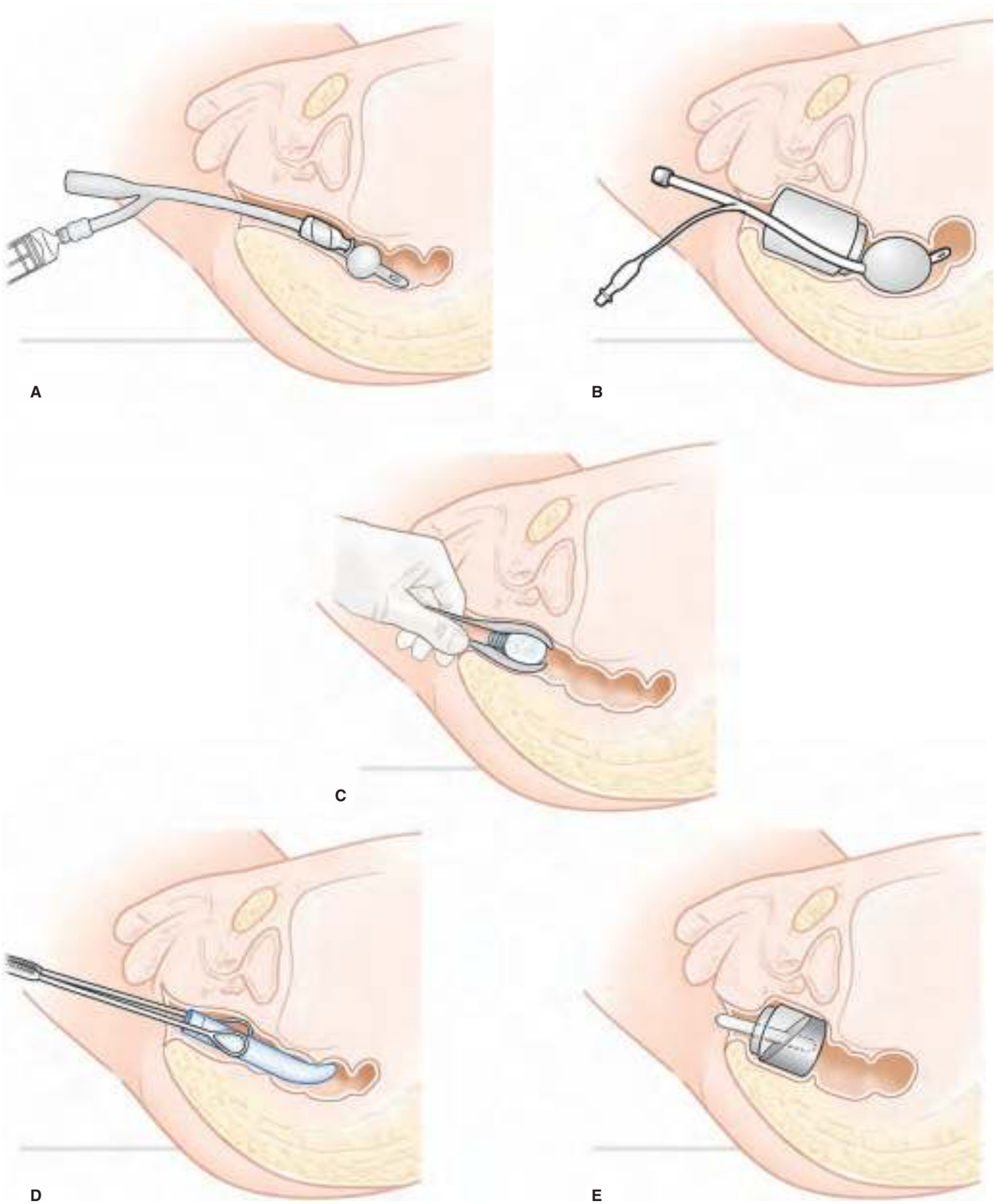

FIGURE 90-5. A sampling of methods to remove rectal foreign bodies. ***A.*** Foley catheter technique. ***B.*** Endotracheal tube technique. ***C.*** Spoons to remove a fragile object. ***D.*** Ring forceps technique. ***E.*** Plaster and a tongue depressor are placed in a jar. After the plaster cures, the tongue depressor can be used as a handle to remove the jar.

relaxation of the anal sphincter; firm attachment to the foreign body and slow, firm traction extraction; and postextraction rectosigmoidoscopy. Inpatient observation is indicated if the rectal mucosa is traumatized. The patient should be taken to the Operating Room if the foreign body cannot be removed in the Emergency Department, if pain control cannot be adequately achieved, if a perforation is suspected, or if removal may result in secondary injury.

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91 Proctoclysis

Hao Wang, Tyson Jay Higgins, and Richard Dean Robinson

INTRODUCTION

Proctoclysis is also known as rectal infusion, a Murphy drip, or rectoclysis.[1] It is defined as the slow infusion of fluids and/or medications into the rectum as a therapeutic intervention. John Benjamin Murphy, an American surgeon, introduced the infusion of rectal saline solution to treat patients with peritonitis in 1909.[2,3] This infusion technique was used in World War I to treat soldiers in hypovolemic shock. Proctoclysis was popular during times when intravenous techniques were less advanced.[4] With the widespread use of intravenous (Chapters 59 and 61 to 64) or intraosseous infusion (Chapter 70) techniques, proctoclysis has become less favorable and is rarely used in Emergency Medicine.[1] It still plays an important role in the resuscitation and treatment of critically ill patients in remote or rural settings, especially by Emergency Medical Technicians (EMTs).

Proctoclysis can be used when sterile fluids are scarce. Nonsterile fluids (e.g., boiled water or tap water) can be infused into the rectum to provide hydration. Proctoclysis is a relatively easy procedure to perform and does not require health care providers skilled at intravenous cannulation. Multiple medications can be quickly and efficiently absorbed through rectal mucosa.[5-9] In recent years, the Macy catheter (**Figure 91-1**) has been successfully used to facilitate proctoclysis with fewer complications and higher satisfaction among patients receiving palliative care, those in the Emergency Department, and those in the Intensive Care Unit.[5,6,10-14] The Macy catheter has proven that proctoclysis can be used as an alternative and efficient route for medication and fluid infusion in the modern era.

ANATOMY AND PATHOPHYSIOLOGY

An understanding of the anatomy of the anorectal canal is necessary to perform proctoclysis. This anatomy is covered in Chapter 88 on anoscopy. The rectosigmoid junction is about 10 to 15 cm proximal to the anus, at which point the lumen sharply angulates anteriorly and to the left. Rectal catheters used for proctoclysis are normally inserted 7.5 to 10 cm (i.e., 3 to 4 inches) from the anal verge in adult patients.

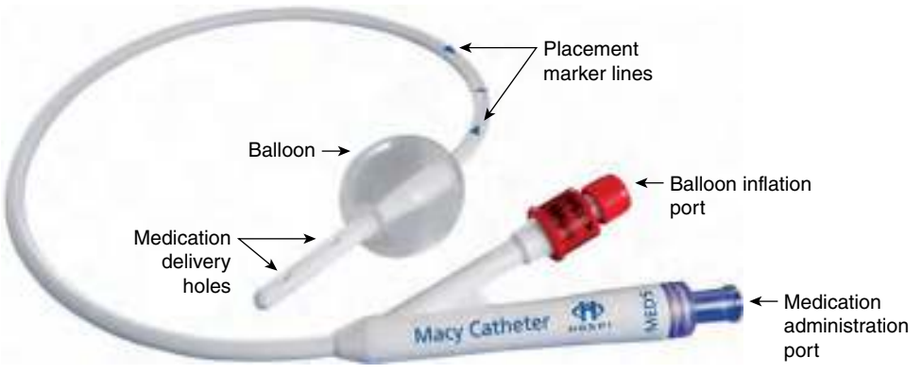
Fluids and medications are absorbed across epithelial cells or via tight junctions between mucosal cells.[15] The rectum does not absorb sodium, chloride, or water well from isotonic solutions. The colon has sufficient capabilities to absorb these substances. When infused via the rectum, drugs migrate into the sigmoid and descending colon. Depending on specific drug composition and administration volume, spreading can vary with a maximal migration generally being reached within 1 hour. The bioavailability is similar to giving the medication orally.

The middle and inferior rectal veins drain venous blood from the lower part of the rectum directly into the inferior vena cava (**Figure 91-2**). Drugs and fluids are directly absorbed into the systemic circulation without entering the liver. This avoids first-pass hepatic metabolism and achieves a greater bioavailability than seen when infusing via other routes (e.g., peripheral venous). A small portion of the absorbent is also delivered directly into the lymphatic system.

INDICATIONS

Proctoclysis can be used in emergent situations for fluid replacement and/or medical treatment when other routes (e.g., oral, sublingual, intravenous, and interosseous) are suboptimal. It can also be used

A



B

FIGURE 91-1. The Macy catheter. ***A.*** The catheter. ***B.*** The labeled catheter. (Photographs courtesy of Hospi Corp.)

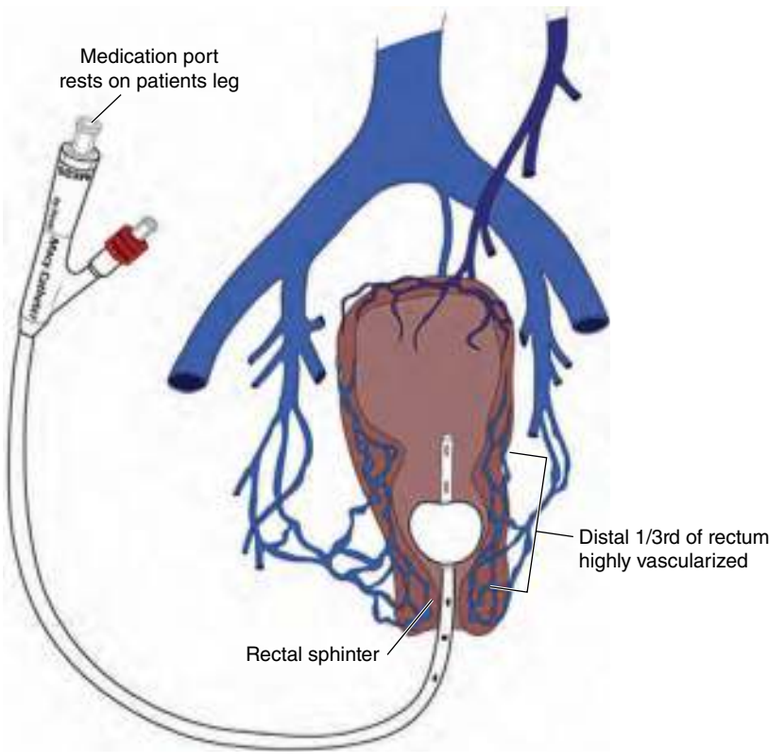


FIGURE 91-2. The venous drainage of the rectum. Note that a Macy catheter has been inserted. (Used with permission from reference 12.)

TABLE 91-1 Some of the Commonly Administered Rectal Drugs[5,6,8,9,16-19]

Classification	Examples
Analgesics	Codeine, hydromorphone, methadone, morphine, oxycodone, tramadol
Antibiotics	Amoxicillin, ceftizoxime, erythromycin, metronidazole
Anticonvulsants	Carbamazepine, diazepam, lamotrigine, pentobarbital, phenobarbital, phenytoin, valproic acid
Antiemetics	Chlorpromazine, cisapride, diphenhydramine, hydroxyzine, metoclopramide, ondansetron, prochlorperazine, promethazine
Anxiolytics	Clonazepam, diazepam, lorazepam, midazolam
Laxatives	Bisacodyl, docusate, glycerin, mineral oil, sodium phosphates
Nonsteroidal anti-inflammatory drugs (NSAIDs)	Acetaminophen, aspirin, diclofenac, ibuprofen, indomethacin, ketoprofen, naproxen
Others	Atropine, cefoxitin, dexamethasone, furosemide, haloperidol, hydrocortisone, Kayexalate (sodium polystyrene sulfonate), ketamine, lactulose, lidocaine, nifedipine, prednisolone, propranolol, theophylline

in remote locations, prehospital settings, or mass casualty situations where health care providers must administer fluids and medications during resuscitations despite having little or no equipment, nursing and ancillary staff, and supplies. Proctoclysis in these situations is often an emergent intervention providing patients with life-sustaining fluids and/or medications until more traditional routes (e.g., intravenous or interosseous) are established. In recent years, proctoclysis has extended into emergent resuscitation and palliative care.

Different types of fluids can be infused during proctoclysis including isotonic solutions (e.g., normal saline or lactated Ringer's). Different concentrations of dextrose solutions can be used to treat patients with hypoglycemia. However, infusing large amounts of dextrose solution is not recommended due to the resultant hypotonic solution raising the potential for water toxicity. Nonsterile solutions (e.g., oral rehydration solution, boiled water, or tap water) can be infused with caution in treating hypovolemic shock via proctoclysis. Different types of medications can be administered during proctoclysis including analgesics, anesthetics, antibiotics, anticonvulsants, and antiemetics (**Table 91-1**).[5,6,8,9,16-19]

CONTRAINDICATIONS

Proctoclysis should not be performed in patients with severe anal strictures or those with a complete imperforate anus. The Emergency Physician should determine whether the rectal catheter will pass through the anus by performing a visual examination and a digital rectal examination. **Digital rectal examination with a well-lubricated, gloved finger is mandatory prior to proctoclysis. Never insert the catheter if resistance to catheter advancement is encountered.** Proctoclysis is also contraindicated if any recent anorectal surgical procedures have been performed and/or in severe genitourinary trauma (e.g., perineal or perianal regions) due to the risk of bowel perforation or active bleeding being relatively high.

Relative contraindications in patients include severe anal pain requiring general anesthesia, anorectal diseases (e.g., abscesses or fistula), or diarrhea. It is also relatively contraindicated as an elective procedure in patients with weak or no anal sphincter tone on digital rectal examination. Proctoclysis should be performed with caution in patients with severe coagulopathies (e.g., thrombocytopenia) and in patients requiring prophylactic antibiotics (e.g., neutropenia, endocarditis, valvular heart disease, or prosthetics) under nonemergent situations. The risk of bacteremia is high. Proctoclysis is relatively contraindicated for long-term use since some drugs (e.g., ergotamine, acetaminophen, aspirin) can cause rectal ulceration and stenosis. Precautions should be given to patients with spinal cord injury, especially above the T6 level. Such individuals may experience autonomic dysreflexia triggering extreme hypertension leading to stroke, hemorrhage, seizure, and death. Proctoclysis should also be used with caution in patients with increased intracranial pressure or glaucoma. Large amounts of hypertonic solution are relatively contraindicated to avoid dehydration. This is especially important when treating infants.

EQUIPMENT

- Personal protective equipment
- Water-soluble lubricant or Lidocaine jelly
- Drapes
- Rectal tube
- Nonsterile examination gloves
- Bright overhead light source
- Tincture of benzoin
- 2 inch adhesive tape
- Sheets
- Blankets
- Toilet tissues
- Suction (optional)
- Absorbent pad (optional)
- Bedpan (optional)
- Intravenous stand (optional)

Appropriate personal protective equipment (e.g., gown, clothing, gloves, face mask, and goggles) needs to be worn to reduce exposure to patient body fluids and/or bowel contents.

There are many different types of rectal tubes. A relatively novel device is the Macy catheter (Hospi Corp., Newark, CA) designed specifically for proctoclysis (**Figure 91-1**). The catheter is available in a 14 French size and made of silicone, making it safe for latex-allergic patients. It utilizes a dual-port system on the proximal end with a balloon inflation port and a fluids/medications administration port. The inflatable 15 mL retention balloon secures the catheter in place. This catheter is designed with a blunt distal end with two side holes for medication and/or fluid delivery. Two placement marker lines are located at 10 and 15 cm from the distal end to guide advancement of the catheter. An internal valve prevents fluid backflow. Once the Macy catheter is positioned and secured, its medication port can be secured to the patient's leg or abdomen for continuous infusions. A large Foley catheter, nasogastric tube, or red rubber catheter can be used for proctoclysis if the Macy catheter is not available.

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. Gain informed consent if possible. No special bowel preparation is required, especially in an emergent situation. The patient should be given an opportunity to voluntarily evacuate their bowels prior to the procedure if no contraindications exist. It is necessary to remove the patient's clothing from the waist down and provide appropriate barriers to protect the patient, the patient's clothing, and the surrounding area from spills. Maintain dignity and privacy by screening the area, and request a chaperon if providing care to a minor or if there is a gender difference or a cultural difference. Place waterproof pads (e.g., Chux) under the patient's hip and buttocks area. Cover the patient

with a blanket to expose only the rectal area. Use an overhead light to clearly visualize the anus.

Proctoclysis can be performed with the patient in one of many positions. The Fowler (i.e., sitting) position, knee-chest position, and ventral (i.e., prone) positions were used for proctoclysis during the early twentieth century. The most popular position used is Sims position (**Figure 90-3*C***). This position allows the patient to lay on their left side with the left hip and left lower extremity straight and the right hip and knee bent. It is also called the left lateral recumbent position. Positioning the patient on the left side facilitates fluid migration toward the colon.

A digital rectal examination with a well-lubricated gloved finger is mandatory prior to proctoclysis. The digital rectal examination has many advantages. It will allow the identification of tenderness, strictures, or a complete obstruction that would preclude proctoclysis. It also prelubricates the anal canal making insertion of the rectal tube easier. Lidocaine jelly can be used as a lubricant and anesthetic if the patient has pain.

Fluids and/or medications should be prepared prior to the procedure. A fluid or medication bag can be attached to the patient (e.g., hip, leg, or torso) or on an intravenous stand. The height of the intravenous stand must be adjusted to avoid high-gravity pressure infusion as outlined below.

TECHNIQUE

OLDER CHILDREN, ADOLESCENTS, AND ADULTS

The technique for proctoclysis is similar to performing anoscopy or rigid rectosigmoidoscopy. Choose an appropriate rectal tube and liberally lubricate the distal 15 cm to 20 cm of the tube. **Avoid overlubricating the rectal tube since it will increase the likelihood of tube retraction (i.e., falling out of the rectum and anus).** Grasp the rectal tube with the dominant hand. Use the nondominant hand to gently separate the patient's buttocks. Instruct the patient to relax by breathing out slowly through their mouth.

Slowly insert the rectal tube with the tip of the tube directed toward the patient's umbilicus. **Give time for the internal anal sphincter muscle to relax.** Gently insert the tube 10 to 15 cm into the anus. **Stop inserting the tube if resistance is encountered**. If a Macy catheter is used, stop inserting it between the two marker lines. Inject the recommended volume of normal saline through the balloon inflation port if using a rectal tube with an inflation balloon. Slowly pull the rectal tube back until it meets resistance or reaches an appropriate length of insertion. The length of insertion is between 7.5 and 10 cm in adults, between 5.0 and 7.5 cm in children, and between 2.5 and 3.75 cm in infants. Final catheter positioning may require subtle adjustment in proportion to the patient's height.

Secure the rectal tube. Apply tincture of benzoin to the buttocks and allow it to dry. Place a loop of tape around the rectal tube, close to the anus, and anchor each end of tape to each buttock to minimize tube retraction and to reduce fluid leakage. **Do not allow the tape to adhere to the anus as it can tear it when removed.**

The infusion should be gravity fed, allowing the rectum to absorb only what is needed. This avoids the high-pressure flow of fluid into the rectum. Raise the height of the infusion bag slowly to an appropriate level above the anus. This is initially 10 to 30 cm above the anus. Open the regulating clamp to fill the tubing. Lower the infusion bag to allow the solution to slowly enter the rectum. Lower the infusion bag further or clamp the tubing if the patient complains of cramping or if fluid leaks from the rectum. Ideally, the height of the infusion bag should ultimately rest at, or slightly above, the patient's hip level. If no intravenous stand is used, lay the fluid or medication bag on the patient's hip, leg, or torso. Infusion time varies with the volume of the solution and type of medication administered. Clamp the tubing after all the solution is infused to prevent entrance of air into the rectum.

When proctoclysis is completed deflate the balloon. Keep the rectal tube in place if repeat treatment is planned within short intervals. Remove the rectal tube if no further proctoclysis is required.

YOUNG CHILDREN AND INFANTS

Proctoclysis can be performed in children for the same indications as an adult. A smaller size rectal tube is appropriate for children and infants. The Sims position might be challenging for an uncooperative child. Position the child supine and have an assistant grasp, abduct, and flex the child's thighs so they touch the abdomen without compressing the abdominal wall. Gently and slowly separate the buttocks. Slowly advance the rectal tube into the rectum to the appropriate length. Sedatives may be beneficial in an uncooperative child. The remainder of the procedure is performed as described for adults.

ASSESSMENT

Monitor the patient's vital signs closely. Recheck the infusion bag periodically. Monitor the response to medications as when administering via any other route (e.g., oral, intramuscular, or intravenous). Nursing should record volumes given, time the infusion is initiated, absorption rates, and total infusion time. Contact the receiving hospital, if the patient requires transportation to an advanced care facility, during proctoclysis to avoid delays. Continue the efforts to establish more traditional infusion routes (e.g., intravenous and interosseous).

AFTERCARE

Inform the patient of the completion of the procedure. Document any side effects or complications. Record the types and volumes of fluids and/or medications given to the patient in the medical record. Clean the patient. Cover or dress the patient as appropriate.

COMPLICATIONS

Proctoclysis is generally a safe procedure with minimal or no complications. The most common complication is pain. This is minimized by allowing enough time for the anus to relax while gently inserting the rectal tube. The use of a topical anesthetic gel will usually allow the procedure to proceed with minimal discomfort. Other potential minor complications are flatulence, fluid leakage, and stimulation of bowel movements.

Digital rectal examination is mandatory as it will assist in identifying the obstacles to a safe rectal tube insertion. Advancement of a rectal tube in the setting of anorectal obstruction may result in bowel perforation. Patients sustaining perianal trauma are at risk of local tissue and/or organ damage if rectal tube advancement produces a pseudolumen.

Bleeding is usually minimal or none. Minimal bleeding from mucosal irritation is common and self-limited. Abrasions or lacerations of the very thin anoderm may occur when the rectal tube is inadequately lubricated or is not gently advanced. This is self-limited and normally avoided with adequate equipment preparation and technique. Dislodgement of a clot may result in hemorrhoidal bleeding. This is typically controlled with direct pressure, packing of the anal canal with gauze squares, or a 4–0 chromic gut figure-of-eight suture.

SUMMARY

Proctoclysis is an uncommon procedure in the Emergency Department. It can be used as an alternative route for infusion of fluids and medications. Proctoclysis provides a simple, safe, and effective

means to rehydrate and resuscitate patients without significant risk of severe side effects or complications. Proctoclysis does not require sterile technique, special equipment, or advanced training.

The rectal route of administration is effective because the rectal mucosa is highly vascularized and allows rapid and effective absorption, water and salts are absorbed quickly, and the effectiveness of medication delivery has been established clinically. The technique of proctoclysis is often avoided due to the numerous downsides of traditional forms of rectal drug administration.

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SECTION 6

Orthopedic and Musculoskeletal Procedures

92 Bursitis and Tendonitis Therapy

Dedra R. Tolson, Brandon M. Fetterolf, and Elaine H. Situ-LaCasse

INTRODUCTION

Bursitis and tendonitis are frequent complaints evaluated in the Emergency Department. **Bursitis represents an acute or chronic inflammation of a bursa. Tendonitis involves inflammation surrounding the bony insertion sites of the tendons.** These complaints are usually treated conservatively with the goal of reducing inflammation. Treatment often includes anti-inflammatory agents, application of cold and heat, elevation, and rest. Joint injections (Chapter 97) and soft tissue injections are helpful for the diagnosis and therapy of a variety of musculoskeletal complaints. The goal of aspirating fluid from a joint is to help make a diagnosis or to relieve pressure within a swollen joint. The therapeutic goal of putting a needle into a joint or soft tissue is to deliver local anesthetics and corticosteroid medications for acute pain relief, suppression of inflammation, and increased mobility.[1,2] Injection therapy along with generalized treatment is a component of a multifaceted treatment regimen that should be considered by the Emergency Physician.

Definitive care can be initiated by the administration of a steroid injection during the patient's presentation. The clinical response to injectable corticosteroids is quite positive.[3-6] The techniques of aspiration and injection are easily mastered. These techniques are generally safe and effective when appropriate guidelines are followed.[1] **Injection treatment does not replace cessation or modification of the offending activity if identified.**

Injection therapies are frequently performed based on anatomic landmarks. Ultrasound (US) can be used to guide injections. **US-guided injections are safer and allow the Emergency Physician to visualize the needle during the procedure when done in the long axis and ensure delivery of the medication to the appropriate area. The use of US decreases complications and increases patient satisfaction.**[7] This chapter uses landmarks and US-guided injections to provide relief of the patient's symptoms.

ANATOMY AND PATHOPHYSIOLOGY

Bursae are round, fluid-filled, pad-like sacs or cavities. There are approximately 160 bursae in the body. They are usually located at sites of friction (e.g., over joints or areas where tendons pass over bony prominences). Bursae are lined with a synovial membrane and contain synovial fluid. Their primary purpose is to reduce friction when movement occurs and to provide a mechanical advantage for tendon function. Bursitis often results from trauma, chronic overuse, and inflammatory arthritis (e.g., crystal deposition, infection, and rheumatoid arthritis).

Tendons are fibrous connective tissue bands attaching muscles to bones. A synovial sheath containing synovial fluid surrounds most tendons. Tendons mainly transmit forces from muscle to the skeleton. Pathologic findings are typified by fibrinoid degeneration, inflammation, and mucoid degeneration.[8]

Bursitis and tendonitis are often grouped together because the history, symptomatology, physical examination findings, and treatment for these two inflammatory processes often coincide. **Corticosteroid injections serve to decrease inflammation, provide pain control, and promote healing. The goal of injection into tendon sheaths and bursae is to attain concentrated steroid levels to maximize the local anti-inflammatory effect while minimizing systemic effects.** Bedside US can assist with differentiating between bursitis and tendonitis, and decrease the risk of iatrogenic adverse effects.

INDICATIONS

Perform injections of corticosteroids for an inflammatory bursitis or synovitis when systemic therapy is contraindicated, as an adjunct to physical therapy, or as an adjunct to systemic therapy. Many inflammatory conditions (e.g., articular and nonarticular processes) are improved with corticosteroid injection therapy.[1,2,9,10] The articular processes improved with injection therapy include crystalloid arthropathies, gout, neuritis, osteoarthritis, pseudogout, rheumatoid arthritis, and spondyloarthropathies. The nonarticular processes improved with injection therapy include adhesive capsulitis, bursitis, entrapment syndromes, epicondylitis, ganglion cysts, neuritis, periarthritis, plantar fasciitis, tendonitis, tenosynovitis, and trigger points.

CONTRAINDICATIONS

Assess the absolute and relative contraindications when contemplating corticosteroid injection therapy. Absolute contraindications include an adjacent acute fracture, allergy or anaphylaxis to local anesthetics, bacteremia, joints containing a prosthesis, overlying cellulitis, septic arthritis, and unstable joints.[11] Relative contraindications include anticoagulant therapy, coagulopathy, joints that are inaccessible, joints requiring radiographic guidance to ensure proper needle placement, joints with loose bodies, labral tears, meniscal tears, multiple sclerosis, or greater than three injections annually into a weight-bearing joint. Consider systemic conditions (e.g., cardiac failure, diabetes, hypertension, and renal failure) that may be affected by the injection of corticosteroids prior to performing injection therapy.

EQUIPMENT

GENERAL SUPPLIES

- Povidone iodine or chlorhexidine solution
- Sterile gloves
- Sterile drapes and towels
- Needles, various gauges, 1.5 to 2 inches long
- Syringes (1, 5, and 10 mL)
- Injectable steroidal preparation (**Table 92-1**)
- 1% and 2% lidocaine without epinephrine

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TABLE 92-1 Corticosteroid Preparations Available for Injection

Generic name	Trade name	Strength (mg/mL)	Relative potency	Dose range (mg)	Biological half-life (h)
Hydrocortisone acetate	Cortef, Solu-Cortef	25	1	12.5–100	8–12
Triamcinolone acetonide	Kenalog – 10	10	2.5	4.0–40	18–36
	Kenalog – 40	40	10.0		
Triamcinolone hexacetonide	Aristospan	20	8	4.0–25	18–36
Dexamethasone acetate	Decadron, Hexadrol, Dexone	4, 8	20–30	0.8–4.0	36–54
Betamethasone sodium phosphate	Celestone	6	20–30	1.5–6.0	36–54
Methylprednisolone acetate	Medrol, Depo-Medrol, Solu-Medrol	20, 40, 80	5, 10, 20	4.0–30	18–36

- 0.25% and 0.5% bupivacaine without epinephrine
- 1% mepivacaine without epinephrine
- Adhesive bandages

ULTRASOUND SUPPLIES

- US machine
- High-frequency linear transducer
- Sterile transducer cover
- Sterile towels

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative. Obtain a written informed consent to perform the procedure. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Position the patient so that they are comfortable and the injection site is easily accessible. Identify the injection site using the appropriate anatomic landmarks. It may be necessary to outline structures with a skin marker when landmarks are difficult to palpate. Clean any debris and dirt from the skin. Apply povidone iodine or chlorhexidine solution over the injection site and surrounding skin. Allow it to dry. Sterile drapes and gloves are recommended for the novice but are not necessary depending on operator experience.[12] Apply sterile drapes or towels to delineate a sterile field. **A no-touch technique is indicated if a sterile field is not created.**

Prepare the US transducer if using US with the procedure. Have an assistant apply US gel over the footprint of the transducer. Instruct the assistant to place the gel-coated US transducer in the sterile cover while you hold the cover. Have the assistant place sterile ultrasound gel over the transducer or the patient.

TECHNIQUES

Medication dosing in bursae and soft tissue injections is influenced by the bursa size, the presence or absence of synovial fluid, the presence or absence of edema, the severity of synovitis, and the steroid preparation selected.[13-15] **Table 92-1** summarizes the characteristics of commonly available corticosteroid preparations. The duration of action is chiefly dependent on the solubility of the preparation. A dose of 20 to 30 mg of methylprednisolone acetate or equivalent is appropriate for large spaces (e.g., the olecranon, subacromial, and trochanteric bursae). This can be increased to 30 to 40 mg if a large amount of synovial fluid is present. A dose of 10 to 20 mg is appropriate for intermediate-size bursae (e.g., heel, knee, and wrist). A dose of 5 to 15 mg is appropriate for tendon sheaths.

Triamcinolone hexacetonide and acetonide are the least soluble and most potent preparations available. The local effects of these steroid preparations may take several days to be noticed but will last weeks or months. More soluble preparations such as hydrocortisone acetate have effects that last a few days. Many prefer triamcinolone due to its prolonged duration of action.

Corticosteroids are often mixed with a local anesthetic solution prior to injection. The local anesthetic solution provides immediate pain relief for the patient. It confirms that the injection was placed at the appropriate anatomic site. Consider using higher concentrations of local anesthetics (e.g., 2% lidocaine instead of 1%) if less volume is needed for the injection.

The sites for joint injections are typically based on anatomic landmarks when superficial bursitis, tendonitis, and large effusions are involved. US may be helpful if available and depending on experience when deep bursae are involved or the target structure is difficult to reach.[8,16,17] Deep injections requiring US guidance may require a specialist. The procedure can be time consuming and difficult to perform for the Emergency Physician who may not possess the skills and experience with US-guided procedures. These injection procedures can be performed with experience.[8,16,17] An Emergency Physician can guide the needle to the exact location to inject if they can find the inflamed bursa or tendon using US.

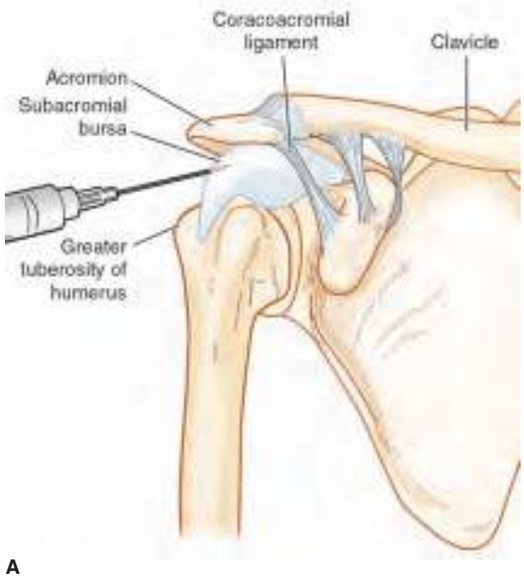
SUBACROMIAL BURSITIS

The subacromial bursa lies between the rotator cuff muscles inferiorly (i.e., supraspinatus, infraspinatus, teres minor, and subscapularis muscles) and the overlying acromion, teres major muscle, and deltoid muscle.[18] It cushions the coracoacromial ligament from the supraspinatus muscle. The subacromial space contains the long head of the biceps, the rotator cuff tendons, and the subacromial bursa. The syndromes of calcific tendonitis, supraspinatus tendonitis, and subacromial bursitis are so similar that the signs of each are difficult to distinguish. Anatomic proximity may cause associated irritative inflammation of adjacent structures so that these conditions often overlap and coexist. Pain from these syndromes is elicited by shoulder abduction.

Patients often present holding the affected arm in a protective fashion against the chest wall. The classic sign of subacromial bursitis is tenderness over the greater trochanter that disappears with arm adduction.[18] The "subacromial painful arc" is painful active abduction of the shoulder with maximal pain occurring between 70° and 100° of abduction. This differs from acromioclavicular joint inflammation which results in a painful arc from 120° to 180° of shoulder abduction.

Three techniques are described to inject the subacromial bursa: the lateral approach, the anterior approach, and the posterior approach. The lateral approach is the most commonly used technique. Some prefer the anterior or subcoracoid approach provided that the Emergency Physician has good familiarity with the anatomic landmarks. This approach is more difficult for the less experienced to perform.

Place the patient seated upright or supine on a gurney. Palpate the indentation under the acromion process of the scapula for the lateral approach. This indentation is located between the acromion process and the greater tuberosity of the humerus. Fill a syringe with



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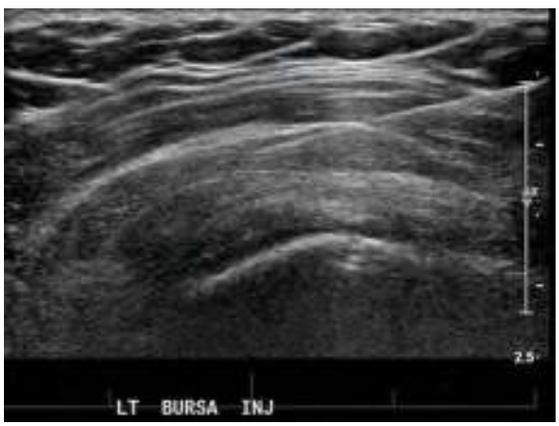


B

FIGURE 92-1. Lateral approach for subacromial bursitis. ***A.*** Schematic illustration. ***B.*** US image. (Courtesy of Dr. George Koulouris, MD, Melbourne Radiology Clinic.)

1 mL of local anesthetic solution and 1 mL of a selected corticosteroid such as dexamethasone. Apply a 22 to 25 gauge needle to the syringe. Insert the needle into the indentation and direct it superomedially. Advance the needle until the tip touches the inferior surface of the acromion (**Figure 92-1*A***). Inject the steroid-anesthetic mixture. Withdraw the needle and apply a bandage.

US can be used to perform the injection (**Figure 92-1*B***).[18,19] Identify the subacromial bursa with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the bursa. Inject the steroid-anesthetic mixture under US guidance.

Alternative approaches include the anterior and posterior approaches. Position the patient as above for the anterior approach. Identify the coracoid process of the scapula. Insert the needle 1.5 cm lateral to the coracoid process. Direct the needle horizontally and posteriorly. Advance the needle approximately 2.5 cm to gain direct access to the subacromial space. Inject the steroid-anesthetic mixture. Withdraw the needle and apply a bandage.

The posterior approach may be used. The distance from the coracoacromial ligamentous arch in this approach is great and may limit efficacy. Place the patient sitting upright with the affected forearm resting in their lap. Palpate the most lateral point of the acromion process posteriorly. Insert the needle at this landmark. Aim the needle toward the center of the humeral head and at an upward angle of 10° (**Figure 92-2**). Advance the needle 3 to 5 cm until the bursa is entered. Inject the steroid-anesthetic mixture. Withdraw the needle and apply a bandage.

FIGURE 92-2. Posterior approach for subacromial bursitis.

SHOULDER IMPINGEMENT SYNDROME

A shoulder impingement syndrome results from rotator cuff, particularly the supraspinatus muscle, compression between the humerus and the coracoacromial arch.[18,20] This is part of a pathophysiologic continuum with an endpoint of complete rotator cuff rupture in some cases. A history of overhead activities (e.g., painting a ceiling) is common. The clinical hallmark is a painful arch of abduction from 60° to 120°. Pain typically begins at 60° to 70° of abduction and is maximal from 100° to 120°. Tenderness at the tendon insertion site over the greater tuberosity of the humerus may be present.

The impingement injection test may separate the pain of impingement from other causes of shoulder pain. Prevent scapular rotation by holding the patient's scapula against their rib cage. Extend the patient's affected arm in forced forward elevation. Pain with this movement may signify numerous inflammatory shoulder conditions. Repeat this maneuver after injecting 10 mL of local anesthetic solution beneath the anterior acromion process. The absence of pain after local anesthetic injection defines an impingement syndrome.

Place the patient sitting upright or supine on a gurney. Identify the coracoacromial ligament, which is found by palpating the coracoid process and the tip of the acromion process. The coracoacromial ligament connects these two bony points (**Figure 92-3**). This

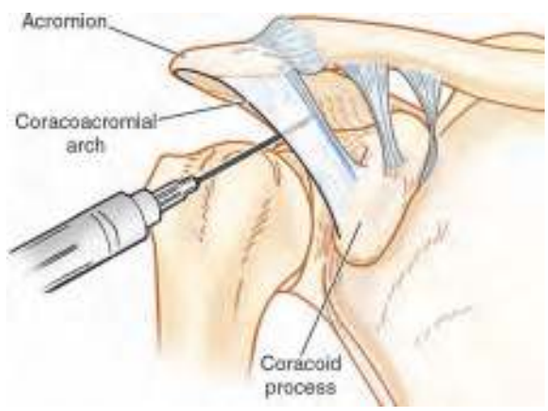


FIGURE 92-3. Injection for painful arch (impingement) syndrome.

ligament is a thick, dense, and fibrous band. Fill a syringe with 80 mg of triamcinolone and 2 mL of bupivacaine or mepivacaine. Apply a 22 to 25 gauge needle to the syringe.[21] Insert the needle under the coracoacromial arch and inject the steroid-anesthetic mixture (**Figure 92-3**). **The plunger should depress easily and without resistance. Forceful injection indicates that the tip of the needle is within the rotator cuff tendons.** Advance the needle another 0.5 cm and reattempt the injection, seeking minimal resistance. Withdraw the needle and apply a bandage.

BICIPITAL TENDONITIS

The long head of the biceps tendon passes through the bicipital groove of the humerus (**Figure 92-4**). The inflamed biceps tendon is tender to palpation along the anterior humerus.[22] Yergason's test is a clinical indicator of bicipital tendonitis.[22] Flex the patient's elbow 90°. Grasp the patient's affected arm as if shaking hands. A positive test is the elicitation of pain in the biceps muscle while providing resisted supination to the patient's hand. Lipman's test is another clinical indicator of bicipital tendonitis.[22] Tenderness of the bicipital tendon as it is rolled or plucked within the bicipital groove is considered a positive test. Pain causes restricted motion. Shoulder elevation will aggravate the patient's symptoms.

Place the patient seated with the affected arm externally rotated 20°. The bicipital groove and tendon are now pointing directly anterior. Fill a syringe with 10 to 15 mg of triamcinolone and 2 mL of local anesthetic solution. Apply a 22 to 25 gauge needle to the syringe. Palpate the bicipital tendon and identify the point of maximal tenderness. Insert and direct the needle into the tendon sheath and aimed toward the border of the bicipital groove at the site of maximal tenderness (**Figure 92-4*A***). Inject one-third of the dose into the peritendinous space (**Figure 92-4*A-1***). **Confirmation of the needle placement within the tendon sheath is made by free flow of the steroid-anesthetic mixture with minimal resistance. Difficulty depressing the plunger indicates that the tip of the needle is within the tendon.** If resistance to injection occurs, withdraw the needle slightly and aim more parallel to the tendon to allow penetration of the sheath and not the tendon substance. Withdraw the needle to just under the skin and redirect it 2.5 cm superiorly. Touch the border of the bicipital groove and inject another one-third of the dose (**Figure 92-4*A-2***). The final one-third of the dose is deposited by again withdrawing the needle to the subcutaneous area and redirecting the tip 2.5 cm inferiorly to the first injection site and touching the border of the bicipital groove (**Figure 92-4*A-3***). Withdraw the needle and apply a bandage.

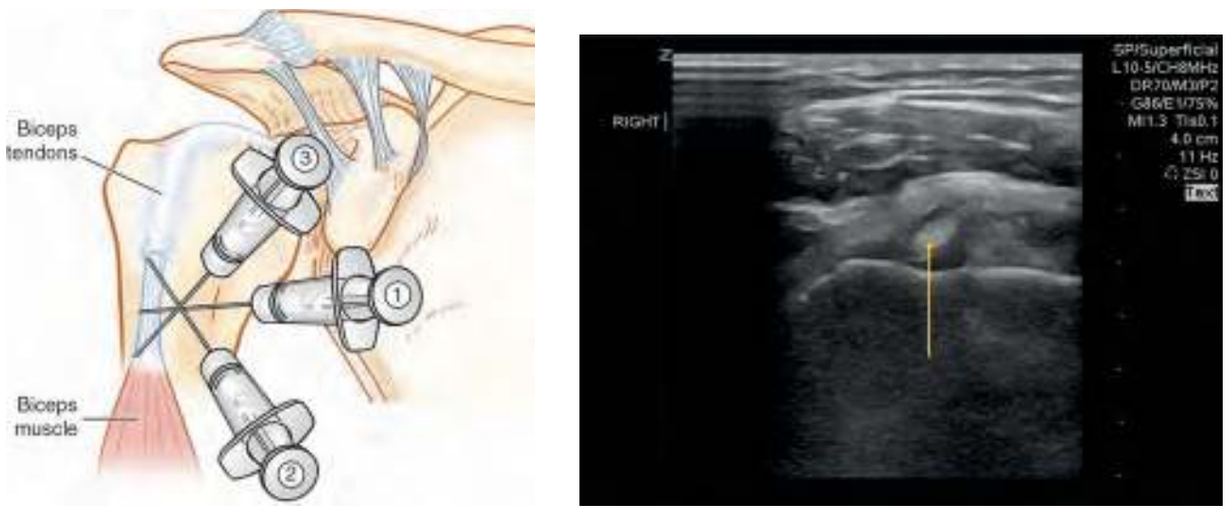
US can be used to perform the injection (**Figure 92-4*B***).[22] Identify the biceps tendon with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the biceps tendon. Inject the steroid-anesthetic mixture under US guidance.

LATERAL EPICONDYLITIS (TENNIS ELBOW)

Lateral epicondylitis, or tennis elbow, is pain at the origin of the wrist and finger extensor muscles.[14,23-25] Pain is elicited on palpation of the lateral epicondyle of the humerus. It is also elicited during resisted wrist extension. A history of biomechanical exposure at work, manual labor, and playing racquet sports is common.[26]

Locate the injection site by palpating the base of the lateral epicondyle with the elbow flexed 90°. Fill a syringe with 1 mL of local anesthetic solution, 1 mL of methylprednisolone, and 0.5 mL of dexamethasone. Apply a 22 to 25 gauge needle to the syringe. Alternatively, mix the local anesthetic solution with 40 mg of triamcinolone. A total volume of 2 mL is required. Insert the needle in the indentation between the lateral epicondyle and the radial head, beginning at the radial head (**Figure 92-5*A***). Slowly advance the needle toward the lateral epicondyle. **The radial nerve runs in this area, and care must be taken not to penetrate and inject the nerve.** Paresthesias and pain will be felt if the needle enters the nerve.

Inject 0.5 mL of the steroid-anesthetic mixture into the tenoperiosteal junction at the base of the lateral epicondyle. Withdraw the needle until the tip is at the level of the radial head while simultaneously infiltrating with 0.25 mL of the steroid-anesthetic mixture. Infiltrate 0.5 mL of the steroid-anesthetic mixture when the tip of the needle reaches the level of the radial head. Redirect the needle over the proximal extensor muscle bellies (**Figure 92-5*B***). Inject the remaining steroid-anesthetic mixture in a fan-like pattern over the extensor muscle bellies (**Figure 92-5*B***). Withdraw the needle and apply a bandage.



A B

FIGURE 92-4. Injection for bicipital tendonitis. ***A.*** Schematic illustration. ***B.*** US image. The hyperechoic ovoid structure in the center of the screen is the biceps tendon (*arrow*) in short axis with fluid surrounding it (i.e., the "halo sign"). (Courtesy of Srikar Adhikari, MD.)

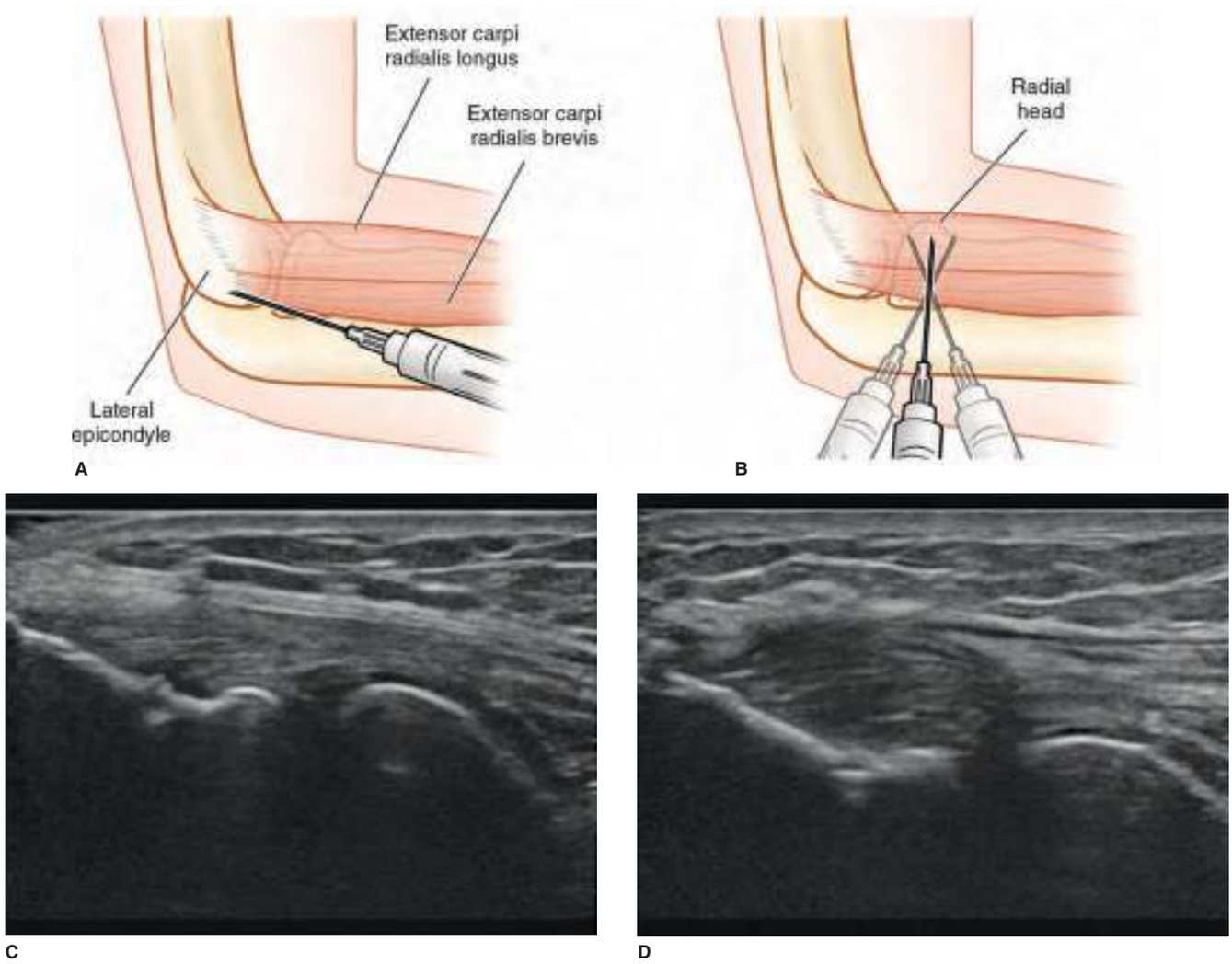


FIGURE 92-5. Injection for lateral epicondylitis. ***A.*** The needle is inserted at the level of the radial head and advanced to the base of the lateral epicondyle. ***B.*** The needle is redirected in a fan-like pattern over the muscle bellies. ***C.*** Normal US image. ***D.*** Thickened extensor tendons at insertion. (US images courtesy of www.ultrasoundcases.info.)

US can be used to perform the injection (**Figures 92-5*C* and 92-5*D***).[23,24,27] Identify the origin of the extensor muscle bellies with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the area. Inject the steroid-anesthetic mixture under US guidance.

MEDIAL EPICONDYLITIS (GOLFER'S ELBOW)

Patients affected with medial epicondylitis, or golfer's elbow, are most commonly Little League pitchers, golfers, and bowlers.[14,28] Pain is felt in the medial aspect of the elbow upon flexion and supination of the wrist.[28] Tenderness is elicited on palpation just distal to the medial epicondyle. Exclude an avulsion fracture of the medial epicondyle, a compression fracture of the subchondral bone of the lateral condyle, or a radial head compression fracture in children with nonfused epiphyses. Radiographs are suggested to exclude these causes.

Place the patient supine on a gurney. Place the arm in 90° of external rotation and abducted 90°. Flex the elbow 90°. Prepare a steroid-anesthetic mixture similar to that used for lateral epicondylitis. Fill a syringe with 1 mL of local anesthetic solution, 1 mL of methylprednisolone, and 0.5 mL of dexamethasone. Apply a 22 to 25 gauge needle to the syringe. Alternatively, mix the local anesthetic solution with 40 mg of triamcinolone. Identify by palpation the volar surface of the medial epicondyle. Insert the needle 2 cm proximal to the medial epicondyle and advance it distally to the tenoperiosteal region (**Figure 92-6*A***). **The ulnar nerve is better protected than the radial nerve as it runs posterior to the epicondyle. Proximal needle insertion prevents striking the ulnar nerve.** Paresthesias and pain are indicators of nerve penetration. Inject 1.5 mL of the steroid-anesthetic mixture over the medial epicondyle while withdrawing the needle. Withdraw the needle and apply a bandage.

US can be used to perform the injection (**Figures 92-6*B* and 92-6*C***). Identify the flexor muscle bellies with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the area. Inject the steroid-anesthetic mixture under US guidance.

OLECRANON BURSITIS

Olecranon bursitis is usually sterile even though the olecranon bursa is the most frequent site of a septic bursitis.[29,30] The bursa is located subcutaneously overlying the olecranon process of the

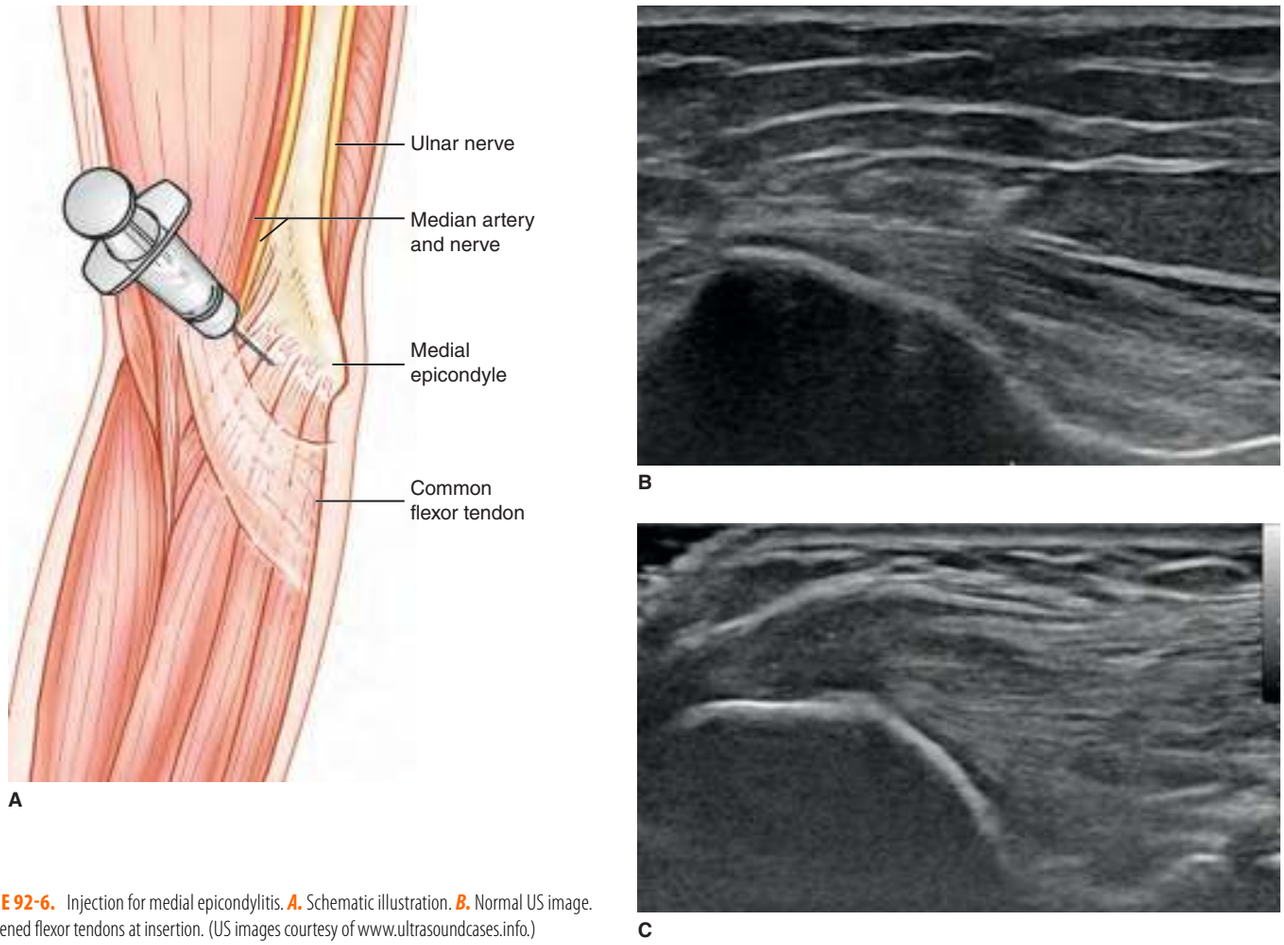


FIGURE 92-6. Injection for medial epicondylitis. ***A.*** Schematic illustration. ***B.*** Normal US image. ***C.*** Thickened flexor tendons at insertion. (US images courtesy of www.ultrasoundcases.info.)

ulna. Olecranon bursitis is not very painful except for the discomfort due to bursal expansion.[29,30] An enlarged olecranon bursa may limit elbow extension. Studies have demonstrated that corticosteroid injection is superior to oral regimens for resolution of bursal inflammation.[21] Simple aspiration without corticosteroid injection is often followed by reoccurrence. **Aspiration with fluid analysis is recommended before corticosteroid injection unless infection can be ruled out clinically.**

Seat the patient upright with their elbow flexed 90°. Fill a syringe with 30 to 40 mg of triamcinolone and 1 mL of local anesthetic solution. Insert an 18 gauge needle on an empty syringe into the most dependent aspect of the bursal sac. Aspirate the bursal fluid to drain it completely. The bursa may be "milked" by palpation and compression of the tissues toward the draining needle. Hold the needle securely. Remove the syringe while the tip of the needle remains within the bursa. Attach the syringe containing the steroid-anesthetic mixture. Inject the steroid-anesthetic mixture. Withdraw the needle and apply a bandage. Wrap the elbow region with an elastic compression bandage (e.g., Jones compression dressing) for 7 to 10 days. Instruct the patient to limit elbow movement for 7 to 10 days to prevent reaccumulation of the fluid.[31]

US can be used to perform the injection (**Figures 92-7*A* and 92-7*B***).[30] Identify the olecranon bursa with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the bursa. Inject the steroid-anesthetic mixture under US guidance.

DE QUERVAIN'S TENOSYNOVITIS

De Quervain's tenosynovitis is an inflammation of the tendon sheaths of the abductor pollicis longus and extensor pollicis brevis muscles as they cross the wrist (**Figure 92-8*A***).[14,32] The precise cause is unknown. Excessive friction resulting from overuse of the thumb and wrist (e.g., repetitive and excessive gripping) may be a possible etiology. Cases have been seen in bricklayers, golfers, piano players, and those who sew. Pain is elicited by the classic Finkelstein test. The patient is asked to make a fist while flexing the thumb across the palm of the hand. The patient is then asked to sharply ulnar deviate the wrist (**Figure 92-8*B***). Palpation along the course of the tendon will also cause pain.

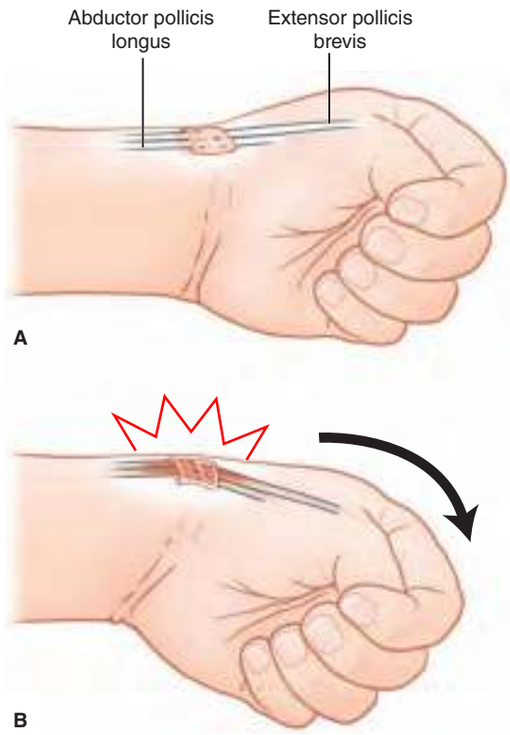
Fill a syringe with 40 mg of triamcinolone and 1 to 2 mL of mepivacaine or bupivacaine. Apply a 25 gauge needle to the syringe. Bending the needle approximately 30° at its base makes it easier to negotiate the area and insert the needle alongside the tendon.[31] Introduce the needle through the skin overlying the point of maximal tenderness. This is usually just distal to the radial styloid process (**Figure 92-9**). Inject the steroid-anesthetic mixture into the tendon sheath. **Resistance to injection signifies that the tip of the needle is within the tendon.** Withdraw the needle slightly and reinject, feeling for the loss of resistance. Withdraw the needle and apply a bandage. Apply a light thumb splint for approximately 10 days. The splint is especially useful at night or when there is significant activity.

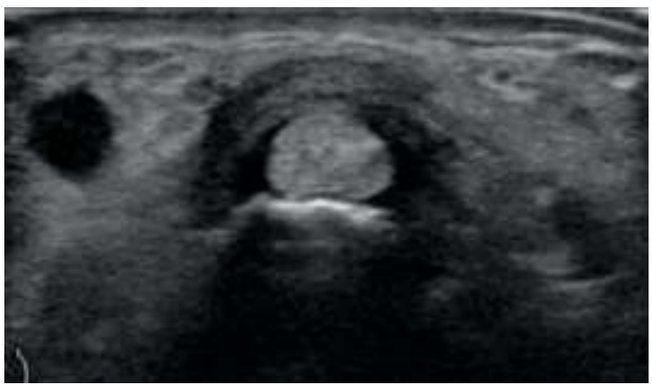
US can be used to perform the injection (**Figure 92-8*C***).[32] Identify the inflamed tendon with US. Prepare the skin and US

A

B

FIGURE 92-7. Olecranon bursitis. ***A.*** Sagittal US image. ***B.*** Transverse US image. The bursa is markedly enlarged and measured. (Courtesy of Srikar Adhikari, MD.)





C

FIGURE 92-8. De Quervain's tenosynovitis. ***A.*** Anatomy. ***B.*** Finkelstein test. ***C.*** US image. Note the fluid surrounding the affected tendon sheath and the thickened retinaculum. (US image courtesy of www.ultrasoundcases.info.)

transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the tendon. Inject the steroid-anesthetic mixture under US guidance.

TROCHANTERIC BURSITIS

Trochanteric bursitis typically affects women in the fourth to sixth decades of life.[29] It also occurs in ballet dancers from overuse, in runners, or from trauma to the hip. Hip pain often prevents the patient from sleeping on the affected side. Pain is severe when ambulating up any stairs. The deep trochanteric bursa lies between the tendon of the gluteus maximus and the greater trochanter of the femur (**Figure 92-10**). Another bursa lies between the gluteus medius and the greater trochanter (**Figure 92-10**). Pain is elicited on palpation of the greater trochanter of the femur. Pain can be reproduced by hip adduction in superficial bursitis or on a resisted active abduction in deep bursitis.[29]

The principal bursa lies between the gluteus maximus and the posterolateral prominence of the greater trochanter. Pain may radiate from the greater trochanter down the lateral or posterior thigh and mimics sciatica or hip joint disease. Passive range of motion of the hip is nearly painless in the trochanteric bursitis in contrast to the previous listed syndromes. Active abduction of the affected hip while the patient is lying on the unaffected side increases symptoms. Pain is also elicited by abduction and external rotation of the hip.

Place the patient prone on a gurney. Fill a syringe with 80 mg of triamcinolone and 3 to 10 mL of local anesthetic solution. Apply a 22 to 23 gauge needle on the syringe. Insert the needle at the point of maximal tenderness and aimed toward the greater trochanter. Advance the needle until the tip strikes the greater trochanter. Withdraw the needle 1 to 2 mm and inject the steroid-anesthetic mixture. Withdraw the needle and apply a bandage.

US can be used to perform the injection (**Figures 92-11*A* and 92-11*B***).[33,34] Identify the trochanteric bursa with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the bursa. Inject the steroid-anesthetic mixture under US guidance.

FIGURE 92-9. Injection for De Quervain's tenosynovitis.

ISCHIAL BURSITIS

The ischial bursa lies between the ischial tuberosity and the overlying gluteus maximus (**Figure 92-10**). It becomes inflamed from trauma or prolonged sitting on a hard surface. Pain may radiate down the back of the thigh and mimic sciatica. Pain can be elicited by applying pressure over the ischial tuberosity.

Place the patient prone on a gurney. Fill a syringe with 30 to 40 mg of triamcinolone and 5 to 10 mL of local anesthetic solution. Apply a 22 to 23 gauge needle to the syringe. Insert the needle over the most prominent section of the ischium. Hip flexion may facilitate palpation of the ischium in obese patients. **Care must be taken not to injure the sciatic nerve.** Striking the nerve will cause paresthesias across the buttocks and down the leg. Advance the needle until it contacts the ischial tuberosity. Withdraw the needle 2 to 3 mm and inject the steroid-anesthetic mixture. Withdraw the needle and apply a bandage.

US can be used to perform the injection (**Figure 92-12**). Identify the ischial bursa with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the bursa. Inject the steroid-anesthetic mixture under US guidance.

ILIOTIBIAL BAND SYNDROME

Patients with iliotibial band syndrome present with lateral knee pain.[35-37] This condition is commonly seen in cyclists, dancers, football players, long-distance runners, and long-distance walkers. These patients have a painful limp that is exacerbated with walking or running. Climbing stairs or walking up an incline will increase their pain. Tenderness is elicited with the patient lying supine and knee flexed 90°. Instruct the patient to extend their knee and press over the lateral femoral condyle. Pain will be localized to the lateral femoral condyle. The patient will have pain at 30° of flexion as the iliotibial band slides over the condyle. A positive Renne test occurs when the patient stands with their weight on the affected leg and flexes their knee. Pain at 30° of flexion is considered a positive test.

Place the patient supine on a gurney. Fill a syringe with 80 mg of triamcinolone and 2 mL of local anesthetic solution. Apply a 22 to 25 gauge needle to the syringe. Typically, at 30° of flexion, the iliotibial band is at the midpoint of the lateral femoral condyle (**Figure 92-13*A***). Support the patient's leg in this position to bring the tendon to its most superficial point. Identify the point of maximal tenderness as the patient flexes their knee. Insert the needle perpendicular to the skin and 1 cm inferior to the point of maximal tenderness. Aim the needle superiorly. Inject the steroid-anesthetic

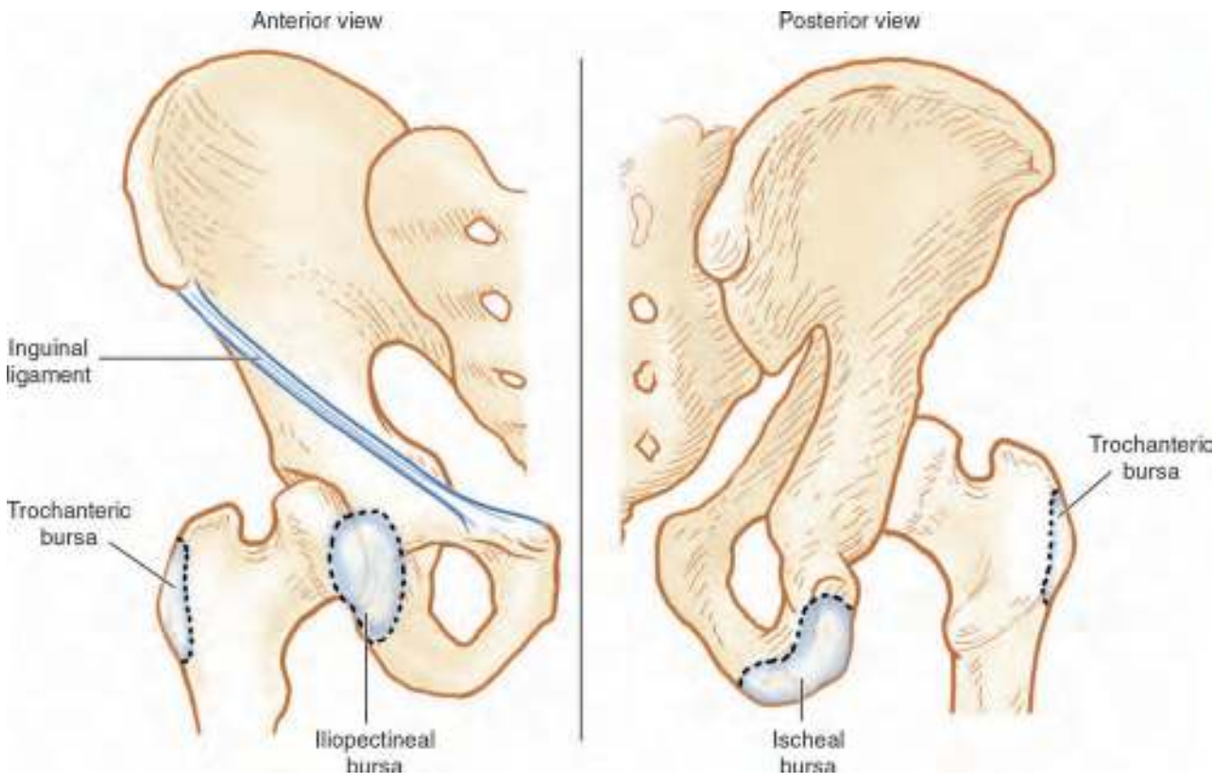
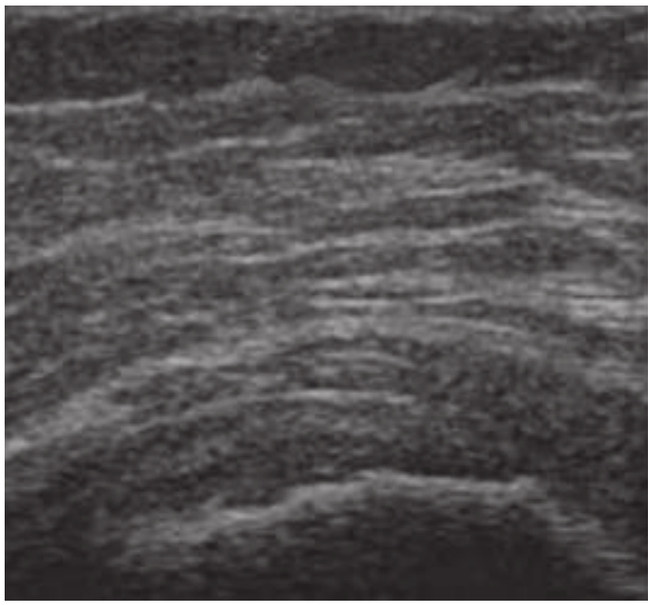


FIGURE 92-10. Selected bursa of the hip region.



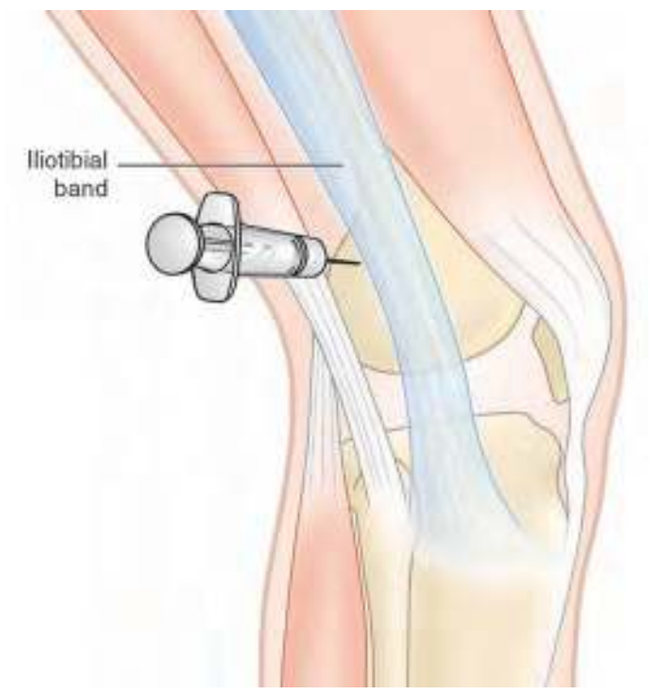
A

B

FIGURE 92-11. Trochanteric bursitis. ***A.*** Normal US image. ***B.*** US image of fluid in the bursa (arrow). (US images courtesy of www.ultrasoundcases.info.)

mixture in an arc from anterior to posterior. The goal is to deposit corticosteroid in the tendon sheath and the surrounding inflamed tissues. **Resistance to injection indicates that the tip of the needle is within the tendon.** Withdraw the needle slightly and reinject, feeling for the loss of resistance. Withdraw the needle and apply a bandage.

FIGURE 92-12. US image of ischial bursitis. Note the mixed echogenicity in the enlarged bursa (*arrow*). (US image courtesy of www.ultrasoundcases.info.)



A

B

FIGURE 92-13. Injection for iliotibial band syndrome. The knee is flexed 30° to bring the tendon to its most superficial position overlying the midportion of the lateral femoral condyle. ***A.*** Schematic illustration. ***B.*** US image. (US image courtesy of www.ultrasoundcases.info.)

US can be used to perform the injection (**Figure 92-13*B***).[36] Identify the iliotibial band with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the iliotibial band. Inject the steroid-anesthetic mixture under US guidance.

ANSERINE BURSITIS

Anserine or pes anserine bursitis is an inflammation of the bursa located 5 cm below the medial joint line of the knee at the tibial insertion of the gracilis, sartorius, and semitendinosus muscles (**Figure 92-14**).[38] The anserine bursa is superficial to the tibial insertion of the medial collateral ligament. The syndrome occurs predominately in overweight women with osteoarthritis of the knees. It may be found in equestrians. The anserine bursa will be tender to palpation.

Place the patient supine with the affected leg externally rotated. Identify the anteromedial joint line of the knee. The bursa is located inferior to the joint line at the insertion of the sartorius, gracilis, and semitendinosus tendons (**Figure 92-14**). Palpate the area of maximal tenderness at this site. Fill a syringe with 40 mg of triamcinolone and 2 to 4 mL of local anesthetic solution. Apply a 23 to 25 gauge needle to the syringe. Insert the needle and direct its tip into the bursa at the point of maximal tenderness. Inject the steroid-anesthetic mixture into the bursa. Withdraw the needle and apply a bandage.

US can be used to perform the injection (**Figure 92-15**).[38] Identify the anserine bursa with US. Prepare the skin and US transducer.

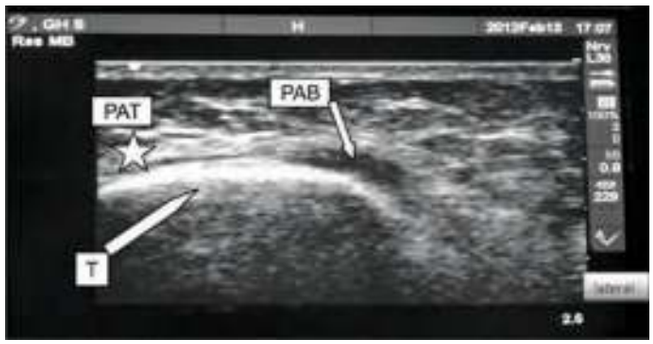


FIGURE 92-15. Anserine bursitis. US image of fluid in the pes anserine bursa (PAB), the tibia (T), and pes anserine tendon thickening (PAT, *star*). (Used with permission from Imani F, et al: Sonoanatomic variation of pes anserine bursa. *Korean J Pain* 2013; 26(3):249-254.)

Prepare the syringe with the steroid-anesthetic mixture. Reidentify the bursa. Inject the steroid-anesthetic mixture under US guidance.

PREPATELLAR BURSITIS

Prepatellar bursitis is caused by direct pressure, such as when a person is kneeling on a firm surface.[29,30,39] It is also known as nun's, rug cutter's, or housemaid's knee. Tenderness and/or crepitance is elicited by direct palpation overlying the patella. Extreme knee flexion causes pain. There is often a fluctuant, well-circumscribed,

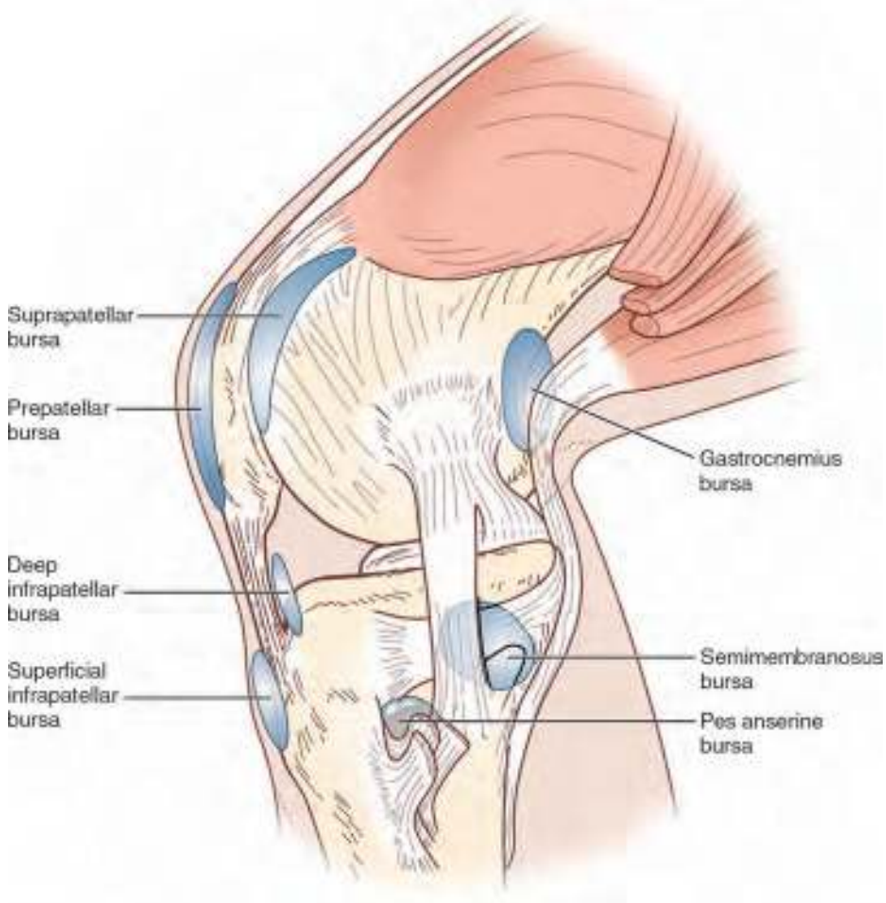


FIGURE 92-14. Bursae of the knee.

FIGURE 92-16. A transverse US image of prepatellar bursitis. The hyperechoic region to the left of the image is the patella, and the anechoic fluid superficial to that is the inflamed bursa. (Courtesy of Srikar Adhikari, MD.)

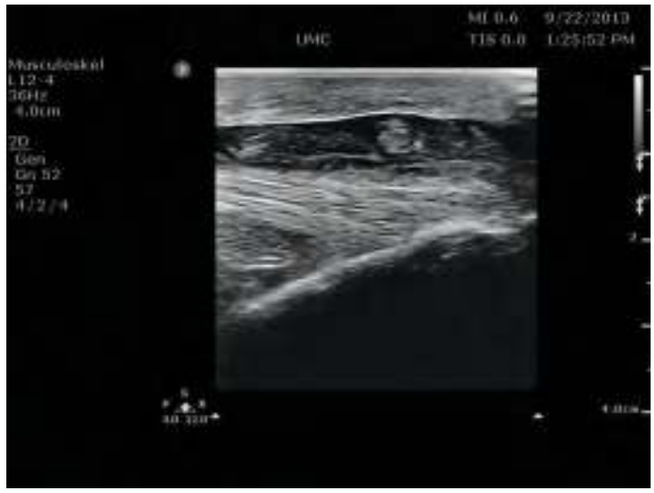


FIGURE 92-17. US image of infrapatellar bursitis. The patellar tendon is attached to the tibia in the middle of the image. The hypoechoic region superficial to it with mixed echogenicity is the inflamed deep infrapatellar bursa. (Courtesy of Srikar Adhikari, MD.)

and warm bursal pouch overlying the patella. The prepatellar space is a common site for septic bursitis. Aspiration with fluid analysis is recommended to rule out an infection before any corticosteroid injection is considered.

Place the patient supine on a gurney with the affected knee slightly flexed. Note that the bursa is very superficial and may be entered by passing the needle just through the skin and subcuticular tissues (**Figure 92-14**). Fill a syringe with 30 to 40 mg triamcinolone and 1 to 2 mL of local anesthetic solution. Apply a 23 to 25 gauge needle to the syringe.[31] Insert the needle into the bursa at the point of maximal fluctuance. Inject the steroid-anesthetic mixture into the bursa. Withdraw the needle and apply a bandage.

US can be used to perform the injection (**Figure 92-16**).[30] Identify the prepatellar bursa with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the bursa. Inject the steroid-anesthetic mixture under US guidance.

INFRAPATELLAR BURSITIS

The infrapatellar bursa has two components (**Figure 92-14**).[29] The superficial infrapatellar bursa lies between the patellar ligament and the skin. The deep infrapatellar bursa lies between the patellar ligament and the anterior tibia. Inflammation of the superficial bursa occurs due to friction from the overlying skin. Clinically, there is no pain with passive flexion. Active knee flexion and extension causes pain in the deep infrapatellar bursa. Edema and tenderness may be found on both sides of the patellar tendon.[29]

Place the patient supine on a gurney with the affected leg and knee extended. Palpate the patellar tendon. Identify the superficial inflamed bursa. Fill a syringe with 20 mg of triamcinolone and 1 mL of local anesthetic solution. Apply a 23 to 25 gauge needle to the syringe. Insert the needle into the superficial bursa and inject the steroid-anesthetic mixture. Withdraw the needle and apply a bandage.

Patients with deep infrapatellar bursitis have maximal tenderness and swelling both medially and laterally to the patellar tendon. Fill a syringe with 30 mg of triamcinolone and 1 to 2 mL of local anesthetic solution. Apply a 23 to 25 gauge needle on the syringe. **Insert the needle into the infrapatellar bursa, either medially or laterally to the patellar tendon.** Attempt to aspirate, although fluid accumulation is minimal and usually no return will be found. Inject the steroid-anesthetic mixture. Withdraw the needle and apply a bandage.

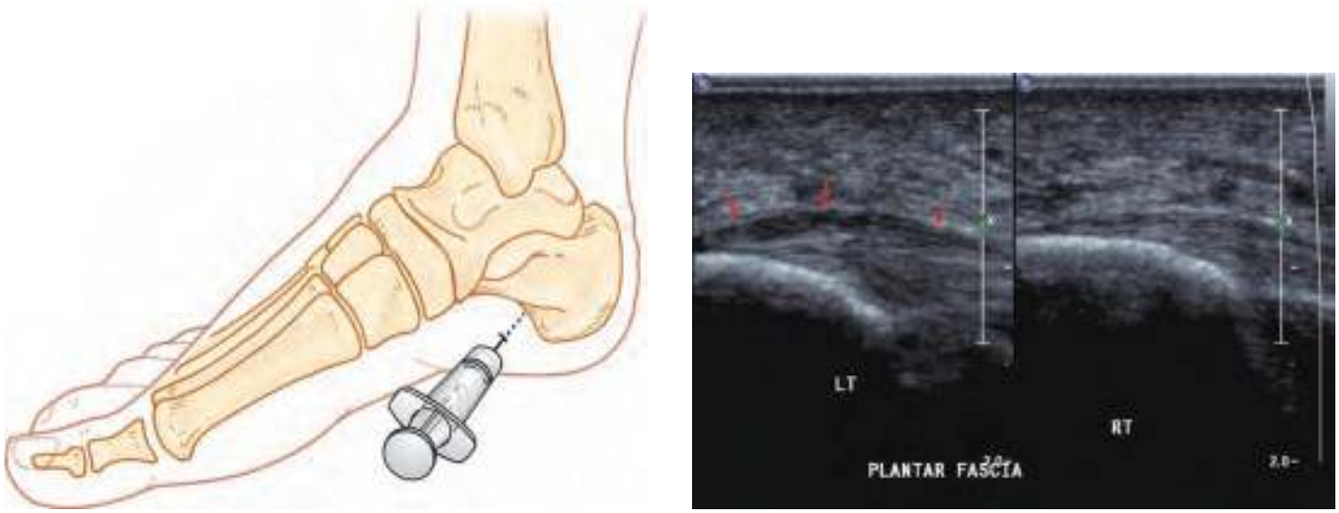
US can be used to perform the injection (**Figure 92-17**). Identify the infrapatellar bursa with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the bursa. Inject the steroid-anesthetic mixture under US guidance.

ACHILLES TENDONITIS

The Achilles tendon is the largest tendon in the body. Achilles tendonitis is a common condition that causes tightness and pain in the posterior heel region upon first awaking.[40] **The tendonitis can occur anywhere from its insertion on the calcaneus to its junction with the muscle.** This discomfort improves with ambulation. The Achilles tendon will be tender to palpation and may be visually swollen. Noninjection and nonsurgical treatments include calf stretching, ice, nonsteroidal anti-inflammatory drugs, orthotics, physical therapy, supportive shoes, and rest. **Corticosteroid injection around the Achilles tendon has been associated with tendon rupture.**[40-42] Consider reserving this injection for the Podiatrist, Rheumatologist, or Orthopedic Surgeon.[42]

US can be used to perform the injection (**Figure 92-18**). Identify the Achilles tendon with US. Prepare the skin and US transducer.

FIGURE 92-18. Achilles tendonitis. Fluid is noted around the Achilles tendon (*red dot*) consistent with Achilles tendonitis. (Courtesy of Srikar Adhikari, MD.)



A B

FIGURE 92-19. Injection for plantar fasciitis. ***A.*** Schematic illustration. ***B.*** The thickened plantar fascia (left, *red arrows*) is seen next to the normal plantar fascia (right). (US image courtesy of www.ultrasoundpaedia.com.)

Prepare the syringe with the steroid-anesthetic mixture. Reidentify the tendon. Inject the steroid-anesthetic mixture under US guidance.

PLANTAR FASCIITIS

Plantar fasciitis is a common problem presenting to the Emergency Department and the Primary Care Physician.[43,44] Patients typically complain of medial heel pain, particularly after standing for a long period of time. These patients have minimal to no swelling but feel acute tenderness to palpation over the calcaneal insertion of the plantar fascia. Maximum tenderness is palpated just beneath the spring ligament at the insertion of the plantar fascia on the calcaneus. Radiographs may demonstrate a calcaneal spur. **The presence of a spur does not correlate with plantar fasciitis.** Patients may have plantar fasciitis with or without a calcaneal spur. The optimal therapy for these patients is to elevate the heel with a felt heel pad inserted in the shoe. The patient may begin stretching exercises designed to stretch the plantar fascia. This often relieves the condition without an injection. Injection of the calcaneal insertion of the plantar fascia with a steroid-anesthetic mixture is advocated in significant cases where conservative therapy is unsuccessful.[43]

Place the patient supine on a gurney with the affected leg externally rotated. Fill a syringe with 20 mg of triamcinolone and 1 mL of local anesthetic solution. Apply a 25 gauge needle on the syringe. Insert the needle into the medial aspect of the foot and aimed just anterior to the base of the calcaneus (**Figure 92-19*A***). Advance the needle 1.5 cm and inject the steroid-anesthetic mixture. Withdraw the needle and apply a bandage. The patient should avoid weight bearing for 3 to 4 days and immediately begin oral nonsteroidal anti-inflammatory drugs. Refer to Chapter 223 for more details regarding plantar fasciitis.

US can be used to perform the injection (**Figure 92-19*B***).[45,46] Identify the plantar fascia with US. Prepare the skin and US transducer. Prepare the syringe with the steroid-anesthetic mixture. Reidentify the bursa. Inject the steroid-anesthetic mixture under US guidance.

PEDIATRIC CONSIDERATIONS

Injection techniques for pediatric patients are not different than those for adults. Smaller injected volumes may be required depending on the age and size of the child. Soft tissue corticosteroid injections should be performed by a Rheumatologist or an Orthopedic Surgeon as these injections can affect bone growth, affect cartilage growth, and can damage growth plates.

ASSESSMENT

The patient must be observed for several minutes after the injection. Reexamine the patient to compare preinjection and postinjection tenderness and mobility. The patient's symptoms should abate within a few minutes from the local anesthetic. Lack of relief indicates deposition of the steroid-anesthetic mixture away from the target structure. A second attempt may be performed if the injection site can be properly identified. Otherwise, refer the patient to their Primary Care Physician, a Rheumatologist, or an Orthopedic Surgeon for reevaluation and reassessment.

AFTERCARE

A treatment algorithm is provided in **Figure 92-20**. Instruct the patient to limit movement and weight bearing of the affected area after a corticosteroid injection. The duration of rest is dependent on the injection site. Larger and weight-bearing joints may require up to 2 to 3 weeks of rest, with range-of-motion exercises encouraged. Immobilization with splints or bandages may be necessary to prevent weight bearing. A rehabilitation program including range-of-motion exercises, stretching, and strengthening may be recommended depending on the chronicity and severity of the presenting condition.

COMPLICATIONS

Local infection is rare after corticosteroid joint injection and occurs in 1 in 17,000 to 50,000 injections.[1,10,47,48] A local reaction consisting of swelling, tenderness, and warmth may occur a few hours after injection and last up to 2 days. This is known as the "postinjection flare" or "steroid flare."[12] It is self-limited, responds to ice packs, and is treated with nonsteroidal anti-inflammatory drugs. The etiology of the steroid flare is attributed to preservatives in the steroid suspension inducing a local synovitis. Steroid flares occur in approximately 2% of patients injected with corticosteroids.[1]

Tendon rupture is a theoretical complication thought to be due to corticosteroids weakening the collagen matrix. Tendon ruptures

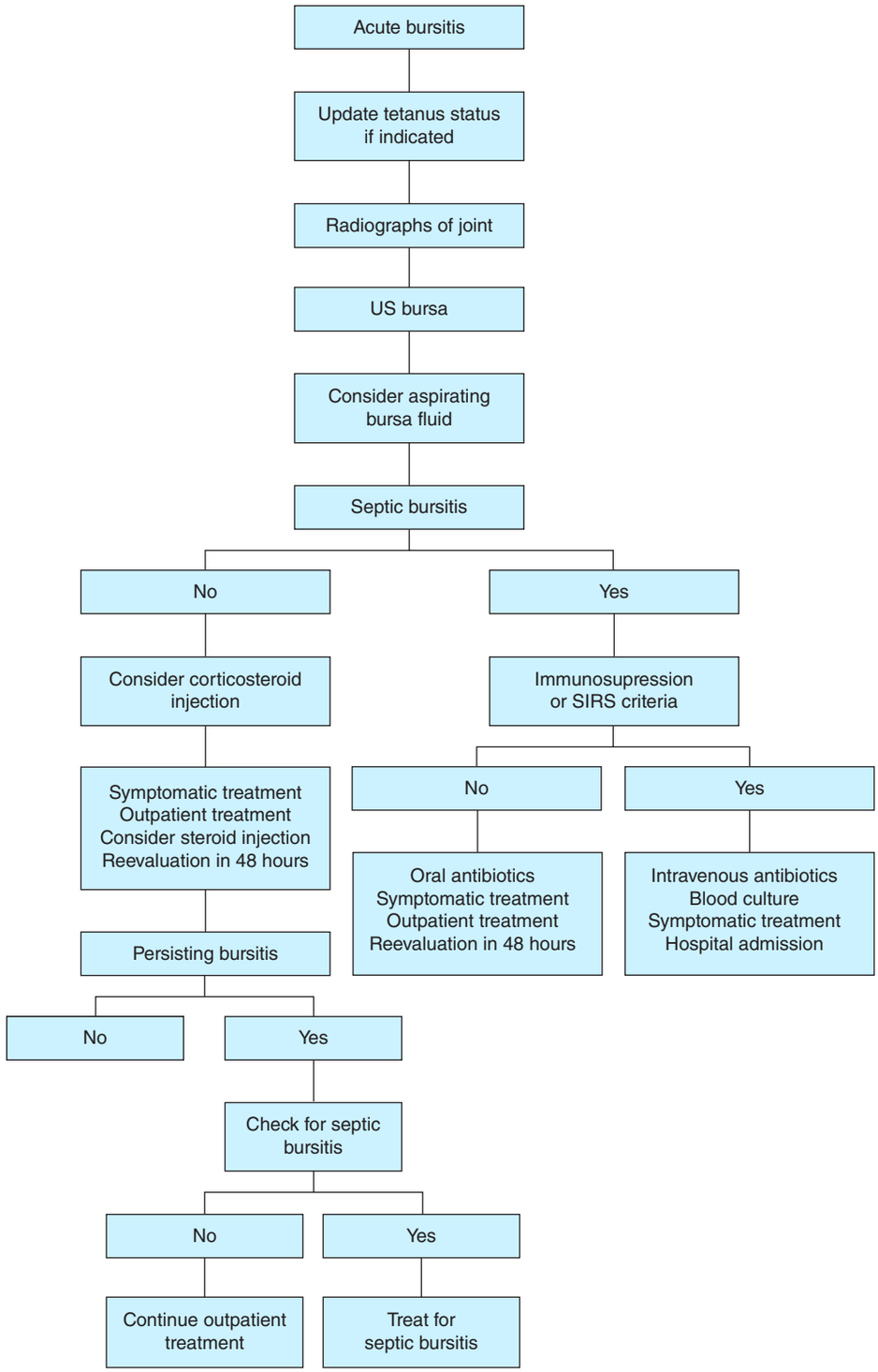


FIGURE 92-20. Treatment algorithm for bursitis. SIRS, systemic inflammatory response syndrome.

after corticosteroid injections have been reported, but direct causality has not been established.[42,49-52] Rupture is more likely if the injection is made into the tendon matrix rather than the synovial sheath, if the patient does not rest the tendon appropriately after the injection, or with multiple repeat injections.

Subcutaneous fat atrophy and atrophy of the overlying skin may develop if the steroid is injected less than 5 mm beneath the skin surface. A depigmentation in darker-skinned individuals may occur due to superficial steroid injections. The depigmentation usually resolves spontaneously over a period of 6 months to 1 year.

SUMMARY

Local corticosteroid injections are useful diagnostic and therapeutic adjuncts for the Emergency Physician. Many patients with an inflammatory bursitis or tendonitis will benefit greatly from these simple

and effective injections (**Figure 92-20**). Mastery of the varied techniques is quite easy. They require a familiarity with the indications, the local anatomy, and the injectable corticosteroid preparations. Complications associated with the injection of a steroid-anesthetic mixture are minimal if it is not injected into an infected area.

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93 Compartment Pressure Measurement

Danielle D. Campagne and Scott T. Owens

INTRODUCTION

The ability to diagnose a compartment syndrome is a critical skill for the Emergency Physician (EP). Early identification of a compartment syndrome can enable the appropriate treatment and may facilitate limb salvage. **A compartment syndrome begins when pressure within a myofascial compartment increases to the point**

that it results in diminished blood flow.[1,2] A compartment syndrome has been classically described in the early literature as a Volkmann ischemic contracture following vascular insufficiency in the forearm.[3] A compartment syndrome is described as the 6 Ps (i.e., pain out of proportion, pallor, paralysis, paresthesias, pressure, and pulselessness).

A compartment syndrome can occur in almost any muscle group contained within a confined fascial space. Common locations include the leg, forearm, and gluteal area. There are many causes of a compartment syndrome. These include protracted muscle ischemia (e.g., necrosis from a contusion), swelling (e.g., fracture or volume overload), or a thrombus in a vessel that traverses the compartment. A compartment syndrome in the Emergency Department (ED) is most commonly associated with long bone fractures or blunt trauma.[4] Most compartment syndromes are caused by trauma.[2] Approximately 58% of cases are due to fractures of the tibia or forearm.[5] Other etiologies for a compartment syndrome include complications from a coagulopathy, dialysis, surgery, or states of obtundation (**Table 93-1**).[2,6-18]

Identifying a compartment syndrome in a timely fashion can be challenging. The sensitivity and specificity of manual palpation to identify a compartment syndrome are 24% and 55%, respectively.[19] Manual palpation has a positive predictive value of 19% and a negative predictive value of 63%.[19] **Manual palpation cannot be used to rule-in or rule-out a compartment syndrome.**

The hallmark symptom of a compartment syndrome is persistent and progressive pain disproportionate to the underlying cause. The pain typically increases with passive motion. **A common and dangerous mistake is to attribute the etiology of the patient's pain solely to the underlying problem (e.g., fracture or trauma).**[20,21] Other signs and symptoms associated with a compartment syndrome occur late in the disease course and include paresthesias of the involved nerve, paralysis of the involved muscle group, pallor of the skin, and diminished pulses.[22] **Waiting for the development of all the clinical signs and symptoms to manifest before considering the diagnosis increases the possibility of permanent sequelae (e.g., muscle necrosis and limb loss). Measurement of elevated tissue pressure within the muscle compartment is currently the most common and expedient objective means of diagnosing a compartment syndrome.** Compartment pressure measurement provides objective support for the diagnosis of a clinically suspected compartment syndrome and leads to treatments designed to salvage the afflicted limb.

Numerous methods have been evaluated to diagnose a compartment syndrome.[23-28] The inflammatory biomarkers and proteins released from damaged muscle are late markers or nonspecific to distinguish between muscle injury from trauma versus a compartment syndrome. Magnetic resonance imaging (MRI) is not reliable or specific to identify the early changes of a compartment syndrome. Pulse phase-locked loop (PPLL) ultrasound and near-infrared spectroscopy (i.e., changes in oxygenated hemoglobin and tissue perfusion) are diagnostic modalities that have been studied and show promise in diagnosing compartment syndromes. These are not yet viable diagnostic tools in the ED due to inadequate sensitivity and specificity.[29] Radionuclide imaging is not feasible in the ED due to its lack of specificity, the complexity of the procedure, the time required for testing, and the inability to perform repeated measurements. Pulse oximetry cannot be used to measure intracompartmental oxygenation. It can measure whether distal blood flow is present but is limited by collateral circulation and the loss of pulses being a late finding for a compartment syndrome. Laser Doppler flowmetry measures microvascular perfusion and has potential in the future when more clinical information regarding its use becomes available. Tissue ultrafiltration requires the extraction of tissue fluid and the time-consuming measurement of biomarkers. Evaluating differences in vibratory sensation using a 256 cycle tuning fork shows some promise in the diagnosis of a compartment syndrome. It cannot be used in young children, patients who cannot appropriately communicate, and those with an altered sensorium. Tissue hardness measurement techniques evaluate skin surface pressure and are in the early stages of testing. Direct nerve stimulation is not useful for the early evaluation of a compartment syndrome because nerve damage is a later finding. **None of the above techniques is an adequate substitute for the gold standard of invasive measurement, which is the focus of this chapter.**

TABLE 93-1 Etiologies of a Compartment Syndrome

Anticoagulation therapy or coagulopathy
Blast injuries
Bleeding into compartment
Burns involving muscle (i.e., electrical or thermal)
Chronic exertional compartment syndrome
Eclampsia
Envenomation
Exercise-induced
External compression
Iatrogenic
Iatrogenic closure of fascial injuries
Immobility
Improper casting or tight dressings
Infections
Infiltration of infusion fluids into muscle compartment
Intraosseous infusion leak
Myositis
Nephrotic syndrome (e.g., lower extremity swelling)
Reperfusion after ischemia
Reperfusion injury after prolonged positioning or tourniquet use
Seizures causing increased capillary permeability
Snake bite
Tetany
Trauma
Contusions
Long bone fractures
Blunt trauma (i.e., crush injury)
Vascular injury
Vasculitis
Venous thrombus

ANATOMY AND PATHOPHYSIOLOGY

The anatomy of a compartment syndrome is variable, as it can occur in any enclosed muscle group. Any muscle tissue confined by fascia, skin, or external forces (e.g., casting material) is a potential site for the development of a compartment syndrome. The muscles, nerves, and vasculature within the affected muscle group are all potentially compromised by a prolonged ischemic state followed by swelling. A basic knowledge of the anatomy of commonly affected compartments is necessary to successfully and safely perform compartment pressure measurement.

The initial imbalance of a compartment syndrome occurs between the volume and pressure within the myofascial compartment. The arterial inflow and venous outflow diminish as intracompartmental volume or pressure increases. The blood begins to be shunted via capillaries into the muscle tissue. This compensatory shunting of blood further disturbs the volume-pressure balance and results in impaired tissue oxygenation.[4,22,30]

The extent of the tissue damage is determined by the duration of ischemia. Numerous experimental studies have documented a

lack of muscle viability after 6 to 8 hours of total ischemia and a lack of nerve viability after 8 hours of total ischemia.[31,32] **Reversing ischemia before this time is crucial to restoring tissue function.** The definitive factor in the development of a compartment syndrome is the alteration of the pressure gradient between arterial and venous flow. Measurement of the intracompartmental pressures is used to determine the extent of ischemia.

The pressure of a healthy muscle compartment ranges between 0 and 8 mmHg.[33] **There is no absolute value that determines a compartment syndrome.** Consider a fasciotomy at values above 30 to 35 mmHg.[22,34,35] Several studies have confirmed that a compartment syndrome does not develop definitively at or above 30 to 35 mmHg.[36,37] A prospective study of patients with isolated lower leg fractures without clinical signs of compartment syndrome showed significant elevations in compartment pressure compared to the contralateral normal leg.[38] Other studies use a compartmental pressure that is within 10 to 30 mmHg of the patient's diastolic pressure as one of the indications for a fasciotomy.[4,31] This "delta pressure" (i.e., diastolic blood pressure minus intracompartmental pressure) has gained popularity in recent years based on literature demonstrating safety and reduction in the number of unnecessary fasciotomies.[39] A measurement within 10 to 30 mmHg of the patient's mean arterial pressure is as an indication for a fasciotomy.[40] **A compartmental pressure that is 30 mmHg or greater requires one to consider the development of a compartment syndrome and the need to perform a fasciotomy**.

Studies have investigated the correlation between the delta pressure or perfusion pressure and a compartment syndrome. A compartment syndrome is suggested if the delta pressure is less than 30 mmHg.[4,5,31] Adjunctive and noninvasive testing (e.g., T2-weighted MRI and near-infrared spectroscopy) may be helpful in equivocal cases.[41,42] There is as yet no gold standard for the diagnosis of a compartment syndrome, and it remains a clinical and operative diagnosis despite advances in testing.

A compartment syndrome can occur in any muscle group. This includes the foot, forearm, hand, intercostal spaces, and thigh, to name a few. This chapter reviews the anatomy of the two most common sites of a compartment syndrome, the leg and the forearm.

Special mention should be made of regional anesthesia with peripheral nerve blocks in the setting of acute musculoskeletal trauma. Regional anesthesia is an increasingly popular approach to the management of pain in traumatic injuries. There exists a theoretical risk of masking the pain symptoms of a compartment syndrome. This is unproven, and regional anesthesia is increasingly common in trauma. **Use extra vigilance in assessing for a compartment syndrome when regional anesthesia has been used for musculoskeletal trauma.**[43]

LOWER EXTREMITY: LEG

The lower leg consists of four distinct muscle compartments: anterior, lateral, deep posterior, and superficial posterior (**Figure 93-1**). A general understanding of the components of each compartment is important.[44]

The anterior compartment contains the four extensor muscles of the leg. These muscles function together to dorsiflex the foot. The deep peroneal nerve travels through this compartment to innervate the extensors and provide sensory innervation to the web space between the first and second toes. The anterior tibial artery travels through this compartment and provides blood flow to its contents.

The lateral compartment contains the peroneus longus and brevis muscles. Their chief function is to evert the foot, with some abduction and plantarflexion of the foot. The major nerve in the lateral compartment is the superficial peroneal nerve which supplies motor innervation to the compartment muscles and sensory innervation to the lower leg and dorsum of the foot.

A fascial layer divides the posterior muscle group into superficial and deep compartments. The superficial posterior compartment contains the muscles of plantarflexion (i.e., gastrocnemius, plantaris, and soleus). No major nerves or blood vessels travel in the superficial compartment. The deep posterior compartment contains the four deep flexor muscles (i.e., flexor digitorum longus, flexor hallucis longus, popliteus, and tibialis posterior). This group

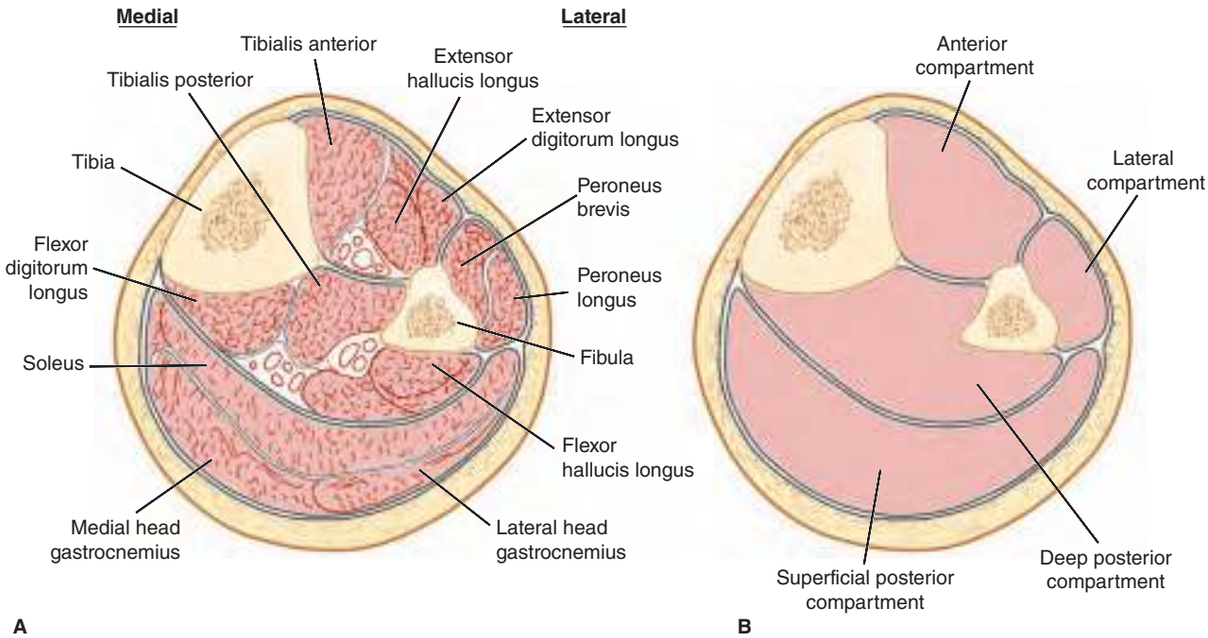

FIGURE 93-1. Cross section through the middle of the right leg demonstrating the four compartments. The size and proportion of the compartments change as one travels proximally or distally from this middle section. ***A.*** The contents of the lower leg. ***B.*** The compartments.

of muscles contributes to inverting and adducting the foot in addition to flexing the foot and toes. The primary sensory innervation is from the tibial nerve which courses through the deep posterior compartment. The tibial nerve supplies most of the muscles of the posterior compartment before dividing into several branches that provide sensory innervation to the sole of the foot. The posterior tibial artery and the peroneal artery are contained within this compartment.

Any of these compartments can suffer from ischemia. **The deep posterior compartment and the anterior compartment have the highest incidence of developing a compartment syndrome.**[37] It is difficult to simply observe the patient in the face of a normal compartmental pressure when they clinically present with increasing pain and the features suggestive of a compartment syndrome. **Obtain multiple compartmental syndrome readings and consult a Surgeon when the clinical suspicion is high but the compartmental pressures are normal**.

UPPER EXTREMITY: FOREARM

The forearm consists of three compartments that are interconnected at various levels (**Figure 93-2**).[44,45] **This interconnection is significant because release of the pressure in one compartment will reduce some of the pressure in the adjacent compartments. The volar compartment is most at risk for development of a compartment syndrome in traumatic injuries of the forearm**.

The forearm includes the volar, dorsal, and mobile wad compartments (**Figure 93-2**). The volar compartment contains the hand and forearm flexor muscles, the median and ulnar nerves, and the interosseous arteries (i.e., common, radial, and ulnar). The mobile wad contains the brachioradialis, the extensor carpi radialis brevis, and the extensor carpi radialis longus muscles. No major arteries or nerves are contained within this compartment. The radial artery and a branch of the radial nerve may sometimes lie between the mobile wad and the volar compartment. The dorsal compartment contains the hand and forearm extensor muscles, the posterior interosseous artery, and the posterior interosseous nerve.

INDICATIONS

The earliest and most reliable indication for measuring compartment pressures is the development of increasing pain in a tense and swollen muscle group.[4] This pain tends to be disproportionate to the underlying cause (e.g., fracture, soft tissue contusion, or thrombus). Pain that increases with passive motion of the affected muscles is an indication for compartmental pressure measurement. Sensory deficits and paresis of the affected muscles are two late findings for the development of compartment syndrome.[22] **The presence of palpable pulses and capillary refill do not rule out an evolving acute compartment syndrome**. The absence of pulses may suggest arterial injury or hypovolemia. **Measure and monitor all compartments at risk if the patient is obtunded or unreliable.** This usually refers to the forearm and lower leg compartments in the multiple trauma patient.

Have a very low threshold for measuring pressures within a muscle compartment. Compartmental pressures must be measured if a compartment syndrome is considered as a diagnosis. Do not rely solely on the compartmental pressure measurements to decide the need for a fasciotomy. A compartment syndrome is primarily a clinical diagnosis. Obtain pressure measurements in each compartment at risk and in at least two sites within the compartment. Perform serial measurements or continuous pressure measurement if the initial pressures are normal or only mildly elevated and concern persists. Interpret compartment pressures in the clinical context of the patient. Normal compartment pressures do not rule out the diagnosis.

CONTRAINDICATIONS

There are no absolute contraindications to the measurement of compartment pressures. Obtain compartment pressure measurements while evaluating for coexistent traumatic injuries given the importance of prompt diagnosis. The EP can still assess the compartmental pressures even though the patient may be undergoing other invasive and/or surgical procedures. **Time is of the essence.**

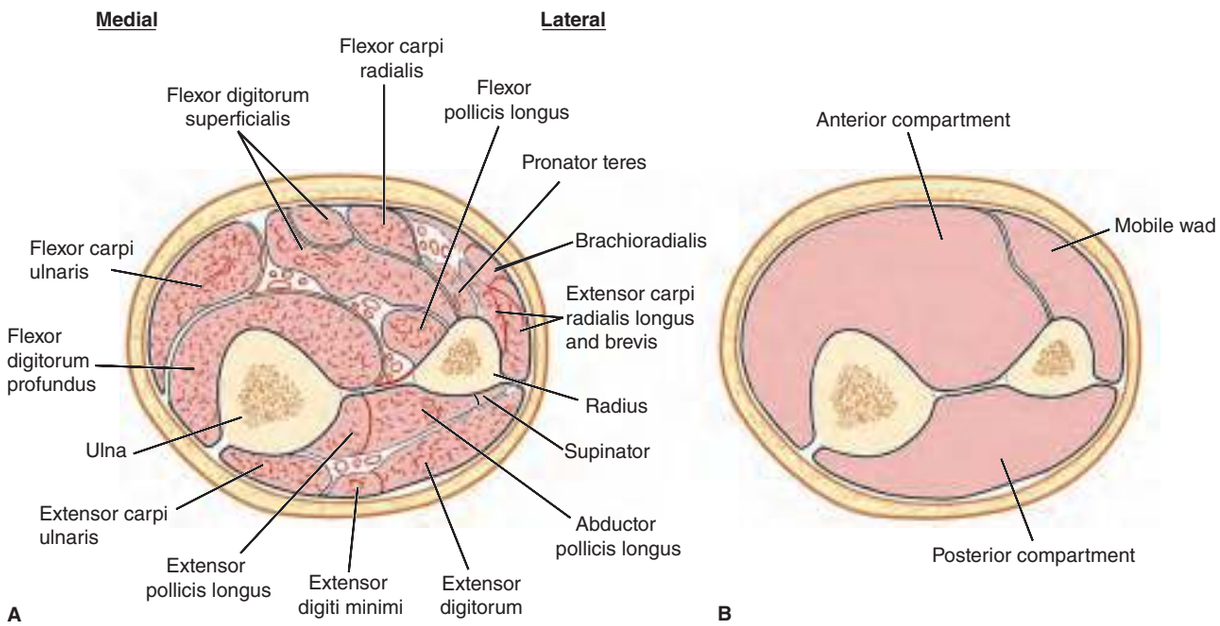

FIGURE 93-2. Cross section through the middle of the right forearm demonstrating the dorsal and volar compartments separated by the line of the radius, the ulna, and the interosseous membrane. ***A.*** The contents of the forearm. ***B.*** The compartments. The mobile wad forms a distinct muscle compartment coursing along the radius.

Postponing compartmental pressure measurements or a fasciotomy may lead to irreversible damage to the affected extremity.

EQUIPMENT

GENERAL SUPPLIES

- Povidone iodine or chlorhexidine solution
- 4×4 gauze squares
- Sterile drapes or towels
- Skin marking pen

NEEDLE (WHITESIDE) MANOMETER TECHNIQUE

- 18 gauge needles, 1.5 inches long
- 20 mL syringe
- Three-port, four-way stopcock
- Intravenous (IV) extension tubing
- Sterile saline
- Mercury manometer

STRYKER MONITOR SYSTEM

- Stryker pressure monitor unit
- Sterile, single-use, quick pressure monitor set (i.e., a needle, diaphragm chamber, and syringe)

ARTERIAL MANOMETER TECHNIQUE

- 18 gauge needle or Angiocath
- Sterile saline
- Arterial line tubing and manometer identical to that used for arterial lines (Chapter 72)

PATIENT PREPARATION

Explain the risks and benefits of the procedure to the patient and/or their representative. Discuss the low risk of bleeding, low risk of infection, and the possibility of obtaining erroneous values. Obtain a written informed consent from the patient and/or their representative if possible. Proceed after documentation of the medical necessity if the patient is unable to consent and no representative is available. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Wash away any blood, dirt, or debris on the patient's skin. Identify the landmarks for needle insertion and mark the skin puncture sites. Apply povidone iodine or chlorhexidine solution to the skin around the puncture sites and allow this to dry. **The measurement of compartment pressures is considered a sterile procedure, and the EP should don a hat, mask, sterile gloves, and a sterile gown.** Create a sterile field with surgical towels or drapes. Reidentify the landmarks for needle insertion and ensure the skin puncture sites are still marked.

The exact locations at which to measure the intracompartmental pressures are not clearly defined. There were no clearly established guidelines for determining the appropriate location for compartmental pressure measurements in patients with fractures prior to the study of Heckman and Whitesides.[46,47] The results of their study suggest that measurements be performed at the level of the fracture "as well as locations proximal and distal to the zone of the fracture." A 5 cm distance was used from the fracture site to the proximal and distal needle insertion sites. Insert the needle at the point of maximal tightness of the compartment in the absence of a fracture and at least two other sites within the compartment. General anatomic landmarks for needle insertion into the various compartments of the lower leg and forearm are described in **Tables 93-2 and 93-3** and graphically depicted in **Figure 93-3**. **The authors recommend using the highest measured intracompartmental pressure in making the decision for further intervention.**

TABLE 93-2 Needle Insertion Sites for the Compartments of the Leg

Compartment	Needle insertion site	Insertion depth (cm)
Anterior	1 cm lateral to the anterior tibial ridge and directed perpendicular to the long axis of the leg	1.0–3.0
Lateral	Just anterior to the posterior border of the fibula and directed toward the fibula	1.0–1.5
Superficial posterior	3 cm medial or lateral to a vertical line drawn through the midcalf	2.0–4.0
Deep posterior	Just posterior to the medial border of the tibia, directed posterolaterally and toward the posterior border of the fibula	2.0–4.0

TECHNIQUES

Several techniques for measuring compartmental pressures have been developed. Some use isolated intracompartmental pressure measurements while others monitor pressure continuously. Obtaining isolated intracompartmental pressure measurements is most important in the ED. The needle manometer method uses a manual manometer and a saline-air meniscus in standard IV tubing connected to a 16 gauge needle.[12] Stryker (Stryker Instruments, Kalamazoo, MI) introduced an electronic system consisting of a reusable digital pressure monitor and a single-use measurement set with a needle, diaphragm chamber, and sterile saline flush.[48] A technique involving a 16 gauge angiocatheter or needle connected by arterial line tubing to a standard manometer has been described.[49] All three of these methods will be discussed in this chapter. Data suggest that the Stryker method and the arterial manometer method are significantly more accurate, are more convenient, and require only a small number of components readily available in most EDs when compared to the needle manometer method.[50,51]

Each of these techniques may be performed with a standard straight needle. All are more accurate if used with a special side-ported needle or slit catheter, if available.[51] Similar measurement systems that introduce a wick or slit catheter into the tissue have been shown to be equally effective. Explanations of the wick and catheter techniques have been omitted as they are rarely performed in the ED.

TABLE 93-3 Needle Insertion Sites for the Compartments of the Forearm

Compartment	Needle insertion site	Insertion depth (cm)
Anterior	1.5 cm medial to a vertical line drawn through the middle of the forearm	1.0–2.0
Mobile wad	Perpendicular to the long axis of the radius and into the muscles lateral to the radius	1.0–1.5
Posterior	1–2 cm lateral to the posterior aspect of the ulna	1.0–2.0

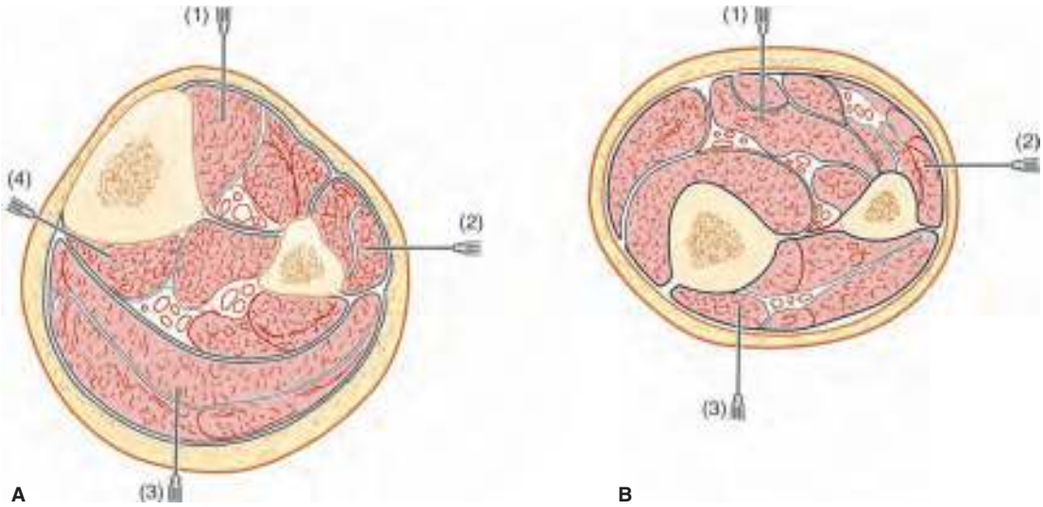

FIGURE 93-3. Needle insertion sites to measure intracompartmental pressures. ***A.*** The leg compartments: anterior (1), lateral (2), superficial posterior (3), and deep posterior (4). ***B.*** The forearm compartments: anterior (1), mobile wad (2), and posterior (3).

NEEDLE MANOMETER TECHNIQUE

Begin by setting up the system (**Figure 93-4*A***). Attach the hub of a 20 mL syringe to the middle port of a three-way stopcock. Attach one end of the IV extension tubing to one of the ports of the stopcock. Attach an 18 gauge needle to the free end of the IV extension tubing. Insert a second 18 gauge needle into a container of sterile normal saline to release the vacuum. Insert the 18 gauge needle attached to the IV extension tubing into the normal saline until the needle port is well immersed. Open the stopcock ports only to the syringe and the extension tubing. Aspirate the saline to fill one-half of the length of the IV extension tubing. **Make sure that no bubbles enter the system**. Turn the stopcock valve so that the port to the saline is closed.

Attach a second piece of IV extension tubing to the remaining open port of the stopcock (**Figure 93-4*B***). Attach the opposite end of this tubing to a manometer or arterial pressure monitor. Remove the 20 mL syringe from the system and aspirate 15 mL of air into the syringe. Reattach the syringe to the stopcock. Remove the extension tubing with the 18 gauge needle from the saline container.

Insert the 18 gauge needle into the affected muscle compartment (**Figure 93-4*B***). Turn the stopcock valve so that all three ports are open. Position the IV extension tubing with the normal saline so that the meniscus of the saline-air interface is exactly level with the tip of the needle inserted into the patient's tissue. **The position of the saline-air interface in relation to the tip of the needle is important for an accurate intracompartmental pressure measurement**.

The saline-air interface will form a meniscus when the needle is in the patient. The meniscus will be convex-shaped away from the patient when the tissue pressure is greater than the pressure within the system (**Figure 93-5*A***). **Depress the plunger of the syringe gradually and delicately to increase the pressure within the system.** The

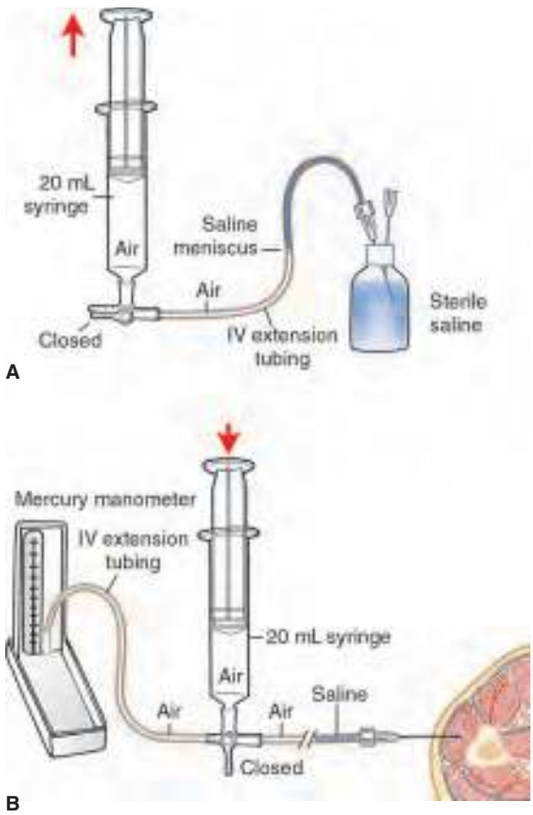

FIGURE 93-4. The needle manometer technique. ***A.*** The initial system setup. ***B.*** The final system should form a closed system from the manometer through the tissue.

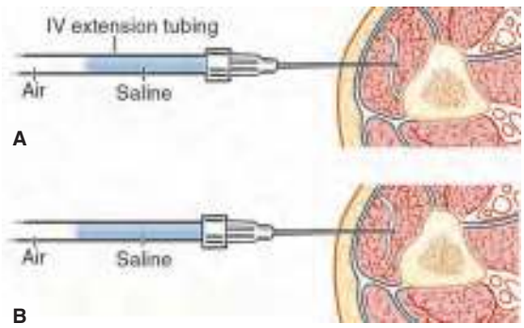

FIGURE 93-5. The air-saline interface. ***A.*** The air-saline meniscus will form a convex shape away from the patient when the tissue pressure is greater than the pressure of the system. ***B.*** The meniscus will flatten out when the pressure within the system equals that of the tissue.

shape of the meniscus begins to flatten as the plunger is depressed. **The point where the meniscus is flat and the saline column begins to move equals the pressure within the compartment (Figure 93-5*B*).** Note and document the pressure on the manometer.

It is important to equilibrate the system between measurements. Pull back on the syringe plunger until the manometer reads 0 mmHg with the needle still positioned in the tissue. Withdraw the needle from the tissue. This will prevent any saline from being deposited in the tissue. The same needle can then be reinserted in another location to obtain additional pressure measurements if sterile technique was used throughout the procedure.

The needle puncture sites will bleed. It is important to complete the procedure by properly dressing the puncture wounds. Apply gauze over the puncture sites and tape it in place. Mild hand-held pressure will tamponade any bleeding.

STRYKER METHOD

The Stryker intracompartmental pressure monitor system is a self-contained device that is convenient, accurate, and relatively easy to use (**Figure 93-6**). Keep the unit in a secure and easily accessible location. The pressure monitor is battery operated and reusable with a digital display (**Figure 93-6*A***). The quick pressure monitor pack is a disposable, single-patient-use kit that contains an 18 gauge side-ported needle, the diaphragm chamber, and a saline-filled syringe (**Figure 93-6*B***).

Turn the pressure monitor unit on. The digital display should read 0 to 9 mmHg. Use sterile technique when setting up the system. Remove the diaphragm chamber from the quick pressure monitor pack. This chamber ensures sterility between the system and the patient. Place the 18 gauge, 2.5 inch side-ported needle firmly on the smaller tapered stem of the diaphragm chamber (**Figure 93-7*A***). **This needle must remain sterile.** Uncap the 3 mL syringe filled with sterile normal saline and screw it onto the larger stem of the diaphragm chamber (**Figure 93-7*A***). Open the clear plastic lid of the monitor unit. Place the needle-diaphragm chamber-syringe unit on the monitor so that the diaphragm chamber sits in the well (**Figure 93-7*B***). Push down gently on the diaphragm chamber until it is firmly and evenly positioned on the monitor. Close the cover of the unit until a snap is heard at the latch site.

Hold the monitor unit with the needle at a 45° angle from the horizontal. Depress the plunger of the syringe slowly to pass saline through the diaphragm chamber and needle until saline drips from the tip of the needle. This removes any air within the system. Position the needle next to the skin at the angle needed for insertion. Press the "zero" button on the pressure monitor. The display should read "00" after a few seconds. **Insert the needle into the desired compartment at the same angle used during the zeroing process (Figure 93-8).** Slowly inject 0.3 mL of saline into the compartment. This volume of fluid is used to equilibrate with the interstitial fluids. Wait a few seconds as the system is measuring the compartmental pressure. The final compartmental pressure measurement will be displayed on the digital screen. Remove the needle from the patient.

Reset the system to zero before taking additional measurements. This is accomplished by positioning the needle at a new site and desired angle of insertion and pressing the zero button. **This must be repeated between each measurement.** Apply bandages over the skin puncture sites. Mild hand-held pressure will tamponade any bleeding.

An optional indwelling slit catheter set is available for use on the Stryker pressure monitor. This set substitutes a slit catheter, breakaway needle, and extension tubing for the side-ported needle. The slit catheter is intended to be left within the patient and allows multiple sequential intracompartmental pressure measurements. This is not intended for use in the ED.

A

B

FIGURE 93-6. The Stryker intracompartmental pressure monitor system. ***A.*** The Stryker pressure monitor unit. Note the power switch, zero button, and digital readout. Under the clear plastic cover, the round well will secure the disposable drum and the syringe will clip into the plastic bracket at right. ***B.*** The quick pressure monitor pack contains (*from left to right*): an 18 gauge 2.5 inch side-ported needle, the diaphragm chamber, and a 3 mL syringe filled with sterile saline.

ARTERIAL MANOMETER TECHNIQUE

The third method of assessing compartment pressures in the ED requires only an 18 gauge needle or angiocatheter, arterial line tubing, and an arterial manometer set up as if for an arterial line (Chapter 72).[52] Use a straight or side-port needle for a one-time reading. Use an 18 gauge angiocatheter if continuous compartment pressure monitoring is preferable. Attach the desired needle or angiocatheter to the end of the arterial line tubing that leads to the prepared arterial manometer. Flush the entire line with sterile saline. Place the tip of the needle adjacent to the skin insertion site

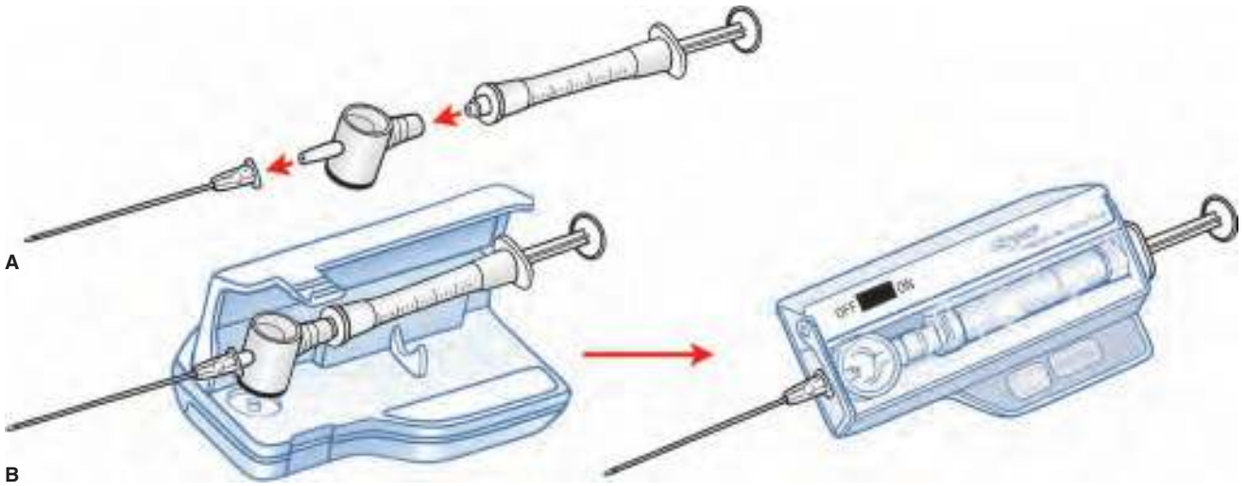

FIGURE 93-7. Assembly of the Stryker system. ***A.*** The contents of the quick pressure monitor pack are assembled. ***B.*** The assembled needle-diaphragm-syringe is placed onto the monitor.

and zero the system. Insert the needle into the compartment and slowly infuse 0.3 mL of sterile saline to equilibrate the system with the interstitial pressure. The manometer will provide continuous real-time measurement of the compartment pressure if the needle or angiocatheter remains in place.

COMPASS PRESSURE MONITOR

The Compass Pressure Monitor (Centurion Medical Products, Williamston, MI) can measure the compartment pressure (**Figure 93-9**). It is a disposable, single-use device that is easy to use. It provides a precise digital qualitative reading instantaneously. Attach a sterile syringe of saline to one end and a sterile needle on the other end. It is used like the Stryker. The device can be used to measure pressure with many procedures (e.g., compartments, central venous lines, intraabdominal, lumbar puncture, and intrapleural).

ASSESSMENT

Measure and document the compartment pressure (**Figure 93-10**). Pressures < 20 mmHg are normal. Pressures of 20 to 30 mmHg are elevated and concerning. They require frequent examination and rechecks of the pressure. Pressures over 30 mmHg or within 30 mmHg of the diastolic blood pressure warrant an emergent evaluation by a General Surgeon or Orthopedic Surgeon for a possible fasciotomy and limb salvage. Obtain an emergent General Surgeon or Orthopedic Surgeon consultation if there is any clinical concern for a compartment syndrome regardless of the pressure.

AFTERCARE

Control any bleeding from the skin puncture sites with direct pressure. Mild hand-held pressure will tamponade any bleeding. Apply gauze and tape dressings to all puncture sites. Perform and document a repeat neurovascular exam of the extremity distal to the procedure site.

COMPLICATIONS

There should be no complications for the patient if sterile technique is maintained throughout this procedure. Inserting a needle into a tissue compartment introduces the theoretical risk of infection or damage to the nerves or vessels. No study has shown this to be a significant complication.

A realistic complication of the procedure is obtaining erroneous values.[53-55] The greatest risk to the patient is if an artificially low pressure is obtained and the needed fasciotomy is not performed. False high-pressure readings may be obtained. The consequences of performing an unnecessary fasciotomy are less disastrous. Erroneous values are due to improper technique or device inaccuracy.[54] Approximately 69% of the time there are technical errors and only 60% with correct technique are within 5 mmHg of the correct pressure.[54]

It is important to understand how the mechanics of the needle manometer system can alter the pressure readings. Injection of

FIGURE 93-8. The needle is inserted into the desired compartment and the pressure measurement read on the digital display.

FIGURE 93-9. The Compass. (Photo courtesy of Centurion Medical Products, Williamston, MI.)

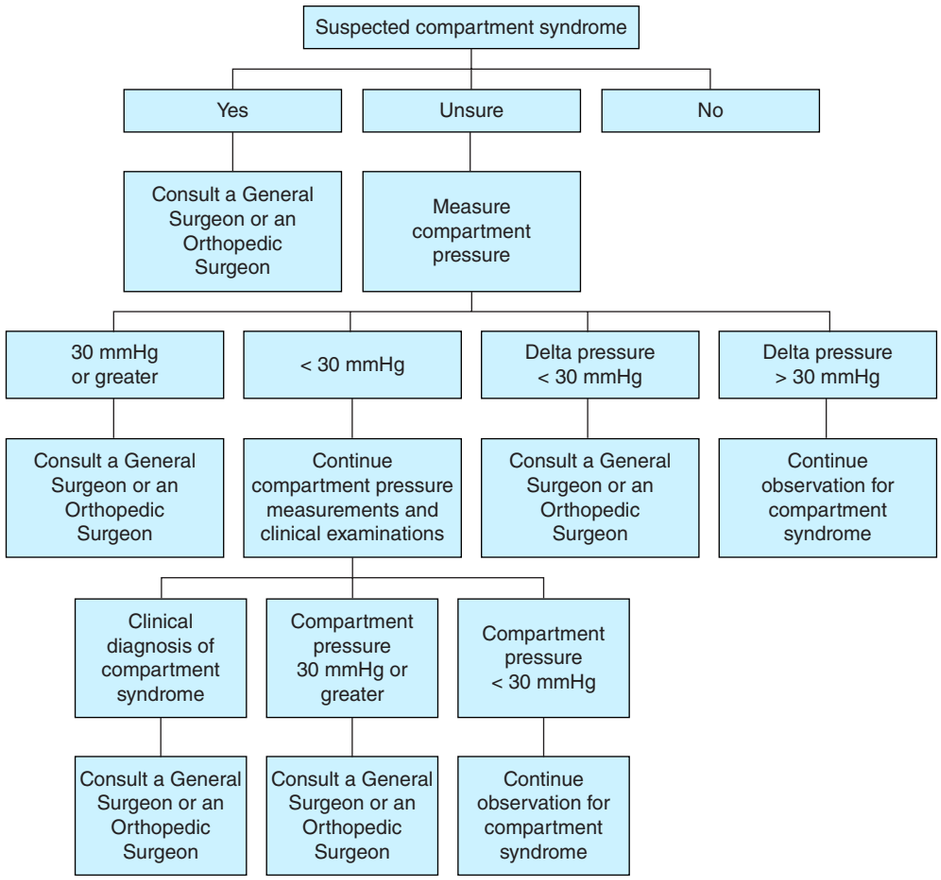

FIGURE 93-10. Algorithm for evaluation of a patient with a suspected compartment syndrome.

normal saline into the tissue will raise the pressure reading. The manometer reading will not accurately reflect the pressure of the compartment if the needle is inserted into a tendon rather than the muscle. A piece of tissue from the compartment can obstruct the needle and lead to erroneous pressure measurements. Failing to match up the saline-air meniscus with the level of the needle in the tissue can produce false pressure measurements.

One must take multiple pressure measurements around the injury site.[56] To perform a fasciotomy on an isolated pressure reading is to proceed without all the necessary data. It becomes difficult to simply observe the patient when they clinically present with increasing pain and features suggestive of a compartment syndrome when the compartmental pressure measurement is normal. Take multiple readings when clinical suspicion is high and the compartment pressures are normal.

SUMMARY

A compartment syndrome is a well-documented phenomenon. The clinical presentation is variable and changes over time. It is a difficult clinical diagnosis that is critical for the EP to make in a timely fashion. The most common sites are the lower leg and forearm. A compartment syndrome can occur in any muscle compartment of the body. Determining the pressure within a compartment is a fundamental and essential tool to aid in this diagnosis. Many methods exist for the measurement of compartment pressures. The traditional needle manometer system, the Stryker pressure monitor kit, and the arterial manometer are three established techniques that can easily be performed in the ED. These techniques represent the standard of practice in most EDs. Obtain an emergent Orthopedic Surgeon or General Surgeon consultation if there is any concern for a compartment syndrome (**Figure 93-10**). Continuous observation and repeated measurements are often indicated. Pressures over 30 mmHg or within 30 mmHg of the diastolic blood pressure warrant an emergent evaluation for a possible fasciotomy and limb salvage.

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94 Fasciotomy

Andrew Rotando and Justin Mazzillo

INTRODUCTION

An acute extremity compartment syndrome is a serious and sometimes catastrophic entity that can lead to irreversible local and systemic damage.[1] It is initiated by a variety of injuries and characterized by a cycle of edema and ischemia. **A compartment syndrome is a condition involving increased interstitial pressure in a closed and confined space that results in inadequate perfusion and impaired tissue function.**[2] The muscles, nerves, and vasculature within the affected muscle group can all be compromised by the edema and prolonged ischemic state with the potential for significant morbidity.

A compartment syndrome may lead to loss of muscle or nerve function, ischemic contractures, rhabdomyolysis, infection, and amputation. This can be followed by systemic complications (e.g., renal failure, sepsis, and possibly death).[3] Ischemic contractures of the extremities were first described over 100 years ago by Richard Von Volkmann and are now referred to as "Volkmann's ischemic contractures." Common causes of a compartment syndrome include

anticoagulation, circumferential burns, constrictive dressings (e.g., tight-fitting bandages, casts, or splints), crush injuries, electrical injuries, exercise, external trauma to the extremity, extravasation (e.g., contrast or misplaced injections), joint dislocations, open or closed fractures, reperfusion after a vascular insult, and snakebites (**Table 93-1**).[1-27] Fractures are the most common etiology of a compartment syndrome.[4,23] Rare causes include unusual circumstances or spontaneous compartment syndrome.[28,29]

The difficulty in diagnosing a compartment syndrome is that the physical examination is a poor indicator of the degree of microcirculatory compromise.[5,29,30,31] Common symptoms historically associated with a compartment syndrome have been shown to be unreliable to diagnose and identify a compartment syndrome, and the diagnosis requires a high level of clinical suspicion on the part of the Emergency Physician.[2,6-8] Refer to Chapter 93 for the complete details of a compartment syndrome.

Treatment of a compartment syndrome with a fasciotomy was first suggested in 1906 by Bardenheuer.[9] **Maintaining a low threshold for performing a fasciotomy can be the safest course for the patient. The prognosis is more favorable if a fasciotomy is performed soon after the onset of symptoms.**[10] There may be little or no benefit to performing a fasciotomy if it is delayed. A basic knowledge of the anatomy of commonly affected compartments is necessary to safely and successfully perform an extremity fasciotomy.

ANATOMY AND PATHOPHYSIOLOGY

The specific anatomic site of an extremity compartment syndrome is variable, as it can occur in any muscle tissue that is confined in a space by fascia, skin, or any external forces (e.g., casting material). Compartmental perfusion is a dynamic process maintained by arterial blood pressure and limited by the absolute compartment pressure. Increased intracompartmental pressure from edema or hemorrhage within or from external compression of the compartment compromises arterial perfusion, causes venous outflow obstruction, and eventually leads to tissue ischemia.

The muscles, nerves, and vasculature within the affected muscle group are all potentially compromised by a prolonged ischemic state followed by swelling. The initial imbalance of a compartment syndrome occurs between the volume and pressure within the myofascial compartment. The arterial inflow and venous outflow diminish as either intracompartmental volume or pressure increases. The blood begins to be shunted via capillaries into the muscle tissue. This compensatory shunting of blood further disturbs the volume-pressure balance and results in impaired tissue oxygenation.

Skeletal muscles, major nerves, and major blood vessels of the extremities are contained within a noncompliant connective tissue membrane known as the investing or deep fascia. Connective tissue septa extend from the investing fascia to the bones of the extremities. The septa separate major muscle groups and form discrete compartments within each extremity.

The individual fascial compartments have a relatively constant range of pressure within which perfusion is maintained.[5] The normal compartment pressure is ≤ 12 mmHg. Any of the previously mentioned intrinsic or extrinsic insults may set off a cascade of events leading to edema and/or hemorrhage, causing the pressure within the compartment to rise. A critical pressure is reached within the compartment and perfusion is impaired. This results in altered metabolic processes, cell wall dysfunction, and muscle cell death begins to occur. This leads to extravasation of intracellular contents and edema within the enclosed space, further raising compartment pressures and causing additional cellular injury, and ultimately forming a positive feedback loop.[2,6]

Skeletal muscle and peripheral nerves can survive ischemic conditions for up to 4 hours before irreversible damage begins to occur. Muscle and nerve injury may still be reversible at 6 hours of ischemia. Ischemia occurring for greater than 8 hours will lead to irreversible damage of muscles and nerves.[11,32]

DIAGNOSIS

Clinical signs of a compartment syndrome consist of the 6 "P's" for any region at risk. These include pain on passive stretch of the muscles that run through the compartment, pain out of proportion to exam, paresthesias, pallor, pulselessness, and paralysis. Some may choose to include poikilothermia as the seventh "P."[6] **These are generally late clinical findings and may be completely absent early in the disease process except for pain. The most important symptom for diagnosing a compartment syndrome is pain**. Compartment syndrome pain is described as progressive, out of proportion to the examination, occurring with passive movement, and persisting despite immobilization. These symptoms are specific but not sensitive. The absence of pain does not rule out a compartment syndrome.[2] Palpation to detect a compartment syndrome is highly unreliable. **It is reasonable to measure the compartment pressures when any signs or a clinical suspicion exists for a compartment syndrome.**[7,8] **The diagnosis of a compartment syndrome relies on the clinical assessment of a combination of factors including mechanism of injury, pain pattern, associated injuries, clinical signs, and compartment pressure measurements.**

The focus of this chapter is to describe the approach to performing fasciotomies of the individual extremity compartments. The pertinent anatomy for each fasciotomy is included. Refer to Chapter 93 for a more detailed discussion of the anatomy, pathophysiology, evaluation, and diagnosis of compartment syndrome.

INDICATIONS

It has been classically taught that a fascial compartment with an absolute pressure of ≥ 30 mmHg will require a fasciotomy. Other factors besides the absolute compartmental pressure contribute to tissue perfusion and should be accounted when deciding to perform a fasciotomy. Hypertension may be protective of perfusion, whereas hypotension may further compromise perfusion.[1] A differential pressure, or "delta P" (ΔP), of ≤ 30 mmHg has been shown to be a better indication for performing a fasciotomy as it accounts for systemic blood pressure.[5,12-14] The ΔP is calculated as the difference between the diastolic blood pressure and the absolute or measured compartment pressure.

Maintaining a low threshold for performing a fasciotomy can be the safest course in patients who are at high risk of extremity ischemia.[10,30,31] Any patient with the appropriate history and any of the clinical signs of a compartment syndrome should be considered a candidate for a fasciotomy. Strongly consider performing a fasciotomy if the patient has a fascial compartment with a ΔP < 30 mmHg. An absolute compartment pressure ≥ 30 mmHg, or ≥ 20 mmHg if the patient is hypotensive, combined with any clinical signs of a compartment syndrome is an indication for a fasciotomy. The prognosis is favorable if the fasciotomy is performed within 30 hours of symptom onset.[15] There is usually little or no benefit if the fasciotomy is delayed longer.[32] This is particularly true if the fasciotomy is performed greater than 2 days after the onset of symptoms.

CONTRAINDICATIONS

A fasciotomy in the setting of a true compartment syndrome has few absolute contraindications. Any life-threatening conditions must first be addressed. There is a high incidence of severe

infections if a fasciotomy is performed beyond the third or fourth day after symptom onset.[15] A fasciotomy should not be performed if the Emergency Physician is not familiar with the local anatomy or the technique.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Face mask and cap
- Sterile gloves and gown
- Sterile drapes or towels
- Equipment for compartment pressure measurement (Chapter 93)
- 4 × 4 gauze squares
- #10 scalpel blade on a handle
- Tissue forceps
- Metzenbaum scissors
- Curved forceps
- Surgical retractors
- Kerlix dressings
- Splint material

PATIENT PREPARATION

Perform and document a thorough neurological and vascular examination of the affected extremity.[1] Remove any constrictive dressings, casts, and/or splints that may be contributing to the increased compartment pressures. Place the patient supine on the gurney so that the limb at risk is at heart level. It may be more appropriate for the fasciotomy to be performed in the Operating Room by an experienced Surgeon if time is permissible and a Surgeon is available. This is often not possible and the fasciotomy must be performed in the Emergency Department. Consider consulting a Surgeon to assist in the decision to perform a fasciotomy.

Inform the patient and/or their representative of the risks, benefits, complications, and aftercare associated with a fasciotomy. **The procedure must be performed using strict aseptic technique.** The Emergency Physician and any assistants must be wearing sterile gloves, a sterile gown, a face mask, and a cap. Clean the extremity of any blood, dirt, and debris. Apply povidone iodine or chlorhexidine solution to the extremity and allow it to dry. Apply sterile drapes or towels to isolate the extremity and form a sterile field. Collect and place all required supplies on a bedside table covered with a sterile drape. **Use a sterile marking pen to draw the incision lines onto the patient's skin prior to making any incisions.**

A fasciotomy is a painful procedure. Some form of analgesia is required if the patient is conscious. The infiltration of local anesthetic solution provides appropriate analgesia. Ensure that the toxic dose of the selected local anesthetic solution is not exceeded (Chapter 153). Alternatives include a regional nerve block (Chapter 156) or procedural sedation (Chapter 159).

TECHNIQUES

THE ARM

The arm consists of three compartments: anterior, posterior, and deltoid (**Table 94-1 and Figure 94-1**).[16] The anterior compartment contains the biceps, brachialis, and coracobrachialis muscles. The posterior compartment contains the triceps muscle. The proximal anterolateral arm contains the deltoid compartment.

TABLE 94-1 The Compartments of the Arm and Their Contents

Compartment	Contents
Anterior	Biceps muscle
	Brachialis muscle
	Coracobrachialis muscle
	Brachial artery
	Median nerve
	Ulnar nerve
	Musculocutaneous nerve
	Lateral cutaneous nerve
	Antebrachial nerve
	Radial nerve
Posterior	Triceps muscle
	Radial nerve
	Ulnar nerve
Deltoid	Anterior deltoid muscle
	Lateral deltoid muscle
	Posterior deltoid muscle
	Axillary nerve

A fasciotomy of the arm requires two longitudinal incisions to decompress the anterior and posterior compartments (**Figure 94-2**). Decompress the anterior compartment with a longitudinal incision over the anterior surface of the arm. Make the incision along the midline overlying the biceps muscle extending from the inferior border of the deltoid to the antecubital fossa (**Figures 94-1 and 94-2*A***). Decompress the posterior compartment with a longitudinal incision. Make the incision along the midline of the posterior surface of the arm overlying the triceps muscle from the inferior border of the deltoid to the olecranon (**Figures 94-1 and 94-2*B***). Use a #10 scalpel blade to cut through the skin and subcutaneous tissues down to the level of the investing fascia. Carefully cut the fascia parallel to the skin incision with scissors. **The fascial incision should be the same length as the skin incision. Use caution so as not to cut the muscle itself.**

A fasciotomy of the deltoid compartment muscle may occasionally be indicated. The deltoid muscle has three heads that are separated by septa. It is often necessary to decompress each muscle belly separately.[17] Further complicating the deltoid muscle decompression

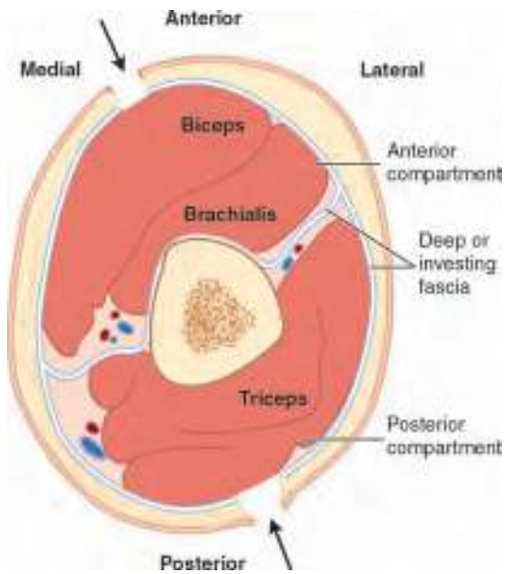


FIGURE 94-1. The compartments of the arm. Fasciotomy incisions have been made (*arrows*) by cutting through the skin, subcutaneous tissues, and deep or investing fascia.

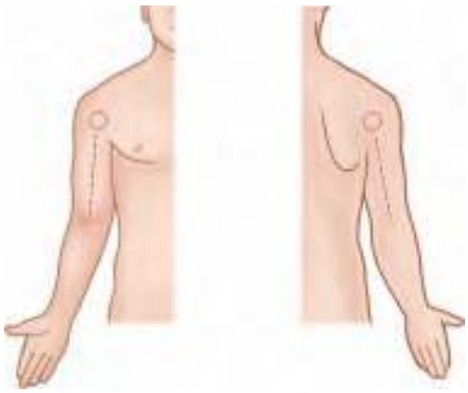


FIGURE 94-2. Fasciotomy incisions of the arm. ***A.*** Anterior compartment. ***B.*** Posterior compartment.

is the presence of multiple septa within the respective heads necessitating individual epimysiotomies.[17]

Begin the skin incision over the anterior origin of the deltoid muscle (**Figure 94-3**). Extend the incision superiorly and laterally, just lateral to the acromion process. Continue the incision posteriorly along the origins of the posterior deltoid muscle. Use a #10 scalpel blade to cut through the skin and subcutaneous tissues down to the level of the investing fascia. **The cutaneous branch of the axillary nerve becomes superficial inferior and posterior to the acromion and continues anteriorly over the lateral deltoid muscle. It should not be cut when performing a fasciotomy.** Carefully cut open the fascia of the anterior deltoid muscle with scissors. **Use caution so as not to cut the muscle itself.** Measure the compartment pressure in the remaining two muscle heads. Decompressing one head allows the remaining head(s) to sometimes spontaneously decompress. Incise the fascia of the middle head longitudinally. Measure the pressure of the posterior head. Incise the investing fascia of the posterior head if the compartment pressure is still elevated. Explore the full extent of each muscle belly and perform any necessary epimysiotomies to achieve full decompression. To fully decompress the anterior deltoid, it may be necessary to extend the incision distally along the deltopectoral groove. To adequately decompress the posterior head, the incision may need to extend down along the midline posterior arm beginning from the posterior aspect of the acromion.

Alternative techniques have been described in several case reports involving anterior, mid-lateral, or posterior single incisions for isolated deltoid head involvement or two-incision approaches for multiple deltoid head decompression.[18-20] The described two-incision approaches involve a combination of the anterior longitudinal incision for anterior head decompression and either a mid-lateral or posterior longitudinal incision to affect decompression of either, or both, of the lateral and posterior heads. These techniques may be warranted based on patient presentation and physician comfort that full decompression of the affected heads will be achieved.

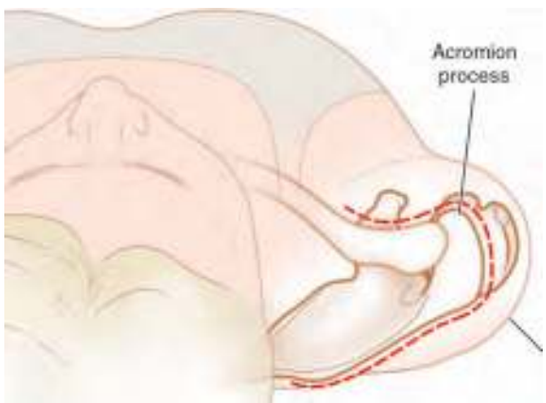


FIGURE 94-3. Fasciotomy incision for the deltoid compartment.

THE FOREARM

The forearm is one of the most common regions afflicted by a compartment syndrome.[1,33-35] The forearm consists of three compartments: volar, dorsal, and lateral (i.e., or mobile wad). They are interconnected at various levels (**Figure 94-4**). This interconnection is significant because release of the pressure in one compartment will reduce some of the pressure in the adjacent compartments. **The volar compartment is most at risk for development of a compartment syndrome in traumatic injuries of the forearm.**[34] This is followed by the dorsal compartment and the lateral or mobile wad compartment. The lateral or mobile wad compartment is the easiest compartment to decompress because it is located superficially.[11]

The contents of each forearm compartment are listed in **Table 94-2**. The volar compartment contains all the hand and forearm flexor muscles, the median nerve, the ulnar nerve, the radial artery, the ulnar artery, and common interosseous artery. The dorsal compartment contains the digital and wrist extensor muscles, the posterior interosseous artery, and the posterior interosseous nerve. The mobile wad contains the brachioradialis, the extensor carpi radialis brevis, and the extensor carpi radialis longus muscles. No major arteries or nerves are contained within this compartment. The radial artery and a branch of the radial nerve may sometimes lie between the mobile wad and the volar compartment.

The volar and dorsal compartments can be further subdivided into superficial and deep muscles. Evaluation of the deep muscles becomes important when these muscles are preferentially injured. This includes electrical injury as the bone transmits thermal injury to adjacent muscles, crush injuries, sepsis, and prolonged external pressure on the forearm.

Decompression of the volar compartment can lead to the simultaneous decompression of the other compartments. Thus, a volar fasciotomy is generally performed first.[34] There are two approaches employed to perform the volar fasciotomy and one approach for the dorsal fasciotomy (**Figure 94-5**). This text describes the volar-ulnar incision for the anterior fasciotomy (**Figure 94-5*B***) and the dorsal approach (**Figure 94-5*A***). Some authors feel that the volar-ulnar incision is associated with the least amount of iatrogenic injury.[1,35,36]

Place the patient supine with their arm supinated on a bedside table to decompress the volar compartment. Begin the incision 1 to 2 cm proximally and 2 to 3 cm laterally to the antecubital fossa. Extend the incision obliquely across the antecubital fossa until it reaches the anterolateral aspect of the distal antecubital fossa. Continue the incision distally on the anterolateral surface of the forearm toward the wrist. Transecting the wrist and extending the incision into the palmar aspect of the hand involves an S-shaped incision. Just proximal to the wrist, extend the incision toward the radial aspect of the forearm being careful to not yet traverse the wrist. **Do not extend the incision as lateral as the radial artery pulse.** Continue the incision back medially and return to the midline of the wrist. Proceed with slight ulnar deviation distally along the midline into the proximal mid-palm. Make a lateral turn along the thenar crease and terminate the incision at the level of the mid-thenar eminence. **Be sure to incise deep enough to include the superficial fascia along the length of the entire forearm without cutting into the muscles or tendons.**

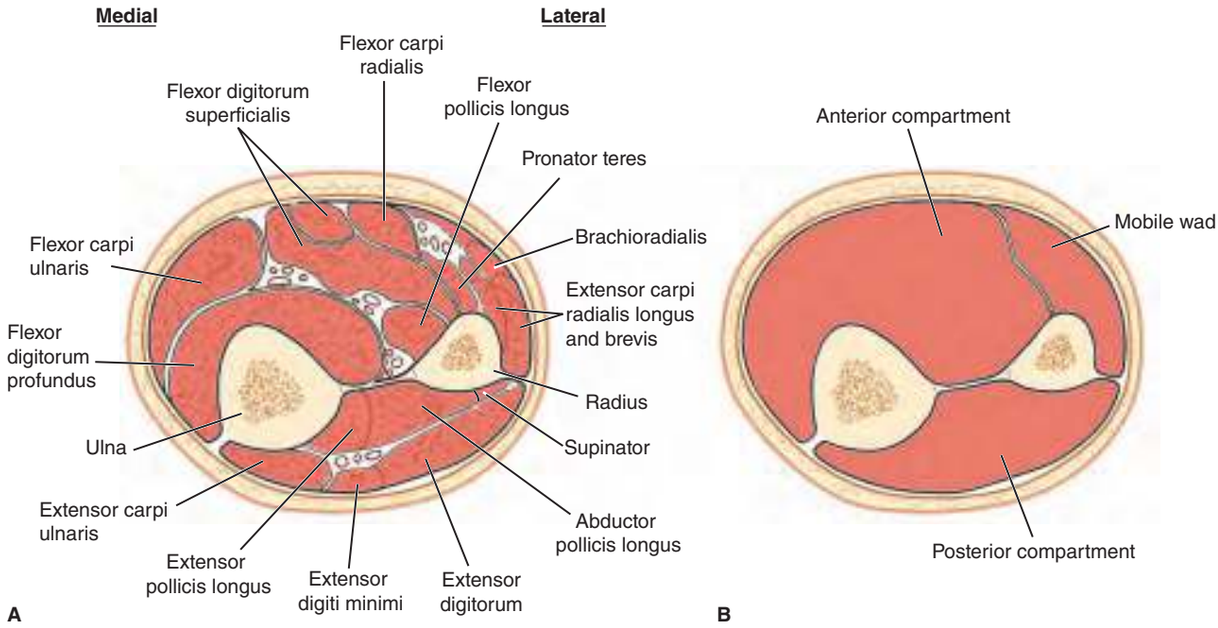


FIGURE 94-4. The compartments of the forearm.

Undertake blunt exploration of the exposed superficial compartment. Identify the median nerve proximally near the antecubital fossa. Release the bicipital aponeurosis at the level of the antecubital fossa where it overlies the median nerve. Retract the flexor carpi ulnaris muscle medially and the flexor digitorum superficialis muscle laterally. Follow the median nerve all the way to the carpal tunnel. Decompress the median nerve at the carpal tunnel. **Be careful not to injure the ulnar artery, ulnar nerve, and the palmar cutaneous branch of the median nerve.** Incise the superficial palmar fascia in the midline followed by incising the transverse carpal ligament in the midline.

The superficial forearm contents are completely open at this point, and the deep contents of the volar compartment are exposed and should be explored. Inspect the flexor digitorum profundus, flexor pollicis longus, and pronator quadratus muscles. Use aseptic technique and measure the pressures within the deep muscles to determine whether the fascia covering these muscles needs to be incised. Incise the deep fascial coverings if the muscle pressures are > 20 mmHg.

The major disadvantage to the volar-ulnar incision is that the mobile wad is not easily accessible via this incision. If the mobile wad is at risk, it may be reached through the dorsal incision (**Figure 94-5*A***) as described below. It may also be reached through a different volar approach such as the volar curvilinear incision (**Figure 94-5*C***). Some authors contend they can successfully decompress all three compartments via this single incision (**Figure 94-5*C***).[37]

Perform a fasciotomy if needed for the dorsal compartment. Place the patient prone with their palm resting on a bedside table. If the patient is supine, completely flex the supinated arm so that the dorsal surface is exposed. Make the longitudinal incision beginning 3 to 4 cm distal to the lateral epicondyle of the humerus and incise to

TABLE 94-2 The Compartments of the Forearm and Their Contents

Compartment	Contents
Lateral (mobile wad)	Brachioradialis muscle
	Extensor carpi radialis longus muscle
	Extensor carpi radialis brevis muscle
Dorsal	Extensor carpi ulnaris muscle
	Extensor pollicis longus muscle
	Abductor pollicis longus muscle
	Extensor pollicis brevis muscle
	Extensor digitorum communis muscle
	Posterior interosseous artery
	Interosseous perforators from the anterior interosseous artery
	Posterior interosseous nerve
Volar	Flexor pollicis longus
	Flexor carpi ulnaris
	Flexor carpi radialis longus
	Flexor carpi radialis brevis
	Palmaris longus
	Flexor digitorum superficialis
	Flexor digitorum profundus
	Pronator teres
	Pronator quadratus
	Radial artery
	Ulnar artery
	Anterior interosseous arteries
	Median nerve
	Ulnar nerve
Carpal tunnel	Extrinsic flexor tendons
	Median nerve

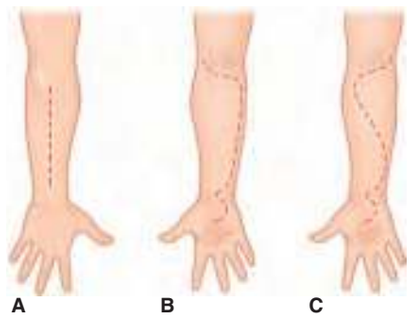


FIGURE 94-5. Fasciotomy incisions for the forearm. ***A.*** The dorsal incision. ***B.*** The anterior volar-ulnar incision. ***C.*** The volar curvilinear incision.

the distal radius (**Figure 94-5*A***).[38] Be sure to incise the fascia along the entire length of the incision. **It is important to avoid incising too deep at the wrist and cutting the extensor retinaculum.** If the skin is adequately undermined, the mobile wad will be accessible via this approach.

The carpal tunnel does not contain muscle bellies and is thus not a true compartment. It can achieve acute compartment syndrome physiology.[17,39] Some authors recommend routinely decompressing the carpal tunnel during a forearm fasciotomy while others maintain that doing so is of limited benefit.[10,40] This decision is left to the Emergency Physician performing the fasciotomy. **Many important structures run through the carpal tunnel including the median nerve.** Structures within the carpal tunnel become compromised at pressures greater than 40 mmHg. The signs and symptoms will be similar but more severe than a chronic carpal tunnel syndrome.

A carpal tunnel release can be performed as part of the forearm fasciotomy as described above or as an isolated procedure. To perform an isolated carpal tunnel release, begin the skin incision on the proximal portion of the hand between the thenar and hypothenar creases.[17,40] Extend the incision proximally toward the flexor crease of the wrist. Make a slight ulnar deviation of the incision so that the flexor crease is not crossed at a 90° angle. This will prevent injury to the palmar branch of the medial nerve as it courses between the palmaris longus and the flexor carpi radialis tendons. Reflect the skin edges to identify the superficial palmar fascia and the transverse carpal ligament. Make a longitudinal midline incision though the superficial palmar fascia and the transverse carpal ligament.

THE HAND

There are of 11 compartments in the hand. This includes the mid-palm, hypothenar, thenar, adductor pollicis, four dorsal interosseous, and three palmar or volar interosseous compartments (**Figure 94-6 and Table 94-3**).[17] **Each hand compartment is completely isolated. Decompression or release of one compartment will not help to decompress any of the others.** The pressures required to cause an acute compartment syndrome in a hand compartment may be as low as 15 to 20 mmHg.

Decompress the dorsal interosseous, palmar interosseous, and adductor compartments through dorsal incisions (**Figure 94-7**). Decompress the first and second dorsal interosseous compartments with a 3 to 4 cm long incision over the second metacarpal (**Figure 94-7*A1***). Extend the incision into the fascia on both sides of the metacarpal to access the compartments. Using gentle blunt dissection and continue deeper on the radial aspect to access the adductor compartment and on the ulnar aspect to access the first palmar interosseous compartment (**Figure 94-7*B***). Decompress the third and fourth dorsal interosseous compartments with a 3 to 4 cm long incision over the fourth metacarpal (**Figure 94-7*A2***). Incise the fascia on both sides of the metacarpal to access the compartments. Use gentle blunt dissection on the radial aspect to access the second palmar interosseous compartment and on the ulnar aspect to access the third palmar interosseous compartment.

Access the thenar compartment. Make a 3 to 4 cm long longitudinal skin incision over the thenar muscles on the mediolateral aspect of the hand and just medial to the first metacarpal (**Figures 94-7*D* and 94-8**). Continue the incision through the subcutaneous tissues until the thenar muscle fascia is visible. Carefully incise the fascia with scissors. **Be careful to not cut the underlying muscle.**

Access the hypothenar compartment. Make a 3 to 4 cm long longitudinal incision along the ulnar aspect of the fifth metacarpal over the hypothenar muscles (**Figures 94-7*E* and 94-8**). Continue the incision through the subcutaneous tissues until the hypothenar muscle fascia is visible. Carefully incise the fascia with scissors. **Be careful to not cut the underlying muscle.**

The mid-palm compartment of the hand is located between the thenar and hypothenar compartments (**Figure 94-6**). Make a 3 to 4 cm long longitudinal incision in the middle of the hand, between the thenar and hypothenar muscles (**Figure 94-7*C***). The incision of a fasciotomy of the forearm or carpal tunnel can be extended distally

TABLE 94-3 The Compartments of the Hand and Their Contents

Compartment	Contents
Mid-palm	Extrinsic flexor tendons
	Lumbrical muscles
	Superficial palmar vascular arches
	Deep palmar arches
	Common digital arteries
	Common digital nerves
	Proper digital nerves
Thenar	Abductor pollicis brevis muscle
	Flexor pollicis brevis muscle
	Opponens pollicis muscle
Hypothenar	Abductor digiti minimi muscle
	Flexor digiti minimi muscle
	Opponens digiti minimi muscle
Dorsal interossei	Dorsal interossei muscles
Volar interossei	Volar interossei muscles
Adductor pollicis	Adductor pollicis muscle

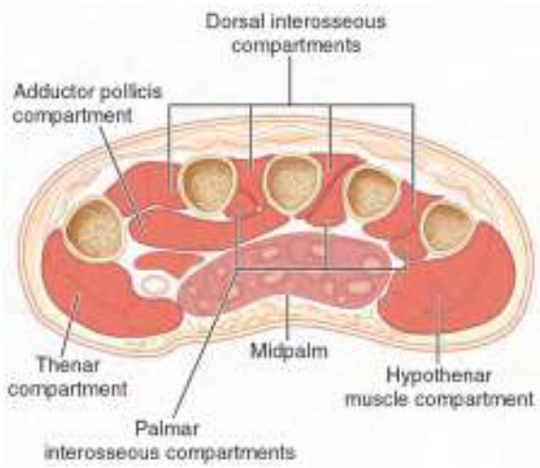


FIGURE 94-6. The 11 compartments of the hand.

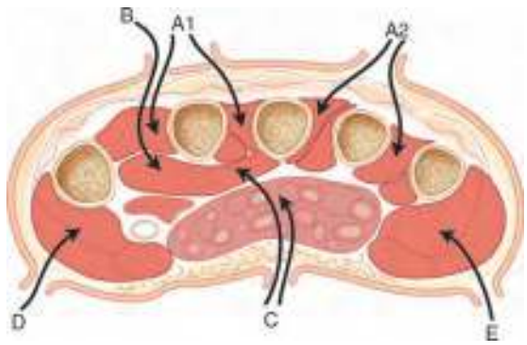


FIGURE 94-7. Fasciotomy incisions for the hand. ***A.*** Dorsal and palmar interosseous compartments. ***B.*** The adductor pollicis compartment. ***C.*** The mid-palm compartment. ***D.*** The thenar compartment. ***E.*** The hypothenar compartment.

FIGURE 94-8. Fasciotomy incisions for the hand and fingers.

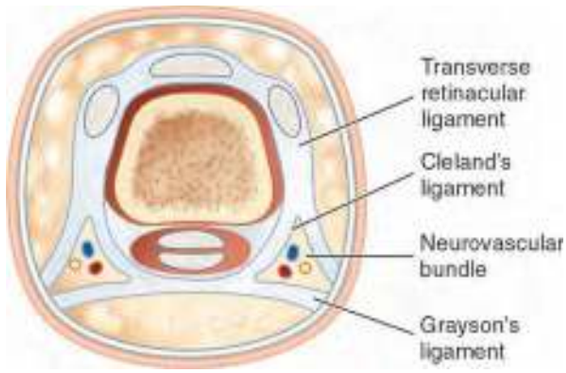


FIGURE 94-9. Cross-section through a digit.

to gain access to the fascia of the mid-palm compartment. Continue the incision through the subcutaneous tissues until the fascia is visible. Carefully incise the fascia with scissors. **Be careful to not cut the underlying muscle.**

THE DIGITS

The digits do not contain muscle bellies or true fascial compartments (**Table 94-4 and Figure 94-8**). The ligaments and restrictive skin create a contained compartment that can exhibit large pressure increases.[17] Cleland's and Grayson's ligaments are constrictive ligaments that encircle the finger and overlie the neurovascular bundles (**Figure 94-9**). **Measurement of pressures in these "pseudo-compartments" is very difficult. The indications for decompression are based more on clinical findings and suspicion. The fingers and toes have very similar anatomy and are managed in the same manner.**

A fasciotomy of the digit is performed using a single midaxial longitudinal incision along the length of the digit. Make the incision along the ulnar aspect of the index, long, and ring fingers and along the radial aspect of the thumb and small fingers (**Figure 94-8**). **Be careful to avoid injuring the underlying neurovascular bundle.** Identify the neurovascular bundle and release any constrictive fascial bands from Cleland's or Grayson's ligaments (**Figure 94-9**). Continue dissecting along the palmar aspect of the flexor tendon sheath and release any vertical bands of connective tissue. A second longitudinal incision can be placed on the opposite side of the digit to decompress the underlying neurovascular bundle if severe swelling is present with dysfunction of both the ulnar and radial neurovascular bundles.

THE THIGH

A compartment syndrome of the thigh is most frequently due to blunt trauma with associated femur fracture, but it is uncommon relative to the overall number of femur fractures.[3,41] The pressure at which a compartment syndrome of the thigh develops is not known. A case report of three patients who developed a thigh compartment syndrome in association with a femur fracture noted two of the patients had significant arterial bleeding into the thigh and the third had prolonged extrinsic compression of the leg. The Emergency Physician will need to rely on clinical signs and symptoms, measured pressures, and the mechanism of injury to make the diagnosis of a thigh compartment syndrome.

The thigh contains three compartments. These are the anterior, medial, and posterior compartments (**Figure 94-10**). They are separated from each other by intermuscular septa. The lateral intermuscular septum is the thickest of the three and separates the anterior and posterior compartments. The contents of the compartments are listed in **Table 94-5**.

Place the patient supine with their foot pointing upward. Expose the thigh from the iliac crest to the lateral epicondyle.[15] Begin the incision just distal to the intertrochanteric line and extend it to the lateral epicondyle (**Figure 94-11*A***). Expose the iliotibial band, a thickening of the investing fascia, by subcutaneous dissection. Release the anterior compartment of the thigh by making an incision in the iliotibial band that parallels the skin incision. Retract the vastus lateralis muscle medially, carefully separating it from the lateral intermuscular septum, and cauterizing or suturing any bleeding perforating vessels (**Figure 94-11*B***).[42] Make a 1.5 cm incision in the lateral intermuscular septum using Metzenbaum scissors. Extend the incision proximally and distally to decompress the posterior compartment. Determine the pressure in the medial or adductor

TABLE 94-4 The Compartments of the Digits and Their Contents

Compartment	Contents
Digits	Common palmar digital arteries Proper palmar digital arteries Proper palmar digital nerves Tendons Ligaments

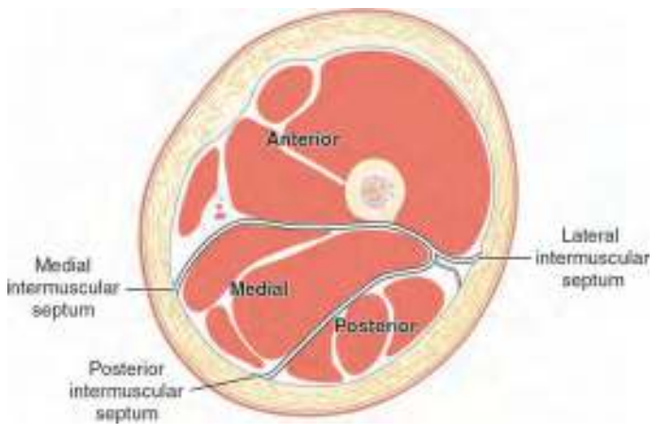
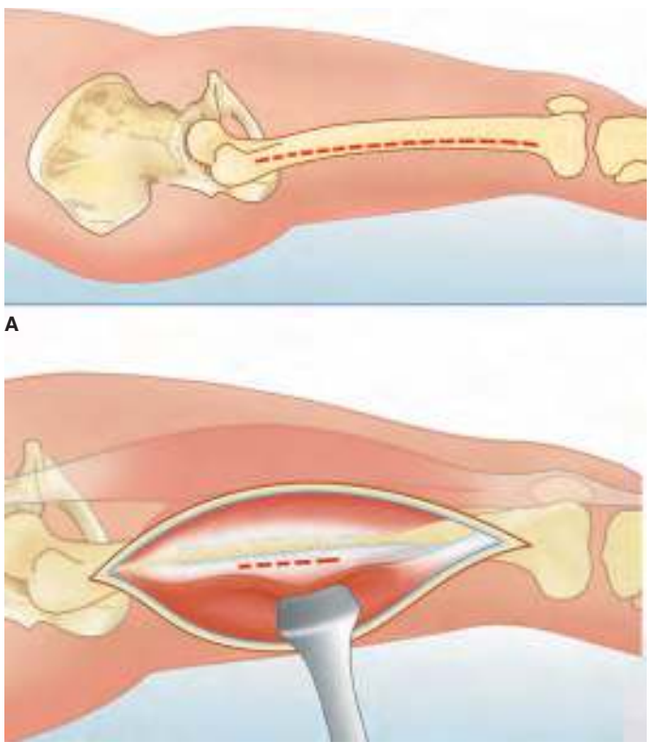


FIGURE 94-10. The compartments of the thigh.

TABLE 94-5 The Compartments of the Thigh and Their Contents

Compartment	Contents
Anterior	Sartorius muscle
	Iliacus muscle
	Pectineus muscle
	Psoas muscle
	Quadriceps femoris muscle
	Femoral artery and vein
	Femoral nerve
	Lateral femoral cutaneous nerves
Medial	Adductor longus muscle
	Adductor magnus muscle
	Adductor brevis muscle
	Obturator externus muscle
	Gracilis muscle
	Obturator artery and veins
	Profundus femoris artery and veins
	Obturator nerve
Posterior	Semitendinosus muscle
	Semimembranosus muscle
	Biceps femoris muscle
	Adductor magnus muscle
	Profundus femoris artery
	Sciatic nerve
	Posterior femoral cutaneous nerves

compartment once the anterior and posterior compartments have been decompressed. Make a longitudinal incision several centimeters medial to the femoral pulse to release the medial compartment if the pressure is elevated.



A

B

FIGURE 94-11. Fasciotomy of the thigh. ***A.*** The fasciotomy incision. ***B.*** The vastus lateralis muscle is retracted to expose the lateral intermuscular septum.

THE LEG

The leg is the most common location for a compartment syndrome, and a tibial fracture is the most common injury leading to a leg compartment syndrome.[1,9,43] The lower leg consists of four distinct muscle compartments: anterior, lateral, deep posterior, and superficial posterior (**Figure 94-12**).[15,44] A detailed list of the contents of each compartment is noted in **Table 94-6**.

The anterior compartment contains the four extensor muscles of the leg. These muscles function together to dorsiflex the foot. The deep peroneal nerve travels through this compartment to innervate the extensors and provide sensory innervation to the web space between the first and second toes. The anterior tibial artery travels through this compartment and provides blood flow to its contents.

The lateral compartment contains the peroneus longus and brevis muscles. Their chief function is to evert the foot, with some consequent abduction and plantarflexion of the foot. The major nerve in the lateral compartment is the superficial peroneal nerve to supply motor innervation to the compartment muscles and sensory innervation to the lower leg and dorsum of the foot.

The transverse intermuscular septum divides the posterior muscle group into superficial and deep compartments. The superficial posterior compartment contains the muscles of plantarflexion (i.e., gastrocnemius, soleus, and plantaris muscles). No major nerves or blood vessels travel in the superficial compartment. The deep posterior compartment contains the four deep flexor muscles (i.e., flexor digitorum longus, flexor hallucis longus, tibialis posterior, and popliteus muscles). This group of muscles contributes to inverting and adducting the foot in addition to flexing the toes and foot. The primary sensory innervation is from the tibial nerve which courses through the deep posterior compartment. The tibial nerve supplies most of the muscles of the posterior compartment before dividing into several branches that provide sensory innervation to the sole of the foot. The posterior tibial artery and the peroneal artery are contained within this compartment.

The deep posterior compartment and the anterior compartment have the highest incidence of developing a compartment syndrome.[45] Recall that a compartment syndrome is progressive, developing over time, and patients may initially present with normal compartment pressures. **Multiple compartmental readings must be taken when the suspicion is high and the compartmental pressures are initially normal**.

A single-incision fasciotomy can be performed if the physical examination of the leg reveals a clinical concern for a compartment syndrome of only one compartment. Decompression of all compartments will be required in cases of prolonged limb compression or severe trauma.[44,46] Several different techniques are used to decompress all four compartments of the lower leg and involve either a single incision or a double incision. This text describes the double-incision approach. It remains the most frequently employed technique, although there may be advantages to the single-incision approach that could ultimately lead to its widespread use.[47,48] Researchers have developed an algorithmic approach to acute compartment syndrome of the leg involving the initial decompression of the anterior and lateral compartments with subsequent repeated pressure measurements of the posterior compartments to determine if further decompression remains warranted.[47] This approach could potentially avoid unnecessary decompression of the posterior compartment if indirect pressure from the anterior and lateral compartments is relieved by the lateral incision, although this necessitates sufficient hospital resources to ensure close monitoring.[49]

Begin the double-incision approach. First perform an anterolateral incision to decompress the anterior and lateral compartments. Make a 20 to 25 cm long longitudinal incision between the fibular

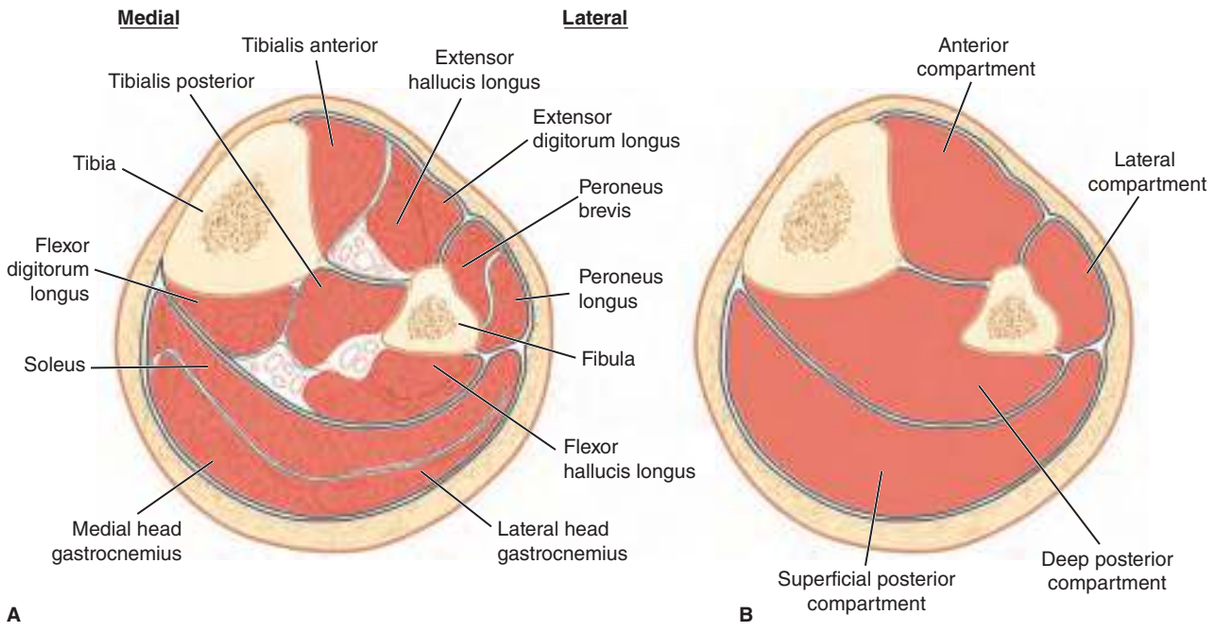


FIGURE 94-12. The compartments of the leg.

shaft and tibial crest (**Figure 94-13*A***).[1] **The aim is for the incision to be located over the anterior intermuscular septum, the landmark that guides access to both the anterior and lateral compartments (Figure 94-13*C*).** Ultrasound could potentially be used to identify the anterior intermuscular septum and allow for a more precise skin incision. How this would perform in the clinical setting has yet to be determined.[50] Undermine the skin edges to expose more of the fascial covering. Make a small transverse incision through the fascia at the midpoint of the incision to expose the intermuscular septum. **Be cautious not to cut the superficial peroneal nerve that lies just lateral to the septum.** Instruct an assistant to retract the skin edges. Open the anterior compartment using Metzenbaum scissors to cut the investing fascia. Extend the fascial incision longitudinally, both distally and proximally, the length of the skin incision. Decompress the lateral compartment by incising its investing fascia longitudinally in a similar manner.

TABLE 94-6 The Compartments of the Leg and Their Contents

Compartment	Contents
Anterior	Tibialis anterior muscle
	Extensor hallucis longus muscle
	Extensor digitorum communis muscle
	Anterior tibial artery and vein
	Deep peroneal nerve
Lateral	Peroneus longus muscle
	Peroneus brevis muscle
	Superficial peroneal nerve
Superficial posterior	Gastrocnemius muscle
	Soleus muscle
	Plantaris muscle
	Sural nerve
Deep posterior	Tibialis posterior muscle
	Flexor hallucis longus muscle
	Flexor digitorum longus muscle
	Posterior tibial artery and vein
	Peroneal artery and vein
	Tibial nerve

Perform a second incision along the posteromedial leg to access the posterior compartments (**Figure 94-13*B***). The two posterior compartments can be decompressed with one skin incision (**Figure 94-13*C***). Make a 20 to 25 cm long longitudinal incision 2 to 3 cm posterior to the posterior tibial margin (**Figure 94-13*B***). Undermine the skin and subcutaneous tissues on both sides of the incision. Instruct an assistant to retract the skin and subcutaneous tissue on both sides of the incision. Incise the crural fascia transversely to expose the medial edge of the posterior intermuscular septum. It is located anterior to the soleus muscle, leads inwardly to the transverse intermuscular septum, and divides the deep and superficial posterior compartments. Decompress the superficial posterior compartment. Make a longitudinal incision posterior to the posterior intramuscular septum overlying the soleus muscle. Extend the incision the length of the skin incision. Decompress the deep posterior compartment. Retract the soleus muscle laterally and make a longitudinal fascial incision just posterior to the tibia and anterior to the soleus muscle. This results in incising the transverse intermuscular septum overlying the flexor digitorum longus to access the deep compartment. Extend this incision both proximally and distally.

THE FOOT

The number of compartments within the foot is a matter of controversy (**Figure 94-14**).[51] It was initially believed that there were four compartments. Further research has shown that up to nine compartments exist. Subsequent studies have shown that the barriers between many of these compartments break down at pressures greater than 10 mmHg, far less than that required to cause a compartment syndrome. For the sake of this chapter we will divide the nine compartments into four groups: the interosseous compartment, the medial compartment, the central compartment, and the lateral compartment.[15] A list of the contents of each compartment is noted in **Table 94-7** and **Figure 94-14**.

There are two main approaches used to access the compartments of the foot and both can potentially reach all the compartments.[52-54] The most popular approach is the medial approach which most readily accesses the medial and central compartments (**Figure 94-15*A***).

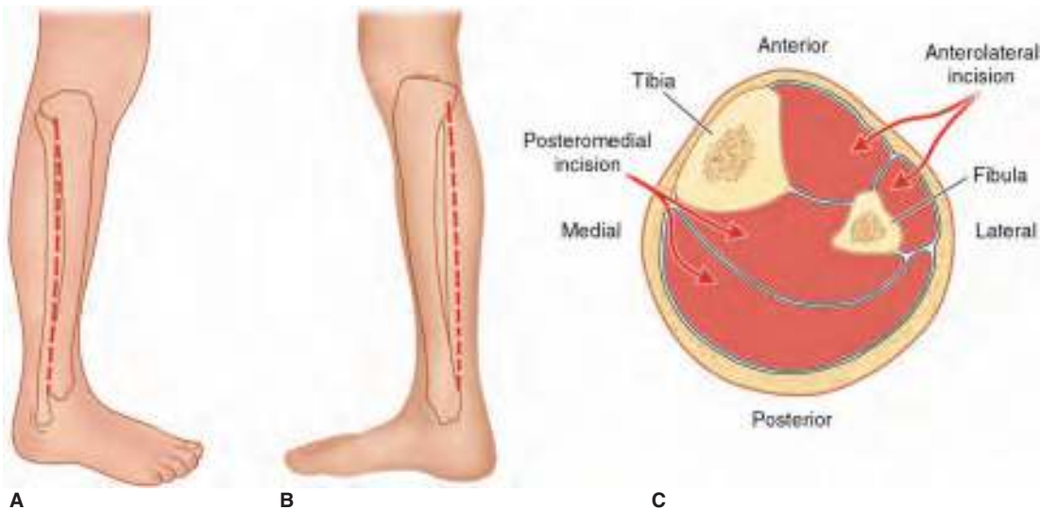


FIGURE 94-13. Fasciotomy incisions for the leg. ***A.*** The anterolateral incision. ***B.*** The posteromedial incision. ***C.*** Cross-section through the leg demonstrating the fasciotomy incisions.

Make the skin incision starting just below the medial malleolus and over the superior aspect of the adductor hallucis muscle. Continue the incision distally along the inferior border of the first metatarsal and superior to the abductor muscle. Retract the skin edges. Identify the neurovascular bundle and retract it out of the way. Incise the fascia of the medial compartment. Continue incising laterally through the medial intermuscular septum of the medial compartment. The remaining compartments can be accessed by blunt dissection with a clamp or by performing additional incisions via the dorsal approach.

The dorsal approach involves two incisions and is best used to access the interosseous compartments and the lateral compartment (**Figure 94-15*B***). This approach is superior to the medial approach in the setting of metatarsal or Lisfranc fractures. Make the first incision along the medial aspect of the second metatarsal. Make the second incision along the lateral aspect of the fourth metatarsal. Incise the superficial fascia of each intermetatarsal space and elevate the

TABLE 94-7 The Compartments of the Foot and Their Contents

Compartment	Contents
Interosseous	Interosseous muscles
	Digital nerves
Medial	Abductor hallucis muscle and tendon
	Flexor hallucis brevis muscle
	Tendon of the flexor hallucis longus
	Medial plantar vessels and nerves
Central	Flexor digitorum brevis muscle
	Quadratus plantae muscle
	Adductor hallucis muscle
	Tendons of flexor digitorum longus
	Lateral planter vessels and nerves
Lateral	Flexor digiti minimi muscle
	Abductor digiti minimi muscle

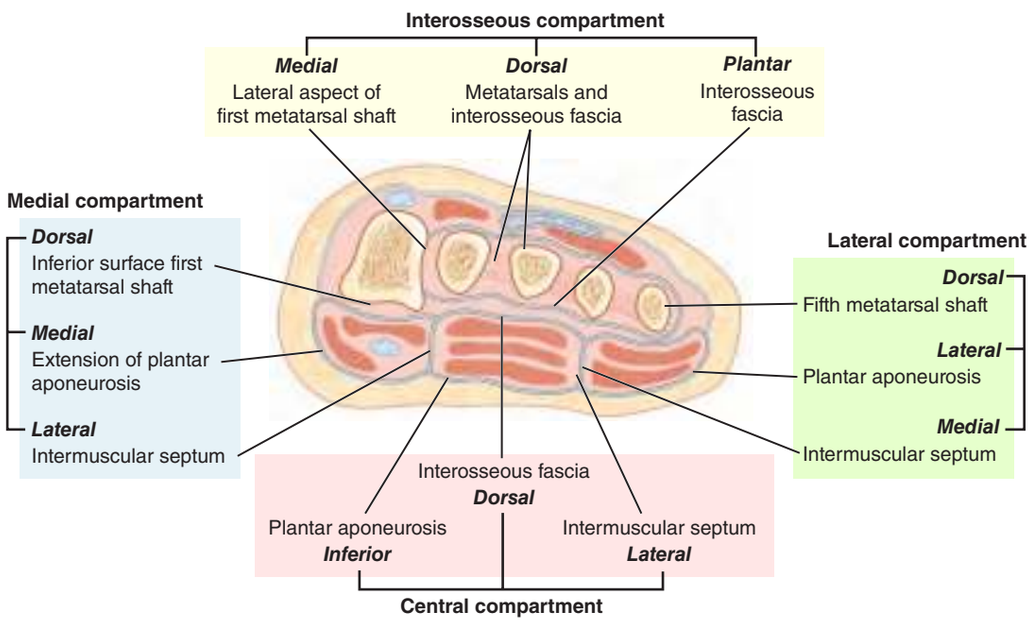


FIGURE 94-14. The compartments of the foot.

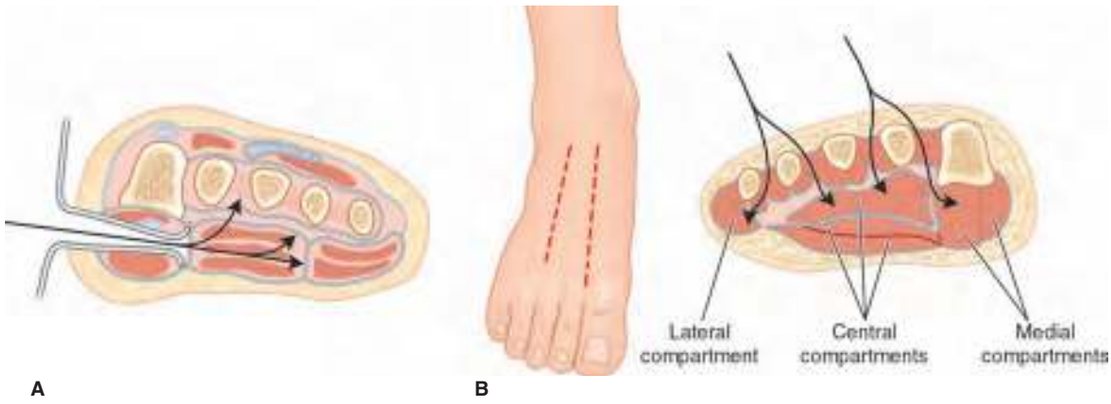


FIGURE 94-15. Fasciotomy incisions for the foot. ***A.*** The medial approach. ***B.*** The dorsal approach.

muscles off the bone for more effective decompression. Use a clamp to bluntly dissect, access, and decompress the remainder of the compartments (**Figures 94-14 and 94-15*B***). The lateral compartment can be accessed and decompressed through the second incision. The central compartment can be accessed and decompressed through either incision. The medial compartment can be accessed and decompressed through the first incision.

ASSESSMENT

Aseptically measure all compartment pressures after each fasciotomy. **Many compartments communicate and a fasciotomy of one compartment may relieve the pressure in others and obviate the need for additional fasciotomies. After the fasciotomy is complete, compartment pressures should be measured again to ensure successful decompression.** Check multiple readings throughout the same compartment.[1] **Ensure that the fascia is completely incised and consider increasing the length of the fasciotomy if the compartment pressures are still elevated.**

AFTERCARE

The compartments and wounds are left open once decompressed. Apply sterile saline-soaked gauze into the incisions followed by a nonrestrictive and bulky dressing wrapped around the limb. The dressing should be loose and allow the wound to easily drain.[35] Splint the limb to prevent contractures. The limb must be splinted so that the fractured bones are in appropriate alignment in cases where the decompressed limb has a fracture.[36] Consult the appropriate Surgeon (e.g., Hand, Orthopedic, Plastic, or Trauma) to manage the patient. The decision to administer prophylactic antibiotics should be made in consultation with the Surgeon. Admit the patient for further care.

COMPLICATIONS

The goal of surgical decompression is to open the tight fascial layers.[36] **Minimizing the length of the skin incisions or limiting the number of compartments decompressed will lead to substandard results.** Continued edema and postischemic hyperemia in the first few hours after a decompressive fasciotomy can lead to increased compartment pressures in adjacent compartments that were not decompressed.

There is a significant risk of an iatrogenic injury to arteries, nerves, tendons, and veins during the fasciotomy.[1] A thorough understanding of the local anatomy and the procedure will help to minimize these injuries. Adequate visualization using proper overhead lighting and proper retraction will further minimize injury.

The diagnosis of a compartment syndrome may be delayed because of a missed diagnosis or because the patient does not present immediately to the Emergency Department. The Emergency Physician may be faced with a situation in which the muscles of the limb are in varying stages of ischemia, irreversible injury, and contracture. Performing a fasciotomy may predispose the limb to secondary infection with subsequent gram-negative sepsis.[35] **The Emergency Physician must weigh the risks and benefits before proceeding. The decision to perform a fasciotomy should be made in consultation with a Surgeon.**

Infection after a fasciotomy is a definite possibility. The use of aseptic technique is mandatory but does not prevent subsequent infections. The presence of crush injuries, myonecrosis, poor perfusion, preexisting peripheral vascular disease, and a compromised immune status can predispose the site to an infection. The decision to use broad-spectrum prophylactic antibiotics should be made in consultation with a Surgeon.

Hemorrhage is often minimal when performing a fasciotomy. Most hemorrhage can be controlled with direct pressure. Small subcutaneous blood vessels transected during the procedure can be controlled with electrocautery or figure-of-eight sutures.

SUMMARY

Emergency Physicians must understand the signs and symptoms of a compartment syndrome. Always maintain a high level of suspicion in patients presenting with limb injuries. The technique itself is quite simple when the incision is made in the proper location. Performing a fasciotomy in a timely fashion can prevent serious and irreversible consequences.

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95 Field Amputation of the Extremity

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INTRODUCTION

An amputation is an old procedure. Early amputations were crude: amputations were performed without anesthesia, hemostasis was achieved by dipping the amputated extremity in hot oil, and the mortality was high. The first reference to an amputation was found in the ancient Babylonian code of Hammurabi in 1700 BC. Hippocrates described the amputation for vascular gangrene in 385 BC. William Cloves performed the first successful above-the-knee amputation in 1588. The tourniquet was described in the 16th century by Botallus and Holdani. The development of anesthesia in the 16th century made the procedure easier for all involved. Jean Domnique performed 200 limb amputations and 11 shoulder disarticulations. Norman Kick used the guillotine amputation during World War II. This procedure has undergone many advancements since the first

amputations. It is a common procedure, with approximately 65,000 amputations occurring annually usually due to peripheral vascular disease of the lower extremities. An amputation is often performed in a hospital but the acceptance by the patient is still low.

Relatively common situations in which rescuers may find victims entrapped include machinery limb entanglement (e.g., agricultural, construction, or industrial), motor vehicle collisions, confined space rescues, and structural collapses. Rescuers have been able to achieve success in situations that were previously futile as extrication technology has progressed. The methods of safe extrication may be exhausted or the patient's clinical course may not be stable enough to endure prolonged extrication attempts. The actions of extrication can sometimes put rescuers or other victims at risk. This is particularly true in structural collapse situations. A prehospital field amputation of entrapped or entangled limbs may be the only way to save the patient.

Prehospital field amputation is performed infrequently. Very little is published on the overall incidence of the procedure except in case reports.[1-10] It is a lifesaving procedure with several indications to be performed in the prehospital environment. Training in limb amputations is not standardized and is infrequently available in Emergency Medical Services (EMS) agencies for their providers. An interview occurred with Medical Directors at the National Association of EMS Physicians (NAEMSP) meeting in 1992.[5] The number of prehospital extremity amputations in the prior 5 years was reported to be 26 by the 143 Medical Directors. Almost all (96%) reported there was no formal training in the procedure or its indications.[5] This highlights the issue of lack of standardization.

Formal training, protocols, and preformed teams are rare.[5] It is conceivable that a local Emergency Physician or Surgeon may be called upon to assist in a prehospital amputation with no prior formal training or equipment preparation.

The amputation may be achieved with the cutting of a long bone or with a disarticulation at a joint. The joint capsule, tendons, and ligaments are transected in a disarticulation as opposed to cutting the bone in an amputation. Severely mutilated limb injuries may only have minimal connective tissue remaining (e.g., nearly complete traumatic amputations).

ANATOMY AND PATHOPHYSIOLOGY

The anatomy involved in such a procedure is dependent on the limb(s) involved. The entanglement may be as simple as a single digit or as complicated as multiple limbs with proximal involvement. Torso entrapment or crush injury may not be amenable to amputation as a method for rescue. Such situations will not be discussed. Any prehospital amputation procedure of an entrapped extremity includes exposure to skin, tendons and ligaments, muscles, blood vessels, nerves, and bone. The injury may distort the anatomy to the point of being unrecognizable (e.g., a mangled or rotated extremity). The basic anatomy of the extremities is sufficient knowledge to perform a field amputation.

The entrapped patient may be subject to numerous assaults on their body's pathophysiology. Trauma to the limbs may be blunt or penetrating. The patient may have sustained multisystem trauma (e.g., structural collapse or motor vehicle collisions). **Attention must be paid to the patient's overall hemodynamics**. External and internal blood loss from the traumatic injuries can lead to shock and coagulopathy. Fluid resuscitation of the entrapped patient can allow additional field time and further attempts at extrication prior to resorting to an amputation. A large area of limb entrapment may lead to a crush syndrome. The pressure applied to muscle groups and blood vessels by external forces may result in areas of ischemia if the entrapment is prolonged. Subsequent rhabdomyolysis results in the release of large amounts of myoglobin, potassium, and other cellular contents. This can result in metabolic acidosis and renal failure.[11] The management of crush syndrome includes large-volume hydration that must be balanced with permissive hypotension to avoid the dilution of clotting factors.

INDICATIONS

Two sets of indications have been developed for prehospital amputations.[12,13] They are not mutually exclusive and have some overlap. One set of criteria was developed from experiences from prior case reports and incidents.[12] These criteria tend to be more conservative. They include the dismemberment of a deceased patient to access live patients. Emergency Physicians should be aware that dismemberment of a deceased individual may be taboo in some cultures.[6] The situation should be discussed, if possible, with the family of the patient prior to proceeding to dismember the deceased.[6] The other indications established a prehospital extremity amputation protocol for EMS Physicians through the Medical College of Wisconsin which included four situations that may warrant prehospital amputation.[13] The indications for prehospital amputation are summarized in **Table 95-1**.

CONTRAINDICATIONS

The only absolute contraindication to the procedure is when the Emergency Physician performing the procedure is placed at risk and it is deemed too dangerous. The safety of the rescuers is paramount.[12] One may consider a relative contraindication to be the informed refusal of the procedure by a conscious patient who understands the risk of clinical deterioration during continued extrication attempts. The ethical considerations of such a refusal are beyond the scope of this text. The local hospital ethics board should be emergently consulted in such a situation.

EQUIPMENT

- Trauma shears*
- Gigli bone saw* (**Figure 95-1**)
- Several hemostats or Kelly clamps*
- Several trauma tourniquets*
- Scalpel*
- Medications for anesthesia, preferably procedural sedation* (Chapter 159)

TABLE 95-1 The Indications for a Prehospital Amputation

- An immediate and real risk to the patient's life due to a scene safety emergency.
- A deteriorating patient physically trapped by a limb and they will almost certainly die during the time taken to secure extrication.
- A completely mutilated and nonsurvivable limb retaining minimal attachment and delaying extrication and evacuation from the scene.
- The patient is dead and their limbs or body are blocking access to potentially live casualties.
- Extrication will not occur rapidly, the patient is hypotensive and the patient is considered a nonresponder to initial IV fluids (i.e., life before limb).
- Extrication will not occur rapidly and further structural collapse or bodily injury is imminent if they are not rapidly extricated.
- Extrication will be extended, the patient was initially hypotensive but responded to initial IV fluids, and the patient currently has an adequate blood pressure.
- The patient is hemodynamically normal and in the best judgment of the rescue personnel and field physician extrication is likely to take many hours if it can be done at all.

Source: Modified from references 12 and 13.

FIGURE 95-1. The Gigli saw.

- Sterile gloves
- Sterile gown
- Face mask
- Goggles
- Foot protection
- Vascular access (e.g., intravenous [IV] or intraosseous [IO])
- Sterile towels or drapes
- Povidone iodine or chlorhexidine solution
- Liston amputation knife
- Laparotomy pads
- Trauma dressings
- Bone wax
- Elastic bandage
- Mayo scissors
- Sterile saline or sterile water for irrigation
- Portable battery-powered oscillating bone saw (optional) (**Figure 95-2**)

The minimum equipment needed in an austere environment or a confined space is noted with an asterisk (*) in the above list and shown in **Figure 95-3**. It is not always possible to have the required equipment in an emergency. A field amputation has been performed with equipment borrowed from firefighters.[14,15] This includes a hacksaw, a reciprocating saw, or a specialized cutting tool (**Figure 95-4**). Rescue tools serve as acceptable alternatives to surgical equipment. A comparison of the classic Gigli wire bone saw with rescue service hacksaws, reciprocating saws, and hydraulic cutting tools was performed in a simulated prehospital amputation scenario on cadavers.[14] All of the tools were capable of amputation in less than 2 minutes. Each technique had various benefits and drawbacks.

FIGURE 95-2. The Stryker oscillating bone saw is battery operated and portable.

FIGURE 95-3. The minimum required equipment for the field amputation of an extremity. Clockwise from top left: anesthesia and analgesia (ketamine), Kelly clamps and/or hemostats, trauma shears, combat tourniquet, Gigli saw, and a scalpel.

PATIENT PREPARATION

The conscious and oriented patient should be involved in the discussion regarding a potential prehospital amputation. Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. The postprocedural care should also be discussed. It is sometimes the case that the Emergency Physician may be able to obtain written consent. Some Emergency Physicians may omit the signed patient consent and place in the procedure note a documentation such as the following: "The risks, benefits, and complications were described and discussed with the patient. They understood this and gave verbal consent for the procedure." Have a witness also sign this if it was witnessed. Attempt to discuss the procedure with next of kin and obtain consent if the patient is unable to participate in the decision-making process. Two Emergency Physicians should agree on a consensus if no decision-maker is available for the unconscious or delirious patient. Advances in telecommunication technology have provided cellular phones and mobile videoconferencing. Such methods are available when a second Emergency Physician is only available remotely. A verbal discussion over a recorded line is ideal. A consensus agreement between the Emergency Physician and officer in charge of the scene will do if only one physician is present and no other physician is available. **Clearly document the decision-making process and attempts at consent.**[6,7,10,13,16]

The subsequent extrication route should be preplanned prior to the field amputation procedure. Resuscitate the patient. The patient's airway should be controlled but it should not be considered mandatory to intubate. This is especially true as space and equipment constraints may preclude airway management. Effective IV or IO access will allow for adequate analgesia, anesthesia, and fluid resuscitation. Ketamine has the ideal properties for a prehospital amputation. It

A

B

C

D

E

FIGURE 95-4. Equipment that may be available at a scene for amputation. ***A.*** DeWalt hacksaw. ***B.*** Rigid Prohack hacksaw. ***C.*** Durofix 18 volt reciprocating saw. ***D.*** Homatro cable cutter. ***E.*** Homatro cutter.

allows for analgesia, anesthesia, retention of airway reflexes, and retention of respirations (Chapters 12 and 159).

The Emergency Physician should be wearing a gown, gloves, and a face mask with an eye shield or goggles. Foot protectors are desirable. A face mask and eye protection will prevent a mucous membrane exposure if the contents spray out of the incision. The gown will protect clothing from contamination. The patient's injury may cause significant external hemorrhage with subsequent bloodborne exposure hazards throughout the rescue area.[6,12,13]

Remove any overlying clothing or debris from the limb. Apply a tourniquet proximally on the affected limb. Crushing and entrapment forces to the deep vasculature often tamponade the hemorrhage. The tamponade is relieved during the field amputation. Note the time of the tourniquet application and write this on the limb or tourniquet. Some EMS protocols include dispatch notification of tourniquet application. The first responders have often exposed the affected limb and applied a tourniquet. Administer broad-spectrum antibiotics prior to the procedure if they are available and the patient has no allergies. The conditions in which the amputation will be performed are far from sterile (e.g., industrial machinery, rubble of a structural collapse, and mangled passenger compartment of a motor vehicle). Establish a sterile field as best as possible with sterile drapes and towels. This allows a safe area to place and prepare the necessary equipment. Gross decontamination is initially achieved using sterile water or saline to remove particulate matter and debris. Perform an antiseptic cleaning with povidone iodine or chlorhexidine gluconate and allow it to dry.[6,12,13]

TECHNIQUES

FIELD AMPUTATION

The general techniques remain the same regardless of the extremity involved or the level at which it is entrapped. The procedure may be scaled up or down depending on whether it is a phalanx or a proximal long bone involved in the entrapment. **All amputations in the prehospital environment are performed as guillotine amputations. The anatomic structures are transected in an axial plane as if a guillotine dropped onto the limb.**[8,9,12,13,17,18] This is in contrast to a skin and soft tissue flap formed in the Operating Room for the purpose of forming a mature stump. The ultimate technique and equipment used may depend on what is available at the time of the emergency.

Perform the amputation as distally on the limb as possible to allow the best possible functional outcome for the patient. For example, a proximal humerus amputation provides far less function, even with a prosthesis, than a distal forearm amputation. Make sure the tourniquet is placed proximally on the limb and at least 5 cm

A B

FIGURE 95-5. An example of a joint disarticulation. ***A.*** The level of the disarticulation. ***B.*** The disarticulation. (Used with permission from reference 15.)

(i.e., 2 inches) above the site of amputation. Tighten the tourniquet to stop blood flow into the limb. Place a second tourniquet if needed for continued hemorrhage from the limb prior to the amputation. Cut the skin and soft tissue with the scalpel or Liston amputation knife. The Liston amputation knife is more expensive than a scalpel, takes up more space than a scalpel, and is usually not available. Some patients may have a nearly complete traumatic amputation with only soft tissue remaining on the limb. Continue cutting in the same plane as the initial cut to transect any tendons, ligaments, and or muscles. Keep cutting down to the bone. The procedure may require more than one scalpel because the blade quickly dulls. The Liston amputation knife provides the benefit of a double-edged knife with a greater cutting surface which is less likely to dull.

Use blunt dissection once the area around the bone has been transected. Insert a Kelly clamp behind the bone. Push the closed Kelly clamp behind the bone and through to the other side. Grasp a laparotomy pad with the clamps and pull it pull back through the wound. Allow approximately equal halves of the laparotomy pad to hang out from behind the bone. Use the laparotomy pad to pull proximal traction and expose the bone surface. Cut the bone with the powered oscillating bone saw, a Gigli saw, or another device. Use an assistant to apply a gentle downward stabilizing force on the limb while cutting the bone. Use trauma shears, Mayo scissors, or the scalpel to divide all remaining tissue of the limb in the same axial plane as the rest of the procedure.

Attention must now be paid to ensuring hemostasis. Clamp any large blood vessels identified with hemostats or Kelly clamps. **This redundancy will minimize hemorrhage if the tourniquet becomes dislodged.** The tourniquet will stop most blood vessel hemorrhage. The vascular matrix of the bone is not amenable to external pressure. Apply bone wax to the end of the transected bone surfaces in the amputation stump to decrease bleeding. Hemostatic agents may be effective to manage continued bleeding from the soft tissue, but this has not yet been described in the literature. Apply a sterile dressing to the stump. Secure the dressing in place.

FIELD DISARTICULATION

The general technique is similar to the technique for an amputation. **Perform the disarticulation at the most proximal joint above the injury and as distally on the limb as possible to allow the best possible functional outcome for the patient.** Make sure the tourniquet is placed proximally on the limb and at least 5 cm (i.e., 2 inches) above the site of disarticulation. Tighten the tourniquet to stop blood flow into the limb. Place a second tourniquet if needed for continued hemorrhage from the limb prior to the disarticulation. Identify the joint line and mark it on the skin (**Figure 95-5*A***). Cut the skin and soft tissue with the scalpel or Liston amputation knife. Continue cutting along the joint line to transect any tendons, ligaments, and/or muscles. The procedure may require more than one scalpel as the blade quickly dulls. Separate the limb at the joint (**Figure 95-5*B***).

Attention must now be paid to ensuring hemostasis. Clamp any large blood vessels identified with hemostats or Kelly clamps. **This redundancy will minimize hemorrhage if the tourniquet becomes dislodged.** The tourniquet will stop most blood vessel hemorrhage. The vascular matrix of the bone will not bleed in a disarticulation. Hemostatic agents may be effective to manage continued bleeding from the soft tissue, but this has not yet been described in the literature. Apply a sterile dressing to the stump. Secure the dressing in place.

PEDIATRIC CONSIDERATIONS

The procedure for pediatric patients is largely unchanged from that of a bigger person. Some techniques may need to be slightly altered. Pediatric limbs are significantly shorter. Preserve as much length as possible and the growth plates. A disarticulation is preferred over an amputation. The pediatric limb allows for less space above the injury for tourniquet application. Commercially available tourniquets may not work on smaller children due to their length and width. Use an improvised tourniquet or a manual pediatric blood pressure cuff. Ensure the cuff has no air leaks prior to its application. Apply tape circumferentially around the cuff to ensure there is no dislodgement of the cuff. Apply a Kelly clamp to the cuff tubing to decrease any leakage from the cuff via the thumbscrew valve after inflation of the cuff. Proceed with the amputation or disarticulation procedure.

AFTERCARE

Promptly transport the patient to the closest hospital capable of managing the injuries. The Emergency Physician who performed the amputation should accompany the patient to the hospital to manage any ongoing bleeding or complications. Continuing patient care needs at the scene may require that the Emergency Physician stay behind (e.g., in the event of a mass casualty). Retrieve the

amputated limb if safe to do so. Place the limb in a bag covered with sterile dressings soaked in sterile saline. **Do not place the limb on ice as this may cause further tissue damage.** The reimplantation of the amputated extremity is unlikely. The decision is best left to the Surgeons at the hospital. The amputated extremity can be used at the very least for autologous skin grafting by the Surgeon when modifying the stump.[6,12,13]

COMPLICATIONS

There is often little preparation of predefined Physicians or equipment prior to the sudden unexpected need for a field amputation.[5] This can lead to the complications of hastily prepared equipment or unprepared surgical teams being deployed into a prehospital environment for the first time. There may be practical complications associated with the attempts to integrate hospital providers into a prehospital environment and the chain of command with no prior field experience. Hospital providers will need to be quickly briefed on safety concerns and risks on the scene. Various hazards to the hospital provider on the scene may include traffic awareness on a highway, bloodborne pathogens, unstable structures, safety around rescue tools, and safety when working in confined space environments.

There may be legal, cultural, or religious ramifications for amputations of living patients or the dismemberment of deceased victims. Early involvement of family and local leaders may aid in the decision-making process and long-term quality of life for the patient.[6] Providers at the scene may endure psychological trauma from the severity of injury and the amputation procedure. Scene supervisors should assess health care providers for the need for further critical incident stress management.[7]

There are numerous complications associated with the procedure. Bleeding is possible with any prehospital amputation. Tourniquets should be checked multiple times to ensure they are well placed and adequately secured prior to the procedure and prior to the transport. Care must be taken to avoid dislodgement of the tourniquet when extricating the patient after the amputation. **Infection and gas gangrene of the wound are concerns.** Perform the procedure as cleanly as possible. A formal washout of the amputated extremity in the Operating Room is mandatory. Multiple revisions of the amputated stump will likely be required. Hemorrhagic shock and multisystem trauma are possible. Follow resuscitation protocols to minimize complications.[13] Complications are common in survivors of field amputations. These include depression, loss of body image, loss of function, loss of independence, loss of mobility, loss of sensation, and the feeling of suicidal ideation. The patient may need psychiatric therapy for these issues.

The late complications can occur with any amputation no matter where it is performed (i.e., field or hospital). A bone spur often forms on the end of the amputated bone. This is usually painless but can cause an ulceration of the skin. The bone can become osteoporotic from disuse and easily fracture. The joint above the amputation may become deformed and stiff. A painful and tender neuroma may develop at the amputation site. Numerous conditions, including breakdown of the muscle and skin due to poor circulation, phantom limb feelings, and phantom limb pain can develop. The preservation of a life by performing the amputation is worth these complications.

SUMMARY

The field amputation of an entrapped extremity is a very low-frequency, high-acuity procedure that Emergency Physicians are generally ill-prepared to handle.[5] A major EMS system may go years without a single prehospital amputation request, only to have several performed within a short time.[7] Ensuring a predefined team with established protocols and prepared equipment will improve the chances of a successful resuscitation and rescue of an entrapped patient. The presence of an Emergency Physician on scene directing the resuscitation, bringing additional medications, and bringing blood products may stabilize a patient long enough for a successful extrication without amputation. Familiarity with the anatomy, equipment, indications, and procedure will go a long way to reduce apprehension and improve outcomes if the need for a prehospital amputation is determined.

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96 Extensor Tendon Repair

JoAnna Leuck and Keegan Bradley

INTRODUCTION

The Emergency Physician (EP) commonly encounters lacerations or trauma to the dorsum of the hand and forearm. **The possibility of extensor tendon lacerations must be considered in evaluating these patients.** A recent study found that tendon injuries in the hand and wrist occur at a rate of 33.2 injuries per 100,000

person-years, with extensor tendon injuries being the most common.[1] Single, shallow lacerations account for 54.4% of the tendon injuries studied and the most common location is the long finger.[1] The extensor mechanism of the hand and forearm is typically disrupted in association with penetrating trauma. Blunt trauma (e.g., sudden forced flexion) can result in injury to extensor tendons. Performing an extensor tendon repair is an important skill in the EP's surgical armamentarium.

The diagnosis of an extensor tendon injury must be identified during the initial examination. The timing of the tendon repair is not a critical aspect of its management. Successful repair of extensor tendons may be accomplished within a 7 day window following the injury.[2] Splint immobilization of the damaged tendon can produce a similar outcome to surgical reapproximation at some anatomic sites. Laceration of < 50% of any tendon in all zones that the patient can extend against resistance can be immobilized with early protected motion.[3]

Repair of an extensor tendon by an EP requires familiarity with the anatomy of the region and skill in the surgical technique. Complications of tendon repair are more frequently associated with flexor tendons. Follow-up studies of extensor tendon repairs reveal similar pitfalls and problems.[4] Adhesions, loss of length, tendon rupture, Swan neck deformity, Boutonniere deformity, and diminished flexion can all complicate the repair of an extensor tendon.[5]

The anatomy of the extensor mechanism prevents tendon retraction far from the site of a laceration or partial disruption.[6] This is mostly due to the tethering of tendons by multiple interconnections as tendons cross the dorsum of the hand. Tendons over the dorsum of the hand are ensheathed in a paratenon layer of tissue. This covering is extrasynovial and contains the cut ends of tendons in a tissue layer that prevents their wide separation. These properties frequently allow both ends of a lacerated extensor tendon to be located with local wound exploration.

The techniques for extensor tendon repair originate in studies of flexor tendons. **The goal of extensor tendon repair is to restore tendon continuity and function while minimizing interference from the repair itself.** The suture techniques of Kessler and Bunnell are two of the methods traditionally used in this repair. Modifications of these original methods have resulted in the greatest outcome measurements of tendon strength.[7] **Familiarity with these two suture techniques, knowledge of extensor tendon anatomy, and knowledge of surrounding structure anatomy is essential to the successful repair of any extensor tendon in the Emergency Department (ED).**

ANATOMY AND PATHOPHYSIOLOGY

The extensor tendon mechanism is an intricate system of pulleys and levers coursing along the dorsum of the forearm, wrist, and hand.[8-11] The function of these tendons is to extend the fingers and wrist from a flexed position. This finger motion is complemented by the actions of the intrinsic hand muscle groups (i.e., lumbricals and interossei).

The elegant anatomy of the hand extensor mechanism is best appreciated in diagrammatic representation (**Figure 96-1**). The forearm tendons pass through the extensor retinaculum of the wrist

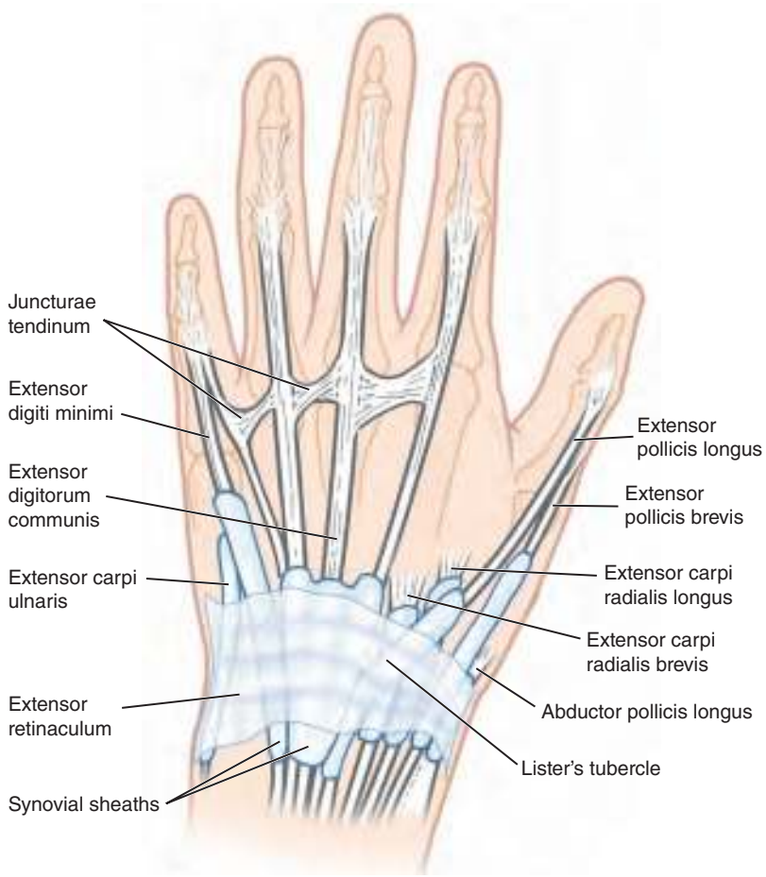

FIGURE 96-1. The extensor tendons of the hand begin just proximal to the extensor retinaculum. After exiting from under this encasing sheath, the tendons form an interconnected network as they cross the dorsum of the hand.

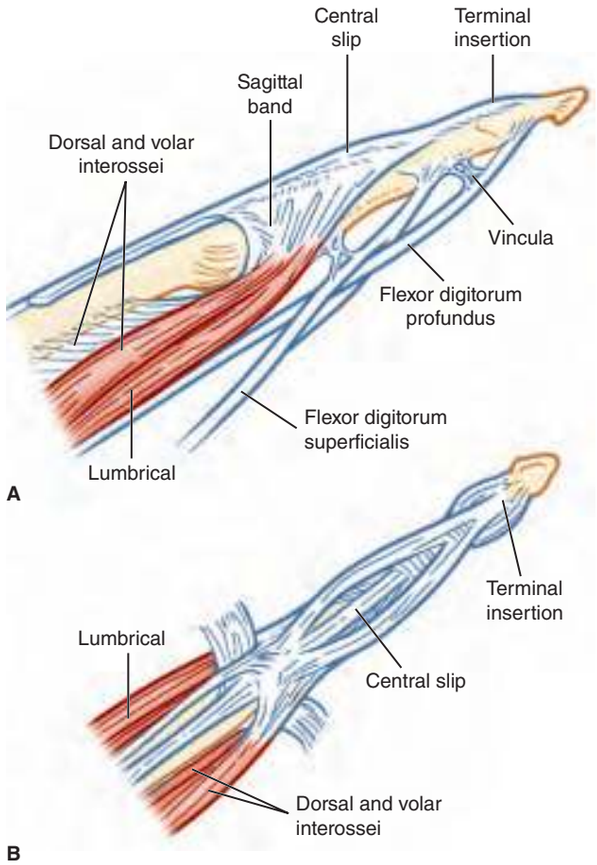

FIGURE 96-2. The central extensor tendons of the digits are reinforced and aided by the lateral bands of the interosseous and lumbrical muscles. The extensor tendon is tethered to the joint surface by a sagittal band, which forms a protective hood. ***A.*** Lateral view. ***B.*** Superior view.

FIGURE 96-3. Division of an extensor tendon in the dorsum of the hand may still allow for full finger extension because of the communicating juncturae tendinum.

to travel across the dorsum of the hand to each of the fingers. The extensor tendons are reinforced in the fingers by the lateral bands of the intrinsic hand muscles to form a complex tendon that inserts onto the distal phalanx (**Figure 96-2**).

Knowledge of the anatomy of the extensor mechanism reveals certain salient points. The extensor retinaculum of the wrist is a complicated structure of fibrous canals through which the tendons pass and whose repair necessitates a Hand Surgeon's intervention. The paucity of soft tissue covering the dorsum of the hand makes extensor tendons quite vulnerable to penetrating trauma. The extensor tendons of the fingers are interconnected across the dorsum of the hand by the juncturae tendinum (**Figure 96-1**). This interconnection of tendons distributes the work of extension among the involved fingers. Redundancy is built into the extension of the first and small fingers with the presence of two separate extensor systems. An isolated tear of the extensor tendon of the index or small finger may not result in lack of function because of the contributing band from the extensor digitorum communis (**Figure 96-3**). There is great anatomic variability in the detailed distribution of these tendons.

The location of an extensor tendon injury is important in determining whether tendon repair in the acute ED setting is feasible. Eight zones define the location of an extensor tendon injury (**Figure 96-4 and Table 96-1**). The zone chart was first described by Kleinert and Verdan to classify and organize the modes of repair.[12,13] It is most commonly referred to as the Kleinert system. The odd-numbered zones refer to areas over the joints. The even-numbered zones refer to the bony areas. Any zone can experience injury and undergo repair. The outcome is variable, depending on the suture technique and the mode of rehabilitation.

INDICATIONS

The decision to surgically repair a lacerated extensor tendon is multifactorial. One must consider the extent of the tendon laceration, the involvement of other tissues (e.g., bone or joint space), and the location of the tendon injury. Studies that define a minimum laceration width that requires tendon repair have not been performed. **It is generally accepted that a laceration greater than 50% of the tendon width requires surgical repair.**[2] A tendon that has a laceration less than 50% of its width will usually heal with conservative management. **One must put the finger through its entire range of motion to confirm normal function and movement of the affected tendon prior to making the decision as to whether conservative management is appropriate.** A lacerated tendon that is associated with significant overlying skin loss, joint space penetration, or a bony fracture will require repair by an Orthopedic or Hand Surgeon.

Defining the location of the tendon injury to the Kleinert zone system guides the decision of which tendons can be safely repaired in the ED. There are no published guidelines on whether these procedures should be performed at the bedside or in the Operating Room. A thorough understanding of the extensor tendon's anatomy and a good dose of common sense are imperative.

The following zones and types of injury represent sites where extensor tendon repair in the ED is feasible with minimal complications. The thickness of the extensor pollicis longus allows for a

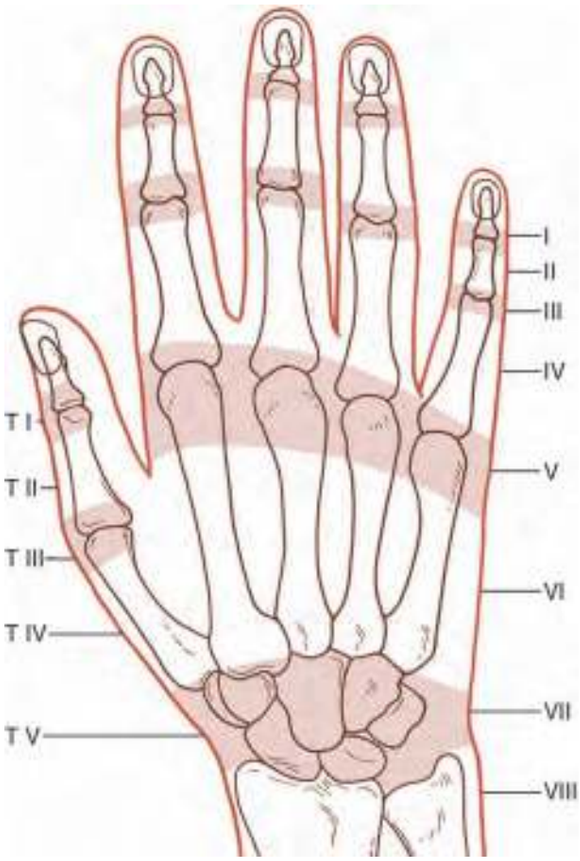

FIGURE 96-4. The extensor tendons of the hand are easily classified by the Kleinert zone system. The odd numbers represent joint spaces. The even numbers represent long bones. Note that the thumb zones are distinct from the other digits.

TABLE 96-1 The Characteristics of Injuries to the Zones of the Extensor Tendons

Zone	Characteristics
Zone 1	• Disruption of terminal extensor tendon distal to or at the DIP joint of the fingers and IP joint of the thumb • Forceful flexion of the DIP joint with an extended finger • Open or closed • Flexion deformity over DIP joint • Often accompanied by a fracture resulting in a mallet finger • Can result in swan neck deformity if untreated
Zone 2	• Disruption of tendon over middle phalanx or proximal phalanx of thumb • Results from crushing injury or lacerations
Zone 3	• Disruption of central slip over the PIP joint of digit or MCP joint of the thumb • Open or closed • May be associated with a fracture • Often results in a Boutonniere deformity
Zone 4	• Disruption over the proximal phalanx of digit or metacarpal of thumb • Lateral bands are spared
Zone 5	• Disruption over MCP joint of digit or CMC joint of thumb • Commonly referred to as a fight bite • Rupture of the sagittal attachment • Usually open
Zone 6	• Disruption over the metacarpals • Nerve and vessel injury
Zone 7	• Disruption at the wrist joint • Must repair retinaculum to prevent complications (e.g., bowstringing) • Tendon repair followed by immobilization
Zone 8	• Disruption at the distal forearm above the wrist • May involve the musculotendinous junction

CMC, carpometacarpal; DIP, distal interphalangeal; IP, interphalangeal; MCP, metacarpophalangeal.
Source: Modified from references 3, 13, and 16.

core-type suture in zone 2 of the thumb. Repair of other zone 2 isolated injuries can be considered depending on the thickness of the tendon. The broad configuration of the extensor tendon allows for a core-type suture in zone 4 of all digits. Isolated involvement of the extensor tendon allows for a core-type suture unless the joint is involved in zone 5 of the fingers. Be alert for the possibility of zone 5 injuries being caused by a "fight bite." Extensor tendon injury from a human bite should be copiously washed out and consultation with a specialist is required. Isolated involvement of the extensor tendon allows for a core-type suture and good prognosis in zone 6 of the hand.

CONTRAINDICATIONS

There are definite contraindications to repairing an extensor tendon in the ED. First and foremost is the lack of skill on the part of the EP. Performing this repair may result in loss of function or may further compromise the patient's result. Consult an Orthopedic or Hand Surgeon if any doubt exists. Skin wounds can be closed loosely, the hand splinted, and the patient referred for delayed repair. Surgical repair of the tendon is not recommended if less than 50% of the tendon is lacerated and the finger functions as well as the corresponding finger on the unaffected opposite hand. The presence of a bony fracture, an open joint space, or the lack of adequate soft tissue or skin covering are contraindications to repairing an extensor tendon in the ED. **A tendon laceration from a human bite wound is an absolute contraindication to closure and repair of the injury.** The management and rehabilitation of these wounds necessitate consultation with the appropriate surgical specialist. Injuries to the thumb should be referred to a Hand Surgeon. Contraindications to immediate bedside repair by the EP are not wound-specific and include whether the patient has rheumatoid arthritis, is immunocompromised, or is a professional athlete.

The Kleinert zone chart can be used to help determine the suitability of repairing an extensor tendon in the ED. The following zones on the forearm, wrist, and hand represent areas where wound care, skin closure, and splinting are highly recommended until an Orthopedic or Hand Surgeon can perform definitive surgical repair. Extensor tendon remnants may be too short in zone 1 of all digits. Extensor tendons of the fingers that are very thin in zone 2 should not be repaired except for those in the thumb. Actual or potential joint or lateral band involvement in zone 3 of all digits should not be repaired. Actual or potential joint or sagittal band involvement in zone 5 of all digits should not be repaired. Actual or potential extensor retinaculum involvement in zone 7 should not be repaired because of its complex anatomy. The actual or potential need for a tendon transfer in zone 8 requires an Orthopedic or Hand Surgeon. Patients discharged from ED with extensor tendon injury must have acute follow-up with an Orthopedic or Hand Surgeon whether primarily repaired or splinted.

EQUIPMENT

DIGITAL OR HAND ANESTHESIA

- Povidone iodine or chlorhexidine solution
- 10 mL syringe
- 25 to 27 gauge needle, 2 inches long
- 16 to 18 gauge needle
- Local anesthetic solution without epinephrine

EXTENSOR WOUND IRRIGATION AND PREPARATION

- 250 to 500 mL of sterile saline or Ringer's lactate solution
- Irrigation set
- 60 mL syringe
- 16 gauge angiocatheter
- Intravenous tubing
- Personal protective equipment
- Blood pressure cuff, preferably automatic
- Overhead lamp or source of intense lighting

EXTENSOR TENDON AND SKIN REPAIR

- Sterile surgical towels
- 4×4 gauze squares
- Forceps
- #11 blade disposable scalpel
- Needle driver
- Nonabsorbable, synthetic, and braided suture (e.g., 4–0, 5–0, and 6–0 Ethibond or Mersilene)
- Nylon suture (e.g., 4–0 and 5–0) for skin closure

WOUND DRESSING AND SPLINT

- Topical antibiotic ointment
- Gauze squares
- Elastic gauze bandage
- Splinting materials

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the planned procedure to the patient and/or their representative. The broad risks to the procedure include, but are not limited to, bleeding, infection, nerve damage, additional tendon damage, temporary or permanent stiffness, and a need for additional operations in the future. Alternative forms of therapy should be discussed. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Proceed with prepping and positioning the patient. Place the patient in a supine and comfortable position to minimize movement during the procedure. Place the involved hand on a bedside procedure table ideally at heart level so that the patient's arm is resting comfortably. Temporarily diminish arterial blood flow with a digital tourniquet or a blood pressure cuff applied to the arm if vascular bleeding is active and obscures the site of repair. Apply padding under the tourniquet. **The maximum tourniquet time should be less than 2 hours.**[12] Any surgical procedure that takes this long should not be performed in the ED. A Penrose drain can make an effective tourniquet for the finger (**Figure 96-5**). Other methods of finger hemostasis are discussed in Chapters 129 and 137.

Anesthesia to the affected area can be accomplished with a digital or wrist block (Chapter 156).[14] The choice of procedure depends on the location and extent of the injury. Clean any dirt or debris from the hand. Apply povidone iodine or chlorhexidine solution and allow it to dry. Debride the wound and copiously irrigate it with 250 to 500 mL of sterile saline if no open joint is involved. The wound is now considered sterile. All techniques must be aseptic from this point forward. Apply sterile drapes or towels to delineate a sterile field. Place a sterile drape over the bedside procedure table. Lay out the required instruments and suture material on the bedside procedure table.

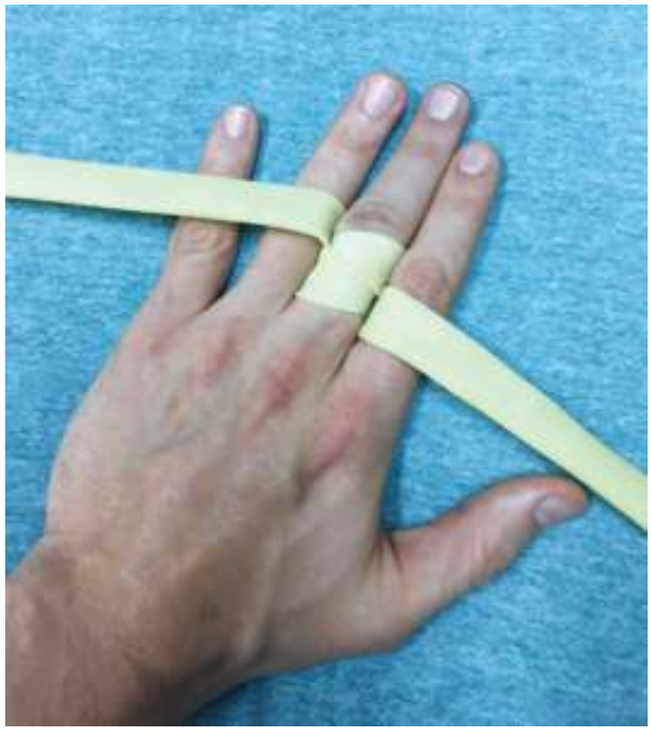

FIGURE 96-5. The Penrose drain method to obtain digit hemostasis. Wrap the Penrose drain from a distal to proximal direction. Continue wrapping until the end of the digit is reached. Begin unwrapping the drain from distal to proximal to expose the laceration. (Photo courtesy of Keegan Bradley, MD.)

TECHNIQUES

Many techniques are used to repair lacerated tendons. All the original techniques were described referring to flexor tendons. These techniques have been expanded to include the repair of extensor tendons. Four common suture techniques used for a "core" repair of the extensor tendon include the mattress stitch, the figure-of-eight stitch, the modified Bunnell stitch, and the modified Kessler stitch (**Figure 96-6**).[15,16] **No single stitch is optimal for any one zone of injury.** The type of stitch must be individualized based on the properties of the tendon at the site of injury. Evidence exists that the modified Kessler and Bunnell stitches produce the greatest strength for a core-type tendon repair (**Figures 96-6*C* and 96-6*D***).[7] These stitches may not be as well suited for extremely thin tendons (e.g., zones 4 or 6) as are figure-of-eight or mattress stitches (**Figures 96-6*A* and 96-6*B***). The Bunnell and Kessler techniques are more useful in round and thicker tendons (e.g., flexor tendons or selected extensor tendons).[7,17]

MODIFIED KESSLER STITCH

This "core" stitch is designed to place the direction of force perpendicular to the longitudinal axis of the tendon (**Figure 96-6*C***). If the force of the suture is placed in the tendon's longitudinal axis, there is a tendency for the suture to pull through and shred the tendon.

Identify the two ends of the lacerated extensor tendon. **Handle the cut ends with maximal care. Blindly or bluntly grabbing the ends traumatizes tendons and compromises the repair.** A retracted tendon can be held in place with a needle piercing it perpendicularly.

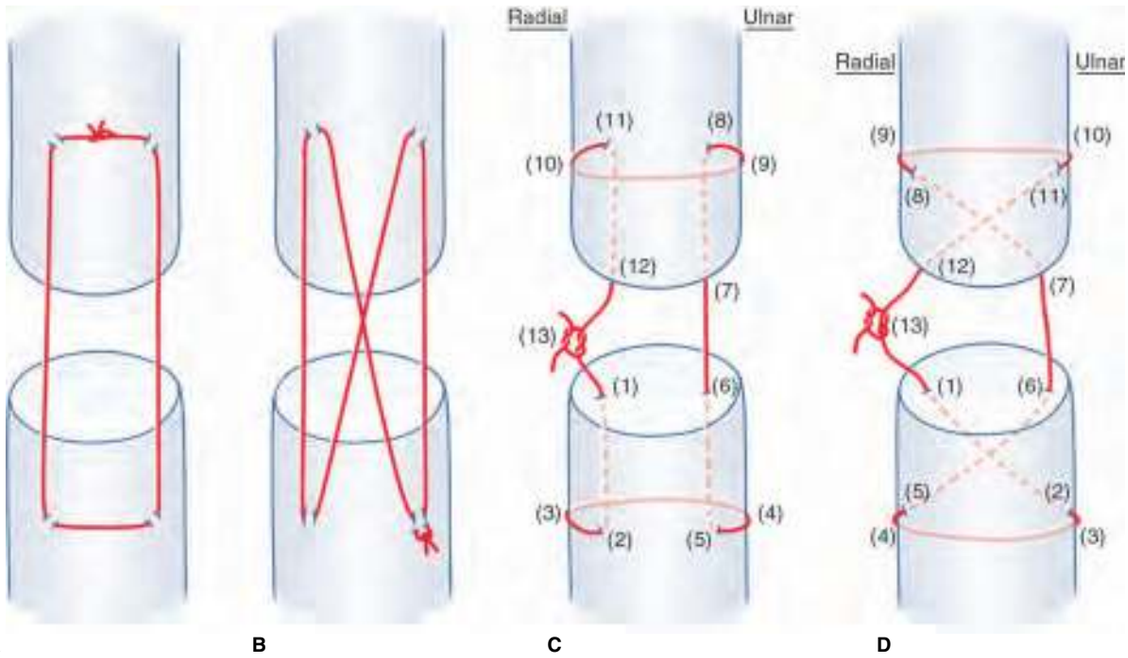

FIGURE 96-6. Suture techniques for extensor tendon repair. ***A.*** Mattress stitch. ***B.*** Figure-of-eight stitch. ***C.*** The modified Kessler stitch. The numbers represent the sequence of steps. ***D.*** The modified Bunnell stitch. The numbers represent the sequence of steps.

Judiciously debride the tendon ends if they are jagged or dirty. This can be performed with a fresh #11 blade scalpel cutting down against a wooden tongue depressor. **Do not remove too much length from the tendon.** Place the affected digit and wrist in maximum extension to facilitate approximation of the tendon ends.

Hold one end of the tendon gently with your fingers or a stitch. Introduce the needle into the cut end of the tendon (**Figure 96-6*C*-1**). The entrance point should be about one-third of the diameter of the tendon, beginning on either its ulnar or radial side. For simplicity, we begin on the radial side. Pass the suture approximately 1 cm through the length of the tendon and exit dorsally (**Figure 96-6*C*-2**). Wrap the suture around the tendon (**Figure 96-6*C*-3**). Reenter the radial side of the tendon perpendicularly and 1 to 2 mm closer to the tendon end (**Figure 96-6*C*-3**). Pull the suture straight through the tendon to exit on its ulnar side (**Figure 96-6*C*-4**). Wrap the suture around the tendon (**Figure 96-6*C*-5**). Enter the dorsal aspect of the ulnar half of the tendon (**Figure 96-6*C*-5**). This entrance stitch must line up with the first dorsal stitch (**Figure 96-6*C*-2**). Pass the needle through the length of the tendon to exit the end of the tendon (**Figure 96-6*C*-6**). The needle must exit on the ulnar one-third of the tendon.

Repeat the same stitch on the opposing piece of the tendon. Pass the needle into the ulnar one-third of the tendon (**Figure 96-6*C*-7**). Pass the suture approximately 1 cm through the length of the tendon and exit dorsally (**Figure 96-6*C*-8**). Wrap the suture around the tendon (**Figure 96-6*C*-9**). Reenter the ulnar side of the tendon perpendicularly and 1 to 2 mm closer to the tendon end (**Figure 96-6*C*-9**). Pull the suture straight through the tendon to exit its radial side (**Figure 96-6*C*-10**). Reenter the dorsal aspect of the radial half of the tendon (**Figure 96-6*C*-11**). The stitch must line up with the previous dorsal stitch (**Figure 96-6*C*-8**). Pass the needle through the length of the tendon to exit the end of the tendon (**Figure 96-6*C*-12**). The needle must exit on the radial one-third of the tendon.

The two free ends of the suture should be on the radial side of the tendon. Apply tension to the two free ends of the suture to gently approximate the ends of the lacerated tendon. **Do not apply so much force that the ends bunch up.** Secure the stitch with a knot that will remain buried between the tendon ends (**Figure 96-6*C*-13**).

MODIFIED BUNNELL STITCH

This stitch follows the same principle as the Kessler stitch. It incorporates a crossing of the suture in its pathway (**Figure 96-6*D***). Minimize the handling of the tendon in performing this stitch. **Instrumentation of the tissue is detrimental to its nutritional supply and can lead to adhesions.** One should ideally immobilize the tendon ends with one's fingers or a single suture.

Enter the tendon end on the radial half and at approximately one-third of the diameter of the tendon (**Figure 96-6*D*-1**). Pass the needle diagonally through the tendon and exit on the ulnar side (**Figure 96-6*D*-2**). Wrap the suture around the tendon (**Figure 96-6*D*-3**). Reenter the tendon on its dorsal half (**Figure 96-5*D*-3**). Pass the needle directly through the tendon to exit the dorsal surface of the radial aspect of the tendon (**Figure 96-6*D*-4**). Enter the radial side of the tendon (**Figure 96-6*D*-5**). Cross the suture diagonally through the tendon to exit its ulnar end (**Figure 96-6*D*-6**). The needle must exit through the ulnar one-third of the tendon end.

Repeat the same stitch on the opposing piece of the tendon. Pass the needle into the ulnar one-third of the tendon (**Figure 96-6*D*-7**). Pass the needle diagonally through the tendon and exit on the radial side (**Figure 96-6*D*-8**). Wrap the suture around the tendon (**Figure 96-6*D*-9**). Reenter the tendon on its dorsal half (**Figure 96-6*D*-9**). Pass the needle directly through the tendon to exit the dorsal surface of the ulnar aspect of the tendon (**Figure 96-6*D*-10**). Wrap the suture around the tendon (**Figure 96-6*D*-11**). Enter the ulnar side of the tendon (**Figure 96-6*D*-11**). Cross the suture diagonally through the tendon to exit the radial end of the tendon (**Figure 96-6*D*-12**). The needle must exit through the ulnar one-third of the tendon end.

The two free ends of the suture should be on the radial side of the tendon. Pull gently on the suture ends to approximate the ends of the lacerated tendon. **Do not apply so much force that**

the ends bunch up. Secure the stitch with a knot, which will remain buried within the tendon ends (**Figure 96-6*D*-13**).

AFTERCARE

Close the overlying skin with nylon suture, ideally everting the skin edges with a horizontal mattress suture. Apply topical antibiotic ointment. Apply 4×4 gauze squares to cover the wound. Apply an elastic bandage for protection. Splint the extremity to immobilize the repaired tendon and its associated muscle belly.

The patient may be discharged home with follow-up arranged within 24 to 48 hours with an Orthopedic or Hand Surgeon. Instruct the patient to elevate the extremity. They should return to the ED immediately if there is increased pain, numbness, or tingling in the digits; if the digits become cold or blue; or if a fever develops. Pain can be controlled with nonsteroidal anti-inflammatory drugs (NSAIDS) supplemented with an occasional narcotic analgesic. Many physicians prescribe prophylactic antibiotics. There is little evidence supporting this practice.[18]

Static immobilization may not be the optimal postoperative management for extensor tendon repairs. The affected tendon may benefit from early mobilization exercises or dynamic splinting depending on the zone of injury. Dynamic extension splinting may produce fewer complications and less postoperative adhesions.[19-21] Tendon injuries in zones 1 to 4 can be temporarily immobilized in extension until follow-up by an Orthopedic or Hand Surgeon determines definitive splinting management. Tendon injuries in zones 5 to 8 can be placed in a temporary static splint with the wrist in 30° of extension, the metacarpophalangeal joints in 15° of flexion, and the interphalangeal joints in full extension.

The aftercare of extensor tendon lacerations necessitates close follow-up by an Orthopedic or Hand Surgeon for the evaluation of tendon function, wound evaluation, and rehabilitation strategies.[22] **Proper aftercare and hand rehabilitation are crucial to ensure successful results of a tendon repair.**[22] The rehabilitation and recovery are different than those in children.[23]

COMPLICATIONS

Surgical repair of any open wound or tendon laceration can result in unforeseen infections. Careful instructions for wound care and follow-up evaluations are important for early detection and treatment of tissue infection. The use of sterile technique and copious irrigation before tendon repair will minimize infections.

Failure of the tendon repair can be due to several etiologies.[24] Disruption of the surgical repair, the formation of adhesions, and joint stiffness can produce an inadequate outcome.[25] It is rare to encounter these complications in the ED. Nonetheless, they are real complications that both the EP and patient must discuss before proceeding with the repair procedure.

SUMMARY

The repair of an extensor tendon laceration can and does occur in the ED. The literature and authors have found that the use of the Bunnell and Kessler stitches for repair of extensor tendon injuries produce good results. The mattress technique and figure-of-eight stitch may be more useful for repair of thinner extensor tendons. Effective communication with an Orthopedic or Hand Surgeon is imperative in optimizing patient outcomes. The wound must be irrigated, explored, closed, and appropriately splinted for delayed surgical repair by an Orthopedic or Hand Surgeon. Postoperative rehabilitation is crucial to ensure an optimal outcome. Splinting in the ED should only be a temporizing immobilization procedure until rehabilitation therapy occurs.

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97 Arthrocentesis

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INTRODUCTION

Arthrocentesis is the insertion of a needle into a joint cavity for the removal of synovial fluid and/or the injection of pharmaceutical agents into the joint cavity. Fluid can be aspirated from almost any joint. Arthrocentesis is used to diagnose and make treatment decisions regarding a joint. Obtaining synovial fluid is safe, simple,

relatively pain free, inexpensive, and extremely beneficial to the patient.

Arthrocentesis may be diagnostic or therapeutic.[1] Diagnostically, it is performed to identify the cause of an acute arthritis, to identify an intraarticular fracture, to identify the causes of an effusion, or to give a therapeutic trial of pharmaceuticals. It can also be therapeutic by relieving pain from elevated intraarticular pressure, to drain septic or crystal-laden fluid, or to inject pharmaceuticals. Ultrasound (US)-guided joint aspiration is a supplemental technique that improves several aspects of the arthrocentesis procedure.

Synovial fluid analysis will provide unique and valuable information about the affected joint.[2,3] It is the only method to definitively diagnose or rule out a septic arthritis.[4,5] The fluid should be analyzed for the presence of crystals. A white blood cell count and differential may help identify the causes of an effusion. A Gram's stain can be quickly performed to identify bacteria in the synovial fluid. Bedside gross analysis of the fluid's physical properties such as clarity, viscosity, and color has been shown to be a reliable clinical predictor of a potentially septic joint.[6] A culture of the synovial fluid should be performed to definitively identify any microbiologic pathogen in the joint.

There are several general principles that should be followed when performing an arthrocentesis (**Table 97-1**). The Emergency Physician (EP) must know the anatomic relationships around the joint. The needle should go around and not through any tendons. Avoid piercing the articular cartilage, which is avascular and may not heal. Do not bounce the needle off the bone, as this is extremely painful for the patient. Insert the needle through the extensor surface of the joint. The synovial cavity is closest to the skin over the extensor surface of the joint. The extensor surface has fewer tendons and ligaments than the flexor surface. Most of the blood vessels and nerves are located on the flexor surface of the joint. Using the extensor surface of the joint for the procedure will avoid potential injury to these structures. Place the joint in slight flexion to maximize the size of the joint cavity. Apply distal in-line traction to the small joints of the wrists, hands, and feet to enlarge the joint cavity. This allows the needle to more easily enter the small joint spaces. Compression of large joints will mobilize peripheral fluid. This is helpful when the volume of synovial fluid is small. Compression may be applied manually or with an elastic bandage.

It is critical to use sterile technique to prevent the infection of a nonseptic joint as well as to ensure the joint fluid is uncontaminated for microbiological cultures. Sterile drapes, sterile gloves, and face masks are not needed to perform the procedure.[7-9] It is necessary to wear gloves to prevent transmission of bloodborne diseases to the EP.[7,10] The skin should be cleansed and prepped in the usual manner. The needle may then be grasped, with clean gloves, at the hub and inserted. As long as the skin and needle are not handled, sterile gloves are not required. This is sometimes difficult to master if the EP has little practice with the technique of arthrocentesis. It may be better to err on the side of conservatism and use sterile gloves and drapes, particularly with inexperienced EPs and in training situations.

US has been used by EPs, Interventional Musculoskeletal Radiologists, and Rheumatologists to guide needles for joint injections and aspirations.[11-16] US guidance allows these procedures to be performed in a safer manner because the needle is less likely to injure nerves, tendons, and blood vessels. US also allows a comparison with the asymptomatic opposite-side joint. As bedside ultrasonography has become used more commonly in the ED, more EPs are appreciating and reporting on its benefits of guiding arthrocentesis.[17-21] Arthrocentesis is commonly performed on the shoulder, elbow, hip, knee, and ankle joints. It may also be performed on other joints. US guidance can be a useful adjunct in all of these procedures.

There are several possible approaches for the use of US-guided arthrocentesis in the ED. Knee arthrocentesis is commonly performed with a high success rate using the landmark-palpation technique. Many EPs feel comfortable performing this procedure without the routine use of US guidance. Wiler and colleagues compared US-guided versus standard landmark techniques for knee arthrocentesis and found that US guidance did not improve overall success.[21] Other EPs use US guidance in cases anticipated to be difficult, such as in patients with small effusions or those with prior joint surgery. Some EPs feel that US guidance improves the safety of arthrocentesis and thus choose to use it whenever an arthrocentesis is performed. This approach is especially appealing in light of the fact that US is noninvasive and does not add any risk to the procedure.

INDICATIONS

Arthrocentesis is indicated to evaluate the cause of an arthritis or a joint effusion (**Table 97-2**).[10,22-24] All patients presenting with an acute monoarthritis or an acute nontraumatic effusion should undergo arthrocentesis when the diagnosis is not clear based on the history and physical examination. Analysis of the synovial fluid is essential to help differentiate inflammatory from noninflammatory causes of joint disease. **Arthrocentesis is the only reliable method to confirm the presence of an infectious agent as the cause of an arthritis**. The presence of crystals within the synovial fluid can diagnose gout or pseudogout from other crystal-induced arthritides. **Always keep in mind that two or more types of arthritis can coexist in a patient or in a single joint.[10]**

Arthrocentesis may be therapeutic.[25] A large effusion, regardless of the cause, stretches the joint capsule, causing pain and limiting range of motion. Removal of the fluid will decrease pain and increase the joint's range of motion. An effusion caused by inflammation or sepsis contains numerous mediators of inflammation. Removal of this synovial fluid will help to relieve the patient's discomfort. The removal of purulent fluid will decrease the number of

TABLE 97-1 General Principles for Arthrocentesis

Know the anatomic relationships
Avoid piercing any tendons
Do not damage articular cartilage
Do not penetrate bone
Use sterile technique
Provide adequate analgesia
Place the joint in slight flexion
Apply distal traction to small joints
Apply compression to large joints
Enter the extensor surface
Use US guidance if available

TABLE 97-2 The Indications for Performing an Arthrocentesis

To evaluate a monoarticular arthritis
To diagnose and treat a traumatic arthritis
To diagnose an intraarticular fracture
To diagnose an intraarticular ligamentous disruption
To relieve pain
To diagnose inflammatory versus noninflammatory disorders
To identify the cause of an effusion
To rule out an infection
To identify a crystal-induced arthritis
To inject therapeutic agents

organisms in the joint cavity and, theoretically, may limit further joint destruction.[26]

Injection of therapeutic agents into nonseptic joints is commonly performed in the Emergency Department (ED). Local anesthetic solutions may be injected to relieve pain. A joint examination after trauma may be limited secondary to pain. Injection of local anesthetic solutions can relieve pain and allow an examination for ligamentous and joint instability. Corticosteroids are injected into joints to control inflammation and arthritis pain. Sometimes, corticosteroids and local anesthetic solutions are combined into one syringe and injected intraarticularly. The local anesthetic provides immediate pain relief and assures the EP of proper needle placement. However, there are downsides. **Local anesthetic agents should be used sparingly as they may have chondrotoxic effects.[27-29] As with anything we do, we must weigh the benefits versus the risks.**

Arthrocentesis can be used to diagnose and treat traumatic arthritides. The traumatic event is usually acute, obvious, and followed by joint pain and swelling. Occasionally, the trauma is minimal or remote and not recalled by the patient. A traumatic effusion can be grossly bloody and, if acute, may contain a large amount of red blood cells. **The bloody synovial fluid represents an intraarticular fracture or a disruption of an intraarticular structure.[30]** An intraarticular fracture may be suspected based on mechanism of injury and yet radiographic findings may be negative. The synovial fluid may then be evaluated for fat globules, which are released from the marrow cavity of the fractured bone, which confirm the presence of an intraarticular fracture.

The temporomandibular joint is a highly specialized joint that has a unique set of problems associated with it. Arthrocentesis can be an easy, minimally invasive, and efficient procedure used to solve some of these issues. Degenerative joint disease and joint lock are two examples of conditions that are amenable to therapeutic arthrocentesis, although much debate still exists concerning efficacy and therapeutic value of this procedure.

CONTRAINDICATIONS

There are no absolute contraindications to arthrocentesis. All contraindications are relative. The risks and benefits of the procedure should be evaluated and a decision made with the informed consent of the patient. **If a septic joint is suspected, it should be aspirated despite the presence of any relative contraindication. The benefit of the procedure outweighs any relative contraindication when compared to the morbidity of an undiagnosed septic arthritis.**

The presence of a suspected or known skin cellulitis, or other infection overlying the joint, is a relative contraindication. A dermatitis or skin lesion overlying the joint should also be avoided. The skin or subcutaneous tissue can harbor organisms that may contaminate the joint when the needle passes through the dermatitis or skin lesion. Often, an alternative site can be found to perform the arthrocentesis and avoid the above obstacles. If the needle is inserted into the joint through any potential or obvious source of infection, antibiotic treatment is required due to the theoretical risk of introducing an infection into the joint cavity.[22] In these cases, patients should be placed in observation for 23 hours of intravenous antibiotics with a spectrum that covers skin flora.

Infections after arthrocentesis, in previously sterile joints, have been reported in bacteremic patients.[22] It is not clear whether the source of the septic arthritis was from the arthrocentesis or bacteremia coincidentally seeding the joint. It is recommended to avoid arthrocentesis in any patients with bacteremia or sepsis except to rule out a septic arthritis.[31]

Patients may be coagulopathic due to pharmacologic therapy, factor deficiencies, liver dysfunction, or many other causes. When possible, the coagulopathy should be reversed prior to arthrocentesis. Unfortunately, this is not always possible or practical. An experienced EP can safely perform the procedure without reversing the coagulopathy. Use the smallest needle gauge possible (22 or 23 gauge) to aspirate the joint fluid. Avoid injury to the articular cartilage by identifying the anatomic landmarks prior to the procedure. Do not bounce the needle off any bony surfaces.

The procedure may be difficult in some patients. In the morbidly obese, it may be difficult to identify anatomic landmarks. The standard needle may be too short to enter the joint cavity. A spinal needle may be required to perform an arthrocentesis in an obese patient. Uncooperative patients require sedation and/or restraint prior to performing the procedure.

A prosthetic joint or an arthroscopic procedure increases the risk of a septic arthritis.[32-35] Arthrocentesis is technically more difficult secondary to scar formation and alteration of the normal anatomic relationships. **Joints that contain a prosthesis should be aspirated only to rule out a septic arthritis. Arthrocentesis for other reasons, including joint injection, should be referred to a consultant**.

Corticosteroids are instilled into joints for a variety of conditions. If the patient has no response to the injection within a few weeks, it may be repeated. If multiple injections cause no improvement, an alternative form of therapy should be explored.[36,37] Multiple injections into a joint increase the risk of complications.

EQUIPMENT

- Clean or sterile gloves
- Gauze squares
- Povidone iodine or chlorhexidine solution
- Sterile skin marking pen
- Local anesthetic (e.g., injectable, vapor coolant, or ice)
- 25 to 27 gauge needle
- 3 mL syringe
- Needles to aspirate fluid (18/20/22 gauge)
- Syringes to aspirate fluid (1/3/5/10/20/30/60 mL)
- Hemostat
- Specimen tubes
- Culturettes or culture tubes

OPTIONAL EQUIPMENT

- US machine
- US gel
- US transducers (5 to 10 MHz)
- Sterile US transducer covers

Arthrocentesis should be performed with a needle of sufficient bore to allow the aspiration of thick fluid, fluid with debris, or purulent fluid. An 18 to 20 gauge needle is recommended for large joints (e.g., shoulder, elbow, ankle, knee, and hip). A 22 to 23 gauge needle is recommended for all other joints. Using too small a needle makes the procedure technically more difficult and more painful for the patient.

A high-frequency (5 to 10 MHz) linear array transducer is preferred for arthrocentesis and should be used whenever possible.[11] If this type of transducer is not available or if more depth of field is required, a curvilinear array may be used. Cavalier and colleagues described the use of color Doppler and a sterile needle guide attached to the transducer when performing US-guided arthrocentesis in children.[38] Doppler was used to localize the blood vessels

so that they could be avoided by the needle. While Doppler may occasionally provide additional anatomic information, it is not used routinely when performing an arthrocentesis. The use of a needle guide depends on the personal preference of the sonographer.

PATIENT PREPARATION

A complete history and physical examination should be performed prior to the arthrocentesis. The affected joint should be thoroughly assessed. Inspect the skin overlying the joint for breaks, infection, old scars, prior incisions, superficial lesions, or any wounds. Palpate the joint to identify any warmth, tenderness, or effusion. Evaluate the joint for any crepitation, deformity, ligamentous instability, or limitations in motion.

As with any nonemergent procedure, consent should be obtained from the patient or their representative. Ideally, the consent should be documented in the medical record and signed by the patient. Some prefer to note on the patient's chart "indications, risks, and benefits were discussed with the patient" rather than having the patient sign a consent form.[10,39] The following is a sample consent for performing an arthrocentesis (which may be written on the medical record and signed by the patient):

Arthrocentesis involves inserting a small needle into your _____ joint. The skin is anesthetized prior to the procedure. Ordinarily, the procedure has no significant complications. Occasionally, a patient may experience bleeding into the joint, infection of the joint or skin, pain, bruising, nerve injury, or an allergic reaction to the medications administered. These complications are minimized by the use of sterile technique and proper techniques.

Position the patient based on the specific joint to be aspirated and the approach to be used. Expose the joint and surrounding areas. Identify the anatomic landmarks required for proper needle placement. The landmarks may be difficult to identify on a swollen and tender joint. Compare the "affected" joint to the "normal" joint on the opposite side of the body. Identify the joint and a landmark on the normal joint and transfer this to the affected joint. Clean any dirt and debris from the skin. Scrub the needle insertion site with povidone iodine or chlorhexidine solution and allow it to dry.

Apply anesthesia to the skin and subcutaneous tissue using 1% lidocaine, topical vapor spray, or ice.[40] The administration of some form of local anesthesia is recommended but not required.[10,41] The most common local anesthetic used is a short-acting injectable anesthetic solution of 1% lidocaine. **Do not inject the local anesthetic solution deeper than the subcutaneous tissues**. Deep injections may instill anesthetic solution into the joint cavity which may interfere with the synovial fluid analysis. There is disagreement regarding whether the additional needlestick to administer the anesthesia causes as much discomfort as aspiration without any anesthesia. This decision is specific to each patient and EP.

Alternative methods of anesthesia include ice and topical vapor coolants. A sterile drape may be placed over the prepped skin and a bag of ice water placed over the drape. Remove the ice water bag and drape after 5 minutes and perform the procedure. Ethinyl chloride topical vapor coolant may be used as an anesthetic. Spray the solution onto the area of skin in which the needle will be inserted. Apply the spray from 6 inches above the skin. Spray until the skin turns white and frosty. This usually takes 5 to 10 seconds. Immediately perform the procedure, as the anesthesia lasts only 30 to 60 seconds.

TECHNIQUES

THE BASIC TECHNIQUE

The general procedure preferred by one of the authors and the editor (E.F.R.) will be described. Some may prefer to insert the needle into the joint space and then attach the syringe. Specifics will be addressed with each individual joint.

Apply the needle to the syringe and break the resistance. This avoids any sudden and painful movements of the needle within the joint cavity. Stretch the skin over the site where the needle will be inserted. Penetrate the skin briskly with the needle and enter the joint cavity. Gently aspirate synovial fluid. If bone is encountered, slightly withdraw the needle and readvance it in a different direction. If no fluid is obtained, reevaluate the joint to determine if an effusion is present, if another site is more appropriate for the procedure, or if another physician may offer a different perspective. For diagnostic aspirations, it is not necessary to aspirate all of the fluid from the joint. Synovial fluid analysis can be performed on 1 to 5 mL of fluid.

If additional fluid is to be removed after the original syringe is filled, or if pharmaceuticals are to be injected into the joint, do not remove the needle from the joint cavity. Grasp the hub of the needle with a hemostat. Remove the syringe and attach the second syringe. Continue to aspirate fluid or inject the desired pharmaceutical. Remove the needle when the procedure has been completed. Apply a bandage to the skin. Transfer the synovial fluid into appropriately labeled tubes or containers. Document the procedure in the medical record. A sample procedure note is described below:

After informed consent, the skin overlying the _____ joint was cleaned and prepped with povidone iodine/chlorhexidine solution. The skin was anesthetized with (_____ mL of _____% lidocaine, ethyl chloride vapor coolant, ice for _____ minutes). Using sterile technique, a(n) _____ gauge needle was inserted on the (supero-/infero-, medial/lateral/inferior/superior) surface of the joint. It was directed (supero-/infero-, medially/laterally/inferiorly/superiorly). _____ mL of fluid was obtained. It was (thin, thick, yellow, clear, straw-colored, bloody, purulent, with debris, without debris). No complications were noted.

The joint was injected with _____ mL of _____% (name of local anesthetic) and/or _____ mL of _____% _____ (name of corticosteroid). No complications were noted.

THE BASIC ULTRASOUND TECHNIQUE

The acoustic windows used for US-guided arthrocentesis are the soft tissues overlying the joint. The transducer is most commonly oriented across a joint so that the bones on each side of the joint and the joint space between them are visualized.[42] Bone is highly reflective of US waves. The cortex is seen as a bright white echogenic line on the US image. Synovial fluid will be seen as an anechoic line or hypoechoic collection within the joint space. When visualized in-plane, the needle will appear as a narrow, linear hyperechoic structure with a posterior reverberation artifact (**Figure 97-1**). When visualized out-of-plane, the needle appears as a small hyperechoic round object.[12]

The general procedure and technique depend on whether the static or dynamic method is used.[42] For the static technique, manipulate the US transducer over the joint until the optimal spot for needle entry is found based on fluid accessibility and a needle path that avoids major blood vessels, nerves, and tendons. Mark the skin on the midpoint of each side of the US transducer. Rotate the transducer 90° and repeat the process. Connect the two sets of lines to form an "X" that marks the spot for needle entry (**Figure 97-2**). Set aside the US transducer, prepare the skin in a sterile manner, and proceed with the arthrocentesis using the standard landmark-based technique.

The dynamic or real-time method is slightly more demanding technically but offers the advantage of allowing visualization of the

FIGURE 97-1. In-plane view of a needle approaching a joint effusion.

needle and needle tip.[11] Prepare the patient's skin in a sterile manner. Place a sterile transducer cover over the US transducer and apply sterile US gel to the skin. Place the transducer over the joint in the appropriate orientation and manipulate it until the synovial fluid is visualized. Inject a local anesthetic agent into the skin and subcutaneous tissue along the projected course of the arthrocentesis needle. In many cases, it is possible to observe the infusion of the anesthetic agent on the US image.[11] Advance the needle through the skin and soft tissue and direct it toward the synovial fluid. The needle can be advanced either in-plane or out-of-plane with regard to the US beam. The in-plane orientation allows visualization of the entire needle along its path and is the technique used by most EPs. Visualization in the out-of-plane orientation allows only one cross-sectional area of the needle to be seen, so it can be difficult to identify the needle and its tip. Regardless of orientation, continue to advance the needle until its tip is visible within the synovial fluid. Aspirate the synovial fluid. It is often possible to watch the synovial fluid diminish in size as it is aspirated.

FIGURE 97-2. The "X" marks the spot for needle entry.

TEMPOROMANDIBULAR JOINT (TMJ) ARTHROCENTESIS

Landmarks: The landmarks for TMJ arthrocentesis are found by first drawing a line on the patient's face from the corner of the eye to the ipsilateral tragus (canthal-tragus line). Mark the point 10 mm from the tragus along the line and 0.5 mm below the line. This marks the posterior border of the superior compartment. Mark a second point 20 mm from the tragus along the line and 1 mm below the line. This marks the anterior border of the superior compartment.

Patient positioning: Place the patient sitting upright with their jaw held slightly open.

Needle insertion and direction: Use two 21 gauge needles to access the joint space compartments. Insert the first needle at the first mark and advance it into the joint space. Inject 2 to 3 mL of sterile saline to insufflate the joint space. Insert and advance the second needle at the second point and into the joint space. Correct placement is confirmed when the saline flows out of the needle.[43]

US-guided arthrocentesis: With the patient positioned as above, place the US transducer inferior and lateral to the TMJ. Verify the hypoechoic space of the TMJ. The remainder of the technique is described above.

Remarks: Arthrocentesis of this joint is a unique solution to a specialized TMJ disorder called closed lock, in which the patient's jaw is felt to be locked and will not open greater than 20 to 25 mm. It is theorized to be the result of small adhesions and nongliding of the intraarticular disk in the TMJ space. Arthrocentesis and subsequent lavage can relieve the symptoms of closed lock. This is rarely performed by the EP. It is usually performed by Dentists and Oral Surgeons. The technique is easier if a double-needle-single-cannula is used.[44]

Joint injection: Once the two needles are in place in the superior compartment, massive lavage with normal saline or lactated Ringer's solution can be achieved with the first needle as input and the second needle as output. Continuous fluid can be infused and removed using a 10 mL syringe or attaching intravenous (IV) tubing to each needle.[45]

STERNOCLAVICULAR JOINT ARTHROCENTESIS

Landmarks: The sternal end of the clavicle and the suprasternal notch are the landmarks for this joint (**Figure 97-3**). The joint can be palpated just medial to the sternal end of the clavicle and just lateral to the suprasternal notch.

Patient positioning: Place the patient supine on a stretcher with their arm hanging over the edge of the table (**Figure 97-3*A***). Abduct the arm 90°. Externally rotate the arm so that the palm faces upward. This position maximally opens the sternoclavicular joint to allow easy access.

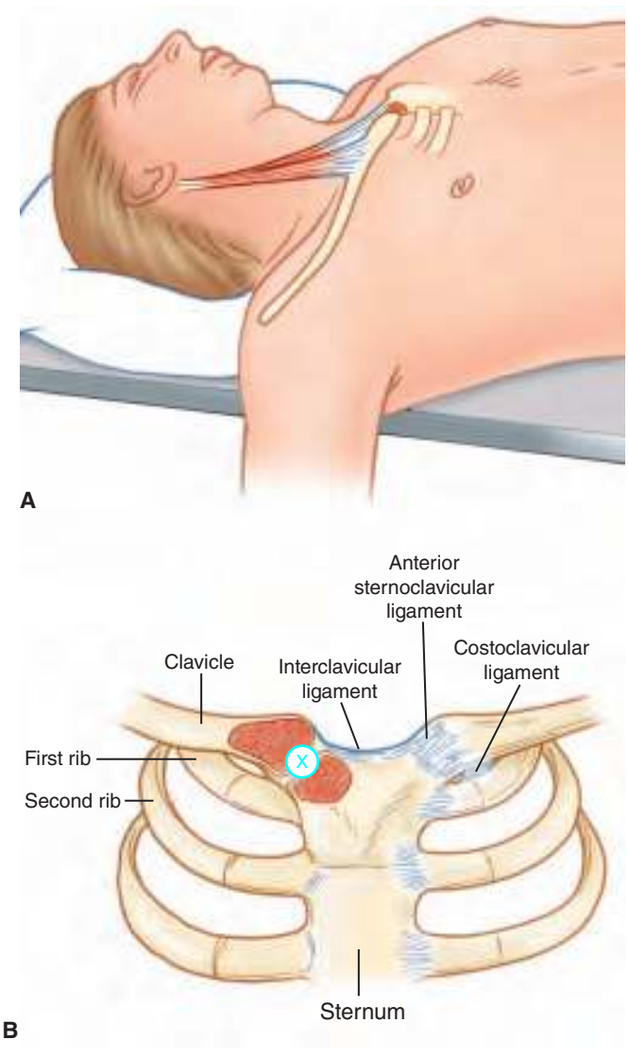

FIGURE 97-3. Sternoclavicular joint arthrocentesis. ***A.*** Patient positioning. ***B.*** Anatomy and needle insertion. The site of needle insertion is represented by an ⊗.

Needle insertion and direction: Insert a 23 gauge needle through the anterior joint surface and perpendicular to the skin (**Figure 97-3*B***). Advance the needle to a depth of 2 to 5 mm.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer superior to the joint. Verify the hypoechoic and small space of the joint. The remainder of the technique is described above.

Remarks: This joint is often involved with degenerative arthritis. Septic arthritis is commonly seen in IV drug abusers who inject drugs into the great vessels, also known as "pocket shooting." This can also be seen as a spontaneous infection.[46] This joint may only contain 0.25 to 0.50 mL of fluid.

Joint injection: A maximum volume of 1 mL may be instilled into this joint. A maximum dose of 10 mg of corticosteroids may be instilled into this joint.

ACROMIOCLAVICULAR JOINT ARTHROCENTESIS

Landmarks: The acromioclavicular (AC) joint is very superficial (**Figure 97-4**). Palpate the clavicle and move laterally until a prominence is felt. This is the AC joint. Have the patient move their arm to confirm the joint location.

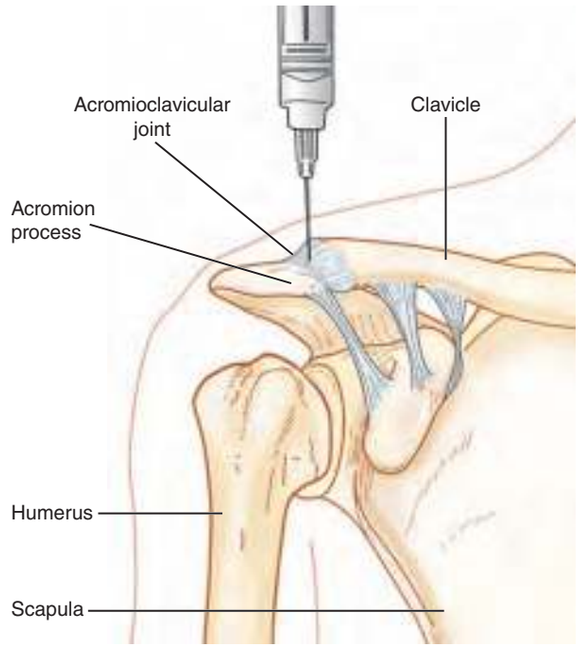

FIGURE 97-4. Acromioclavicular joint arthrocentesis.

Patient positioning: Place the patient sitting upright on a stretcher with the affected arm hanging by their side. A weight may be placed in the patient's hand to distract and open the joint space.

Needle insertion and direction: Insert a 22 gauge needle through the superior surface of the AC joint and perpendicular to the skin (**Figure 97-4**). Advance the needle to a depth of 3 to 5 mm.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer superior to the joint. Verify the hypoechoic and small space of the joint. The remainder of the technique is described above.

Remarks: The AC joint is very superficial. It has a dense, thick capsule anteriorly that is lacking on its superior surface. The joint may contain only 0.25 to 0.50 mL of fluid. US increases the accuracy of joint aspiration.[47]

Joint injection: A maximum volume of 1.5 mL may be instilled into this joint. A maximum dose of 10 mg of corticosteroids may be instilled into this joint.

GLENOHUMERAL JOINT (SHOULDER) ARTHROCENTESIS, ANTERIOR APPROACH

Landmarks: Palpate the coracoid process of the scapula (**Figure 97-5**). It will be found below the lateral third of the clavicle. Internally rotate and adduct the humerus. Palpate the groove between the coracoid process and the humeral head. This groove is the landmark for introduction of the needle.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Flex the elbow 90°. Internally rotate and adduct the arm. In the proper position, the forearm is resting against the patient's abdomen.

Needle insertion and direction: Insert an 18 gauge needle perpendicular to the skin and into the groove just lateral to the coracoid process (**Figure 97-5*B***). Aim the needle directly posterior. Advance the needle until a loss of resistance is felt as the joint cavity is entered.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer inferior and lateral to the coracoid process. Rotate the transducer so that the marker is cephalad and angled

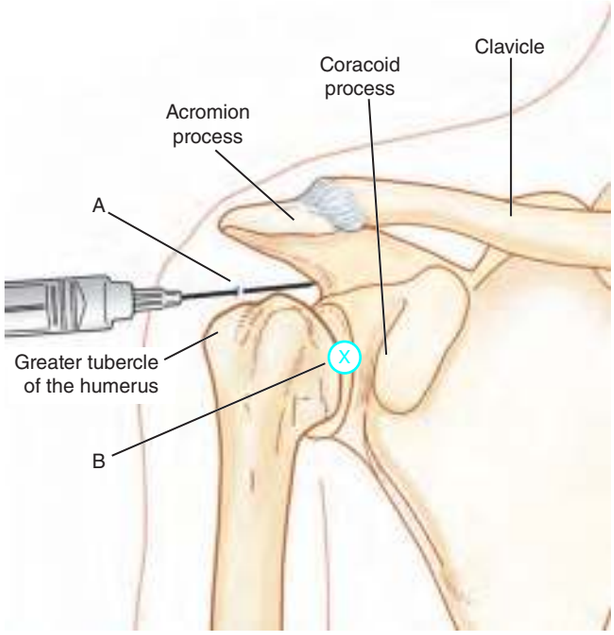

FIGURE 97-5. Shoulder joint arthrocentesis. ***A.*** Lateral approach. ***B.*** Anterior approach. The site of needle insertion is represented by an ⊗.

medially toward the patient's head. The curve of the humeral head will appear different from the flat portion of the glenoid. Between these hyperechoic surfaces lies the V-shaped hypoechoic joint space.

Remarks: This approach is the simplest yet most painful of the three approaches. The needle must penetrate the tendons of the coracobrachialis, subscapularis, biceps, and pectoralis major muscles in addition to the very tough anterior joint capsule. The major disadvantage of this approach is the possible, but rare, penetration of the brachial plexus or the axillary vessels with the needle. The patient can watch the large needle as it approaches the skin and this may increase their anxiety level. The posterior approach is preferred when using US guidance.[12] US increases the accuracy of joint aspiration.[48]

Joint injection: A maximum volume of 15 mL may be instilled into this joint. A maximum dose of 30 mg of corticosteroids may be instilled into this joint.

GLENOHUMERAL JOINT (SHOULDER) ARTHROCENTESIS, LATERAL APPROACH

Landmarks: Identify the acromion process of the scapula (**Figure 97-5**). A groove can be found just inferior to the lateral surface of the acromion and above the greater tubercle of the humerus.

Patient positioning: Place the patient sitting upright on a stretcher with the affected arm hanging by their side. A weight may be placed in the patient's hand to distract and open the joint cavity.

Needle insertion and direction: Insert an 18 gauge needle into the midpoint of the groove (**Figure 97-5*A***). Direct the needle medially and slightly posterior. Advance the needle to a depth of 2.5 to 3 cm.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer under the acromion coracoid process. Identify the joint space. The remainder of the technique is described above.

Remarks: Immediately below the deltoid muscle is the subacromial bursa. This bursa does not communicate with the shoulder joint. The needle must be inserted 2.5 to 3 cm to ensure the needle is in the shoulder joint and not in the subacromial bursa. The anterior or posterior approach to shoulder arthrocentesis avoids this potential problem.

Joint injection: A maximum volume of 15 mL may be instilled into this joint. A maximum dose of 30 mg of corticosteroids may be instilled into this joint.

GLENOHUMERAL JOINT (SHOULDER) ARTHROCENTESIS, POSTERIOR APPROACH

Landmarks: Identify the spine of the scapula (**Figure 97-6**). Follow the spine laterally until it turns anterior to become the acromion process. This posterior border of the acromion process is the landmark for this technique. Locate the coracoid process of the scapula, just inferior to the lateral third of the clavicle.

Patient positioning: Place the patient sitting upright on a stretcher. Place the palm of the hand of the affected shoulder on the anterior surface of the opposite shoulder. The arm and forearm should be held against the chest. This position maximally opens the posterior joint space.

Needle insertion and direction: Place the nondominant thumb on the posterior border of the acromion process. Place the nondominant index finger on the coracoid process. Insert an 18 gauge needle 1 to 2 cm below the thumb and parallel to the floor (**Figure 97-6**). Aim the needle toward the tip of the index finger, approximately 30° medially. Advance the needle to a depth of 2 to 3 cm.

US-guided arthrocentesis: Position the patient as described above. Place the US transducer across the posterior aspect of the shoulder in a transverse orientation (**Figure 97-7**). The marker may be directed medially or laterally. The echogenic cortex of the humeral head is visible along with the adjacent glenoid rim (**Figure 97-8**). The joint fluid is visible as an anechoic area between these two bony structures (**Figure 97-8**). Insert and advance the needle from the lateral edge of the US transducer, oriented along the long axis of the transducer, and into the joint fluid.

Remarks: This is felt by some physicians to be the preferred approach to shoulder arthrocentesis. The needle will pierce the deltoid and infraspinatus muscles and avoid the tendons of the rotator cuff. This approach avoids the anxiety associated with the patient observing the large needle and syringe used for the procedure during the anterior or lateral approach. The posterior joint capsule is much thinner and more easily penetrated than the anterior joint capsule. There are no significant neurovascular structures that may

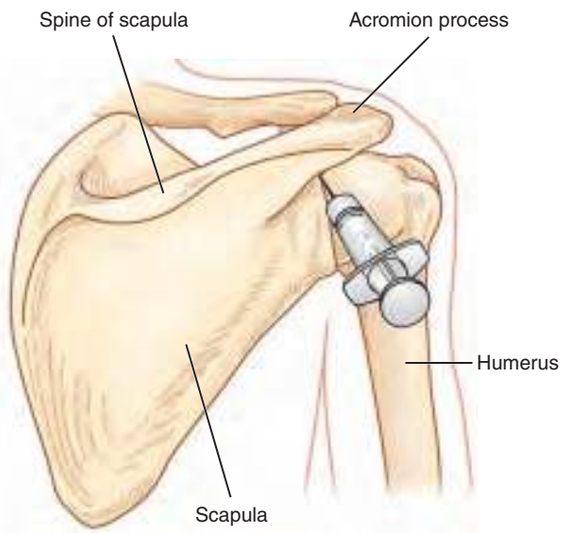

FIGURE 97-6. Posterior approach for shoulder joint arthrocentesis.

FIGURE 97-7. In the posterior approach to the shoulder, the US transducer is placed in a transverse orientation.

be injured from this approach. US increases the accuracy of joint aspiration.[48]

Joint injection: A maximum volume of 15 mL may be instilled into this joint. A maximum dose of 30 mg of corticosteroids may be instilled into this joint.

FIGURE 97-8. US image of the posterior shoulder. The hypoechoic joint fluid (*asterisk*) is located between the echogenic cortices of the humeral head (*arrow*) and the glenoid rim (*arrowhead*).

HUMERORADIOULNAR JOINT (ELBOW) ARTHROCENTESIS, LATERAL APPROACH

Landmarks: The lateral epicondyle of the humerus, the radial head, and the tip of the olecranon process of the ulna are the landmarks for this joint (**Figure 97-9*A***). Flex the elbow 45° and pronate the hand. Pronation of the hand stretches the radial collateral ligament, moves the radial nerve out of the needle's path, and widens the synovial cavity. Identify the depression between the three bony landmarks. The depression is located proximal to the radial head in the area where no bony structures are palpated.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Flex the elbow 45° and pronate the hand. This position widens the joint cavity and avoids any neurovascular structures.

Needle insertion and direction: Insert a 22 gauge needle perpendicular to the skin and into the depression just proximal to the radial head (**Figure 97-9*A***). Advance the needle to a depth of 0.75 to 2.0 cm.

US-guided arthrocentesis: With the patient positioned as above, place the US probe at the landmark. Verify the hypoechoic space of the joint. The remainder of the technique is described above.

Remarks: This is the preferred approach for elbow arthrocentesis. It avoids tendons and neurovascular structures, thereby reducing the risk of complications.[49]

Joint injection: A maximum volume of 5 mL may be instilled into this joint. A maximum dose of 20 mg of corticosteroids may be instilled into this joint.

HUMERORADIOULNAR JOINT (ELBOW) ARTHROCENTESIS, POSTERIOR APPROACH

Landmarks: Identify the top of the olecranon process of the ulna and the triceps muscle insertion into the olecranon process (**Figure 97-9*B***). Find the point just proximal to the top of the olecranon and just lateral to the triceps insertion. This point is the landmark for insertion of the needle.

Patient positioning: Place the patient sitting upright on a stretcher. Flex the elbow 90° with the hand supinated. The forearm and hand should be resting on a tabletop or on the patient's ipsilateral leg.

Needle insertion and direction: Approach the joint from the posterolateral surface with the needle parallel to the radial shaft. Insert a 22 gauge needle perpendicular to the skin at the landmark (**Figure 97-9*B***). Advance the needle to a depth of 1 cm.

US-guided arthrocentesis: Position the patient as described above. Place the US transducer along the posterior elbow in a longitudinal orientation with the marker toward the shoulder (**Figure 97-10**). This produces an image with the humerus located to the left, the olecranon located to the right, the olecranon fossa at the bottom, and the triceps tendon passing from side to side across the top of the screen (**Figure 97-11**). Keep the US transducer in the same orientation and slide it laterally to move off the triceps tendon. The joint effusion will be visualized in or adjacent to the olecranon fossa (**Figure 97-11**). Be sure to avoid the ulnar nerve, which passes over the medial epicondyle, by choosing a location as far lateral as possible.[50] Advance the needle over the superior edge of the US transducer and into the synovial fluid.

Remarks: Potential complications include needle penetration of the triceps tendon or the radial nerve. This approach is reserved for patients in whom the lateral approach is contraindicated. US is helpful to guide the procedure.[51]

Joint injection: A maximum volume of 5 mL may be instilled into this joint. A maximum dose of 20 mg of corticosteroids may be instilled into this joint.

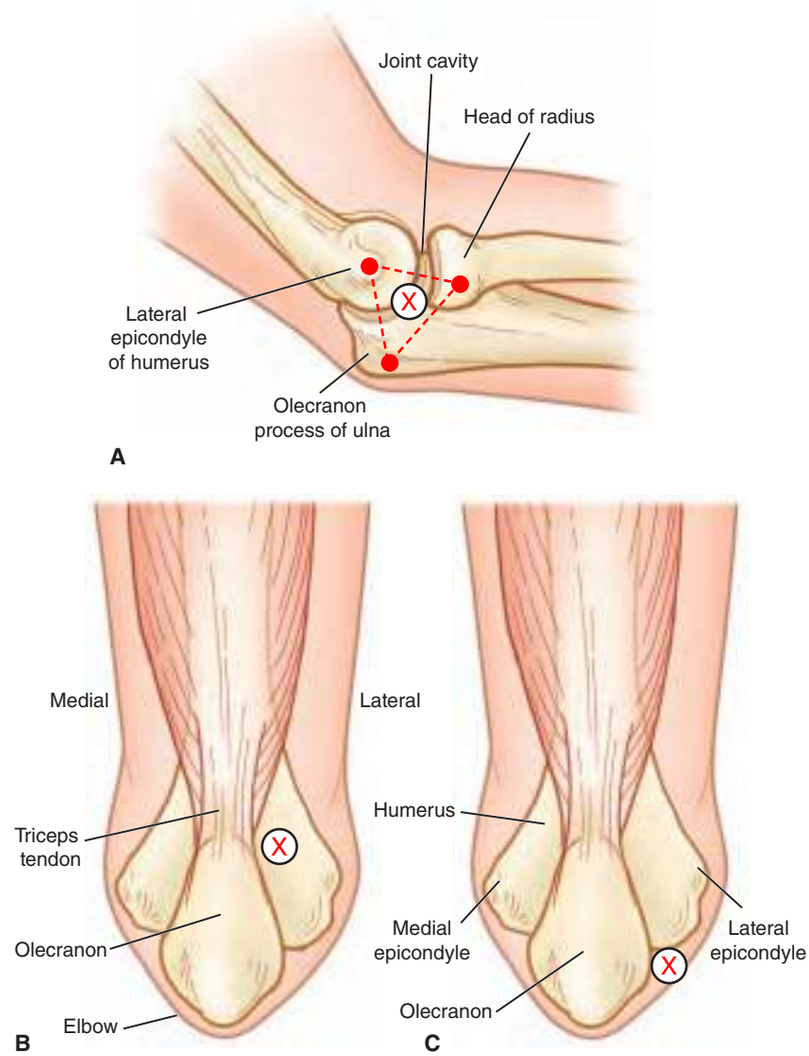

FIGURE 97-9. Elbow joint arthrocentesis. The site of needle insertion is represented by an ⊗. ***A.*** Lateral approach. ***B.*** Posterior approach. ***C.*** Posterolateral approach.

FIGURE 97-10. The US transducer is placed along the posterior elbow in a longitudinal orientation.

HUMERORADIOULNAR JOINT (ELBOW) ARTHROCENTESIS, POSTEROLATERAL APPROACH

Landmarks: Identify the lateral surface of the olecranon process of the ulna and the lateral epicondyle of the humerus (**Figure 97-9*C***). Find the indentation just lateral to the olecranon and just distal to the lateral epicondyle. This point is the landmark for the insertion of the needle.

Patient positioning: Place the patient sitting upright on a stretcher. Flex the elbow 90° with the hand supinated. The forearm and hand should be resting on a tabletop or on the patient's ipsilateral leg.

Needle insertion and direction: Approach the joint from the posterolateral surface with the needle parallel to the radial shaft. Insert a 22 gauge needle perpendicular to the skin at the landmark (**Figure 97-9*C***). Advance the needle to a depth of 1 cm.

US-guided arthrocentesis: Position the patient as described above. Place the US transducer along the posterior elbow in a longitudinal orientation, with the marker toward the shoulder (**Figure 97-10**). This produces an image with the humerus located to the left, the olecranon located to the right, the olecranon fossa at the bottom, and the triceps tendon passing from side to side across

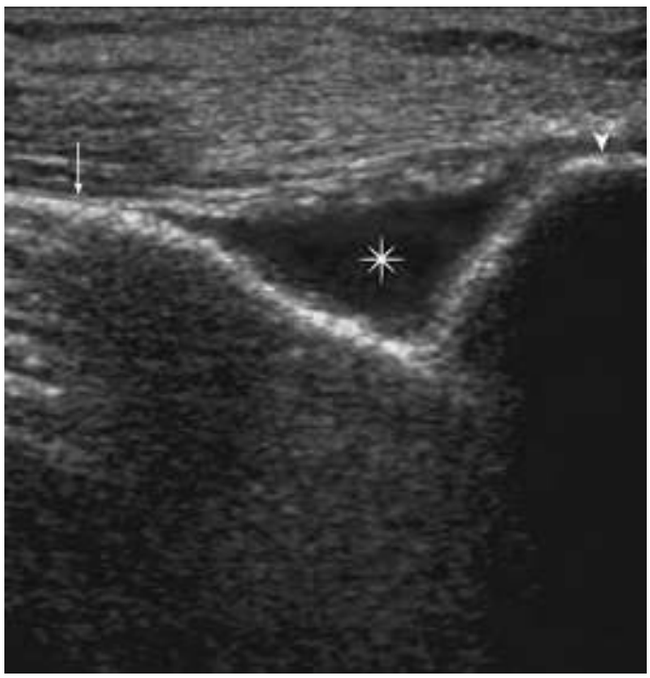

FIGURE 97-11. US image of the longitudinal view of an elbow joint. The hypoechoic joint fluid (*asterisk*) is located between the echogenic cortices of the humerus (*arrow*) and the olecranon (*arrowhead*).

the top of the screen (**Figure 97-11**). Keep the US transducer in the same orientation and slide it laterally to move off the triceps tendon. The joint effusion will be visualized in or adjacent to the olecranon fossa (**Figure 97-11**). Be sure to avoid the ulnar nerve, which passes over the medial epicondyle, by choosing a location as far lateral as possible.[50] Advance the needle over the superior edge of the US transducer and into the synovial fluid.

Remarks: This approach is an alternative when the lateral approach is contraindicated. US is helpful to guide the procedure.[51]

Joint injection: A maximum volume of 5 mL may be instilled into this joint. A maximum dose of 20 mg of corticosteroids may be instilled into this joint.

RADIOCARPAL JOINT (WRIST) ARTHROCENTESIS

Landmarks: Identify the lunate bone (**Figure 97-12**). Palpate the middle (i.e., third) metacarpal and follow it proximally until a rounded bean-like elevation is felt (**Figure 97-12*A***). This is the lunate bone. Palpate the indentations proximal and distal to the lunate bone. The indentation proximal to the lunate bone is the landmark for needle insertion.

Alternatively, identify Lister's tubercle and the extensor pollicis longus (EPL) tendon (**Figure 97-12*B***). Lister's tubercle is a bony prominence in the center of the dorsal aspect of the distal radius. The EPL tendon can be found lateral (i.e., radial) to Lister's tubercle. If the EPL tendon is difficult to find, extend the hand and thumb against resistance and it will become prominent. The lunate bone is the bony prominence distal to Lister's tubercle. The indentation proximal to the lunate bone is the landmark for needle insertion.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Pronate the hand with the wrist in slight (20° to 50°) flexion and ulnar deviation (**Figure 97-12*A***). The palm of the physician's nondominant hand should be holding the patient's palm. Reidentify the lunate bone with the nondominant thumb.

Needle insertion and direction: Insert a 22 gauge needle perpendicular to the skin in the indentation proximal to the lunate bone (**Figure 97-12*A***). Advance the needle to a depth of 0.75 to 1.25 cm.

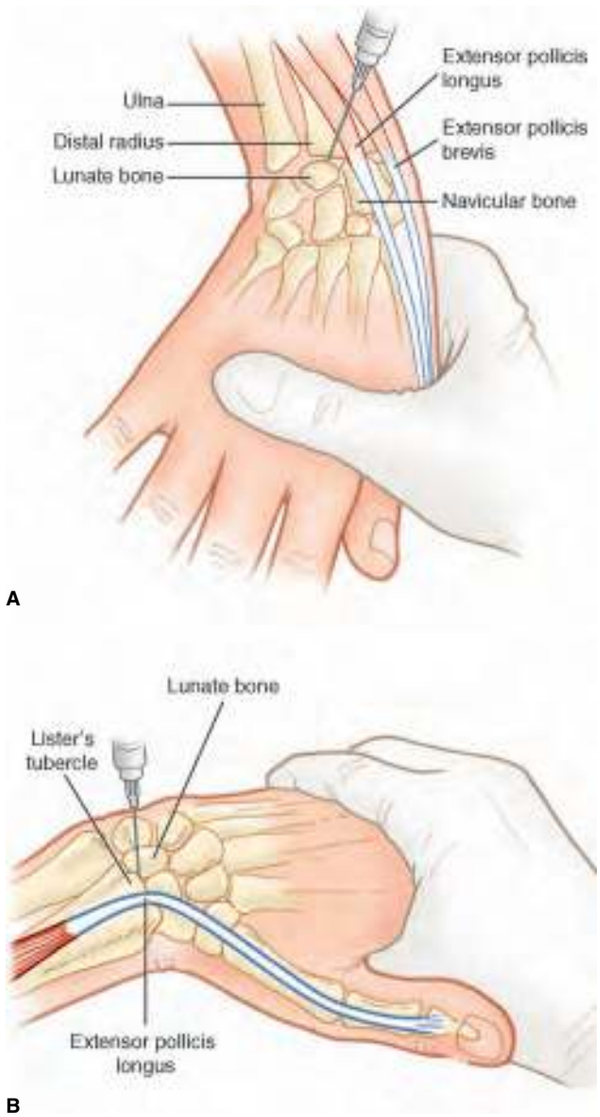

FIGURE 97-12. Radiocarpal joint arthrocentesis.

Alternatively, insert the needle in the indentation just distal to Lister's tubercle and ulnar to the EPL tendon (**Figure 97-12*B***).

US-guided arthrocentesis: With the patient positioned as above, place the US transducer at the indentation proximal to the lunate bone. Verify the hypoechoic and small space of the joint. The remainder of the technique is described above.

Remarks: The preferred, and easiest, method is to identify the lunate bone and then insert the needle in the indentation just proximal to it.

Joint injection: A maximum volume of 2 mL may be instilled into this joint. A maximum dose of 20 mg of corticosteroids may be instilled into this joint.

INTERCARPAL JOINT ARTHROCENTESIS

Landmarks: Identify the lunate bone (**Figure 97-12**). Palpate the middle (i.e., third) metacarpal and follow it proximally until a rounded bean-like elevation is felt (**Figure 97-12A**). This is the lunate bone. Palpate an indentation distal to the lunate bone and proximal to the base of the third metacarpal. This indentation is the landmark for insertion of the needle.

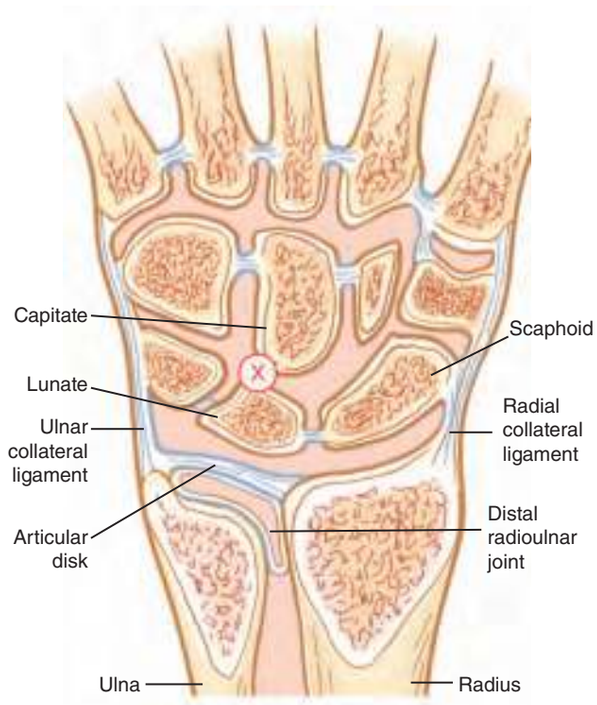

FIGURE 97-13. Intercarpal joint arthrocentesis. Coronal section through the wrist and hand demonstrating that the joints between the carpal bones are all interconnected. The site of needle insertion is represented by an ⊗.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Pronate the hand with the wrist in slight (20° to 50°) flexion and ulnar deviation (**Figure 97-12*A***). The palm of the physician's nondominant hand should be holding the patient's palm. Reidentify the landmarks with the nondominant thumb.

Needle insertion and direction: Insert a 22 gauge needle perpendicular to the skin in the indentation distal to the lunate bone. Advance the needle to a depth of 0.5 to 1.0 cm.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer at the indentation distal to the lunate bone. Verify the hypoechoic and small space of the joint. The remainder of the technique is described above.

Remarks: The joints between the carpal bones are all connected to each other (**Figure 97-13**). Fluid aspirated from one of these joints is representative of all the joints. Likewise, injection of one joint allows the pharmaceutical to be distributed to all the joints.

Joint injection: A maximum volume of 1.5 mL may be instilled into this joint. A maximum dose of 15 mg of corticosteroids may be instilled into this joint.

CARPOMETACARPAL JOINT OF THE THUMB ARTHROCENTESIS

Landmarks: The base of the first metacarpal and the abductor pollicis longus (APL) tendon are the landmarks for this joint (**Figure 97-14**). Identify the radial aspect of the base of the first metacarpal. Have the patient rotate the affected thumb to help identify the joint. Identify the APL tendon by extending the patient's thumb against resistance.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Flex the thumb into the palm with the tip of the thumb touching the fifth metacarpal head. Clench the remaining fingers into a fist (**Figure 97-14*A***).

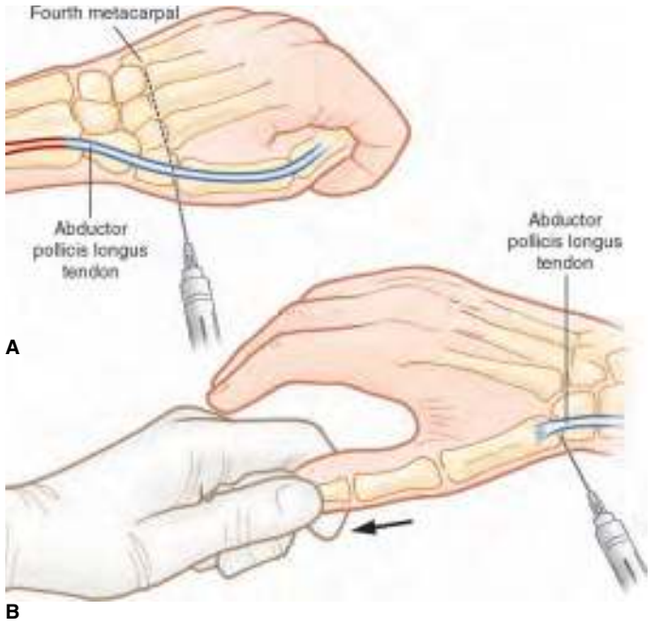

FIGURE 97-14. Carpometacarpal joint of the thumb arthrocentesis. ***A.*** Recommended approach. The *dotted line* represents the proper direction for needle insertion, toward the base of the fourth metacarpal. ***B.*** An alternative technique. The *arrow* represents the application of distal traction.

Needle insertion and direction: Insert a 22 gauge needle into the joint space at the radial aspect of the base of the first metacarpal and just lateral (i.e., radial) to the APL tendon (**Figure 97-14*A***). Direct the tip of the needle toward the base of the fourth metacarpal. Advance the needle to a depth of 0.5 to 1.0 cm.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer superior to the carpometacarpal joint. Rotate the transducer so that the marker is cephalad and angled medially toward the patient's head. Between the hyperechoic surfaces of the bones lies the small and narrow hypoechoic joint space. The remainder of the technique is described above.

Remarks: If difficulty is encountered when inserting the needle, try an alternative thumb position (**Figure 97-14*B***). Flex the first carpometacarpal joint 45° and apply distally directed traction to the distal thumb. This in-line traction will enlarge the joint cavity and allow easier access.

Joint injection: A maximum volume of 1.5 mL may be instilled into this joint. A maximum dose of 10 mg of corticosteroids may be instilled into this joint.

METACARPOPHALANGEAL JOINT OF THE FINGER ARTHROCENTESIS

Landmarks: Identify the metacarpophalangeal (MCP) joint and the extensor digitorum tendon (**Figure 97-15*A***). The MCP joint can be located just proximal to the prominence at the base of the proximal phalanx of the finger. Identify the extensor tendon by having the patient extend the finger against resistance.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Pronate the hand and abduct the fingers. Grasp the finger and apply distally directed traction.

Needle insertion and direction: Insert a 22 gauge needle into the dorsal joint space just medial or lateral to the extensor tendon (**Figure 97-15*B***). Direct the tip of the needle toward the center of the joint. Advance the needle to a depth of 0.3 to 0.5 cm.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer superior to the MCP joint. Rotate the transducer

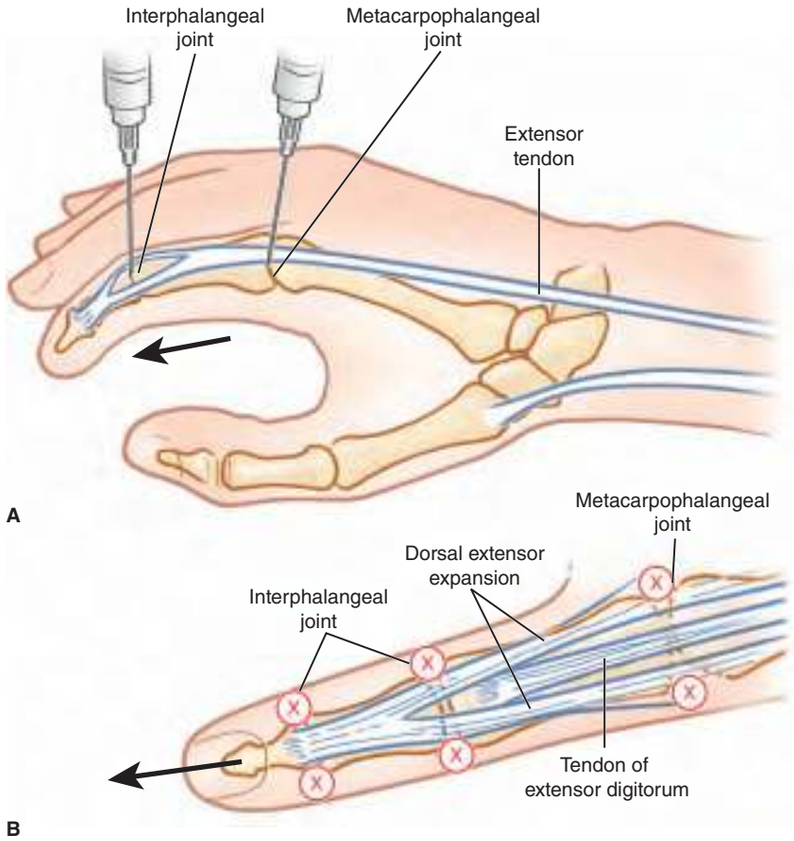

FIGURE 97-15. Arthrocentesis of the metacarpophalangeal and interphalangeal joints. ***A.*** Proper needle placement. ***B.*** Distal traction will cause dimpling of the skin in the areas noted by the ⊗. These areas of skin dimpling are the landmarks for needle insertion. The *arrow* represents the application of distal traction.

so that the marker is cephalad and angled medially towards the patient's head. Between the hyperechoic surfaces of the bones lies the small and narrow hypoechoic joint space. The remainder of the technique is described above.

Remarks: The application of distal traction often causes a depression to appear on both sides of the extensor tendon (**Figure 97-15*B***). These depressions can be used as landmarks for the site of needle insertion into the joint cavity.

Joint injection: A maximum volume of 1 mL may be instilled into this joint. A maximum dose of 5 mg of corticosteroids may be instilled into this joint.

INTERPHALANGEAL JOINT OF THE FINGER ARTHROCENTESIS

Landmarks: Identify the interphalangeal (IP) joint and the extensor tendon of the fingers (**Figure 97-15*A***). The IP joint can be located just proximal to the prominence at the base of the middle or distal phalanx of the finger. Identify the extensor tendon by having the patient extend the finger against resistance.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Pronate the hand and abduct the fingers. Grasp the finger and apply distally applied traction.

Needle insertion and direction: Insert a 22 gauge needle into the dorsal joint space just medial or lateral to the extensor tendon (**Figure 97-15*B***). Direct the tip of the needle toward the center of the joint. Advance the needle to a depth of 0.3 to 0.5 cm.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer superior to the IP joint. Rotate the transducer so that the marker is cephalad and angled medially towards the patient's head. Between the hyperechoic surfaces of the bones lies the small and narrow hypoechoic joint space. The remainder of the technique is described above.

Remarks: The application of distal traction often causes a depression to appear on both sides of the extensor tendon (**Figure 97-15*B***). These depressions can be used as landmarks for the site of needle insertion into the joint cavity. The joints are small and normally contain almost no synovial fluid. When inflamed or infected, the joint cavity may contain up to 2 mL of synovial fluid.

Joint injection: A maximum volume of 1 mL may be instilled into this joint. A maximum dose of 5 mg of corticosteroids may be instilled into this joint.

HIP JOINT ARTHROCENTESIS, ANTERIOR APPROACH

Landmarks: Identify the anterior superior iliac spine (ASIS) and the femoral pulse (**Figure 97-16**). The landmark for insertion of the needle is 2 to 3 cm below the ASIS and 2 to 3 cm lateral to the femoral artery pulse.

Patient positioning: Place the patient supine on a stretcher with the affected leg internally rotated and the knee slightly flexed.

Needle insertion and direction: Insert a 3.5 inch, 18 gauge needle at the landmark (**Figure 97-16**). Direct the tip of the needle posteromedially. Insert the needle at a 60° angle to the skin of the thigh. Advance the needle until bone is encountered. Slightly withdraw the needle and begin aspirating the synovial fluid.

US-guided arthrocentesis: Position the patient as described above.[20] Place the US transducer over the anterior hip and aligned with the long axis of the femoral neck, with the marker in a superior-medial direction and aimed toward the umbilicus (**Figure 97-17**).[11] The echogenic cortex of the femoral head is visible to the left of the image and the femoral neck to the right. The concave transition

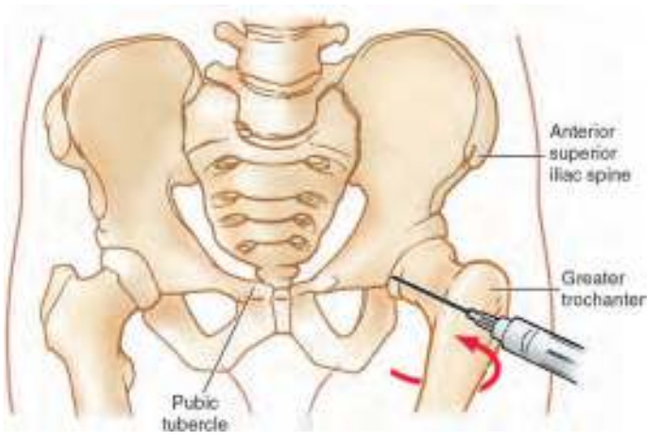

FIGURE 97-16. Anterior approach for hip joint arthrocentesis. The *curved arrow* represents internal rotation of the femur.

between these two areas is the anterior synovial recess.[11,20] Synovial fluid will be seen in this recess and tends to displace the joint capsule anteriorly (**Figure 97-18**).[11] Insert the needle from the inferolateral edge of the US transducer and advance it into the synovial fluid.

Remarks: This approach is technically easier and recommended by many authors.[52] A long spinal needle is usually required for this procedure. Use caution as the needle may injure the articular cartilage. This procedure should be performed by a consultant (e.g., an Orthopedist, a Rheumatologist, or a Radiologist) under fluoroscopic guidance if US is not available in the ED.

Joint injection: A maximum volume of 10 mL may be instilled into this joint. A maximum dose of 40 mg of corticosteroids may be instilled into this joint.

FIGURE 97-17. The US transducer is placed anteriorly over the hip joint and aligned with the long axis of the femoral neck.

FIGURE 97-18. US image of a hip effusion. The hypoechoic effusion (*asterisk*) is located in the anterior synovial recess between the echogenic cortices of the femoral neck (*arrowhead*) and the femoral head (*arrow*).

HIP JOINT ARTHROCENTESIS, LATERAL APPROACH

Landmarks: The greater trochanter is the landmark for this technique (**Figure 97-19**). Place the patient supine with the affected leg internally rotated. Palpate the greater trochanter.

Patient positioning: Place the patient supine on a stretcher with the affected leg internally rotated (**Figure 97-19*A***). Identify the greater trochanter and hold it between the thumb and index finger. A second patient position may be used for the alternative approach (**Figure 97-19*B***). Place the patient lying on the unaffected side. Flex the unaffected, lower leg 90°. Fully extend the upper, affected, leg with a pillow supporting the ankle.

Needle insertion and direction: Insert a 3.5 inch, 18 gauge needle just superior to the superior margin of the greater trochanter (**Figure 97-19*A***). Advance the needle horizontally, parallel to the stretcher, until it contacts the femoral neck. Withdraw the needle 2 to 4 mm and redirect it slightly cephalad. Advance the needle while applying negative pressure to the syringe until synovial fluid enters the syringe. Stop advancing the needle and continue to aspirate the synovial fluid. The alternative technique requires the insertion of the needle perpendicular to the skin and 1 cm proximal to the greater trochanter (**Figure 97-19*B***). Advance the needle until the femoral neck is contacted. Withdraw the needle 2 mm and begin aspirating synovial fluid.

Remarks: In the morbidly obese, the greater trochanter may not be palpable. This approach is technically more difficult than the anterior approach. The advantage of this approach is that the articular cartilage will not be in the needle's path and so avoids injury. This procedure should be performed by a consultant (e.g., an Orthopedist, a Rheumatologist, or a Radiologist) under fluoroscopic guidance.

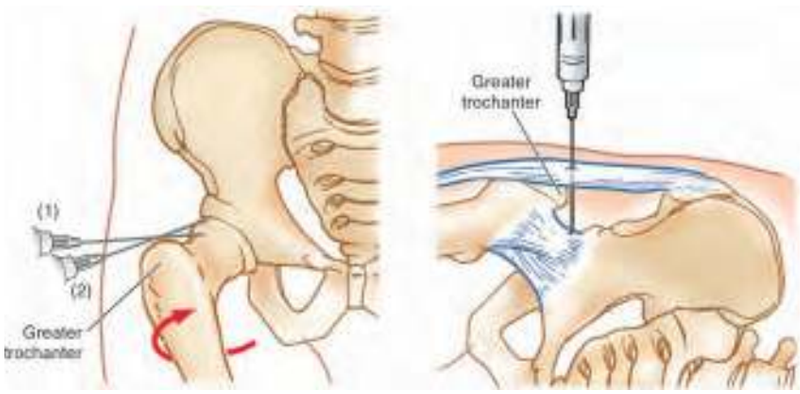

FIGURE 97-19. Lateral approach for hip joint arthrocentesis. ***A.*** The patient is supine with the leg internally rotated (*curved arrow*). A needle is inserted above the greater trochanter and advanced until the femoral neck is encountered (*1*). The needle has been withdrawn slightly, redirected cephalad, then readvanced into the joint cavity (*2*). ***B.*** An alternative approach.

Joint injection: A maximum volume of 10 mL may be instilled into this joint. A maximum dose of 40 mg of corticosteroids may be instilled into this joint.

PATELLOFEMOROTIBIAL JOINT (KNEE) ARTHROCENTESIS, SUPRAPATELLAR APPROACH

Landmarks: Identify the midpoint of the superolateral or superomedial border of the patella (**Figure 97-20*A***). Either of these landmarks may be used as the site for needle insertion.

Patient positioning: Place the patient supine on a stretcher with the affected knee fully extended.

Needle insertion and direction: Insert an 18 gauge needle through the midpoint of the superolateral or superomedial border of the patella (**Figure 97-20*A***). Direct the tip of the needle toward the intercondylar notch of the femur. Advance the needle to a depth of 1.5 to 3.0 cm.

US-guided arthrocentesis: Position the patient as described above. A small roll or towel can be used behind the knee to provide slight flexion. Place the US transducer in a longitudinal orientation superior to the patella, with the marker directed toward the patient's head. The cortex of the distal femur is visible as a bright echogenic line to the right of the image and the synovial fluid is visible deep to the quadriceps tendon (**Figure 97-21**). Keep the US transducer oriented longitudinally and slowly slide it medially or laterally to move off the quadriceps tendon. Insert the needle from the superior edge of the US transducer in a longitudinal orientation and direct it toward the synovial fluid. Advance the needle into the synovial fluid.

Remarks: The needle enters the suprapatellar bursa and avoids any potential damage to the articular cartilage. The bursa is a direct continuation of the synovial cavity.[11] There are no neurovascular structures of significance to injure with this approach. If the knee effusion is minimal, synovial fluid might not be aspirated from this approach. Approximately 10% to 15% of the population has a plica completely separating the suprapatellar bursa from the knee joint. If this variation exists in the patient, the bursa fluid will not

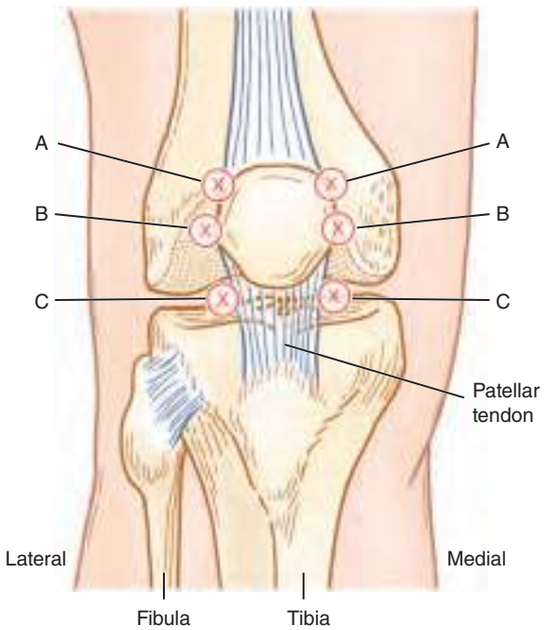

FIGURE 97-20. Knee joint arthrocentesis. The site of needle insertion is represented by an ⊗. ***A.*** Medial and lateral suprapatellar approach. ***B.*** Medial and lateral parapatellar approach. ***C.*** Medial and lateral infrapatellar approach.

FIGURE 97-21. US image of a knee effusion. The hypoechoic joint fluid (*asterisk*) is located above the echogenic cortex of the distal femur (*arrow*).

represent synovial fluid. The use of US has been shown to improve the patient-reported clinical outcomes.[53]

Joint injection: A maximum volume of 10 mL may be instilled into this joint. A maximum dose of 40 mg of corticosteroids may be instilled into this joint.

PATELLOFEMOROTIBIAL JOINT (KNEE) ARTHROCENTESIS, PARAPATELLAR APPROACH

Landmarks: Identify the midpoint of the lateral or medial border of the patella (**Figure 97-20*B***). Either of these landmarks may be used as the site for needle insertion.

Patient positioning: Place the patient supine on a stretcher with the affected knee fully extended.

Needle insertion and direction: Insert an 18 gauge needle just below the midpoint of the lateral or medial border of the patella (**Figure 97-20*B***). Direct the needle perpendicular to the long axis of the leg and aimed toward the intercondylar notch of the femur. Advance the needle to a depth of 1 to 2 cm.

US-guided arthrocentesis: Place the US transducer oriented longitudinally, and either lateral or medial to the patella, with the marker directed toward the patient's head. Identify the joint fluid. Slowly rotate the US transducer to a horizontal orientation to identify the area with the largest anechoic fluid collection.[11] Insert and advance the needle along the superior edge of the US transducer and into the joint fluid.

Remarks: The easiest site for arthrocentesis is the medial parapatellar region. There are no disadvantages to using the medial parapatellar site.

Joint injection: A maximum volume of 10 mL may be instilled into this joint. A maximum dose of 40 mg of corticosteroids may be instilled into this joint.

PATELLOFEMOROTIBIAL JOINT (KNEE) ARTHROCENTESIS, INFRAPATELLAR APPROACH

Landmarks: Identify the inferior border of the patella and the patellar tendon (**Figure 97-20*C***). The tendon is a thick band that passes from the inferior border of the patella to the tibial tuberosity.

Patient positioning: Place the patient sitting upright on a stretcher with the affected knee bent 90° over the edge of the bed and the leg hanging freely and unsupported.

Needle insertion and direction: Insert an 18 gauge needle 0.5 cm below the inferior border of the patella at the level of the joint line and just medial or lateral to the patellar tendon (**Figure 97-20*C***). Direct the needle perpendicular to the long axis of the leg and aimed toward the intercondylar notch of the femur. Advance the needle to a depth of 1.5 to 2.0 cm.

Remarks: The weight of the leg helps to open the joint cavity. The risk of injury to the articular cartilage is minimal. This approach was popular in the past but is not often used today. **Do not pierce the patellar tendon with the needle.**

Joint injection: A maximum volume of 10 mL may be instilled into this joint. A maximum dose of 40 mg of corticosteroids may be instilled into this joint.

TIBIOTALAR JOINT (ANKLE) ARTHROCENTESIS, ANTEROLATERAL APPROACH

Landmarks: Identify the joint cavity, the lateral malleolus, and the extensor digitorum longus (EDL) tendons (**Figure 97-22**). The joint cavity is located below the distal edge of the fibula and between the bases of the malleoli. Extend the toes against resistance to identify the EDL tendons. Palpate the base of the lateral malleolus.

Patient positioning: Place the patient sitting upright or supine on a stretcher. The patient can also be placed sitting upright on a stretcher with affected knee bent 90° over the edge of the bed and the foot hanging freely and unsupported. Plantar flex the ankle.

Needle insertion and direction: Insert a 22 gauge needle perpendicular to the fibular shaft at the level of the base of the lateral malleolus; midway between the malleolus and the lateral border of the EDL tendon (**Figure 97-22*A***). Advance the needle to a depth of 0.5 to 1.0 cm.

US-guided arthrocentesis: Position the patient as described above. Place the US transducer over the anterior tibiotalar joint in a longitudinal orientation, with the marker of the US transducer toward the patient's head (**Figure 97-23**).[11,17] Manipulate the US transducer with a slight rotation until the echogenic cortex of the tibia and talus bones are both visible (**Figure 97-24**). The tibia is visible to the left of the image and the talus to the right. During the rotation of the US transducer, note the location of the dorsalis pedis artery and the extensor tendons so that they may be avoided when inserting the needle.[11] The joint fluid is visible as a hypoechoic area in the V-shaped recess between the tibia and talus (**Figure 97-24**). Insert the needle at the inferior end of the transducer and advance it into the joint fluid.

Remarks: This approach avoids potential injury to the dorsalis pedis vessels and the deep peroneal nerve.

Joint injection: A maximum volume of 3 mL may be instilled into this joint. A maximum dose of 20 mg of corticosteroids may be instilled into this joint.

TIBIOTALAR JOINT (ANKLE) ARTHROCENTESIS, ANTEROMEDIAL APPROACH

Landmarks: Identify the joint cavity, the medial malleolus, and the tendons of the tibialis anterior (TA) and the extensor hallucis longus (EHL) muscles (**Figure 97-22**). The joint cavity is located below the distal edge of the tibia and between the bases of the malleoli. Extend the great toe against resistance to identify the EHL tendon. Plantar flex the ankle against resistance to identify the TA tendon. Palpate the base of the medial malleolus.

Patient positioning: Place the patient sitting upright or supine on a stretcher. The patient can also be placed sitting upright on a stretcher with the affected knee bent 90° over the edge of the bed and the foot hanging freely and unsupported. Plantar flex the ankle to use the EHL tendon as the landmark for the procedure. Alternatively, plantar flex the ankle and invert the subtalar joint to use the TA tendon as the landmark for the procedure.

Needle insertion and direction: Insert a 22 gauge needle perpendicular to the tibial shaft at the level of the base of the malleolus and medial to the EHL tendon (**Figure 97-22*B***). Alternatively, insert the needle medial to the TA tendon (**Figure 97-22*C***).

US-guided arthrocentesis: The procedure is exactly as described above for the anterolateral approach.

Remarks: If using the EHL tendon as the landmark, use caution to avoid the dorsalis pedis vessels and the deep peroneal nerve that usually lie immediately lateral to the EHL tendon.

Joint injection: A maximum volume of 3 mL may be instilled into this joint. A maximum dose of 20 mg of corticosteroids may be instilled into this joint.

SUBTALAR JOINT ARTHROCENTESIS

Landmarks: Identify the tip of the medial malleolus. Palpate the sustentaculum tali of the calcaneus (**Figure 97-25**). It is approximately 1.5 to 2.0 cm below the tip of the medial malleolus.

Patient positioning: Place the patient supine on a stretcher with the foot held at a right angle to the leg. Externally rotate the hip until the medial malleolus is pointing upward.

Needle insertion and direction: Insert a 22 gauge needle immediately above and slightly posterior to the sustentaculum tali (**Figure 97-25**). Advance the needle to a depth of 1.5 to 2.0 cm.

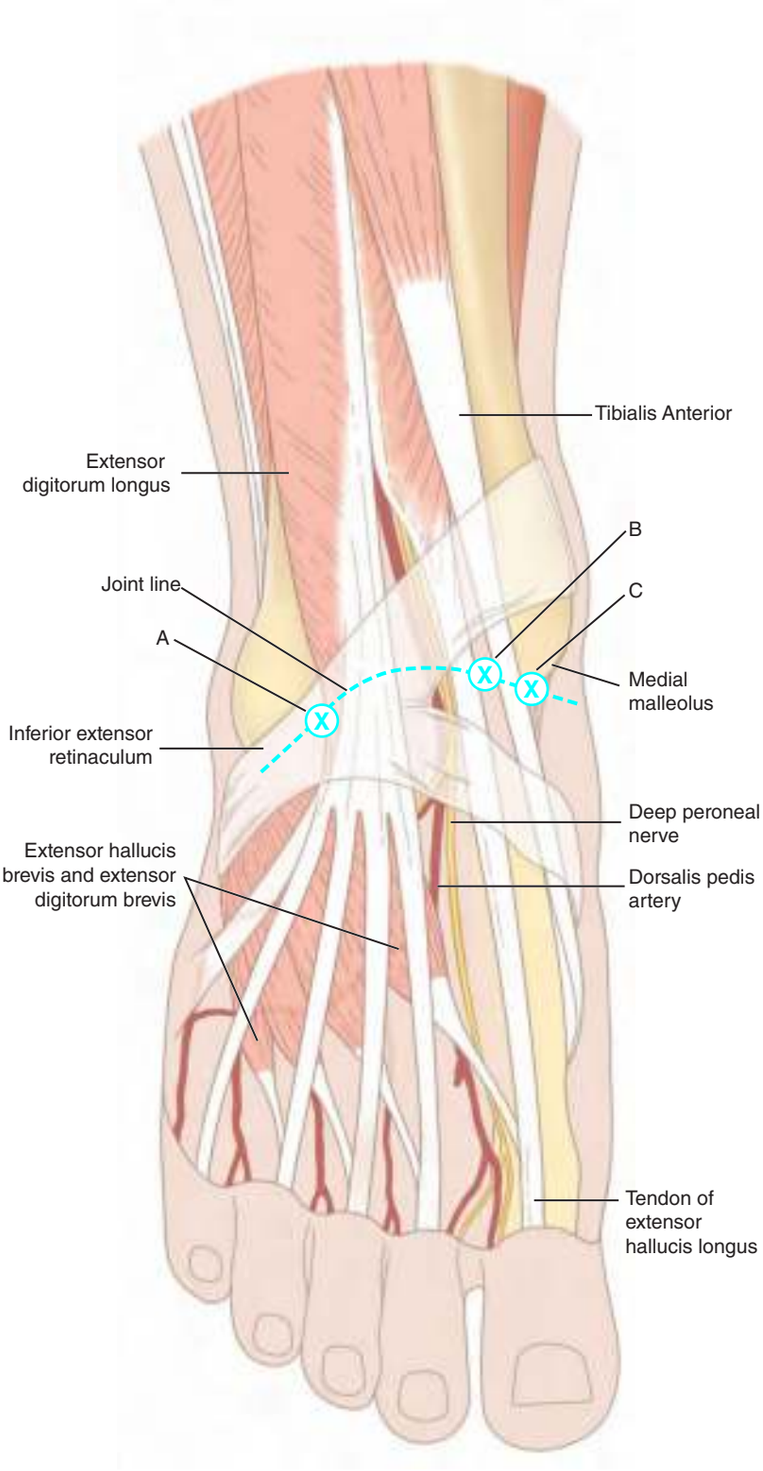

FIGURE 97-22. Arthrocentesis of the ankle joint. The site of needle insertion is represented by an ⊗. ***A.*** Anterolateral approach. ***B.*** Anteromedial approach. ***C.*** An alternative technique for the anteromedial approach.

Remarks: This is a difficult procedure because the joint space is very small. Fluoroscopy may be required to gain entry into the joint cavity. This procedure is seldom performed in the ED. US is not very helpful due to the small joint space and irregular body and bony surfaces.

Joint injection: A maximum volume of 1.5 mL may be instilled into this joint. A maximum dose of 20 mg of corticosteroids may be instilled into this joint.

INTERTARSAL JOINT ARTHROCENTESIS

There are no specific landmarks for the many intertarsal joints. Radiographs are required to identify the location of a specific joint. Plain radiographs are often not helpful and fluoroscopic guidance is usually required. For these reasons, arthrocentesis of these joints is not routinely performed in the ED. Some authors recommend

FIGURE 97-23. The US transducer is placed over the anterior tibiotalar joint in a longitudinal orientation.

probing the general area of the joint with a needle until it enters the joint cavity. This is extremely painful for the patient and risks injury to the articular cartilage. Therefore, this technique cannot be recommended.

METATARSOPHALANGEAL JOINT ARTHROCENTESIS

Landmarks: Identify the metatarsophalangeal (MTP) joint and the extensor tendon. The MTP joint can be located just proximal to the prominence at the base of the proximal phalanx of the toe. Identify the extensor tendon by having the patient extend the toe against resistance.

FIGURE 97-24. US image of the longitudinal view of the anterior tibiotalar joint. The hypoechoic joint fluid (*asterisk*) is located in the recess between the echogenic cortices of the tibia (*arrow*) and talus (*arrowhead*).

FIGURE 97-25. Subtalar joint arthrocentesis.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Plantar flex the foot. Grasp and plantar flex the toe 15° to 20° and apply distal traction (**Figure 97-26*A***).

Needle insertion and direction: Insert a 22 gauge needle into the dorsal joint space and just medial or lateral to the extensor tendon (**Figure 97-26*B***). Direct the tip of the needle toward the center of the MTP joint. Advance the needle to a depth of 0.3 to 0.5 cm.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer superior to the MTP joint. Rotate the transducer so that the marker is cephalad and angled medially towards the patient's head. Between the hyperechoic surfaces of the bones lies the small and narrow hypoechoic joint space. The remainder of the technique is described above.

Remarks: The application of distal traction often causes a depression to appear on both sides of the extensor tendon. These depressions can be used as landmarks for the site of needle insertion into the joint cavity.

Joint injection: A maximum volume of 1.5 mL may be instilled into this joint. A maximum dose of 10 mg of corticosteroids may be instilled into this joint.

INTERPHALANGEAL JOINT OF THE TOE ARTHROCENTESIS

Landmarks: Identify the interphalangeal (IP) joint and the extensor tendon. The IP joint can be located just proximal to the prominence at the base of the middle or distal phalanx of the toe. Identify the extensor tendon by having the patient extend their toes against resistance.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Plantar flex the foot. Grasp and plantar flex the toe 15° to 20° and apply distal traction (**Figure 97-26*A***).

Needle insertion and direction: Insert a 23 gauge needle into the dorsal joint space and just medial or lateral to the extensor tendon (**Figure 97-26*B***). The needle should be aimed toward the center of the joint. Advance the needle to a depth of 0.3 to 0.5 cm.

US-guided arthrocentesis: With the patient positioned as above, place the US transducer superior to the IP joint. Rotate the transducer so that the marker is cephalad and angled medially toward the patient's head. Between the hyperechoic surfaces of the bones lies the small and narrow hypoechoic joint space. The remainder of the technique is described above.

Remarks: The application of distal traction often causes a depression to appear on both sides of the extensor tendon. These depressions can be used as landmarks for the site of needle insertion into

FIGURE 97-26. Arthrocentesis of the metatarsophalangeal and interphalangeal joints of the foot. ***A.*** The ankle is plantar flexed. The affected toe is flexed 15° to 20° at the metatarsophalangeal joint and distal traction is applied (*arrow*). ***B.*** The needle is inserted just inferomedial or inferolateral to the extensor tendon at the level of the metatarsophalangeal joint (*1*) or the interphalangeal joint (*2*).

the joint cavity. These joints are small and normally contain almost no synovial fluid. When inflamed or infected, the joint cavity may contain up to 1.5 mL of synovial fluid.

Joint injection: A maximum volume of 1 mL may be instilled into this joint. A maximum dose of 5 mg of corticosteroids may be instilled into this joint.

JOINT INJECTION TECHNIQUE

A joint may be injected with corticosteroids and/or local anesthetic solution to relieve pain and inflammation. Identify the anatomic landmarks, prepare the patient, and insert the needle as if performing an arthrocentesis. Inject the pharmaceutical(s), remove the needle, and apply a bandage.

Occasionally, synovial fluid may be required for analysis prior to the injection of pharmaceuticals. A prospective randomized study in rheumatoid arthritis patients found that aspirating all the synovial fluid prior to therapeutic steroid injection reduced the risk of relapse, led to better outcomes, and increased symptom relief.[54] Identify the anatomic landmarks, prepare the patient, and insert the needle as if performing an arthrocentesis. Aspirate the synovial fluid into the syringe. With the needle still in the joint, grasp the hub of the needle with a hemostat. Remove the original syringe containing the aspirated synovial fluid. Attach a second syringe containing the pharmaceutical(s) to be injected into the synovial cavity. Re-aspirate to confirm that the needle tip remained within the synovial cavity. Inject the pharmaceutical(s). While injecting, no resistance should be felt. If resistance is encountered, the needle may have dislodged from the joint cavity. Remove the needle, reinsert it into the synovial cavity, aspirate to confirm proper positioning, and then inject the pharmaceutical(s). After injection, remove the needle and apply a bandage.

ULTRASOUND AND PEDIATRIC HIP EFFUSIONS

Evaluation of a suspected pediatric hip joint effusion requires consideration of septic arthritis versus a toxic synovitis.[20,55,56] Toxic synovitis is the most common cause of hip pain in children and occurs most frequently between the ages of 3 and 6 years. It is a nonpyogenic, inflammatory condition of the joint synovium with an unknown etiology. It occurs most commonly in the hip joint and occasionally in the knee joint.

In contrast, septic arthritis is an acute pyogenic inflammation that occurs most commonly in children below the age of 4 years, with a peak incidence between 6 and 24 months of age. Useful studies include serum white blood cell (WBC) count, erythrocyte sedimentation rate (ESR), blood cultures, plain films, computed tomography (CT), magnetic resonance imaging (MRI), and US. The most critical diagnostic information comes from aspiration and analysis of joint fluid.

US has been shown to be as accurate as MRI in detecting effusions. It is most easily performed using an anterior approach with the US transducer oriented in the sagittal plane and parallel to the femoral neck. An effusion is visible as a convex, rather than concave, joint capsule more than 5 mm thick. Transudates are more often hypoechoic, whereas exudates and hemorrhage are usually more hyperechoic.[57]

Many imaging techniques such as plain film, scintigraphy, MRI, and CT are useful in the differentiation of these two clinical entities. MRI and CT provide the highest quality information. However, these studies usually require sedation or even general anesthesia in young children due to the long examination times and the requirement for no motion. US-guided hip joint arthrocentesis is a useful bedside imaging technique to help solve this problem.

US can detect the presence of a joint effusion. It cannot reliably differentiate between toxic synovitis and septic arthritis without joint fluid aspiration and analysis. US should not be used as the primary means of ruling out a suspected septic arthritis.[56-58]

AFTERCARE

No special care or precautions are required after performing an arthrocentesis. Pain can be relieved with the use of ice, elevation, and nonsteroidal anti-inflammatory drugs. Joint injection, on the other hand, often requires some precautions. Some recommend limiting joint activity for 4 to 8 hours if an anesthetic solution is injected into a joint. An anesthetized joint, especially weight-bearing joints, may be susceptible to further injury when the joint has decreased sensation. Injection of corticosteroids into a joint cavity often requires a period of immobilization. This discussion is beyond the scope of this chapter. The readers should refer to a rheumatology textbook, orthopedic textbook, or the medical literature for this information.

COMPLICATIONS

Complications can occur with joint aspiration and/or injection (**Table 97-3**). Most of the complications are minor. Significant complications are rare but do occur.

TABLE 97-3 Complications Associated with the Aspiration and/or Injection of Joints

Allergic reactions
Bleeding
Cartilage injury
Dry tap
Infection
Joint instability
Needle-associated complications
Corticosteroid-induced complications
Vasovagal reactions

ALLERGIC REACTIONS

Allergic reactions can occur from hypersensitivity to the local anesthetic solution. Symptoms can range from mild itching and urticaria to circulatory collapse and death. Severe allergic reactions are extremely rare but may occur. Taking a thorough history can prevent many allergic reactions. The preservative in the local anesthetic solution is often the cause of the allergic reaction. Local anesthetic solutions containing no preservatives are an alternative. Some authors believe local anesthesia is not required, as the pain of anesthetic injection is equal to performing the procedure without injectable anesthesia.[10,41] If one is concerned about a potential allergic reaction associated with local anesthetic solutions, topical ice or vapor coolant is an acceptable alternative to nothing. A solution of 1% or 2% diphenhydramine can also be used as an injectable local anesthetic agent for the skin and soft tissues. Do not inject diphenhydramine into the joint cavity as this can result in significant inflammatory reactions and possible crystal deposition.

CARTILAGE INJURY

The articular cartilage can be damaged from improper needle insertion or needle movement within the joint cavity. The actual incidence of cartilage injury is unknown. The cartilage is avascular and injuries do not heal. Damaged cartilage can lead to focal degenerative changes and be a nidus for future infection. Injury to the articular cartilage can be prevented with a few simple steps. Select a site for the procedure and a needle path that avoids the articular cartilage. Aspirate as you slowly enter the joint cavity and stop inserting the needle when synovial fluid enters the syringe. There is no advantage to plunging the needle into the middle of the joint cavity. Avoid needle movement once the joint cavity has been entered. Finally, do not try to completely aspirate all the fluid within the joint cavity.

DRY TAP

A needle inserted into a joint does not always guarantee fluid will be aspirated.[59] A dry tap can occur due to improper needle placement, a small or absent effusion, mechanical obstruction, or chronic inflammation.

A dry tap can result from improper needle placement. If the needle is not within the joint cavity, no fluid will be aspirated. Slightly withdraw the needle and reinsert it at a different angle. Alternatively, remove the needle, re-identify the anatomic landmarks, and then reinsert the needle.

One of the most common reasons for a dry tap is the lack of an effusion or a small one. It may be difficult on physical examination to determine if an effusion is present. This is especially true if the patient is obese or if a large amount of subcutaneous edema is present. Try using the non–syringe-bearing hand to "milk" fluid toward the needle while aspirating. Alternatively, if a small effusion is suspected, inject a small amount of sterile saline into the joint cavity. Allow the saline to remain for 30 to 60 seconds, and then aspirate the fluid.

Synovial fluid may be loculated or inaccessible by the chosen site of needle entry if the joint has been previously injured. Choose an alternate approach or site to enter the joint cavity. This may facilitate synovial fluid aspiration.

Mechanical obstruction can result in a dry tap. Gently move the needle tip to determine if it can move freely. If it doesn't, it may be caught or embedded in cartilage, periosteum, or synovium. Withdraw the needle 1 to 2 mm and re-aspirate. The needle may also be within the intraarticular fat pad. Slightly withdraw the needle and re-aspirate. A plica may be obstructing the lumen of the needle. Rotate the needle and re-aspirate. If no fluid is aspirated, slightly advance the needle and re-aspirate. If still no fluid is obtained, attempt an alternate approach or reconsider whether an effusion is present and whether the procedure is actually required.[59]

The synovial fluid itself may be the cause of a dry tap. The presence of purulent fluid or fluid with debris may clog the needle. Remove the needle and repeat the procedure with a larger gauge needle. If the needle clogs during the aspiration, try reinjecting a small amount of the synovial fluid to dislodge the obstruction and then re-aspirate. A larger gauge needle may be required to complete the procedure.

Aspiration of synovial fluid from a chronically inflamed joint may be problematic. The synovium may undergo fatty replacement known as lipoma arborescens. Long-standing inflammatory fluid may be resorbed, leaving a thick gelatinous material that is difficult to aspirate. In these cases, arthrocentesis should be referred to the experienced clinician or a consultant.

HEMORRHAGE

Significant bleeding is extremely rare. External hemorrhage can be controlled with direct pressure over the puncture site. Hemarthroses are usually small, self-limited, and only require observation. If a significant hemarthrosis or external hemorrhage occurs, treatment may be required to reverse the anticoagulant or replace clotting factors. The readers should refer to another source for management of these complications, as a detailed discussion is beyond the scope of this chapter.

Arthrocentesis can be safely performed in patients who are anticoagulated or have a bleeding disorder. The most experienced EP should perform the procedure to limit any potential complications and bleeding. Also reconsider if the procedure must be done immediately in the ED. The only true indication to perform an arthrocentesis in a coagulopathic patient in the ED is to rule out a septic joint.

INFECTION

Infection of a sterile joint can occur when the needle penetrates unclean skin, cellulitic skin, infected subcutaneous tissues, or skin lesions. If proper aseptic technique is used, the risk of infecting a sterile joint occurs in less than 1:10,000 arthrocenteses.[10,60] The risk of infection is negligible when the skin is properly cleansed, aseptic technique is used, and the skin is not punctured through an obvious infection or through a skin lesion that may harbor micro-organisms. An added benefit to the use of ethyl chloride topical vapor coolant is that it decreases bacteria on the skin surface.[61]

HYPODERMIC NEEDLE-ASSOCIATED COMPLICATIONS

The hypodermic needle can be the source of complications in rare circumstances. The needle may separate from the hub during the procedure and require a minor surgical procedure to recover it.[62]

The needle tip may be advanced too deep and become embedded in the bony skeleton surrounding the joint. Upon withdrawing the syringe, the needle tip may break off and remain embedded in the bone or the needle may separate from the hub.

CORTICOSTEROID-INDUCED COMPLICATIONS

Corticosteroid-induced complications can be acute or chronic.[9,10,37,63-67] **The most severe acute complication is injection of corticosteroids into an infected joint. A septic arthritis must be ruled out prior to instillation of corticosteroids into a joint cavity**. Other local complications include steroid arthropathy, Charcot arthropathy, osteonecrosis, aseptic necrosis, tissue atrophy, tendon rupture, fat necrosis, formation of calcifications, joint instability, intraneural injection, and postinjection flare. Systemic complications include flushing, pancreatitis, posterior subcapsular cataracts, and hyperglycemia. Due to the potential for complications, many clinicians defer corticosteroid injections to the Orthopedist, Rheumatologist, or Sports Medicine consultant.

VASOVAGAL REACTIONS

The patient may experience an increase in vagal tone from apprehension, needle phobias, and/or pain. Vasovagal reactions are relatively common and may be associated with light-headedness and/or fainting. To prevent secondary injury to the patient, arthrocentesis should be performed with the patient on a stretcher or in a chair that reclines. These vasovagal reactions are self-limited and only require reassurance.

SYNOVIAL FLUID ANALYSIS

Hailed as the most valuable test in rheumatology, synovial fluid analysis provides essential diagnostic information for the appropriate management and treatment of urgent and emergent arthritic conditions.[6,10,68] It has been established as a fundamental component to the complete and appropriate work-up of arthritic diseases. With the possibility of potential joint destruction and chronic disability, **the role of synovial fluid analysis in the expedient diagnosis and treatment of acute joint disease cannot be overemphasized**.[69]

Controversy exists concerning what constitutes the appropriate guidelines for a "routine" synovial fluid analysis.[10,70-74] This controversy arises from multiple issues that include the clinical scenario, physician competency with appropriate arthrocentesis techniques, the sensitivity and specificity of individual tests, the availability and proficiency of laboratories, and cost. Classification schemes have been established based upon gross, microscopic, biochemical, and microbiological analyses. The most traditional and cited classification for synovial fluid is normal, noninflammatory, inflammatory, septic, or hemorrhagic (**Table 97-4**).[10,70,71,74] Despite the controversy that exists with guidelines and classification schemes, **it is critical to differentiate between an inflammatory and noninflammatory process, with the intent of expediting the diagnosis and treatment of a possible infectious etiology**.[10]

A detailed discussion of synovial fluid analysis is beyond the scope of this book. A brief discussion of the most essential components that can be performed in the ED will be presented.

TABLE 97-4 Synovial Fluid Analysis

	Normal	Noninflammatory	Inflammatory	Septic	Hemorrhagic
Gross analysis					
Color	Clear/yellow/straw	Straw/xanthochromic	Xanthochromic/cloudy/white	White/variable	Red
Clarity	Transparent	Transparent	Translucent/opaque	Opaque	Opaque
Viscosity	Very high	High	Low	Very low/variable	
Mucin clot	Good/firm	Fair-to-good/firm	Fair-to-poor/friable	Poor/friable	
Microscopic analysis					
Total leukocyte count (WBC/mm^3)	< 150	< 3000	3000–50,000	> 50,000	
Polymorphonuclear leukocytes (%)	< 25	< 25	> 70	> 90	
Biochemical analysis					
Glucose (mg/dL)*	Normal	Normal	70–90	> 90	
Protein (mg/dL)*	1.3–1.8	3–3.5	> 4.0	> 4.0	
Microbiological analysis					
Gram's stain	Negative	Negative	Negative	Positive†	Negative
culture	Negative	Negative	Negative	Positive†	Negative
Differential diagnosis					
		Osteoarthritis	Rheumatoid arthritis	Bacterial infections	Trauma coagulopathy
		Traumatic arthritis	Acute crystal		Anticoagulant therapy
		Early rheumatoid arthritis	Synovitis		Tumor
		Avascular necrosis	Viral arthritis		Charcot's arthropathy
		Crystal synovitis§	Psoriatic arthritis		Hemangioma
		Osteochondritis dissecans	Reiter's syndrome		Arteriovenous malformation
		SLE	Arthritis of IBD		Sickle cell disease
		Polyarteritis	SLE		Postsurgical
		Scleroderma	Polyarteritis		Joint prosthesis
		Amyloidosis	Scleroderma		
			Amyloidosis		

IBD, inflammatory bowel disease; SLE, systemic lupus erythematosus; WBC, white blood cells.

*Variable interpretation (refer to text).

†Variable results depending on organism (refer to text).

§Chronic or subsiding.

PATHOPHYSIOLOGY OF SYNOVIAL FLUID

Synovium refers to the one to three cell thick structure that lines the joint space and terminates at the articular cartilage margin.[70] This structure overlies a highly vascularized subsynovium, both of which are supported by the dense fibrous joint capsule.[70] The synovium produces synovia, an ultrafiltrate of plasma that includes hyaluronate. The synovia serves to lubricate, nourish, and clear the metabolic waste of the avascular articular cartilage.[71] Synovial fluid has been demonstrated to possess potent bactericidal activity against the most common gram-positive organisms responsible for septic arthritis.[75]

The synovium has been described as a double barrier in which molecules must pass through the endothelial microvasculature as well as the synovium and its matrix.[71] This double barrier is responsible for the retention of plasma protein. In the presence of an inflammatory process, the barrier is disrupted and protein can leak through the synovium. Difficulty arises in the diagnostic interpretation of protein and smaller molecules (i.e., sodium, chloride, urea, urate, lactate) found in synovial fluid secondary to the effects of damaged endothelial permeability and variable lymphatic drainage.[71,73,74]

GROSS ANALYSIS OF SYNOVIAL FLUID

The color of synovial fluid varies depending on the amount of protein, blood, and breakdown products of hemoglobin. Normal synovial fluid usually appears clear to a straw or yellow color. Inflammatory and purulent synovial fluid may appear xanthochromic to white. Hemorrhagic synovial fluid is red and must be distinguished from a traumatic arthrocentesis. A traumatic aspiration usually clots and is more than often nonhomogenous.[70,71] A hematocrit may be sent on a bloody aspirate to distinguish between a traumatic tap and hemorrhagic fluid. A vein was pierced by the needle (i.e., a traumatic tap) if the synovial fluid hematocrit is equal to the serum hematocrit.[71]

The following synovial fluid properties observed during bedside gross analysis were found to better predict a potentially septic joint when compared to synovial fluid cell count alone.[6]

The clarity of synovial fluid refers to the amount and type of particles within the fluid. Normal synovial fluid is usually transparent and newspaper print can be easily read through a glass tube containing this fluid.[70] Inflammatory and purulent synovial fluid is translucent to opaque secondary to the presence of leukocytes. Opaque fluid can also represent crystals and other particulate matter. Infected synovial fluid cannot be differentiated from noninfected synovial fluid based on gross appearance alone.[10]

Synovial fluid viscosity is determined by the intactness and concentration of hyaluronate. Viscosity can grossly be assessed by observing a drop of fluid fall from the tip of the needle. The "string" formed will normally be 5 to 10 cm in length.[76] In inflammatory and septic synovial fluid, the hyaluronidase is depolymerized and degraded.[71] The string formed in these conditions is shorter, or not formed at all, and the fluid falls as a drop. Processes resulting in a significant effusion without inflammation may also dilute hyaluronate without degrading it and result in decreased viscosity. Clotted white blood cells may enhance viscosity in the presence of inflammation.[71] EPs must be cautious with their interpretations of viscosity because even quantitative measures often fail to distinguish between inflammatory and noninflammatory states.[10]

The mucin clot test evaluates the degree of polymerization of hyaluronate.[10,70] The mucin clot test is performed by one of two methods. The first method involves mixing the supernatant of a centrifuged specimen with a few drops of glacial acetic acid. The second method involves mixing 1 mL of synovial fluid to 4 mL of 2% acetic acid. A "good" clot consists of a dense white precipitant that indicates a high degree of polymerization and a high viscosity.[70] A "poor" clot consists of little to no precipitate and suggests an inflammatory process that has depolymerized the hyaluronate. Controversy exists concerning the subjectiveness of the clot's endpoint.[10]

MICROSCOPIC ANALYSIS OF SYNOVIAL FLUID

The total leukocyte count, more than any other test, aids in distinguishing between an inflammatory, noninflammatory, and septic process.[71] Although a significant overlap may exist, the total leukocyte count can be used to identify synovial fluid as normal, noninflammatory, inflammatory, or septic (**Table 97-4**). Using this classification scheme, a total leukocyte count of less than 3000 cells/L is considered noninflammatory. A count between 3000 and 20,000 cells/L is considered inflammatory. The range of 20,000 to 50,000 cells/L may be inflammatory or septic. A count greater than 50,000 cells/L is considered septic until proven otherwise. **The overlap between categories is considerable and clinicians must be cautious of basing a diagnosis solely on the total leukocyte count**. Depending on the acuteness of the inflammation, several arthritic conditions such as gout, pseudogout, and rheumatoid arthritis may yield a significantly elevated total leukocyte count approaching 100,000 cells/L.[72] Immunocompromised hosts and some infectious diseases, such as tuberculosis and *Neisseria gonorrhoeae,* may have lower absolute counts than expected.[10]

The differential leukocyte count may further aid in distinguishing between inflammatory, noninflammatory, and septic synovial fluid. The cell count and WBC differential of the joint fluid is the best diagnostic test for septic arthritis, whereas the serum ESR and WBC perform poorer in the identification of infection.[77] However, using the traditional cut-off value of 50,000 WBC/mm^3 lacks enough sensitivity to safely rule out a septic arthritis.[78] Inflammatory processes generally have greater than 70% neutrophils, whereas septic synovial fluid has greater than 90% neutrophils.[70] Again, the overlap can be significant depending on the arthritic process and its acuteness. Crystal-induced processes may present with high neutrophil counts, while immunocompromised hosts, fungal infections, and tuberculosis may present with lower neutrophil counts.[10,72] **In general, a synovial fluid containing greater than 90% neutrophils in the presence of an elevated total leukocyte count should be highly suspicious of a septic process**. High eosinophil counts may suggest parasitic infection, allergic reactions, tumor, or Lyme disease.[71,73] High monocyte counts may suggest viral infection (e.g., rubella and hepatitis B) or serum sickness.[76]

Crystal identification is an essential component of synovial fluid analysis.[79,80] Crystal identification requires the use of a polarized light microscope with higher-powered lenses and oil immersion capabilities. Crystals can be identified based on their shape, size, and birefringence. Birefringence is defined as the crystals' ability to bend the light passing through it into two distinct directions, negative or positive. The light's ability to bend negatively or positively is transformed into a specific color (e.g., yellow or blue) under the polarized microscope.[70,71] Caution must be taken as artifact and tissue debris can often imitate birefringent material.

Crystal analysis requires an experienced technician and is rarely, if ever, performed in the ED. Monosodium urate crystals are commonly seen in gout. These crystals are needle-shaped, 2 to 25 μm in length, and have strong negative birefringence. They may clump together in sheets.[76,81] Local anesthetic solutions have the ability to dissolve monosodium urate crystals. Therefore, the joint cavity should not be penetrated with the needle when anesthetizing the skin and subcutaneous tissue.[72] Calcium pyrophosphate dihydrate crystals are seen in pseudogout. These crystals are

rhomboidal or rectangular, 2 to 10 μm in length, and have weak positive birefringence.[76,81] These crystals may be more difficult to detect than monosodium urate crystals because of the weaker birefringence. Cholesterol crystals may present in multiple forms and sizes. They are typically flat rhomboidal plates, 5 to 50 μm in length, and have both negative and positive birefringence.[76] Artifact "crystals" can be produced by a variety of substances. Corticosteroids can be detected weeks after injection and have variable shapes but no regular geometric form. Maltese crosses are strong birefringent particles that are secondary to multiple compounds such as talc powder, lipids, calcium oxalate, and dust.[76]

BIOCHEMICAL ANALYSIS OF SYNOVIAL FLUID

As discussed previously under the pathophysiology section, difficulty arises with the interpretation of total protein secondary to the effects of damaged endothelial permeability and variable lymphatic drainage.[71] In theory, the damage caused by an inflammatory process should increase the permeability of proteins into the synovial fluid. Multiple studies have shown that protein samples were only able to classify synovial fluid into an inflammatory or noninflammatory process in approximately 50% of the cases.[73] Furthermore, the total protein count was unable to differentiate among various groups of arthritides, including rheumatoid arthritis and osteoarthritis.[10]

Theoretically, inflammatory and infectious processes consume glucose and thus lower the level present in synovial fluid. One study has shown that glucose levels were able to classify synovial fluid into an inflammatory or noninflammatory process in less than 50% of cases.[73] In 50% of the septic joints analyzed, the glucose level was not significantly decreased. Another study reports glucose analysis having a sensitivity of 20% and specificity of 84% in detecting inflammatory joint disease.[74] Synovial fluid glucose levels can vary from serum glucose levels when taken less than 6 hours after oral intake.[73] These studies are just a few of many that confirm synovial fluid glucose levels are not reliable to diagnose or rule out a septic joint.

Other biochemical markers have been studied to elicit a marker to differentiate a septic from a nonseptic joint.[82] These include lactate, lactate dehydrogenase, and numerous immunologic and inflammatory mediators. These biochemical markers, at present, are not sensitive or specific to rule out a septic joint. They are not recommended for routine synovial fluid analysis. Bacterial polymerase chain reaction (PCR) techniques with aspirated joint fluid are currently being studied and are theoretically superior tests, able to identify difficult-to-culture organisms. However, many drawbacks, including high false positives due to contamination, limit usage until further studies are done.[78]

MICROBIOLOGICAL ANALYSIS OF SYNOVIAL FLUID

The Gram's stain is an easily performed test that yields rapid results and can lead to the expedient diagnosis and treatment of a septic joint. The Gram's stain has a sensitivity of 50% to 70% for nongonococcal infections and less than 10% for gonococcal joint infections.[74] Although the sensitivity of this test is low, the specificity of the Gram's stain approaches 100%. This makes it an essential component of routine synovial analysis. *Neisseria gonorrhoeae* is identified as a gram-negative intracellular diplococcus.[83] *Staphylococcus aureus* and *Streptococcus* are responsible for approximately 70% of nongonococcal septic arthritis and can be identified as gram-positive cocci in clusters and gram-positive cocci in chains, respectively.[57,81,84,85] Recent studies have identified an increased prevalence of methicillin-resistant *S. aureus* (MRSA)-associated osteoarticular pathology such as septic arthritis and subperiosteal abscesses.[69] These diagnoses should be considered in patients with and without risk factors for community-acquired MRSA infection.[69]

Bacterial identification is essential when confronted with the possibility of a septic joint. Synovial fluid cultures have a sensitivity of 75% to 95% for nongonococcal bacteria and 10% to 50% for gonococcal bacteria in the absence of previous antibiotic treatment. Difficulty arises from the low sensitivity of cultures for some organisms, culture methods, specimen preparation, and the length of time for some bacteria to grow.[74] Recent advances with the use of PCR techniques have shown increased sensitivity and specificity for detecting *N. gonorrhoeae*.[70,83]

SPECIMEN COLLECTION

Minimal quantities of synovial fluid can yield valuable information. Analysis can be performed with as little as two drops of synovial fluid.[72] The total leukocyte count, differential leukocyte count, crystal analysis, and Gram's stain can be obtained from the first drop, while the second drop can be sent for cultures.[72] Unfortunately, few institutions or laboratories can perform synovial fluid analysis with only two drops of fluid.

The specimen may be transported for analysis by one of two methods. First, the syringe with the synovial fluid may be capped and sent to the laboratory. The laboratory technicians will then divide the specimen for analysis. Alternatively, the synovial fluid may be placed into tubes. A new sterile needle, different from the arthrocentesis needle, should be used to place fluid in the test tubes. A needle used for steroid injection should never be used secondary to the formation of a crystal-like substance.[10] Synovial fluid for microscopic analysis should be collected in test tubes with and without preservatives. Fluid should be placed in a Culturette or culture bottle for microbiological analysis. Synovial fluid for crystal analysis should be sent in a test tube with liquid heparin (e.g., green-topped tube) because EDTA and powdered anticoagulants interfere with crystal identification.[72] A tube (e.g., red-topped) containing no preservatives should be used to test for chemistries, serology, and viscosity. A tube with an anticoagulant (e.g., purple-topped tube) is used for the cell count, cell differential, and cytology. Prompt examination of fluid specimens is urgent to avoid the following problems: misdiagnosing borderline inflammatory fluids because of decreased WBC counts, not identifying crystals that dissolve with time, or overinterpreting findings because of new, artifactual crystals.[86]

SUMMARY

Arthrocentesis is used to diagnose and make treatment decisions regarding a joint. It is a safe, easy, and simple procedure. Arthrocentesis is relatively painless and extremely beneficial to the patient. It may be performed for diagnostic information and/or therapeutic treatment. Analysis of the synovial fluid provides unique and valuable information about a joint. It is the only method to accurately and definitively diagnose or rule out a septic arthritis. Arthrocentesis is indicated to evaluate the cause of an arthritis or a joint effusion. All patients presenting with an acute monoarthritis or an acute, nontraumatic effusion should undergo arthrocentesis when the diagnosis is not clear based on the history and physical examination.

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98 Methylene Blue Joint Injection

Pholaphat Charles Inboriboon and Katherine Gloor Willet

INTRODUCTION

Trauma can breach the integrity of the joint capsule and result in infection, long-term arthritis, and other serious potentially permanent morbidity. Any breach of the joint capsule can introduce contaminants and risk septic inoculation of the joint. Synovial joint fluid provides nutrition to the articular cartilage. Loss of this fluid without prompt joint capsule closure can lead to cartilage wasting and arthritis. The injection of joints with methylene blue provides a rapid and definitive way to assess joint capsule integrity in cases of periarticular trauma where the clinical examination is inconclusive.

ANATOMY AND PATHOPHYSIOLOGY

Synovial joints (e.g., the fingers, wrist, shoulder, and knee) consist of a fibrous capsule that overlies a thin and delicate synovial membrane (**Figure 98-1**). The synovial membrane is a highly vascular structure and the site of synovial fluid production. The synovial

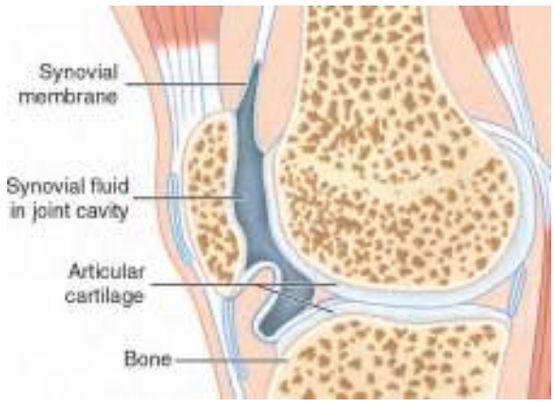


FIGURE 98-1. The anatomy of the knee joint.

fluid provides nutritional support to the relatively avascular articular cartilage. Joint capsules contain varying volumes of fluid that can be greatly expanded under conditions of inflammation or injection.

Methylene blue is a relatively safe and stable deep blue dye used in both chemistry and medicine. It is frequently used to treat methemoglobinemia and for marking skin and lymphatic tracts for oncologic surgery. The proposed basis for its vasoconstrictive effects is its ability to be readily oxidized.[1] Undiluted methylene blue can cause direct tissue necrosis without prior immune sensitization.[2]

The literature regarding the safety of methylene blue supports its use, even during direct intradermal injection. When used for lymph node mapping by direct intradermal and intraparenchymal injection, there is consistent evidence of local inflammatory reactions ranging from local wheal and flare in 0.5% of patients to focal erythema with induration in 5% of patients.[3,4] Superficial tissue toxicity occurred in only 1.25% of patients.[4] These reactions were proportional to the concentration of methylene blue ranging from a dilution of 1:1 to 1:7 and the superficiality of the injection.[4] **Emergency Physicians (EP) need to be aware of the potential complications of methylene blue. The tissue toxic effects are believed to be much less for intraarticular injections than direct intradermal injection.**

INDICATIONS

The primary indication for injecting a joint with methylene blue is to assess the integrity of the articular joint capsule (**Figure 98-2**). This includes any of the following injuries in proximity to a joint: skin laceration, a visible joint capsule through a wound, an open fracture, extravasation of serous or serosanguineous fluid from a wound, or a traumatic loading of the joint with evidence of a deformity or an acute effusion. The finger, wrist, elbow, shoulder, toe, ankle, knee, and hip joints can all be injected with methylene blue.

CONTRAINDICATIONS

There are few absolute contraindications to injecting methylene blue into a joint. It should be avoided in patients with a known allergy to methylene blue or patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency. Patients with G6PD deficiency do not generate sufficient NADPH to efficiently reduce methylene blue to leukomethylene blue. This makes them prone to methylene blue–induced hemolysis, oxidative hemolysis, and hemolytic anemia. Methylene blue can itself initiate methemoglobin formation. It is reasonable to consider using a saline load test in patients with a contraindication to methylene blue or if methylene blue is not

FIGURE 98-2. Methylene blue dye injection into a knee joint. The loss of capsule integrity allows the dye to flow out of the joint cavity.

available.[5,6] This involves the joint injection of a high volume of saline to stretch the joint capsule and watch for extravasation. It may be more difficult to appreciate extravasation of saline without dye. Methylene blue should not be injected intraspinally or intrathecally.

There are a few relative contraindications to the injection of methylene blue. Any joint injection should be used cautiously in a patient with a known coagulopathy, especially a factor deficiency, to prevent intraarticular hemorrhage. Consider reversing the coagulopathy before the injection. Methylene blue injection should not be performed if there is a lack of procedural knowledge or skill by the EP.

Not all synovial joints can be injected due to structural instability. Joint injection is often unnecessary when definitive operative treatment of the joint is indicated. This includes a visibly open joint capsule, a fracture with obvious joint involvement, intraarticular air on radiographs, and an intraarticular foreign body.

The articular capsule will be breached in many injuries but not externally open to the environment if there is no wound tracking to the skin surface. These injuries may present with an acute joint effusion or radiographic evidence of a periarticular fracture with or without intraarticular air. Methylene blue is not helpful in such cases despite capsular rupture.

Use caution when using methylene blue on psychiatric patients or those taking psychiatric medications.[7,8] Methylene blue has potent monoamine oxidase inhibitor properties. It can react with certain psychiatric medications to cause a serotonin syndrome (**Table 98-1**). This adverse interaction has not been documented in the literature with the intraarticular instillation of methylene blue.

EQUIPMENT

- Sterile drapes
- Sterile gloves
- Sterile gown
- Face mask
- Eye protection
- Povidone iodine or chlorhexidine solution
- Sterile gauze
- Sterile dressing or tape
- Sterile saline
- Sterile basin
- Local anesthetic solution (e.g., 1% or 2% lidocaine)
- 25 or 27 gauge needles for infiltrating local anesthetic
- 18 or 21 gauge needles for joint injection
- 5 mL syringes for local anesthetic injection
- 10 mL syringes for fingers or toe injection
- 30 mL syringes for wrist or elbow injection
- 60 mL syringes for hip, knee, or shoulder injection
- 1% methylene blue dye
- Ultrasound machine (recommended)
- Sterile ultrasound gel
- Sterile ultrasound transducer cover
- Linear transducer with small footprint

PATIENT PREPARATION

A complete history and physical examination should be performed prior to the injection of methylene blue. The affected joint should be thoroughly assessed. Inspect the skin overlying the joint for breaks, infection, old scars, prior incisions, superficial lesions, or any wounds. Palpate the joint to identify any warmth, tenderness, or effusion. Evaluate the joint for any crepitation, deformity, ligamentous instability, or limitations in motion.

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. Consent the patient for both the arthrocentesis procedure (i.e., joint injection) and the use of methylene blue. Obtain a written consent from the patient or their representative. Place the consent in the medical record.

Prepare the ultrasound transducer if using ultrasound with the procedure. Have an assistant apply ultrasound gel over the footprint of the transducer. Instruct the assistant to place the gel-coated ultrasound transducer in the sterile cover while you hold it. Have the

TABLE 98-1 Psychiatric Medications That Can Adversely React with Methylene Blue

Selective serotonin reuptake inhibitors (SSRIs)	Serotonin-norepinephrine reuptake inhibitors (SNRIs)	Tricyclic Antidepressants (TCAs)	Monoamine oxidase inhibitors (MAOIs)	Miscellaneous psychiatric medications
Citalopram	Desvenlafaxine	Amitriptyline	Phenelzine	Amoxapine
Escitalopram	Duloxetine	Clomipramine	Selegiline	Bupropion
Fluoxetine	Venlafaxine	Desipramine	Tranylcypromine	Buspirone
Fluvoxamine		Doxepin		Maprotiline
Paroxetine		Imipramine		Mirtazapine
Sertraline		Nortriptyline		Nefazodone
		Protriptyline		Trazodone
		Trimipramine		Vilazodone

Source: Adapted from references 7 and 8.

assistant place sterile ultrasound gel over the patient's joint. Reidentify the target.

Position the patient based on the specific joint to be injected and the planned approach. Expose the joint, the wound, and the surrounding areas. Identify the anatomic landmarks required for proper needle placement. The landmarks may be difficult to identify on a swollen and tender joint. Compare the "affected" joint to the "normal" joint on the opposite side of the body. Identify the joint and a landmark on the normal joint and transfer this to the affected joint. The injection of the joint should be ideally performed through a site that is not adjacent to the wound (**Figure 98-3*B***). Leakage from the injection site into the wound could lead to a false-positive dye extravasation. **Avoid injecting through skin that is infected.**

Prepare the skin. Clean any dirt and debris from the skin. Scrub the needle insertion site with povidone iodine or chlorhexidine solution and allow it to dry. Apply sterile drapes or a clear sterile dressing to maintain sterility of the injection site (**Figure 98-3**). The rate of infection resulting from intraarticular injections is very low if sterile technique is used.[9]

A comfortable and relaxed patient will facilitate arthrocentesis. Tense muscles can distort the anatomy of the joint and make palpation more difficult. Oral analgesics for the precipitating injury along with adequate local anesthesia for the procedure are often sufficient. The articular cartilage has no intrinsic pain fibers. The synovial membrane, joint capsule, and skin are richly innervated. Adequate time should be taken for the infiltration of local anesthetic solution. The subcutaneous injection of local anesthetic solution is often most practical for larger joints (e.g., the knee, shoulder, and hip). A regional block is often easier for small and more distal joints (e.g., the finger, ankle, and toe).

The administration of local or topical anesthesia is recommended but not required. The most common local anesthetic used is a short-acting injectable anesthetic solution of 1% lidocaine. There is disagreement regarding whether the additional needle stick to administer the anesthesia causes as much discomfort as performing the procedure without any anesthesia. This decision is specific to each EP and patient. Topical anesthetics (e.g., LET [lidocaine, epinephrine, tetracaine] or EMLA [eutectic mixture of local anesthetics]) can be considered, but the time it takes to achieve anesthesia can delay prompt evaluation of the joint.

Alternative methods of anesthesia include ice and topical vapor coolants. A sterile drape may be placed over the prepped skin and a bag of ice water placed over the drape. Remove the ice water bag and drape after 5 minutes and perform the procedure. Ethyl chloride topical vapor coolant may be used as an anesthetic. Spray the solution onto the area of skin in which the needle will be inserted. Apply the spray from 6 inches above the skin. Spray until the skin turns white and frosty. This usually takes 5 to 10 seconds. Immediately perform the procedure as the anesthesia lasts only 30 to 60 seconds.

It is common to dilute the dye in normal saline before the injection. Draw up one to two drops of 1% methylene blue into an appropriately sized syringe. Fill the remainder of the syringe with sterile saline to dilute the dye.

TECHNIQUES

THE BASIC TECHNIQUE

The use of musculoskeletal ultrasound is useful to assist in arthrocentesis and has been shown to have many benefits including greater fluid aspiration, greater physician confidence, and greater accuracy.[10,11] It does not result in more pain for the patient and takes minimal additional time to perform at the bedside. Bone will appear hyperechoic and easily differentiated from muscle and subcutaneous tissue. Tendons will appear fibrillar like a bundle of drinking straws. Joint fluid will appear hypoechoic and dark. The "seagull sign" is a shape that can be seen on most joint images (**Figure 98-4**). It represents the joint space between the articular surfaces of two opposing bones (**Figure 98-4**). It is a V-shaped hypoechoic area surrounded by hyperechoic bone. The needle can be inserted with or without ultrasound guidance once the landmarks are identified.

Apply the needle to the syringe and break the resistance. This helps avoids any sudden and painful movements of the needle within the joint cavity. Stretch the skin over the site where the needle will be inserted. Penetrate the skin briskly with the needle and enter the joint cavity. Gently aspirate synovial fluid to confirm the proper needle position within the joint cavity. If bone is encountered, slightly withdraw the needle and advance it in a different direction. Grasp and stabilize the hub of the needle with your fingers or a hemostat. Remove the syringe and attach the one with methylene blue.

Inject the methylene blue slowly into the joint cavity. There should be no resistance to flow if the needle is within the joint capsule.

A B

FIGURE 98-3. An injury at the level of the knee joint. The patient is prepared by draping the lateral joint where the needle will be inserted (***A***) or by dressing the area with a sterile clear dressing (***B***).

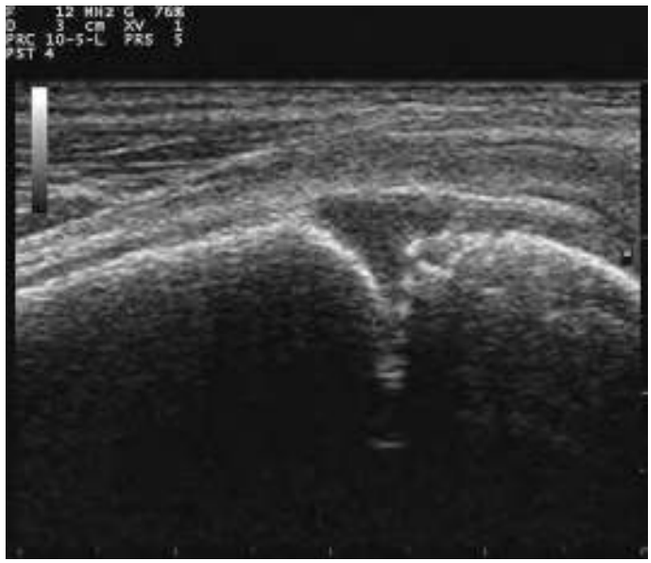


FIGURE 98-4. Ultrasound of a joint. Note the "seagull sign," which is a V-shaped hypoechoic area surrounded by hyperechoic bone. (Used from Devit Dilmen from www.commons.wikimedia.org.)

The injection should not be painful if the needle is within the synovial capsule. The patient may feel a slight discomfort as the joint capsule fills with fluid. Observe the skin wound for extravasation of blue dye while injecting the joint. Remove the needle when the procedure has been completed. Apply a bandage to the skin.

Different joints will accommodate various amounts of injected volume. The knee may allow for 30 mL or more, whereas the finger may accommodate only 1 mL of fluid. Often, it is only necessary to inject a minimal amount of dye before extravasation is seen. If the joint capsule is ruptured, a greater amount of fluid can be injected, as it will escape through the breach. The capsule will expand in intact joints. This results in increasing pressure and resistance to continued injection. There is often visible swelling of the skin around the injected joint if there is no breach.

SPECIFIC SITES

Methylene blue joint injection can be reasonably accomplished at any joint. The concern for joint capsule rupture without the concomitant need for operative exploration and fixation is rare in joints other than the knee and fingers. The knee is relatively easy to inject while the fingers and toes are more difficult. Arthrocentesis with methylene blue injection in the knee and finger is discussed below. The principles and techniques described can be used on any synovial joint for which there is concern of a capsule breach and for which the EP feels competent to perform arthrocentesis (Chapter 97).

PATELLOFEMOROTIBIAL JOINT (KNEE), PARAPATELLAR APPROACH

Landmarks: The knee joint can be injected in a variety of locations and with the knee extended or flexed. Palpate the superior, lateral, and inferior borders of the patella and the patellar tendon (**Figure 98-5*A* and 98-5*B***). Identify the midpoint of the lateral or medial border of the patella (**Figure 98-5*B***). Either of these landmarks may be used as the site for needle insertion.

Patient positioning: Place the patient in a position of comfort. Ideally, position the patient supine with the affected knee fully extended.

Needle insertion and direction: Insert an 18 gauge needle just below the midpoint of the lateral or medial border of the patella (**Figure 98-5*C***). Direct the needle perpendicular to the long axis of the leg and aimed toward the intercondylar notch of the femur. Advance the needle to a depth of 1 to 2 cm and aspirate until synovial fluid is obtained. Inject the methylene blue dye.

Ultrasound transducer placement: Start with transducer placement longitudinally and lateral or medial to the patella for a first view of the possible fluid collection. Change the transducer to a horizontal orientation. Rotate the transducer like the hands of a clock around the patella 360° to discover the area with the largest anechoic fluid collection. Advance the needle into the fluid space under ultrasound guidance. Grasp and stabilize the hub of the needle with your fingers or a hemostat. Inject the methylene blue.

Remarks: The blind lateral and medial parapatellar approaches are used with high relative success. This is most likely due to the large joint space and minimal accessory structures. Pooled studies demonstrate an overall lower success rate with the blind medial midpatellar approach (64%) compared with the blind superior lateral patellar approach (87%).[11] Success was increased with the use of ultrasound.[11]

Joint injection: The volume injected into the knee joint is a topic of debate. Some studies suggest that 150 to 180 mL may be necessary for ruling out joint capsule involvement.[5,6] The additional injection is not necessary once extravasation is noted.

METACARPOPHALANGEAL (MCP) JOINT

The relatively thin dermal and subcuticular layers over the phalanges often make one wonder about deep soft tissue avulsions or lacerations and the potential involvement of the joint capsule. Injection with methylene blue is an ideal method to assess joint capsule integrity. The success rate of arthrocentesis is much lower in the phalangeal joints than larger joints. The overlying ligaments and tendons are more prominent and the synovial capsule is smaller. A failure rate of 15% for finger arthrocentesis was found among skilled surgeons and as high as 32% among first-year residents.[12] Successful arthrocentesis was highest in the proximal interphalangeal joint compared to the distal interphalangeal joint or carpometacarpal joint of the thumb.[12]

Landmarks: Identify the MCP joint and the extensor digitorum tendon (**Figure 98-6*A* and 98-6*B***). The MCP joint can be located just proximal to the prominence at the base of the proximal phalanx of the finger. Identify the extensor tendon by having the patient extend the finger against resistance.

Patient positioning: Place the patient sitting upright or supine on a stretcher. Pronate the hand and abduct the fingers. Grasp the finger and apply distal traction.

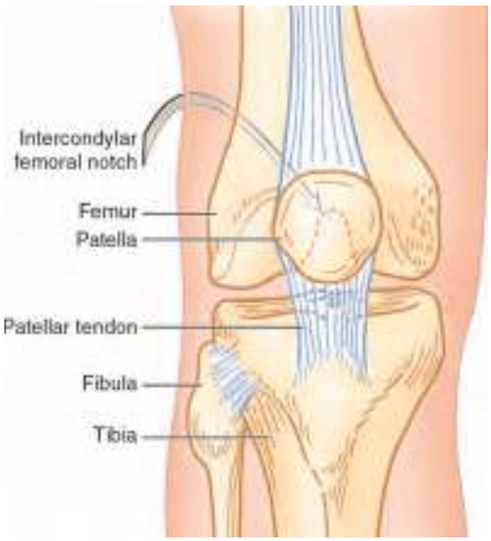
Needle insertion and direction: Insert a 22 gauge needle into the dorsal joint space just medial or lateral to the extensor tendon (**Figure 98-6*C***). Direct the tip of the needle toward the center of the joint. Advance the needle to a depth of 0.3 to 0.5 cm. Inject the methylene blue.

Remarks: The application of distal traction often causes a depression to appear on both sides of the extensor tendon. These depressions can be used as landmarks for the site of needle insertion into the joint cavity.

Joint injection: A maximum volume of 1 mL may be instilled into this joint.

ASSESSMENT

The lack of methylene blue extravasation reassures the EP of an intact joint capsule. Extravasation of methylene blue through the injury site is indicative of a ruptured joint capsule. These require exploration, high-volume irrigation, and adjunctive medical treatment (e.g., antibiotics). Although some wounds can be closed primarily in the Emergency Department after consultation with an



A

B

C

FIGURE 98-5. Methylene blue injection of the knee. ***A.*** Anatomy of the region. ***B.*** Palpating the boundaries of the patella. ***C.*** Injection of methylene blue using the medial parapatellar approach.

Orthopedic or Hand Surgeon, open joints require management in the Operating Room.

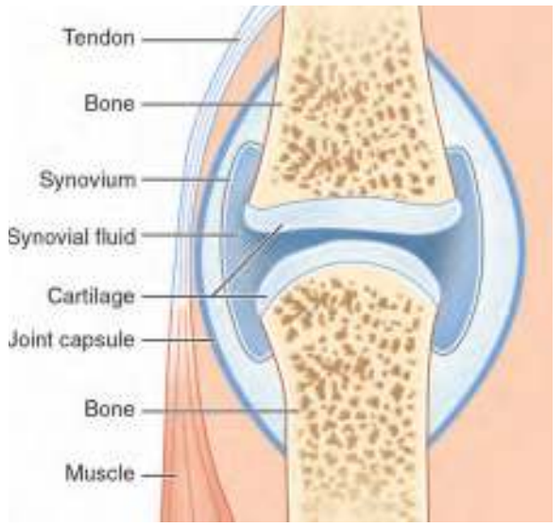
A minimal amount of methylene blue needs to be injected before visible extravasation will occur. Upon injection, if no extravasation occurs, attempt to aspirate fluid. This may be difficult in small joints. Aspiration of blue fluid provides additional confirmation of intracapsular needle placement. **Maintain a high index of suspicion for a joint capsule rupture in the face of a negative study given the significant rate of ectopic needle placements (i.e., extrasynovial, intraligamentous, or intratendinous). Clinical intuition and judgment should guide all decisions.** Methylene blue injections may not be sensitive enough to identify violation of the joint capsule and may lead to an unacceptable rate of false negatives in the setting of puncture or stab wounds.[13] Patients needed injections of up to 155 mL of fluid to obtain a positive result in 95% of subjects in a study looking at 1 cm arthroscopic incisions and methylene blue testing.[5]

AFTERCARE

Arthrocentesis with methylene blue is a relatively benign procedure. The postprocedural care consists of monitoring for external bleeding and swelling. Musculoskeletal and neurovascular checks should be performed at the area and distal to the joint. Apply a bandage to the injection site. Pain should be minimal, and analgesia can be achieved with oral nonsteroidal anti-inflammatory drugs (NSAIDs). The patient may already be receiving opioid analgesics in the setting of a significant traumatic injury. Warn the patient or their representative that urine excretion of methylene blue can change the color of their urine.[14]

COMPLICATIONS

Complications are rare and include hemarthrosis, nerve injury, tendon injury, cartilage damage, and infection. External bleeding and nerve damage should be immediately apparent. A hemarthrosis may be insidious, appear with progressive swelling and pain with joint motion, and often without joint warmth. Tendon and cartilage damage may not be apparent for some time and may present with joint stiffness or arthritis. Septic arthritis is the most concerning complication, evidenced by swelling, erythema, warmth, pain with range of motion, and systemic symptoms. Cellulitis may also complicate the procedure and appear with local warmth, erythema, and induration over the site. Refer to Chapter 97 for a more complete discussion regarding the complications of arthrocentesis.



A

B

C

FIGURE 98-6. Methylene blue injection of the metacarpophalangeal (MCP) joint. ***A.*** Anatomy of the region. ***B.*** Palpation of the MCP joint. ***C.*** Injection of methylene blue.

SUMMARY

Synovial joint injection with methylene blue provides the EP with a simple, rapid, and definitive method of assessing joint capsule integrity in cases of periarticular trauma. Ultrasound may be useful to improve success rates in some joints. This procedure can help to determine between repairing a wound and sending the patient home or admitting a patient to the hospital for joint exploration and closure. Joint injection with methylene blue has a low rate of complications.

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99 Basic Principles of Fracture and Joint Reductions

Scott C. Sherman and John Hardwick

INTRODUCTION

Orthopedic injuries are some of the most common presenting complaints facing the Emergency Physician (EP). Forces that cause injury can be large enough to result in fractures, displaced fractures, and joint dislocations. While each injury is different, some general principles can be applied to all displaced fractures and joint dislocations. Specific instructions on the techniques to reduce common fractures and dislocations are in Chapters 101 through 113.

ANATOMY AND PATHOPHYSIOLOGY

The most common reason for a fracture to be displaced or a joint to be dislocated in a particular direction depends on the mechanism of injury. A fall forward on an outstretched arm is the most common mechanism of injury of the upper extremity. It results in elbow dislocations occurring most frequently in a posterior direction. Distal radius fractures occur most often as Colles fractures, and supracondylar fractures are extension-type fractures in 95% of cases.[1]

The deforming forces of muscles and ligaments play an important role in the appearance of a fracture. The muscles surrounding a fracture contract or spasm. This leads to further deformity (e.g., shortening, angulation, and rotation) of the bone fragments distal and proximal to the fracture. The bone fragments will displace in different directions depending on the location of a fracture (**Figure 99-1**). Fractures that occur between the insertions of the pectoralis major and deltoid muscles will result in a proximal humerus that is adducted from contraction of the pectoralis major muscle and a distal humerus that is abducted from contraction of the deltoid muscle (**Figure 99-1*A***). The proximal humerus will be abducted from contraction of the deltoid muscle and the distal humerus will be adducted from contraction of the biceps and triceps muscles if the fracture occurs distal to the deltoid insertion (**Figure 99-1*B***).

INDICATIONS

The reduction of fractures and joint dislocations in the Emergency Department (ED) is more frequently indicated than it is contraindicated. **Reduction is more readily achieved soon after an injury.** No fracture benefits from a prolonged period of angulation or displacement because the reduction becomes more difficult the longer the fracture has been present. No joint benefits from a prolonged dislocation as damage to articular cartilage increases with time.

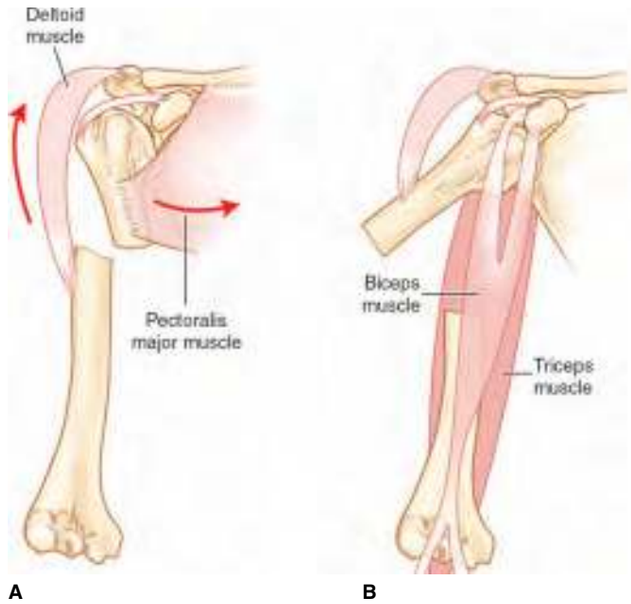

FIGURE 99-1. The direction of fracture displacement is influenced by muscle contraction following the injury. ***A.*** Humeral shaft fracture between the insertions of the deltoid and pectoralis major muscles. *Arrows* represent the direction of pull on the fracture fragments. ***B.*** Humeral shaft fracture distal to the deltoid muscle insertion.

Reduction should occur on an emergent basis when perfusion to the extremity is absent. A nonperfused extremity has a finite time before nerve and muscle death occurs. Reduction should occur as soon as possible. **The earlier perfusion is restored, the better is the chance of avoiding tissue necrosis.**

Vascular injury can occur after any displaced fracture or dislocation. Note the presence of an expanding hematoma, absent distal pulses, or delayed capillary refill. Orthopedic injuries more commonly associated with vascular injury include knee dislocations, posterior sternoclavicular joint dislocations, and supracondylar fractures. The importance of early reduction and repair of vascular injuries is emphasized in knee dislocations. Up to 86% of patients will require an amputation if surgery is delayed more than 8 hours.[2]

Urgent reduction of displaced fractures and joint dislocations is indicated to reduce the incidence of other potential complications. The incidence of a compartment syndrome of the forearm is reduced by early reduction of displaced supracondylar fractures.[3] Reduce a posterior hip dislocation in a timely manner. The likelihood of avascular necrosis rises exponentially from a rate of 5% with less than 6 hours of being dislocated to 50% with greater than 6 hours of being dislocated.[4]

Neurologic injury results from the original injury, traction on a nerve, or compression of a nerve. Patients may experience altered sensation, decreased sensation, and/or paresthesias. **These injuries require urgent reduction to decrease potential long-term complications.**

CONTRAINDICATIONS

Be aware of potential relative and absolute contraindications despite the potential advantages of early reduction of fractures and dislocations in the ED. Chronic or previously unrecognized dislocations are difficult to reduce in a closed manner and overly forceful

attempts increase the chances of causing a fracture. An example is a surgical neck of the humerus fracture in a patient when attempting to reduce a chronic shoulder dislocation. **Reduction is contraindicated when the patient requires immediate operative treatment.** An open fracture in a perfused extremity should be reduced in the Operating Room where an appropriate surgical washout can occur.[5]

Not all displaced fractures and dislocations suspected of causing vascular injury should be reduced in the ED. An actual or suspected vascular injury associated with a posterior sternoclavicular joint dislocation is best performed in the Operating Room with a Cardiothoracic Surgeon available because the distal clavicle may be tamponading a lacerated subclavian vessel.[6] Supracondylar fractures require immediate reduction only when the extremity is pulseless and perfusion is absent.[7]

Reduction is unnecessary when the potential for remodeling is such that the fracture angulation will correct without the need of a painful reduction or the risk of procedural sedation.[8,9] Distal radius fractures in children heal well when they are 15° to 20° angulated.[10-13] Other fractures where some degree of displacement or angulation may be acceptable and that frequently do not require reduction include fractures of the clavicle, humeral shaft, and neck of the fifth metacarpal.[1,14-20]

Reduction may be contraindicated in the ED for numerous other reasons. Adequate analgesia may not be possible due to the patient's medical condition or the inability to appropriately monitor the patient. These cases may require general anesthesia in the Operating Room. It is prudent to consult and wait for an Orthopedic Surgeon when the potential complications of a reduction attempt are high or the EP is uncomfortable performing the technique. A busy EP in a chaotic ED with single coverage may not have the time or resources to adequately sedate a patient and reduce the fracture or dislocation. This situation may require calling in an additional EP or an Orthopedic Surgeon.

EQUIPMENT

- Procedural sedation equipment and supplies (Chapter 159), if applicable
- Hematoma block supplies (Chapter 155), if applicable
- Regional nerve block supplies (Chapter 156), if applicable
- Gurney
- Weights for distraction
- Bed sheets for traction-countertraction techniques
- Splinting/casting material or commercially made splints (e.g., knee immobilizer)
- Finger traps for upper extremity reductions
- Ultrasound machine and supplies
- Fluoroscopy, if available, to assist with the reduction and postreduction radiographs
- Assistants, depending on the reduction technique

PATIENT PREPARATION

Reduction techniques require a well-informed patient and a signed consent if possible. Explain the risks, benefits, and alternatives to the patient and/or their representative. Tell the patient what to expect. This knowledge may assist in the successful performance of the procedure. An anterior shoulder dislocation reduction can be performed with minimal or no sedation if the patient is able and willing to relax their musculature while the EP slowly manipulates the humeral head back into position.[21] **Consent the patient for the anesthesia technique because this is a second procedure with potential complications much different from the reduction.**

The physical preparation of a patient for the reduction of a fracture or dislocation is dependent on the type of injury and the clinical setting. The patient should be resting as comfortably as possible on a gurney. Place the patient supine whenever possible. Fully expose the involved extremity. Remove any constricting pieces of clothing or jewelry proximal and distal to the injury. Move the fluoroscopy unit into position if it is judged to be useful and is available. Splint material is frequently measured and set up prior to the start of the procedure so that it may be immediately applied to the extremity in the setting of an unstable fracture or dislocation.

Always consider a nerve block (Chapter 156) or hematoma block (Chapter 155) when approaching a reduction.[22] Avoiding procedural sedation (Chapter 159) can result in better patient throughput, free nurses to complete other clinical tasks, and decrease the risk of procedural complications.[4,6,14]

TECHNIQUE

The basic principles to reduce a displaced fracture or a joint dislocation are similar. The procedure can be divided into four steps.[23] **These are distraction, disengagement, reapposition, and release. The four steps can be used to reduce most displaced fractures or joint dislocations.**

The first step is distraction. It involves creating a longitudinal force to pull the fracture fragments or bones involved with a dislocated joint apart. Perform this step gradually. It sometimes requires time to be most effective in overcoming muscle spasm. Distraction is important when the fractured ends of the bone are overriding. It can be applied manually, with the help of an assistant, or by using weights. One common technique to distract the extremity injury uses finger traps to hold the hand or toes in place while weights are applied more proximally (**Figure 99-2**). Saline bags placed in a stockinette are equally effective and comfortable for the patient when weights are not available (**Figure 99-3**). Each liter bag of saline is equivalent to 1 kg or 2.2 pounds.

Disengagement is the second step to reduction. It allows for further disimpaction of the fracture fragments than distraction alone. Achieve disengagement by rotation of the distal fragment or more

FIGURE 99-2. Distraction for reduction of a forearm fracture.

FIGURE 99-3. Four 1 L bags of saline in a stockinette are the equivalent of 8.8 pounds. This setup is useful when weights are not available.

classically by "recreating the fracture deformity." This relieves tension on the surrounding soft tissues to allow the interlocking fracture fragments to reposition (**Figure 99-4**). Disengagement is important for dislocation reduction. It is most frequently achieved by rotation of the bone distal to the joint. An example is the external rotation technique to reduce an anterior shoulder dislocation.

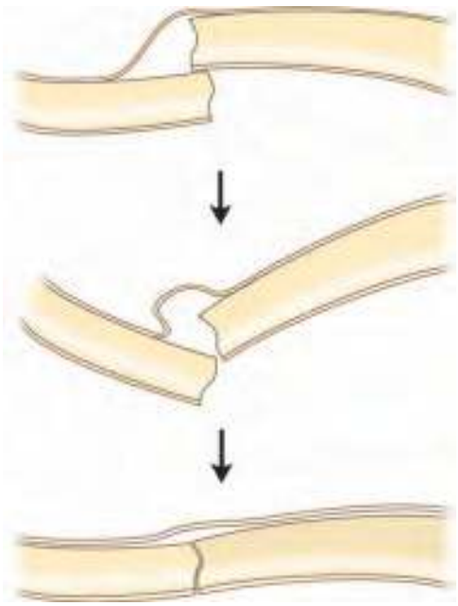

FIGURE 99-4. Disengagement and reapposition. Recreating the fracture deformity allows the release of interlocking fragments by lengthening and relaxing the soft tissue hinge.

FIGURE 99-5. Reapposition of a forearm fracture.

The third step is reapposition. This is achieved by reversing the forces that caused the injury to bring the bony fragments back into alignment (**Figures 99-4 and 99-5**). This step seems simple conceptually, but it may not be easy in clinical practice. One important pitfall to avoid is ignoring a rotational deformity that might create functional problems if the bone healed with a rotational deformity. This is especially important when treating fractures of the hand where a 5° rotational deformity of a proximal metacarpal fracture can translate into a distal fingertip that is 1.5 cm out of position (**Figure 99-6**).[24]

Release refers to the removal of the initial distracting force with the intent that alignment will be maintained. It is at this point that forces such as muscle contraction and gravity return. The fracture fragments are at risk for becoming malaligned again. A properly applied splint or cast can protect from loss of fracture alignment. It is advisable to have the splint measured and ready to apply before manual reduction is initiated.

ALTERNATIVE TECHNIQUES

There are frequently alternative techniques or variations of the four steps outlined above that are available to the EP depending on the circumstance or the success of previous attempts. The next maneuver is attempted if the initial technique is unsuccessful. The authors use the following sequence of maneuvers to reduce an anterior shoulder dislocation: scapular manipulation with downward arm traction (i.e., disengagement and distraction), then external rotation (i.e., disengagement), followed by the Milch technique (i.e., disengagement), and finally traction-countertraction (i.e., distraction).[21,25-28] **Be aware of the limitations of the closed reduction technique.** No amount of distraction and no alternative technique will reduce the dislocation without taking the patient to the Operating Room if soft tissue is interposed in an interphalangeal finger dislocation.

FIGURE 99-6. Rotational deformity of the fifth digit.

ULTRASOUND

Ultrasound has become increasingly useful in the diagnosis and management of fractures and dislocations. Ultrasound has proven to be an effective means of improving success of hematoma and nerve blocks which can eliminate the need for procedural sedation.[5,12,13] Many dislocations and fractures can be reliably diagnosed using ultrasound. Ultrasound can be used to determine the success of reduction. This is useful when radiography or fluoroscopy is not available and the clinical examination is not definitive.[1-3,11] The radiograph remains a mainstay of management and should not be abandoned in lieu of ultrasound.[10]

ASSESSMENT

Assess the neurovascular status of the extremity to ensure that pulses are present, the extremity is perfusing, and nerve function has not been compromised before and after any reduction attempt. Assess the neurovascular status after any casting or splinting to the extremity. Postreduction radiographs assess the success of the reduction, are used to identify any fractures that may have been missed on the prereduction radiographs, and assess any complications from the reduction.

AFTERCARE

Immobilize the extremity as required for the specific fracture or dislocation. Place a record of the procedure in the chart. Include any complications and the method of reduction. Obtain a repeat plain radiograph in most cases to document the success of the reduction. Refer patients with reduced fractures and dislocations to an Orthopedic Surgeon. A fracture that is properly reduced and immobilized will occasionally be unstable and become displaced again. This requires operative fixation.

COMPLICATIONS

Complications of fracture and dislocation reduction are uncommon when properly performed. Complications can occur with proper techniques. These include converting a closed fracture to an open fracture, soft tissue trauma that produces a compartment syndrome, a reduction attempt that causes injury to the soft tissues making the fracture more unstable, producing a fracture during the dislocation reduction attempt by using excessive force, neurovascular injury/compromise due to bony laceration, or compression of a nerve or blood vessel.

SUMMARY

Displaced fractures and joint dislocations requiring reduction are commonly seen in the ED. The initial step in assessment is to determine the neurovascular status of the extremity. Emergent reduction is the rule when neurologic or vascular compromise is present with few exceptions. All dislocations require reduction. Not all displaced fractures require reduction. Become familiar with displaced fractures that benefit from reduction. The basic elements of fracture dislocation and joint dislocation are distraction, disengagement, reapposition, and release.

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100 Ultrasound for Fracture and Dislocation Identification and Management

Adrian Flores

INTRODUCTION

Point-of-care ultrasound (POCUS) for the evaluation of fractures and dislocations in the Emergency Department (ED) has been established in the literature and is not difficult to perform with minimal ultrasound (US) experience or brief focused training. Benefits include reduced cost, increased patient satisfaction, increased ED throughput, and decreased time to pain control.[1,2]

Acute fractures and dislocations are frequently seen in the ED. Radiographs can be normal and the next step is computed tomography (CT) or magnetic resonance imaging (MRI). These modalities are expensive, time consuming, and may not be available. US is an option for the initial diagnosis. US has advantages over radiographs including multiplanar imaging, ability to be performed at the bedside, and no ionizing radiation.

US as an initial imaging modality may obviate the need for a radiograph in a patient with a low suspicion for fracture or dislocation. It can be done at the bedside at the time of the physical examination. The patient does not have to wait for a radiograph, would not incur the cost of a radiograph, is more likely to be happy with their care than if they had received no imaging at all, and receives no ionizing radiation.[2] Patients with a fracture often go on to require a radiograph to better characterize a fracture. There are circumstances (e.g., sternal fractures) where US can be more sensitive and a radiograph is unlikely to change patient management.[3,4] Rapid evaluation with US allows the Emergency Physician to make a better and more specific choice to control pain. An intraarticular injection or a peripheral nerve block may be a better choice than intravenous (IV) opioids.

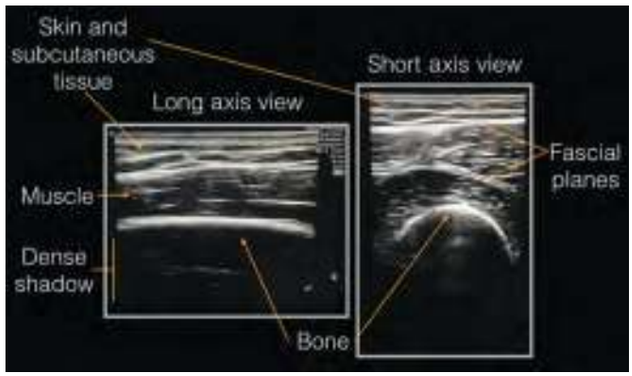


FIGURE 100-1. US of a typical bone.

This chapter covers the basics of US for long bone fractures and two of the most common dislocations in adults (e.g., the shoulder and elbow). Its use in other joint dislocations is similar.

ANATOMY AND PATHOPHYSIOLOGY

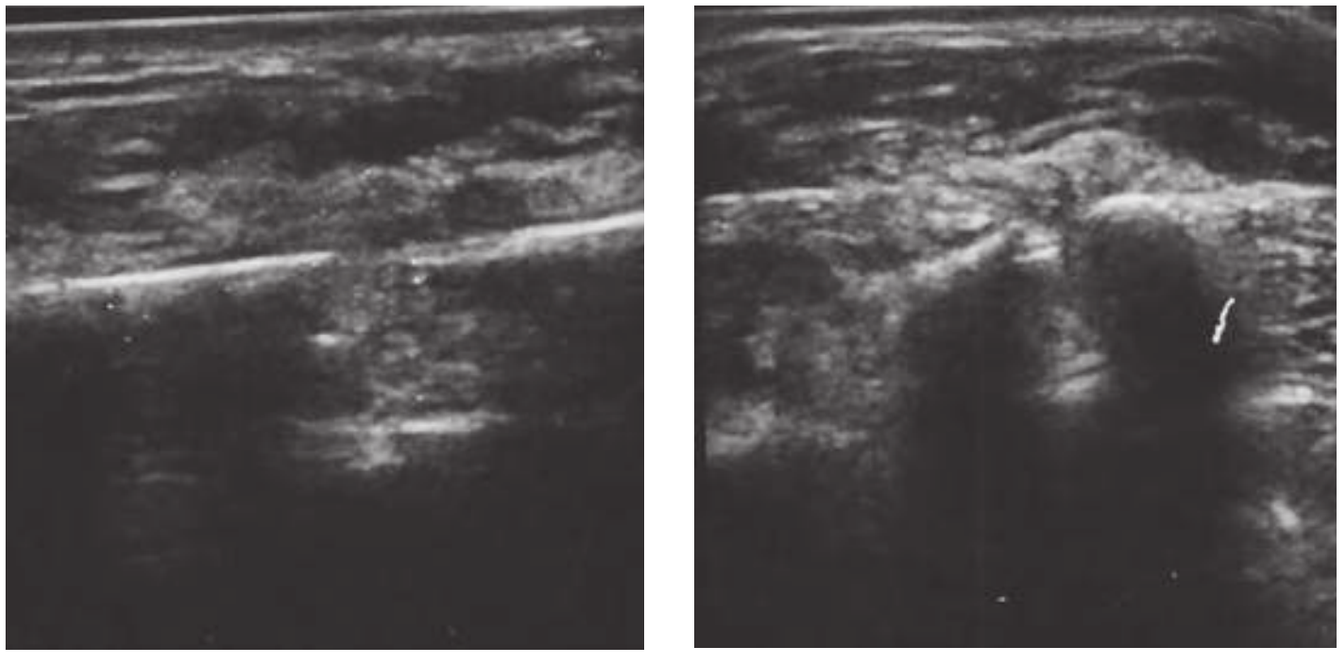
Bone is hyperechoic or bright white on the US screen and casts a dense shadow (**Figure 100-1**). The hypergenicity is from the differences in acoustic impedance between bone and surrounding soft tissues. The cortex is bright white and continuous, has posterior shadowing, and often shows a reverberation artifact. It is the brightest soft tissue structure seen on musculoskeletal US. This includes fascia, fat, muscle, subcutaneous tissue, tendons, and vessels. It can be brighter when the US transducer is directed transverse to the bone. The deep structures of the bone are not visible due to the high acoustic impedance of the cortex.

The rest of the musculoskeletal structures are visible adjacent and superficial to the bone. Fascial planes can be similarly bright as bone on US but do not produce a shadow. Air is visualized as bright and small scattered foci. It is not continuous like bone. Air casts a much dirtier shadow (e.g., various shades of gray and black) and not the uniform black shadow cast by bone.[5]

A bone fracture is visualized on US as a disruption of the hyperechoic bright white cortex of bone (Figure 100-2). This makes fracture identification easier in areas that are straight. A higher sensitivity and specificity are noted in these straight areas.[6] There is often edema, hematoma, hyperemia on Doppler, and periosteal thickening surrounding the fracture. A healing fracture can show the callus that is forming at the fracture site on US.

INDICATIONS

An US is indicated in patients with a potential fracture or joint dislocation.[7-17] It is helpful when reducing a fracture or joint dislocation.[16-20] An US is useful to evaluate a bone when a fracture is not seen on a radiograph but suspected on clinical examination.[21,22] This is especially true for rib fractures when the chest radiograph is negative. When indirect evidence of fractures (e.g., fat pads on radiographs of the elbow or tenderness in the snuffbox) appears on radiographs, the fractures can often be seen on US. US can identify stress fractures when a radiograph is negative and saves the cost and time of a bone scan or MRI. The portability of US allows the athlete to be diagnosed in the field where there is no accessibility to radiographs and can identify a contusion which allows return to play versus a fracture. US can be used for persistent pain after an injury with negative radiographs or without radiographs. It may visualize a callus before the radiograph.



A B

FIGURE 100-2. The cortical disruption seen in a fracture. ***A.*** Longitudinal view. ***B.*** Transverse view. (Used with permission from reference 6.)

US is ideal for close and overlapping small bones (e.g., the wrist and foot) to identify a fracture.

Evaluation of fractures and dislocations with US has the potential to take a much larger role, including as the primary diagnostic modality, in remote settings and low-resource settings. The portability of US allows for its use in remote military and wilderness settings where radiology equipment is too large and heavy. The World Health Organization (WHO) estimates that up to 75% of the population has no access to diagnostic imaging.[23] The low cost, portability, and bedside interpretation of US could potentially have a large impact.

CONTRAINDICATIONS

A contraindication to performing an US examination is an open fracture. Relative contraindications are issues that limit a thorough evaluation. Pain and tenderness are usually the only factors that limit an US examination. These can be overcome with the application of analgesics before the US examination proceeds.

EQUIPMENT

A brief description of the equipment required is provided in this section. A complete description is contained in Chapter 6, Basic Principles of Ultrasonography.

ULTRASOUND MACHINE

Most bedside US machines are portable machines on a cart. Many handheld devices are now available and may be suitable for evaluating fractures and dislocations.[24,25] The machines vary greatly but have three basic buttons to obtain good images. The power button can sometimes be in a place that is not obvious or even hidden. The gain adjusts the brightness of the image. The depth determines how deep into the tissue the machine images. It allows the sonologist to bring the object into the middle of the monitor screen.

TRANSDUCER

The linear, high-frequency transducer (e.g., 6 to 15 MHz) is appropriate for most musculoskeletal applications. It is also known as the vascular transducer due to its common use for evaluation of vasculature for venous or arterial access. This transducer provides the best resolution for superficial structures. A lower frequency transducer (e.g., a phased array of 1 to 5 MHz or a curvilinear of 2 to 5 MHz) might be a better choice in circumstances requiring images deeper in the body (e.g., a femur in a patient with obesity or thick musculature).

ULTRASOUND GEL

Commercial US gels are widely available. Single-use sterile lubricant packets can be used in situations where there may be concern for introducing an infection. Locally made US gels have been proposed as an alternative in remote settings.[26]

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain a verbal informed consent for the procedure. Raise the gurney to a comfortable height and lower the rail to be in a comfortable ergonomic position for scanning. The examiner stands on the patient's right side by convention with the US machine in front and near the patient's head. Constraints (e.g., room size and room configuration) sometimes do not allow for this positioning, and the Emergency Physician should use their best judgment. Warn the patient that the US gel might feel cold. Patients are often surprised by how cold the US gel feels despite it being at stored room temperature.

Have a clean towel available to wipe the US gel off the patient after obtaining images. Make the appropriate preparations to disinfect the US transducer after scanning.

FIGURE 100-3. Proximal phalanx fracture of the fifth finger. ***A.*** Radiograph. ***B.*** US showing the cortical disruption (*arrow*). (Used with permission from reference 28.)

TECHNIQUE FOR FRACTURES

Evaluation of fractures is straightforward. Evaluate the bone in two planes at 90° to each other. This usually comprises a transverse (i.e., short axis or cross-section) view and a longitudinal (i.e., long axis) view. Place the transducer with the marker pointed to the patient's right for short axis views. Place the transducer with the marker toward the head for long axis views. Start in the short axis and identify the bone of interest. Rotate the transducer 90° to the long axis. Scan the bone from the distal to proximal portion. Pay special attention to the area of maximal tenderness.[23] **A fracture is identified by a cortical disruption or irregularity of the cortical line (e.g., depressions, displacements, or gaps).**[6,27]

This same technique can be used for all bones.[14,28-38] Scanning small bones of the hand (**Figure 100-3**), wrist (**Figures 100-4, 100-5, and 100-6**), and foot (**Figure 100-7**) through a 100 mL bag

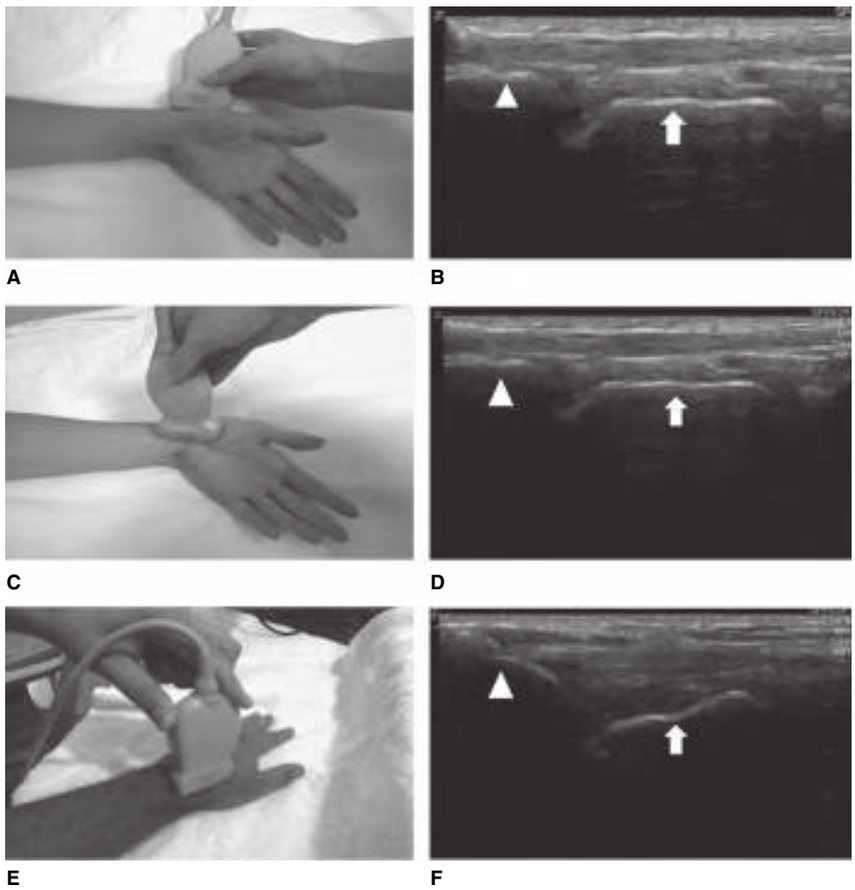
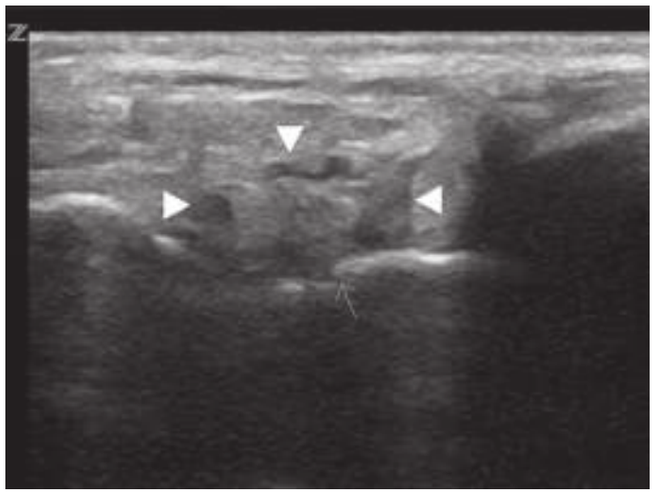


FIGURE 100-4. Positioning of the transducer and normal US findings for the scaphoid. The cortex of the distal radius (arrowheads) and scaphoid (arrows) is noted in the US images. ***A.*** Longitudinal position over the lateral wrist with the hand ulnar deviated. ***B.*** Longitudinal view US. ***C.*** Palmar positioning with the hand ulnar deviated. ***D.*** Palmar US view. ***E.*** Dorsal positioning with the hand neutral. ***F.*** Dorsal US view. (Used with permission from reference 29.)

A



B

FIGURE 100-5. A scaphoid fracture. ***A.*** Posteroanterior radiograph. ***B.*** Longitudinal US view of the wrist with a cortical disruption (*arrow*) and an overlying hematoma (*arrowheads*). (Used with permission from reference 29.)

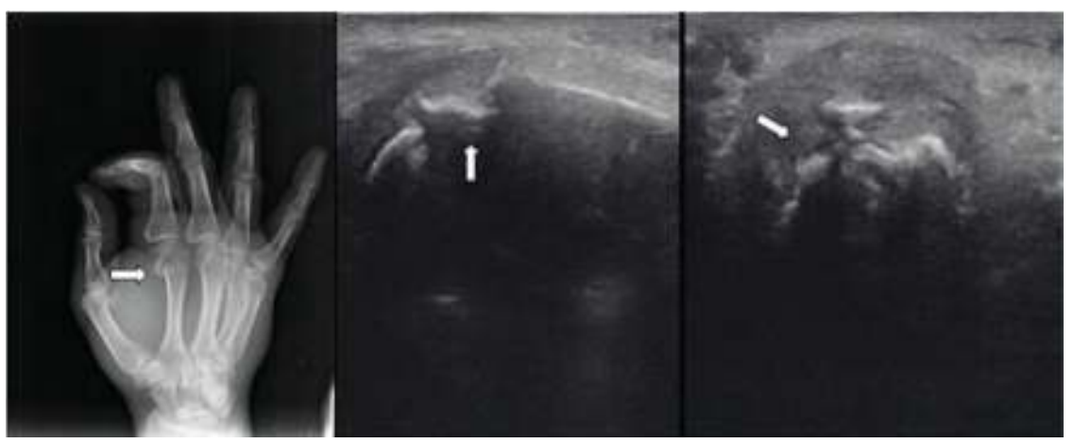
of normal saline or submerged in a water bath can facilitate obtaining a good image. The humerus, radius (**Figures 100-8, 100-9, and 100-10**), ulna (**Figure 100-11**), femur (**Figure 100-12**), tibia, and fibula (**Figure 100-13**) are easy long bones to evaluate using US. The clavicle (**Figure 100-14**), patella (**Figure 100-15**), and sternum (**Figure 100-16**) are easy non-long bones to evaluate because of their superficial location and the limited area needed to be scanned. Evaluation of the skull, mandible, ribs (**Figure 100-17**), metacarpals, phalanges, and metatarsals with confidence and accuracy takes more focused teaching and practice.[39]

A few fractures deserve special mention. Rib fractures are commonly suspected. Radiographs are not sensitive in diagnosing a costal cartilage fracture, a costochondral junction fracture, or a minimally displaced rib fracture. US can diagnose these fractures.[40,41] The transducer can move along the rib as it changes direction.[33] It is important to diagnose rib fractures for the patient's knowledge, expectations, pain management, and missing work. It can affect the discharge instructions to prevent atelectasis and pneumonia. US can be used to evaluate subacute traumatic injuries to evaluate healing fractures (**Figure 100-18**).[33] Real-time US can help in the reduction of fractures.[9] The use of US in children can save time, prevent them from leaving the department, and help build rapport.[15,42] US can identify fractures not seen using radiographs (i.e., occult fractures).[43,44]

TECHNIQUE FOR DISLOCATIONS

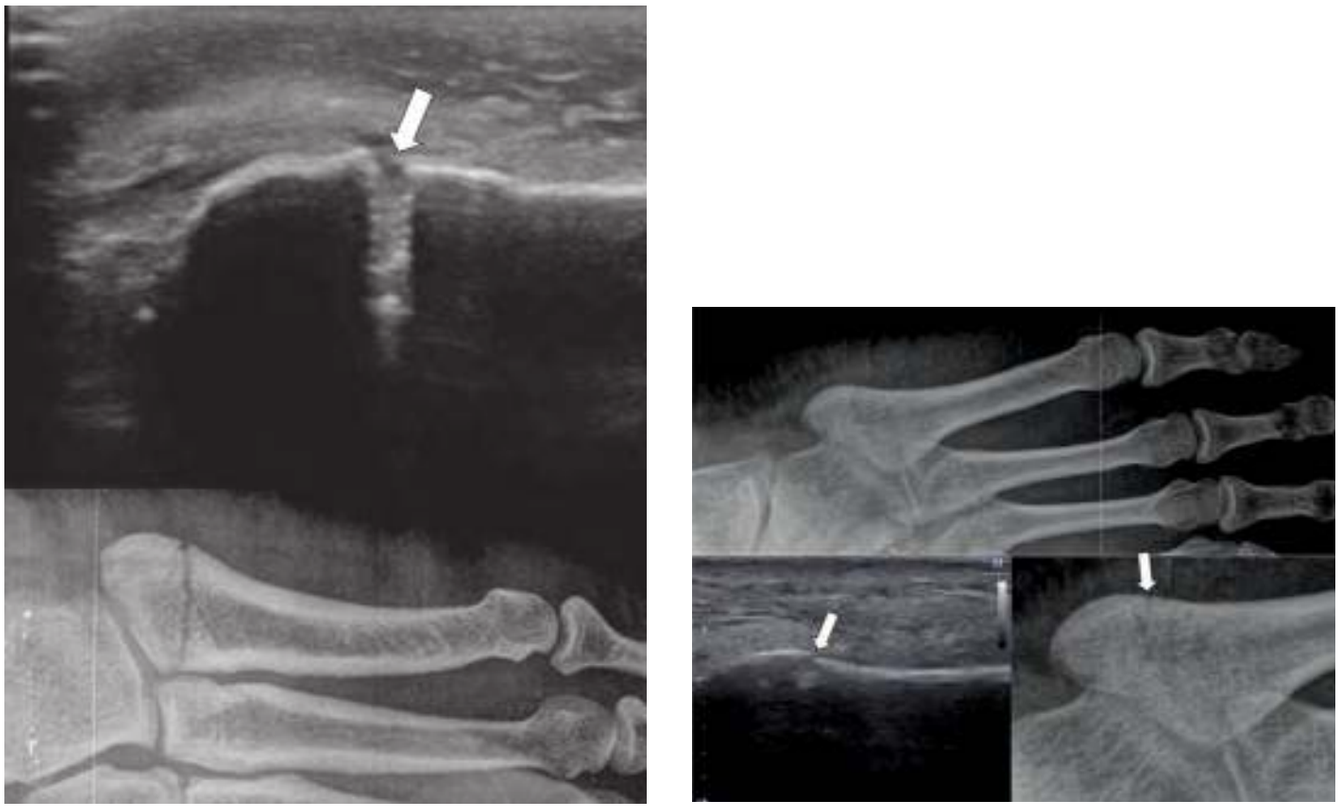
SHOULDER

The shoulder is the most commonly dislocated major joint in adults. Place the patient on the edge of the bed with their arm adducted and the elbow flexed to 90°. Stand behind the patient and position the US machine in front of the patient. Place the US monitor screen toward the examiner. Use the curvilinear transducer. Place it with the marker facing to the patient's left rather than the right because



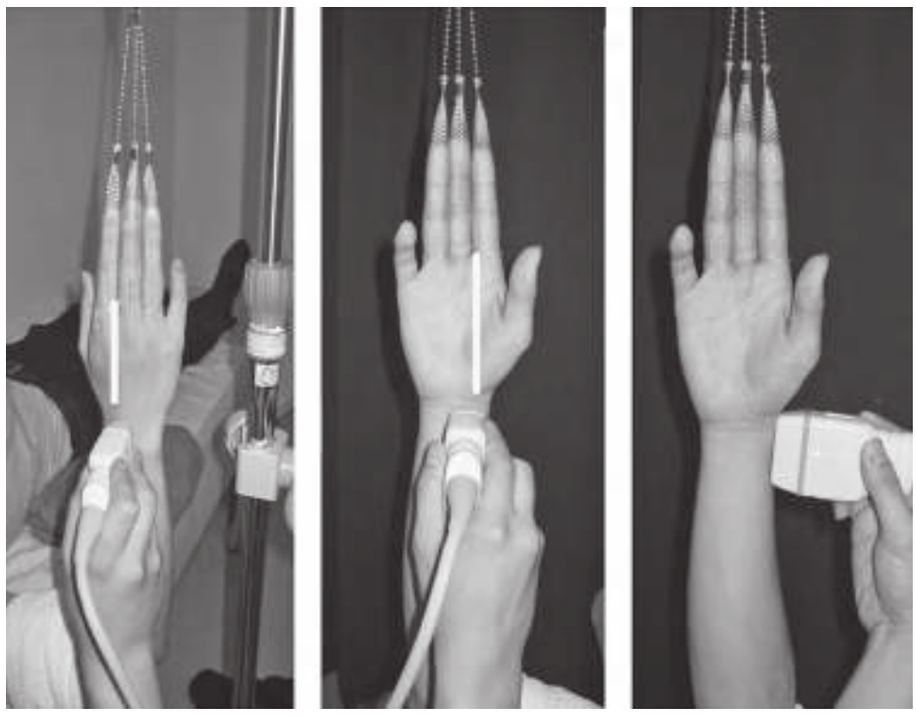
A B C

FIGURE 100-6. A metacarpal fracture. ***A.*** Oblique radiograph. ***B.*** Longitudinal US view. ***C.*** Transverse US view. (Used with permission from reference 14.)



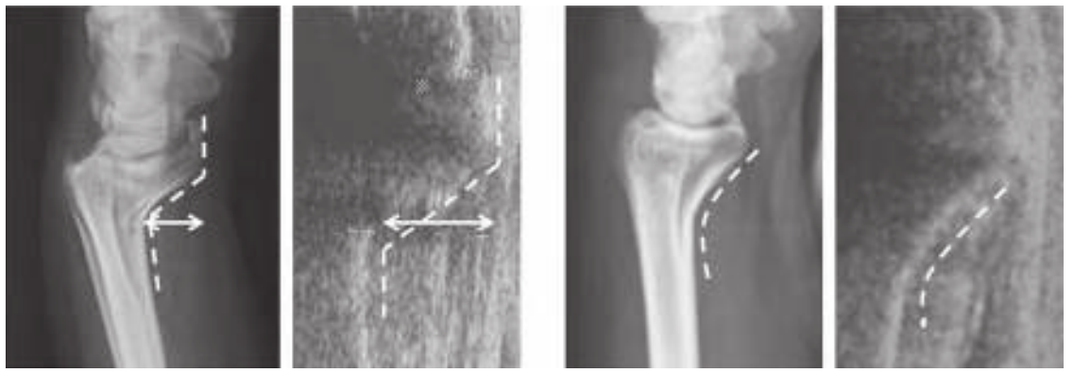
A B

FIGURE 100-7. A metatarsal fracture. ***A.*** A Jones fracture on radiography is seen on the US as a cortical disruption (*arrow*). ***B.*** An avulsion fracture on the radiograph is seen on the US as cortical disruptions (*arrows*). (Used with permission from reference 30.)



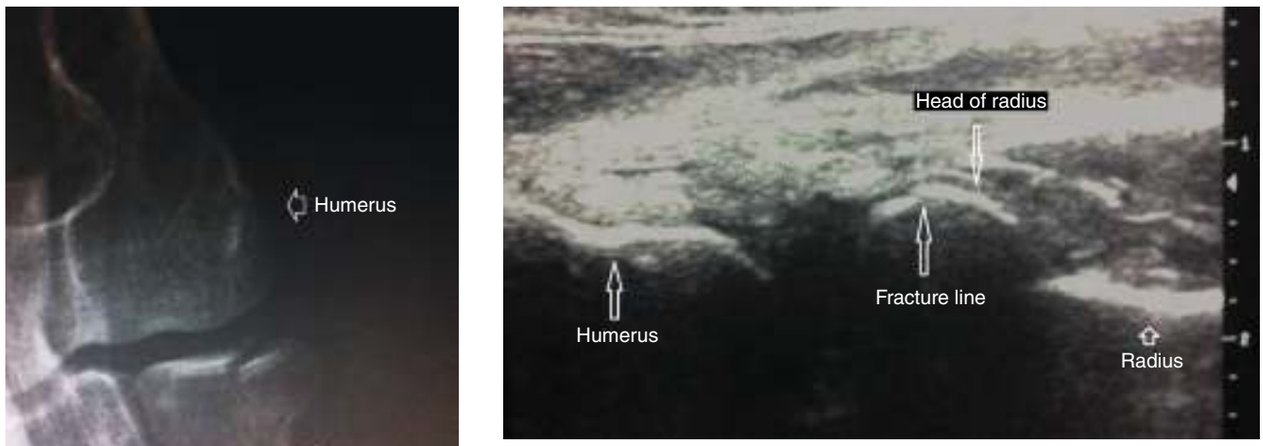
A B C

FIGURE 100-8. Positioning of the transducer to evaluate a distal radius fracture. ***A.*** Dorsal positioning between the index and middle fingers. ***B.*** Palmar positioning. ***C.*** Radial positioning. (Used with permission from reference 31.)



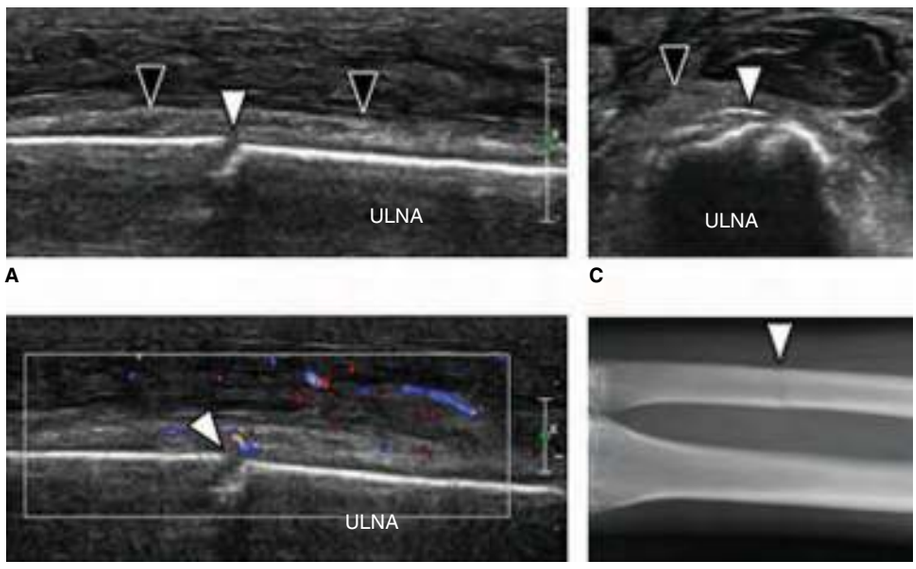
A B

FIGURE 100-9. A distal radius fracture. ***A.*** Prereduction view with the cortex (*dotted line*) displaced (*arrow*). ***B.*** Postreduction view shows alignment over the volar cortex. (Used with permission from reference 31.)



A B

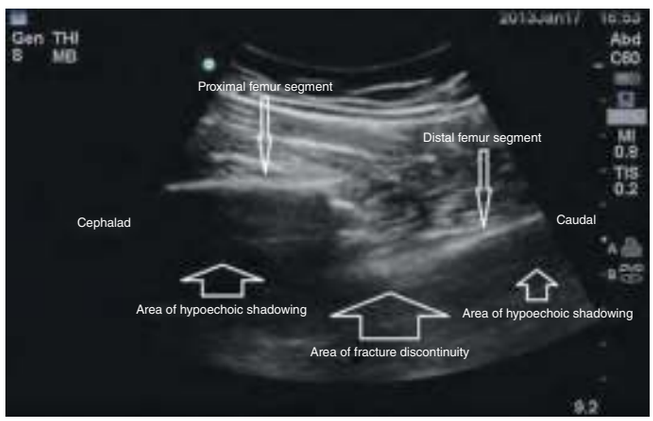
FIGURE 100-10. A radial head fracture. ***A.*** Radiograph. ***B.*** US view. (Used with permission from reference 32.)



A C B D

FIGURE 100-11. A midshaft ulna fracture. The *white arrowheads* are cortical disruptions, and the *black arrowheads* are thickenings of the periosteum. ***A.*** Longitudinal US view. ***B.*** Longitudinal US view with Doppler. ***C.*** Transverse US view. ***D.*** Anteroposterior radiograph. (Used with permission from reference 33.)

A



B

FIGURE 100-12. A femur fracture. ***A.*** Anteroposterior radiograph. ***B.*** US view. (Used with permission from reference 34.)

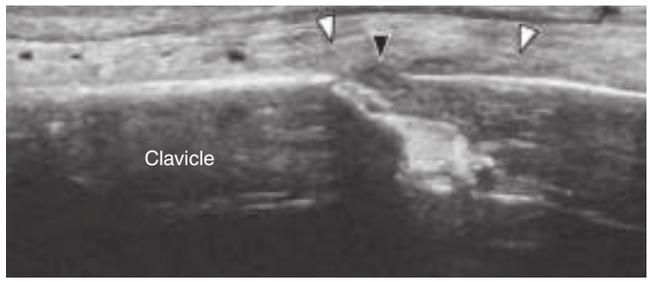


FIGURE 100-14. Longitudinal US view of a clavicle fracture. Periosteal thickening (*white arrowheads*) and edema (*black arrowheads*) are noted. (Used with permission from reference 33.)

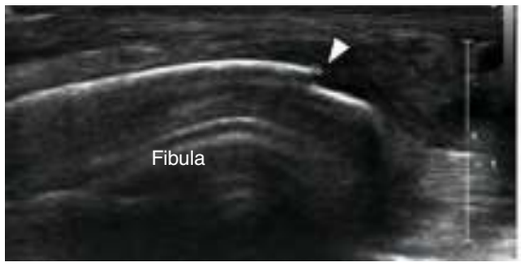
you are behind them and scanning posterior rather than anterior.[45,46] Start scanning the posterior shoulder just inferior to the scapular spine (**Figure 100-19**). Slide laterally and identify the glenoid fossa and humeral head (**Figure 100-19**). The humeral head is displaced at least 20 mm from the glenoid fossa toward the far-field or bottom of the screen in an anterior dislocation (**Figure 100-20**). The humeral head will be displaced toward the near-field or top of the screen in a posterior dislocation (**Figure 100-20**).[47,48]

US can be used to confirm the reduction in addition to the initial evaluation. The humeral head will be aligned with the glenoid fossa, and the shoulder should freely internally and externally rotate on the screen. This is especially helpful when using procedural sedation where a rapid evaluation with US can be performed much faster than radiographs. This limits the time for patient sedation and prevents the need for resedation if the radiograph shows a persistent dislocation.[47]

This chapter does not cover shoulder reduction techniques (Chapter 102). It is important to note that US is extremely helpful in ensuring entry into the joint for intraarticular lidocaine injections. The intraarticular instillation of local anesthetics has been shown to be as effective as procedural sedation for analgesia for the reduction of anterior shoulder dislocations.[49,50]

ELBOW

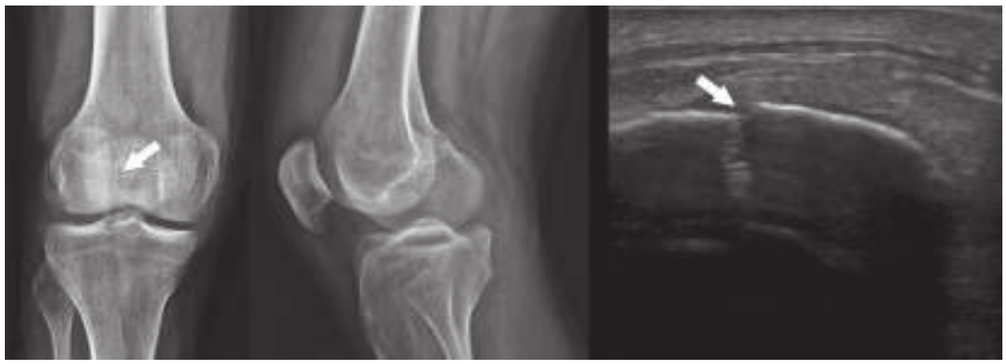
The proximal ulna often dislocates posterior to the distal humerus. Place the patient with their arm in 90° of flexion (**Figure 100-21**).[51] Place the US transducer on the posterior aspect of the distal humerus in a longitudinal plane with the marker toward the patient's head (**Figure 100-21*A***). Slide the transducer caudally until the olecranon is visualized. The olecranon is aligned with the distal humerus in a normal elbow (**Figure 100-22**). Turn the transducer 90° and repeat the US scan (**Figure 100-21*B***). The olecranon is displaced posteriorly from the distal humerus or displaced toward the top of US monitor screen in a dislocation (**Figure 100-23**).[52]



A

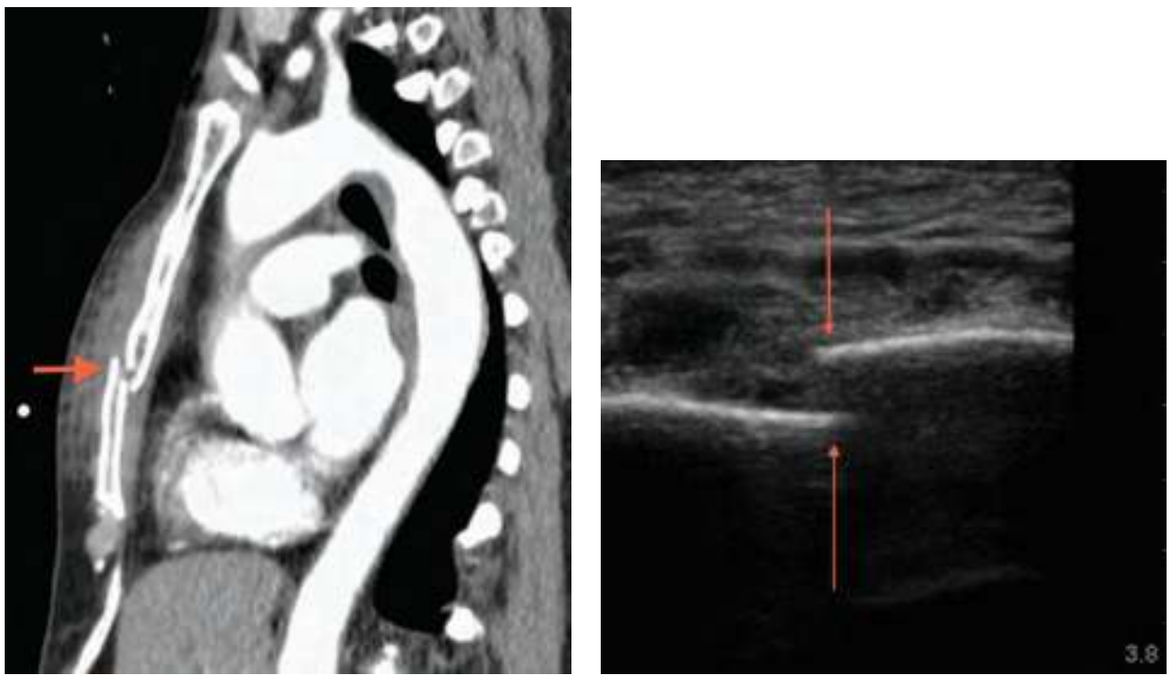
B

FIGURE 100-13. A fibula fracture. ***A.*** Longitudinal US view. ***B.*** Anteroposterior radiograph. (Used with permission from reference 33.)



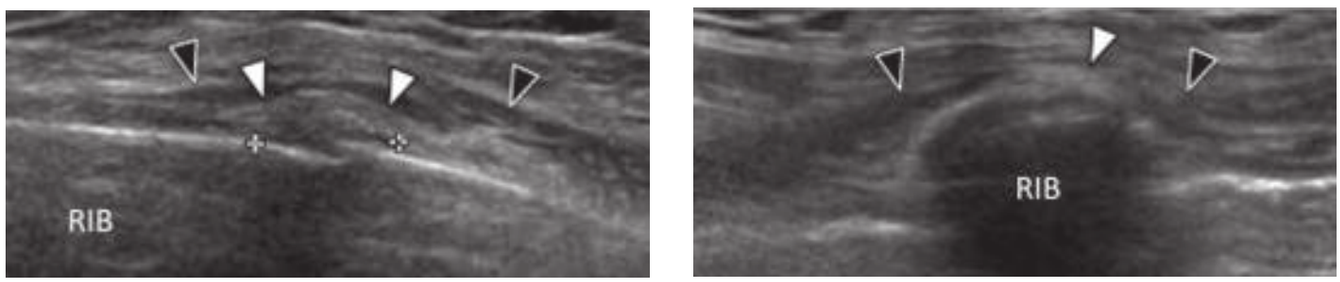
A B C

FIGURE 100-15. A patella fracture. ***A.*** Anteroposterior radiograph. ***B.*** Lateral radiograph. ***C.*** Longitudinal US view with the *arrow* pointing toward the fracture. (Used with permission from reference 35.)



A B

FIGURE 100-16. A sternal fracture. The *red arrows* identify the fracture. ***A.*** CT scan. ***B.*** US view. (Used with permission from reference 36.)



A B

FIGURE 100-17. A rib fracture. The fracture is seen as a cortical disruption (between calipers) with elevation and thickening of the periosteum (*white arrowheads*) and adjacent soft tissue edema (*black arrowheads*). ***A.*** Longitudinal US view. ***B.*** Transverse US view. (Used with permission from reference 17.)

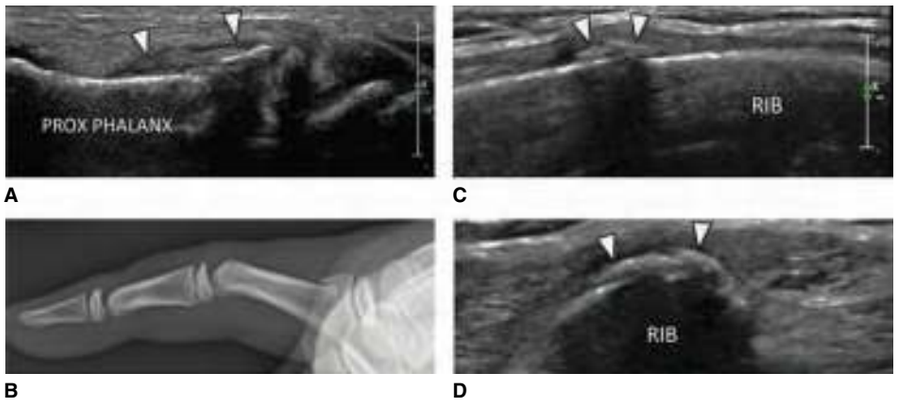


FIGURE 100-18. Bone callus formation (*arrowheads*). ***A.*** Longitudinal US view of a 13 day old proximal phalanx fracture. ***B.*** Lateral radiograph of the proximal phalanx. ***C.*** Longitudinal US view of a 6 week old rib fracture. ***D.*** Transverse US view of a 6 week old rib fracture. (Used with permission from reference 33.)

ASSESSMENT

Review and interpret the US scans. Physicians with a minimal US background and short focused training sessions can perform many US scans.[23,25] A systematic review on the diagnostic accuracy of bedside US in the ED found an overall 85% to 100% sensitivity and 73% to 100% specificity.[1] The best option is to determine the scan inconclusive when there is uncertainty regarding the diagnosis or interpretation of the images. The use of US allows the Emergency Physician to continuously manipulate the fracture until it is reduced without the delays of obtaining radiographs and having to sedate the patient for a long time (**Figure 100-24**).

AFTERCARE

No specific aftercare is needed for the patient. Wipe the excess US gel off the patient. Wipe down and clean any blood, debris, and dirt from the US transducer. Disinfect the US transducer according to manufacturer directions and hospital policy.

COMPLICATIONS

US is operator dependent and the images can be misinterpreted. It depends on an individual's experience, training, and familiarity with the scan being performed. There are no other complications from obtaining a US scan of the affected area. Pain can be overcome by not pushing as hard with the transducer or providing IV analgesia before the scan.

Ensure the transducer is clean before and after scanning. A clean transducer before scanning will not cross-contaminate the patient. A clean transducer after scanning prevents the next patient from being contaminated. The US machine and transducer are used by multiple people in the ED. It is recommended to wipe the transducer with disinfecting wipes before and after scanning. Use wipes approved by the manufacturer and hospital infection control.

SUMMARY

US is as sensitive as radiographs for most long bone fractures and more sensitive for some fractures (e.g., sternum). Novice sonographers can learn these scans and apply them in clinical practice to

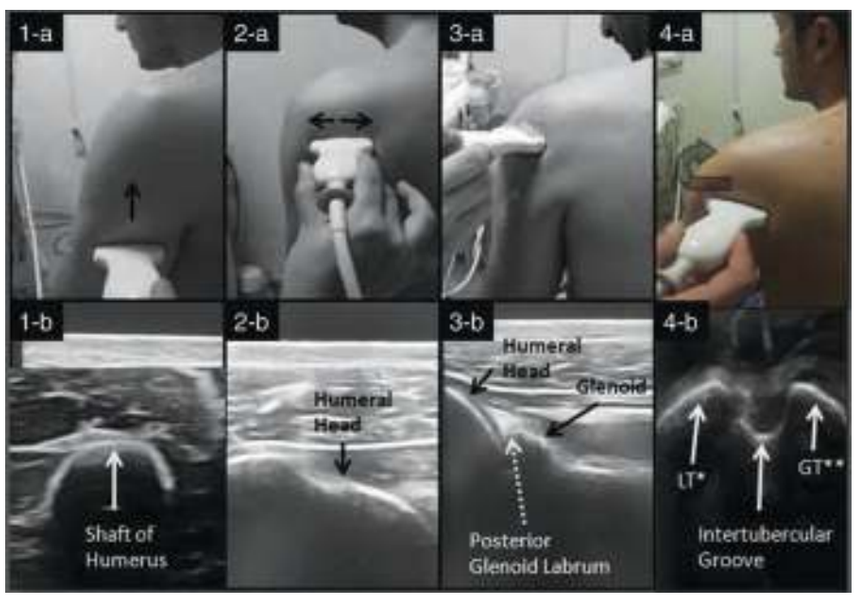


FIGURE 100-19. The steps in performing a shoulder US. View the humeral shaft (1-a and 1-b). Slide the transducer to view the humeral head (2-a and 2-b). Slide the transducer toward the posterior shoulder to view the glenohumeral joint and posterior glenoid labrum (3-a and 3-b). Slide the transducer anteriorly to view the humeral head, greater tuberosity (GT), lesser tuberosity (LT), and intertubercular groove (4-a and 4-b). (Used with permission from reference 46.)

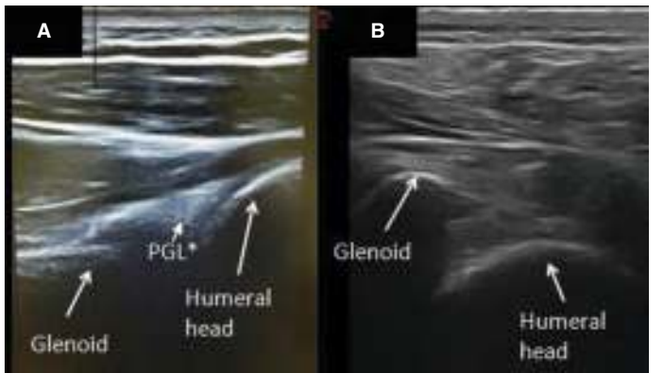


FIGURE 100-20. Shoulder dislocation on US. ***A.*** Anterior dislocation. PGL, posterior glenoid labrum. ***B.*** Posterior dislocation. (Used with permission from reference 46.)

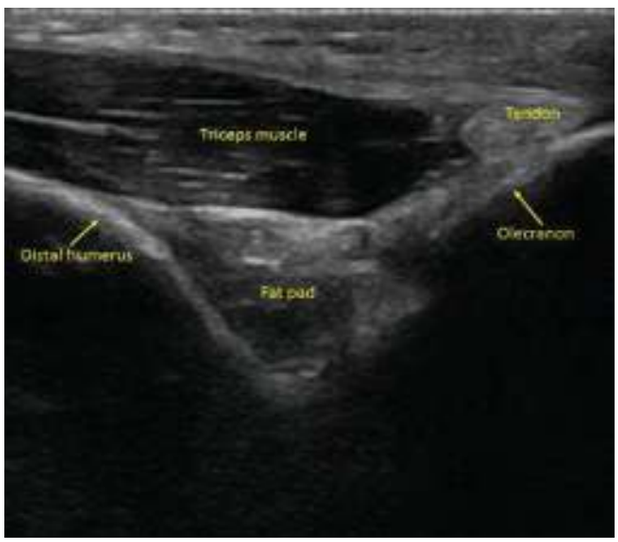
FIGURE 100-22. The elbow US anatomy.

A

B

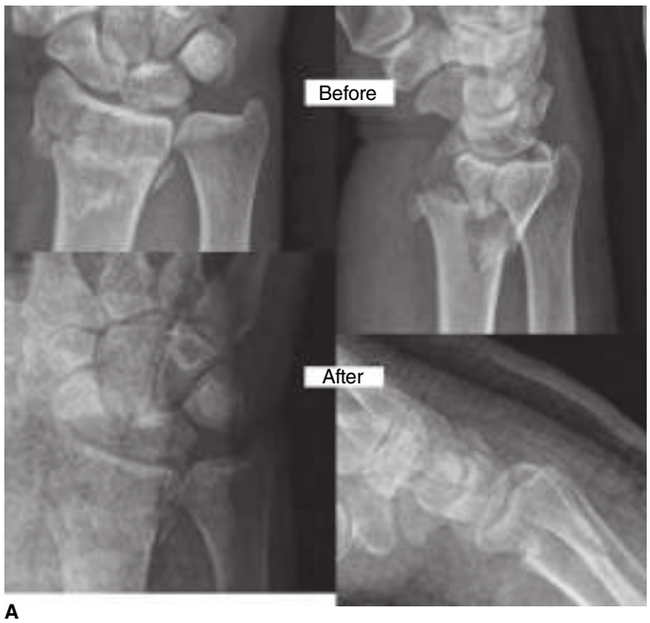
FIGURE 100-21. The placement of the transducer for the elbow evaluation. (Used with permission from reference 52.)

A

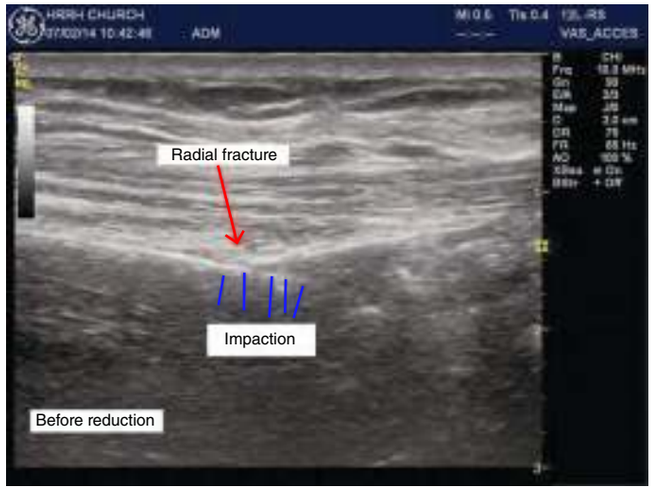


B

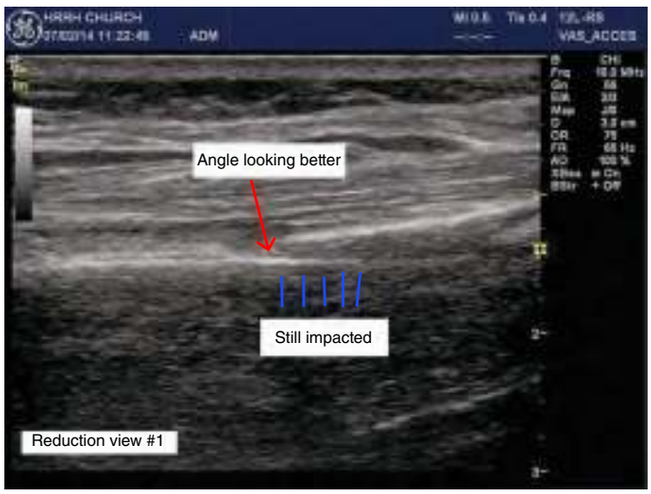
FIGURE 100-23. An US of a posterior elbow dislocation. ***A.*** The dislocation. ***B.*** After reduction. (Photos courtesy of Srikar Adhikari, MD.)



A



B



C

D

E

FIGURE 100-24. US reduction of a Colles fracture. ***A.*** Radiograph. ***B.*** Initial US. ***C.*** First reduction. ***D.*** Second reduction. ***E.*** Third and final reduction. (Photos courtesy of Lloyd Gordon, MD.)

improve time to pain control, choose a better pain control modality, reduce the number of unnecessary radiographs, decrease patient length of stay, and improve patient satisfaction. US can be used by the Emergency Physician in the ED or low-resource settings.[53]

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101 Sternoclavicular Joint Dislocation Reduction

Michael D. Burg

INTRODUCTION

Sternoclavicular dislocations are uncommon, accounting for less than 3% of shoulder girdle injuries.[1] The medial clavicle may be displaced anteriorly, posteriorly, or rarely superiorly (**Figure 101-1**).[2] Bipolar clavicular dislocations (i.e., simultaneous dislocation of both clavicular articulations) also occur rarely.[3] Anterior dislocations are considerably more common than posterior dislocations. However, posterior dislocations deserve more attention due to the far higher incidence of associated complications.[1] Also, since the medial clavicular physis closes relatively late in life, what appears to be a posterior sternoclavicular dislocation in young patients may actually be a medial epiphyseal disruption with posterior displacement.[4]

Sternoclavicular dislocations result from direct trauma to the sternoclavicular joint or to the glenohumeral joint with the force directed toward the sternoclavicular joint. **Tremendous force is usually required to disrupt the sternoclavicular joint.** Common mechanisms of injury include motor vehicle collisions and contact sports.[3] Falls, minor trauma, or various medical conditions without trauma result in sternoclavicular dislocations less commonly.[5-7]

Anterior dislocations are often due to indirect forces transmitted through the anteromedial shoulder. As the shoulder is externally compressed and rolled backward, the lateral clavicle is pulled back and down beyond its limit of motion. The first rib acts as a fulcrum to spring the sternal end of the clavicle anteriorly from its articulation (**Figure 101-2**).[1,8,9]

Posterior dislocations may be due to direct or indirect forces.[1,10-13] With indirect trauma, the shoulder is externally compressed and rolled forward from a force applied posterolaterally to the shoulder. The costoclavicular ligament acts as a fulcrum that produces displacement of the sternal end of the clavicle posteriorly from its articulation (**Figure 101-3**).[11] Less commonly, a posterior dislocation may be due to a direct blow to the anteromedial clavicle.[12]

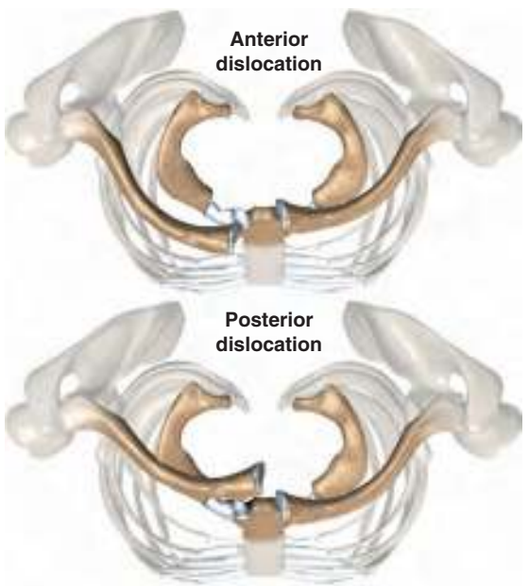

FIGURE 101-1. Superior view of the position of the medial end of the clavicle in a sternoclavicular joint dislocation. (Used with permission from eOrthopod.org.)

FIGURE 101-2. Clinical photo of an anterior sternoclavicular joint dislocation. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill, 2016. Photo contributor: R. Jason Thurman, MD.)

Spontaneous and atraumatic sternoclavicular subluxations and dislocations do occur, often in those with laxity in other joints or in association with inflammatory conditions.[7,8,14-16] Both anterior and posterior subluxations have been described, as have atraumatic anterior and posterior dislocations.[14-16] These are usually seen in females less than 20 years of age with sternoclavicular joint laxity. The clavicle dislocates anteriorly during abduction or flexion of the arm to the overhead position and reduces spontaneously when the arm is returned to the side.

ANATOMY AND PATHOPHYSIOLOGY

Both surfaces of the maximally mobile sternoclavicular joint are covered by fibrocartilage (**Figure 101-4**). The intraarticular disk ligament divides the joint into two separate compartments, each of which is lined with synovium.[17] This joint is freely mobile and functions like a ball-and-socket joint, with motion (including rotation) in almost all planes.[1,18] This includes 30° to 35° of upward elevation, 35° of combined forward-backward movement, and 40° to 45° of rotation about its long axis.[18] Less than half of the medial clavicle articulates with the sternum's upper angle, giving the sternoclavicular joint the least bony stability of any major joint.[1] Given this amount of joint incongruity, it is surprising that sternoclavicular joint dislocations are uncommon. However, the joint's stability is

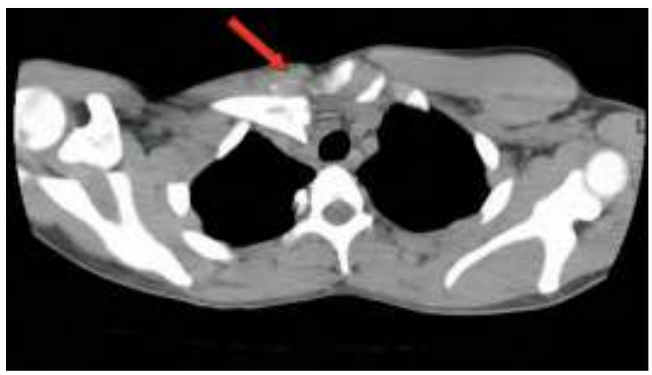

FIGURE 101-3. CT scan of a posterior sternoclavicular joint dislocation. (Used with permission from Tintinalli JE, et al: *Tintinalli's Emergency Medicine*: *A Comprehensive Study Guide*, 8th ed. New York: McGraw-Hill; 2016.)

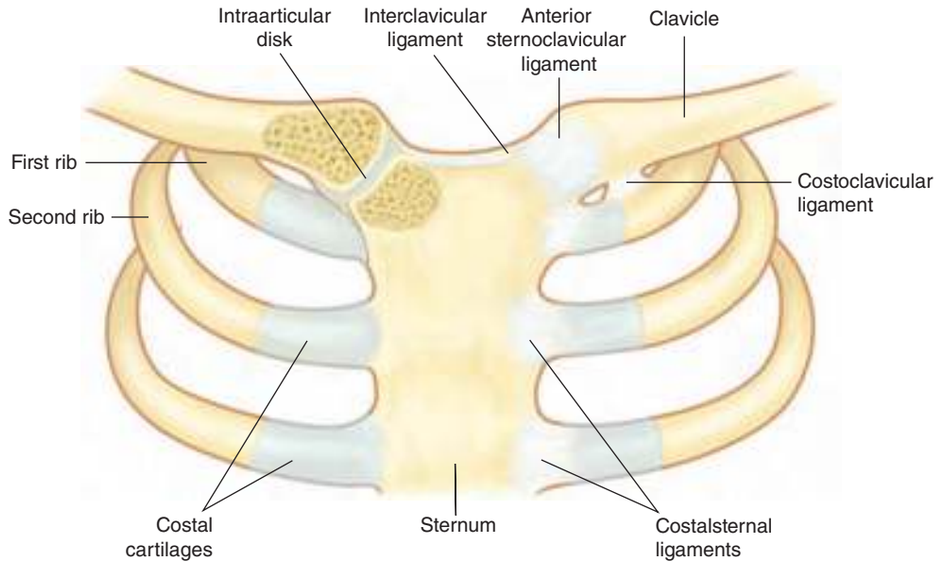

FIGURE 101-4. Anatomy of the sternoclavicular joint and surrounding structures. The extremely strong posterior sternoclavicular ligament lies immediately behind the sternoclavicular joint and is not shown.

due to strong surrounding structures (**Figure 101-4**), including the intraarticular disk, the extraarticular costoclavicular ligament, the anterior and posterior sternoclavicular ligaments, and the interclavicular ligament.[17]

Numerous vital structures are directly posterior to the sternoclavicular joint including the trachea, esophagus, lungs, and great vessels (Figure 101-5). Injuries to these structures frequently occur with posterior sternoclavicular joint fractures and dislocations due to this proximity, the thoracic inlet's small size, and the forces involved.

The medial clavicular epiphysis is the last long bone epiphysis to appear, usually ossifying by 18 to 20 years of age, but occasionally not until the age of 25.[1,19] It is also the last to fuse.[1,19] This epiphysis is difficult, or frankly impossible, to see on plain radiographs. Many injuries in patients less than 25 years of age initially thought to be true sternoclavicular joint dislocations are actually Salter-Harris I or II epiphyseal injuries.[1,20-24]

Sternoclavicular joint dislocation diagnosis may be difficult and/or delayed, particularly in those with multiple injuries.[25,26] Several case reports exist detailing diagnostic delays, occasionally with catastrophic outcomes.[27-29]

Severe local pain that increases with movement of the ipsilateral arm may signal a sternoclavicular joint disruption. Edema, ecchymosis, crepitus, and tenderness may be present in the region overlying the sternoclavicular joint. Those with sternoclavicular joint dislocations usually hold their affected arm adducted across the trunk. A head tilt toward the affected side may be seen in an attempt to relieve the pain caused by traction of the sternocleidomastoid muscle on the medial clavicle.[30]

With anterior dislocations, the clavicle's medial end may be palpable anterior to the sternum (**Figure 101-2**). It may be either fixed or mobile. With posterior dislocations, a depression may be visible or a hollow palpable over the region of the sternoclavicular joint.[31] Edema may obscure these physical findings. The affected shoulder may be held forward. The affected shoulder may not lie flat against the bed when the patient is supine.

Additional signs and symptoms associated with posterior sternoclavicular joint dislocations may be due to mediastinal injuries.[1,31-34] **Perform a careful and complete physical examination. Associated injuries are common.** Tracheal or esophageal compression may result in cyanosis, dyspnea, or dysphagia. Ipsilateral arm circulation may be reduced if the subclavian artery is compressed or otherwise damaged. Venous congestion of the upper extremity or neck can result from compression of, or injury to, the subclavian or jugular veins. Shock may be due to compression or injury of the great vessels. Upper extremity paresthesias may result from brachial plexus injuries. Voice changes may be due to compression of the recurrent laryngeal nerve. Tracheal or lung injuries with resultant pneumothoraces can occur.

Do not rely on clinical findings gleaned from observation and palpation to distinguish between anterior and posterior sternoclavicular joint dislocations.[1] Appropriate radiologic studies are recommended prior to treatment decisions. Routine radiographs that include the sternoclavicular joint are difficult to interpret due to overlapping structures. Several different radiographic projections are reported to improve the ability to detect sternoclavicular joint asymmetry.[1,11,12,35,36] Computed tomography (CT) is the optimal imaging modality for the sternoclavicular joint (**Figure 101-3**). It should always be performed if the diagnosis is uncertain.[1,12,13] Ultrasound may also provide useful diagnostic and therapeutic information.[37-39]

With posterior sternoclavicular joint dislocations, consider appropriate studies to evaluate for injuries to neighboring structures. A chest radiograph may reveal mediastinal widening, pneumomediastinum, or a pneumothorax. An intravenous contrast-enhanced chest CT is definitive (**Figure 101-3**). It will reveal the relationship of the clavicles to the great vessels, esophagus, and trachea. The CT may also demonstrate compression of, or injury to, these structures as well as other findings. Angiography, venography, and Doppler studies can further investigate potential vascular injuries. The esophagus may be evaluated with esophagoscopy and/or an esophagram. Bronchoscopy is indicated if a tracheal or bronchial injury is suspected.

INDICATIONS

It was generally held that closed reduction should be attempted on all acute traumatic anterior and posterior sternoclavicular joint dislocations.[1,15] A case series describes successful closed reduction of an anterior sternoclavicular joint dislocation 10 days after injury.[11] Successful closed reduction of posterior sternoclavicular joint dislocations has been reported up to 5 days after injury.[37] However, in at least one of these late-presenting cases, reduction was done in the Operating Room with ultrasound guidance.[37]

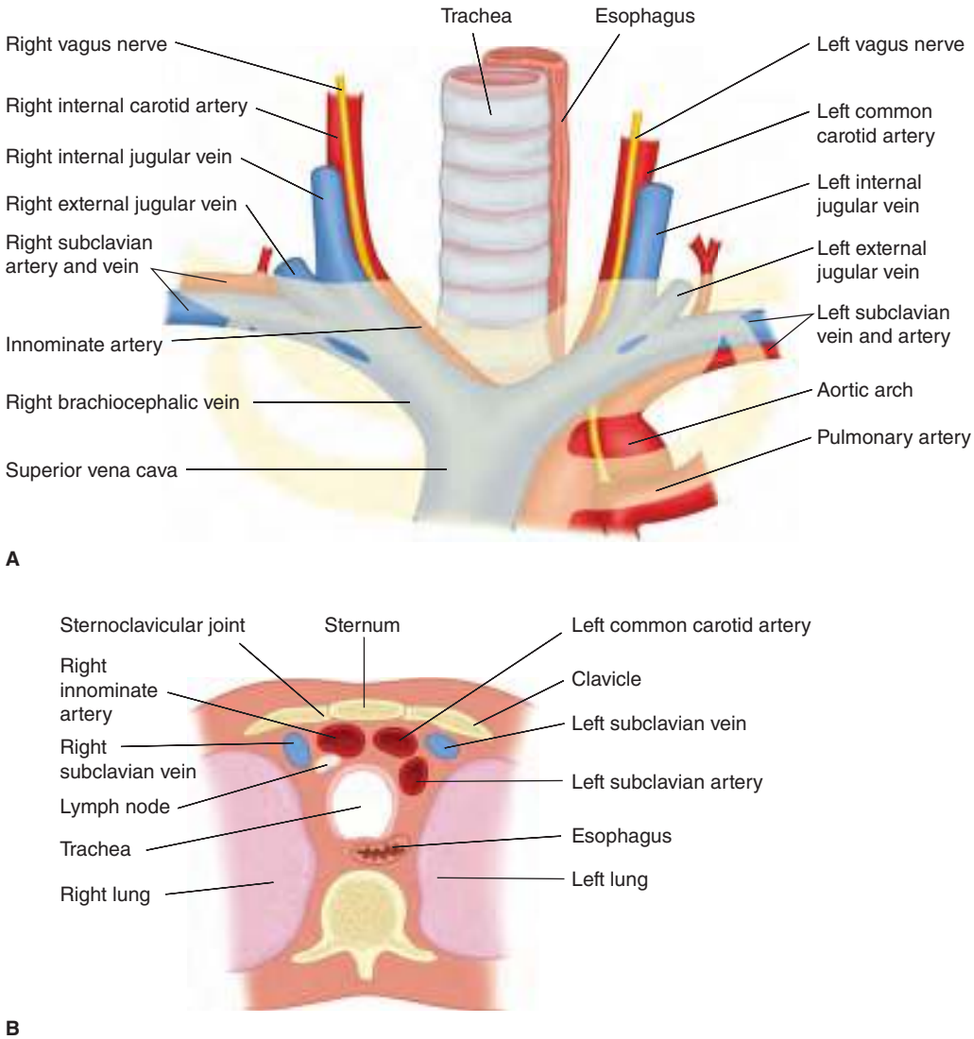

FIGURE 101-5. Anatomic relationships of structures to the sternoclavicular joint. ***A.*** Anteroposterior view. ***B.*** Cross-section through the level of the sternoclavicular joint.

Early consultation with an Orthopedic Surgeon is recommended for the less common, potentially more serious, and difficult to manage posterior sternoclavicular joint dislocations. Prior to the reduction, carefully assess the patient's airway, breathing, circulation, and neurologic status. Obtain appropriate diagnostic imaging studies as described above. Obtain immediate Thoracic Surgery consultation for patients with intrathoracic injuries due to posterior dislocations. **Emergent reduction in the Operating Room with an Orthopedic Surgeon and a Thoracic Surgeon in attendance is required for those with mediastinal injuries due to posterior dislocations. If the consultants are not available, consider emergent reduction in the Emergency Department if neurologic or vascular compromise exists in the affected extremity.**

In patients under the age of 25, many sternoclavicular disruptions initially thought to be true posterior dislocations are Salter-Harris I or II epiphyseal injuries with posterior malposition of the distal clavicular segment.[1,20-24] Closed reduction of these injuries can still be attempted after Orthopedic Surgeon consultation.

CONTRAINDICATIONS

Postpone reduction to attend to airway, breathing, circulation, and more serious injuries. If a posterior sternoclavicular joint dislocation is present and compromising adjacent structures, prioritize reduction in your critical patient care actions. Open reduction of posterior sternoclavicular joint dislocations may be preferred if surgery is planned for associated injuries.

Chronic anterior dislocations should not be reduced in the Emergency Department.[40-43] These patients usually have minimal discomfort, normal range of motion and can return to normal activity as tolerated.[1,43] They generally do well with symptomatic treatments, nonsteroidal anti-inflammatory drugs, heat application, and a sling. However, a small number will develop persistent symptomatic instability requiring surgery.[40-44]

The reduction of chronic posterior sternoclavicular joint dislocations is controversial. Most will require an open procedure due to the long-term potential risk of damage to mediastinal structures.[1,45,46] In asymptomatic young adults, a strategy of watchful waiting for physeal plate remodeling that removes the posteriorly displaced bone may be acceptable.[1,34,47] Notably, chronic posterior dislocations without serious sequelae are reported.[15,48] Treatment decisions for chronic posterior fractures or dislocations should be deferred to an Orthopedic Surgeon.

EQUIPMENT

- Sandbags or folded towels
- Povidone iodine or chlorhexidine solution

- Sterile towel clamps
- Sterile gloves
- Local anesthetic solution
- 18 gauge needles
- 25 gauge needles
- 10 mL syringe

PATIENT PREPARATION

Explain the risks, benefits, alternatives, potential complications, and aftercare of the procedure to the patient and/or their representative. Obtain a signed consent to perform the reduction.

Place the patient supine with the affected side near the gurney's edge. Place sandbags or towels between the patient's scapulae. They should be thick enough to raise the patient 5 cm off the gurney.[1]

Anesthesia and analgesia are required to reduce a sternoclavicular joint dislocation. Closed reduction of an anterior sternoclavicular joint dislocation may be performed with local anesthetic solution infiltrated about the medial clavicle and sternoclavicular joint. Consider using supplementary procedural sedation and analgesia (Chapter 159). Posterior sternoclavicular joint dislocations have also been reduced using local anesthetic infiltrated about the medial clavicle and sternoclavicular joint. However, procedural sedation and analgesia (Chapter 159) or general anesthesia is highly recommended. Obtain a consent for the anesthesia technique used to reduce the dislocation.

TECHNIQUES

ANTERIOR STERNOCLAVICULAR JOINT DISLOCATION REDUCTION

Position the patient as described above. The patient's arms should be at their sides (**Figure 101-6*A***). Administer analgesia and sedation as needed. Instruct an assistant to apply downward pressure to the anterior surface of both shoulders (**Figure 101-6*B***). Pushing the shoulders posteriorly pulls the clavicles laterally and distracts the dislocated medial clavicle. Push the medial clavicle posteriorly and into anatomic position (**Figure 101-6*C***). Clavicular relocation usually occurs promptly. Be aware that a sizeable percentage of these dislocations will immediately recur.[1,40] Carefully sit the patient upright while the assistant maintains the patient's shoulders in a posterior position. Apply a figure-of-eight splint (**Figure 112-3*C***).

POSTERIOR STERNOCLAVICULAR JOINT DISLOCATION REDUCTION

Position the patient as described above. Administer anesthesia, analgesia, and sedation as needed. Abduct the affected arm 90° and extend it 20° in-line with the clavicle (**Figure 101-7*A***). Spontaneous reduction may occur at this point. If not, instruct an assistant to apply distal inline traction to the extremity (**Figure 101-7*B***). This may be aided by wrapping a sheet about the patient's upper torso to provide countertraction. The clavicle may reduce under traction. Keep the patient's arm abducted, extended, and under traction if the sternoclavicular joint does not reduce. Manually manipulate the clavicle into position. Grasp the medial clavicle and pull it upward (i.e., anteriorly) into anatomic position (**Figure 101-7*C***).

It may be difficult to firmly grasp the medial clavicle. To overcome this problem, cleanse the skin overlying the medial clavicle of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. Apply a towel clamp to grasp through the skin and around, and not through, the shaft of the medial clavicle (**Figure 101-7*D***). **Ensure that the towel clamp follows the contours of the clavicle. Grasping too deeply can puncture the subclavian artery and/or vein.** Be aware that the clavicle's thick cortical bone often prevents purchase of the towel clamp into the clavicle.[1] Elevate the medial clavicle into anatomic position while an assistant maintains traction on the abducted and extended extremity (**Figure 101-7*D***).

ALTERNATIVE TECHNIQUES

An alternate technique uses the first rib as a lever to reduce a posterior sternoclavicular joint dislocation and has been reported to be successful when the previous technique has failed.[9,49] Position the patient supine with their arms adducted (**Figure 101-8*A***). Administer analgesia and sedation as necessary. Instruct an assistant to apply distal in-line traction to the adducted arm (**Figure 101-8*B***). This will lever the medial clavicle over the first rib and above the superior sternum (**Figure 101-8*B*, inset**). Apply posterior (i.e., downward) pressure to the anterior shoulder, forcing it into retraction (**Figure 101-8*C***). This will lever the medial clavicle anteriorly and laterally into anatomic position (**Figure 101-8*C*, inset**). Reduction may occur at this point. If it does not, elevate the medial clavicle by one of the two methods (i.e., grasping or clamping) described above.[50] It is postulated that this technique requires less force than the more commonly done procedure previously described.[9] A variation of this technique involves applying lateral traction to the upper humerus using a sheet looped around the upper arm.[49]

FIGURE 101-6. Reduction of an anterior sternoclavicular joint dislocation. ***A.*** Patient positioning. ***B.*** An assistant applies anterior pressure to both shoulders. ***C.*** The medial clavicle is pushed posteriorly.

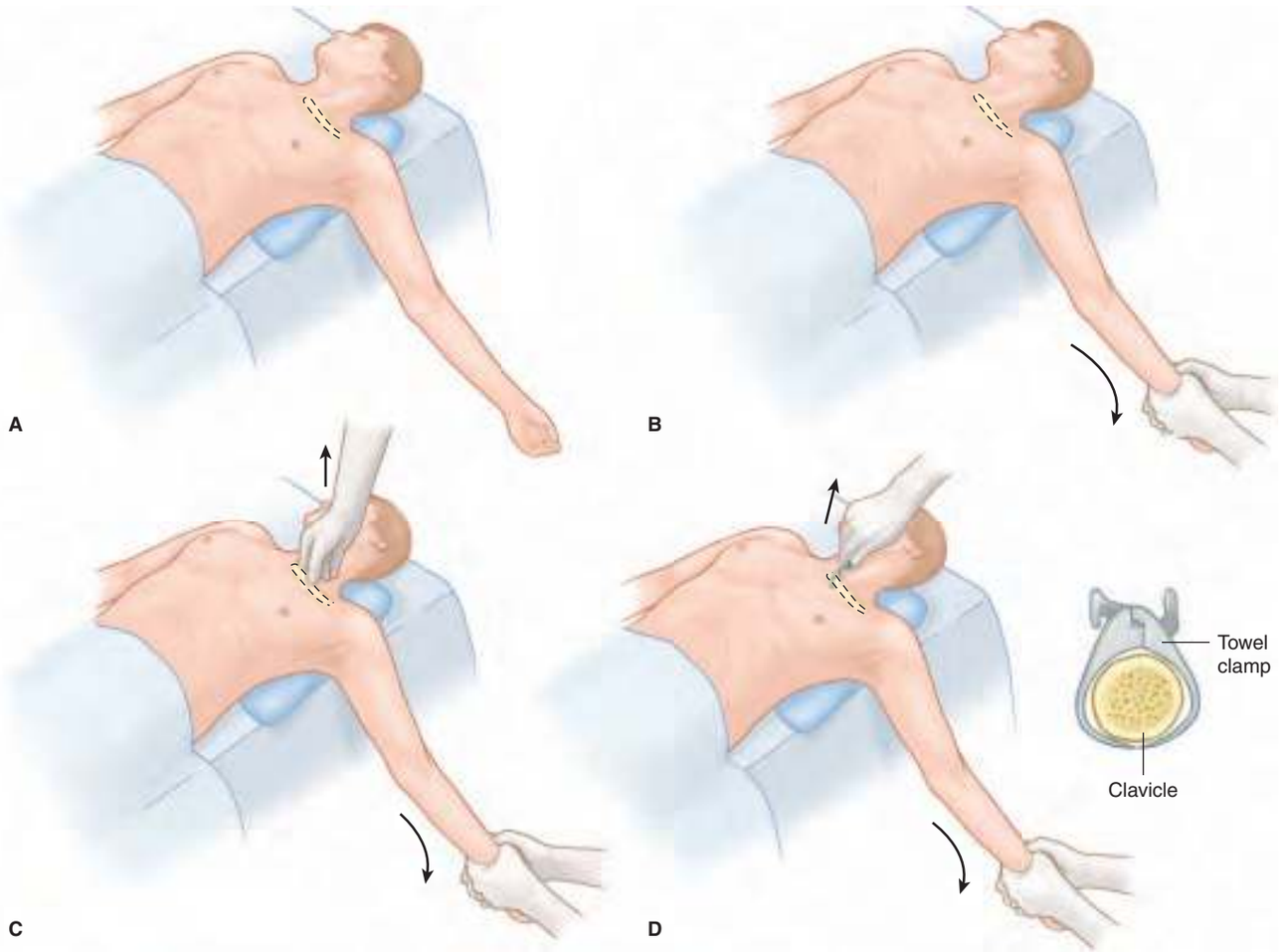

FIGURE 101-7. Reduction of a posterior sternoclavicular joint dislocation. ***A.*** Patient positioning. ***B.*** An assistant applies distal in-line traction. ***C.*** The medial clavicle is grasped and elevated while maintaining distal traction on the extremity. ***D.*** An alternative technique. A towel clamp is placed around the medial clavicle. The clamp is used to elevate the medial clavicle while maintaining distal traction on the extremity.

Other less commonly performed methods for closed reduction of posterior sternoclavicular joint dislocations have been described. Reduction was achieved using 4.5 kg of lateral skin traction on the abducted arm for 30 minutes.[11] Reduction has also been achieved by simple forced retraction of the lateral clavicle.[13] Reduction has been reported in a sedated patient positioned on an interscapular towel roll for 8 hours.[51]

ASSESSMENT

Assess the vascular integrity and neurologic function of all patients initially and following any reduction attempts. It may be prudent to confirm proper bony positioning with post-procedure imaging although studies on this topic have not been done. Additional reduction attempts may be performed if imaging shows incomplete dislocation resolution.

Anatomically correct positioning is not essential in reductions performed for neurologic or vascular compromise. The primary consideration is resolution of the neurologic or vascular compromise. The Orthopedic Surgeon can later reduce the defect that remains.

AFTERCARE

General orthopedic care principles apply. Recommend rest with activity as tolerated, local ice application, immobilization of the injured joint, and nonsteroidal anti-inflammatory drugs. Prescribe supplemental narcotic analgesics as necessary.

ANTERIOR STERNOCLAVICULAR JOINT DISLOCATIONS

The sternoclavicular joint is often unstable following reduction of anterior sternoclavicular joint dislocations and requires splinting to maintain normal anatomic relationships and allow ligamentous healing.[1,40] This is best accomplished with a figure-of-eight splint (**Figure 112-3*C***). However, many patients cannot tolerate this splint. Alternatives include a sling, a sling and swath, or a sling and Velpeau dressing (**Figures 112-3*A* and 112-3*B***). Apply one of these immobilizers. Arrange Orthopedic Surgeon follow-up within 5 to 7 days.

POSTERIOR STERNOCLAVICULAR JOINT DISLOCATIONS

The sternoclavicular joint is usually stable following reduction. The splinting and follow-up are the same as with an anterior sternoclavicular joint dislocation.

COMPLICATIONS

ANTERIOR STERNOCLAVICULAR JOINT DISLOCATIONS

Complications of the reduction are relatively minor. The sternoclavicular joint is often unstable after reduction and may redislocate

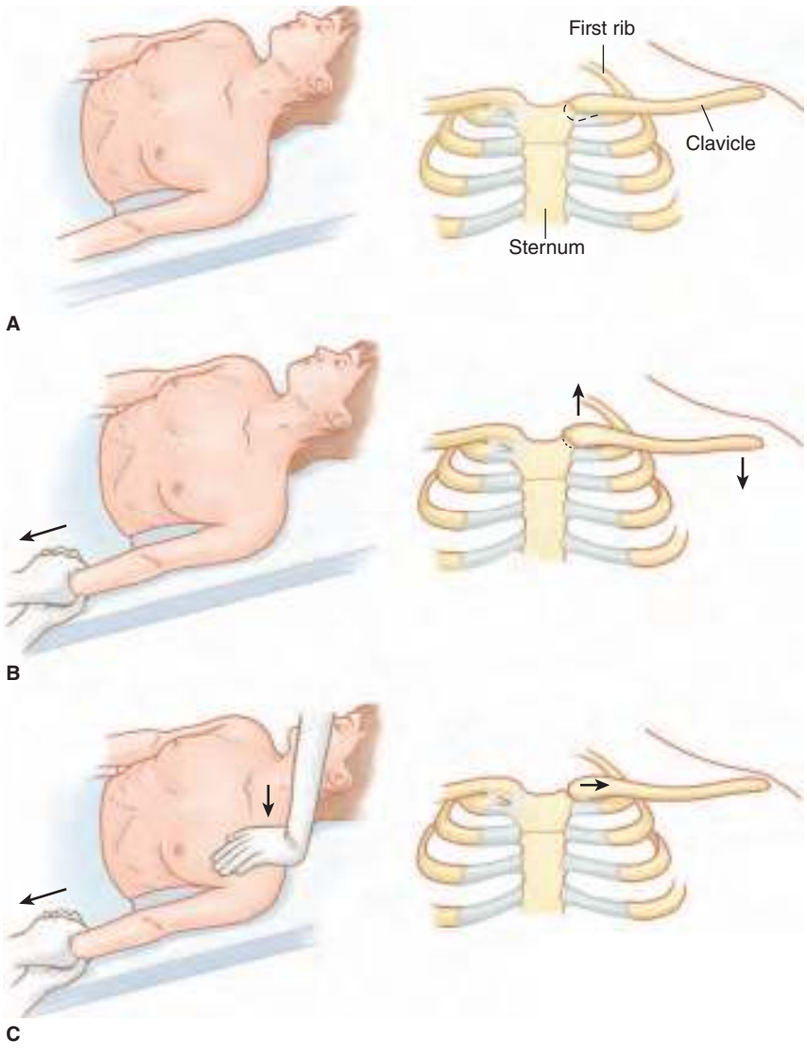

FIGURE 101-8. An alternative technique for reducing a posterior sternoclavicular joint dislocation. (Modified from Buckerfield and Castle.[9]) ***A.*** Patient positioning. ***B.*** An assistant applies distal in-line traction (*large arrow*). The medial clavicle will be elevated above the sternum as traction is placed on the arm (*small arrows*). ***C.*** A posteriorly directed force is applied to the shoulder to draw the medial clavicle anteriorly and laterally into its normal anatomic position.

spontaneously. Explain this to the patient prior to reduction attempts. A "cosmetic bump" can remain even if reduction is achieved and/or maintained. Even after reductions that hold, some patients have persistent pain or joint instability requiring surgery.[1,40-44]

POSTERIOR STERNOCLAVICULAR JOINT DISLOCATIONS

Most complications associated with posterior dislocations are from the original injury and are due to compression or injury of proximate structures. The incidence of associated injuries is commonly reported as approximately 25%.[1,8] Deaths due to these injuries have been reported.[8,29,43,52] Potential reduction-associated complications include pain, incomplete reduction, subclavian vessel injury, and brachial plexus injury.

Chronic posterior dislocations should be reduced in an open fashion in the Operating Room. Fibrous adhesions form between the damaged joint and deeper structures and can tear the brachial plexus if the clavicle is manipulated blindly. This particular complication has also not been documented in the literature.

A higher incidence of injury to the subclavian vessels may be seen if a towel clamp is used in the reduction. This is theoretical based on the towel camp injuring the underlying neurovascular structures in relation to the clavicle. This particular complication has also not been documented in the literature.

The reduction of a posterior dislocation can result in an anterior subluxation or dislocation.[54] This conversion of a posterior-to-anterior dislocation should not be re-reduced since it may resolve spontaneously or require open management.

SUMMARY

Sternoclavicular joint dislocations are uncommon injuries. They are usually due to high-impact forces from motor vehicle collisions or contact sports. Anterior sternoclavicular joint dislocations are easily reduced but often do not remain reduced. However, most patients do well with persistent anterior dislocations. Posterior sternoclavicular joint dislocations are even less common. Emergency Physicians should be aware of the high incidence of associated injuries. These dislocations are usually stable after reduction.

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102 Shoulder Joint Dislocation Reduction

Damali N. Nakitende, Tina Sundaram, and Michael Gottlieb

INTRODUCTION

The shoulder joint is the most commonly dislocated of all joints.[1-5] Shoulder dislocations were depicted in Egyptian murals as early as 3000 BC.[1] Despite 5000 years of medical advancements, shoulder dislocations continue to be a major cause of Emergency Department (ED) visits. They account for more than 50% of all joint complications treated by the Emergency Physicians (EPs).[2]

The human shoulder is remarkable for its degree of mobility. The same anatomic features that contribute to this mobility also contribute to its instability.[3] The shallow glenohumeral joint allows the shoulder to be dislocated anteriorly, posteriorly, or inferiorly. Anterior shoulder dislocations are the most common and account for 95% of all shoulder dislocations.[1-4] The overall incidence of shoulder dislocations is 17 per 100,000. There is a bimodal age distribution.[1,4,6] It occurs most commonly in males from 20 to 30 years of age, typically related to athletics and trauma. The other large group is women from 60 to 80 years of age primarily due to falls.

ANATOMY AND PATHOPHYSIOLOGY

The shoulder or glenohumeral joint is a multiaxial ball-and-socket type of synovial joint that permits a wide range of motion. This range of motion is at the expense of stability.[7] The shoulder has greater than 180° of motion in the sagittal and coronal planes and 180° of rotary movement.[8] The spheroidal head of the humerus articulates with the shallow glenoid fossa of the scapula. The glenoid fossa accommodates roughly one-third of the humeral head. The bony landmarks surrounding the shoulder joint are the coracoid and acromion processes of the scapula. A loose, thin fibrous capsule encloses the glenohumeral joint. The muscular component of the shoulder is a fusion of four separate muscles (i.e., supraspinatus, infraspinatus, teres minor, and subscapularis) that together form the rotator cuff. **These muscles are torn and injured in shoulder dislocations, especially with posterior and inferior dislocations.[9,10]**

The shoulder receives its blood supply from branches of the axillary artery (i.e., the anterior and posterior circumflex humeral arteries). Innervation of the shoulder is from branches of the suprascapular, axillary, and lateral pectoral nerves. The axillary nerve lies at the level of the humeral neck. The humeral head can be dislocated anteriorly into the quadrangular space where it may compress and damage the axillary nerve. This can result in a neurapraxia, paralysis of the deltoid muscle, and/or a sensory loss to the skin over the shoulder.

Shoulder dislocations can occur anteriorly, posteriorly, or inferiorly depending on the mechanism of injury. **Anterior shoulder dislocations are the most common and account for 95% of all dislocations (Figure 102-1).** An anterior dislocation usually results from direct or indirect forces causing abduction, extension, and external rotation of the limb. Anterior dislocations are classified as subcoracoid, subglenoid, subclavicular, or intrathoracic based on the location of the humeral head. **Subcoracoid dislocations account for 75% of all anterior shoulder dislocations (Figure 102-2*A*).** The dislocated humeral head can shift between the first three positions but generally remains in one anatomic location.[2] The injury is usually linked to athletics (e.g., spiking a volleyball or blocking a basketball shot) in younger patients. Older patients may sustain anterior shoulder dislocations from falling on an outstretched arm or from a direct blow on the posterior shoulder.[4] The patient will present in obvious distress. They are holding the affected arm in slight abduction and internal rotation. Their elbow is flexed and supported by the unaffected arm. The shoulder will have the typical "squared off" appearance with loss of the normal deltoid contour (**Figure 102-2*A***). The humeral head may be palpable anteriorly.[1]

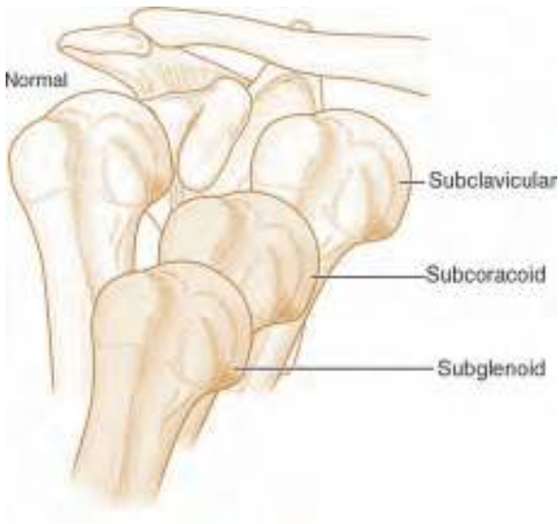


FIGURE 102-1. The types of anterior shoulder dislocations.

Posterior shoulder dislocations account for 4% of all shoulder dislocations.[1-4] They are often missed, even by experienced EPs.[11] Delayed diagnoses have been made up to 1 year after the initial injury.[12] The mechanism of injury is usually indirect, with a combination of internal rotation, adduction, and flexion. The most common precipitating mechanism is a seizure.[13] Other etiologies include electrocution, direct trauma, and falls.[3,13] Direct trauma (e.g., a head-on motor vehicle collision in which the patient braces their hands against the dashboard) can result in bilateral posterior shoulder dislocations in rare instances. Posterior shoulder dislocations are classified based on the location of the humeral head into subacromial, subglenoid, or subspinous spaces. **Subacromial dislocations account for 98% of posterior shoulder dislocations.[2,12]** The patient presents with their arm held in adduction and internal rotation (**Figure 102-2*B***).[14] The shoulder will appear flat anteriorly with the coracoid process visually prominent and palpable.[1]

Inferior shoulder dislocations are the least common type.[1-4] They represent less than 0.5% of all shoulder dislocations. An inferior shoulder dislocation is also known as luxatio erecta because the dislocated extremity is extended upward. Inferior shoulder dislocations are usually sustained from indirect forces with the arm hyperabducted causing the rotator cuff to tear and the arm to rotate 180° externally.[9] Alternatively, a direct axial force applied to the arm above the head (e.g., Olympic-style weight lifting) will drive the humeral head inferiorly.[15] The patient will present with the affected arm shortened, fixed above their head, and the hand rotated as if asking a question (**Figure 102-2*C***).[9,16,17] The humeral head may be palpable along the lateral chest wall. Inferior shoulder dislocations are often associated with fractures.[1] The fractures can involve the acromion process, coracoid process, clavicle, greater tuberosity, humeral head, and/or glenoid rim.[1,2,8,18] Complete disruption of the rotator cuff often occurs with inferior shoulder dislocations.[1,2,8,18] Dislocations can cause capsular and rotator cuff tears, compression or tears of the axillary artery and its branches, and/or injury to the subclavian vein. Damage by traction and compression of the brachial plexus, suprascapular nerve, and the axillary nerve occurs at a rate of 21% to 36% because of their anatomic proximity.[12] Prompt reduction of the dislocation may alleviate compression injuries, enable a more

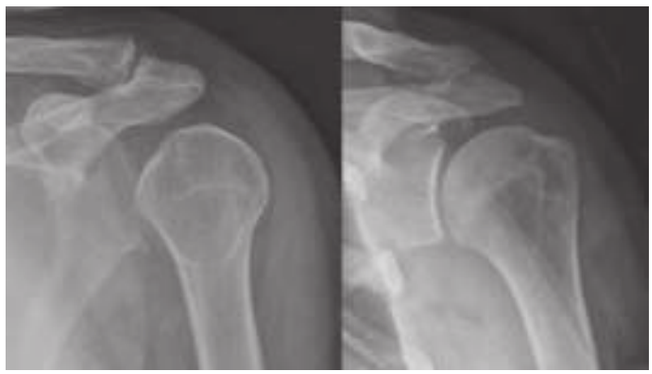
FIGURE 102-2. Clinical photos of shoulder dislocations. ***A.*** Anterior shoulder dislocation. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill, 2016. Photo contributor: Kevin J. Knoop, MD, MS.) ***B.*** Posterior shoulder dislocation. (Used with permission from Schwartz DT: *Emergency Radiology: Case Studies*. New York: McGraw-Hill, 2008.) ***C.*** Inferior shoulder dislocation. (Used with permission from Sherman SC: *Simon's Emergency Orthopedics*, 7th ed. New York: McGraw-Hill; 2015.)

thorough examination, and allow a thorough evaluation of all the components of the shoulder.

Radiographs are required to classify the type of dislocation and diagnose fractures (**Figure 102-3**). Associated fractures are detected in up to 24% of anterior shoulder dislocations.[3] They include fractures of the greater tuberosity, humeral head, coracoid process, acromion process, clavicle, and glenoid. Recent evidence suggests that clinically obvious dislocations without a high-energy mechanism can be reduced without prior radiographs.[19-26] There is no consensus at present to eliminate prereduction radiographs versus the current recommendation that all patients have at least two-view prereduction plain radiographs of the affected joint.[19,27] Postreduction films may be considered to document the reduction of the joint, to assess injury caused by the reduction technique, to document bony abnormalities (e.g., Hill-Sachs lesions and Bankart lesions), and to identify previously hidden fractures that were not visible on the initial radiographs. There is some evidence that postreduction radiographs may be unnecessary.[24-26,28-32] Further study is required before this can be made the standard of care.

The anteroposterior (AP) view will clearly demonstrate anterior dislocations, inferior dislocations, and almost all humeral fractures (**Figure 102-3**). A Hill-Sachs deformity is an impaction fracture of the posterolateral portion of the humeral head (**Figure 102-4**). These are found in up to 50% of all anterior shoulder dislocations.[2] A Bankart lesion is an avulsed fragment of the glenoid labrum with contiguous bone (**Figure 102-5**).[8] Both lesions tend to get worse

A



B

FIGURE 102-3. Radiographs of shoulder dislocations. ***A.*** Anterior dislocation. *Red arrow* identifies the glenoid fossa. *Blue arrow* identifies the humeral head. (Used from James Heilman, MD, www.commons.wikimedia.org.) ***B.*** Posterior dislocation. (Used from Hellerhoff, www.commons.wikimedia.org.)

the longer the humeral head remains dislocated and can further increase shoulder instability.

The AP view often shows a "normal" picture in patients with posterior shoulder dislocations. This accounts for the high incidence of missed dislocations (**Figure 102-3*B***). Up to 50% of posterior shoulder dislocations were missed using only the AP view. The lateral view increased the diagnostic accuracy to 100%.[33] There are four features that suggest a posterior dislocation on AP films. There is the loss of the normal elliptical pattern produced by overlap of the humeral head and the posterior glenoid rim. The distance between the anterior glenoid rim and the articular surface of the humeral head will be increased. This is known as the "rim sign." Internal rotation of the greater tuberosity gives the humerus the appearance of a "light bulb" or "ice cream cone."[2,4] An isolated fracture of the lesser tuberosity on the AP view is suggestive of a posterior shoulder dislocation until proven otherwise.[2] A lateral view (e.g., the Y-view, an axillary view, or an apical oblique view) will help delineate the posterior position of the humeral head behind the glenoid fossa when in doubt.[1,34,35]

FIGURE 102-4. The Hill-Sachs deformity. (Used from Hellerhoff, www.commons.wikimedia.org.)

Bedside ultrasound (US) can be useful to diagnose a shoulder dislocation and verify the reduction (**Figure 102-6**).[36-44] A high-frequency (e.g., 7 to 13 MHz) US transducer is preferred, but a lower frequency transducer can also be used if available. Place the US transducer transversely on the patient's back just inferior to the lateral aspect of the scapular spine to obtain a posterior US.[43] It may be more practical to obtain an anterior or lateral US image in the supine patient.[44] The advantages of bedside US over plain radiographs include decreased time, lack of radiation, not waiting for procedural sedation to wear off before transporting the patient to the radiology suite, ability to perform US in any position, and minimal need for patient cooperation.

INDICATIONS

Shoulder dislocations are typically very painful and distressing for the patient. It does not matter if the dislocation is initial or recurrent. **All attempts should be made to reduce the joint as quickly as possible once the diagnosis is made**. Uncomplicated joint dislocations should be reduced within 20 to 30 minutes, or as soon as possible, to alleviate further injury to surrounding neurologic and vascular structures.[45] **It becomes more difficult to reduce the longer a shoulder is dislocated.[45] A patient with a neurologic deficit or a compromised distal pulse in the setting of a shoulder dislocation should undergo immediate reduction**.

CONTRAINDICATIONS

There are no absolute contraindications to reducing a dislocated shoulder. The patient's airway, breathing, and circulation should be assessed and managed prior to reducing the dislocated shoulder.

A

B

FIGURE 102-5. The Bankart lesion. ***A.*** Plain radiograph. ***B.*** CT scan. (Photos used from RSatUSZ, www.commons.wikimedia.org.)

A

B

FIGURE 102-6. US for an anterior shoulder dislocation. ***A.*** The characteristic empty space between the humeral head and glenoid fossa. ***B.*** Postreduction with the humeral head in close proximity to the glenoid fossa. (Photos used with permission from reference 69.)

Any life- or limb-threatening injuries should be managed before the shoulder reduction is attempted.

An Orthopedic Surgeon should be consulted prior to the reduction of a shoulder dislocation in patients with posterior and inferior dislocations. They are relatively rare, there is a high incidence of complications requiring operative management, associated fractures may make the reduction difficult, and there may be indications for surgical management.[46] The indications for surgical intervention in anterior shoulder dislocations include complete rotator cuff tears, fracture of the greater tuberosity with displacement of more than 1 cm, or fractures of the glenoid rim that are displaced more than 5 mm.[2] Posterior shoulder dislocations with major displacement of a fractured lesser tuberosity or an impression defect greater than 20% of the articular surface necessitate surgical intervention for open reduction.[2,3] Open dislocations require operative management but may be reduced in the ED if there is a delay in getting the patient to the Operating Room. Surgical reduction is indicated in patients with evidence of hemorrhagic shock from a suspected axillary artery injury sustained during a shoulder dislocation.

Consult an Orthopedic Surgeon before reducing a dislocated shoulder that presents greater than 7 to 10 days after the acute

injury.[47,48] There is a higher risk of vascular injury when an "old" dislocation is mistaken for an acute injury and subsequently reduced. The axillary artery, which is bound down by the pectoralis major muscle and anterior pericapsular scarring, becomes brittle and may not withstand the traction required to reduce an "old" dislocation. This is especially true in the elderly. A 50% rate of hemorrhage-related mortality was reported in patients who had shoulder reductions performed several weeks after the initial injury.[18]

Shoulder dislocations in children present unique problems. Pediatric patients whose ossification centers have not yet fused may have associated Salter-Harris fractures of the epiphyseal plate. **Consult an Orthopedic Surgeon prior to the reduction of a pediatric shoulder dislocation unless neurologic or vascular compromise is present in the affected extremity**. The same techniques for reduction can be applied to both adult and pediatric patients.[49]

EQUIPMENT

GENERAL SUPPLIES

- Equipment and supplies for procedural sedation (Chapter 159)
- Assistants
- Sheets
- 10 to 15 pounds of weights
- Sling and swath or shoulder immobilizer
- Splinting material (e.g., plaster, fiberglass, prepackaged casting material)

INTRAARTICULAR ANALGESIA

- Povidone iodine or chlorhexidine solution
- 20 mL syringe
- 25 gauge, 2.5 inch needle
- Local anesthetic solution (e.g., lidocaine or bupivacaine)

MISCELLANEOUS

- Equipment and supplies for nitrous oxide anesthesia (Chapter 158)
- Equipment and supplies for regional anesthesia (Chapter 156)

PATIENT PREPARATION

Explain the risks, benefits, potential complications, and aftercare of the procedure to the patient and/or their representative. Obtain an informed consent for the reduction procedure. An informed consent should be obtained for procedural sedation and/or an intraarticular injection if they are performed in addition to the reduction procedure.

Place the patient in a position of maximal comfort, usually sitting upright or at a 45° angle, with the affected extremity supported and its motion limited. Placing the patient supine or prone is difficult, painful, and often requires analgesia. The patient presenting in spinal immobilization should remain supine. If the spine cannot be cleared, shoulder reduction can be performed without changing the patient's position. Intravenous access should be obtained if indicated. Pain should be addressed quickly and aggressively.

ANALGESIA

There are several methods to control pain for patient comfort before and during the joint reduction procedure. Intravenous or intramuscular opioids should not be withheld pending prolonged radiographic studies. Commonly administered medications include morphine, hydromorphone, and fentanyl.

An alternative to intramuscular or intravenous narcotics is the intraarticular instillation of local anesthetic solution (Chapter 97).[34,50-58] This is an effective method of analgesia for anterior shoulder dislocations and is often used to supplement procedural sedation. It can also be used as the only method of analgesia, when procedural sedation is contraindicated, or to decrease the agents used for procedural sedation. Clean the anterolateral shoulder of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the shoulder area and allow it to dry. Identify the hollow space 2 cm inferior to the lateral border of the acromion process (**Figure 102-7**). Using sterile technique, insert a 25 gauge needle perpendicular to the skin

A

B

FIGURE 102-7. Local anesthesia for a shoulder dislocation. The needle is introduced in the hollow under the lateral surface of the acromion process. ***A.*** Using landmarks. ***B.*** Using US. (Photos used with permission from reference 69.)

and into the hollow to a depth of 2 cm (**Figure 102-7**). Inject 10 to 20 mL of a 50:50 mixture of sterile saline and local anesthetic solution. This technique is effective in controlling muscle spasm and pain. **The book editor believes this should be performed on every dislocated shoulder before attempts at reduction.**

Alternative methods for providing analgesia include the use of nitrous oxide (Chapter 158), procedural sedation (Chapter 159), and regional nerve blocks (Chapter 156). Nitrous oxide has numerous advantages including a short onset, short duration of action, self-administration by the patient as needed, and a decreased ED length of stay.[59] Unfortunately, the use of nitrous oxide produces a quite variable analgesic response. Procedural sedation is commonly used as an aid in the reduction of a dislocated shoulder. US-guided brachial plexus blocks can provide excellent analgesia if the EP is trained in this technique.[60-69] Please refer to Chapter 130 for the details of this technique.

Several sources suggest that patients with an anterior shoulder dislocation without a significant trauma history may accept some degree of discomfort as a trade-off for the prompt resolution of pain by reduction without anesthesia.[70-72] **Patient comfort should not be sacrificed for expediency.** Anterior shoulder dislocations may require procedural sedation prior to reduction, depending on the patient's level of discomfort and the reduction method chosen. Posterior and inferior shoulder dislocations always require procedural sedation prior to reduction.

ANTERIOR SHOULDER DISLOCATION REDUCTION TECHNIQUES

The methods for treating a closed shoulder dislocation depend on overcoming muscular spasm to relocate the humeral head into the glenoid fossa. Reduction techniques are classified into traction techniques, leverage techniques, scapular manipulation, and combined techniques. A study evaluating various reduction techniques found similar success rates of 70% to 90% regardless of the technique.[73] Postreduction complications rates are variable.[73] The major considerations in deciding which technique to use are EP experience, EP familiarity with the technique, availability of time, and the presence or absence of an assistant.[73]

HENNIPEN TECHNIQUE

The Hennipen technique was popularized at Hennipen County Medical Center and is often the preferred method to reduce anterior shoulder dislocations (**Figure 102-8**). This technique can be accomplished with no anesthesia or with the intraarticular instillation of local anesthetic solution. Procedural sedation is not required but may be used if the patient has severe pain and muscle spasms.

Place the patient seated, supine, or reclined 45° on a gurney (**Figure 102-8*A***). Place the affected arm in adduction with the elbow in 90° of flexion. Support the patient's flexed elbow against their torso with the nondominant hand. Grasp the patient's forearm with the dominant hand. Slowly rotate the arm externally. **Stop the motion and wait until the spasm subsides if the pain becomes severe from rotator cuff spasm. Do not release the arm or return it to its original position**. Continue to rotate the arm externally until the humeral head reduces or the arm reaches the coronal plane (i.e., 90° of external rotation). This can take up to 10 minutes to accomplish. If the humeral head is still dislocated, slowly abduct the arm until the humeral head reduces or full abduction is obtained (**Figure 102-8*B***).[2] Full abduction occurs when the patient's hand crosses over their head and touches the contralateral ear. Adduct the arm until it is against the patient's torso. Examine the shoulder. Another technique should be attempted if the humeral head is still dislocated.

The advantages of this technique include little or no patient manipulation and positioning, the relative ease of reduction, minimal equipment, the requirement of only a single person, and the ability to perform the reduction without analgesia. The success rate when performed by EPs is approximately 80%, with 36% of patients not requiring analgesia.[3] The major disadvantage is that patients are too apprehensive and experience too much discomfort to relax their arms sufficiently to allow for reduction to occur. This problem can be eliminated by the intraarticular instillation of local anesthetic solution. The patient will occasionally require procedural sedation.

EXTERNAL ROTATION TECHNIQUE

This is a modified version of the Hennipen technique.[74-79] The technique is identical to the Hennipen technique except that the

A B

FIGURE 102-8. The Hennepin technique to reduce an anterior shoulder dislocation. ***A.*** Patient positioning and external rotation of the humerus. ***B.*** Abduction of the arm with the elbow flexed 90°.

procedure is terminated when the arm reaches 90° of external rotation. The step of abduction is not performed. The advantages and disadvantages are as listed for the Hennipen technique. The success rate ranges between 78% and 97%.[74,76,78-80]

STIMSON TECHNIQUE

The Stimson technique is a safe first-line technique that uses gravity and weights to overcome muscle spasm and reduce the dislocated shoulder (**Figure 102-9**).[81-84] Instill intraarticular local anesthetic solution into the shoulder joint prior to attempting the reduction. Procedural sedation is usually not necessary. **The patient must be under constant observation to monitor pulse oximetry and respiratory status if procedural sedation is used because of the patient's prone positioning.**

Place the patient prone with the dislocated arm hanging over the side of the gurney (**Figure 102-9**). Place the shoulder in 90° of flexion. A pillow or folded sheets may be placed beneath the affected shoulder for patient comfort. Tie a sheet around the patient's hips and the gurney to prevent them from falling off the bed (**Figure 102-9**). Apply 3 to 5 pounds of weight to the patient's wrist or forearm. The weights can be attached by a commercially available device, hung off a padded wrist restraint, or hung off gauze wrapped circumferentially around the wrist. Raise the gurney so that the weights are suspended off the ground (**Figure 102-9**). Every few minutes, add an additional 3 to 5 pounds of weights until a total of 10 to 15 pounds is achieved. The weights will provide traction in a position of forward flexion and are usually sufficient for reduction to take place within 15 to 30 minutes.[4] If the reduction is unsuccessful after 30 minutes, grasp the patient's forearm and twist it to gently rotate the humerus externally and then internally while the patient is prone and the arm is maintained under traction.[85] This additional maneuver will often reduce the dislocation if the weights alone are unsuccessful.

An alternative method is to have the patient grip a bucket half-filled with water. This will provide the necessary traction weight to reduce the joint. The disadvantage of the bucket technique is that the patient must grip the bucket for a considerable length of time without releasing it.

FIGURE 102-9. The Stimson technique to reduce an anterior shoulder dislocation. Weights are progressively added to the wrist to distract the humerus.

The advantage of the Stimson technique is that it is safe and does not require the presence of an assistant. A 96% success rate has been reported with this technique.[3] The disadvantage of the procedure is that the patient must be placed in a prone position that may be painful, uncomfortable, or impossible because of other injuries. There is a small risk of the patient slipping off the elevated gurney. **Apply a strap or sheet tied around the patient and the gurney to prevent this.** Procedural sedation is not recommended due to the prolonged prone positioning which may interfere with the patient's respirations.[86] Procedural sedation for 15 to 30 minutes is relatively contraindicated and difficult to maintain without potential complications.

SCAPULAR MANIPULATION TECHNIQUE

Scapular manipulation accomplishes reduction by repositioning the glenoid fossa rather than manipulating the humeral head (**Figure 102-10**).[4,87-92] This is a popular technique due to its low complication rate and high patient satisfaction. This technique can be accomplished with no anesthesia and without the intraarticular instillation of local anesthetic solution. Procedural sedation is not required.

Place the patient prone with the dislocated extremity hanging over the side of the gurney (**Figures 102-10*A* and 102-10*B***). Place the shoulder in 90° of flexion. A pillow or folded sheets may be placed below the affected shoulder for patient comfort. Place 5 to 15 pounds of weights suspended from the patient's wrist or in their hand. An assistant may apply downward traction on the extremity if weights are not available (**Figure 102-10*B***). The weights or the assistant will provide in-line traction to the arm.

Identify the scapula and its borders. The scapular tip will "wing" laterally. Stabilize the superior portion of the scapula with one hand (**Figure 102-10*A***). Place the thumb of the stabilization hand along the superolateral border of the scapula. Apply constant and firm medial and upward pressure to the inferior tip of the scapula using the other hand or thumb (**Figure 102-10*B***). The thumb of the stabilizing hand can also be used to apply medially directed pressure to the tip of the scapula. Push the tip of the scapula as far medially as possible. The shoulder should reduce within 1 to 3 minutes. A small degree of dorsal displacement of the scapular tip has also been recommended.[3,92] Slight external rotation of the humerus by an assistant while the scapula is being manipulated and the arm remains under traction may facilitate reduction (**Figure 102-10*B***). This maneuver releases the superior glenohumeral ligament and presents a favorable profile of the humeral head to the glenoid fossa.[90,92]

The scapular manipulation technique may be performed with the patient supine (**Figure 102-10*C***). This is particularly helpful when other injuries or conditions preclude using the prone position. Place the affected shoulder in 90° of flexion. Instruct an assistant to grasp the forearm and apply upward traction to elevate the shoulder off the bed. Apply your hands to stabilize and manipulate the scapula as described previously.

This technique may be performed with the patient sitting (**Figure 102-10*D***). This is particularly helpful when other injuries or conditions preclude using the prone or supine position. Place the affected shoulder in 90° of flexion. Instruct an assistant to grasp the forearm and apply horizontal traction. Apply your hands to stabilize and manipulate the scapula as described previously. This method is technically more difficult because the patient's torso is not stabilized and moves during the traction and scapular manipulation.[3,90,92]

The reduction of an anterior shoulder dislocation by the scapular manipulation technique is usually quite subtle and may be missed by

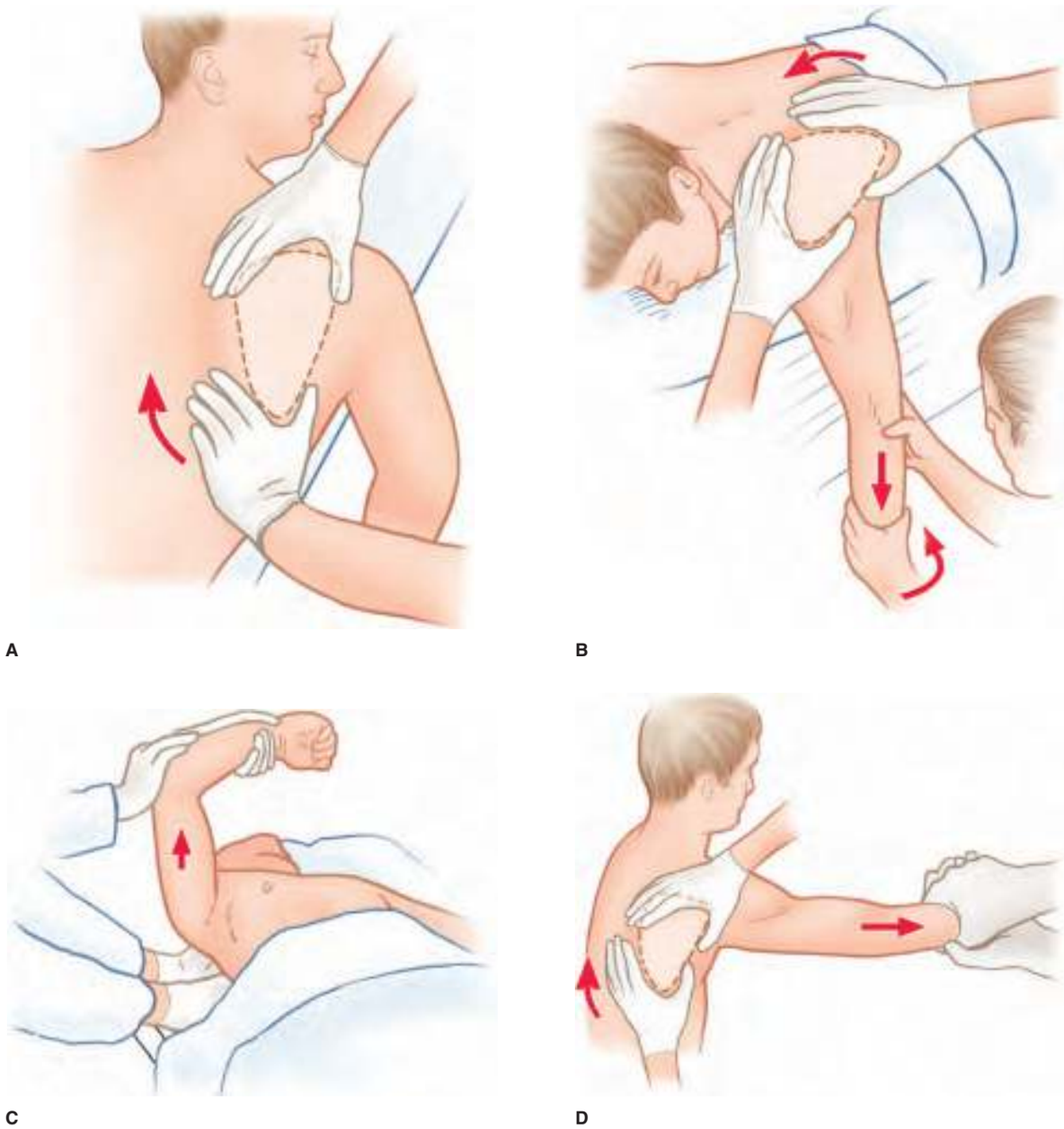


FIGURE 102-10. The scapular manipulation technique to reduce an anterior shoulder dislocation. ***A.*** Proper hand positioning. The upper hand stabilizes the base of the scapula while the lower hand applies medial and upward pressure on the tip of the scapula (*curved arrow*). ***B.*** Reduction with the patient prone. Traction is applied by an assistant (*straight arrow*). Occasionally, external rotation is also required (*curved arrow*). ***C.*** Reduction with the patient supine. Traction is applied on the humerus to elevate the shoulder off the bed (*arrow*). ***D.*** Reduction with the patient sitting. Traction is applied on the humerus (*arrow*).

both the patient and the EP. The act of lying prone will be sufficient to relocate the shoulder in a few rare cases. Success rates of 79% to 90% have been reported with this technique.[3,4,87-92] The procedure is well tolerated.[3,4,87-92] There is a very low incidence of complications with this procedure and it can be performed without analgesia and monitoring.[87] Disadvantages include the prone position and the need for an assistant to apply traction on the arm.

TRACTION-COUNTERTRACTION TECHNIQUE

The traction-countertraction technique is commonly performed in the ED out of tradition (**Figure 102-11**). It may be used as a first-line technique or a backup for patients who have failed alternate techniques.[2] This technique requires anesthesia and analgesia. Instill local anesthetic solution intraarticularly. This may be sufficient. Many patients will require procedural sedation. This technique can cause significant patient discomfort.

Place the patient supine. Pass a sheet around the chest and axilla of the affected arm (**Figure 102-11*A***). Place the affected arm in 45° of abduction. Instruct an assistant to firmly grasp the loose ends of the sheet. Grasp the patient's wrist and apply slow and steady traction. Instruct the assistant to apply countertraction. The assistant and EP should be exerting equal and opposite forces. **Stop the motion and wait until the spasm subsides if the pain becomes severe from rotator cuff muscle spasm. Do not release the arm or return it to its original position.** Continue applying traction and countertraction until the shoulder reduces after the spasm subsides.

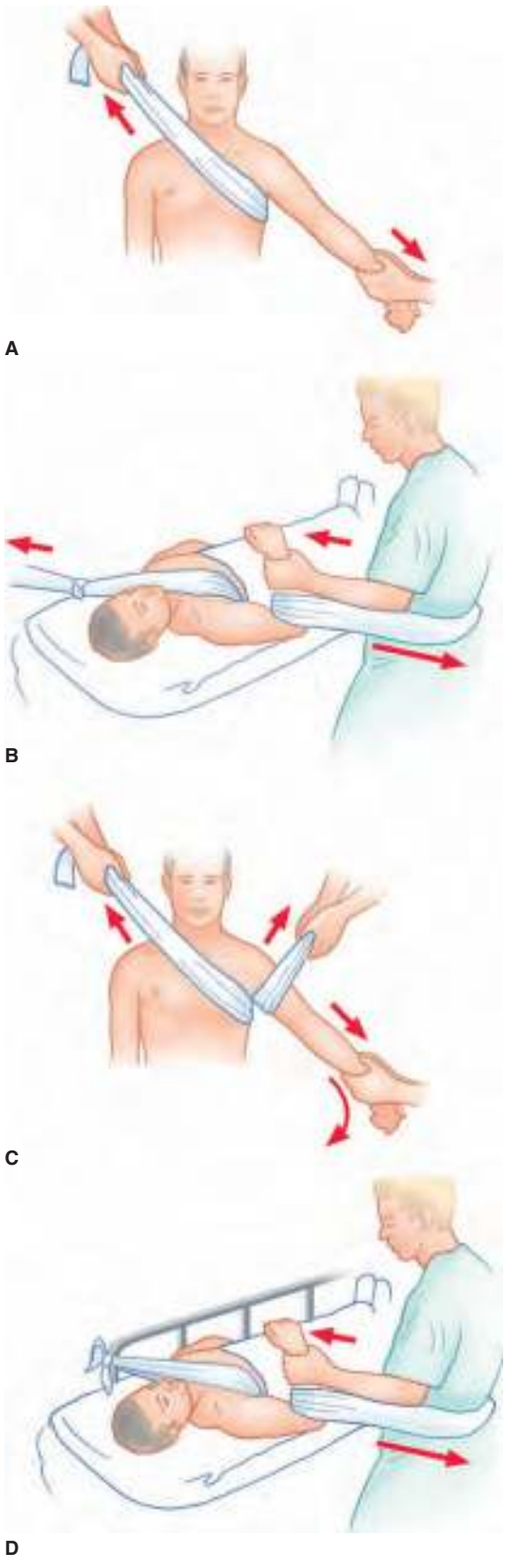


FIGURE 102-11. The traction-countertraction technique to reduce an anterior or posterior shoulder dislocation. ***A.*** In-line traction is applied to the affected extremity after it is abducted 45°. An assistant provides equal and opposite countertraction with a sheet. ***B.*** An alternative method. A sheet is looped around the flexed forearm and the EP's hips. The EP leans back (*arrow*) to allow their body to do the work of reduction while their hand maintains the patient's elbow in 90° of flexion. ***C.*** An additional assistant is applying traction 90° to the traction-countertraction axis with a sheet in the axilla. Simultaneous adduction (*curved arrow*) and in-line traction by the EP may aid in the reduction. ***D.*** The single-person technique.

Direct traction on the extended arm may result in rapid operator fatigue. This is especially true if the EP is creating most of the force of traction through contraction of their biceps (**Figure 102-11*A***). A less strenuous alternative is available and may be preferred (**Figure 102-11*B***). Position the patient as above. Place the elbow of the affected arm in 90° of flexion. Place a looped sheet over the proximal forearm and the EP's hips. **Do not loop the sheet around their back, as this can cause low back strain.** This method allows the EP to lean back slowly and use their body weight to supply the traction force. The EP's arms should be extended with their hands grasping the patient's distal forearm. The EP's hands should maintain the patient's forearm upright with the elbow flexed to 90° while applying traction on the forearm with the sheet (**Figure 102-11*B***).

Traction may need to be applied for several minutes. The application of gentle and limited external rotation to the affected arm while under traction may speed up the reduction. Alternatively, a second assistant can apply lateral pressure (i.e., lateral traction) on the humeral head with their hands. A variation of this technique involves a second assistant with a looped sheet placed high in the patient's axilla (**Figure 102-11*C***). This second assistant is used to create lateral traction at the proximal humerus that is perpendicular to the traction-countertraction axis.[2] The EP continues in-line traction and can simultaneously adduct the patient's arm to maneuver the humeral head back into position as lateral traction is applied (**Figure 102-11*C***). The second assistant may also be used with the technique demonstrated in **Figures 102-11*A* or 102-11*B***.

Successful reduction is noted by a lengthening of the arm, a noticeable "clunk," and/or a brief fasciculation of the deltoid muscle. Disadvantages of the traction-countertraction technique include the need for more than one person, the significant degree of force required, the prolonged time and endurance required of the EP, and the need for procedural sedation. **This technique should not be used to reduce shoulder dislocations associated with significant fractures. The force required for this technique can displace fracture fragments and necessitate an open reduction or operative management of the displaced fragments.**

A technique has been developed to use this technique with a single person (**Figure 102-11*D***).[93] The sheet to place countertraction is tied to the gurney instead of being wrapped around the waist of an assistant. The remainder of the procedure is the same as described previously.

SNOWBIRD TECHNIQUE

The Snowbird technique was named after a ski area in Utah where this technique originated.[94] It was developed to reduce the large number of ski-related dislocations quickly while conserving time and resources. This is an effective alternative reduction technique as compared with the more traditional methods. This technique can be accomplished with no anesthesia. The intraarticular instillation of local anesthetic solution is highly recommended. Procedural sedation is usually not required for this reduction technique.

Place the patient in a seated position on a chair with a back (**Figure 102-12**). Completely adduct the affected arm. Flex the elbow to greater than 90°. Support the affected arm with the other arm or a pillow. Make a 3 foot long loop of 4 inch wide cast stockinette. Place the stockinette around the proximal forearm. Instruct the patient to sit as straight as possible. Instruct an assistant to maintain the patient in an upright position by standing adjacent to the unaffected shoulder and clasping their hands around the chest (i.e., in the axilla) of the affected shoulder (**Figure 102-12**). Place one foot in the stockinette loop and apply firm downward traction with the foot while the patient tries to keep the shoulder relaxed and the affected elbow flexed. Instruct the assistant to provide

FIGURE 102-12. The Snowbird technique to reduce an anterior shoulder dislocation. Downward traction is applied to the humerus (*arrow*) while the humeral head is manipulated back into the glenoid fossa.

countertraction to keep the patient from moving. The shoulder may reduce. If not, with continued downward traction on the stockinette, the EP will have both hands free to apply gentle rotation and pressure on the humeral head until the shoulder is reduced.[94]

The Snowbird physicians had a 97% success rate and reduced 93% of anterior shoulder dislocations without any form of analgesia.[94] The advantages of this technique include the relative ease of setup, the rapid nature of the technique, limited use of analgesia, and limited patient positioning. Potential disadvantages include the use of an assistant and the fact that this technique was used and developed on a limited patient population (i.e., young and healthy skiers).

MILCH TECHNIQUE

Milch wrote that a fully abducted arm is in a natural and neutral position in which there is little tension on the muscles of the shoulder girdle.[95-97] The technique that Milch developed relies on gentle manipulation through abduction, external rotation, and traction on the arm.[72,95-100] The patient's affected arm moves in a gradual arc, assisted by the EP, to reduce the dislocation without extensive or forceful manipulation (**Figure 102-13**). The intraarticular instillation of local anesthetic solution is highly recommended even though this technique can be performed with no anesthesia.[101] Procedural sedation is not usually required for this reduction technique.

Place the patient supine. Position one hand with the thumb under the dislocated humeral head (**Figure 102-13*A***). Slowly abduct the affected arm 180° to an overhead position (**Figure 102-13*B***). This can be accomplished by having the patient lift the affected arm. Many patients are unable to do this due to pain, muscle spasm, or

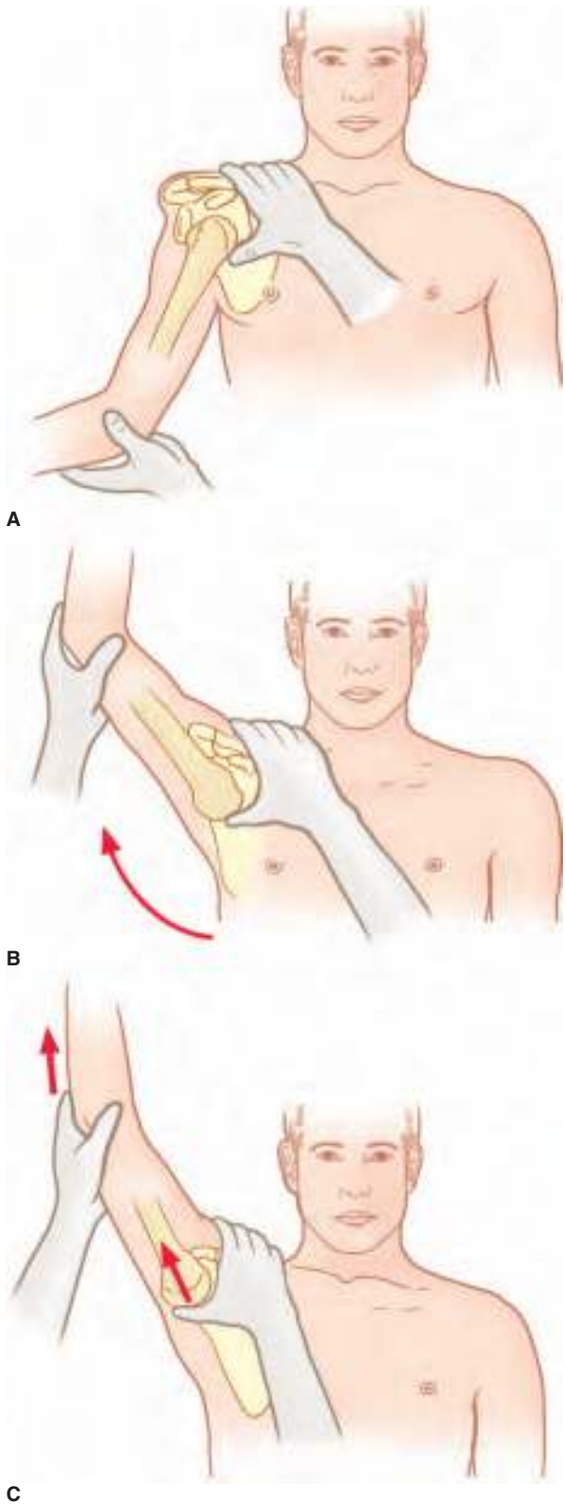


FIGURE 102-13. The Milch technique to reduce an anterior shoulder dislocation. ***A.*** The distal humerus is grasped with one hand while the thumb of the other hand is placed under the dislocated humeral head. ***B.*** The arm is abducted to 180°. ***C.*** In-line traction is applied to the humerus while the thumb pushes the humeral head into the glenoid fossa.

apprehension. Gently grip the elbow or wrist and slowly abduct the arm to 180° (**Figure 102-13*B***). Grasp the patient's distal arm or proximal forearm with one hand once the arm is fully abducted. Apply gentle longitudinal traction with slight external rotation to

the arm (**Figure 102-13*C***). The humeral head may reduce. If not, while maintaining traction with external rotation, apply upward pressure under the humeral head with the other hand to guide it into the glenoid fossa (**Figure 102-13*C***).

Successful reduction is attained in 70% to 90% of the cases with no requirement for assistance, other equipment, or medications.[72,95-100,102] Advantages of the Milch technique include its gentleness, high success rate, limited complications, and good patient tolerance.[103,104] Disadvantages include positioning the arm in full abduction with or without analgesia, severe pain, muscle spasm, and/or apprehension. This technique can be modified slightly so that the patient can perform a self-reduction under the instruction of the EP.[105]

ALTERNATIVE ANTERIOR SHOULDER DISLOCATION REDUCTION TECHNIQUES

Numerous alternative and less commonly used techniques are available to reduce an anterior shoulder dislocation. Some of these are modifications of existing methods. Others are original techniques that had too many associated complications and were modified with time and experience. Some are well-known and effective techniques that have been used for many years. There is very little research regarding many of these techniques. Some of these techniques may prove to be useful in the reduction of an anterior shoulder reduction. **None of these techniques are advocated as first-line treatments for the reduction of shoulder dislocations. Their inclusion here is for the sake of completeness and does not necessarily constitute an endorsement for their use**. Only a select few of these techniques are discussed.

HIPPOCRATIC TECHNIQUE

The Hippocratic technique is the original traction-countertraction technique (**Figure 102-14**).[106] It is one of the oldest documented techniques to reduce a shoulder dislocation.[2,18,82,106] This technique has the advantage that it can be performed by a single operator in any setting. **The Hippocratic technique is efficient but not recommended due to the great force required to achieve reduction.** Common complications of the technique include fractures, brachial plexus injury, vascular injury, and poor patient tolerability.[2,8,18]

Place the patient supine (**Figure 102-14**). Grasp the wrist of the affected arm and abduct the arm 20° to 30°. Place one foot into the axilla of the affected arm. Firmly grasp the patient's wrist and apply traction to the arm while extending the foot in the axilla to provide countertraction.

FIGURE 102-14. The Hippocratic technique to reduce an anterior shoulder dislocation. Traction is applied to the arm while countertraction is applied using a foot in the axilla.

KOCHER TECHNIQUE

The Kocher technique was first recorded on an Egyptian mural dated 1200 BC.[107] It is another traditional method that has come into disfavor. This maneuver relies upon leverage and humeral manipulation to reduce the shoulder (**Figure 102-15**).[108] The humeral head is levered on the anterior glenoid while the humeral shaft is levered against the anterior thoracic wall until reduction is achieved.[18,107,109] A substantial amount of force must be applied while adducting and externally rotating the arm to reduce the joint.

Place the patient sitting at a 45° incline or supine. Abduct the affected arm to 45° and flex the elbow to 90° (**Figure 102-15*A***). Grasp the patient's distal humerus with the dominant hand and the patient's wrist with the nondominant hand (**Figure 102-15*A***). Apply in-line traction to the distal humerus. Rotate the arm externally to its maximum extent while maintaining in-line traction (**Figure 102-15*B***). Move the patient's elbow across their chest and to the midline while maintaining in-line traction; the elbow is held tightly against the patient's chest (**Figure 102-15*C***). Internally rotate the arm while in-line traction is maintained and until the patient's hand touches their opposite shoulder (**Figure 102-15*D***).

The main advantage of this method is that it is a single-person technique that has withstood the test of time. Studies have shown that the forces generated are sufficient to cause fractures of the humeral neck, spiral humeral fractures, vascular trauma, and brachial plexus injury.[18]

ESKIMO TECHNIQUE

The Eskimo technique uses the patient's body weight and gravity as a traction mechanism to reduce an anterior shoulder dislocation.[110] It can be performed in the field, where access to a health care facility may be limited. Disadvantages of this technique include the strength and stamina required to lift the patient, injury due to heavy lifting, poor patient tolerability, and increased stress on the brachial plexus and axillary vessels.

Place the patient on the floor and lying on the unaffected side (**Figure 102-16**). Instruct the patient to place the affected arm tightly adducted with the elbow flexed to 45° (**Figure 102-16*A***). Grasp the injured arm and slowly lift the patient 6 to 12 inches off the ground (**Figure 102-16*A***) so that the patient's body weight produces enough traction to reduce the joint. This technique was initially described and reported to have a 74% success rate.[110]

Two alternatives exist. The patient can be positioned on the unaffected side with the affected arm abducted to 90° (**Figure 102-16*B***). One or two people can then grasp the patient's wrist and forearm and lift the patient 6 to 12 inches off the ground (**Figure 102-16*B***). Some patients cannot move to the floor. Position the patient with the unaffected side on the gurney. Stand on the gurney while an assistant holds the EP so they do not fall. This risks the EP falling off the gurney and becoming injured.

CHAIR TECHNIQUE

The chair technique is a simple method to reduce an anterior shoulder dislocation.[111-114] It is a variation of the traction-countertraction technique. Place the patient sitting sideways or backward in a chair with the affected arm draped over the back rest (**Figure 102-17**). Supinate the patient's wrist and apply downward traction while the patient attempts to stand and provide countertraction (**Figure 102-17**).

This technique has a 72% success rate.[3] The advantages include the simplicity of the technique, not requiring an assistant, not needing any equipment, and not requiring analgesia. A large amount of force is required to reduce the shoulder. These forces can cause

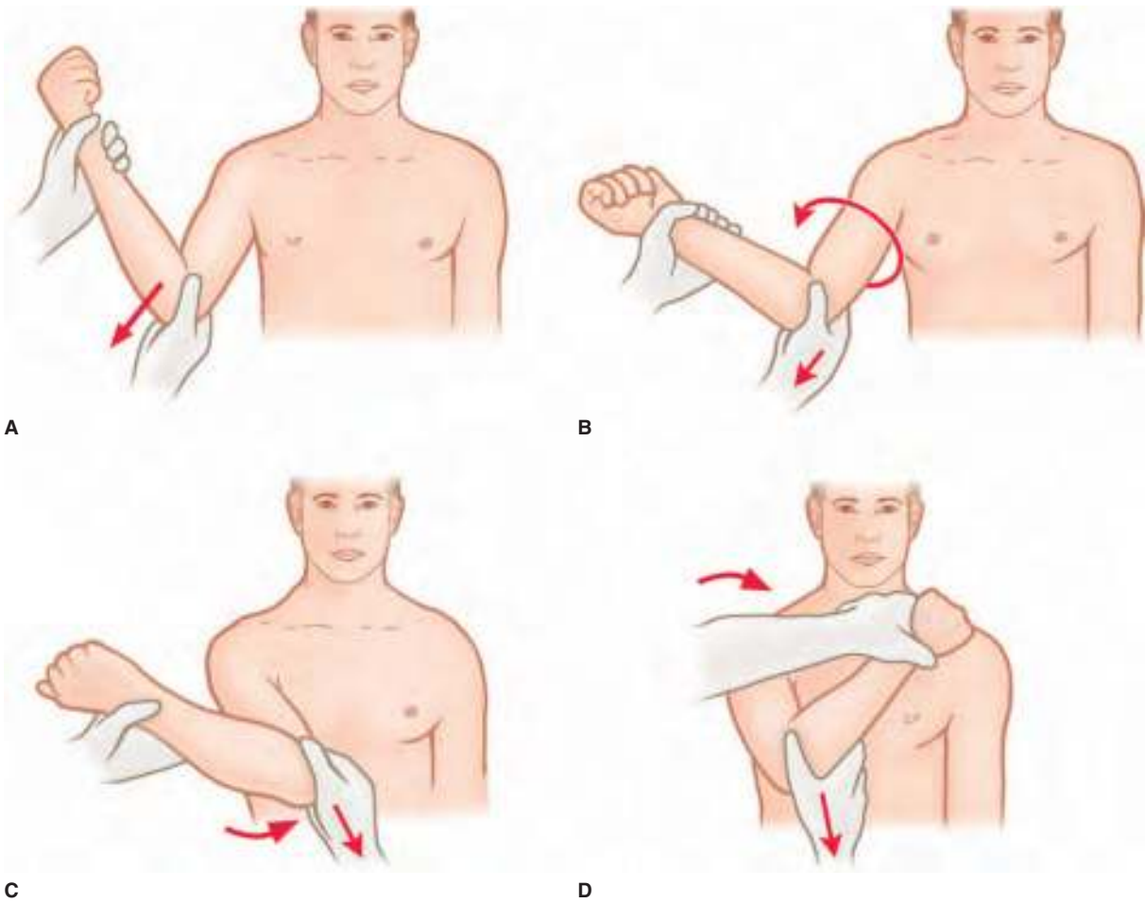


FIGURE 102-15. The Kocher technique to reduce an anterior shoulder dislocation. ***A.*** The arm is abducted to 45° with the elbow flexed to 90°. In-line traction is applied to the humerus (*arrow*). ***B.*** The arm is rotated externally (*curved arrow*) while traction is maintained (*straight arrow*). ***C.*** The elbow is brought across the chest to the midline while the arm is still rotated externally and traction on the arm is maintained (*straight arrow*). ***D.*** Traction is maintained while the arm is rotated internally until the patient's hand touches their opposite shoulder.

injury to the brachial plexus and axillary vessels as the axilla is impinged on the back of the chair.

WRESTLING TECHNIQUE

A new technique was recently described based on optimal anatomic positioning with limited complications.[115] Place the patient supine with the elbow of the affected shoulder flexed to 120°. Grasp the dislocated arm just above the humeral condyles and apply distal traction to the arm. Grasp the distal forearm overhanded with the opposite hand and move the hand from the condyles through the acute angle of the arm, grasping the wrist of the hand holding the forearm. The wrestling hold is now established (**Figure 102-18**). The patient's forearm will be used as a fulcrum. Abduct the patient's shoulder to 45° while maintaining constant traction. Externally rotate the arm in a slow, smooth motion. Move the patient's arm over their chest wall while maintaining traction and maintaining external rotation, and then rotate it internally.[1] The shoulder should subsequently reduce.

This technique has the advantages of requiring no equipment, no analgesia, and no assistants. It may be used in the field where a health care facility is not readily available. The main disadvantage is the amount of force applied to the shoulder and surrounding structures. The twisting of the forearm as a lever can displace fracture fragments or cause new fractures. This technique requires the EP to have a significant amount of upper body strength and arms long enough to accomplish the wrestling hold. It is difficult to apply the wrestling hold if the patient has large arms. The series of movements is difficult to accomplish while always maintaining distal traction.

PNEUMATIC STRETCHER TECHNIQUE

This technique was developed to reduce a shoulder dislocation when assistants were not available or if the physician did not have the physical strength required to use other techniques.[116] It is a modification of the traction-countertraction technique. **This technique should never be used to reduce a shoulder dislocation**. It will cause stretching and possible rupture of the brachial plexus, ligaments of the shoulder region, muscles and tendons crossing the shoulder, nerves of the upper extremity, vascular structures of the upper extremity, and injury to other joints. **Tremendous forces are applied to the extremity with this technique.**

Place the patient prone with the affected arm hanging off the gurney (**Figure 102-19**). Wrap a sheet around the patient's hips and the gurney to hold the patient in position. Tie the ends of the sheet into a knot or have them held by an assistant. Wrap the patient's wrist with gauze and tie it to the base or wheel of the gurney. Begin elevating the bed until the dislocation is reduced.

SPASO TECHNIQUE

The Spaso technique is a single person technique that is simple to perform.[117-121] It uses minimal force and can be performed without procedural sedation. This technique is similar to the Stimson

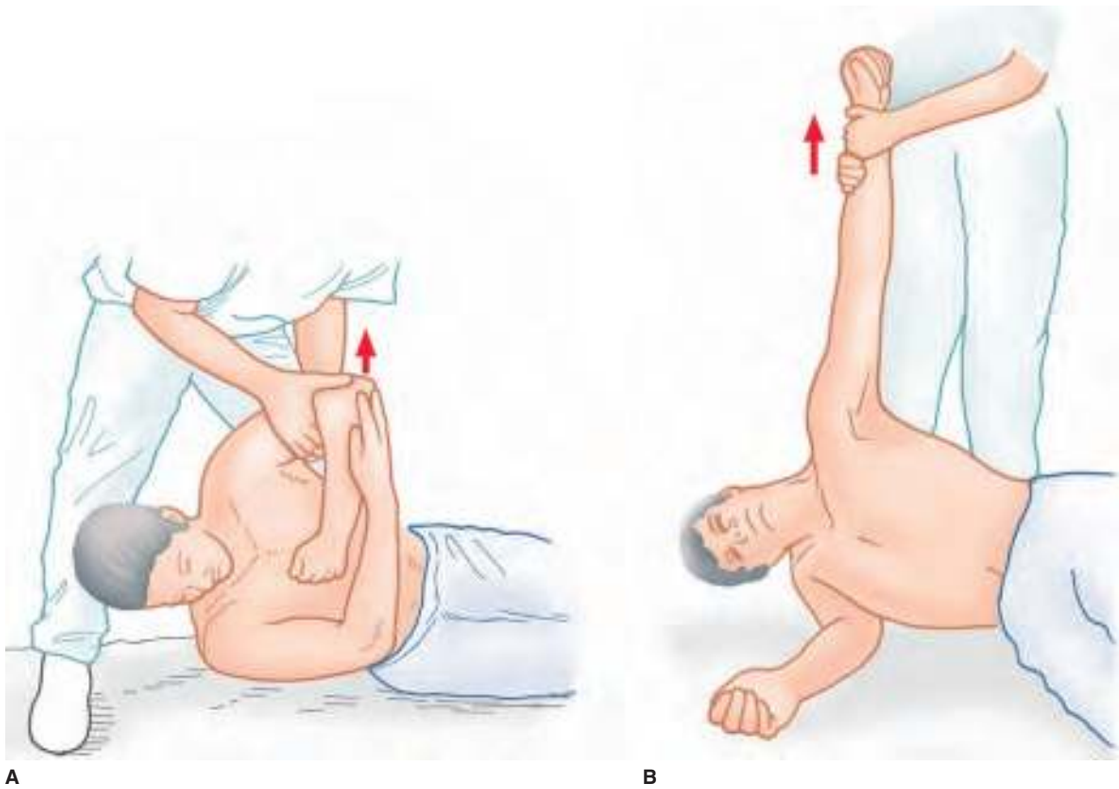


FIGURE 102-16. The Eskimo technique to reduce an anterior shoulder dislocation. The patient is lying on the floor on the unaffected side. ***A.*** The patient is lifted off the ground by grasping the adducted arm. ***B.*** The patient is lifted off the ground by grasping the distal forearm of the affected arm.

technique, with the main difference being the patient's position.[118] Successful rates of reduction can be seen in up to 87% of patients.[119-121]

Place the patient supine. Grasp the wrist of the affected arm with the dominant hand and the patent's proximal forearm with the nondominant hand (**Figure 102-20**). Slowly flex the patient's shoulder until the arm is upright and the shoulder is flexed to 90° (**Figure 102-20**). Apply upward traction to the arm while gently externally rotating the arm. Slowly increase the upward traction until the shoulder reduces. If reduction does not occur, use the nondominant hand to apply externally directed pressure to the humeral head to push it into the glenoid fossa while maintaining the arm in traction and external rotation with the dominant hand. An assistant may be required to push on the humeral head while the EP uses both hands to apply upward traction.

FIGURE 102-17. The chair technique to reduce an anterior shoulder dislocation. Traction is applied to the arm as the patient attempts to stand and provide countertraction.

FIGURE 102-18. The wrestling technique to reduce an anterior shoulder dislocation. Traction and external rotation are applied to the arm after the hold is established.

FIGURE 102-19. The pneumatic stretcher technique to reduce an anterior shoulder dislocation. The gurney is raised to provide countertraction to the traction applied to the wrist that is by being tied to the base of the gurney.

OZA MANEUVER

Direct manipulation of the humeral head with simultaneous arm traction may allow for a simple reduction of the humeral head.[122] This technique is a modification of the traction-countertraction technique. Very little information exists in the literature regarding the effectiveness of this maneuver.

Place the patient supine with the affected arm and their body against the edge of the gurney (**Figure 102-21**). Instruct an assistant to grasp the patient's wrist and slowly abduct the arm until it is at 45° to 90°. Stand between the patient's abducted wrist and their body, with your back against the patient's body. Place both thumbs against the dislocated humeral head. Wrap the fingers of both hands around the proximal humerus and interlace the fingers. Instruct the assistant to apply traction to the arm while simultaneously using the thumbs to lift and push the humeral head up and over the anterior glenoid lip and back into the glenoid fossa. Alternatively, the EP can stand outside the patient's abducted arm, wrap their fingers around the proximal humerus, and pull the humeral head back into the glenoid fossa.

FIGURE 102-20. The Spaso technique to reduce an anterior shoulder dislocation. Upward traction is applied (*arrow*) as the arm is externally rotated (*curved arrow*).

FIGURE 102-21. The Oza maneuver to reduce an anterior shoulder dislocation. Traction is applied to the arm as the humeral head is pushed outward.

BEST OF BOTH (BOB) MANEUVER

The BOB maneuver is a combination of distally applied traction and scapular manipulation.[123] This maneuver is quick to perform and does not use procedural sedation. It requires the patient to sit upright with their legs dangling off the gurney. This maneuver is not appropriate to use with procedural sedation, intubated patients, multitrauma patients, those who cannot sit upright, or anyone with altered mentation. The BOB maneuver can be performed with no analgesia and without the instillation of local anesthetic solution intraarticularly.

Lower the gurney as much as possible. Elevate the head of the gurney to 90°. Place the patient sitting upright on the side of the gurney with their unaffected arm against the upright head of the gurney. The patient's feet may touch the floor or dangle. Instruct the patient to "scoot over" until their unaffected shoulder and hip are tight against the head of the gurney. Kneel on the gurney facing the patient (**Figure 102-22**). Flex the elbow of the affected arm to 90°. Place one hand on the patient's proximal forearm adjacent to the antecubital fossa. Grasp the patient's wrist with the other hand. Lean down with your body weight and pull the patient's humerus downward (**Figure 102-22**). Instruct an assistant to simultaneously stand behind the patient and perform the scapular manipulation technique. The combination of traction of the humerus and scapular manipulation will allow the humeral head to relocate in the glenoid fossa.

LEGG REDUCTION MANEUVER

This maneuver is a simple technique to reduce an anteriorly dislocated shoulder.[124] It was developed and used to treat athletes in the field. Many patients may not tolerate the pain associated with this maneuver. It is not appropriate to use this technique with procedural sedation, intubated patients, multitrauma patients, those who cannot sit upright in a chair, or anyone with altered mentation. The maneuver can be performed with no analgesia and without the instillation of local anesthetic solution intraarticularly.

FIGURE 102-22. The best of both (BOB) maneuver to reduce an anterior shoulder dislocation. Traction is applied to the humerus as the tip of the scapula is pushed medially. ***A.*** Superior view. ***B.*** Lateral view.

Place the patient sitting upright in a chair. Instruct an assistant to provide slight downward pressure on the unaffected shoulder (**Figure 102-23*A***). Instruct the assistant to maintain downward pressure on the shoulder throughout the maneuver. Place one hand on the affected shoulder and apply downward pressure while abducting the patient's arm with the other hand (**Figure 102-23*A***). Externally rotate the arm and flex the elbow to 90° until the patient's palm faces outward (**Figure 102-23*B***). Pull the elbow and forearm posteriorly until it crosses a plane through the occiput (**Figure 102-23*C***). Adduct the arm while simultaneously flexing the elbow from 90° to full flexion (**Figure 102-23*D***). Internally rotate the arm until the hand touches the unaffected shoulder and the humerus relocates (**Figure 102-23*E***).

CUNNINGHAM TECHNIQUE

Neil Cunningham developed a technique to simply, quickly, and painlessly reduce an anterior shoulder dislocation without the use of medications.[125,126] His technique is based on the principle of relaxing the biceps brachii muscle with massage while the rhomboids retrovert the scapula to allow the humeral head to relocate.

Place the patient sitting upright in a chair or on a gurney (**Figure 102-24**). Stand next to the affected arm and face the patient. Fully adduct the humerus and flex the elbow to 90°. Insert one hand between the patient's forearm and their body. Grasp the patient's proximal forearm so that their hand and wrist are resting on your upper forearm (**Figure 102-24**). Instruct the patient to shrug their

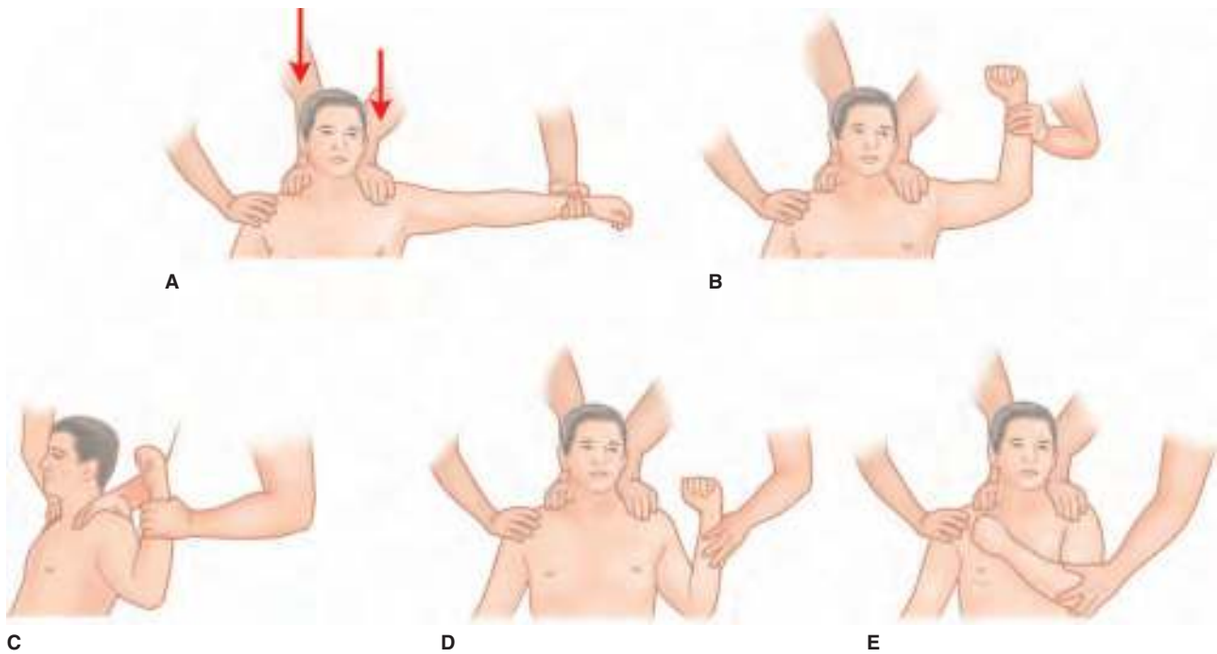


FIGURE 102-23. The Legg reduction maneuver to reduce an anterior shoulder dislocation. ***A.*** Initial patient positioning. ***B.*** The affected arm is externally rotated and the elbow flexed to 90°. ***C.*** The arm is moved posteriorly. ***D.*** The arm is adducted while fully flexing the elbow. ***E.*** Internal rotation of the arm.

FIGURE 102-24. The Cunningham technique to reduce an anterior shoulder dislocation. Traction is applied to the humerus while the trapezius, deltoid, and biceps muscles are massaged sequentially.

shoulders superiorly and posteriorly to square-off the angle of their shoulder. Apply steady and downward pressure to the patient's forearm. With your other hand simultaneously massage the patient's trapezius, deltoid, and biceps muscles sequentially. Repeat the massage process while maintaining the downward traction and concentrating on the biceps until the shoulder reduces.

BOSS-HOLZACH-MATTER TECHNIQUE

This technique allows the patient to self-reduce an anterior shoulder dislocation.[127-130] The patient can control the application of force to minimize their pain during the reduction. It is a hybrid of the traction-countertraction and scapular manipulation techniques. The EP instructs the patient through the technique. This technique is only successful in 60% of anterior shoulder dislocations. This is significantly lower than many other commonly used reduction techniques.

Place the patient supine on a gurney. Elevate the head of the gurney to 90°. Bind the patient's wrists together with an elastic or gauze bandage. Instruct the patient to flex their knee on the affected side. Instruct the patient to place their bound wrists in front of the flexed knee (**Figure 102-25**). Lower the head of the gurney completely. Instruct the patient to slowly lean back while simultaneously hyperextending their neck and shrugging their shoulders anteriorly to move the glenoid fossa anteriorly and inferiorly. The humeral head will reduce as the glenoid fossa moves toward the humeral head while traction is applied to the humerus.

FIGURE 102-25. The Boss-Holzach-Matter technique to reduce an anterior shoulder dislocation. The patient leans back to provide countertraction while the wrists are secured over the knee to provide distal traction.

FARES METHOD

This technique can reduce an anteriorly dislocated shoulder in an average of 2 to 3 minutes with the use of procedural sedation.[131] The FARES method has a 78% success rate and only requires a single person to perform the reduction.

Place the patient supine with the affected arm at their side. Fully extend the elbow and place the forearm in the neutral position with the patient's thumb pointing upward. Grasp the distal forearm and hand. Apply longitudinal traction on the arm (**Figure 102-26*A***). Oscillate the forearm continuously with brief (e.g., 2 to 3 cycles/second) and short range (e.g., 5 cm above and below the horizontal plane) vertical movements of the arm (**Figure 102-26*A***). Slowly

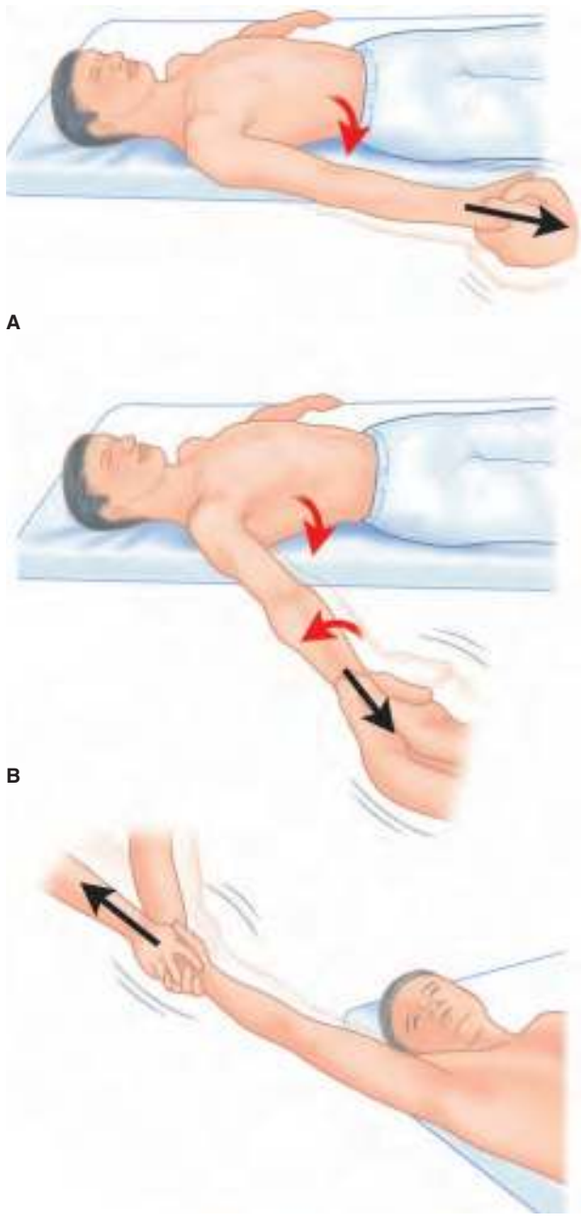


FIGURE 102-26. The FARES method to reduce an anterior shoulder dislocation. ***A.*** Longitudinal traction is applied to the abducted arm as vertical oscillatory movements are made with the forearm. ***B.*** The arm is further abducted and externally rotated as longitudinal traction and vertical oscillatory movements are continued. ***C.*** The arm is further abducted as longitudinal traction and vertical oscillatory movements are continued until the shoulder reduces.

adduct the arm while maintaining longitudinal traction and vertical oscillatory movements (**Figure 102-26*B***). Externally rotate the arm until the palm is facing upward and the arm is approximately 90° abducted. Continue to slowly abduct the arm while maintaining longitudinal traction and vertical oscillatory movements until the shoulder reduces (**Figure 102-26*C***). The shoulder usually reduces at approximately 120° of abduction. Gently internally rotate the arm and adduct it until the humerus is against the chest and the forearm is resting across the chest.

GONAIS TECHNIQUE

The GONAIS technique is a self-reduction method that involves squatting.[132] The word GONAIS is an acronym for "grasp a waist-high object, opposite arm assists, nonsedated, autoreduction/autotraction, immobilize the grasped object, and squatting and stooping." The advantages of this method include the following: self-reduction, quick reduction, the patient does not have to lie down or sit, minimal discomfort, minimal amount of force required, and the opposite hand can assist. The disadvantage is having to be able to squat.

Position the patient next to the locked gurney so it will not move (**Figure 102-27*A***). Instruct the patient to grasp the waist-high gurney with the affected arm (**Figure 102-27*A***). They can use the opposite arm to support the affected arm. The patient stands straight (**Figure 102-27*B***). The patient applies gentle traction on the affected arm and gradually steps back (**Figure 102-27*C***). The patient has stepped back and flexes their upper body forward (**Figure 102-27*C***). The patient squats down and abducts the dislocated shoulder (**Figure 102-27*D***). The patient squats until they have completely squatted (**Figure 102-27*E***). Instruct the patient to lean forward and move a few steps backward while remaining in a squat (**Figure 102-27*F***). Instruct the patient to use the opposite hand to apply pressure and push the humerus backward to reduce the dislocation (**Figure 102-27*G***).

SOOL'S METHOD

Sool's method was developed to overcome muscle spasm and reduce pain.[133,134] It is easy to perform, reduces the dislocation quickly, and has a 75% success rate. The addition of intraarticular local anesthetic would probably increase the rate of successful reduction. The disadvantages of this technique include the requirement for the patient to sit upright, severe pain limiting the lifting of the affected arm, the strength required of the EP, and the lack of sedation in the sitting patient.

The patient is placed in a sitting position with their back straightened. The EP stands in front of the patient (**Figure 102-28*A***). The EP grasps the patient's elbow (**Figure 102-28*A***). The patient's hand and arm are raised and angled toward the EP's shoulder. The patient raises their affected arm and hand until the angle of the arm and trunk reaches 90° (**Figures 102-28*A* and 102-28*B***). The EP is still holding the patient's elbow. The patient's hand and arm are resting on the EP's shoulder (**Figures 102-28*C* and 102-28*D***). Maintain hold of the patient's elbow and grasp the anterior part of the deltoid and pectoralis muscles with the other hand (**Figure 102-28*D***). Slowly pull the elbow upward and out while massaging the anterior part of the deltoid and pectoralis muscles (**Figure 102-28*E***). A clunk is felt as the shoulder is reduced. Instruct the patient to maintain this position.

DAVOS TECHNIQUE

The Davos technique is named after the hospital in Switzerland in which the physicians described it had worked.[128,129,135,136] It is one of the self-reduction techniques that can be used outside the hospital. There is confusion about the name. This technique is the same as the Boss-Holzen-Matter technique discussed earlier.

POSTERIOR SHOULDER DISLOCATION REDUCTION TECHNIQUE

An Orthopedic Surgeon should be consulted before attempting to reduce the shoulder due to the rarity of posterior shoulder dislocations, the difficulty of reduction, the high incidence of associated injuries, and the need to operate to repair the associated injuries.[2,4,12,18,137,138] **The only exception is when the affected extremity has signs of neurologic or vascular compromise and the Orthopedic Surgeon is not immediately available to reduce the shoulder**. The patient will require intraarticular instillation of local anesthetic solution and procedural sedation for the performance of this technique.

TRACTION-COUNTERTRACTION

Place the patient supine. Perform procedural sedation. Pass a sheet around the axilla and torso of the affected arm in the same manner as in the traction-countertraction technique (**Figure 102-11*A***). Grasp the distal forearm. Apply axial traction in-line with the humerus. Instruct an assistant to apply countertraction on the sheet looped around the patient's torso. The traction and countertraction should be equal in force and in opposite directions. Gently internally rotate

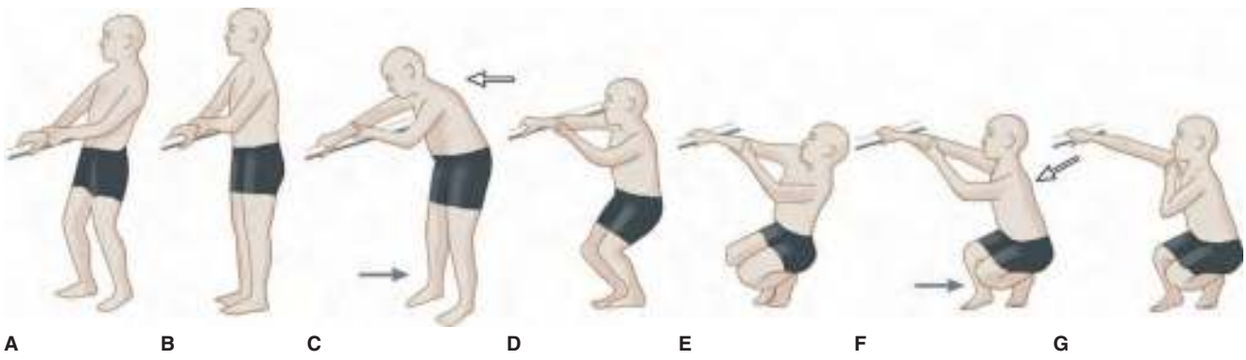


FIGURE 102-27. The GONAIS technique to reduce an anterior shoulder dislocation. ***A.*** The patient is positioned next to the locked gurney. They grasp the waist-high gurney with the affected arm. ***B.*** The patient stands straight. ***C.*** The patient applies gentle traction on the affected arm and gradually steps back (*gray arrow*). The patient has stepped back and flexes their upper body forward (*clear arrow*). ***D.*** The patient squats down and abducts the dislocated shoulder. ***E.*** The patient squats completely. ***F.*** The patient leans forward (*clear arrow*) and moves a few steps backward (*gray arrow*) while remaining in a squat. ***G.*** The patient uses the opposite hand to apply pressure and push the humerus backward to reduce the dislocation.

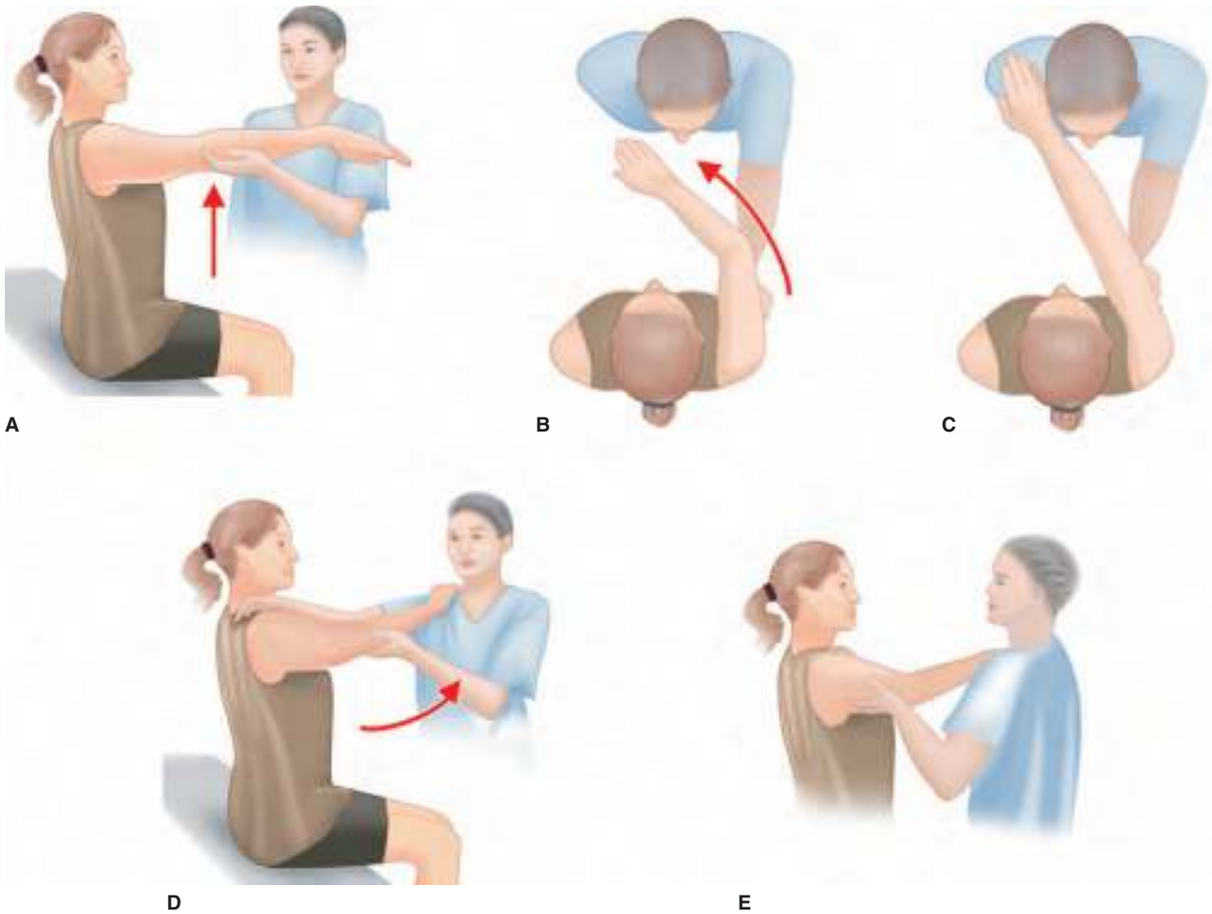


FIGURE 102-28. Sool's method to reduce an anterior shoulder dislocation. ***A.*** The patient is placed sitting on the edge of the gurney with their back straightened. The EP stands in front of the patient. The EP grasps the patient's elbow. ***B.*** The patient's hand and arm are raised and angled toward the EP's shoulder. ***C.*** The patient raises their affected arm and hand until the angle of the arm and trunk reaches 90°. ***D.*** The EP holds the patient's elbow and grasps the anterior part of the deltoid and pectoralis muscles with the other hand. The EP slowly pushes the elbow upward and out while massaging the anterior part of the deltoid and pectoralis muscles. ***E.*** The physician feels a clunk as the shoulder is reduced.

and adduct the arm while maintaining traction. The shoulder may reduce. If not, instruct a second assistant to apply simultaneous lateral pressure on the humeral head.[3] Continue to exert pressure on the humeral head to work it over the glenoid rim. The arm may need to be gently rotated externally. If this fails to reduce the shoulder, instruct a second assistant to use a sheet looped around the proximal humerus to apply lateral traction and repeat the process (**Figure 102-11*C***). If the shoulder will still not reduce, this is an indication for general anesthesia and an open or closed reduction in the Operating Room.[4,14] The shoulder joint is usually unstable and may not remain articulated once it is reduced.

An alternative to grasping the forearm is to apply a padded wrist restraint. Tie the loose ends of the restraint straps to create a loop around the EP's hips. A second alternative is to loop a sheet around the patient's flexed forearm and the EP's hips as in the traction-countertraction technique (**Figure 102-11*B***). These two alternatives are preferred, as they allow the EP's body to reduce the shoulder rather than depending on biceps strength.

INFERIOR SHOULDER DISLOCATION REDUCTION TECHNIQUE

Inferior shoulder dislocations require an Orthopedic Surgeon to be consulted before attempting closed reduction unless neurologic or vascular compromise is present in the affected extremity. Consultation is required because of the rarity of inferior shoulder dislocations, the difficulty of reduction, the high incidence of associated injuries, and the need to operate to repair the associated injuries.[2-4,15,138-142] The patient will require intraarticular instillation of local anesthetic solution and procedural sedation for the performance of these techniques.[4] An inferior shoulder dislocation is usually reduced using the traction-countertraction technique.[15,138,142,143]

TRACTION-COUNTERTRACTION

Place the patient supine. Loop a sheet over the clavicle of the affected shoulder with the loose ends of the sheet at the opposite hip (**Figure 102-29**). Stand above the patient's head and grasp the distal forearm. Apply axial traction to the arm. Instruct an assistant to apply equal countertraction on the sheet. Gently adduct the arm in a full arc from the patient's head to their side (**Figure 102-29**). The shoulder should reduce. The shoulder joint is usually unstable and may not remain articulated once reduced. In rare instances, buttonholing of the joint capsule will prevent closed reduction and require an open reduction.[2]

TWO-STEP MANEUVER

This maneuver consists of two steps.[144-146] The first step converts an inferior shoulder dislocation into an anterior shoulder dislocation. Step two is the reduction of the anterior shoulder dislocation

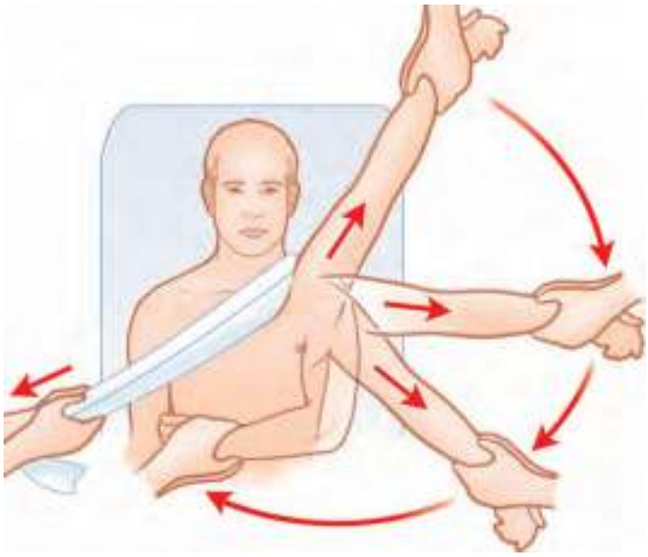


FIGURE 102-29. Reduction of an inferior shoulder dislocation. Axial traction (*straight arrows*) is applied and maintained on the dislocated extremity while it is simultaneously hyperadducted (*curved arrows*).

using the external rotation technique described previously. The two-step maneuver has several advantages over using the traction-countertraction technique. This includes requiring a single person, using a single reduction attempt, possibly using less sedation, and not requiring a trip to the Operating Room for the reduction.

Place the patient supine. Stand adjacent to the affected shoulder and facing the patient's feet. Place one hand on the distal aspect of the lateral humerus and the other hand on the medial epicondyle (**Figure 102-30**). Use the hand on the lateral aspect of the distal humerus to push inward while simultaneously pulling the elbow outwards (**Figure 102-30**). This will convert the inferior dislocation into an anterior dislocation. Apply the external rotation technique to reduce the anterior dislocation (**Figure 102-8*A***).

FIGURE 102-30. The two-step maneuver to reduce an inferior shoulder dislocation. Step one involves pushing the lateral aspect of the distal humerus while pulling on the medial condyle to convert the inferior dislocation into an anterior dislocation. Step two is the external rotation technique (Figure 102-8).

ASSESSMENT

The postprocedural care of the dislocated shoulder is as important as the initial reduction. Successful shoulder reduction can be sensed as a shift or "clunk" in the shoulder joint. Sometimes this can be a very subtle sign. The normal contour of the shoulder is restored and patients often report marked improvement in their pain. A simple test to evaluate the success of the reduced joint is to have the patient touch their contralateral ear or shoulder with the hand of the affected limb.[3] The ability to do so usually signifies a relocated shoulder joint. **The patient should have a thorough examination to evaluate the extremity for vascular and/or neurologic compromise**. Any compromise requires immediate consultation with an Orthopedic Surgeon.

It is important to immobilize the shoulder to prevent further external rotation or abduction of the reduced shoulder by the patient. Place the affected extremity in a shoulder immobilizer (**Figure 102-31*A***) or a conventional sling with a swath (**Figure 102-31*B***).[1,2,18,23,147-150] The shoulder should be immobilized in external rotation in a spica cast if a successful reduction is unstable (**Figure 102-31*C***).[3,151] Splinting the shoulder with the arm adducted and in external rotation may prevent recurrent dislocations.[152-160] Patients should remain in this position for 3 weeks.[152] A splint to hold the shoulder in external rotation can be made with splinting material, or a commercially available product may be purchased (**Figure 102-31*D***).[152-155,157,161]

Postreduction films are indicated after immobilization to confirm the reduction of the joint, to rule out missed fractures on the original radiographs, to rule out a fracture from the reduction procedure, and to evaluate for displacement of fracture fragments. There is some evidence suggesting that postreduction radiographs may be unnecessary, especially if atraumatic.[28,29,35] Further study is warranted before this can be routinely recommended.

US can be used to verify the shoulder is reduced (**Figure 102-6**).[162,163] Only small studies and case reports are in the literature. This technique needs to be validated before US can become standard practice and replace postreduction radiographs.

AFTERCARE

The patient will need to be observed following procedural sedation before being discharged home in the care of friends or family. It is necessary to have the patient awake, alert, oriented, and to have the pain adequately controlled before discharge. The patient should be discharged with adequate pain control and follow-up care by an Orthopedic Surgeon. Control pain during the acute inflammatory response period using oral nonsteroidal anti-inflammatory drugs supplemented with narcotics as needed for 2 to 3 days. Arrange Orthopedic follow-up within 24 hours for anterior shoulder dislocations complicated by fractures or soft tissue injuries beyond ligamentous strain. Follow-up within 5 to 7 days is generally sufficient for uncomplicated anterior shoulder reductions.

The duration of immobilization depends on the patient's age.[2,18,73] The only large-scale prospective study of first-time anterior shoulder dislocations followed patients over a 10-year period. It found that the duration of immobilization had no effect on the incidence of recurrence. **Age was the only prognostic factor for recurrence.** Patients under 20 years of age should be immobilized for 3 weeks and then begin active range-of-motion exercises. Patients age 20 to 40 years should be immobilized for 1 to 2 weeks and begin active range-of-motion exercises 5 days after reduction. Patients older than 60 years of age should have minimal immobilization (i.e., less than 1 week) and begin active range-of-motion exercises within 72 hours after reduction to limit subsequent shoulder stiffness.[2,18] Patients should be instructed to avoid external rotation and

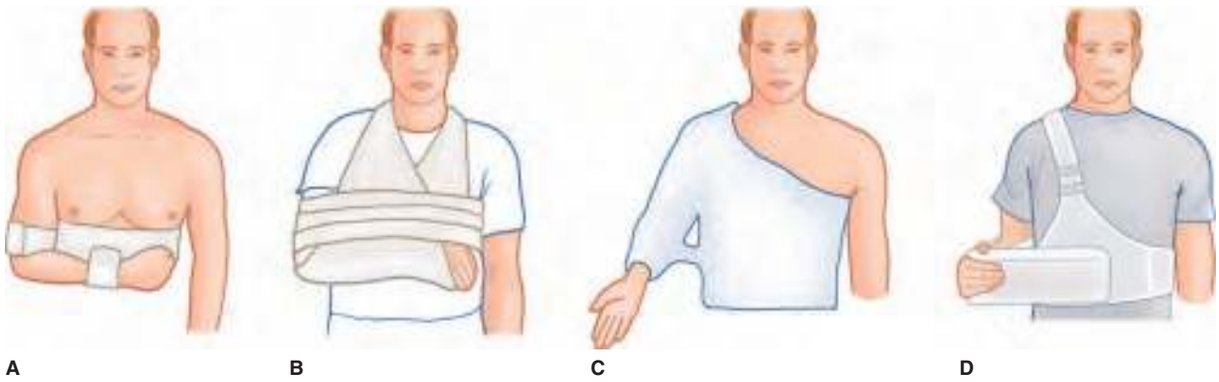


FIGURE 102-31. Methods of shoulder immobilization after reduction of a dislocation. ***A.*** Shoulder immobilizer. ***B.*** Velpeau dressing. ***C.*** Spica cast. ***D.*** Immobilization in external rotation.

abduction activities (e.g., combing their hair) to avoid a recurrent dislocation.[1]

Range-of-motion exercises should include dangling-arm rotation.[2,18] While supporting the torso with the other arm, the patient makes a small circular motion with the injured arm against the force of gravity. Strengthening the subscapularis muscle by doing internal rotation against a resistance band with the elbow flexed to 90° is advocated for anterior dislocations.[2]

COMPLICATIONS

Complications of shoulder dislocations can occur from the initial injury, the reduction technique, or a combination of both. Complications are discussed with respect to both the initial injury and the reduction. They include fractures, displacement of fracture fragments, rotator cuff tears, vascular injury, neurologic injury, recurrence of the dislocation, hemarthroses, and the inability to reduce the shoulder.

FRACTURES

Most fractures are caused during the dislocation and rarely during the reduction procedure if the proper techniques are used.[2,4,12,18,139] Prereduction radiographs will identify most fractures. Postreduction radiographs will identify fractures initially missed or new ones associated with the reduction. Fractures of the humerus and coracoid process are rare and almost always associated with traumatic anterior shoulder dislocations.[1,2,12] These fractures make closed reduction very difficult and should generally be treated under general anesthesia by an Orthopedic Surgeon.

More common bony injuries include the Hill-Sachs deformity (**Figure 102-4**) and the Bankart lesion (**Figure 102-5**), both caused from the dislocation. The Hill-Sachs lesion occurs in up to 50% of shoulder dislocations.[1,2] More significant fractures can occur during reduction in rare situations in which the humeral head is dislocated anteriorly with impaction on the glenoid rim.[1,2,12,18] The Bankart lesion is more commonly seen in recurrent dislocations and is associated with rupture of the joint capsule but not the rotator cuff.[1,2,12,18]

DISPLACEMENT OF FRACTURE FRAGMENTS

Obtain prereduction radiographs on all traumatic shoulder dislocations. They will identify most fractures and any associated displacement. Many of the reduction techniques use significant force and may displace a fracture fragment.[164] Postreduction radiographs are required to evaluate displacement of fracture fragments. The displacement of fracture fragments may make reduction difficult or impossible. They sometimes necessitate operative reduction under general anesthesia.

ROTATOR CUFF TEARS

Rotator cuff injury is most commonly seen in inferior dislocations and in patients greater than 60 years of age.[18,140,141] Approximately 38% of shoulder dislocations will have associated rotator cuff tears at the time of injury. This injury is not typically seen as a complication of the reduction technique.[3,18] Rotator cuff tears generally do not impede reduction, as they are often missed during the initial evaluation. One study showed that 86% of shoulder dislocations had rotator cuff tears diagnosed by arthroscopy an average of 7 months after the dislocation.[1,4] Rotator cuff injuries complicate restoration of normal shoulder function and may require surgical correction.

VASCULAR INJURY

Vascular injuries are seen in the arteries and veins of the shoulder region in association with shoulder dislocations.[1,12,18,139,165-167] **An evaluation for the signs of an axillary artery injury should be sought before and after any reduction attempt**. The most common vessel injured during a dislocation and forceful reduction is the axillary artery.[168-170] Such an injury is usually seen in older patients who have brittle vessels that have lost some elasticity. Inferior dislocations have the highest association with vascular injuries.[18] The second and third parts of the axillary artery are deep to the pectoralis major muscle and sustain the most damage.[12] Arterial injury may occur when attempting the reduction of a chronically dislocated shoulder. Arterial injuries can present with a decreased radial pulse, an axillary mass, an axillary bruit, or lateral chest wall bruising.[1] An angiogram is indicated if a vascular injury is suspected.

Direct injuries to venous vessels are atypical. The most common injury is a venous thrombosis.[171,172] Physical signs include extremity edema and occasionally paresthesias. These signs are typically seen days after the reduction. The diagnostic test of choice for venous evaluation is a US with Doppler study.[12]

NEUROLOGIC INJURY

Neurologic injury is seen in up to 12% of all shoulder dislocations.[12,18,139,167,173-175] **An evaluation for the signs of any neurologic injury should be sought before and after any reduction attempt**. Anterior shoulder dislocations with humeral fractures have a 45% incidence of nerve injury, with the axillary nerve being injured in up to 36% of cases.[12] Older patients tend to be more prone to nerve injury from the dislocation and the reduction techniques.[12] The techniques described that cause significant downward traction typically cause more reduction-induced neurologic injuries. Most neurologic injuries are neurapraxias and will completely resolve

within 2 to 5 months.[18,176-179] A small percentage of axillary nerve injuries that do not resolve may require nerve grafting. Brachial plexus injuries are much more common in posterior and inferior shoulder dislocations. Injuries to the artery should raise suspicion for a brachial plexus injury because the brachial plexus surrounds the axillary artery.

DISLOCATION RECURRENCE

The incidence of recurrence is variable, age-dependent, and gender-dependent.[149,180-182] Among patients under the age of 20 years, 90% will dislocate again, whereas only 14% of those over the age of 40 will dislocate again.[1,183,184] Recurrences are much more common in men with a ratio of 4:1 as compared to 6:1 in women.[18] Recurrent shoulder dislocations have other associated morbidities. A triad of lesions (e.g., a detached labrum and anterior capsule, a Hill-Sachs deformity, and erosion of the anterior glenoid) develops in 85% of recurrent shoulder dislocations.[4] The methods for reduction are not different from those of a first-time shoulder dislocation. Patients who have had multiple shoulder dislocations are generally easier to reduce using nonanalgesic manipulation techniques. The orthopedic literature suggests that three shoulder dislocations in a single extremity indicate the need for surgical repair.[18]

HEMARTHROSIS

Blood collections in the shoulder joint are rare complications and are seen almost exclusively in traumatic shoulder dislocations associated with fractures. Patients older than 60 years of age will return to the Emergency Department within 24 to 48 hours with a tense, swollen, and painful shoulder. The shoulder joint should be aspirated (Chapter 97). Aspiration is usually sufficient to relieve pain and restore function.

INABILITY TO REDUCE

There are a few reasons for the inability to reduce a dislocated shoulder completely.[185,186] The most common is inadequate medication and sedation to overcome muscle spasm and pain. The humeral head may be "buttonholed" through the joint capsule.[18] A fracture fragment may be impinged or interposed between the humeral head and the glenoid cavity. Significant or complete disruption of ligamentous structures or soft tissue interposition will not allow the humeral head to remain in the glenoid cavity.[185] The inability to reduce a shoulder dislocation is an indication for reduction, open or closed, under general anesthesia in the Operating Room.[187]

SUMMARY

Shoulder dislocations are common due to the inherent instability of the glenohumeral joint. There are three different types of dislocation, each with different mechanisms of injury and risks of associated injuries. Most dislocations are anterior shoulder dislocations. The diagnosis of a shoulder dislocation is generally uncomplicated given the history and patient presentation. The EP must be expeditious in reducing the dislocated joint once the patient is stabilized, other injuries have been ruled out, pain control has been addressed, and radiographs have been obtained to confirm the type of dislocation along with associated bony injuries.

Orthopedic Surgeons may need to be involved with the acute care of a dislocated shoulder. They should be involved in the initial reduction care of all posterior and inferior dislocations because of the rarity of these shoulder dislocations, the difficulty of reduction, the high incidence of associated injuries, and the need to operate to repair the associated injuries.

TABLE 102-1 The Techniques and Their Success Rates

	Technique	Success rate (%)	References
Anterior shoulder dislocation	Hennepin	80	3
	External rotation	78–97	74, 76, 78–80
	Stimson	92–96	3, 4, 84
	Scapular manipulation	79–96	3, 4, 87–92
	Traction-countertraction	> 90	2
	Snowbird	97	94
	Milch	70–90	72, 95–102
	Hippocratic	> 90	2, 18, 106
	Kocher	98–100	2, 18, 109
	Eskimo	74–77	82, 110
	Chair	62–100	3, 11, 113, 114
	Wrestling	85	115
	Pneumatic stretcher	100	116
	Spaso	67–88	117–121
	Oza	100*	122
	Best of both (BOB)	Unknown	123
	Legg reduction	Unknown	124
	Cunningham	100*	125,126
	Boss-Holzach-Matter	60–90	127–129, 135
	Fares	78–95	131, 188
	Gonais	Unknown	132
	Sool	75	133
	Davos	60–90	128, 129, 135, 136
Posterior shoulder dislocation	Traction-countertraction	> 90	2, 4, 18
Inferior shoulder dislocation	Traction-countertraction	98–100	2, 3, 101
	Two-step	100*	103, 144

*Small number of patients and unreliable results.

Multiple closed reduction techniques are available. They have similar success rates (**Table 102-1**). The method chosen, as well as the decision to use analgesia, is individualized to the EP and the patient. Certain traditional techniques (i.e., Hippocratic and Kocher) have been demonstrated to have a higher incidence of complications and should be avoided. Patients should be thoroughly evaluated before and after any closed reduction attempt for neurologic, vascular, soft tissue, or bony injury. The shoulder should be immobilized after it is reduced.

Patients should be instructed on proper aftercare and provided with adequate oral analgesia. This can be accomplished with nonsteroidal anti-inflammatory drugs supplemented with narcotic analgesics. All patients discharged from the Emergency Department should follow-up with an Orthopedic Surgeon within 1 day to 1 week, depending on the associated injuries and the patient age.

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103 Elbow Joint Dislocation Reduction

Angelique S. Kelly Campen

INTRODUCTION

The elbow is inherently subjected to dislocations because of its mechanical structure.[1] Elbow dislocations are one of the more common joint dislocations in the body, second only to dislocations of the shoulders and fingers.[2] Injuries to the elbow have a high potential for complications and residual disability.[3] **Timely reduction is imperative to relieve pain and reduce the possibility of neurovascular sequelae.**[3] Closed reduction of the elbow is unlikely to be successful if not performed promptly.[4]

The most common mechanism for a dislocation is a fall onto an extended and abducted arm.[5] The patient usually presents with a swollen and painful arm that is held in flexion. Elbow dislocations require a significant amount of force. Up to 20% of elbow dislocations are associated with fractures.[2] Simple elbow dislocations have a better prognosis and are less likely to require surgical intervention than complex ones (i.e., fracture-dislocations). This chapter deals with the closed reduction of simple elbow dislocations.

One particular type of dislocation pertains primarily to the pediatric population. Subluxation of the radial head, often referred to as a "nursemaid's elbow," occurs commonly in preschool children. It is rarely seen after age 7 and represents 20% of upper extremity injuries in children.[5] It occurs after sudden traction on the radius with an extended elbow, as when an adult pulls a child up into a standing position by one arm. The annular ligament slips between the capitellum and the head of the radius impeding supination of the arm. The patient will present with the arm held in slight flexion and pronation, usually in not much distress, and not using the affected arm. The simple reduction of this dislocation is also addressed. Refer to Chapter 104 for a more detailed discussion of this topic.

ANATOMY AND PATHOPHYSIOLOGY

The elbow is a hinge joint comprising articulations between the humerus, the ulna, and the radius (**Figure 103-1**). The distal humerus consists of the extraarticular medial and lateral epicondyles which are diverging columns separated by the intraarticular trochlea and capitellum. The trochlea articulates with the proximal ulna. The articular surfaces of the trochlea extend from the coronoid fossa anteriorly to the olecranon fossa posteriorly. The anterior and posterior fossae provide space for the coronoid and olecranon, respectively, at the extremes of motion. The capitellum is a spherical structure that articulates with the concave radial head.

Numerous neurovascular structures cross the elbow (**Figure 103-2**). The prominent medial epicondyle protects the ulnar nerve which travels in its posterior sulcus. The radial nerve travels just anterior to the lateral epicondyle. The median nerve travels with the brachial artery through the antecubital fossa.

There are four ligamentous structures of importance in considering injuries to the elbow: the radial collateral ligament, the ulnar collateral ligament, the annular ligament, and the anterior capsule (**Figure 103-3**). The annular ligament and radial collateral ligament hold the radial head in position. The annular ligament allows the radial head to rotate under it during pronation and supination.

The relationship of the radius and ulna to the humerus is used to classify elbow dislocations into posterior, anterior, medial, lateral, and divergent (**Figure 103-4**).[6,7] The majority of elbow dislocations are posterior in direction (**Figure 103-5**) and account for 80% to 90% of dislocations.[7-9] The other types of elbow dislocations are uncommon.[9] The radius and ulna are held tightly together by the annular ligament and the interosseous membrane (**Figure 103-3**). Posterior elbow dislocations result in the radius and ulna projecting posterior to the humerus (**Figures 103-4*A* and 103-5**). The radius and ulna may be slightly lateral or medial in posterior elbow dislocations in addition to being posteriorly displaced. This does not affect the management or prognosis. The presence of a fracture of the radial head or coronoid process may frequently render any attempt at reduction unstable and will usually require an open reduction.[10]

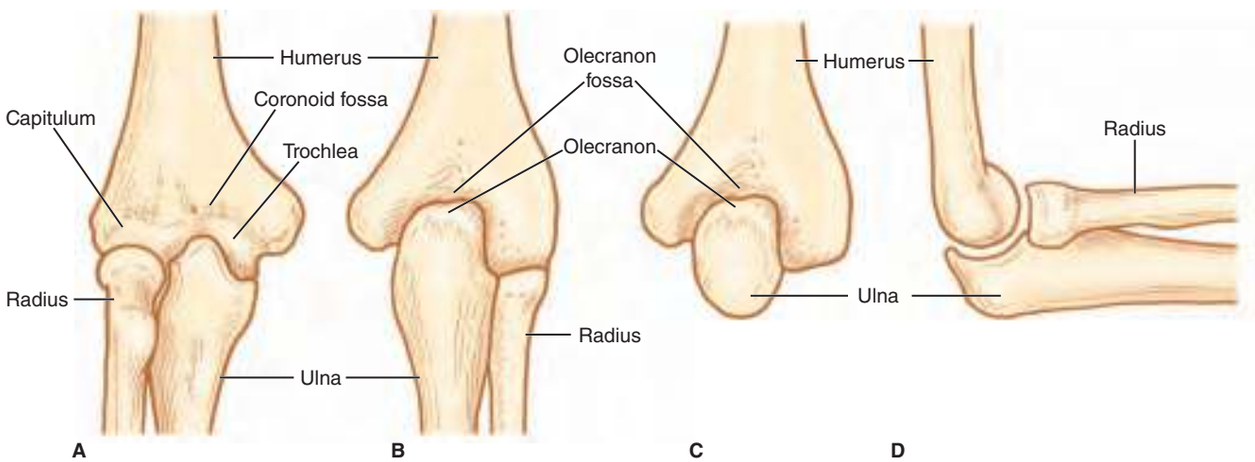

FIGURE 103-1. Bony anatomy of the elbow region. The right arm is demonstrated in these illustrations. ***A.*** Anterior view. ***B.*** Posterior view. ***C.*** Posterior view of the elbow in 90° of flexion. ***D.*** Lateral view of the elbow in 90° of flexion.

FIGURE 103-2. Major neurovascular structures that cross the elbow.

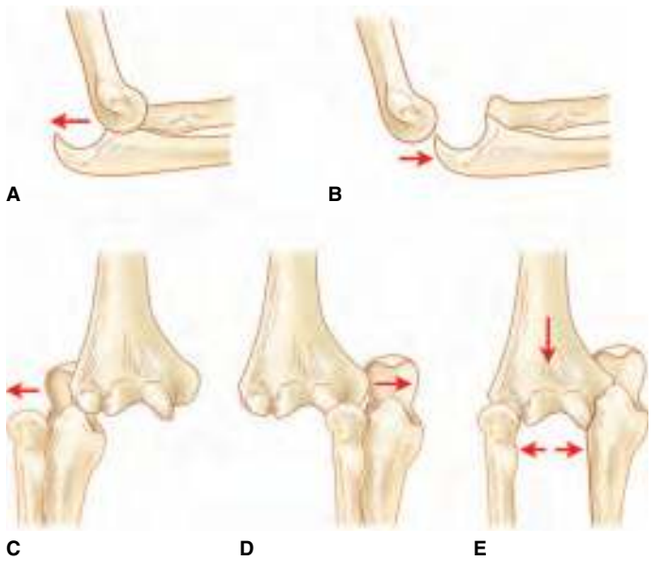

FIGURE 103-4. Classification of elbow dislocations. ***A.*** Posterior. ***B.*** Anterior. ***C.*** Lateral. ***D.*** Medial. ***E.*** Divergent.

Anterior elbow dislocations occur from traction of the forearm with the elbow extended or a blow to the posterior aspect of the flexed elbow. Anterior elbow dislocations result in the radius and ulna projecting anterior to the humerus (**Figure 103-4*B***). Medial (**Figure 103-4*C***) and lateral (**Figure 103-4*D***) dislocations are rare injuries with poorer prognoses. Divergent dislocations (**Figure 103-4*E***) are rare injuries that are distinct from the other types of elbow dislocations because there is dissociation of the radius and ulna. The annular ligament and interosseous membrane must be torn for a divergent dislocation to occur.

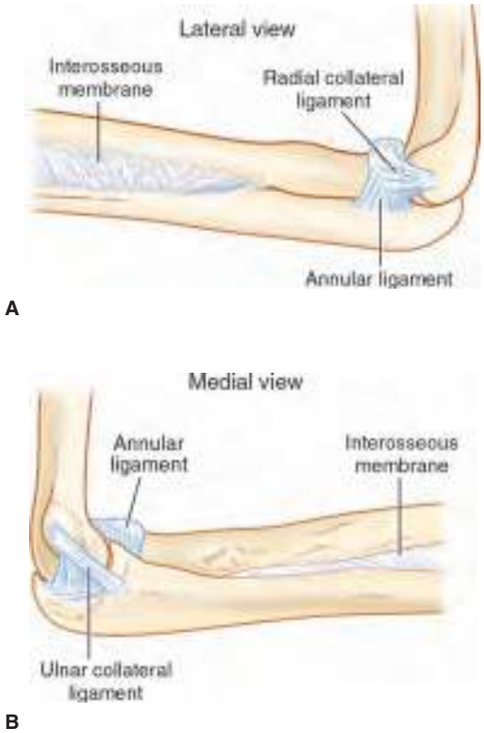

FIGURE 103-3. Major ligamentous structures of the elbow. ***A.*** Lateral view. ***B.*** Medial view.

The patient usually presents with pain and swelling of the elbow. All elbow dislocations are characterized by loss of the normal relationship of the humeral epicondyles to the tip of the olecranon (**Figure 103-5**). The bony landmarks may be identified if the patient is seen immediately after the injury. However, the swelling and hemarthrosis that develop over time make it difficult to palpate these landmarks. Posterior dislocations are further apparent by a shortening of the forearm and the elbow being fixed in flexion.

INDICATIONS

All elbow dislocations require reduction. Early reduction of a dislocation, by open or closed means, is of paramount importance if good functional results are to be obtained. Closed reduction is unlikely to be successful if attempted later than 14 days after the injury.[4] The more promptly the reduction is attempted, the more likely it is to be successful.

The vascular status of the extremity also dictates the need for emergent relocation. **Emergent and immediate relocation is necessary when there is neurologic or vascular compromise of the distal extremity.** Relocation can await titration of sedation and analgesia when the extremity is neurovascularly intact.

CONTRAINDICATIONS

There are no absolute contraindications to the closed reduction of an elbow dislocation. A relative contraindication to the procedure is when there is uncertainty, before radiographic evaluation, regarding whether the injury is a dislocation or a fracture. Closed reduction is not indicated if there is an interposed osteochondral fragment preventing concentric reduction or when there is a concomitant displaced fracture of the radial head or neck. Elbows that have been dislocated for a prolonged period of time may have closed reduction attempted but will most likely require an open procedure.

EQUIPMENT

- Towel or sheet to aid in applying traction
- Splinting materials
- Weight (sandbag, bucket with water, or any other weight)
- Procedural sedation supplies (Chapter 159)

A

B

FIGURE 103-5. The posterior elbow dislocation. ***A.*** Photo of the patient's arm. (Used with Permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 3rd ed. New York: McGraw-Hill, 2009. Photo contributor: Frank Birinyi, MD.) ***B.*** Radiograph.

PATIENT PREPARATION

Explain the risks, benefits, and potential complications to the patient and/or their representative. The postprocedural care should also be discussed. Obtain an informed consent for the reduction procedure.

Carefully assess and document the preprocedural neurologic (median, radial, and ulnar nerves) and vascular (brachial, radial, and ulnar arteries) status of the extremity. Splint and/or sling the affected extremity until radiographs are obtained and a closed reduction can be performed. Obtain anteroposterior and lateral radiographs to confirm the diagnosis of an elbow dislocation. Oblique views may be helpful to further define the relationship between the distal humerus, radius, and ulna. **The only case for which a preprocedure radiograph is not indicated is when neurologic or vascular compromise exists in the distal extremity and an expeditious reduction is required.**

Closed reduction of elbow dislocations requires adequate analgesia and muscle relaxation. In most cases, procedural sedation is useful.[5,7] Regional anesthesia (axillary nerve or Bier blocks) is useful if procedural sedation is contraindicated.[11] Refer to Chapters 156 and 157 for details regarding regional anesthesia of the upper extremity. General anesthesia with fluoroscopy is rarely necessary unless the dislocation is associated with an undisplaced fracture of the radial head or neck.

TECHNIQUES

POSTERIOR ELBOW DISLOCATION REDUCTION TECHNIQUES

The goal of closed reduction is to distract the radius and ulna, allowing them to relocate. **The preferred technique that provides the least complications is a modification of the Stimson technique used for shoulder dislocations.**[12] Place the patient prone with the affected arm hanging off the gurney (**Figure 103-6**). Place padding anteriorly in front of the arm and shoulder so that the arm does not drag along the side of the gurney. **Care must be taken that the patient does not fall from the gurney.** Tie a sheet circumferentially around the patient's hips and the gurney. **Ensure that the patient does not have any respiratory difficulty while in the prone position.** Suspend a weight from the patient's wrist to apply traction to the arm (**Figure 103-6**). The weight should be approximately 5 pounds and may go up to 15 pounds, depending on the patient's musculature and weight. Allow the arm to dangle over the edge of the gurney for 10 minutes. If the elbow dislocation will not reduce, attempt the traction-countertraction technique.

The traction-countertraction technique can be performed but is not the optimal approach. Place the patient's arm in slight flexion. Grasp the patient's mid-humerus with the nondominant hand

FIGURE 103-6. The modified Stimson technique for reducing posterior elbow dislocations.

A B

FIGURE 103-7. The traction-countertraction technique to reduce a posterior elbow dislocation. ***A.*** The one-person technique. The physician stabilizes the humerus with one hand and distracts the forearm with the other hand. ***B.*** The two-person technique. The assistant stabilizes the humerus and provides countertraction while the physician applies traction to the forearm.

and the patient's wrist with the dominant hand (**Figure 103-7*A***). Alternatively, an assistant can grasp the humerus while the physician uses both hands to grasp the patient's wrist (**Figure 103-7*B***). Stabilize the patient's humerus. Apply steady and constant traction to the patient's wrist to distract the coronoid process and allow it to slip past the humerus and back into anatomic position. The application of downward pressure on the proximal forearm can help to disengage the coronoid process from the olecranon fossa and ease the reduction.

MEDIAL AND LATERAL ELBOW DISLOCATION REDUCTION TECHNIQUE

Medial and lateral dislocations of the elbow are extraordinarily uncommon. They are usually associated with a posterior elbow dislocation. These dislocations can be reduced in a similar manner using the traction-countertraction technique used for posterior elbow dislocations. For lateral dislocations, apply traction to the patient's wrist with one hand while using your other hand to guide the proximal ulna downward, then medially, and finally backward. For medial dislocations, apply traction to the patient's wrist with one hand while using your other hand to guide the proximal ulna downward then laterally.

Emergency Physicians should not reduce a medial or lateral elbow dislocation. They are uncommon, may be associated with neurovascular complications, have severe ligamentous tears, and should be reduced by an Orthopedic Surgeon. **The only exception to this is if there is neurologic and/or vascular compromise of the distal extremity and no Orthopedic Surgeon is immediately available.**

ANTERIOR ELBOW DISLOCATION REDUCTION TECHNIQUE

Anterior elbow dislocations are uncommon. They are often associated with intimal injuries to the brachial artery being stretched during the injury.

Anterior elbow dislocations should be reduced by an Orthopedic Surgeon for the same reasons as a medial or lateral elbow dislocation. **The only exception to this is if there is neurologic and/or vascular compromise of the distal extremity and no Orthopedic Surgeon is immediately available.**

DIVERGENT ELBOW DISLOCATION REDUCTION TECHNIQUE

Fortunately, divergent dislocations are exceedingly rare. They are commonly associated with severe articular damage, interosseous ligamentous tears, neurologic injuries, and vascular injuries. The reduction technique is complex; the elbow is reduced as a two-part dislocation and often requires surgical fixation to be stabilized. Simple traction can be applied to relieve stress on the neurovascular structures but will not reduce the dislocation due to the instability of the joint.

Divergent elbow dislocations should be reduced by an Orthopedic Surgeon in the Operating Room. **The only exception to this is if there is neurologic and/or vascular compromise of the distal extremity and no Orthopedic Surgeon is immediately available.**

RADIAL HEAD SUBLUXATION REDUCTION

This condition is also referred to as a "nursemaid's elbow." Reduction of a radial head subluxation is a maneuver involving supination or hyperpronation flexion of the affected forearm. The forearm is quickly supination or hyperpronation the elbow flexed completely in one smooth motion. A pop or click is sometimes heard or felt by the Emergency Physician as the subluxation is reduced. Most patients are asymptomatic within 5 to 10 minutes and 90% within 30 minutes.[13] The Emergency Physician should leave the room for 5 to 10 minutes after the procedure. Ask the parents to distract the child to see if they begin to move the arm. A fearful child will often not use a successfully reduced arm in fear of pain. Place an object (e.g., car keys, a popsicle, a toy, or another object the child may want) within grasp of the reduced extremity to encourage the child to use the extremity. Refer to Chapter 104 for the complete details regarding the reduction of a radial head subluxation.

ASSESSMENT

After a reduction, gently move the joint through the entire range of motion to ensure smooth movement and proper joint reduction. This also tests for joint stability and whether or not the joint will easily redislocate. The joint may need to be repaired operatively if it dislocates during this examination. **The neurovascular status of the extremity must again be evaluated and documented.** The integrity

FIGURE 103-8. Postreduction radiograph demonstrating a fracture of the coronoid process.

of the median, ulnar, and radial nerves as well as the brachial artery and its distal branches must be evaluated and documented. All reductions except radial head subluxations should have postprocedural radiographs to ensure proper bony alignment and the lack of a fracture (**Figure 103-8**). If full and smooth passive range of motion is not possible, which is especially common in children, postreduction radiographs should be examined for entrapment of the medial epicondyle.

AFTERCARE

Place the reduced extremity in a posterior long arm splint, from the mid-humerus to the base of the fingers, with the elbow in 90° of flexion. **Do not apply a circumferential cast due to the subsequent swelling and edema.** Suspend the arm with a sling to aid in elevating the extremity.[14] The patient should be carefully observed for 12 to 36 hours for vascular impairment. Instruct the patient to return to the Emergency Department if they develop weakness, numbness, paresthesias, cold fingers, or cyanotic fingers. The patient should be admitted for observation if there is any question of neurovascular compromise. Gentle range-of-motion exercises can be started as early as 3 to 5 days after reduction if the elbow is stable.[14-18] Schedule a follow-up with an Orthopedic Surgeon 3 to 4 days after reduction to test for joint stability. Prescribe nonsteroidal anti-inflammatory drugs supplemented with narcotic analgesics to control pain.

No immobilization is necessary for a radial head subluxation that is reduced. Immobilization in a sling with follow-up by an Orthopedic Surgeon is recommended only for recurrent radial head subluxations.

COMPLICATIONS

Several serious complications exist with simple elbow dislocations.[19,20] The most serious and first to happen are ischemic complications. Damage to and obstruction of the brachial artery can occur with any of the elbow dislocations. Brachial artery injury occurs in 5% to 13% of patients with an elbow dislocation.[21] It is a serious complication that can occur even without an associated fracture.[21] **The presence of a distal pulse is not proof that there is no vascular injury.** Collateral circulation around the elbow can result in a distal pulse despite a complete brachial artery laceration or occlusion.[22] Signs of an arterial injury include cyanosis, an expanding hematoma, pallor, pulselessness, and severe pain. Any suspicion of a brachial artery injury necessitates prompt angiography.[22,23]

The second serious complication resulting from an elbow dislocation is nerve injury from traction or entrapment. Loss of median nerve function after reduction should prompt an immediate consultation with an Orthopedic Surgeon. The ulnar nerve is most commonly injured.[24,25] It is seen in 8% to 20% of patients with posterior elbow dislocations.[24]

Fractures commonly occur with elbow dislocations. **Radiographs should always be obtained before and after any attempt at reduction. The only exception to obtaining prereduction radiographs is if the extremity has signs of distal neurovascular compromise and obtaining radiographs will delay the reduction.** A fractured coronoid process can sometimes become entrapped in the joint requiring an open reduction. Fractures of the coronoid process are commonly associated injuries and will usually come into near-normal opposition once reduction occurs (**Figure 103-8**). Large fragments that are displaced may require operative fixation.

Late complications of simple elbow dislocations include ectopic ossification, occult distal radioulnar posttraumatic stiffness, posterolateral joint instability, and residual pain.[16,26]

SUMMARY

Elbow dislocations are the second most common large joint dislocations that occur in adults. The majority of dislocations are posterior elbow dislocations, although the radius and ulna can dislocate into just about any other position. Relocation involves distracting the forearm while stabilizing the humerus and putting pressure counter to the direction of the dislocation. It is not uncommon to have an associated fracture, so radiographic studies are imperative. The neurovascular status of the extremity must be carefully monitored and documented both before and after any attempts at reduction. Splint the elbow in flexion after reduction. Follow-up with an Orthopedic Surgeon and early range-of-motion exercises are recommended to ensure proper joint function.

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104 Radial Head Subluxation (Nursemaid's Elbow) Reduction

Mark P. Kling

INTRODUCTION

Subluxation of the radial head is one of the most common pediatric orthopedic injuries. It is also known as nursemaid's elbow and a pulled elbow. This can occur in children whose age ranges from less than 6 months to the preteens. Most radial head subluxations occur between 1 and 3 years of age.[1,2] It is a rare injury before 1 year of age and after 8 years of age. The basic cause is a pulling of the child's arm even though the exact cause varies.[3,4] The injury is limited to small children because the radial head is less bulbous than in older children, adolescents, and adults making it easier to become subluxed. Fortunately, it can be easily reduced in the Emergency Department.[5]

ANATOMY AND PATHOPHYSIOLOGY

The classic mechanism involves axial or longitudinal traction on an extended elbow with the forearm pronated. This often occurs while someone is holding onto the child by the hand or wrist. The child is then pulled or falls while being held and is suspended by the arm. The subluxation is seen more often in the left arm than the right.[6] This is due to more people being right-handed and holding the child's left hand or wrist while walking. It is not uncommon, however, for the child to present with a history of a fall or rolling over in bed.[1]

This orthopedic injury involves the region of the elbow (**Figure 104-1**). The annular ligament is a thick band that wraps around the upper radial neck and radial head (**Figure 104-1*A***). It guides the radial head as the forearm moves through pronation and supination. **The injury causes the radial head to become partially dislocated from its articulation with the ulna and the capitellum of the humerus while the forearm is in a pronated state.** The annular ligament then slips proximally and its lateral end becomes entrapped between the radial head and the capitellum (**Figure 104-1*B***). **The forearm becomes locked in pronation due to the entrapped annular ligament.** This condition is painless when the forearm is held in pronation. Supination of the forearm causes pain so the child holds the extremity in pronation. The act of supination would also spontaneously return the annular ligament to its anatomic position and reduce the subluxation.

FIGURE 104-1. Anatomy of the elbow region. ***A.*** Normal anatomy. ***B.*** A radial head subluxation. Note the entrapped annular ligament.

Children will present in no apparent distress.[7] They are usually resting comfortably and have some reservation in using the affected extremity. The arm will be held with slight flexion of the elbow and pronation of the forearm (**Figure 104-2*A***). The child may point to an area of pain, but this is not often the case. A child may be much more comfortable with the parent examining and questioning areas of tenderness as opposed to the unknown and sometimes intimidating Emergency Physician.

Radiographs are not required unless other trauma or diagnoses are suspected. The child often returns from the radiology suite, if radiographs are ordered, using the affected extremity. Radial head subluxations often reduce spontaneously during positioning for radiographs.

Bedside ultrasound can be used to quickly exclude other diagnoses.[8-10] Ultrasound is accurate, easy to perform, quick, and uses no ionizing radiation. The ultrasound can be used to see the annular ligament is displaced from its normal position around the proximal radial head.[8] The normal posterior fat pad rules out other bony pathology.[9] A "hook sign" may be seen on ultrasound.[10] This is when the supinator muscle is pulled into the joint space giving the appearance of a "hook" over the area of the radial head.

INDICATIONS

Any child presenting with the inability to utilize the partially flexed elbow, the forearm pronated and adducted, and a mechanism for a radial head subluxation should be reduced. Many Emergency Physicians may be hesitant to repeat the procedure multiple times if a child was not using the arm normally within 15 to 30 minutes after a clinically successful reduction. A decision to repeat the reduction should be considered if the radiographs appear normal and a repeat history and physical examination are consistent with the original diagnosis.

CONTRAINDICATIONS

The presence of edema, ecchymoses, tenderness other than over the radial head, suspicion of a fracture, or a mechanism of injury not consistent with a radial head subluxation should first be

A

B

C

FIGURE 104-2. A child with a radial head subluxation. ***A.*** The subluxed left forearm is held flexed, pronated, and adducted. ***B.*** Reduction technique. ***C.*** The forearm is freely mobile after reduction with normal extension and abduction.

evaluated radiographically.[11] **The presence of any distal neurologic or vascular compromise excludes the diagnosis of a radial head subluxation.**

EQUIPMENT

No equipment is required for the reduction of a radial head subluxation.

PATIENT PREPARATION

Explain the risks, benefits, potential complications, and aftercare of the procedure to the child and their representative. Obtain a signed consent to perform the reduction. Place the patient sitting in the parent's lap or supine on an examination table or gurney. No premedication is required.

TECHNIQUES

SUPINATION AND FLEXION

Place one hand on the child's elbow with the thumb over the radial head (**Figure 104-3*A***). This will aid in palpation of the traditional "click" of reduction. Gently grasp the child's wrist with the other hand (**Figure 104-3*A***). Perform the following maneuvers in one smooth motion to reduce the subluxation. Apply distal traction while supinating the forearm (**Figure 104-3*B***) followed by flexion of the elbow (**Figure 104-3*C***). A click may be felt as the radial head is reduced.[1,12] If not, flex the forearm until the hand is upright. Other methods of reduction are often compared to this method.[13,14]

HYPERPRONATION

Some feel that hyperpronation causes less pain to the child and is more successful than the supination technique.[14-23] This is because the arm is already being held in pronation and the additional pronation (i.e., hyperpronation) requires less force and motion than the supination technique.[19,20]

Position the patient as described above. There are two variations on this technique. The first holds the child's elbow at 90° of flexion and hyperpronates the forearm without any additional flexion at the elbow (**Figure 104-4**).[14] The second holds the child's elbow at 90° of flexion and hyperpronates the forearm while simultaneously fully flexing the elbow (**Figure 104-5**).[15] Both hyperpronation techniques can successfully reduce a radial head subluxation.

ASSESSMENT

Allow 5 to 15 minutes to pass and return for a repeat examination. Children may cry at the end of the procedure but will generally only do so for a moment. As the child feels more comfortable, they will proceed to use the arm (**Figure 104-2*C***). This freedom of use can be accelerated by the caregiver or physician stimulating the patient

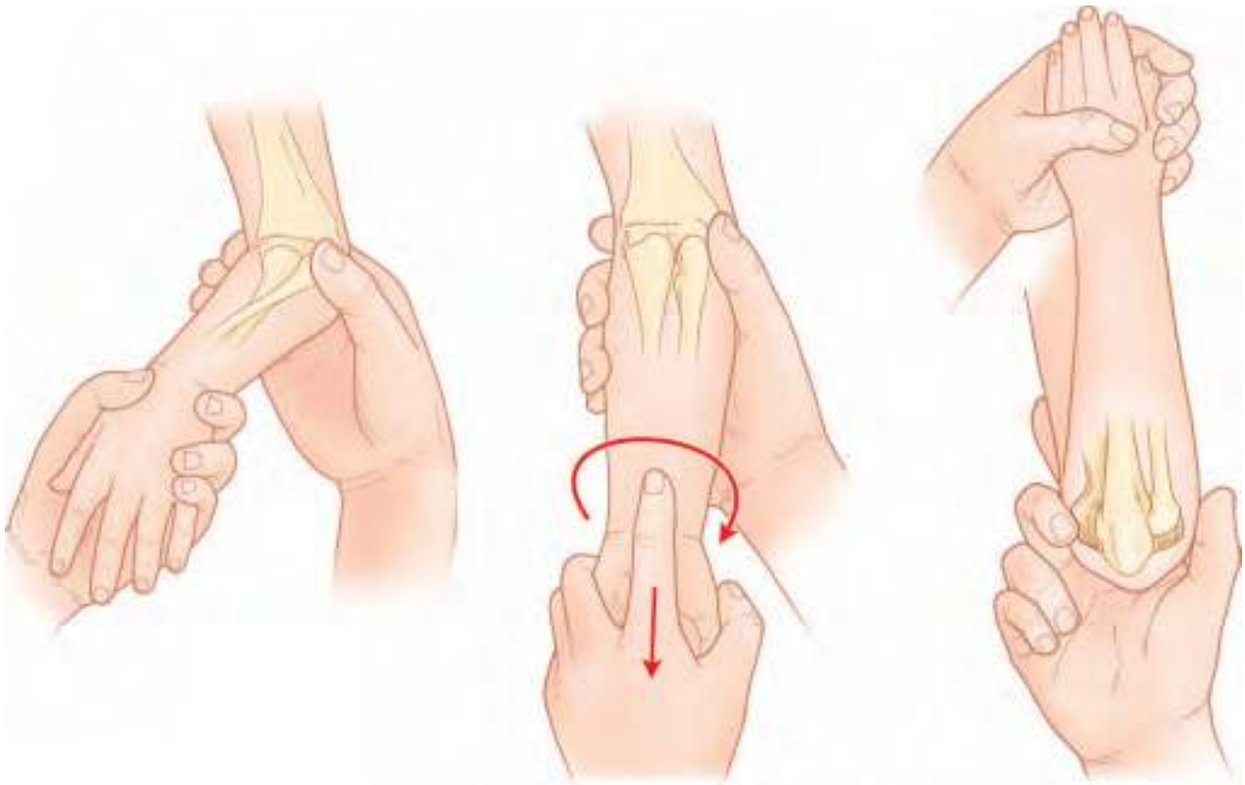

FIGURE 104-3. Reduction of a radial head subluxation using supination and flexion. ***A.*** Positioning of the physician's hands. ***B.*** Distal traction is applied (*straight arrow*) while supinating the forearm (*curved arrow*). ***C.*** The forearm is maximally flexed.

to use the arm with an incentive (e.g., by offering candy, a pen, or a popsicle for the child to grab).

The child should have uninhibited use of the forearm within 30 minutes. **Reconsider the diagnosis if the child is not using the extremity.** Consider radiography or ultrasonography of the affected extremity. Alternative diagnoses include clavicular fractures, distal humeral fractures, osteomyelitis, radial head fractures, septic arthritis, stress fractures, and Monteggia fractures. Reevaluate the elbow joint for signs of trauma. Obtain plain radiographs if not done previously or consider ultrasound for evaluation and reevaluation. Full recovery may take 24 to 48 hours if the reduction is delayed for more than 8 hours from the time of injury.

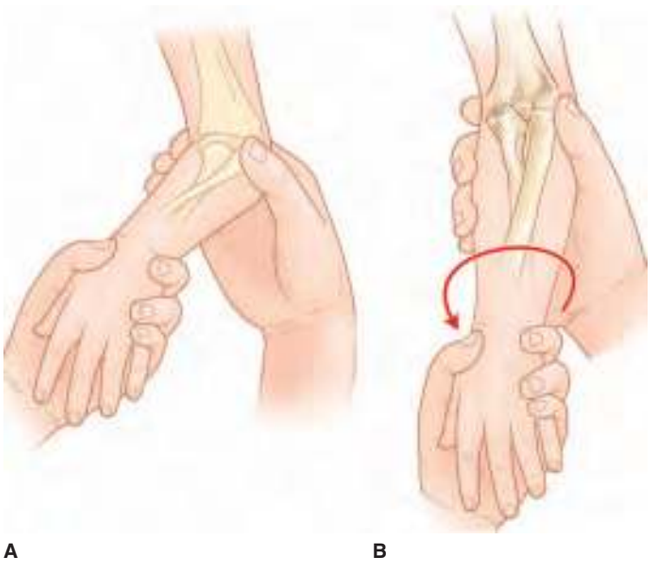

FIGURE 104-4. Reduction of a radial head subluxation using hyperpronation. ***A.*** Positioning of the physician's hands. ***B.*** Hyperpronation of the forearm.

AFTERCARE

Radiographs, immobilization, splinting, analgesics, and orthopedic follow-up are not necessary if the subluxation is reduced and the child is using their arm. Educate the caregiver regarding the mechanism of injury and prevention of future subluxations.

Phone consultation with an Orthopedic Surgeon is recommended if the reduction is unsuccessful. It is not unusual to have a spontaneous reduction on repeat examination and follow-up. Immobilize the arm until the child is evaluated by an Orthopedic Surgeon. Immobilization will aid in pain relief. Consider the use of nonsteroidal anti-inflammatory drugs.

COMPLICATIONS

There are no complications associated with the reduction of a radial head subluxation. Rarely, the subluxed radial head will not reduce.[24] This is due to buttonholing of the radial head through the annular ligament or the brachialis tendon. Consult an Orthopedic Surgeon in these cases.

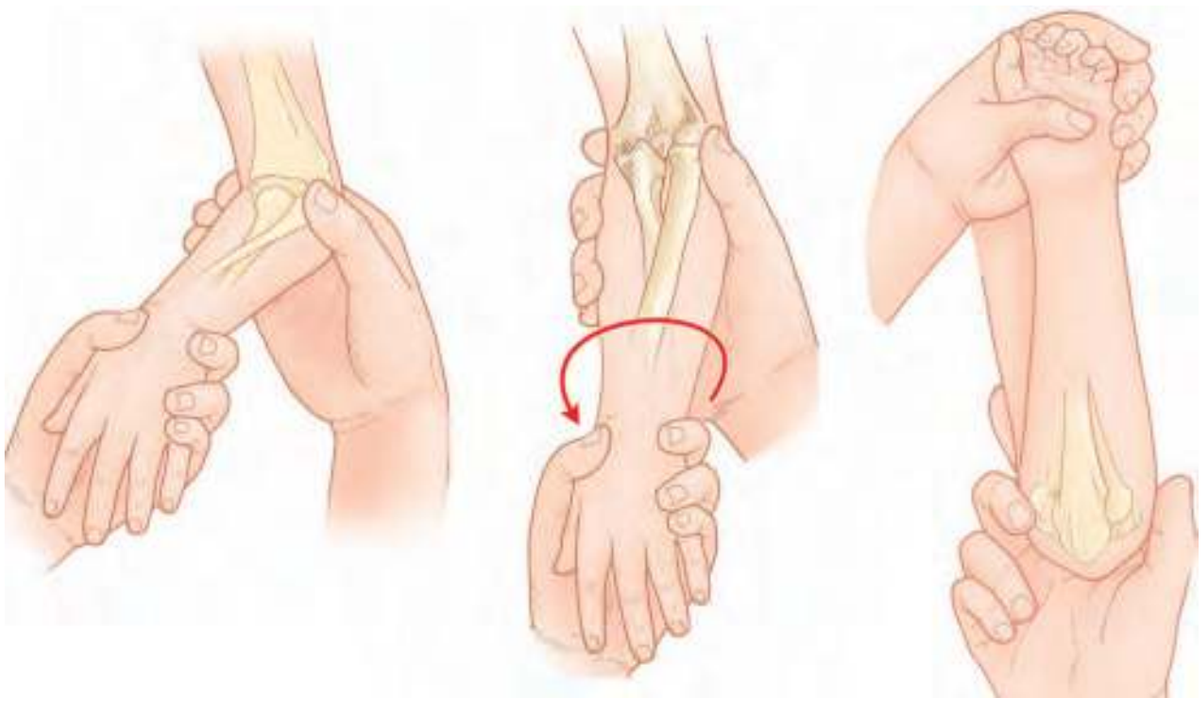

FIGURE 104-5. Reduction of a radial head subluxation using hyperpronation and flexion. ***A.*** Positioning of the physician's hands. ***B.*** Hyperpronation of the forearm. ***C.*** Flexion of the hyperpronated forearm.

SUMMARY

A radial head subluxation is one of the more common orthopedic injuries of childhood. Children present with the inability to use the affected upper extremity. They hold the forearm flexed, pronated, and adducted. The reduction technique is quick and simple. Some more recent reviews suggest more effective and less painful results with hyperpronation techniques versus the supination method. It is important to educate the caregivers regarding the mechanism of injury and prevention of future subluxations.

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105 Carpometacarpal Dislocation Reduction

Rene Pineda Carizey and Priya Perumalsamy

INTRODUCTION

The hand is frequently injured. Carpometacarpal (CMC) dislocations are an uncommon injury, composing less than 1% of all hand and wrist injuries.[1,2] The mechanism usually occurs during hyperextension from high-velocity blunt trauma, mainly punching or falling.[2] The most commonly involved joints are the fourth and fifth CMC joints. Most injuries are dorsal dislocations, with volar dislocations being less common. These dislocations are usually seen in adults.

Missed diagnosis of a CMC dislocation can result in chronic pain and decreased function of the affected hand. Up to 87% of patients with CMC injuries will return to full daily activities with negligible pain if they receive appropriate management.[3] The method of optimal treatment is still being debated. CMC dislocations are intrinsically unstable and usually require a Hand Surgeon evaluation for surgical fixation with percutaneous pinning, plates, or screw placement after closed or open reduction. There have been rare cases of closed reduction with conservative immobilization for CMC dislocations without associated fracture.[4-8]

ANATOMY AND PATHOPHYSIOLOGY

There are numerous bones in the hand and wrist (**Figure 105-1**). The CMC joints of the hand are gliding-type joints (i.e., arthrodial diarthrosis). The bases of the metacarpals articulate with the distal row of the carpal bones and with each other using an interlocking mechanism (**Figure 105-1**). Stability at the CMC joints is provided by four ligaments (i.e., the dorsal and palmer metacarpal ligaments and the dorsal and palmar interosseous ligaments). Intermetacarpal ligaments, wrist extensor ligaments, and wrist flexor ligaments that insert at the bases of the second, third, and fifth metacarpals further reinforce and stabilize the joints.[9] Strong anterior joint capsules often result in dorsal dislocations. The thumb and fifth finger have a wide range of motion. The CMC joints of the ring and little finger are relatively more mobile than the rest of the CMC joints. This makes them more susceptible to dislocation injury. The ulnar nerve passes adjacent to the hook of the hamate and just volar to the fifth CMC joint. Dislocation of the fourth or fifth CMC joint can cause traumatic nerve injury. Monitoring for a compartment syndrome (Chapter 93) is essential as there are 10 separate osteofascial compartments in the hand. These compartments are accessible for surgical release (Chapter 94).

CMC dislocations are rare.[1,2] The thumb CMC joint is rarely dislocated. It usually results from an axial load with flexion of the thumb metacarpal resulting in a dorsal dislocation. A dislocation commonly occurs in association with a fracture. Closed reduction is usually unstable and requires operative stabilization. CMC dislocation of fingers 2 through 5 is also uncommon. It usually affects the fifth CMC joint. A dislocation commonly occurs in association with a fracture. Closed reduction is usually unstable and requires operative stabilization.

FIGURE 105-1. The hand and wrist bones. (Used with permission from Hand Specialists of TX.)

DIAGNOSTICS

Tenderness, swelling, and deformity of the proximal hand are signs of CMC dislocations (**Figure 105-2**).[10] There may be a raised deformity of the dorsal hand with a palmar depression from a dorsal dislocation of the metacarpal base. The proximal metacarpals are palpable. A dorsal depression will be seen with a palpable palmar mass from a volar dislocation. Impaired hand function with decreased motion and strength are usual findings.[11,12] Comparison between the two hands will most likely show asymmetry. Extensive soft tissue swelling may obscure some or all the findings.

Conventional radiographs remain the first-line imaging modality in the acute posttraumatic setting. This can be overwhelming given the complexity of the hand anatomy. CMC dislocations are frequently missed on routine plain radiographs.[5,13,14] Up to 70% of CMC dislocations are missed or misdiagnosed.[14] **Obtain a complete set of hand radiographs (i.e., posteroanterior [PA], oblique, and true lateral) if high clinical suspicion exists of injury and the mechanism of injury is associated with pain or deformity of the hand.** Basic radiographs allow examination of the CMC joints and intercarpal joints (**Figures 105-3 to 105-6**).[15,16]

Recognition of common radiographic findings can prevent delays in diagnosis and treatment. PA and lateral views provide visualization of the second through fifth CMC joints. The 30° internal oblique view helps to isolate the fourth and fifth CMC joints, whereas the 30° external oblique view is useful in evaluating the second and third CMC joints.[12,17] **The apposing joint surfaces should be parallel with a uniform 1 to 2 mm joint space. Overlap of the normally parallel joint surfaces suggests a subluxation or dislocation if the wrist is in a neutral position.**[5] A cortical rim should be

FIGURE 105-2. The hand deformity immediately after trauma. (Used with permission from reference 10.)

FIGURE 105-3. Radiograph of a thumb CMC dislocation. (Used with permission from reference 15.)

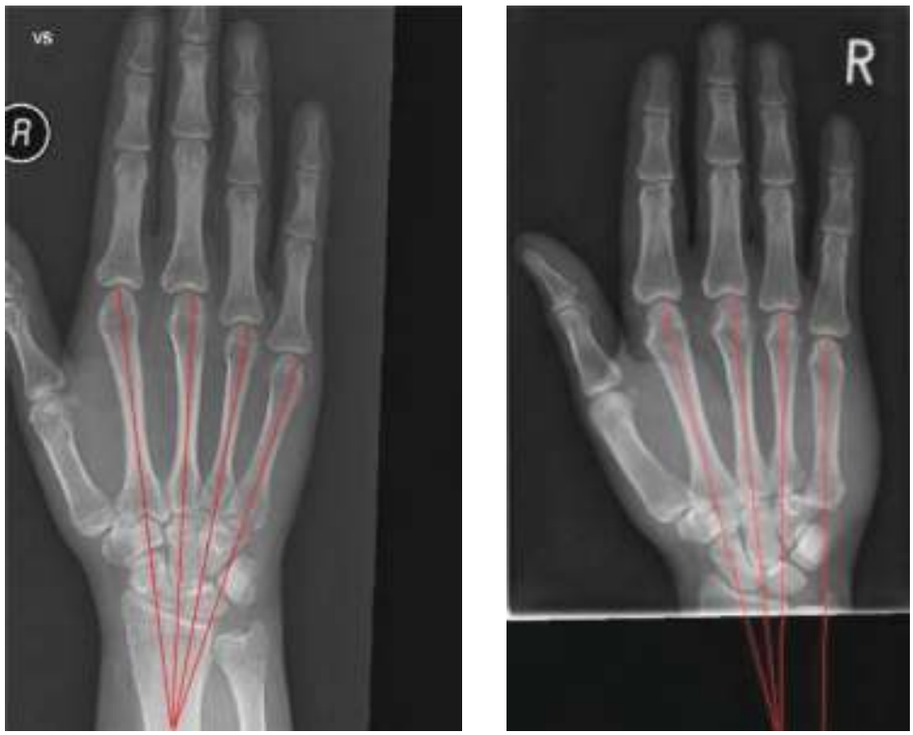
FIGURE 105-5. A lateral radiograph of a fourth and fifth finger CMC dislocation.

seen at apposing margins. Blurring of the cortical rim can be a sign of abnormal angulation of the CMC bones.

A break in the parallel M-line made by following the distal edges of the trapezoid, capitate, hamate, and the bases of the second through first metacarpals indicates an abnormality in the CMC joint (**Figure 105-7**).[11,17] It is also termed the metacarpal cascade line technique. The metacarpal cascade line technique is one method in viewing PA radiographs to help diagnose CMC dislocations. It is more sensitive in diagnosing volar CMC dislocations when compared to other radiograph evaluation techniques.

FIGURE 105-4. A PA radiograph of a fifth finger CMC dislocation. (Used with permission from reference 16.)

FIGURE 105-6. A lateral radiograph of a second and third finger CMC dislocation.



A B

FIGURE 105-7. The PA radiograph metacarpal cascade line technique. ***A.*** Normal convergence. ***B.*** Abnormal convergence seen in a dislocation. (Used with permission from reference 18.)

Superimpose lines over the central longitudinal axis of each metacarpal on the PA radiograph.[18] The lines will converge to a common point approximately 2 cm proximal to the distal radius articular surface when the hand is in neutral position (**Figure 105-7*A***). A divergent metacarpal cascade line(s) indicates a CMC abnormality (**Figure 105-7*B***). Fractures of the metacarpal base or distal carpal bones should heighten suspicion for an associated CMC dislocation. The lateral view can show soft tissue swelling, a shortening of the longitudinal axis, and the direction of dislocation (**Figures 105-4 and 105-5**).[11,17]

Zigzag lines can be drawn between the articular surfaces of the distal row of carpal bones and the metacarpal bases (**Figure 105-8**).[19,20] These zigzag lines represent the hand bone arches. The zigzag lines are normally parallel to each other throughout the CMC joints (**Figure 105-8*A***). Disruption of the parallel lines signifies a dislocation (**Figure 105-8*B***).

Computed tomography (CT) and magnetic resonance imaging (MRI) have traditionally been second-line imaging modalities. Evidence suggests conventional radiograph has limited sensitivity in posttraumatic injury. MRI has the added advantage of detecting

A B

FIGURE 105-8. The zigzag line. ***A.*** Schematic. (Used with permission from reference 19.) ***B.*** A radiograph of a dislocation. (Used with permission from reference 20.)

ligamentous injury in the absence of bone damage. **A hand injury with clinical symptoms suggestive of a CMC subluxation or dislocation with nonspecific or negative radiographic findings is an indication for CT or MRI.**[12,17]

INDICATIONS

Management of these injuries includes prompt reduction of the CMC joint.[5] The goal is a pain-free hand with minimal disability.[5,7,12,15,21] Reduce any dislocation with neurologic or vascular compromise while avoiding additional trauma.

CONTRAINDICATIONS

Contraindications to closed reduction include displaced avulsion fractures, multiple dislocations or fractures, neurologic injury, overlying open wounds, significant intraarticular damage, and vascular injury. These are indications for emergent evaluation and treatment by a Hand Surgeon.[12] Chronic dislocations will require capsulorrhaphy, K-wire fixation, and/or ligament reconstruction.[15] The inability to achieve adequate anesthesia will prevent closed reduction.[22] Reduction with fixation may be more appropriate due to the unstable nature of the joint after closed reduction and risk of chronic posttraumatic disability.[1,12,21,23]

EQUIPMENT

- Local anesthetic solution
- Regional nerve block supplies (Chapter 156)
- Procedural analgesia and sedation supplies (Chapter 159 for details)
- Finger trap (**Figure 105-9**)

FIGURE 105-9. A hand suspended in the finger trap with a counterweight on the arm for traction. (Courtesy of Instrument Specialists Inc., Boerne, TX.)

- Splinting material, fiberglass or plaster
- Elastic bandages (e.g., Ace wrap)
- Sling

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Obtain an additional signed consent for any anesthesia or sedation used for the reduction procedure. Perform a "time out" before the procedure to verify that the patient is correct, the site is correct, and the procedure being performed is correct. Reconcile all medications and allergies. Remove all jewelry or potentially constricting objects from the affected hand or wrist.

Prepare the patient. Position the patient in a position of comfort with the head of the bed at 45°. Place the patient on the cardiac monitor, noninvasive blood pressure cuff, pulse oximetry, and supplemental oxygen to monitor them during and after the procedure if sedation is used. Clean any dirt or debris from the skin. Apply povidone iodine or chlorhexidine solution to the skin surface and allow it to dry. Perform the local injection, regional anesthesia (Chapter 156), or procedural sedation (Chapter 159). Apply the finger trap (**Figure 105-9**).

TECHNIQUES

The closed reduction technique consists mainly of longitudinal traction with direct pressure at the base of the affected metacarpal.

CMC DISLOCATION OF JOINTS 2 TO 5

These dislocation usually require some form of anesthesia for the reduction. Apply traction using the finger trap and a weight (**Figure 105-9**). This unlocks the metatarsal bases from the proximal row of carpals and stretches the ligaments. Apply simultaneous flexion and longitudinal directed pressure on the base of the metacarpals. The dislocated metacarpal(s) should return to their normal position. Obtain a radiograph to confirm the reduction.

CMC JOINT OF THE THUMB DISLOCATION

The CMC joint of the thumb reduces easier than the other fingers. The dislocation is usually dorsal. The reduction is unstable and may sublux. Apply traction using the finger trap and a weight (**Figure 105-9**). This unlocks the thumb metatarsal base from the proximal carpal bone and stretches the ligaments. Apply simultaneous flexion and longitudinal directed pressure on the base of the metacarpal. The dislocated metacarpal should return to its normal position. Obtain a radiograph to confirm the reduction.

ALTERNATIVE TECHNIQUES

There is no definitive method of treatment of a CMC dislocation in the Emergency Department. Closed reduction may be attempted, but there is a higher risk of redislocation.[1,12] Closed reduction with percutaneous pinning and open reduction with internal fixation are appropriate management options.[23] An early and stable anatomic reduction will produce faster mobilization and decrease posttraumatic disability.[1,4]

ASSESSMENT

Assess the neurologic and vascular status of the hand, joint stability, and passive range of motion to ensure successful reduction.[5,24] **It is important to evaluate the median, radial, and ulnar nerves.**

These nerves are susceptible to damage from the deformity, edema, hematoma formation, or the initial injury.[23]

AFTERCARE

The urgency of Hand Surgeon evaluation is dependent on the patient's clinical status and the success of the closed reduction. Apply a thumb spica splint (Chapter 113) after closed reduction of the first CMC joint.[25] Apply a dorsal-volar splint (Chapter 113) to immobilize the second through fifth CMC joints after closed reduction. Early follow-up with a Hand Surgeon after reduction is necessary to prevent permanent pain and disability. The instability of the reduced CMC joint requires frequent clinical and radiographic evaluation to evaluate for any postreduction subluxation.[5,12]

Provide symptomatic care. Instruct the patient to elevate their hand and apply ice or cold packs several times a day. Nonsteroidal anti-inflammatory drugs (NSAIDs) supplemented with narcotic analgesics will alleviate the pain. Avoid giving the patient a splint to prevent locking of the elbow and shoulder. Instruct the patient to return to the Emergency Department immediately if they develop finger or hand numbness, increased pain, or tingling, which can signify the splint is too tight or a compartment syndrome.

COMPLICATIONS

There are usually no complications from the reduction. Neurologic or vascular damage can result from entrapment. This will be identified on a postreduction examination. **Consult a Hand Surgeon for immediate decompression in the Operating Room.** Capsular damage and edema may contribute to joint instability after closed reduction. Debris within the joint may prevent anatomic reduction. Chronic CMC dislocations or incomplete reductions can cause arthritis, chronic instability, decreased strength, pain, and stiffness.[23] Hand compartment syndromes can occur from the dislocations if the swelling is severe or from delaying the reduction.

SUMMARY

Carpometacarpal dislocations are a rare occurrence, usually stemming from high-velocity trauma. This injury can be easily missed and lead to long-term chronic pain and a loss of hand function. Early detection, evaluation by a Hand Surgeon, and appropriate management can minimize the risk of complications.

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106 Metacarpophalangeal Joint Dislocation Reduction

Michael Gottlieb

INTRODUCTION

Hand injuries are among the most common injuries encountered in the Emergency Department (ED). They are responsible for 5% to 10% of ED visits, with approximately 6% of these patients having significant injuries.[1,2] Many hand injuries occur from sports-related events or in the workplace. Data suggest that hand injuries account for 19% of lost-time injuries and 9% of workers' compensation cases.[3] Approximately 3 to 4 million working days are lost each year as a result of hand injuries.[4] It is estimated that 10% of patients with hand injuries require referral to a hand specialist.[5] Proper motion and function of the hand are intimately related to normal anatomic alignment. The Emergency Physician (EP) must be skilled in the diagnosis and management of injuries about the hand. An improperly managed hand injury can result in significant disability that may include chronic pain, decrease range of motion, stiffness, joint swelling, deformity, or early degenerative arthritis.

Dislocations of the metacarpophalangeal (MCP) joint are relatively uncommon due to the relatively protected location of this joint in the hand.[6] Injuries to the MCP joint of the thumb are more common than injuries to the other digits. Most of these injuries are to the collateral ligaments rather than a true dorsal or volar

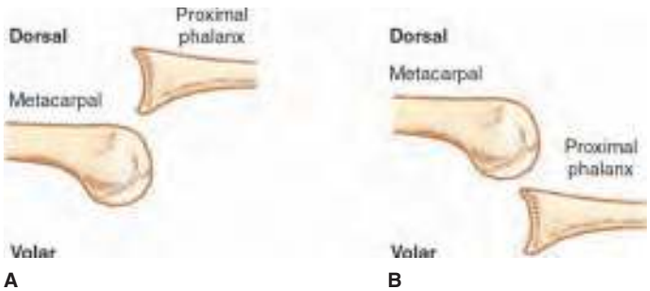

FIGURE 106-1. Dislocations are classified by the position of the distal skeletal unit in relation to its proximal counterpart. ***A.*** Dorsal MCP joint dislocation. ***B.*** Volar MCP joint dislocation.

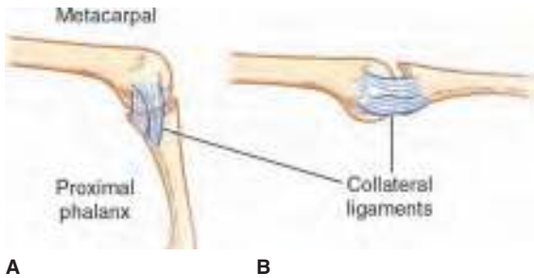

FIGURE 106-2. The shape of the metacarpal head is eccentric, making the collateral ligaments tighter in flexion (***A***) than in extension (***B***).

dislocation.[7] The spectrum of injury to the ligaments extends from a minor stretch or sprain to a complete disruption.

The deformity caused by a joint dislocation is classified by the position of the distal skeletal unit in relation to its proximal counterpart. A dorsal MCP joint dislocation describes a dislocation in which the proximal phalanx is displaced in a dorsal direction relative to the metacarpal bone (**Figure 106-1*A***). A volar MCP joint dislocation describes a dislocation in which the proximal phalanx is displaced in a volar direction relative to the metacarpal bone (**Figure 106-1*B***).

ANATOMY AND PATHOPHYSIOLOGY

FUNCTIONAL ANATOMY

The metacarpals are tubular bones structurally divided into a head, neck, shaft, and base. When viewed in the sagittal plane, the metacarpal head has an increasing diameter beginning dorsally and extending along the articular surface to the volar side. When viewed in the coronal plane, the metacarpal head is pear-shaped or dumbbell-shaped, with the volar surface extending out of each side. The metacarpal head is broader in volar orientation which results in increasing bony stability as the joint is flexed. The volar plate, collateral ligaments, dorsal capsule, deep transverse intermetacarpal ligament, extensor tendon, and intrinsic tendons provide additional support and stability to the MCP joint.

The MCP joints are resistant to ligamentous injury and dislocation because of their inherent ligamentous structure, their surrounding supporting structures, and their protected position at the base of the fingers. The volar plate is a fibrocartilaginous structure that is attached firmly to the base of the proximal phalanx. It originates just proximal to the metacarpal head where it is thin and transparent. This allows for hyperextension of the MCP joint yet makes it vulnerable to injury during dorsal dislocations.

Side-to-side stability of the MCP joint is provided mainly by the collateral and accessory collateral ligaments and, to some extent, by the lumbrical and the interossei muscles and tendons.[8] The collateral ligaments originate from the medial and lateral recesses in the metacarpal head and insert onto the base of the proximal phalanx. The eccentric configuration of the metacarpal head and the relatively fixed length of the collateral ligaments cause it to tighten when the joint is in flexion (**Figure 106-2**). This accounts for the limited abduction and adduction of the MCP joint in flexion as compared to the laxity in extension. The collateral ligaments of the MCP joint are most vulnerable to injury from forces directed ulnarly and dorsally. The accessory collateral ligament spans from the true collateral ligament to the volar plate, providing additional joint stability in extension. The central extensor tendon and sagittal band augment the thin dorsal capsule. The tendons of the palmar and dorsal interossei add a small degree of dynamic stability.

The interphalangeal (IP) joints are hinge joints, so motion only occurs as flexion and extension. The MCP joints are condyloid joints. The range of motion about the MCP joint includes 120° of flexion and 30° of hyperextension. It also has up to 30° of additional mediolateral laxity and a small degree of rotational laxity to facilitate an efficient grasp.[9] **These differences are particularly important to consider when placing a splint that involves hand or finger immobilization. One must ensure proper positioning to minimize the risk of developing contractures. The preferred position of immobilization is with the IP joints in extension and the MCP joints in flexion.**

The opposable thumb is an essential structure for numerous activities. Despite its strong ligamentous and capsular support, the exposed positioning of the thumb makes it a frequent site of dislocations and subluxations. The MCP joint of the thumb is similar to those of the fingers but has a stronger volar plate and collateral ligaments with less side-to-side mobility.[2] The MCP joint of the thumb is also a condyloid joint with a quadrilateral rather than a spherical metacarpal head that allows mainly flexion and extension. However, it also permits some degree of abduction, adduction, and rotation. There is a large variability among individuals in the range of flexion and extension.

The metacarpal head is bicondylar, with the radial condyle being slightly larger. This structural difference provides the proximal phalanx of the thumb with a modest degree of pronation during flexion. Its range of motion consists of 15° to 20° of extension, 80° of flexion, and 10° of medial (i.e., adduction) and lateral (i.e., abduction) motion. Therefore, the range of motion of the MCP joint of the thumb is significantly more limited than that of the IP joints.

PATTERNS OF INJURY

First described by Kaplan in 1957, MCP joint dislocations are considerably less common than dislocations of the IP joints.[10] Most MCP joint dislocations are dorsal, with volar MCP joint dislocations being a rare finding.[11,12] Dislocation of any of the MCP joints in any direction is possible from hyperextension injuries. Dorsal dislocation of the thumb MCP joint is the most common type of all the MCP joint dislocations.

Hyperextension of the MCP joint results in the rupture of the volar plate. Injuries are classified as simple (i.e., reducible with a closed technique) or complex (i.e., irreducible with a closed technique). The collateral ligaments typically remain intact unless there is an associated twisting of the finger. Clinical and radiographic features can be used to differentiate simple from complex dislocations. In cases of simple dislocations, the joint usually hyperextends to approximately 90° and the volar plate is not interposed between the dislocated bones.[13] The deformity is obvious with the finger in a claw position of extreme dorsiflexion at the MCP joint. In complex dislocations, the metacarpal and proximal phalanx usually lie more

parallel to each other with the metacarpal head causing a dimple on the volar skin. Complex MCP joint dislocations occur more frequently in the index finger. Clinical features that suggest a complex MCP dislocation include a proximal phalanx that is less acutely angulated than with a simple dislocation.[2] With complex MCP joint dislocations, the volar plate, sesamoids, flexor tendon, adductor tendons, extensor expansion, collateral ligaments, or joint capsule may become entrapped and prevent reduction.[2,14-16] A widened joint space is seen on radiographs when the volar plate is interposed in the joint. This makes complex MCP dislocations difficult to manage.

The MCP joint of the thumb is very mobile. Dislocations of the thumb MCP joint are more common than the MCP joints of the fingers. The MCP joint can be dorsally dislocated by a hyperextension injury or shearing forces that ruptures the volar plate, joint capsule, and at least part of the collateral ligament. The proximal phalanx will come to rest in a position dorsal to the first metacarpal. Displacement of the proximal phalanx varies from a subluxation to the complete dislocation. For the latter to occur, the volar plate and the collateral ligaments must completely tear. Volar dislocations are rare and result from extensive tearing of the dorsal capsule and the extensor pollicis brevis tendon, leaving the joint very unstable.[17]

A simple MCP joint dislocation can be converted into a complex one during prolonged or repeated reduction attempts, especially those in which axial traction is the primary component.[2] This should not deter the EP from attempting a closed reduction for any MCP dislocation, but emphasizes the importance of using the appropriate technique.

RADIOGRAPHIC EVALUATION

Radiographic evaluation of all hand injuries is relatively straightforward. It should include at least three views (i.e., anteroposterior, oblique, and lateral) of the injured area. The most important radiographic error in evaluating joint injuries of the hand is failing to get a true lateral view of the injured joint. This may result in missing a fracture or a loose body in the joint.[2] Radiographic examination will show an obvious dislocation in the lateral view. Anteroposterior views may reveal widening of the joint space in complex dislocations. In addition, the sesamoid bones may be seen in the joint space, a pathognomonic sign for a complex MCP joint dislocation.

TOES

Metatarsophalangeal (MTP) joint dislocations of the toes are primarily dorsal. They occur secondary to an axial compression load to the digit, such as kicking a toe against a wall. MTP joint dislocations of the toe are reduced similarly to MCP dislocations of the finger.[18,19] MTP joint dislocations can be difficult to reduce, as are MCP dislocations.[20]

INDICATIONS

All MCP joint dislocations should have an initial attempt at reduction in the ED unless they are unstable, chronic, open, or associated with a fracture.[21] This is true even if it is suspected to be a complex MCP joint dislocation.[22] Reduction of the dislocation will provide significant pain relief. If a Hand Surgeon is not immediately available, an open MCP joint dislocation should be reduced after copious irrigation with sterile saline to remove any dirt and debris.

CONTRAINDICATIONS

Certain MCP joint dislocations require an immediate evaluation by a Hand Surgeon for open reduction and repair of any ligamentous or associated injuries. Closed reduction of an MCP joint dislocation should not be performed in the ED for unstable, chronic, and open injuries. Instability in a joint through active range of motion indicates complete and multiple ligament disruption requiring an open surgical repair.[2,23] Chronic injuries greater than 3 weeks old generally require surgical repair.[24]

If the closed reduction attempt is unsuccessful in the ED, repeated attempts at reduction are contraindicated as a simple dislocation can be converted to a complex dislocation requiring an open surgical reduction. The ligaments, tendons, or sesamoid bones may become entrapped by bony and soft tissue structures in and around the MCP joint and prevent the reduction. Complex dislocations usually require open surgical reduction.[22,25] Open MCP joint dislocations, whether reduced in the ED or not, will require operative management after initiation of antibiotics.

There are few contraindications to the use of local anesthesia solutions.[26] Relative contraindications include injection through infected skin, history of coagulopathy from heparin or warfarin therapy, factor deficiencies, liver dysfunction or bleeding disorder, or an allergy to the anesthetic medication. Another relative contraindication includes preexisting neurovascular damage prior to the procedure. Uncooperative patients can make the injection procedure technically more difficult and dangerous to perform. Refer to Chapter 153 for the details of the complications and contraindications of local anesthetics. Procedural sedation should be considered for these patients and children.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- 18 gauge needle to draw up the local anesthetic solution
- 27 or 30 gauge needle, 2 inches long
- 10 mL syringe
- Alcohol swab
- Sterile gloves
- Sterile drapes
- Local anesthetic solution without epinephrine
- Equipment and supplies for procedural sedation (Chapter 159), if used
- Adhesive tape, ½ inch
- Webril padding
- Casting material
- Ace bandage
- Scissors

PATIENT PREPARATION

Explain the risks, benefits, potential complications, and aftercare of the reduction to the patient and/or their representative. The patient must be aware that irreducible dislocations will require an open surgical reduction and possible repair of damaged structures within and surrounding the joint. Inform the patient that the injury can result in joint swelling for weeks to months and possibly permanent joint enlargement. Loss of motion with stiffness and residual soreness may last for months. Obtain an informed consent for the reduction procedure. An informed consent should also be obtained for the procedural sedation (Chapter 159), wrist block (Chapter 156), or the intraarticular injection (Chapter 97) if they are performed in addition to the reduction procedure.

Clean the involved joint of any dirt or debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. Perform the wrist block (Chapter 156), the instillation of the local anesthetic

solution into the joint (Chapter 97), or procedural sedation and analgesia (Chapter 159) to facilitate the reduction attempt. **Make sure to perform a neurologic examination prior to anesthetizing the digit.**

TECHNIQUES

Early closed reduction may be easy provided that the thumb is maintained in adduction, the fingers are flexed, and the wrist is flexed to relax the intrinsic hand muscles.[27,28] The major obstruction preventing reduction of the dislocated MCP joint is the displaced volar fibrocartilaginous plate lying between the proximal phalanx and the metacarpal head. Approximately 50% of MCP joint dislocations can be reduced in the ED using the closed technique.

DORSAL MCP JOINT DISLOCATIONS

Dorsal MCP joint dislocations (**Figures 106-3 and 106-4*A***) require minimal, if any, distally applied traction for reduction. **It is important to remember that applying simple traction alone as an initial maneuver risks trapping the volar plate and transforming a simple dislocation into a complex dislocation.**

Hyperextend the MCP joint while simultaneously pushing the proximal end of the proximal phalanx volarly and over the metacarpal head (**Figure 106-4*B***). This maneuver diminishes the potential buttonhole effect on the metacarpal neck that is caused by excess traction. Flex the MCP joint as the proximal phalanx returns to position (**Figure 106-4*C***). Simultaneously flexing the IP joint and the wrist can help to relax the flexor tendons and make the reduction easier. This maneuver can sometimes relocate the displaced fibrocartilaginous plate to its normal position anterior to the metacarpal head.

FIGURE 106-3. Radiograph of a dorsal MCP dislocation of the index finger. (Used with permission from Kodama A, et al: Arthroscopic reduction of complex dorsal metacarpophalangeal dislocation of index finger. *Arthroscopy Tech* 2014; 3(2):e261-e264.)

FIGURE 106-4. Reduction of a dorsal MCP joint dislocation. ***A.*** Dorsal dislocation of the MCP joint. ***B.*** The deformity is exaggerated by hyperextension of the MCP joint as the proximal end of the proximal phalanx is pushed volarly and over the metacarpal head while simultaneously flexing the IP joint to relax the flexor tendons. ***C.*** The reduced MCP joint.

If the reduction is unsuccessful, repeated attempts to reduce the dislocation are contraindicated. Consider open reduction to disengage the metacarpal head from a probable buttonhole slit in the anterior capsule and from the surrounding muscles and tendons. Consult a Hand Surgeon for MCP joint dislocations that cannot be reduced after one attempt in the ED.

VOLAR MCP JOINT DISLOCATIONS

Volar MCP joint dislocations (**Figures 106-5 and 106-6*A***) are generally associated with collateral ligament ruptures. They are commonly irreducible due to interposition of the extensor tendons and the dorsal joint capsule.[29] Consultation with a Hand Surgeon is often required for operative reduction.

FIGURE 106-5. Radiograph of a volar MCP dislocation of the thumb. (Used with permission from reference 29.)

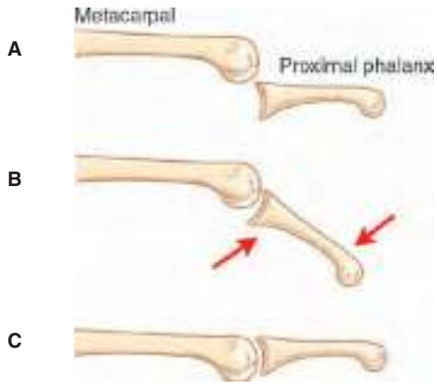

FIGURE 106-6. Reduction of a volar MCP joint dislocation. ***A.*** Volar dislocation of the MCP joint. ***B.*** The deformity is flexed at the MCP joint as the proximal end of the proximal phalanx is pushed dorsally and over the metacarpal head. ***C.*** The reduced MCP joint.

Volar MCP joint dislocations require minimal, if any, distally applied traction for reduction. **It is important to remember that applying simple traction alone as an initial maneuver risks trapping the volar plate and transforming a simple dislocation into a complex dislocation.** Flex the MCP joint while simultaneously pushing the proximal end of the proximal phalanx dorsally and over the metacarpal head (**Figure 106-6*B***). Extend the MCP joint as the proximal phalanx returns to position (**Figure 106-6*C***).

If the reduction is unsuccessful, repeated attempts to reduce the dislocation are contraindicated. Consider open reduction to disengage the metacarpal head from a probable buttonhole slit in the anterior capsule and from the surrounding muscles and tendons. Consult a Hand Surgeon for MCP joint dislocations that cannot be reduced after one attempt in the ED.

MTP JOINT DISLOCATIONS OF THE TOES

MTP joint dislocations of the toes are primarily dorsal (**Figure 106-7**). They are reduced similarly to MCP dislocations of the finger.[18,19] Like MCP dislocations, MTP joint dislocations can be difficult to reduce.[20]

ALTERNATIVE TECHNIQUE

Some physicians use a finger trap (Instrument Specialists, Inc., Boerne, TX) with all the above mentioned reduction techniques. Insert the finger with the MCP dislocation and adjacent fingers in the finger trap (**Figure 106-8**). Adjust the height of the hanging finger trap so the elbow is flexed 90°. Attach a weight to the hanging arm, usually 5 or 10 pounds (**Figure 106-8**). This results in a distraction of the MCP joint and possibly a smoother and easier reduction.

ASSESSMENT

Assess the collateral ligaments for stability. **Because the goal of treatment is to restore functional stability, it is essential to perform a thorough evaluation of joint stability.** An accurate assessment generally requires adequate pain control, even in the most cooperative patients. Active stability is tested by allowing the patient to move the joint through the normal range of motion. Completion of a full range without displacement indicates adequate joint stability. Passive stability is assessed by applying gentle radial and ulnar stress to each collateral ligament and posteroanterior stress to assess volar plate integrity. Stress testing should be performed in both extension and flexion to avoid the stabilizing effect of the volar plate. Comparisons with the same joint of the uninvolved hand may

FIGURE 106-7. Radiograph of an MTP dislocation of the toe. (Used with permission from Bussouga M, et al: Irreducible dorsal metatarsophalangeal joint dislocation of the fifth toe: a case report. *J Foot Ankle Surg* 2010; 49:298.e17-298.e20.)

FIGURE 106-8. The hand and arm suspended in a finger trap. (Photo courtesy of Instrument Specialists, Inc., Boerne, TX.)

assist in the diagnosis.[2] A stable joint suggests that optimal functional recovery would result from short-term immobilization without requiring surgical intervention.

Joint stability of the thumb MCP joint is tested in full extension as well as in 30° of flexion. Complete ligament disruption is suspected if the joint can be stressed in a radial direction 25° to 35° beyond a similar stress to the patient's opposite thumb MCP joint. This instability should be demonstrated in both full extension and 30° of flexion. Flexion at 30° lessens the stabilizing effects of the volar plate.[2] The presence of ecchymosis suggests a complete ligament tear as well as a volar subluxation of the proximal phalanx.[26]

Postreduction radiographs are important to demonstrate adequate reduction. Occasionally, a fracture is only visible on these views and not on the prereduction films.[1,30] Joint subluxation after reduction is associated with interposed soft tissue or severe capsular injury.

AFTERCARE

Splinting of any MCP joint dislocation should focus on adequate immobilization and protection. Splinting is maintained for 1 week to 3 weeks. Prolonged splint use (e.g., greater than 3 weeks) can lead to joint stiffness. Simple dorsal finger MCP joint dislocation injuries should be splinted with the joints in 45° to 50° of flexion.[2] Volar finger MCP joint dislocations require splinting in complete extension. The thumb should be immobilized in a thumb spica splint in 20° of flexion for dorsal dislocations and in extension for volar dislocations for 3 weeks. A proper program of gradual active range-of-motion exercises should follow splinting.[25] With adequate protection and splinting of uncomplicated dislocations, athletes may return to activities within 1 to 2 weeks. Athletes involved in low-risk sports with minor injuries may return sooner while those requiring surgery will necessitate a longer recovery period.[30]

COMPLICATIONS

Allergic reactions can occur from hypersensitivity to the local anesthetic solution. Symptoms can range from mild pruritus and urticarial rash to anaphylaxis. Severe allergic reactions to local anesthetics are extremely rare and the preservative in the anesthetic is often the culprit. The possibility of injury to structures in the joint may occur from improper insertion of the needle or needle movement within the joint cavity. Infection of the joint can also occur when the needle penetrates unclean skin, infected skin, or infected subcutaneous tissue. If proper aseptic technique is followed, the risk of infection is negligible. Refer to Chapters 97 and 153 regarding the complete details of joint injection complications and local anesthetic complications.

Complications of the reduction procedure are primarily related to failure of reduction, especially with complex dislocations. Entrapment of ligaments, tendons, or sesamoid bones can lead to an unsuccessful reduction.[14] A simple MCP joint dislocation can be converted into a complex one during prolonged or repeated reduction attempts, especially those in which axial traction is a primary component of the reduction attempt.[2] Irreducible MCP joint dislocations require an immediate evaluation by a Hand Surgeon.[31]

Collateral ligament injuries occur infrequently and are often missed in the acute setting. The presence of an avulsion fracture from the metacarpal head or corner fracture of the base of the proximal phalanx suggests a collateral ligament injury. Consultation with a Hand Surgeon is recommended for patients with a collateral ligament injury. Some surgeons prefer immediate operative repair of these injuries. ED care involves immobilization in an ulnar gutter or clam digger splint with the MCP joint in 45° to 50° of flexion and appropriate referral.

Rupture of the ulnar collateral ligament of the thumb, also known as gamekeeper's or skier's thumb, results from a laterally directed force at the thumb MCP joint. This ligament is important to the grasping function of the thumb. **Early recognition of this injury is key.** The diagnosis is generally made through stress testing of the reduced MCP joint. Radiographs occasionally demonstrate an avulsion-type fracture. Treatment of partial ligament tears requires immobilization in a thumb spica splint for 6 weeks whereas complete rupture requires operative repair.

Radial collateral ligament injuries of the MCP joint of the thumb are less common but equally debilitating. The usual mechanism is forced adduction with or without hyperextension. The diagnosis is generally made through stress testing of the reduced MCP joint. Radiographs occasionally demonstrate an avulsion-type fracture. Treatment of partial ligament tears requires immobilization in a thumb spica splint for 6 weeks whereas complete rupture requires operative repair.

Degenerative arthritis may occur after multiple closed reductions or unrecognized chronic dislocation.[32] Inadequate immobilization or early return to high-stress activities may result in ligamentous laxity or recurrent instability. Excessive joint contractures unresponsive to physical therapy may require surgical release.

SUMMARY

Injuries to the MCP joints of the hand are occasionally encountered in the ED. They may be associated with significant morbidity if not properly managed. The most common dorsal MCP joint dislocation seen is that of the thumb. Volar dislocations of the MCP joints are rarely seen. A thorough understanding of the anatomy and function of the MCP joint is essential to diagnose and manage these injuries. A detailed physical assessment of the soft tissues, bones, and neurovascular structures is essential to prevent occult injuries. Radiographic evaluation is required for all potential injuries. This should include anteroposterior, lateral, and oblique views in order to not miss associated avulsion fractures or evidence of complex dislocations. All MCP joint dislocations should have an attempt at reduction except those that are unstable, chronic, open, or associated with a fracture. A Hand Surgeon should evaluate any unstable, chronic, open, or irreducible dislocations. MCP joint dislocations reduced in the ED must be appropriately splinted and follow-up arranged with a Hand Surgeon.

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107 Interphalangeal Joint Dislocation Reduction

Michael Gottlieb

INTRODUCTION

Dislocation of the interphalangeal (IP) joints is one of the most common orthopedic injuries seen in the Emergency Department (ED).[1-3] Most of these injuries occur during athletic activities. The proximal interphalangeal (PIP) joint is especially susceptible to injury during ball-handling sports.[2-5] Among dislocations, IP joint injuries are second only to shoulder dislocations in incidence.[4] While IP joint dislocations are generally easy to reduce, improperly treated injuries can result in chronic pain, swelling, restricted range of motion, deformity, and early degenerative arthritis.[1,6,7] Emergency Physicians must be proficient in diagnosing and treating IP joint dislocations.

ANATOMY AND PATHOPHYSIOLOGY

The bicondylar conformation of the PIP joint creates an inherently stable hinge joint limited to flexion and extension within a range from 0° to 120°.[1,8,9] Additional stability comes from the complex of ligaments and tendons which form a box around the joint (**Figure 107-1**). The elements of this complex include the volar plate, lateral and collateral accessory ligaments, and the dorsal extensor tendons.[10] The volar plate's dense, fibrous distal aspect attaches firmly to the middle phalanx while its more membranous proximal portion is continuous with the synovial reflection. This conformation resists dorsal dislocation at the joint.[4] The three bands of the extensor tendon mechanism (i.e., the central slip with a lateral band on each side) provide dorsal support that resists joint dislocation (**Figures 107-2 and 107-3**). Lateral collateral ligaments bridge the PIP on the radial and ulnar sides, stabilizing it against lateral dislocation.[3] Most dislocations are the result of hyperextension injuries. This results in the distal bone displacing dorsally. This damages the volar plate. The volar plate can be interposed in the joint space making the dislocation irreducible. This can sometimes be unlocked during the reduction with hyperextension.

The less commonly dislocated IP joints (i.e., finger distal interphalangeal [DIP] and thumb IP joints) are similar in anatomy to one another. They are more broad-based than the PIP and range from 0° in extension to 90° in flexion with no significant lateral or rotary movement.[3] The distal phalanx in both joints is firmly attached to the skin, accounting for the high percentage of open dislocations involving these joints.

Dislocations of the PIP joint are the most common and may be classified as dorsal, volar, and lateral (**Figure 107-4**).[1,4,11] Each type of dislocation results from a different mechanism of injury and has specific associated complications.

Dorsal or posterior dislocations are the most common type of PIP joint dislocation (**Figures 107-4*A***).[12] They usually result from hyperextension injuries.[4,8,9] A dorsal dislocation occurs when the middle phalanx is displaced dorsally from the proximal phalanx. These dislocations involve injury to the volar plate and may be associated with an avulsion fracture of the base of the middle phalanx (**Figures 107-4*B* and 107-5**). Avulsion fractures involving greater than 30% of the articular surface are considered unstable and require referral to an Orthopedic or Hand Surgeon.[4]

Volar or anterior PIP joint dislocations are far less common, but more severe than dorsal PIP joint dislocations (**Figures 107-4*C* and 107-6**).

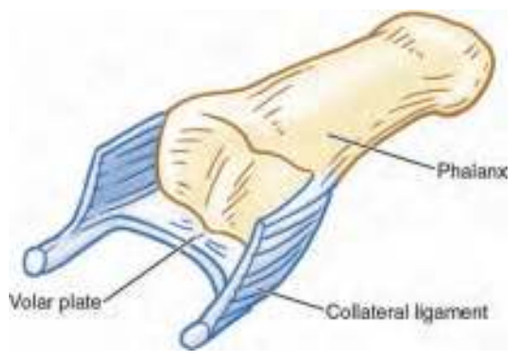

FIGURE 107-1. A schematic drawing of the box complex surrounding the PIP joint.

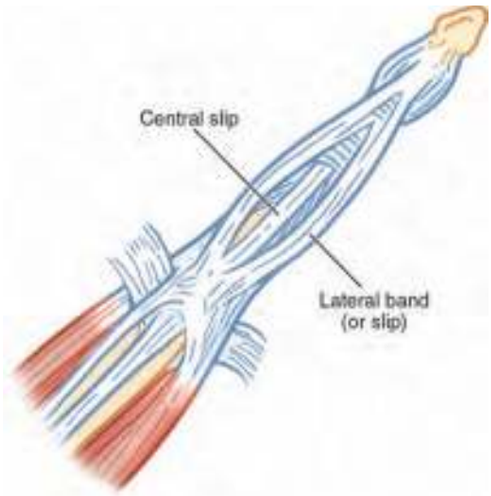

FIGURE 107-2. Dorsal view of the extensor mechanism.

They occur from a simultaneous axial load and a rotational force on the IP joint. Volar PIP joint dislocations are associated with rupture of the collateral ligament and disruption of the central slip of the extensor tendon mechanism. These injuries may preclude closed reduction.[4] These injuries can be more difficult to diagnose than other types of dislocations, often presenting as a subtle subluxation at the PIP joint with rotational deformity of the middle and distal phalanx seen on plain films.[13]

If left untreated, volar PIP joint dislocations may result in a boutonniere deformity (**Figure 107-7**).[1,12,14,15] This injury should be suspected in patients with pain over the PIP joint and the inability to fully extend the digit against active resistance. Plain films are usually unremarkable. Splinting in full extension and early follow-up with an Orthopedic or Hand Surgeon are mandatory in all suspected cases.

Lateral PIP joint dislocations are uncommon (**Figure 107-4*D***).[3] They result from a pure radial or ulnar force on the joint with either partial or complete rupture of the collateral ligament. Ulnar dislocation with radial collateral ligament injury is six times more common than radial dislocation with ulnar collateral ligament disruption.[3] Lateral PIP joint dislocations are often able to be reduced by closed methods.

Distal IP joint dislocations and IP joint dislocations of the thumb (**Figure 107-8**) are rare. They are most commonly caused by a direct blow to the distal portion of the digit.[8] They are most often dorsally

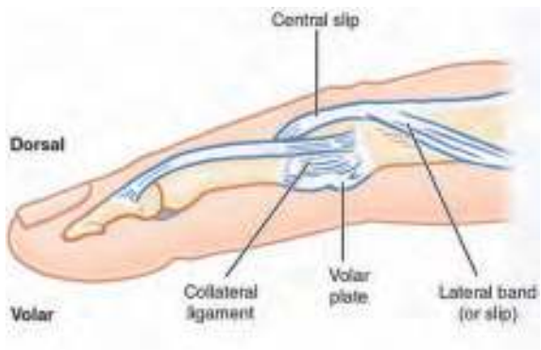

FIGURE 107-3. Lateral view demonstrating the anatomy of supporting structures. The volar plate and collateral ligaments form a box around three sides of the joint, while the extensor mechanism (consisting of central and lateral slips) lies along the dorsal aspect of the joint.

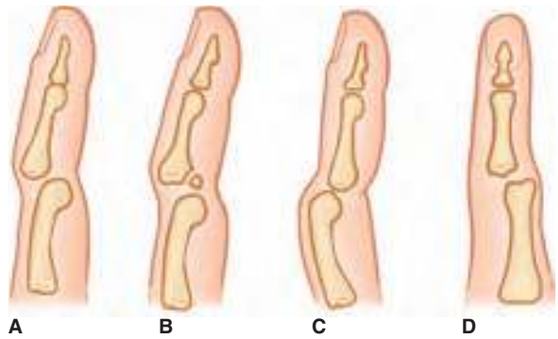

FIGURE 107-4. Dislocations of the PIP joint. They are classified based on the relationship of the distal bone to the proximal bone. ***A.*** Dorsal or posterior dislocation. ***B.*** Dorsal dislocation with an associated fracture. ***C.*** Volar, ventral, or anterior dislocation. ***D.*** Lateral dislocation.

FIGURE 107-5. Lateral view of a dorsal dislocation with minor avulsion fracture involving less than 30% of articular surface. This is a stable fracture-dislocation.

FIGURE 107-6. Volar dislocation with an associated fracture of the middle phalanx.

FIGURE 107-7. The boutonniere deformity.

dislocated and frequently open due to the firm attachment of the distal phalanx to the subcutaneous tissue and skin. All open dislocations require immediate Orthopedic or Hand Surgeon consultation.

IP joint dislocations of the toes are primarily dorsal. They occur secondary to an axial compression load to the digit, such as kicking a toe against a wall. IP joint dislocations of the toe are reduced similarly to dorsal IP dislocations of the finger.[16]

INDICATIONS

Most IP joint dislocations that present to the ED are amenable to closed reduction by the Emergency Physician. Factors to be considered include time from injury, closed versus open dislocation, associated fracture patterns, and direction of the dislocation. The majority of IP joint dislocations present within hours of the injury, and closed reduction may be safely performed up to 3 weeks from the time of injury.[7] If there is no disruption of the skin overlying an IP joint dislocation, it is considered a closed injury and suitable for closed reduction. Small fractures involving less than 30% of the joint surface are also considered safe for closed reduction.[3]

The direction of the dislocation should be considered prior to any attempted reduction. Most dorsal dislocations are reducible by closed methods. Open reduction is occasionally necessary due to interposition of the ruptured volar plate or trapping of the proximal phalanx between the volar plate and flexor tendon.[17] Lateral dislocations are also successfully managed by closed reduction in most cases.[17]

The management of volar dislocations is more controversial. These injuries are often irreducible with closed methods due to trapping of soft tissue in the joint space (e.g., usually the ruptured extensor mechanism) or are unstable after reduction due to extensive damage to the supporting ligaments.[17] **A single attempt at closed reduction may be performed.** If successful, splint the finger in extension and consult an Orthopedic or Hand Surgeon as open repair of the extensor mechanism is usually required to prevent a boutonniere deformity.[13,17] **All irreducible injuries require emergent consultation with an Orthopedic or Hand Surgeon.**

FIGURE 107-8. Dorsal dislocation of the thumb IP joint.

CONTRAINDICATIONS

Contraindications to closed reduction of IP joint dislocations include chronic dislocations, open dislocations, unstable dislocations, or complex dislocations. A chronic dislocation is defined as an IP joint dislocation of longer than 3 weeks in duration. These injuries generally require open reduction and should not be reduced in the ED.[8] The digit should be carefully inspected for any breaks in skin integrity. Any skin wound, especially in the direction of the dislocation, mandates treatment as an open dislocation by an Orthopedic or Hand Surgeon. Unstable injuries are generally fracture-dislocations involving greater than 30% of the articular surface.[3] Complex injuries involve complete disruption of multiple ligaments or tendons surrounding the joint and often require open reduction and repair due to interposition of disrupted soft tissues between the articular surfaces of the IP joint.[1,8,18,19]

EQUIPMENT

DIGITAL BLOCK

- Povidone iodine or chlorhexidine solution
- 27 gauge needle, 2 inches long
- 5 mL syringe
- Alcohol swab
- Local anesthetic solution without epinephrine (1% lidocaine, 0.25% or 0.5% bupivacaine)

POSTREDUCTION SPLINT

- Aluminum finger splint with foam padding
- Gauze padding
- Adhesive tape, ½ inch
- Scissors

PATIENT PREPARATION

Explain the risks and benefits of the procedure to the patient and/or their representative. Obtain a written consent for the reduction procedure. **Inform the patient that up to 30% of PIP and DIP joint injuries may remain swollen for many months and will likely result in permanent joint enlargement. Loss of motion and residual soreness may last several months.**[1,7]

The use of local anesthesia is based on physician and patient preference. Many physicians and patients believe that the pain of reduction is less than that of a digital block and more tolerable. For this reason, many physicians will reduce an IP joint dislocation without the use of local anesthesia.

Clean the finger of any dirt and debris. Apply povidone iodine solution and allow it to dry. Insert a 27 gauge needle into the lateral aspect of the base of the proximal phalanx. Inject 0.5 mL of local anesthetic solution. Redirect the needle dorsally while depositing 1 mL of local anesthetic solution. Withdraw the needle and redirect it volarly while depositing 1 mL of local anesthetic solution. Repeat the procedure on the medial aspect of the base of the proximal phalanx. Refer to Chapter 156 for a more detailed description of the methods to anesthetize a finger.

TECHNIQUES

DORSAL DISLOCATION OF THE PIP JOINT

A dorsal dislocation of the PIP joint involves a partial or complete disruption of the volar plate. Most dorsal dislocations of the PIP joint are easily reduced (**Figure 107-9**). Firmly grasp the middle phalanx of the affected finger with the dominant hand. Grasp the base of the proximal phalanx with the nondominant hand. Hyperextend the PIP joint and apply longitudinal traction to separate the articular surfaces (**Figure 107-9*B***). The nondominant hand is used to stabilize the proximal phalanx and apply countertraction. Flex the PIP joint while maintaining traction and apply dorsal pressure on the base of the middle phalanx (**Figure 107-9*C***). This should restore the proper alignment of the proximal and middle phalanx.

Immobilize the reduced PIP joint in 30° of flexion for approximately 3 weeks (**Figure 107-9*D***). Alternatively, tape the injured finger to an adjacent unaffected finger (i.e., "buddy taping"). Gauze padding must be placed between the fingers before "buddy taping" to prevent skin breakdown. During immobilization, it is important to avoid hyperextension if the finger is buddy taped as this may lead to further injury of the volar plate. The presence of an avulsion fracture involving less than 30% of the articular surface does not alter this management plan.

Rarely, a dorsal dislocation can be irreducible due to interposed soft tissue or impingement of the proximal phalangeal head between the central slip and the lateral bands.[13] This type of dislocation is referred to as "complex." **Failure of two or more attempts at closed reduction should raise the suspicion of an irreducible joint and an Orthopedic or Hand Surgeon should be consulted.**

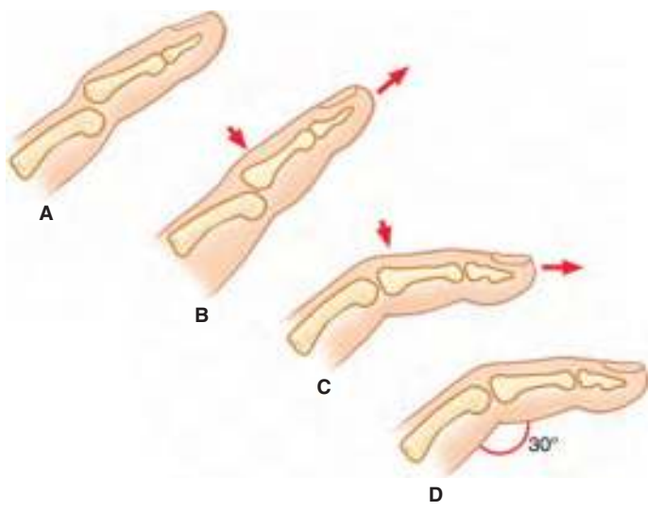

FIGURE 107-9. Reduction technique for an IP joint dislocation. ***A.*** Dorsal PIP joint dislocation. ***B.*** Exaggerate the deformity by hyperextending the middle phalanx and applying longitudinal traction distal to the injury. ***C.*** Flex the PIP joint while applying pressure to the dorsum of the middle phalanx with continued traction. ***D.*** The PIP joint is placed in 30° of flexion for splinting.

VOLAR DISLOCATION OF THE PIP JOINT

Volar dislocations of the PIP joint are almost always accompanied by an injury to the central slip of the extensor tendon causing an inability to extend the PIP joint (**Figure 107-7**). These dislocations are generally irreducible and need early consultation with an Orthopedic or Hand Surgeon for operative repair due to the extensive soft tissue damage.[20] Although controversial, some authors recommend a single attempt at closed reduction by applying a longitudinal (distal) force, hyperextending the joint, and applying dorsal pressure to the base of the middle phalanx. During this procedure, the metacarpophalangeal and DIP joints should be flexed and the wrist extended to relax the anteriorly displaced lateral bands and extensor mechanism. Splint the joint in extension and arrange for early follow-up with an Orthopedic or Hand Surgeon if closed reduction is achieved. **It is important to splint in complete extension, as even mild flexion or "buddy taping" may lead to a boutonniere deformity (Figure 107-7).**

LATERAL DISLOCATION OF THE PIP JOINT

Lateral dislocations of the PIP joint involve a partial or complete rupture of the radial and ulnar collateral ligaments. There is a 6:1 ratio of radial to ulnar collateral ligament tears with the digit being displaced in the opposite direction of the ligament rupture.[3] Generally, reduction is easy and the joint is stable after the procedure. Recreate the injury and apply longitudinal (i.e., distal) traction to the finger. Bring the distal phalanx in line with the proximal phalanx.

After reduction, determine by physical examination if the collateral ligaments are partially or completely torn. "Buddy tape" the finger to the adjacent finger for 3 weeks for incomplete collateral ligament tears. Early active motion of the finger is encouraged after this time. Complete collateral ligament tears are repaired operatively and require early consultation by an Orthopedic or Hand Surgeon.

DISLOCATION OF THE IP JOINT OF THE THUMB

Dislocations of the IP joint of the thumb are rare.[21] The injury is usually dorsal and open. If the dislocation is closed, the joint can be reduced in the same manner as PIP dislocations of the fingers.[18] Splint the reduced joint in slight flexion and arrange for early follow-up by an Orthopedic or Hand Surgeon. It may be irreducible due to the volar plate, flexor pollicis longus tendon, sesamoid bone, or a fracture fragment.[22]

DISLOCATION OF THE DIP JOINT

DIP joint dislocations are rare, usually dorsal, and open.[23] They are easily reduced in a similar manner to other IP joint dislocations and are generally stable after reduction.[19,24] Reduction is accomplished by application of longitudinal (distal) traction, hyperextension of the distal phalanx, and the application of dorsal pressure on the base of the distal phalanx. Immobilize only the DIP joint with a dorsal splint in 5° to 10° of flexion. Arrange for follow-up with an Orthopedic or Hand Surgeon.

The DIP joint may be irreducible if there is an avulsion and entrapment of the volar plate in the joint, entrapment of the long flexor tendon in the joint, or entrapment of a bony fragment.[25] Immediate consultation with an Orthopedic or Hand Surgeon should be sought if the DIP joint is irreducible.

ALTERNATIVE TECHNIQUE

Some physicians use a finger trap (Instrument Specialists, Inc., Boerne, TX) with all the above mentioned reduction techniques. Insert the distal phalanx of the finger with the PIP or DIP

FIGURE 107-10. The hand and arm suspended in a finger trap. (Photo courtesy of Instrument Specialists, Inc., Boerne, TX.)

dislocation and the distal phalanges of the adjacent fingers in the finger trap (**Figure 107-10**). Adjust the height of the hanging finger trap so the elbow is flexed 90°. Attach a weight to the hanging arm, usually 5 or 10 pounds (**Figure 107-10**). This results in a distraction of the PIP and DIP joint and possibly a smoother and easier reduction.

ASSESSMENT

Fully evaluate the finger after the reduction or any reduction attempt. This requires the local anesthetic injection to wear off. This may be the reason to use lidocaine over bupivacaine. **Perform and document a complete neurologic and vascular exam of the finger. An Orthopedic or Hand Surgeon should be consulted immediately if any neurologic or vascular deficits are identified.** Obtain postreduction radiographs of the digit to identify an avulsion injury or an incomplete reduction. Test the joint for functional stability by having the patient actively move the injured finger through a full range of motion. Stability of the joint is maintained if the collateral ligaments and volar plate are intact and no subluxation or dislocation occurs. Test the collateral ligaments by applying radially and ulnarly directed stresses with the joint in 20° of flexion. Test the integrity of the volar plate by having the patient hyperextend the joint and comparing the range of motion to that of the other fingers. The joint is considered stable if there is no displacement during active range of motion and passive stressing of the joint. If stable, place the joint in an appropriate splint and refer the patient to an Orthopedic or Hand Surgeon for follow-up. Immediately consult an Orthopedic or Hand Surgeon if the joint is not easily reduced or if it is not stable after the reduction. All open dislocations require immediate evaluation by an Orthopedic or Hand Surgeon for irrigation, reduction, and closure.

AFTERCARE

Splinting of any finger injury should provide adequate immobilization and protection while allowing maximal range of motion of the unaffected joints. The method of splinting for each specific dislocation is described in the Technique section.

COMPLICATIONS

Most complications of IP joint dislocations are secondary to the injury itself rather than the reduction procedure.[26-28] Even seemingly minor injuries can have complications such as prolonged swelling, pain, and stiffness. A thorough evaluation of the digit, prompt diagnosis, and proper treatment will help minimize these complications.

Complications of the reduction procedure are primarily related to failure of reduction. Entrapment of soft tissues should be suspected in cases with multiple failed attempts at reduction.[20] Numerous attempts at reduction may lead to trauma at the articular surface, predisposing to the development of premature degenerative arthritis. Irreducible or complex IP joint dislocations require an immediate evaluation by an Orthopedic or Hand Surgeon.

Prolonged or improper splinting of a joint can lead to chronic complications. Extended splinting and immobilization can lead to permanent joint stiffness. In general, IP joints should not be immobilized for greater than 3 weeks. **Inappropriate splinting of a volar dislocation in even mild flexion may lead to long-term complications such as the boutonniere deformity.[14,15]**

"Buddy taping" is problematic. There is a low patient compliance rate and it can cause skin adhesion or damage from the tape or rubbing of the fingers.[29] Gauze padding must be placed between the fingers before "buddy taping" to prevent skin breakdown. During immobilization, it is important to avoid hyperextension if the finger is buddy taped as this may lead to further injury of the volar plate. The "buddy taping" can also become loose.

SUMMARY

Injuries to the IP joints of the hand are commonly encountered in the ED and may be associated with significant morbidity. The most common injury encountered is a dorsal IP joint dislocation. Other dislocations include volar and lateral IP joint dislocations. A thorough understanding of the anatomy and function of the IP joint is essential to diagnose and treat these common injuries appropriately. A detailed physical examination of the soft tissues, bones, and neurovascular structures is necessary. Radiographic evaluation is required for all potential injuries, including an anteroposterior and a lateral view of the affected digit. Acute stable dislocations can be reduced immediately in the ED. An Orthopedic or Hand Surgeon should evaluate any unstable, chronic, open, or complex dislocation. Joints reduced in the ED must be splinted and appropriate follow-up arranged.

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108 Hip Joint Dislocation Reduction

Michael Gottlieb

INTRODUCTION

Hip dislocations are true orthopedic emergencies that the Emergency Physician must be capable of reducing. Neurovascular damage to the hip and leg is a known consequence of hip dislocations. Avascular necrosis (AVN) may occur in up to 20% of patients with a hip dislocation, with some studies showing that AVN following a hip dislocation occurs in a time-dependent fashion. **In other words, the longer a hip is dislocated, the higher the incidence of AVN.**[1] Dislocation for more than 6 hours almost universally results in AVN.[2-4] A hip dislocation can be diagnosed clinically with the help of radiologic studies.[5] The advantages of plain films and computed tomography (CT) scans, over the use of ultrasonography, are to identify associated fractures.

The most common causes of hip dislocations are traumatic dislocations of a normal hip, mechanical dislocations of a prosthetic hip, spontaneous dislocations, and pathologic dislocations. Dislocations may occur with minor mechanisms at the extremes of age. Examples include falling from a standing height for elderly patients or during athletic activities in children.[6,7]

Many techniques have been described to reduce dislocated hips.[3,8-15] The Emergency Physician must be familiar with each of these techniques and how to apply them appropriately to optimize patient management and outcome. Dislocations of both normal and prosthetic hips are seen in the Emergency Department, with prosthetic hip dislocations now occurring more commonly than normal hip dislocations.[16-18] Although these are not associated with AVN, the pressure from the dislocated prosthetic head may still result in other neurovascular complications.

ANATOMY AND PATHOPHYSIOLOGY

Ball-and-socket joints are inherently stable. The strong muscles, ligaments, and fibrous joint capsule of the hip further reinforce this innate stability. Consequently, in the average adult, a great deal of force must be transmitted to dislocate the hip. This is significant, as the patient with a hip dislocation may have other life-threatening injuries that take precedence over the management of the hip dislocation. The increased mortality rate associated with a hip dislocation typically results from associated injuries of the head, thorax, or pelvis.

Hip dislocations are classified into anterior, posterior, inferior, and central based on the relationship of the dislocated femoral head to the acetabulum.[2,7] Anterior hip dislocations account for 5% to 10% of hip dislocations and occur with the leg in a neutral or abducted position. The femoral head is pushed anterior to the coronal plane of the acetabulum. These patients present in extreme pain with the hip and knee flexed to 90° and the leg held in external rotation. A slight shortening of the leg may also be noted, but this is difficult to detect with the knee in flexion. There are three subtypes of an anterior hip dislocation: anterior obturator, anterior iliac, and anterior pubic. In anterior obturator dislocations, the femoral head displaces medially and lies in the obturator canal. In anterior iliac dislocations, the femoral head moves superiorly and lies over the iliac wing. In anterior pubic dislocations, the femoral head moves inferiorly over the pubic ramus.

Posterior hip dislocations are the most common type, accounting for nearly 90% of all hip dislocations. This is because the posterolateral half of the femoral neck lies outside the joint capsule and results in weaker posterior support of the hip. Posterior dislocations result from force transmitted along the femoral shaft with the leg adducted. The most common mechanism of injury is a motor vehicle collision where the knees strike the dashboard and the femoral head is pushed posterior to the coronal plane of the acetabulum. The leg will be shortened and internally rotated with marked knee flexion and adduction of the thigh. The femoral head is rarely visible but may be palpable in the buttock region. Posterior hip dislocations are further categorized into posterior ischial and posterior iliac subtypes. In posterior ischial dislocations, the femoral head is displaced inferiorly and lies over the ischium. In posterior iliac dislocations, the femoral head is displaced superiorly and lies over the iliac wing.

Central hip dislocations are the rarest form. In central dislocations, the femoral head remains on the same coronal plane as the acetabulum but is displaced superiorly. Most central hip dislocations are associated with acetabular fractures.

Inferior hip dislocations are the rarest form of hip dislocations. They are difficult to reduce due to buttonholing of the femoral head through the inferior joint capsule. An inferior dislocation usually involves forceful abduction and external rotation. A special type of inferior dislocation is luxatio erecta of the hip.[19] Reduction is usually easy due to the destruction of most of the hip ligaments during the dislocation. This makes the hip unstable and most reductions do not stay reduced.

INDICATIONS

All hip dislocations must be reduced. **Emergent hip reduction by the Emergency Physician is indicated when distal neurologic or vascular deficits are present or if the Orthopedic Surgeon is not immediately available.** The incidence of AVN is time-dependent and necessitates reduction as soon as possible to limit this complication.

CONTRAINDICATIONS

Any life-threatening conditions must be treated before the hip is reduced. Closed reduction is contraindicated if a surgical indication for repair exists. Surgical exploration is required for hip dislocations associated with femoral head fractures, femoral shaft fractures, or the finding of sciatic nerve dysfunction. Surgery is also indicated for an irreducible dislocation, persistent instability of the joint after closed reduction, and any postreduction neurovascular deficits.

EQUIPMENT

- Procedural sedation equipment and supplies (Chapter 159)
- Assistants
- Sheets

PATIENT PREPARATION

The patient must be appropriately stabilized. Life-threatening associated injuries and comorbid conditions must be adequately addressed. Obtain plain radiographs to define the anatomic dislocation pattern, rule out any associated fractures, and guide relocation attempts. Explain the risks, benefits, complications, and aftercare of the reduction procedure and obtain an informed consent from the patient and/or their representative. **The patient must be sedated to achieve optimal muscle relaxation and pain control.** Perform procedural sedation (Chapter 159) after obtaining a separate informed consent for this procedure.[20]

TECHNIQUES

Hip reduction techniques have been described with the patient in every imaginable position.[9-16,21] The relative success rates for each technique have not been reliably reported.[16] Therefore, physicians typically use the technique(s) with which they are most comfortable. The reader should become familiar with multiple reduction techniques in case one or more are unsuccessful.

ALLIS MANEUVER

This is one of the most common hip reduction methods (**Figure 108-1*A***). It was first described by Allis in 1893.[9] The technique has been improved by the addition of procedural sedation. Place the patient supine and perform procedural sedation. Instruct an assistant to stabilize the patient's pelvis to the gurney by pressing down on the anterior superior iliac spines. It may be necessary for the assistant to use both hands on the side of the pelvis associated with the hip dislocation to stabilize the pelvis. The patient's hips may also be stabilized by tying a sheet around the patient's anterior superior iliac spines and the bed. Flex the affected knee and hip to 90°. Grasp the affected knee with both hands behind the knee (**Figure 108-1*A*-(1)**). Apply axial traction to the thigh with incrementally increasing force. Simultaneously rotate the femur laterally and medially until the hip relocates (**Figure 108-1*A*-(2)**).

If relocation is not easily accomplished, instruct a second assistant to apply lateral traction to the inner thigh of the affected proximal femur (**Figure 108-1*B***). Repeat the entire procedure with the addition of lateral traction to reduce the dislocation.

MODIFIED ALLIS MANEUVER

This technique incorporates all of the maneuvers described above. Additionally, place the hip in maximum adduction. Apply longitudinal traction to the femur while an assistant presses down on the pelvis with one hand and pushes the head of the affected femur toward the acetabulum with the other hand.

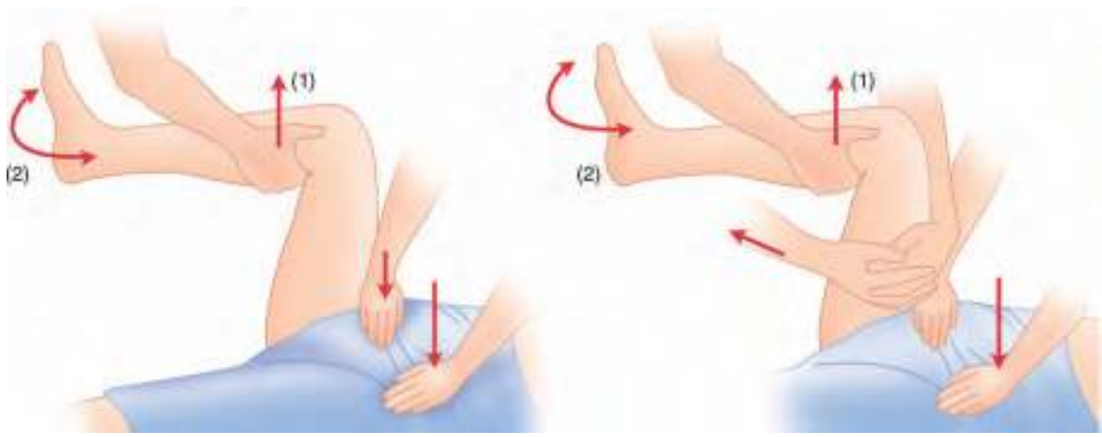

FIGURE 108-1. The Allis maneuver. ***A.*** An assistant stabilizes the pelvis. The physician simultaneously distracts the femur (1) and rocks it medial to lateral (2, *curved arrow*). ***B.*** The same maneuver with the addition of a second assistant to apply lateral traction to the thigh.

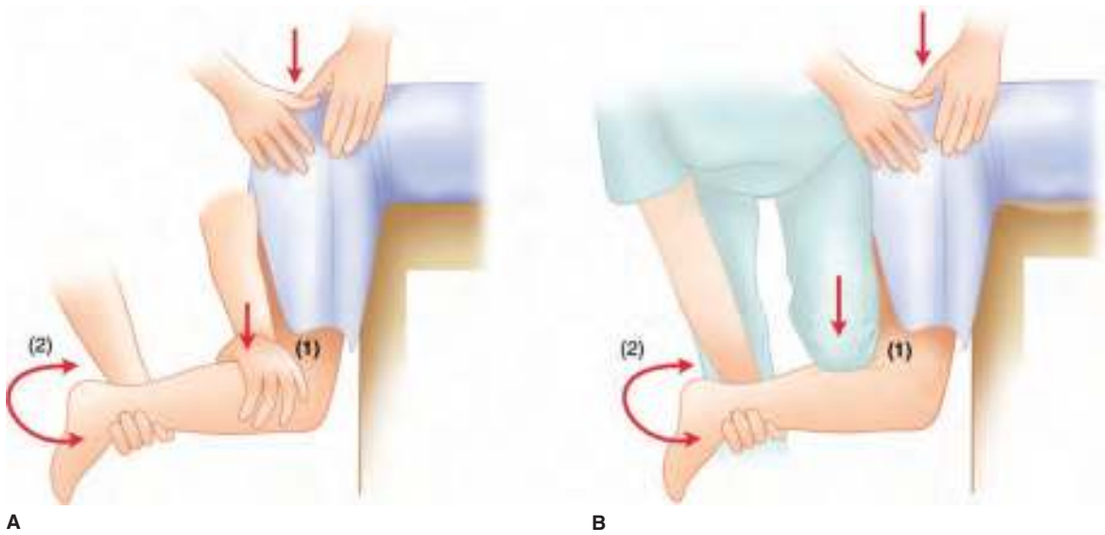

FIGURE 108-2. The gravity method of Stimson. ***A.*** An assistant stabilizes the pelvis. The physician applies downward pressure on the calf (1, *straight arrow*) while applying subtle and external rotation to the femur (2, *curved arrow*). ***B.*** An alternative method.

GRAVITY METHOD OF STIMSON

Place the patient prone on the gurney. Perform procedural sedation. Monitoring may be more difficult due to the prone positioning. **Extra attention must be paid to the patient's airway and breathing when placed prone.** Associated injuries may preclude prone positioning of the patient.

Place the affected leg hanging over the side of the gurney with the knee and hip each flexed 90°. Alternatively, hang both legs off the distal edge of the gurney with the knees and hips flexed 90° (**Figure 108-2*A***). Instruct an assistant to hold the patient down on the gurney by applying downward pressure on the posterior pelvis at the posterior superior iliac spines.

Grasp the ankle in one hand to support the limb and to be able to apply internal and external rotation to the extremity (**Figure 108-2*A*-(1)**). Place the other hand on the proximal posterior calf. Exert gradual longitudinal traction on the femur by placing pressure on the affected calf until the hip relocates. A subtle internal and external rotary motion (**Figure 108-2*A*-(2)**) may help to assist the femoral head over the acetabular rim. **Care must be taken not to compress the structures in the popliteal fossa with excessive pressure behind the knee.**

A much greater degree of force can be applied to the hip if the physician places a knee in the affected popliteal fossa instead of pushing with their arm (**Figure 108-2*B*-(1)**). Pull the affected ankle upward while simultaneously exerting downward force on the calf to reduce the dislocation. A subtle internal and external rotary motion (**Figure 108-2*B*-(2)**) may help to assist the femoral head over the acetabular rim.

WHISTLER/ROCHESTER/TULSA TECHNIQUE

This technique was described at three separate sites (**Figure 108-3**). Whistler Health Care Center in Vancouver, Canada, described it in 1997.[22] The Orthopedic Associates of Rochester described it in 1999. Vosburgh described it in 1995 as the Tulsa method.[23] It is reported to be easier than other techniques and can be performed without the aid of an assistant.[22,23] Pelvic stabilization is provided by a counterforce on the uninjured knee. The force and counterforce occur through the same fulcrum and are therefore exactly equivalent.[21]

Place the patient supine. Perform procedural sedation. Stand to the side of the affected hip. Flex the unaffected knee 130°. Place your elbow under the affected knee, allowing the leg to dangle over your forearm (**Figure 108-3**). Reach to grasp the flexed unaffected knee with the palm. Grasp the affected ankle with your other hand in order to flex the knee and rotate the hip. The hold is now established (**Figure 108-3**).

Elevate the affected knee by raising your shoulder and using your arm as a lever (**Figure 108-3-(1)**). Simultaneously apply a longitudinal force by progressively flexing the patient's knee over your arm (**Figure 108-3-(2)**). This applies traction to the femoral head, moving it anteriorly and around the acetabular rim. Once the acetabular rim is cleared, externally rotate the leg to allow the femoral head to reduce (**Figure 108-3-(3)**). External rotation is achieved by swinging the ankle laterally. A pop should be felt as the femoral head falls into the acetabulum. Reduction can be verified by internal

FIGURE 108-3. The Whistler (or Rochester, or Tulsa) technique. Elevation of the physician's shoulder (1) while simultaneously flexing the patient's knee (2) moves the femoral head anteriorly and around the acetabular rim. Externally rotating the patient's leg (3, *curved arrow*) by swinging the ankle laterally allows the femoral head to reduce.

and external rotation of the hip. An assistant may occasionally be required to stabilize the pelvis.

MODIFIED FULCRUM TECHNIQUE

Lefkowitz described the fulcrum technique in 1993 and it was modified by Bergman as described in 1994.[14] The modified technique has been used successfully in the Emergency Department.[8] The advantage of this technique is that leverage allows greater reduction forces to be applied to the hip with less strength and effort on the part of the physician (**Figure 108-4**). A steady and constant force can easily be applied that reduces the risk of fractures and nerve injuries. This constant traction is superior to the sudden jerks that are inevitable in some of the other reduction techniques.[14]

Place the patient supine and perform procedural sedation. Secure the patient to the gurney with a sheet or use an assistant to stabilize the patient's pelvis. Lower the bed, preferably to within 2 to 3 feet of the floor. Stand on the side of the affected hip. Place one foot on the edge of the bed at the level of the patient's hip. A platform or footstool may be used to gain a mechanical advantage if the level of the bed is too high or you are too short.

Flex the affected knee 90° over your knee (**Figure 108-4**). Grasp and hold the ankle of the affected leg. Apply steady and gentle downward traction on the ankle to flex the knee while simultaneously plantarflexing your foot on the gurney. This will cause the knee to exert an upward force on the patient's knee, raising the femoral head around the edge of the acetabulum and reducing the hip. **Do not use the patient's knee as the fulcrum.** [8,24,25] **Pushing down on the patient's lower leg with the physician's knee fixed creates much force on the patient's knee and can cause damage to the knee ligaments.** It may be necessary to gently rotate the affected foot internally and externally if the hip does not reduce easily.

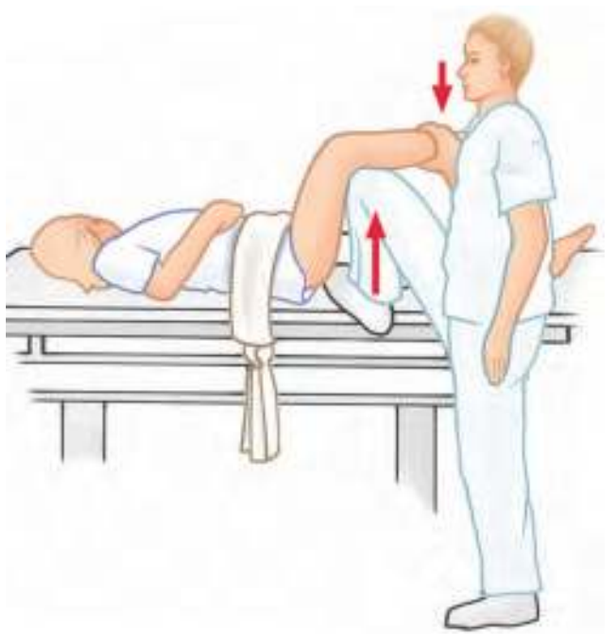

FIGURE 108-4. The fulcrum technique. The physician applies downward pressure on the patient's ankle while simultaneously plantarflexing their foot to move the femoral head around the acetabular rim and reduce the hip.

SIMPLE LONGITUDINAL TRACTION

This technique is similar to the reduction of a shoulder dislocation (**Figure 108-5**). Place the patient supine and perform procedural sedation. Extend the affected lower extremity at the hip and knee.

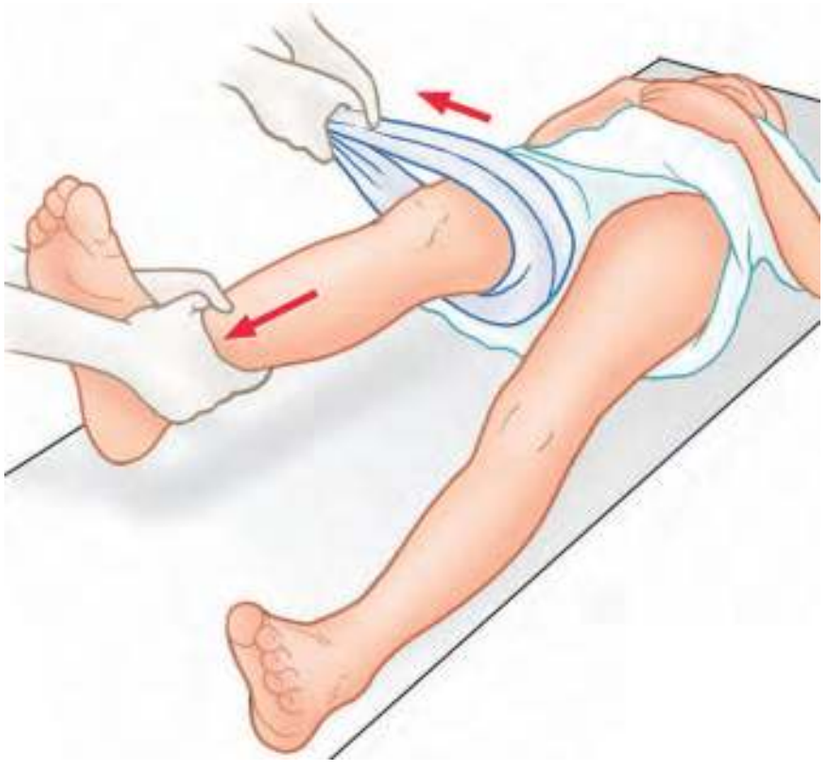

FIGURE 108-5. The simple longitudinal traction technique. An assistant applies lateral traction to the thigh while the physician simultaneously applies in-line traction to the leg.

Wrap a sheet around the affected proximal thigh. Grasp the patient's ankle with both hands. **Do not grasp the foot as this can result in secondary injury.** Instruct an assistant to apply lateral traction to the sheet and proximal thigh to move the femoral head over the acetabular rim while simultaneously exerting longitudinal traction to the leg by pulling on the patient's ankle to reduce the hip (**Figure 108-5**).

The editor prefers to use a padded leather restraint around the affected ankle. Wrap the two ties of the restraint around your hips and secure them with a knot. You can then slowly lean backward to allow your body weight to reduce the hip. This method is especially useful if you are small in stature or do not have significant upper body strength. Do not wrap the ties around your waist as this can cause low back strain.

BIGELOW MANEUVER

Bigelow described this technique in the literature in 1870 (**Figure 108-6**). It was the first documented hip reduction technique. Perform procedural sedation. Place the patient supine with the affected hip and knee flexed 90°. Hold the affected knee in the crook of your flexed elbow with the patient's foot in the opposite hand (**Figure 108-6*A***). Instruct an assistant to stabilize the pelvis

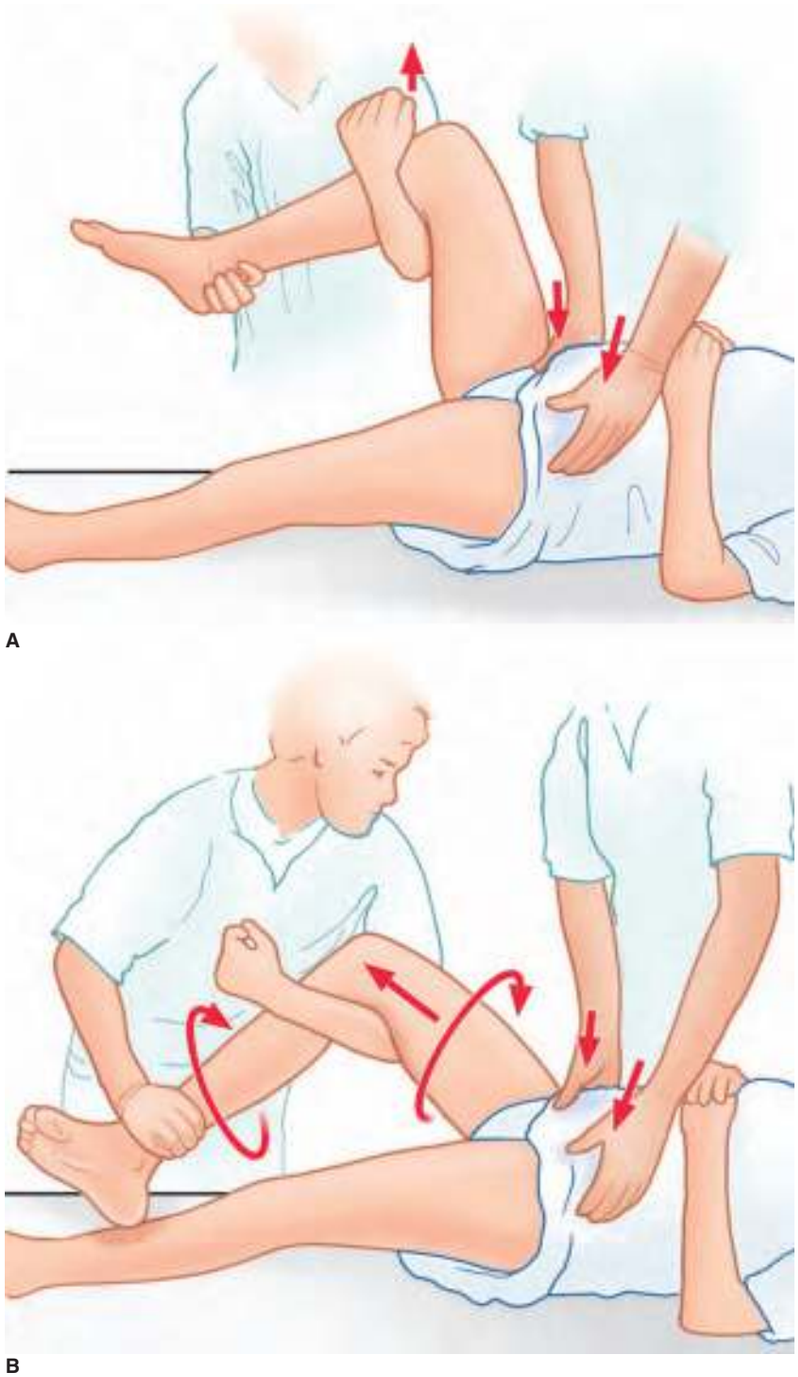

FIGURE 108-6. The Bigelow maneuver. ***A.*** The physician applies upward traction on the femur while an assistant stabilizes the pelvis. ***B.*** The hip is externally rotated and extended while the femur is distracted.

by applying downward pressure to the anterior superior iliac spines. Lift your shoulder and arm supporting the patient's knee to apply distal traction to the femur (**Figure 108-6*A***). Externally rotate and extend the hip while distracting the femur to reduce the hip (**Figure 108-6*B***).

Although this is considered the "classic" reduction technique, it is disadvantageous in that it requires great strength on the part of the physician to reduce the hip. The force applied is often jerking and inconsistent. The aid of a strong assistant is required to stabilize the pelvis.

LATERAL REDUCTION TECHNIQUE

This technique was described in 1986 by Skoff.[16,26] It gives a mechanical advantage to the physician as most of the force exerted is by the physician's own body weight (**Figure 108-7**). It also capitalizes on the principle of recreating the position of injury in order to exactly reverse the forces of the injury to produce the reduction.

Place the patient in the lateral decubitus position lying on the unaffected extremity. Perform procedural sedation. Flex the affected hip 100° and allow it to gravitate to adduction. This position recreates the typical position of the hip during the dislocation. Internally rotate the patient's hip 45° while maintaining 45° of adduction to exaggerate the hip dislocation. Place a looped sheet around the patient's hips and an assistant's hips. Place a second looped sheet around the patient's knee and your own hips. The use of sheets allows optimal leverage by using body weight as the reduction force. Grasp the affected ankle to maintain the patient's knee flexed at 90°. Apply distal traction to the femur by slowly leaning backward while the assistant simultaneously applies posteriorly directed countertraction to the femoral head. The assistant can use their hands to apply a distally directed force to the femoral head to assist in the reduction.

ASSESSMENT

The appropriate evaluation of any dislocation requires a thorough pre- and postreduction neurologic and vascular examination of the distal extremity. Any neurologic or vascular deficits require immediate evaluation by an Orthopedic Surgeon. Obtain a postreduction radiograph to confirm the reduction and rule out any fractures missed on the initial radiographs or as a result of the reduction procedure. Monitor the patient until they recover from the procedural sedation. A CT scan may help identify any acetabular or osteochondral fractures.

AFTERCARE

All patients with a hip dislocation require an evaluation by an Orthopedic Surgeon. All native hip dislocations and most prosthetic hip dislocations require hospitalization and traction after reduction.[27]

COMPLICATIONS

The complications of a hip dislocation itself are fractures, AVN of the femoral head, injury to the sciatic and femoral nerve, and injury to the femoral artery.[27] Posttraumatic arthritis, recurrent dislocation, and myositis ossificans can also occur.[28] Complications may occur despite the most expedient treatment and prosthetic hip replacement may become necessary.

The complication of AVN is time-dependent. Reductions delayed over 6 hours are at significant risk for AVN. The risk of AVN increases as the time of the dislocation increases. Reductions that apply steady, nonjerking force to the limb have a lower incidence of associated fractures as well as fewer neurovascular complications. Multiple attempts at reduction are also associated with an increased

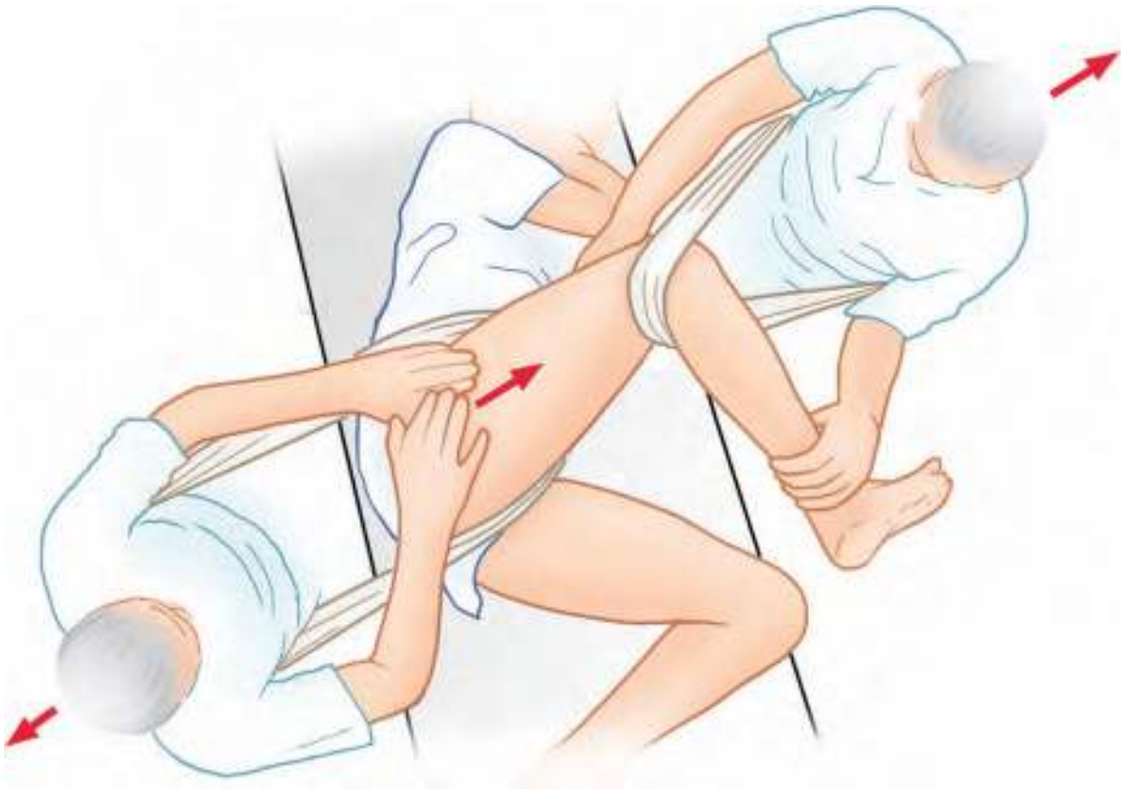

FIGURE 108-7. The lateral reduction technique. The hip is flexed 100°, adducted 45°, and rotated internally 45°. The physician applies traction to the femur. The assistant applies countertraction while simultaneously applying distally directed pressure on the femoral head with their hands.

risk of AVN. More than three attempts at closed reduction are associated with an increased risk of AVN and should prompt the Emergency Physician to seek orthopedic consultation.[29]

Neurologic injury is one of the most common complications of hip dislocations, even when successful closed reduction is achieved. Sciatic nerve neurapraxia, from peroneal nerve branch damage, occurs in up to 15% of adult traumatic dislocations, although symptoms resolve in 60% to 70% of cases.[30] Sciatic nerve dysfunction may lead to an equinus deformity and will need immediate consultation with an Orthopedic Surgeon.[30] Nerve entrapment and dysfunction can occur following closed reduction techniques.[31]

SUMMARY

Multiple techniques exist to reduce hip dislocations. The Emergency Physician should master several techniques in order to provide the best care to these patients, limit complications, and enhance outcomes. The sooner a dislocated hip is reduced, the fewer the potential complications.

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109 Patellar Dislocation Reduction

Mark P. Kling

INTRODUCTION

Dislocation of the patella generally results from a traumatic event.[1,2] It is most commonly due to a direct blow to the flexed knee. It may also occur from a forceful quadriceps contraction while the femur is internally rotated on the tibia with the foot planted (e.g., baseball, football, and soccer). Many patients may not notice the dislocation as it may spontaneously reduce immediately after the injury. There are numerous theories as to the predisposition to a patella dislocation (e.g., adolescents, age 10 to 30, anteverted femur, contracted iliotibial groove, excessive patellar lateral tilt, family history of patella dislocation, females, flat intercondylar groove, joint laxity, "knock-knees" or genu valgus, hypoplastic femoral condyle, large Q-angles, ligamentous laxity, obesity, patella alta, patellar hypermobility, physical activity, shallow intercondylar groove, and vastus medialis muscle atrophy).[3-6] This condition is most commonly seen in adolescents and females.[7,8]

ANATOMY AND PATHOPHYSIOLOGY

The knee consists of the patellofemoral and the tibiofemoral joints. The patellofemoral joint is a gliding joint. The patella is an oval-shaped sesamoid bone that develops in the tendon of the quadriceps muscle. It is suspended between the quadriceps superiorly and the tibial tuberosity inferiorly. The patella articulates between the femoral condyles. It is held in place by the vastus medialis muscle, the medial retinaculum, the medial and lateral patellofemoral ligaments, and the patellotibial ligament.

The patella may dislocate in numerous directions (**Figure 109-1**). Lateral dislocations are the most common type (**Figure 109-2**).[8] The patella usually dislocates laterally due to its asymmetrical shape and the normal upward and lateral pull of the quadriceps muscle. The patella may dislocate intraarticularly, medially, and superiorly in rare instances (**Figure 109-1**).[8-13]

The clinical determination of a lateral patellar dislocation is usually simple and obvious (**Figure 109-3*A***).[8] The knee is held in partial flexion. The patella can be seen and palpated on the lateral surface of the knee. This may be accompanied by edema and/or ecchymoses over the anterolateral knee.[8]

FIGURE 109-1. Types of patellar dislocations.

Pain over the parapatellar ligaments may be the only clinical sign in patients whose patellar dislocation has spontaneously reduced. The physical examination usually reveals mild edema in the parapatellar recesses. There is often laxity in the tendons and ligaments surrounding the patella. A patellar apprehension test is generally positive. The knee joint is usually stable.

The pathophysiology of this dislocation may include abnormalities secondary to hyperelasticity, laxity, and malalignment of the joint. Osteochondral fractures are common and seen on arthroscopy.[3,4,14] Arthroscopy, bone scans, and magnetic resonance imaging scans are considerations for further evaluation and diagnosis of the patellofemoral joint by the Orthopedic Surgeon.

Obtain prereduction radiographs (**Figure 109-3*B***) to document patellar fractures or other bony abnormalities prior to the reduction. Obtain radiographs in multiple views (e.g., anteroposterior, axial, lateral, notch, and sunrise). These may be difficult to obtain if the patient has significant discomfort and may be delayed until after the reduction. Radiographs may be used to identify a foreign body if abrasions or lacerations are present over the knee. **The patella often reduces spontaneously in the radiology suite as the leg is extended to obtain the radiographs.**

INDICATIONS

Reduce any medial or lateral patellar dislocation that does not reduce spontaneously. The goal is prompt reduction to decrease articular surface injury.[8]

CONTRAINDICATIONS

The evaluation and management of the patient's airway, breathing, circulation, and other significant injuries take priority over the reduction of a patellar dislocation. There are a few relative contraindications to the reduction of a patellar dislocation. Consult an Orthopedic Surgeon for the evaluation and reduction if the dislocation is horizontal, intercondylar, superior, or associated with fractures of the distal femur or proximal tibia. **The only exception to this is if there is neurologic and/or vascular compromise of the distal extremity.** This requires immediate reduction by the Emergency Physician if, after phone consultation, the Orthopedic Surgeon is not immediately available to perform the reduction.

EQUIPMENT

No special equipment is required for the reduction of a patellar dislocation. A knee immobilizer or splinting material (i.e., plaster, fiberglass, or prepackaged splints) should be available to temporarily splint the patella after the reduction.

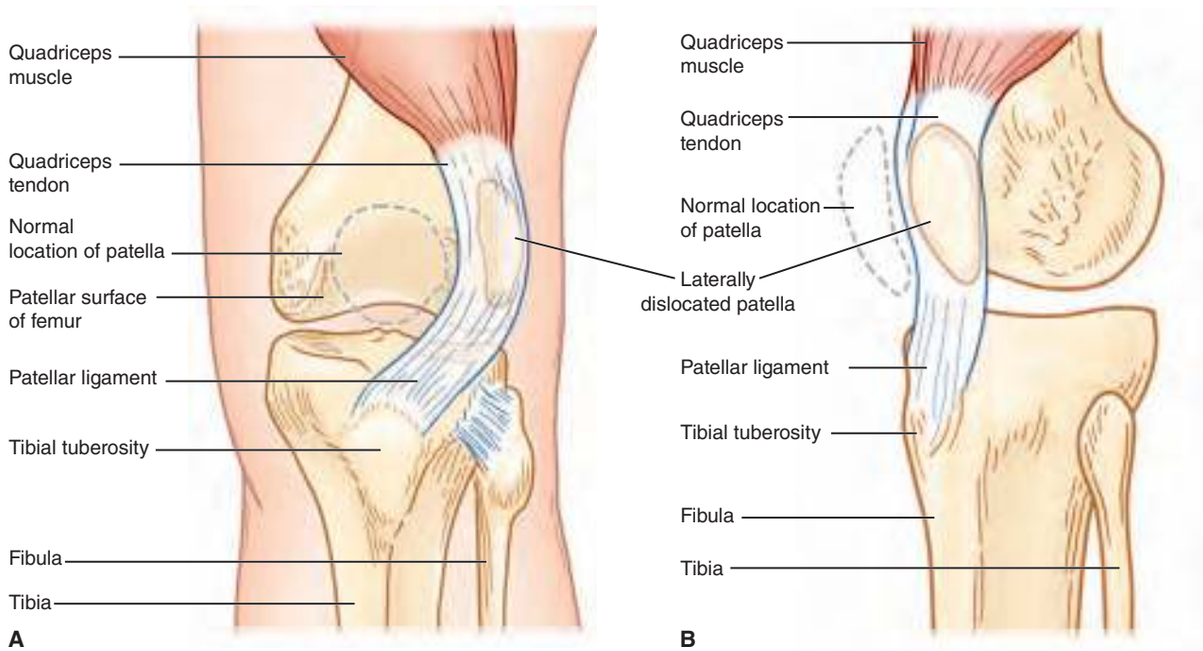


FIGURE 109-2. Anatomy of a lateral patellar dislocation. ***A.*** Anteroposterior view. ***B.*** Lateral view.

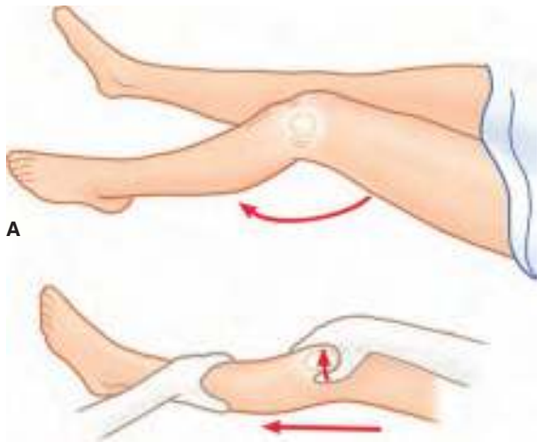
A

B

FIGURE 109-3. The lateral patellar dislocation. ***A.*** The presentation is often clinically dramatic. ***B.*** Radiograph. (Photos used with permission from www.lifeinthefastlane.com.)

PATIENT PREPARATION

Patient preparation is minimal in the case of a lateral or medial patellar dislocation. Explain the risks, benefits, complications, and aftercare to the patient and/or their representative. Obtain an informed consent prior to performing the procedure. Verbal consent is usually sufficient since the reduction of a patellar dislocation is relatively simple with infrequent complications. Place the patient supine on a gurney. No intravenous access, premedication, or sedation is required for this procedure. The Emergency Physician may administer analgesics after the initial examination to make the patient more comfortable during radiography and while waiting for the reduction. Consider the use of a femoral nerve block if anesthesia is required (Chapter 156).[15]



A

B

FIGURE 109-4. Reduction of a lateral patellar dislocation. ***A.*** Manipulation of the knee begins with gradual extension. ***B.*** Medially directed pressure applied to the patella when the knee is fully extended reduces the dislocation.

TECHNIQUES

The technique for the reduction of a lateral patellar dislocation is simple (**Figure 109-4**). Slightly flex the patient's hip to release the tension on the quadriceps muscles. Slowly and gently extend the knee (**Figure 109-4*A***). The patella may relocate spontaneously by simply extending the knee. Apply gentle and medially directed pressure to the lateral surface of the patella if it is still dislocated (**Figure 109-4*B***). This will allow the patella to move into its normal anatomic position in the intercondylar fossa of the femur. The technique to reduce a medially dislocated patella is similar except for the application of a laterally directed force on the patella.

Intraarticular and horizontal patellar dislocations are sometimes reduced by closed manipulation, although most require open reduction. Case reports have described irreducible lateral dislocations.[16] Superior patellar dislocations require operative reduction. **Do not reduce intercondylar and superior dislocations in the Emergency Department unless hemodynamic compromise is present.** Patients with these types of patellar dislocations require urgent consultation with an Orthopedic Surgeon and possible hospital admission for reduction in the Operating Room.

PEDIATRIC CONSIDERATIONS

Most pediatric injuries occur during athlatics.[8] This is in contrast to adults that are often sedentary and overweight. The indications, contrandications, and technique are the same as for adults. Some studies show children do better in the long term with surgical management.[17] Other studies show no difference between nonsurgical and surgical management.[18]

ASSESSMENT

Obtain a postreduction radiograph to rule out any osteochondral fractures that were not diagnosed initially and to ensure positioning of the patella. Maintain the knee in extension by immobilization with a splint or knee immobilizer until follow-up for reevaluation (Chapter 113).

AFTERCARE

The Orthopedic Surgeon may elect to take a conservative approach with the leg in a long leg cast and the knee in full extension for 6 weeks.[6,19] Some Orthopedic Surgeons believe that all first-time dislocations should be repaired surgically. A phone consultation with an Orthopedic Surgeon is recommended before the patient is discharged home.

The general principles of orthopedic care can be applied.[8] These include rest, ice, elevation, and nonsteroidal anti-inflammatory drugs (NSAIDs). Narcotic analgesics are not necessary or required in most cases. The patient should follow up with an Orthopedic Surgeon in 5 to 7 days. Surgical versus conservative treatment will be evaluated at that time to determine the best outcome.[20] The patient will most likely need physical therapy. The instability and resultant tracking abnormalities will require isometric, proprioceptive, and strength rehabilitation.[6,21] Patients placed in splints or casts should use crutches and not bear weight on the affected extremity. Consider crutches for those placed in a knee immobilizer.

COMPLICATIONS

The damage to ligaments from the dislocation can result in a hemarthrosis. Twisting can damage the anterior cruciate ligament, lateral collateral ligament, medial collateral ligament, and/or meniscus. Patellar dislocations are subject to degenerative arthritis, osteochondral fractures that may be difficult to diagnosis initially, and recurrent dislocations or subluxations.[8] No complications are associated with the reduction procedure.

SUMMARY

Patellar dislocations are common. The reduction of a lateral or medial patellar dislocation is a safe, simple, and gratifying procedure. Education of the patient and follow-up with an Orthopedic Surgeon are requirements for successful rehabilitation.

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110 Knee Joint Dislocation Reduction

Michael Gottlieb

INTRODUCTION

Dislocations of the knee are rare. They are true orthopedic emergencies and have a significant association with soft tissue injuries and neurovascular compromise. A dislocated knee occurs most commonly after a major force is applied to the knee joint such as from a motor vehicle collision, other high-speed trauma, or a sports injury. The etiology has recently been changing to also occur during activities of daily living and other low-level trauma, especially in obese patients.[1-4] A dislocated knee can occur after a total joint replacement.[5] The forces necessary to cause a dislocation of the knee joint can often fracture the bones of the leg.

Complete dislocation of the knee joint results in a gross deformity that is confirmed by plain radiographs. Reduction by the Emergency Physician may be reasonable if the Orthopedic Surgeon is not immediately available or if the injured extremity shows signs of distal neurologic or vascular compromise.

A careful examination of the distal extremity must be performed and documented.[6] **It must include an assessment of the capillary refill, the dorsalis pedis pulse, the posterior tibial pulse, peroneal nerve function, and tibial nerve function.**

ANATOMY AND PATHOPHYSIOLOGY

A knee dislocation is the displacement of the tibiofemoral articulation (**Figure 110-1**). It can involve the rupture of the anterior cruciate ligament, the posterior cruciate ligament, the joint capsule, or the collateral ligaments of the knee.[7] Anterior knee dislocations are the most common type of knee dislocation. This injury is defined as anterior displacement of the tibia relative to the femur (**Figures 110-1*A* and 110-2**). It results from an acute hyperextension injury to the knee joint that ruptures the anterior cruciate ligament as well as part of the posterior cruciate ligament and the posterior joint capsule. The collateral ligaments usually remain intact. Tibial spine fractures, osteochondral fractures of the tibia or femur, and meniscal injuries can be associated with the rupture of the anterior cruciate ligament. In children, hyperextension injuries are more likely to cause a distal femoral epiphyseal separation rather than a complete dislocation.

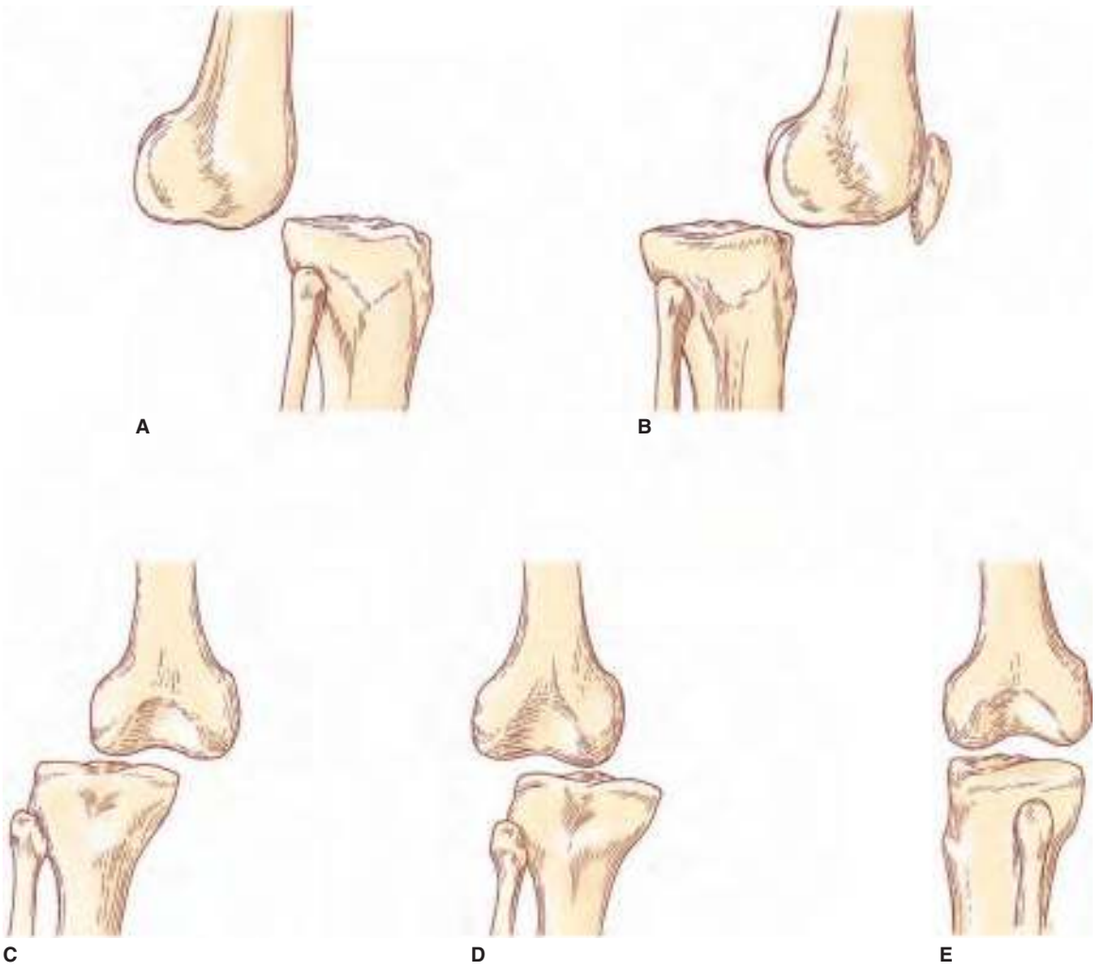

FIGURE 110-1. The classification of knee dislocations. ***A.*** Anterior. ***B.*** Posterior. ***C.*** Lateral. ***D.*** Medial. ***E.*** Rotary.

An anterior knee dislocation may be associated with a popliteal artery injury in 30% to 40% of patients.[8] The popliteal artery is at particular risk for injury because it is anchored proximally at the adductor hiatus and distally at the soleus arch. The collateral circulation around the knee joint is relatively poor. Therefore, disruption of the popliteal artery may result in significant distal ischemia and limb loss if the reduction is delayed. **It is important to note that the presence of distal peripheral pulses and capillary refill does not preclude an arterial injury.[9]**

The peroneal nerve is tethered as it winds around the fibular neck. As a result, peroneal nerve injury is another common complication occurring in up to 23% of patients with knee dislocations. Nearly one-half of patients with peroneal nerve injuries have a permanent deficit.[10] Therefore, it is particularly important to examine the peroneal nerve for dysfunction (i.e., anesthesia or paresthesia on the lateral aspect of the leg and impaired dorsiflexion of the foot).

A posterior knee dislocation is defined as the posterior displacement of the tibia relative to the femur (**Figure 110-1*B***). It occurs less commonly than an anterior knee dislocation. It results from a direct force applied to the anterior tibia with the knee slightly flexed which ruptures the posterior joint capsule and both cruciate ligaments. The collateral ligaments usually remain intact. It is associated with popliteal artery damage and disruption of the extensor mechanism of the knee joint.

Medial, lateral, and rotary dislocations of the knee joint are less common than anterior or posterior knee dislocations (**Figures 110-1*C*, 110-1*D*, and 110-1*E***). Medial knee dislocations result from an adduction force on the tibia that ruptures the lateral collateral ligament, the posterior joint capsule, and both cruciate ligaments. Damage to the peroneal nerve is common while injury to the popliteal artery is not. Lateral knee dislocations result from an abduction force on the tibia that ruptures the medial collateral ligament, the posteromedial joint capsule, and both cruciate ligaments. Neurovascular injuries are uncommon with a lateral knee dislocation.

Rotary dislocations are subdivided into posterolateral and posteromedial types. Posterolateral rotary dislocations result from an anteromedial force on the tibia that ruptures the posterior and medial joint capsule, partially avulses the gastrocnemius, damages the menisci, and has an associated chondral fracture. Posteromedial rotary dislocations result from an anterolateral force on the tibia that ruptures both cruciate ligaments, the medial collateral ligament, and the posteromedial joint capsule; partially avulses the gastrocnemius; damages the menisci; and has an associated chondral fracture. Both of these rotary dislocations are associated with peroneal nerve and popliteal artery injuries.

Medial, lateral, and rotary dislocations of the knee are uncommon injuries that should be managed by an Orthopedic Surgeon.[11,12] The reduction technique for these dislocations is quite similar to that for the reduction of anterior or posterior knee dislocations. The rotary knee dislocation is often irreducible in the Emergency Department.

FIGURE 110-2. Radiograph of an anterior knee dislocation. (Used with permission from reference 16.)

Although the presentation of a knee dislocation is usually clinically obvious, patients can present after a spontaneous reduction.[13] **This type of injury is classified as an occult dislocation and can be easily missed if one does not maintain a high index of suspicion.** A hemarthrosis suggest a knee fracture, knee dislocation, or intraarticular ligamentous rupture.[14]

Anteroposterior and lateral radiographs of the knee will confirm the diagnosis of a knee dislocation (**Figure 110-2**).[2] Postreduction films should include at least two planes to detect occult fractures of the tibial spine, the distal femoral physis, or the proximal tibial physis. Consider a postreduction stress view if damage to the collateral ligaments is suspected. Radiographs of the pelvis and hip should also be considered to rule out any associated injuries.

INDICATIONS

Any dislocation of the knee joint requires prompt reduction, ideally within 6 to 8 hours after the injury. The incidence of limb loss is greater than 85% if the knee is dislocated longer than 6 to 8 hours.[15]

Knee dislocations associated with distal neurologic or vascular insufficiency require immediate reduction. This includes children with open growth plates.[16]

CONTRAINDICATIONS

There are no absolute contraindications to the reduction of a dislocated knee joint. Reduction of the knee joint may be performed intraoperatively if the patient requires surgery for other reasons. An Orthopedic Surgeon should reduce the knee if it is dislocated medially, laterally, or rotatorily; if it is associated with fractures of the extremity; or if the joint is open. **Emergent reduction by the Emergency Physician is indicated if the Orthopedic Surgeon is not immediately available or if there is evidence of distal neurologic or vascular compromise.**

EQUIPMENT

- Procedural sedation equipment and supplies (Chapter 159)
- Assistants
- Compressive cotton wrap (Webril)
- Splinting material
- Elastic bandage

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. The necessary postprocedural care should also be discussed. Obtain an informed consent for the reduction procedure as well as for the procedural sedation. Place the patient supine on a gurney. Prepare for and apply procedural sedation (Chapter 159). **The key to performing this procedure is to have the patient adequately sedated and their muscles relaxed.**

TECHNIQUES

ANTERIOR KNEE DISLOCATION REDUCTION

Reduction of an anterior knee dislocation is usually performed without difficulty using a modified traction-countertraction technique (**Figure 110-3**). Instruct an assistant to grasp the tibia and apply in-line traction while a second assistant grasps the thigh and applies countertraction. **It is extremely important to avoid putting pressure over the popliteal fossa as this can injure the structures traversing that space.** The physician then pushes the proximal tibia posteriorly (**Figure 110-3-(1)**) while the distal femur is simultaneously lifted anteriorly into anatomic position (**Figure 110-3-(2)**).

FIGURE 110-3. Reduction of an anterior knee dislocation. An assistant applies in-line traction to the tibia while a second assistant applies countertraction to the femur. The proximal tibia is pushed posteriorly (*1*) while the distal femur is pulled anteriorly (*2*) to reduce the dislocation.

FIGURE 110-4. Reduction of a posterior knee dislocation. An assistant applies in-line traction to the tibia while a second assistant applies countertraction to the femur. The proximal tibia is pulled anteriorly to reduce the dislocation.

Do not allow the knee to become hyperextended during the reduction as this may further injure the neurovascular structures located within the popliteal fossa.

Some physicians feel that the reduction procedure may be easier to perform if the patient is in the prone position. Performing the procedure in the prone position can be a more challenging position to attain if other injuries are present and makes monitoring during procedural sedation difficult. Therefore, placing the patient in the prone position is not recommended.

POSTERIOR KNEE DISLOCATION REDUCTION

Reduction of a posterior knee dislocation is similar to that of an anterior knee dislocation. The two assistants provide in-line traction and countertraction while the physician grasps the proximal tibia and pulls it anteriorly into anatomic position (**Figure 110-4**).

MEDIAL KNEE DISLOCATION REDUCTION

Reduction of a medial knee dislocation is similar to that of the anterior knee dislocation. The two assistants provide in-line traction and countertraction while the physician grasps the proximal tibia and pulls it laterally into anatomic position.

LATERAL KNEE DISLOCATION REDUCTION

Reduction of a lateral knee dislocation is similar to that of the anterior knee dislocation. The two assistants provide in-line traction and countertraction while the physician grasps the proximal tibia and pulls it medially into anatomic position.

ROTARY KNEE DISLOCATION REDUCTION

Reduction of a posteromedial knee dislocation is similar to that of the anterior knee dislocation. The two assistants provide in-line traction and countertraction while the physician grasps the proximal tibia and simultaneously externally rotates and lifts it upward into anatomic position.

Reduction of a posterolateral knee dislocation should be performed in the Operating Room unless neurologic and/or vascular compromise to the distal leg is present. These dislocations are often irreducible using closed reduction techniques because the medial femoral condyle evaginates through the medial joint capsule in a process known as "buttonholing."[11] This dislocation requires open reduction under general anesthesia.

PROSTHETIC KNEE DISLOCATION

The reduction of prosthetic knees is similar to native knees. The preprocedural and postprocedural care is also the same.

ASSESSMENT

Immediately evaluate and document the neurologic and vascular status of the distal extremity after any attempts at reduction. Any decrease in sensation, motor function, or pulses require immediate angiography or computed tomography (CT) angiography and operative intervention. Immediate operative intervention can avoid limb ischemia and amputation.[17] Obtain postreduction radiographs to confirm proper anatomic alignment, to identify any fractures not evident on the prereduction radiographs, and to rule out the displacement of any fracture fragments.[18] Stress radiographs are recommended if injury to the collateral ligaments is suspected.

AFTERCARE

The postprocedural care of the knee joint is as important as the initial reduction. Immobilize the extremity in a posterior long leg splint with the knee in 15° of flexion. Administer analgesics as necessary to control the patient's pain.

All patients require admission to the hospital for observation and monitoring of the distal neurovascular status of the extremity and the development of a compartment syndrome.[2] Arteriography or CT angiography can be obtained to exclude injury to the popliteal artery, especially if there is any irregularity in the dorsalis pedis pulse, posterior tibial pulse, or ankle-brachial index before or after the reduction (**Figure 110-5**). Arteriography or CT angiography may not be necessary if the distal pulses are normal before and after the reduction and if the ankle-brachial index is normal. However, the vascular status should be closely monitored for 48 to 72 hours after the reduction.[19-21]

The mandatory use of arteriography or CT angiography to evaluate vascular injuries in all patients after reduction may not be necessary.[19,22-24] In select patients who have a normal physical examination and a normal ankle-brachial index angiography may be avoided. However, this is a decision that should be made in consultation with the Orthopedic Surgeon who will be managing the patient.

An inpatient magnetic resonance imaging (i.e., magnetic resonance imaging with magnetic resonance angiography) scan of the knee joint should be obtained to evaluate ligamentous injury.[2] The patient may require reconstructive surgery, especially if they are young and physically active.[25]

COMPLICATIONS

Knee dislocations are true orthopedic emergencies because of the potential for associated vascular and neurologic injuries. Complications are primarily related to injuries of the neurovascular structures within the popliteal fossa. These include the popliteal artery and peroneal nerve. Patients are also at risk of knee instability, knee arthrosis, knee stiffness, and chronic pain.[26,27] Neurovascular

A B

FIGURE 110-5. Angiogram of a popliteal artery disruption. Note the abrupt cutoff. ***A.*** Anteroposterior view. ***B.*** Lateral view with the collaterals backfilling the popliteal artery. (Used with permission from reference 22.)

injuries increase as body mass index increases.[1] These can lead to the development of a compartment syndrome.[2]

Anterior knee dislocations have a high incidence of associated vascular injuries usually involving the popliteal artery. Of these, up to one-half can result in amputation of the leg.[28] Nerve damage has been reported in the literature to occur in 20% to 40% of knee dislocations.[29] **It is important to specifically assess for injuries to these structures. Avoid direct pressure to the popliteal fossa during the reduction and hyperextension of the knee after the reduction to prevent iatrogenic neurovascular damage.** Injuries to neurologic and vascular structures can also occur during the reduction attempt. These include lacerations, traction injuries, and entrapment between the tibial plateau and the femoral condyles.

Irreducible dislocations may be secondary to interposed soft tissue, ligamentous instability, buttonhole tears in the medial joint capsule, or entrapment of the medial femoral condyle.[1,2,30] These patients require operative reduction under general anesthesia by an Orthopedic Surgeon.

SUMMARY

Knee dislocations occur after significant trauma to the knee joint. Fortunately, knee dislocations are rare events. They are associated with significant morbidity and require prompt reduction to restore the normal alignment of the bony structures. Arteriography to rule out damage to the popliteal artery and a magnetic resonance imaging scan to rule out soft tissue injuries should be performed after the knee joint has been reduced and adequately splinted. All patients require admission for observation and may require reconstructive surgery. Frequent neurovascular evaluation is extremely important during the hospitalization. Evidence of distal leg ischemia requires immediate surgical exploration.

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111 Ankle Joint Dislocation Reduction

Crystal Ives Tallman

INTRODUCTION

The foot and the ankle are commonly injured parts of the body. Fractures of the ankle associated with dislocations of the ankle joint (i.e., fracture-dislocations) or isolated ankle dislocations without fracture are serious injuries that can lead to long-term morbidity. Ankle dislocations are high-energy injuries that occur most commonly in young people from sports, falls, or motor vehicle collisions.[1-3] The ankle mortise and surrounding ligaments make the ankle joint strong and stable. This makes isolated ankle dislocations uncommon. Ankle dislocations are usually associated with malleolar fractures or a fracture of the tip of the tibia. They are open 25% of the time. There are limited data on the mechanism of injury. Most ankle dislocations lead to posterior or posteromedial displacement and occur from a force against a plantarflexed foot. Definitive management of fracture-dislocations is most often surgical. The patient benefits from early analgesia and prompt reduction.

Ankle dislocations can be successfully reduced in the Emergency Department with the use of procedural sedation and longitudinal traction-countertraction. The key to a successful outcome is anatomic restoration and healing of the ankle mortise.[1] Postreduction management involves leg immobilization and consultation with an Orthopedic Surgeon. Some closed ankle dislocations may be managed nonoperatively with good long-term results from a closed reduction and casting for 6 to 9 weeks.[4-8]

ANATOMY AND PATHOPHYSIOLOGY

The ankle joint is composed of the talus, tibia, and fibula. The inferior articular surface of the tibia is concave in both the coronal and sagittal planes. The articular surface of the talus is broader anteriorly and longer on its medial and lateral aspects.[9] The ankle mortise limits rotation of the talus and make the ankle joint inherently stable.

There are three groups of ligaments that provide added stability to the ankle joint. It is stabilized laterally by the anterior talofibular, the calcaneofibular, and the posterior talofibular ligaments (**Figure 111-1**). It is stabilized medially by the deltoid ligament. The deltoid ligament comprises a group of four adjoining ligaments: the anterior and posterior tibiotalar, the tibionavicular, and the tibiocalcaneal ligaments (**Figure 111-2**). The third group of ligaments stabilizes the tibia to the fibula and forms the tibiofibular syndesmosis. This includes the anterior and posterior tibiofibular ligaments.

Almost all ankle dislocations are associated with partial or complete ligamentous ruptures (**Figure 111-3**).[10] They are often associated with malleolar and distal fibula fractures due to the relative strength of the ligaments compared to the malleoli.[1,2,11] This results in one or both malleoli fracturing rather than the ligaments tearing with a lateral fracture-dislocation.

Posterior or posteromedial ankle dislocations are the most commonly described dislocations of the ankle joint due to the tendency to land with the ankle pronated and inverted after a fall from a height (**Figures 111-3*A* and 111-4**).[2,6,12] Lateral ankle fracture-dislocations are commonly seen (**Figure 111-3*B***). Posterior ankle dislocations are associated with posterior marginal fractures of the tibia. An anterior ankle dislocation is rare and usually associated with a fracture of the anterior margin of the tibia (**Figure 111-3*C***). The talus may also dislocate laterally or medially if the tibiofibular ligaments are disrupted or a fracture of one or both malleoli occurs. Due to the low incidence of reported ankle dislocations without fractures, data on the mechanism of injury are incomplete.[13] A posteromedial ankle dislocation occurs when a force is applied against the posterior distal tibia with the foot plantarflexed. Anterior ankle dislocations occur from a forcible dorsiflexion of the foot (e.g., fall on the heel with the foot dorsiflexed) or from a force applied to the distal anterior tibia while the foot is fixed. Injury to the tibiofibular joint is variable and the fibula may be dislocated posteriorly or anteriorly. Diastasis of the tibiofibular syndesmosis is uncommon. Lateral ankle dislocations are always associated with fractures of the malleoli. They occur from a force on the distal fibula with the foot fixed to the ground. Superior ankle dislocations are uncommon (**Figure 111-3*D***).[14-16] They occur when a force from above is driven through the leg and to the ankle (e.g., a fall from a height).

Physical examination often reveals the type of ankle dislocation. Prominence of the talus and a change in the length of the foot are common. Neurovascular injury is uncommon with closed ankle dislocations. There is a higher incidence of neurovascular injury with open dislocations. Ankle dislocations are associated with a risk of vascular injury and the development of a compartment syndrome from severe swelling.[12] Most vascular injuries are to the dorsalis pedis or posterior tibial vessels and may be accompanied by damage to the adjacent superficial peroneal nerve or sural nerve, respectively.[1,9] Tibiotalar dislocations rarely result in avascular necrosis.

INDICATIONS

All closed ankle fracture-dislocations and isolated dislocations should be reduced urgently to protect the soft tissues, decrease swelling, and minimize articular injury. Some authors

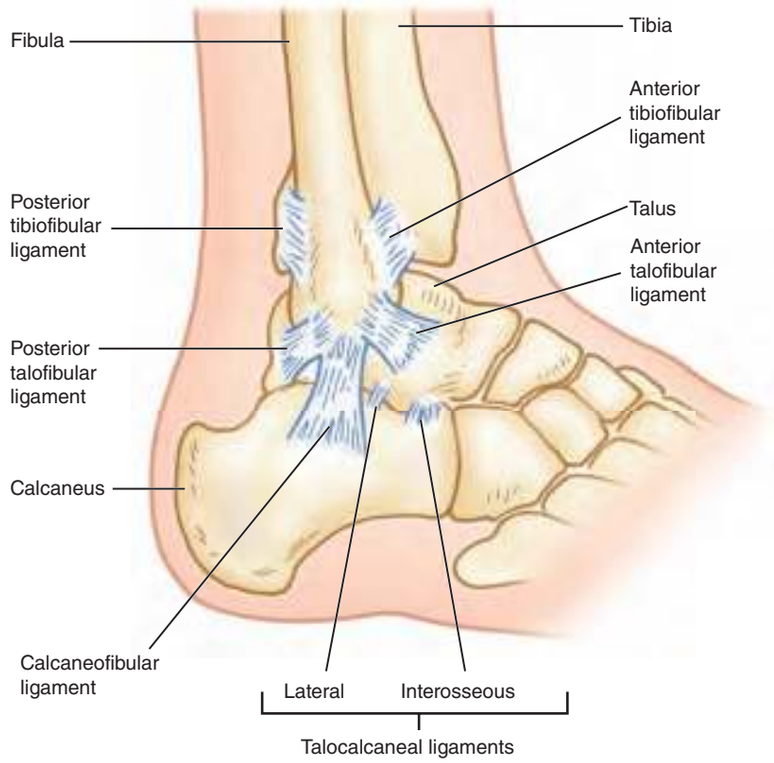


FIGURE 111-1. The bony and ligamentous structures of the lateral ankle.

recommend reduction prior to radiography.[17] **Any dislocation, open or closed, that has evidence of distal neurologic or vascular compromise must be reduced emergently.** Extreme lateral deviation may compromise the dorsalis pedis artery and requires prompt reduction.[17] All open dislocations require intravenous antibiotics, irrigation, surgical debridement, and reduction by an Orthopedic Surgeon in the Operating Room. However, reduction should occur in the Emergency Department if the Orthopedic Surgeon or the Operating Room is not immediately available.[5] Some physicians will reduce dislocations that are intact neurovascularly but the skin

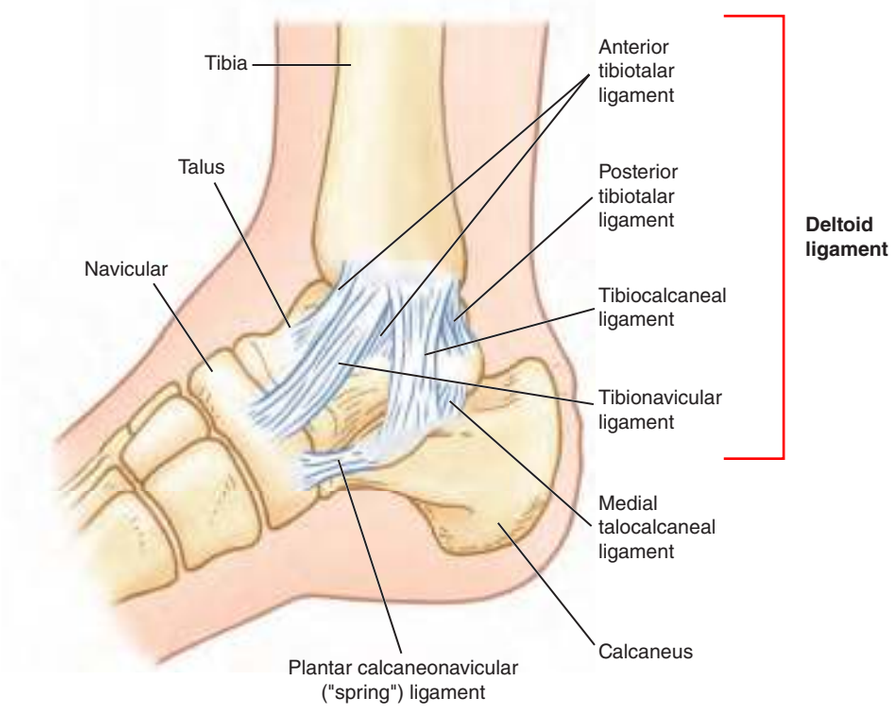


FIGURE 111-2. The bony and ligamentous structures of the medial ankle.

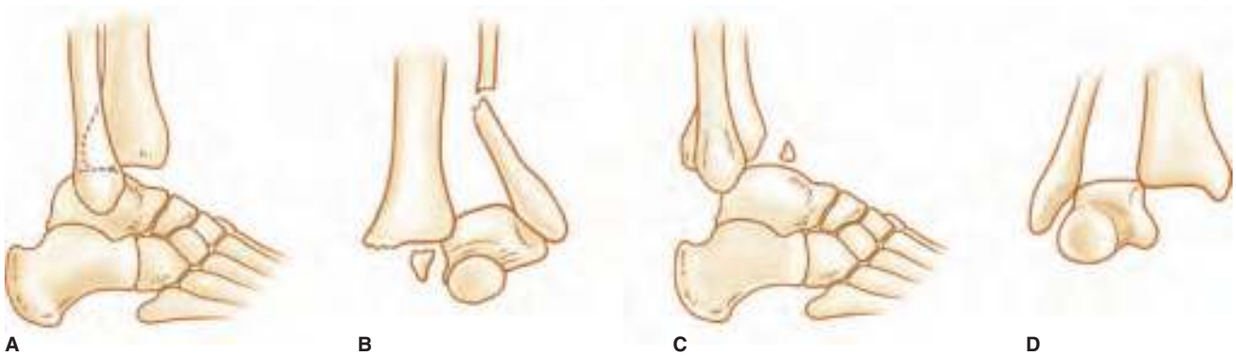


FIGURE 111-3. Types of ankle dislocations. ***A.*** Posterior. ***B.*** Lateral. ***C.*** Anterior. ***D.*** Superior.

is tense from the dislocation. This prevents skin necrosis over the ankle joint.

CONTRAINDICATIONS

There are no absolute contraindications to reducing a dislocated ankle. Some authors would not recommend Emergency Department reduction of an open fracture-dislocation without evidence of neurovascular compromise or in a setting where immediate orthopedic and operative intervention was available.

EQUIPMENT

- Local anesthetic solution
- 18 gauge needle
- 22 gauge needle, 2 inches long

FIGURE 111-4. Radiograph of a posterior ankle dislocation.

- Equipment and supplies for procedural sedation (Chapter 159)
- Equipment and supplies for intravenous (IV) regional anesthesia (Chapter 157)
- Stockinette
- Compressive wrap (e.g., Webril)
- Plaster, fiberglass, or commercially prepared splinting material
- Elastic bandage

PATIENT PREPARATION

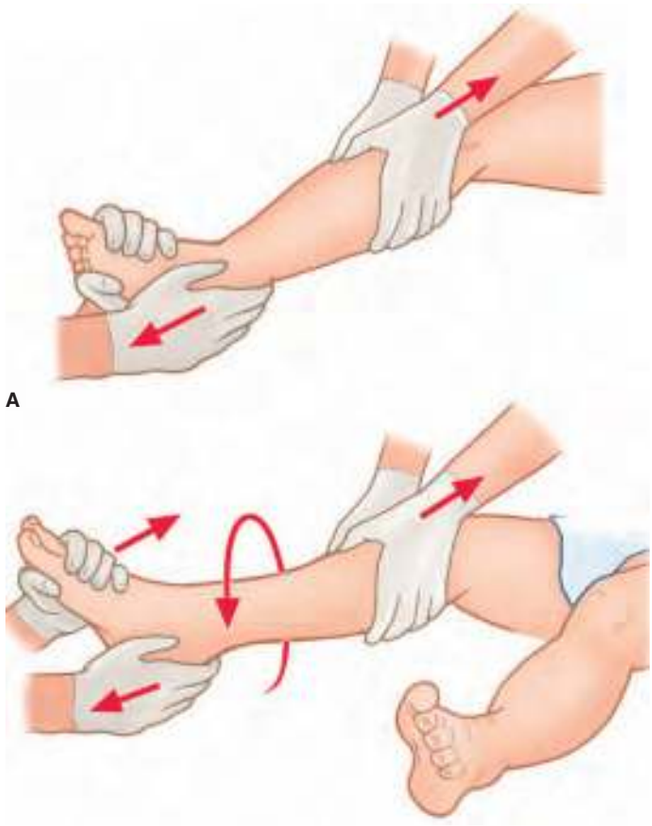
Explain the risks, benefits, and potential complications of the reduction procedure and the procedural sedation to the patient and/or their representative. Obtain an informed consent for both procedures. Place the patient supine with the affected foot at the edge of the gurney. Patients should be premedicated with an opioid analgesic prior to the procedure and ideally prior to radiography. Procedural sedation (Chapter 159) provides excellent analgesia, muscle relaxation, and sedation. It allows the reduction procedure to be more tolerable for both the patient and the Emergency Physician. Intraarticular lidocaine (Chapter 97) has been reported as an effective alternative to conscious sedation for closed reduction of ankle fracture dislocations.[18] Alternatives include IV regional anesthesia (i.e., Bier block) of the lower leg (Chapter 157) and ultrasound-guided distal popliteal sciatic nerve block (Chapter 156).[19]

TECHNIQUES

The techniques described below to reduce ankle dislocations have three things in common. The hip and knee are flexed to relieve the tension on the gastrocnemius and soleus muscles. The foot is flexed (i.e., plantarflexed or dorsiflexed) to unlock or disengage the talus. Finally, the talus is maneuvered into its proper anatomic position.

LATERAL ANKLE DISLOCATIONS

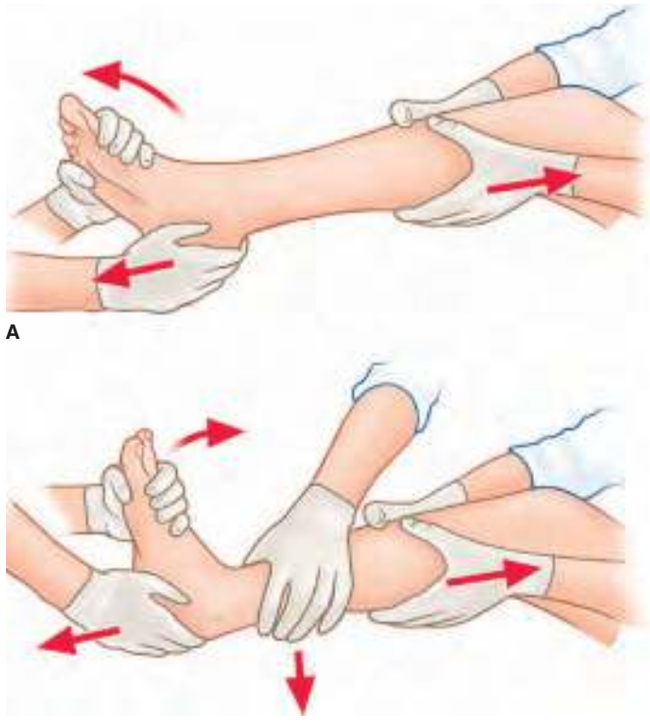
Flex the patient's hip and knee by placing a pillow behind their knee. Grasp the calcaneus with one hand and the forefoot with the other hand (**Figure 111-5*A***). Instruct an assistant to grasp the patient's calf. Apply distal traction to the heel while the assistant provides countertraction to the leg (**Figure 111-5*A***). The next step is to rotate the foot medially so that the great toe is in alignment with the anterior tibia while simultaneously dorsiflexing the foot and distracting the heel (**Figure 111-5*B***). The talus will reduce easily with a palpable and audible "clunk."



A

B

FIGURE 111-5. Closed reduction of a lateral ankle dislocation. ***A.*** The heel is distracted while an assistant provides countertraction. ***B.*** Simultaneously, medially rotate and dorsiflex the foot while distracting the heel.



A

B

FIGURE 111-6. Closed reduction of a posterior ankle dislocation. ***A.*** The heel is distracted and the foot is plantarflexed while an assistant provides countertraction. ***B.*** The foot is dorsiflexed while the heel is distracted and a second assistant applies posterior traction on the distal leg.

POSTERIOR ANKLE DISLOCATIONS

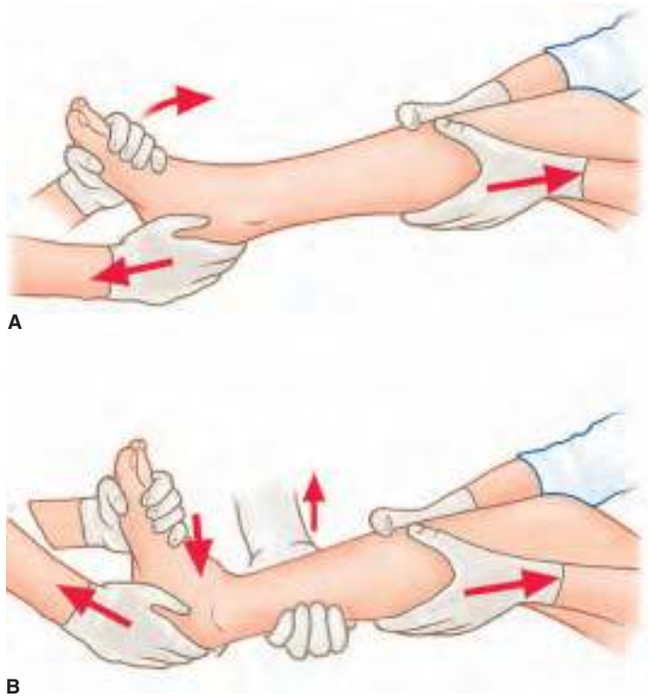
Flex the patient's hip and knee by placing a pillow behind their knee. Grasp the calcaneus with one hand and the forefoot with the other hand (**Figure 111-6*A***). Instruct an assistant to grasp the patient's calf. Simultaneously apply distal traction to the heel and plantarflex the foot while the assistant provides countertraction to the leg (**Figure 111-6*A***). The next step is to dorsiflex the foot while distracting the heel and a second assistant provides posteriorly directed pressure on the distal leg (**Figure 111-6*B***). The talus will reduce with a palpable and audible "clunk."

ANTERIOR ANKLE DISLOCATIONS

Flex the patient's hip and knee by placing a pillow behind their knee. Grasp the calcaneus with one hand and the forefoot with the other hand (**Figure 111-7*A***). Instruct an assistant to grasp the patient's calf. Simultaneously apply distal traction to the heel and dorsiflex the foot until the toes point upright while the assistant provides countertraction to the leg (**Figure 111-7*A***). The next step is to push the foot posteriorly while distracting the heel and a second assistant provides anteriorly directed pressure on the distal leg (**Figure 111-7*B***). The talus will reduce with a palpable and audible "clunk."

SUPERIOR ANKLE DISLOCATIONS

Superior ankle dislocations are associated with significant soft tissue and articular damage. Neurovascular injury is uncommon with



A

B

FIGURE 111-7. Closed reduction of an anterior ankle dislocation. ***A.*** The heel is distracted and the foot is dorsiflexed until the toes are upright while an assistant provides countertraction. ***B.*** The foot is pushed posteriorly while the heel is distracted and a second assistant applies anterior traction on the distal leg.

these dislocations. Superior ankle dislocations should be reduced, splinted, and managed by an Orthopedic Surgeon. The only exception to this is if there is neurologic and/or vascular compromise of the distal extremity and no Orthopedic Surgeon is immediately available.

OPEN ANKLE DISLOCATIONS

The Emergency Physician occasionally reduces open ankle dislocations if neurologic and/or vascular compromise of the foot is present and an Orthopedic Surgeon is not immediately available. Copiously irrigate the wound with sterile saline before attempting the reduction. The technique to reduce an open ankle dislocation is the same as that for a closed ankle dislocation.

ALTERNATIVE TECHNIQUE

A technique was developed by an Orthopedist and should be considered by the Emergency Physician if they do not have an assistant (**Figure 111-8**).[20] It is well described and worth trying. The Emergency Department has plenty of assistants who can help, including the nurse monitoring the patient. Consider this technique if the Emergency Department is busy and understaffed.

Place Kerlix gauze or stockinette as a sling behind the patient's knee (**Figure 111-8*A***). Pull the gauze to flex the hip to 90° or more (**Figure 111-8*B***). Tie the gauze to the gurney to maintain the patient's leg in this position. Use 2 inch gauze or stockinette and tie it into a loop. Slip the tied material between the first two toes (**Figure 111-8*C***). Pull the end of the loop over the toes and secure it over toes 1 and 2 (**Figure 111-8*D***). Lift the foot and ensure the gauze is secure (**Figure 111-8*E***). Hang the gauze over an IV pole and hang the foot (**Figure 111-8*F***).

Reduce the ankle dislocation as described above. Apply Webril (**Figure 111-8*G***) followed by a splint (**Figure 111-8*H***) to the extremity. Cut down the extremity after the splint is applied and hardened.

ASSESSMENT

Verify and document the neurologic and vascular status of the foot before and after any attempts at reduction.[21] Any diminution or the absence of normal neurologic or vascular signs requires emergent consultation with an Orthopedic Surgeon.

AFTERCARE

Splint the extremity (Chapter 113). Apply a three-sided short leg splint from the base of the toes to just below the knee for posterior, lateral, and superior ankle dislocations that have been reduced. Immobilize the ankle in 90° of flexion and in a neutral position with respect to inversion and eversion.[12] Immobilize the reduced anterior ankle dislocation with the ankle in slight plantarflexion. The limb should be elevated, not bear weight, have frequent neurologic and vascular checks, and have frequent assessments for the development of the signs associated with a compartment syndrome.[12]

COMPLICATIONS

Most complications occur from the dislocation or fracture-dislocation and not the reduction procedure. This includes neurologic damage, vascular damage, and compartment syndromes. A posttraumatic peroneal tendon dislocation can occur and may be initially unrecognized. The patient usually becomes symptomatic after the acute stage when the tendon subluxes and dislocates. There is a low rate of subsequently developing avascular necrosis and degenerative joint disease with isolated ankle dislocations.

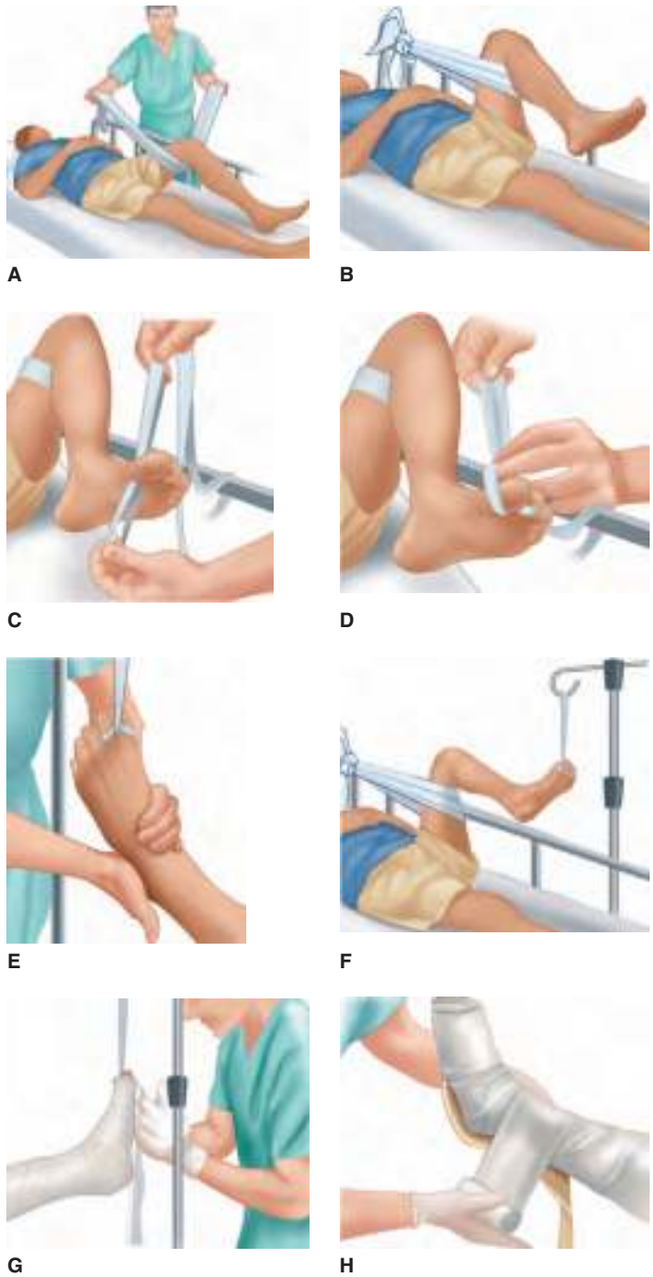


FIGURE 111-8. The one-person technique to care for a dislocated ankle. ***A.*** Gauze is placed behind the patient's knee. ***B.*** The hip is flexed and the gauze is tied to the gurney. ***C.*** Place looped 2 inch gauge between the first two toes. ***D.*** Pull the end of the loop over the toes and secure it. ***E.*** Lift the foot and ensure the gauze is secure. ***F.*** Hang the gauze over an IV pole, hang the foot, and reduce the ankle dislocation. ***G.*** Apply Webril. ***H.*** Apply the splint.

Complications associated with the reduction procedure, if they occur at all, are usually neurologic and vascular injuries. These structures may become impinged in the relocated joint or on a fracture fragment. **Emergently consult an Orthopedic Surgeon if there is any diminished or absent function of any nerve or artery.**

The ankle might not be reducible. Interposition of bony fragments, foreign bodies, ligaments, nerves, tendons, and vasculature can occur in the ankle joint. Inadequate anesthesia can also be a

reason the dislocation is not reducing. Repeated attempts at reducing the dislocated joint can cause additional injury to the soft tissues, make a closed dislocation into an open dislocation, or cause a fracture. Consult an Orthopedic Surgeon and consider reduction in the Operating Room under general anesthesia.

SUMMARY

Ankle dislocations without fractures are uncommon yet serious injuries. Closed ankle dislocations can be reduced emergently and successfully with the use of procedural sedation. Open ankle dislocations should be irrigated in the Emergency Department and reduced rapidly after consultation with an Orthopedic Surgeon. Reduction, irrigation, and debridement are all likely to occur in the Operating Room if an Orthopedic Surgeon is immediately available. Any ankle dislocation with evidence of distal neurovascular compromise should be reduced immediately. All patients with ankle dislocations should have an Orthopedic Surgery consultation. Most closed ankle dislocations are managed nonoperatively with good long-term results.

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112 Common Fracture Reduction

Christopher A. Gee

INTRODUCTION

Extremity fractures are common injuries and often necessitate an Emergency Department (ED) visit. Most closed fractures can be managed conservatively in the ED with splinting and follow-up if there is no neurologic or vascular compromise. This chapter addresses four common fracture patterns of the upper extremity that may require reduction by the Emergency Physician (EP). These include clavicular fractures, dorsally angulated distal radius or Colles fractures, displaced surgical neck fractures of the humerus, and supracondylar fractures of the humerus. **The reduction of fractures in the ED should involve consultation with an Orthopedic Surgeon prior to performing the procedure. The only exception to this is the existence of neurologic or vascular compromise and the Orthopedic Surgeon is not immediately available.**

CLAVICULAR FRACTURES

INTRODUCTION

Clavicular fractures are common and represent approximately 5% of all fractures.[1-4] Most of these occur at the junction of the middle and distal third of the clavicle, just medial to the coracoclavicular ligament. The clavicular fracture is the most common fracture encountered in childhood and occurs most often from a fall.[4] These fractures are usually detectable clinically (**Figure 112-1**) with plain radiographs (**Figure 112-2**) helping to confirm the diagnosis.[5] These fractures are relatively common and have a definite risk of associated complications.

ANATOMY AND PATHOPHYSIOLOGY

The clavicle is the only bony attachment of the upper extremity to the axial skeleton. It serves as a strut to support the shoulder girdle. It provides support and stabilization of the upper limb while allowing a broad range of movements. The clavicle is securely attached at both the acromioclavicular and sternoclavicular joints by ligaments

FIGURE 112-1. Middle third clavicle fracture with soft tissue swelling. (From Sherman SC, et al: *Simon's Emergency Orthopedics*, 7th ed. New York: McGraw-Hill; 2014. Courtesy of Northwestern Emergency Medicine teaching file with permission.)

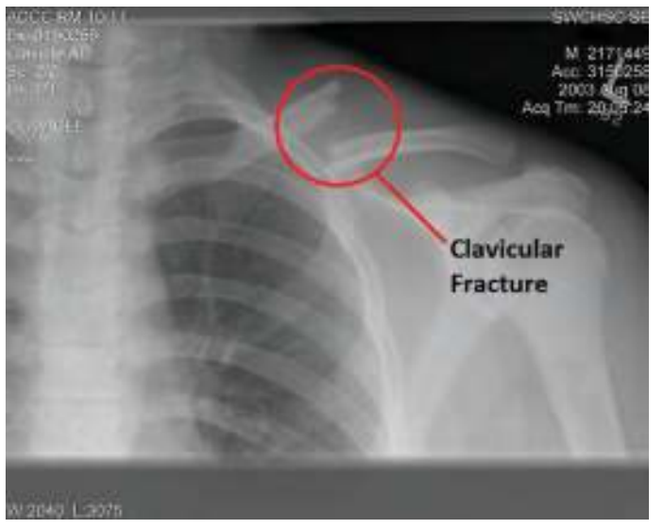


FIGURE 112-2. Radiograph of a clavicle fracture. (Used from Kin412CF from www.commons.wikimedia.org.)

(**Figure 112-3**). The subclavian vessels and nerves of the brachial plexus pass posterior and inferior to the clavicle at its midportion where it overlies the first rib. The proximity of these neurovascular structures and the underlying lung accounts for most of the potential complications of clavicular fractures.

The most commonly used classification for clavicular fractures was proposed by Allman.[6] This simple classification is useful clinically and mechanistically but it does not predict outcomes. Group I fractures are midclavicular and account for approximately 80% of clavicular fractures. These most often result from a shearing force applied to the lateral aspect of the shoulder. Group II fractures involve the distal third of the clavicle and account for approximately 15% of all clavicular fractures. These most often result from a direct blow to the top of the shoulder. Several additional subclassifications have been proposed for these fractures based on the location of the fracture and associated ligamentous injury (e.g., Neer's classification recognizes the importance of the coracoclavicular ligaments in providing support to distal clavicle fractures).[7] Operative repair is suggested for some of these subtypes. Refer all distal clavicular fractures for follow-up within 5 to 7 days to an Orthopedic Surgeon.[1,2] Group III

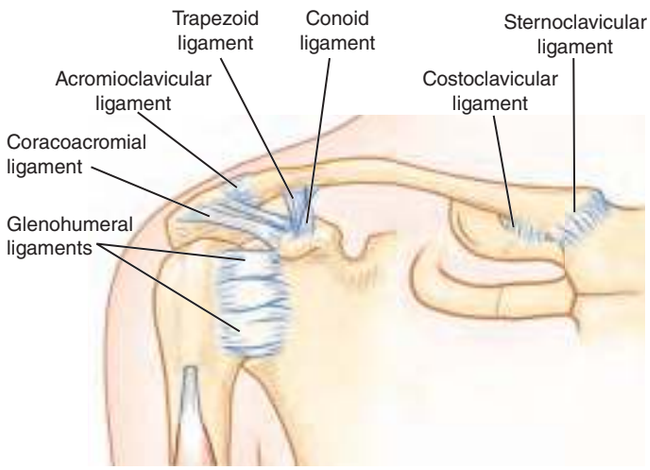


FIGURE 112-3. The clavicle serves as a strut between the torso and upper extremity. The brachial plexus and great vessels pass behind the middle third of the clavicle.

fractures represent about 5% of clavicular fractures and involve the proximal third of the clavicle. They often result from a direct blow to the chest.

Patients with clavicular fractures are easily identified clinically. The clavicle is almost entirely subcutaneous and allows most displaced fractures to be palpated. Presenting signs and symptoms include ecchymoses, edema, and localized pain. Physical examination findings include superior and posterior displacement of the proximal portion of the clavicle due to traction from the sternocleidomastoid muscle (**Figures 112-1 and 112-4**). The shoulder is often displaced inferiorly by the weight of the upper extremity and medially by traction from the pectoral and the latissimus dorsi muscles. This position is protective for the patient but can exacerbate fracture displacement and overlap.

Most fractures are readily identifiable on standard anteroposterior radiographs (**Figure 112-2**). Some group II and III fractures may not be readily identifiable.[1] Additional views at a 45° angle cephalad (i.e., apical lordotic view) or 15° angle posteroanterior may be useful to assess displacement.[1,3,8] Evaluation of the fracture by computed tomography (CT) may be necessary to confirm a clinically suspected fracture or to fully evaluate the degree of displacement. CT is usually required for adequate visualization of medial-end fractures extending into the sternoclavicular joint.[9] Special views (i.e., cone views, tomograms, and upper rib films) may be helpful and are best determined in consultation with an Orthopedic Surgeon.

Clavicular injury and pain in children present two concerns. Nondisplaced greenstick fractures to the clavicle may not be radiographically visible for 7 to 10 days.[2,3] Clinical suspicion of a clavicular fracture with a negative radiograph should prompt conservative management (i.e., rest, sling, icing, and pain control). Arrange follow-up for 7 to 10 days after the injury to obtain repeat radiographs. It may be unclear if the physes are involved in some group II and III fractures.[2,3] **Any fracture through the physis requires an urgent referral to an Orthopedic Surgeon.**

INDICATIONS

Most clavicle fractures are treated nonoperatively. Reduction of clavicular fractures is necessary in a few circumstances. **It is required if neurologic and/or vascular compromise is present in the affected extremity.** This includes medial fracture displacement producing superior mediastinal compromise (e.g., aerodigestive tract compression, head vascular compression, or neck vascular compression). Make an emergent attempt at closed reduction.[8,10] **Tenting of the skin by fracture fragments has the potential to convert a closed fracture to an open injury.** Manipulate or repair the fracture in the Operating Room. Reduce distal clavicular fractures that are significantly displaced. Reduction is otherwise optional and at the discretion of the treating physician.

Refer patients actively involved in competitive athletics or who have jobs that require overhead use of their arms (e.g., painters) for possible operative reduction by an Orthopedic Surgeon. Recent evidence has reversed trends to treat all clavicle fractures nonoperatively.[4,11-13] Up to 20% of sports-related clavicle fractures are treated with surgery.[14] Research has shown that athletes and workers with overhead jobs may get back to play or work sooner with surgical treatment.[15] This is particularly true with complete displacement, comminution, or over 2 cm of shortening.[15]

CONTRAINDICATIONS

Reduction of most clavicular fractures is not usually necessary in either the pediatric or adult patient.[1,2] A sling for simple arm support provides results comparable to the figure-of-eight harness without the risk of brachial plexus injury or patient discomfort.[1-3]

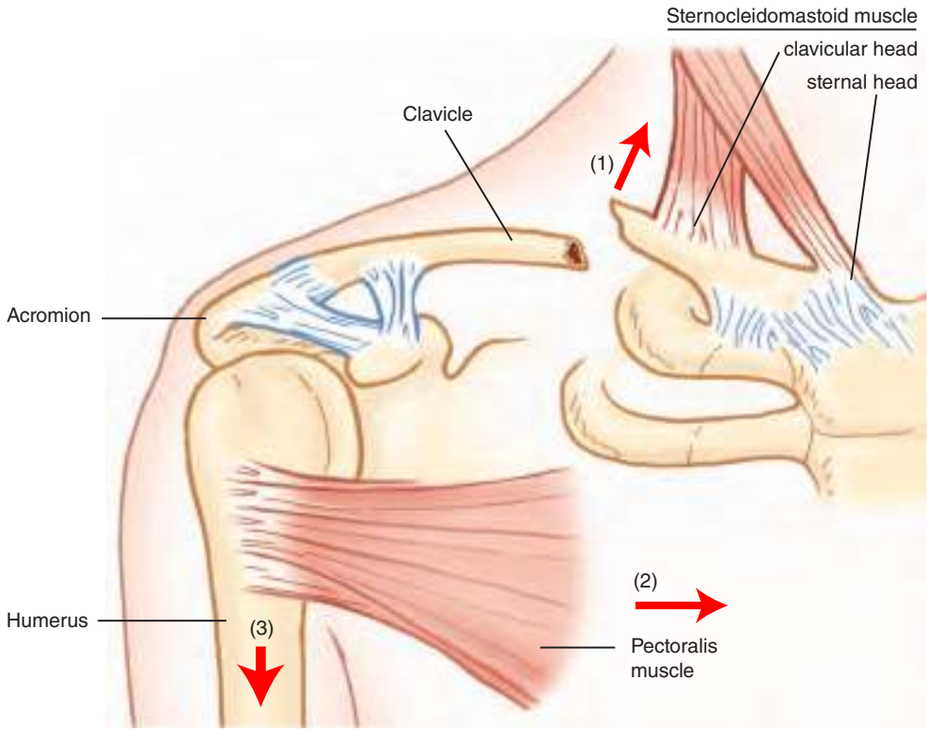


FIGURE 112-4. Displacement of the clavicle and shoulder after a clavicular fracture. The clavicular head of the sternocleidomastoid muscle displaces the proximal clavicular fragment superiorly and posteriorly (*1*). The pectoralis major and latissimus dorsi muscles pull the shoulder medially (*2*). The force of gravity displaces the distal clavicle and shoulder inferiorly (*3*).

The sling may be additionally supported by a swath (**Figure 112-5*A***) or a Velpeau wrap (**Figure 112-5*B***). The Velpeau wrap is a sling-and-swath technique that positions the forearm diagonally rather than horizontally (**Figure 112-5*B***). The Velpeau wrap has no practical advantages over a sling and swath.

Many physicians still prefer the use of a figure-of-eight harness (**Figures 112-5*C*, 112-5*D*, and 112-5*E***). The figure-of-eight harness still represents the treatment of choice in patients over the age of 10 years in the presence of greatly displaced fragments.[2,3] There is no evidence that the figure-of-eight harness offers any advantage over a simple sling for midclavicular fractures. Some patients may not tolerate a figure-of-eight harness initially due to the pain of the harness crossing the fracture line, but it may provide more usability of the extremity with time.[16] The rates of compression of the neurovascular bundle, axillary pressure sores, and nonunion are higher in patients treated with the figure-of-eight harness.[8,9]

Contraindications to the reduction of a clavicular fracture include other injuries that represent a threat to life or limb. The patient's airway, breathing, and circulation must first be addressed and stabilized. Patients with open clavicular fractures require emergent consultation with an Orthopedic Surgeon, intravenous antibiotics, and hospital admission for possible open reduction and internal fixation. Reduction is also contraindicated if an expanding hematoma, indicative of a vascular injury, is observed. Unfamiliarity with the reduction technique is a relative contraindication.

EQUIPMENT

- Figure-of-eight harness
- Sling
- Swath
- Kerlix rolls
- Elastic bandage

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Consider the administration of oral, intramuscular, or intravenous analgesics for patient comfort during the procedure. Procedural sedation (Chapter 159) is not required for this fracture reduction.

TECHNIQUE

Sit the patient upright on the side of the stretcher with their feet on the floor. Alternatively, the patient may be standing upright. Stand behind the patient. Grasp and pull both of the patient's shoulders backward as if the patient were standing at attention. Alternatively, this can be performed from the front with the patient's back against a corner. Instruct an assistant to apply the figure-of-eight splint while the patient is in this position. Apply the harness like a backpack and tighten the harness (**Figures 112-5*C*, 112-5*D*, and 112-5*E***). **Reassess the neurologic and vascular integrity of the affected extremity after applying the harness.**

ASSESSMENT

Assess the neurologic and vascular integrity of the upper extremity for all patients initially, following any reduction attempts, and after the application of a figure-of-eight harness. Any noted neurologic and/or vascular compromise requires the removal of the harness. Reassess the neurologic and vascular integrity. Any residual neurologic and/or vascular compromise requires an emergent consultation with an Orthopedic or Vascular Surgeon.

AFTERCARE

Refer patients with uncomplicated fractures to an Orthopedic Surgeon in 7 to 10 days. Patients with group II distal fractures and

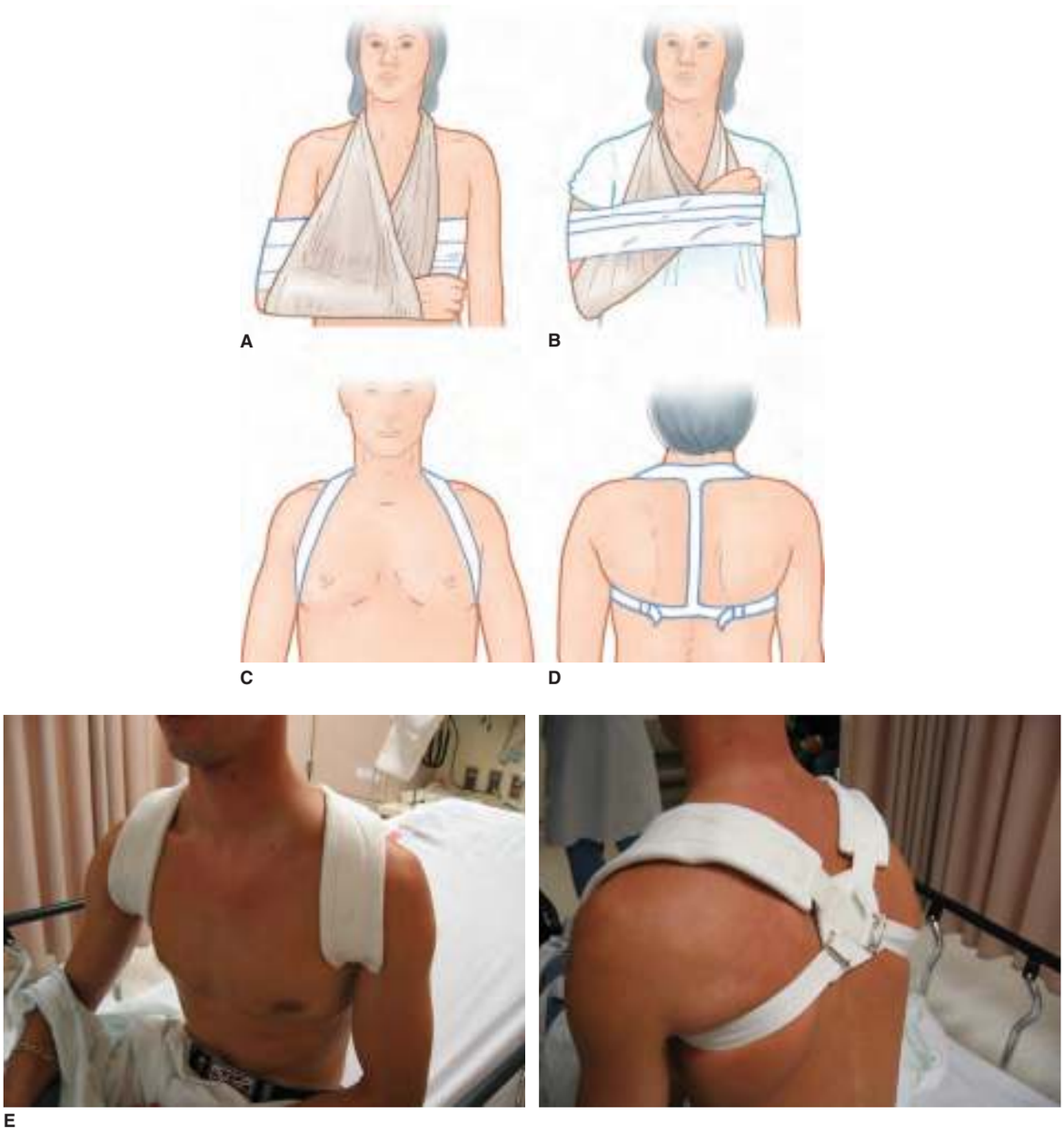


FIGURE 112-5. Treatment of clavicular fractures. ***A.*** Sling-over-swath immobilization. ***B.*** Velpeau sling immobilization. ***C.*** Anterior view of the figure-of-eight harness. ***D.*** Posterior view of the figure-of-eight splint. ***E.*** Photo of the figure-of-eight harness on a patient. (Used with permission from Sherman SC, et al: *Simon's Emergency Orthopedics*, 7th ed. New York: McGraw-Hill; 2014.)

any fracture in a child potentially involving the physis should have an urgent consultation with an Orthopedic Surgeon within 48 to 72 hours. Any patient with neurologic or vascular compromise, a pneumothorax, or signs of vascular injury from a clavicle fracture should be stabilized and admitted to the hospital after an emergent consultation with an Orthopedic Surgeon.

Discontinue the sling and encourage normal activities as pain allows. Instruct the patient to begin self-administered range-of-motion exercises (e.g., shoulder pendulums and "sawing" motion to prevent loss of glenohumeral mobility). Recovery of the range of motion of the shoulder is usually swift. Supervised physiotherapy is rarely required.[10]

General principles of orthopedic care are recommended. These include the application of ice, rest, nonsteroidal anti-inflammatory drugs, and narcotic analgesics. Most patients find the figure-of-eight harness difficult to apply, extremely uncomfortable, and remove it shortly after its application. It is better tolerated after acute pain has improved. If the patient tolerates the harness, it should be tightened daily to ensure a shoulder back "at attention" posture. The figure-of-eight splint should be worn until there is evidence of clinical union and the arm can be abducted without pain. This generally requires 3 to 5 weeks in children and 6 or more weeks in adults.[1,2] It may be more advantageous to apply a sling and swath or a sling and Velpeau wrap for patient comfort and compliance early on (**Figures 112-5*A* and 112-5*B***). The outcomes of applying a figure-of-eight harness versus a sling are equivalent.[17,18]

The sling is used to immobilize and support the elbow, forearm, and hand. They are simple, inexpensive, and effective. **It is imperative that the sling not be too short to allow the wrist and hand to hang over the sling.** This can result in an ulnar nerve neurapraxia.

The addition of a swath to a sling is used to immobilize dislocated shoulders that have been reduced and proximal humeral fractures (**Figure 112-5*A***). The swath immobilizes the humerus against the torso to limit motion at the shoulder. A shoulder immobilizer may be substituted for a sling and swath.

COMPLICATIONS

Complications of the reduction procedure include injuries to the brachial plexus, subclavian artery, and subclavian vein.[1,2] These are usually the result of the initial injury and not the reduction procedure. **It is imperative to perform a neurologic and vascular examination before and after any attempt at reducing a clavicular fracture. Any neurologic or vascular deficit requires an immediate consultation with an Orthopedic or Vascular Surgeon.**

SUMMARY

Clavicular fractures are common, easily diagnosed, and often treated conservatively in the ED. Fractures of the distal or medial third may be more challenging. The incidence of complications is low. A thorough examination for associated injury is required. Any evidence of neurologic, vascular, or aerodigestive compromise requires an emergent consultation with an Orthopedic Surgeon.

DISTAL RADIUS COLLES FRACTURE

INTRODUCTION

A Colles fracture is an eponym for a transverse fracture of the distal radial metaphysis with dorsal displacement and angulation of the distal fragment.[19,20] The fracture usually occurs 2 cm from the distal end of the radius (**Figures 112-6, 112-7, and 112-8**). The most common mechanism producing a Colles fracture is a fall on an outstretched hand.[21,22] Most fractures occur in patients 50 years of age and older.[21,22] This fracture is more commonly seen in women than in men.

The Colles fracture is the most common fracture of the wrist.[21,22] Familiarity with its presentation, indications for reduction, and method of reduction are essential. Consult an Orthopedic Surgeon as these patients will need long-term follow-up and care. This is important due to the high incidence of long-term complications that may result, even when these fractures are managed and reduced appropriately.[21,22]

FIGURE 112-6. Classic "dinner fork" deformity of a Colles fracture. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill, 2016. Photo contributor: Kristin L. Stevens, MD.)

FIGURE 112-7. The Colles fracture. ***A.*** The dinner fork deformity. ***B.*** Anteroposterior view. ***C.*** Lateral view.

ANATOMY AND PATHOPHYSIOLOGY

The distal radius is involved in two important articulations. First is the distal radioulnar joint which in conjunction with the proximal radius is responsible for pronation and supination of the forearm. The second is the wrist articulation. The distal radius normal anatomy can be remembered using the 11, 11, 22 rule. The radial length relative to the ulna should be 11 mm. The radius should have a volar tilt of approximately 11°. The radial inclination (i.e., degree of ramp up from ulnar side) should be approximately 22°. There are variances to this rule, but these numbers work as a target for reduction. **It is important to maintain the anatomic position of the distal radius with the reduction of a Colles fracture so that the patient retains good function of the wrist and distal radioulnar joints.**[22]

A Colles fracture can be associated with several other significant injuries. Up to 60% of patients have a fracture of the ulnar styloid process.[21,22] Other injuries include carpal fractures, distal radioulnar joint subluxations, flexor tendon injuries, median nerve injuries, ulnar neck fractures, and ulnar nerve injuries. A thorough physical

FIGURE 112-8. Radiograph of a Colles fracture. Views include the anteroposterior (left), lateral (middle), and Oblique (right). (Used from Ashish j29 from www.commons.wikimedia.org.)

A B

FIGURE 112-9. Clinical photo of a Smith fracture. (Used with permission from Sherman SC, et al: *Simon's Emergency Orthopedics*, 7th ed. New York: McGraw-Hill; 2014.)

examination and evaluation of the radiographs will uncover these injuries. **The forearm is effectively a ring structure, and a fracture at one location can result in a fracture or dislocation at a distal site.**

Standard radiographs include the anteroposterior and lateral views of the wrist (**Figure 112-8**). The Colles fracture is classically described as a "dinner fork" deformity when seen on lateral view (**Figures 112-6, 112-7*A*, and 112-8**). Intraarticular involvement with the fracture is rare and should prompt an emergent consultation with an Orthopedic or Hand Surgeon for reduction.[22]

A variation of the Colles fracture is the Smith fracture or the reverse Colles fracture (**Figure 112-9**). A Smith fracture is like a Colles fracture except that the distal fracture fragment is displaced in a volar direction. This fracture most often results from a direct blow to the wrist while the hand is flexed. It is more commonly seen in young males. The management of these fractures is like that of the Colles fracture.

INDICATIONS

Nondisplaced distal radius fractures can be placed in a splint and the patient referred for follow-up with an Orthopedic or Hand Surgeon as an outpatient. Displaced fractures should be reduced.[21,22] Simple Colles fractures may be reduced after consultation with an Orthopedic or Hand Surgeon. Some Orthopedic and Hand Surgeons prefer to reduce these fractures themselves often prior to open reduction and internal fixation. **The fracture must be emergently reduced if the patient has any neurologic and/or vascular compromise and the Orthopedic or Hand Surgeon is not immediately available.** The goal is to relieve the neurologic and/or vascular compromise. Ideal positioning is the goal but is not required as the Surgeon can later reduce any nonanatomic bony defect.

CONTRAINDICATIONS

Contraindications to the reduction of a Colles fracture include other injuries that represent a threat to life or limb. Airway, breathing, and circulation must first be addressed and stabilized. An Orthopedic or Hand Surgeon should reduce any fractures that involve the radioulnar or wrist joint. Complex, comminuted, or open fractures require reduction by an Orthopedic or Hand Surgeon. Unfamiliarity with the reduction technique is a relative contraindication.

Any patient presenting with a Colles fracture should be evaluated for the presence of a compartment syndrome (Chapter 93). **Any suspicion of a compartment syndrome necessitates having intracompartmental pressures measured (Chapter 93). Elevated compartmental pressures require an emergent consultation with an Orthopedic, Hand, or General Surgeon.** Reduction and fasciotomies (Chapter 94) should be performed in the Operating Room.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Local anesthetic solution, 1% to 2% lidocaine
- 10 mL syringe
- 18 gauge needle
- Finger trap
- Compressive cotton bandage (e.g., Webril)
- Elastic wrap
- 8 to 10 pounds of weights
- Casting material (e.g., plaster, fiberglass, or prepackaged splints)
- Analgesia or procedural sedation supplies (Chapter 159)
- Ultrasound or fluoroscope

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record.

The patient should be given adequate analgesia for the procedure. This can often be accomplished with a hematoma block (Chapter 155).[23-25] The procedure is briefly described here. Clean the skin overlying the fracture of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the skin and allow it to dry. Place a subcutaneous wheal of local anesthetic solution over the area of the fracture hematoma. Insert an 18 gauge needle attached to a 10 mL syringe containing local anesthetic through the anesthetized skin and into the fracture hematoma. The entry site may need to be proximal from the fracture and the needle angled into the hematoma. Aspirate blood from the hematoma site to confirm proper needle placement. Inject 5 to 10 mL of the local anesthetic solution into the hematoma surrounding the fracture. Although a hematoma block will provide adequate analgesia in most patients, it may be incomplete. Adjunctive or alternative anesthesia includes intramuscular analgesics, intravenous analgesics, regional nerve blocks (Chapter 156), intravenous regional anesthesia (Chapter 157), and procedural sedation (Chapter 159).[26]

TECHNIQUE

Place the patient supine on a stretcher. Perform a hematoma block as described above. Provide supplemental analgesia to the patient if required. Position the patient (**Figure 112-10*A***). Abduct the arm 90° and allow it to hang over the edge of the bed. Flex the elbow 90° with the hand pointing upright. Insert the thumb, index finger, and long finger into the finger trap (**Figure 112-10*B***). Suspend 8 to 10 pounds of weights from the distal humerus (**Figures 112-10*C* and 112-10*D***). Allow the patient to remain in this position for 10 to 20 minutes to distract and disimpact the fracture fragments.

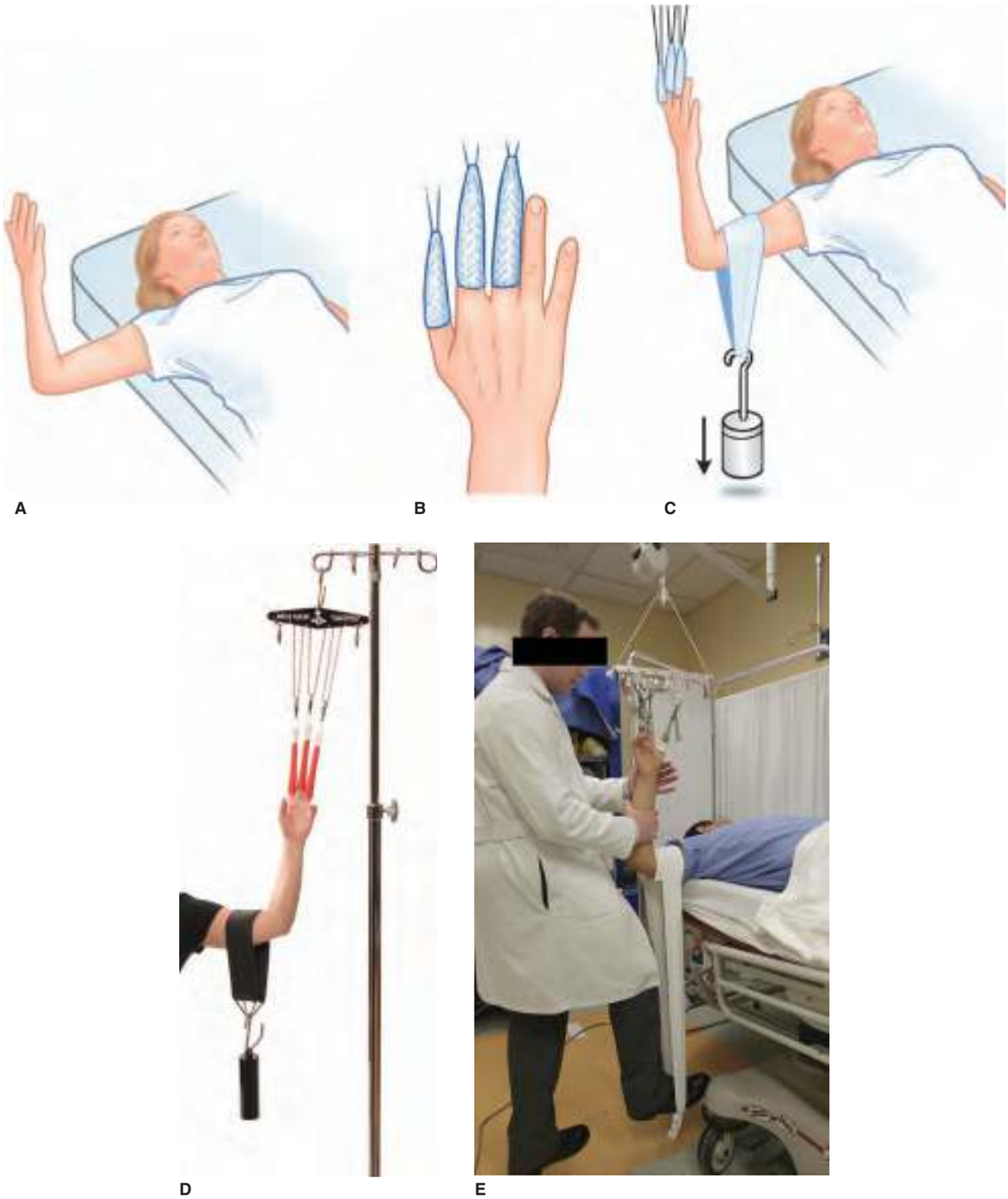


FIGURE 112-10. Patient positioning for the reduction of a Colles fracture. ***A.*** The arm is abducted 90° and the elbow is flexed 90°. ***B.*** The thumb, index finger, and long finger are placed in the finger trap. ***C.*** Weights are suspended from the distal humerus. ***D.*** Clinical photo. (Photo courtesy of Instrument Specialists Inc., Boerne, TX.) ***E.*** Using a loop of stockinette. (Used with permission from reference 27.)

This alone will often improve fracture alignment significantly. A loop of stockinette can be used if no weights are available (**Figure 112-10*E***).[27]

The fracture reduction involves traction followed by manipulation of the distal radial fragment to reverse the action that resulted in the fracture (**Figure 112-11**). Place both hands around the patient's wrist with the thumbs at the base of the fracture site (**Figure 112-11*A***). Displace the fracture fragment distally with your thumbs while maintaining traction with the finger trap and the weights (**Figure 112-11*A***). Firmly grasp the distal fragment with your thumbs over the fracture site. Accentuate the deformity by bending more dorsally and then swing the distal fragment back into position while applying traction. This maneuver allows the distal fragment to become free from any contacts with the proximal radius which may prevent its movement. The wrist should look normal and the dorsal radius should feel flat. Don't let the normal bump of Lister's tubercle throw you off! Ultrasound or fluoroscopy can be

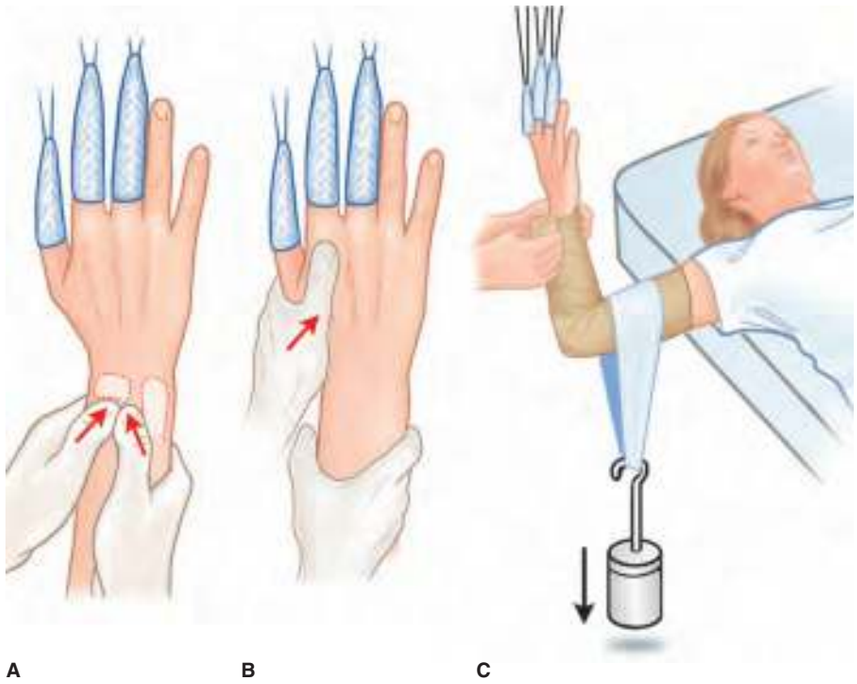


FIGURE 112-11. Reduction of a distal radius fracture. ***A.*** Proper positioning of the EP's hands. The arrows represent the application of a distally directed force. ***B.*** Application of an ulnar-directed force to reduce the radial deviation. ***C.*** The application of a splint to an unstable fracture. The finger trap and weights remain during the splinting.

used to verify reduction. Continue to manipulate the fragment distally while simultaneously manipulating it in a volar direction until the fragment assumes the proper anatomic position.

In the case of a volar angulation or Smith fracture, these manipulations would simply be reversed to reduce the displacement. Slight ulnar deviation of the fragment is often necessary (**Figure 112-11*B***). Remove the weights. Palpation of a smooth surface at the radial and dorsal aspects of the radius indicates an appropriate reduction. Remove the finger trap and apply a splint.

Immobilize the forearm with a sugar tong splint. Place the forearm in a neutral position, halfway between pronation and supination. Place the wrist in 15° to 20° of flexion and 20° of ulnar deviation. Unstable fractures are best splinted immediately after the reduction while the hand is still maintained in the finger trap for traction (**Figure 112-11*C***). Alternatively, the EP can maintain reduction while an assistant places the splint. **If a long arm cast is applied, be sure to bivalve it to prevent complications.** A short arm splint or cast may be used if the fracture is stable and impacted or is stable in an elderly person who needs to maintain mobility of the elbow.

ASSESSMENT

All patients should be assessed initially and following any reduction attempts for neurologic and vascular integrity of the extremity.[28] Obtain postreduction radiographs to confirm proper bony positioning. The procedure may be repeated if the radiographs show an incomplete reduction. Bedside ultrasound is a reliable and convenient method of assessing the reduction.[29-31] It is noninvasive, does not use ionizing radiation, involves minimal contact, and does not require the patient to remain motionless.[32]

The goal is the restoration of the normal relationships and angles of the radius with congruity of the radiocarpal and radioulnar joints. Remember the 11, 11, 22 rule. The radial length relative to ulna should be 11 mm. The radius should have a volar tilt of approximately 11°. The radial inclination (i.e., degree of ramp up from ulnar side) should be approximately 22°.

Positioning is not critical if the reduction was performed for neurologic and/or vascular compromise. The primary consideration is the relief of the compromised nerve and/or artery. The Orthopedic or Hand Surgeon can later reduce the bony defect that remains.

AFTERCARE

Patients with uncomplicated or nondisplaced fractures should be referred to an Orthopedic or Hand Surgeon within 5 to 7 days. Patients with unstable fractures should be evaluated within 24 to 48 hours. **All patients should be given written instructions regarding the signs and symptoms of the splint or cast being too tight or a potential compartment syndrome.** Any patient with an open fracture, evidence of neurologic and/or vascular compromise, or suspicion of a compartment syndrome should be admitted to the hospital after an emergent consultation with an Orthopedic or Hand Surgeon.

General principles of orthopedic care are recommended. These include rest, elevation of the arm, nonsteroidal anti-inflammatory drugs, and narcotic analgesics. Instruct the patient to exercise the fingers and shoulder to prevent weakness, muscular atrophy, and the ligaments surrounding these joints from becoming taut.

COMPLICATIONS

Complications of the reduction procedure include postreduction edema and bleeding into the forearm. These may contribute to the development of a compartment syndrome. A neurapraxia of the median nerve is infrequent but possible. **Most complications result from the injury that produced the fracture and underlie the need for a good neurologic and vascular examination prior to any attempts at reduction.**[28] Any diminished or absent neurologic or vascular function requires an emergent consultation with an Orthopedic or Hand Surgeon. Incomplete reduction can result in future morbidity (e.g., pain, decreased range of motion, and deformity).

SUMMARY

A dorsally displaced distal radius or Colles fracture is the most common fracture of the wrist. Familiarity with its presentation and the method of reduction are essential. Some Orthopedic and Hand Surgeons prefer to reduce these fractures. Consultation is advised prior to reduction unless the extremity has evidence of neurologic or vascular compromise. These fractures have a high incidence of long-term complications, even when appropriately managed and reduced.[21,22]

DISPLACED SURGICAL NECK FRACTURE OF THE HUMERUS

INTRODUCTION

Proximal humeral fractures are relatively common, approximately 5% of injuries to the appendicular skeleton (**Figures 112-12 and 112-13**).[33] Most patients presenting to the ED with a proximal humeral fracture are elderly and usually have osteoporosis. The most common mechanism of injury involves a fall on an outstretched hand with the elbow extended.[3,33] The EP can manage most of these fractures conservatively.[33] The EP must have a basic knowledge of the types of proximal humeral fractures, particularly those that should be managed by the Orthopedic Surgeon, and the indications for emergent reduction.

ANATOMY AND PATHOPHYSIOLOGY

The proximal humerus is composed of the articular segment, the greater and lesser tuberosities, and the proximal humeral shaft. This anatomic division is based on the physeal lines and the development of the humerus.[2,3,33] The commonly used Neer classification uses the observation that proximal humeral fractures separate primarily along these physeal lines.[2,3,33] It is the displacement of fragments, not the total number of fracture lines, that is important. Significant displacement is considered to be a separation of greater than 1 cm, angulation of more than 45°, or displacement of the greater tuberosity greater than 0.5 cm.[3,33] The Neer classification separates fractures into one-part to four-part fractures. Approximately 80% of proximal humeral fractures are one-part or minimally displaced fractures.[3,33] These fractures can be managed conservatively. Multiple-part fractures often require surgical intervention by an Orthopedic Surgeon. **A surgical neck fracture of the humerus is the only proximal humeral fracture that should be reduced by the Emergency Physician. These can be difficult fractures to get back into anatomic alignment as the fragments may be oblique or transverse and slide off one another. The purpose is to get the fragments into acceptable displacement and angulation and not perfect anatomic alignment.**

FIGURE 112-12. Surgical neck fracture of the humerus. (Used with permission from Sherman SC, et al: *Simon's Emergency Orthopedics*, 7th ed. New York: McGraw-Hill; 2014.)

FIGURE 112-13. Radiograph of a displaced surgical neck fracture of the humerus. (Used with permission from Sherman SC, et al: *Simon's Emergency Orthopedics*, 7th ed. New York: McGraw-Hill; 2014.)

INDICATIONS

Surgical neck fractures of the humerus may be reduced in the ED after consultation with an Orthopedic Surgeon. **The reduction should be undertaken emergently after consultation with the Orthopedic Surgeon and if they are not immediately available if the patient has neurologic and/or vascular compromise.**

CONTRAINDICATIONS

Contraindications to the reduction of humeral fractures include other injuries that represent a threat to life or limb. Airway, breathing, and circulation must first be addressed and stabilized. An Orthopedic Surgeon should manage any open fracture, complex comminuted fracture, or multiple-part fracture as the patient often requires operative repair and reduction.[2,3,33] Unfamiliarity with the reduction technique is a relative contraindication. Children presenting with separation of the proximal humeral epiphysis require meticulous realignment. These fractures should be reduced by an Orthopedic Surgeon.[2]

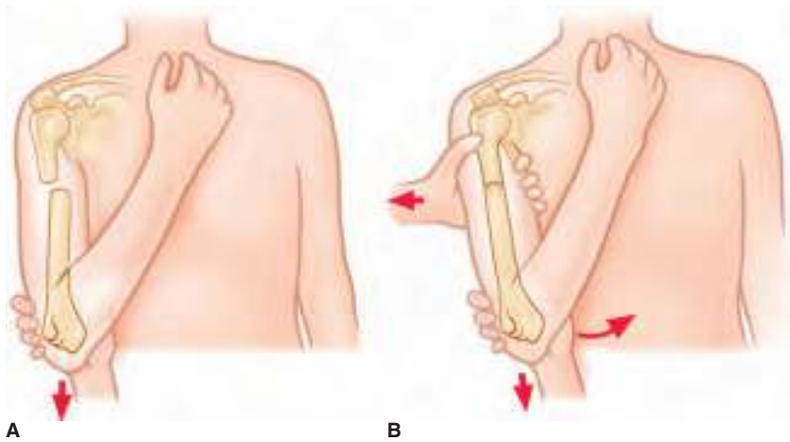


FIGURE 112-14. Reduction of a displaced surgical neck fracture of the humerus. ***A.*** The application of distal traction to the elbow. ***B.*** Lateral pressure is applied to reduce the fracture while maintaining distal traction, adducting the elbow, and slightly flexing the arm.

EQUIPMENT

- Supplies and equipment for procedural sedation (Chapter 159)
- Compressive cotton bandage (e.g., Webril)
- Casting material (e.g., plaster, fiberglass, or prepackaged splints)
- Sling
- Elastic wrap

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Adequate anesthesia for this procedure is best accomplished with procedural sedation (Chapter 159). Obtain an informed consent for the procedural sedation procedure.

TECHNIQUE

Place the patient supine. Perform procedural sedation (Chapter 159). Completely flex the patient's elbow. Apply a distractive force along the long axis of the humerus by applying traction on the patient's elbow (**Figure 112-14*A***). Simultaneously slightly adduct the arm across the chest to allow relaxation of the pectoralis muscle, slightly flex the arm, and apply lateral pressure to the fracture site while distracting the humerus (**Figure 112-14*B***). Slowly release the traction on the elbow as the fragments come into good reduction.

Apply a sugar tong coaptation splint from over top of the shoulder, laterally down the upper arm, under the elbow, and back up into the axilla (**Figure 112-15*A***). A trick to helping this splint stay in place is to put the splint material into a tube gauze and cut strips into the ends of the gauze. Then tie the upper end around the patient's neck and the other end around the top of the shoulder to keep the splint in place. Secure the splint in the usual manner with an elastic wrap. Apply a sling (**Figure 112-15*B***). Place the arm slightly across the chest and apply a sling and swath in the event of an unstable proximal humeral fracture (**Figure 112-5*A***). This can help to limit the pull of the pectoralis muscle on the fracture site. Don't tighten the sling too much as the weight of the arm and splint will help to pull on the fracture and correct any residual misalignment.

ASSESSMENT

All patients should be assessed both initially and following any reduction attempts for neurologic and vascular integrity of the extremity. Obtain postreduction radiographs to confirm proper bony positioning. The procedure may be repeated if the radiographs show incomplete reduction.

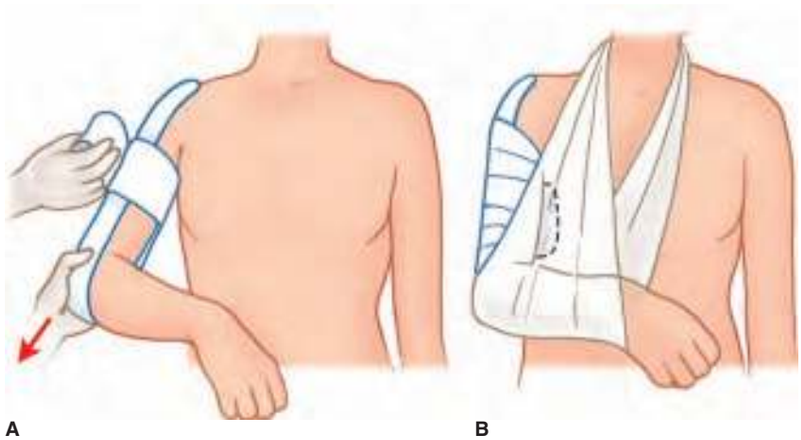


FIGURE 112-15. Immobilization of the reduced surgical neck fracture of the humerus. ***A.*** Application of a sugar tong splint. ***B.*** A sling is applied after padding is placed between the upper arm and the thorax.

Positioning is not critical if the reduction was performed for neurologic or vascular compromise. The primary consideration is the relief of the compromised nerve and/or artery. The Orthopedic Surgeon can later reduce the bony defect that remains.

AFTERCARE

Patients with uncomplicated or nondisplaced fractures should be referred to an Orthopedic Surgeon in 5 to 7 days. **All patients should receive written instructions on the signs and symptoms of the splint being too tight or a potential compartment syndrome.** Patients with unstable fractures should be evaluated within 24 hours. Any patient with an open fracture, evidence of neurologic and/or vascular compromise, or suspicion of a compartment syndrome should be admitted to the hospital after an emergent consultation with an Orthopedic Surgeon.

COMPLICATIONS

Complications of the reduction procedure include primarily neurologic and/or vascular compromise. **Most complications result from the injury that produced the fracture. This underlies the need for a good neurovascular examination prior to any reduction attempt.**

SUMMARY

Proximal humeral fractures are common. The EP can manage most of these conservatively. A displaced surgical neck fracture is the only proximal humeral fracture that should be reduced in the ED. Reduction is often reserved for patients who have evidence of neurologic and/or vascular compromise. Most of these injuries require open reduction and internal fixation in the Operating Room by an Orthopedic Surgeon. Arrange close follow-up for any patient discharged from the ED along with good discharge instructions about any potential complications.

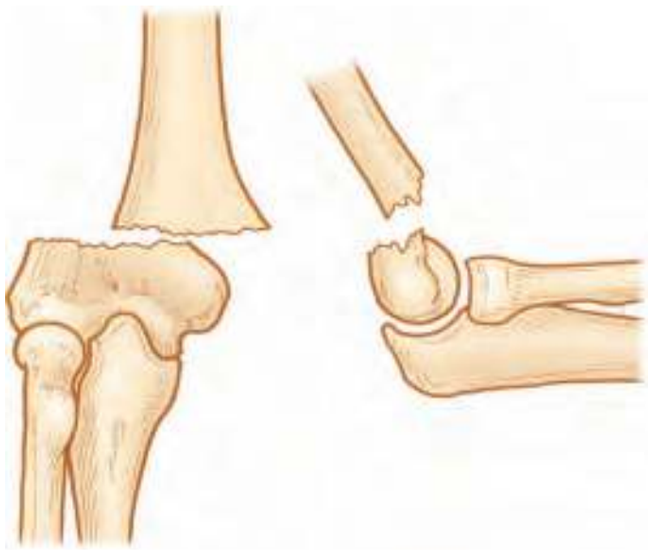
SUPRACONDYLAR FRACTURE OF THE HUMERUS

INTRODUCTION

Distal fractures of the humerus located within 2 cm or just proximal to the epicondyles are known as supracondylar fractures (**Figures 112-16 and 112-17**). Supracondylar fractures are classified as extension or flexion fractures.[34-36] These fractures are common in children under the age of 15 and are rare in people over the age of 20.[34-36] Supracondylar fractures are the most common elbow fractures seen in children.[37] The most common mechanism of injury involves a fall onto an outstretched hand with the elbow locked in extension.[34-36] The same mechanism of injury in an adult often results in a posterior elbow dislocation. The bones of an adult are much stronger than those of a child, so that the ligaments rupture rather than the bone fracturing.[34,35] **These injuries have a significant incidence of associated neurologic and/or vascular injury.[38] There is a significant incidence of a subsequent compartment syndrome.** Refer all supracondylar fractures to an Orthopedic Surgeon for follow-up care.

ANATOMY AND PATHOPHYSIOLOGY

The child will be holding the affected extremity with the elbow flexed 90° and the arm adducted.[34,36] Localized tenderness, swelling, and the occasional deformity will be found upon examination. The patient may have a "pucker sign."[39] Extension fractures have



A B

FIGURE 112-16. Supracondylar fracture of the humerus. ***A.*** Anteroposterior view. ***B.*** Lateral view.

posterior displacement of the distal fragment of the humerus that is exacerbated by the pull of the triceps muscle.[34,36] The posterior displacement of the olecranon may mimic a posterior elbow dislocation. **Anterior angulation of the sharp proximal fragment (Figures 112-16*B* and 112-17) may injure the brachial artery or median nerve (Figure 112-18). A thorough neurovascular examination is essential.** There will be loss of the normal olecranon prominence with flexion injuries. These fractures are frequently open and vascular injury is less frequent.[34,36]

The modified Gartland classification of supracondylar humeral fractures is the most commonly accepted and used system.[40] A Gartland type I supracondylar fracture is nondisplaced or minimally

FIGURE 112-17. Radiograph of a supracondylar fracture. (Used with permission from Shah BR, et al: *Atlas of Pediatric Emergency Medicine*, 2nd ed. New York: McGraw-Hill; 2013. Photo contributors: Binita R. Shah, MD, and John Amodio, MD.)

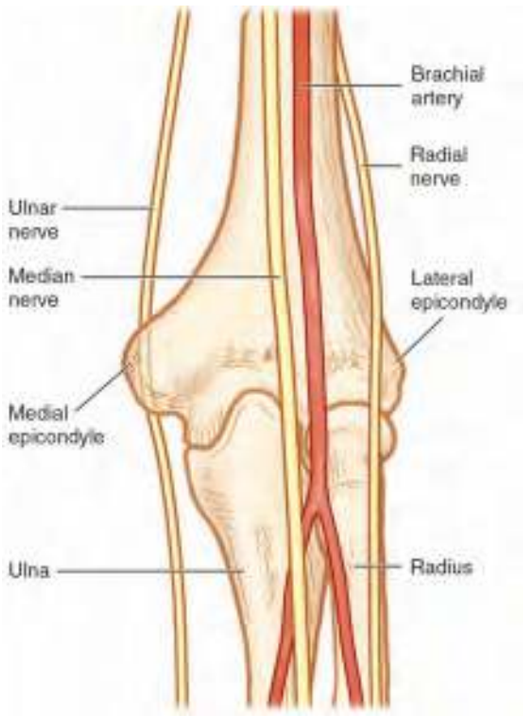


FIGURE 112-18. Major neurologic and vascular structures crossing the elbow.

displaced by < 2 mm and is associated with an intact anterior humeral line. The anterior humeral line is a line drawn on a true lateral radiograph of the elbow down the anterior surface of the humerus. Its distal end should traverse through the middle third of the capitellum. Gartland type I fractures may only require a posterior long arm splint and no reduction.[41-43] Make this decision in conjunction with the consultant. A type II supracondylar fracture is displaced by > 2 mm and the posterior cortex is intact but may be hinged. The anterior humeral line does not go through the middle third of the capitellum. Type III fractures are displaced with no meaningful cortical contact. Gartland type IV fractures are characterized by an incompetent periosteal hinge circumferentially and are defined by instability in flexion and extension.[40] **The EP's role in a supracondylar humeral fracture is to ascertain whether there is any neurologic and/or vascular compromise of the extremity distal to the fracture.**

Radiologic evaluation of supracondylar fractures is best appreciated on the lateral view. One-quarter of these fractures in children are of the greenstick variety.[34,36] The posterior fat pad sign is not visible on a normal elbow and indicates an effusion. Examine the radiograph for the posterior fat pad and anterior humeral line. Initial radiographs may show no evidence of a fracture except for a posterior fat pad sign.[37] Treat a child with localized tenderness in the supracondylar area without any radiologic signs of a fracture conservatively with splinting and referral to an Orthopedic Surgeon.[34,36] The anteroposterior view allows an assessment of any medial to lateral displacement. Displaced fractures should be emergently reduced by an Orthopedic Surgeon.

INDICATIONS

The only indication for reducing a supracondylar fracture of the humerus emergently is if there is any neurologic and/or vascular compromise of the extremity distal to the fracture and an Orthopedic Surgeon is not immediately available.

CONTRAINDICATIONS

Contraindications to the reduction of a supracondylar fracture include other injuries that represent a threat to life or limb. The patient's airway, breathing, and circulation must first be addressed and stabilized. An Orthopedic Surgeon should manage any open or displaced fractures. Unfamiliarity with the reduction technique is a relative contraindication. Simple traction on the extended elbow may be sufficient to restore neurologic and/or vascular integrity if the EP is uncomfortable with the reduction procedure.

EQUIPMENT

- Supplies and equipment for procedural sedation (Chapter 159)
- Compressive cotton bandage (e.g., Webril)
- Elastic wraps
- Casting material (e.g., plaster, fiberglass, or prepackaged splints)
- Prepackaged splinting sheets
- Sling and swath

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Adequate anesthesia for this procedure is best accomplished with procedural sedation (Chapter 159). Obtain an informed consent for this procedure in addition to the reduction procedure.

TECHNIQUE

Place the patient supine. Perform procedural sedation (Chapter 159). Slightly abduct the affected extremity. Place the patient's hand in the midposition between pronation and supination with the thumb pointing upward (**Figure 112-19*A***). Grasp the patient's elbow region with the dominant hand and grasp the wrist with the nondominant hand (**Figure 112-19*A***). Instruct an assistant to stabilize the distal humerus (**Figure 112-19*A***). Apply distal traction in line with the long axis of the arm by pulling on the wrist while simultaneously correcting any medial or lateral displacement at the elbow (**Figure 112-19*A***). Supinate the patient's arm and correct any remaining medial or lateral displacement at the elbow while simultaneously distracting the wrist (**Figure 112-19*B***). Place the thumb of the dominant hand across the joint line of the elbow with the fingers behind the olecranon process and slowly flex the elbow to just beyond 90° while distracting the elbow (**Figure 112-19*C***). Splint the arm in this position. Avoid tight bandaging or splinting as significant swelling may occur up to 24 hours after the reduction. Excessive flexion or extension may compromise the limb's vascularity and increase compartment pressure.[44]

ASSESSMENT

All patients should be assessed before and after any reduction attempts for neurologic and vascular integrity of the extremity. Postprocedural swelling is common. Obtain postreduction radiographs to confirm proper bony positioning. The procedure may be repeated if the radiographs show incomplete reduction.

Positioning is not critical if the reduction was performed for neurologic or vascular compromise. The primary consideration is the relief of the compromised nerve and/or artery. The Orthopedic Surgeon can later reduce the bony defect that remains.

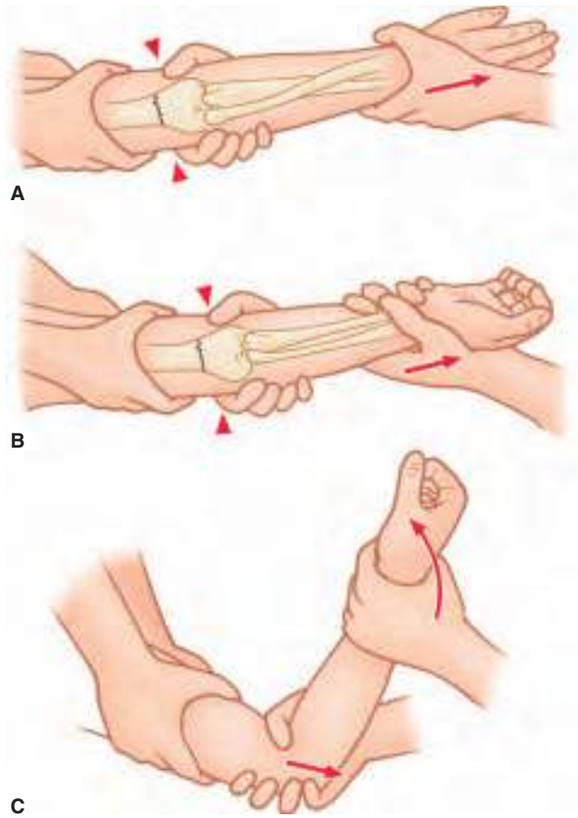


FIGURE 112-19. Reduction of a supracondylar fracture. ***A.*** Positioning of the hands of the EP and the assistant. Distal traction is applied (*arrow*) while reducing the medial or lateral displacement (*arrowheads*). ***B.*** The patient's hand is supinated while maintaining distal traction (*arrow*). Any remaining medial or lateral displacement is corrected (*arrowheads*). ***C.*** The patient's elbow is flexed (*curved arrow*) just beyond 90° while maintaining distal traction (*straight arrow*).

AFTERCARE

Patients with supracondylar fractures should be admitted to the hospital to be monitored for a delayed compartment syndrome, neurologic compromise, or vascular compromise. Any patient with an open fracture, evidence of neurologic or vascular compromise, or suspicion of a compartment syndrome should be admitted to the hospital after an emergent consultation with an Orthopedic Surgeon.

COMPLICATIONS

Most complications result from the injury that produced the fracture and not the reduction.[38] Complications of the reduction procedure include primarily neurologic and vascular compromise. This is most often a neurapraxia and may involve any of the three nerves crossing the fracture. The neurovascular structures crossing the fracture may become lacerated or entrapped during the reduction. **The necessity of an accurate and complete neurovascular examination before and after any reduction attempts cannot be overemphasized.**

SUMMARY

Supracondylar fractures of the humerus are common in children under the age of 15 and rare over the age of 20 years. The most common mechanism of injury involves a fall onto an outstretched hand with the elbow locked in extension. These injuries have a significant incidence of associated neurologic injury, vascular injury, and the subsequent development of a compartment syndrome. The only indication for the reduction of a supracondylar fracture of the humerus by the EP is neurologic and/or vascular compromise distal to the fracture.

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113 Casts and Splints

Eric F. Reichman

INTRODUCTION

External immobilization of the extremities is the oldest form of fracture treatment. References to plaster use and various immobilization techniques are scattered throughout historical records. The use of plaster of Paris, also referred to as plaster, in fracture management dates back to the eighteenth century Turkish Empire. Plaster bandages became commercially available in 1931. Despite the development of plastic (i.e., fiberglass) casting products, the plaster bandage persists as the most economical and versatile material for immobilization techniques.[1]

Immobilization of an injured extremity begins at the scene of the accident. According to Advanced Trauma Life Support guidelines, the injured extremity must be aligned and immobilized after the appropriate management of any life-threatening problems.[2] Prehospital immobilization of fractures is invaluable for pain control, prevention of soft tissue injury, prevention of any new or further injury to neurovascular structures, and management of edema. External immobilization with splinting or casting is often the definitive management of injured extremities in the Emergency Department. Knowledge and expertise in this therapeutic procedure are essential for any Emergency Physician.

Splints are commonly used for the immobilization of upper and lower extremity injuries. **A splint is a hard bandage that is not circumferential and prevents movement of the fracture site.** Splinting may be the definitive management of certain injuries. Splints have the distinct advantage of being quick and easy to apply, and they are designed to accommodate postinjury swelling. The major disadvantages of splints are that they provide slightly less rigid immobilization than casting and require an Orthopedic Physician visit within a few days to be replaced with a cast.

Casts, which are generally circumferential, are better suited for the definitive treatment of fractures and ligamentous injuries. Casts provide superb immobilization and allow for the maintenance of a reduced fracture. The rigidity of a cast limits the amount of swelling and soft tissue edema in the first 24 to 48 hours after the injury and is therefore associated with an increased risk of developing a compartment syndrome. **Casts should be used with caution in the management of acute fractures. They are often split (bivalved) to allow swelling and prevent the development of a compartment syndrome before the patient is discharged from the Emergency Department**.

ANATOMY AND PATHOPHYSIOLOGY

Casts and splints rely on the principle of a three-point mold to maintain fracture reduction (**Figure 113-1**). **When applying a cast or splint, the application of directed force to the underlying bones should be uppermost in one's mind**. To obtain a three-point mold, place one point of contact over the convex side of the fracture site. The other two points of force are aimed in an opposite direction, proximal and distal to the fracture, and from the concave side. This is the classic teaching of Sir John Charnley who noted that "a curved plaster is necessary in order to make a straight limb."[3] A skin-tight cast that closely follows the contours of the extremity will not maintain the fracture in alignment as it does not apply appropriate pressure to the underlying bones.

Casts and splints also rely on hydraulic force to maintain limb length and alignment. One may think of the soft tissues surrounding the broken bones as constituting a flexible cylinder that contains the underlying fracture, hematoma, and edema. Axial loading of the bones will cause the soft tissue to expand and allow the limb to shorten. **A well-applied cast or splint will resist the outward**

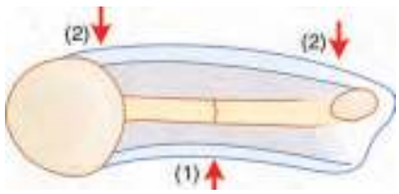


FIGURE 113-1. Three points of force are acting on the injured extremity in a well-applied cast or splint. One force is applied to the convex side of the fracture site (*1*). Two opposing forces are applied at sites proximal and distal to the fracture and on the concave side of the fracture (*2*).

TABLE 113-1 Characteristics of Plaster and Fiberglass

	Plaster	Fiberglass
Allergic reaction	Almost none	Almost none
Clothes	Easily washes off	Resin permanently stains
Cost	Inexpensive	Expensive
Curing	48–72 hours	30 minutes
Moldability	Excellent	Good
Radiolucency	Poor	Good
Skin	Easily washes off	Resin bonds for days
Strength	Good	Excellent
Water resistance	Poor	Excellent
Weight	Heavy	Light

expansion from axial loading and provide additional support for the limb.[4]

Plaster is made of finely ground calcium sulfate that has been dehydrated by heat. The calcium sulfate is impregnated into muslin sheets containing dextrose or starch. The addition of various chemicals (e.g., alum, aluminum, copper, iron, magnesium, salicylic acid, or zinc) to the calcium sulfate alters the rate of hardening after the addition of water. Plaster grades include fast-setting and extra-fast-setting plaster, which are useful for different applications. Long cylindrical crystals form and interlock as the cast sets to give strength to the cast. **For this reason, plaster should not be moved once it begins to set as these crystals may fracture causing the plaster to lose strength. Careful lamination of the plaster while it is still wet will add strength by enabling the formation of longer crystals.**

Crystal formation during the setting of plaster is an exothermic process that is initiated by the addition of water to the plaster. Using warm water will accelerate the chemical reaction, harden or set the plaster sooner, and decrease the setting time. Unfortunately, the use of very warm or hot water will also increase heat production while the plaster sets. The use of very thick splints or fast-setting plaster will also increase the heat produced during setting. Use cold water to activate the plaster, increase the setting time, and decrease the heat produced during setting. **Great care should be taken if warm water is used in applying a cast or splint to an anesthetized patient, unconscious or altered patient, or an insensate limb.**[5,6] **The setting plaster may burn the skin in these cases as the patient will not notice the heat and pain associated with the exothermic reaction**. Rubbing and working with the plaster will also accelerate the setting process.[7]

The same principles described above regarding plaster apply to the use of fiberglass. The fiberglass splinting material consists of cloth impregnated with a plastic, spun glass resin, and a water-activated catalyst. A comparison of plaster and fiberglass can be found in **Table 113-1**.

INDICATIONS

SPLINTS

An injured extremity should be splinted as soon as possible after the injury. Splinting results in the reduction of pain, reduction of edema, relief of pressure on neurovascular structures, and prevention of further soft tissue injury. Any available material can be used to immobilize or realign the affected extremity in the prehospital setting and in the Emergency Department. **A thorough neurologic and vascular examination of the extremity should be performed and documented before temporarily splinting the extremity.**[8] **A thorough neurologic and vascular examination of the extremity should be performed and documented after temporarily splinting the extremity. The initial neurovascular examination, temporary splinting, and post-splinting neurovascular examination should be performed before the patient undergoes radiographic studies**. Immobilize an extremity in the appropriate and definitive splint after diagnosing and stabilizing the fracture. **A thorough neurologic and vascular examination of the extremity should be performed and documented after the placement of the definitive splint.**

An extremity fracture is the most common reason for placement of a splint.[9] A splint is also indicated following the reduction of a dislocated joint. The patient with ligamentous sprains or muscle strains will also receive significant pain relief with splint immobilization. Splints are placed following orthopedic or soft tissue surgery of the extremities.

CASTS

There are few reasons to immobilize an acutely injured extremity with a cast in the Emergency Department. Most injuries can be initially stabilized with a splint. Following reduction of certain fractures, placement of a cast will secure the bones in their proper alignment and allow for a primary union (e.g., distal radius or tibial shaft).[10,11] **Placement of a cast instead of a splint should be performed only if the patient has access to close follow-up or can return to the Emergency Department for a cast check within 24 hours**.

Patients with casts may present to the Emergency Department with various cast-associated problems. The cast may have become wet and lost its strength and integrity. The cast may no longer fit properly if the affected extremity has decreased in size from reduction of swelling. The cast must be removed and the affected area examined if the patient complains of persistent pain.[10,11] All these patients may safely be placed back in a cast. They may also be placed in an appropriate splint and follow-up arranged with an Orthopedic Surgeon for casting.

CONTRAINDICATIONS

SPLINTS

There are no absolute contraindications for the placement of a splint.[9] Relative contraindications include soft tissue injuries or wounds that need regular care and evaluation. In this setting, the wounds should be appropriately dressed, padded, and the splint constructed so that it can easily be removed and replaced. A "window" may also be cut in the splint as it is being applied to facilitate access to the wound. The splint should not place pressure over the wound.

CASTS

Do not cast an extremity that has the potential for significant edema or that may develop a compartment syndrome. An injured extremity with significant edema or soft tissue injuries should not be constrained by a cast. Similarly, infections of joint spaces or soft tissues must remain exposed for frequent evaluations. Any fracture that is not adequately reduced by closed manipulation should not be placed in a cast.[3,11] A cast should not be applied by someone unfamiliar with the technique and unable to manage the associated complications.

EQUIPMENT

- Bucket or basin to hold water
- Source of cool or tepid water
- Cotton cast padding (e.g., Webril), various widths

- Plaster strips or rolls, various widths
- Fiberglass strips or rolls, various widths
- Prefabricated splinting material, various widths
- Bias stockinette, various widths
- Cloth tape, 1 inch wide
- Bias stockinette, optional
- Elastic bandages (e.g., Ace wraps), various widths
- Slings, various sizes

The width of the cotton cast padding, plaster, and bias stockinette required will vary by the site of application. In general, 1 to 3 inch material can be used for the hands and digits, 3 to 4 inch wide material can be used for the upper extremity, and 4 to 5 inch wide material can be used for the lower extremity. Alternative materials include fiberglass strips and rolls instead of plaster strips. Elastic bandages (e.g., Ace wraps) can be substituted for bias stockinette. Prefabricated splinting material is also available, with the padding and fiberglass (or plaster) already assembled and covered with cotton material (e.g., Parker Splints or Orthoglass). **It is important to remember that all of the casts and splints described in this chapter can be constructed using plaster or fiberglass splinting material.**

Fiberglass rolls must be cut to the appropriate length with scissors whereas plaster can easily be torn by hand. The fibers are too strong to be torn by hand. Gloves should always be worn when handling fiberglass because the resin will stick to skin and is exceedingly difficult to remove.[4,7,10] Otherwise, the same principles described for plaster apply to fiberglass.

PATIENT PREPARATION

Whether reducing a fracture or simply manipulating the extremity to place a cast or splint, make sure that the patient has received appropriate analgesia. Conduct a thorough examination of the skin overlying the site of injury. **It is inexcusable to miss the diagnosis of an open fracture. All skin wounds must be inspected and explored**. Exploration of the wound is undertaken cautiously with a sterile probe or gloved finger according to wound size. Fractures may puncture the skin from the "inside out," resulting in an innocuous appearing pinhole in the skin. These are grade I open fractures, and they carry a 5% risk of infection.[12] Cover any open fractures with sterile saline-soaked gauze until formal irrigation and debridement can be undertaken in the Operating Room. Do not examine open wounds repeatedly due to the risk of increased contamination. Administer tetanus prophylaxis as indicated. Administer the appropriate intravenous broad-spectrum antibiotics for an open fracture if present.

Document a thorough neurologic and vascular examination of the injured extremity before and after any splinting or casting.[9] A change in the neurologic or vascular status of an extremity may be the result of the fracture reduction, the splint or cast application, or a compartment syndrome. The fracture may have to be reduced and held in position while a splint or a cast is applied. If no reduction is needed, splint the affected extremity in a position of stability. Techniques for achieving and maintaining fracture reduction are beyond the scope of this chapter.

GENERAL SPLINTING CONSIDERATIONS

The general considerations and techniques common to the application of all splints are discussed in this section. It describes the techniques for splints using cotton cast padding, plaster, and bias stockinette. Many alternative materials are available and may be substituted, such as fiberglass for plaster or elastic wraps for stockinette. Prefabricated splint materials that incorporate padding may also be used. The techniques described in these chapters are applicable for all splinting materials. Where alterations in technique are required, they are so noted.

Splints are constructed of cotton padding overlaid by splinting material (i.e., strips of plaster or fiberglass) and subsequently held in position by an overwrap of bias stockinette or an elastic bandage. Splint padding should be thick enough to provide protection for the skin from the plaster. **The splint or cast will be unable to provide sufficient immobilization of the fracture to maintain a reduction if the padding is too thick**. One to two layers of cotton cast padding are sufficient over the fracture site. Three to four layers of cotton cast padding are required at the proximal and distal extents of the splint to distribute the stresses. Thinner padding can be used when maintenance of fracture reduction is a priority (e.g., with a fracture of the distal radius).

Plaster strips are available in precut slabs measuring either 4 by 15 or 5 by 30 inches. The precut strips have the advantage of speed and ease of application. Alternatively, plaster and fiberglass are available in rolls of various widths ranging from 2 to 8 inches. The rolls of splinting material may be rolled out to the precise length desired and cut appropriately. The rolls of splinting material are useful for splints requiring long strips such as coaptation splints. **The ideal thickness for most upper extremity splints is 10 sheets or layers of plaster or 5 to 6 layers of fiberglass.[13] The use of 15 sheets or layers of plaster or 8 to 10 sheets of fiberglass is preferable for lower extremity splints**. The strength of the splint depends on the number of layers as well as the lamination of the layers during their application.

A piece of cotton cast padding may be used as a template for the length of the required splinting material. Roll a piece of cotton cast padding over the desired location of the splint to determine the length of the splinting material required (**Figure 113-2*A***). Roll out the splinting materials to the appropriate lengths and cut them slightly shorter than the template.

An alternative is to cut the splinting material approximately 1 to 2 inches longer than necessary so that the ends may be folded back on themselves. This will prevent any contact of the sharp splint ends with the patient's skin. Prefabricated splinting material covered with padding has jagged ends and once it has been cut it begins to harden immediately, even prior to the addition of water. These sharp ends will rub against the patient's exposed skin, even through cotton cast padding or bias, and for that reason, they should be folded back. To avoid premature hardening of the prefabricated splinting material, keep it inside of the protective foil and seal it with tape or the manufacturer-provided clips.

Bias stockinette may be applied to the extremity for extra padding and comfort. This may also be omitted depending on Physician preference. Prepare strips of cotton cast padding. **The strips of cotton cast padding should always be longer and wider than the plaster**. This prevents the plaster from touching the skin and causing a pressure sore, abrasion, or burn. Padding is especially important at the proximal and distal edges, as this is where significant pressure originates. Apply additional pieces of padding over pressure points and bony prominences (e.g., the olecranon). **Additional padding may also be created by folding a loose piece of cotton cast padding back on itself several times and placing it over the individual prominences.** Alternatively, cotton cast padding may be applied directly by wrapping the pressure point or bony prominence circumferentially as is done in cast application.

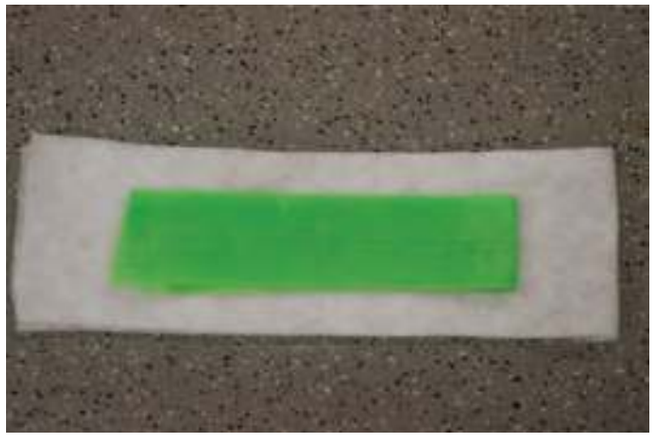
Begin applying the splint once the padding and splinting material have been cut to the appropriate lengths. Be sure that all required materials have been collected before dipping the splinting material

A

B

C

D



E

FIGURE 113-2. Preparing the splint. ***A.*** Cotton cast padding to measure the length of material needed. ***B.*** Plaster is submersed in tepid water with air bubbles arising from it. ***C.*** Fiberglass is submerged in tepid water with air bubbles arising from it. ***D.*** Suspend the lengths of wet splinting material over the bucket and squeeze out the excess water. ***E.*** The splinting material is placed on the cotton cast padding.

in water to activate it. Only a limited amount of time, less than 10 minutes, is available for splint application and molding once the splinting material is wet. Completely immerse all of the splinting material in a bucket of tepid, clean tap water. Keep the splinting material submersed until no more bubbles arise from under the water (**Figures 113-2*B* and 113-2*C***). At this point, the splinting material can absorb no more water. Suspend the splinting material over the bucket and lightly squeeze out the excess water by running your fingers down its length (**Figure 113-2*D***). **It should only take two or three passes of the fingers to remove the excess water. Do not wring the plaster strips like a dish rag, as that will cause loss of plaster into the bucket!** Lay the splinting material on a clean flat surface. Run your hands over the splinting material to laminate the individual strips into one slab. Laminating the strips together adds significant strength. Lay the splinting material onto the cotton cast padding (**Figure 113-2*E***). Fold the edges of the cotton cast padding over the splinting material to cover all the edges completely. Apply the splint to the extremity.

An alternative and more commonly used method that is preferred by many is to apply the cotton cast padding circumferentially over the extremity, overlapping each layer by 50%, and then applying the wet splinting material. In this fashion, the splinting material will "stick" to the cotton cast padding. **Smooth the splinting material with the broad aspect of your hand and not your fingers to help minimize irregular indentations**.

The cotton cast padding should be facing the patient and no splinting material should directly contact the skin regardless of the method used. Secure the splint with a wrap of bias stockinette or an elastic bandage. The wrap must be applied under minimal tension when you are using an elastic wrap to affix the splint. The elastic may cause increasing pressure over time. Gently apply strips of tape to the end of the wrap to secure the bias stockinette or the elastic bandage. **Tape should never be applied circumferentially as this can impede expansion of the splint due to underlying swelling and create a tourniquet effect. Application of the tape under tension before the splint is completely hard will cause indentations in the splinting material and result in pressure points on the underlying skin**.

The splinting material may be molded at this point to achieve greater conformity to the extremity or better reduction of the fracture. **It is paramount to use only the palms and not the fingertips when molding the finished splint. Finger pressure induces deformity in the splint that will result in skin breakdown under those defects. All molding must stop once the splinting material begins to harden**. The plaster is quite fragile and cracks that weaken the splint may be propagated. Apply 1 inch wide tape in a spiral fashion to secure the bias or elastic wrap after the plaster has hardened.

If using prefabricated splinting materials, cut it to length, remove it from the sealed foil, and wet it to activate the splinting materials. Briefly pass the prefabricated splinting material under cool water. Place the wet prefabricated splinting material on a towel and roll it up. This will remove any excess water. **The splinting material should not be soaked or placed in a bucket of water. It requires only one pass under the running water.** The prefabricated splinting material should not be squeezed dry as this tends to deform the splinting material and results in a poorly molded final product.

Apply the splint and mold it to the extremity. **It is paramount to use only the palms and not the fingertips when molding the finished splint. Finger pressure induces deformity in the splint that will result in skin breakdown under those defects.** Any excess length of splinting material should be folded back on itself so that the cut ends do not come in contact with the patient's skin. **The distal part of any extremity should always be left visible so that the Emergency Physician may recheck a neurovascular exam.** The patient will also be able to visualize any changes in color that may occur latter. It is held in place by circumferentially wrapping the splinting material with an elastic bandage. **The elastic bandage should be applied relatively loosely so it will not induce increased compartment pressures.**

GENERAL CASTING CONSIDERATIONS

The general considerations and techniques common to the application of all casts are discussed in this section.[3,7,10,14] **Casting requires careful circumferential turns of material instead of longitudinal layers of material, as for splints.** Casts are constructed of cotton cast padding overlaid with either fiberglass or plaster bandages (i.e., casting materials). The four areas that require particular attention and are discussed in this section are the application of padding, padding pressure points, application of casting material, and molding the cast.

Begin by organizing the required supplies. Cast application requires the same material as that used in splinting (i.e., water, cotton cast padding, stockinette, and casting material). The width of the padding and casting material depends on the size of the extremity. The 4 to 6 inch wide rolls of casting material are generally used for lower extremity casts, whereas 3 or 4 inch wide rolls are generally used for the upper extremity. **Use the widest casting material available and possible in order to limit the number of turns of the fiberglass or plaster roll over joints and other curved surfaces**. Place all materials on a tray near the bedside including a bucket of tepid water. An assistant designated to dip and drain the casting material and help with patient positioning is immensely helpful.

Prepare the patient. Position the extremity and the patient appropriately for cast placement. The patient is frequently able to assist in the process. Cover the patient with gowns or towels to keep casting material off their clothes.

Apply tubular stockinette to the extremity (**Figures 113-3*A* and 113-3*B***). The stockinette is not a necessary component of casting but many Emergency Physicians use it as a first layer.[7] It provides a smooth covering over the skin that wraps neatly over both ends of the cast. Roll up the stockinette. Place it over the distal extremity as if you were putting on a sock (**Figure 113-3*A***). Unroll the stockinette up the extremity (**Figure 113-3*B***). Care must be taken to apply the stockinette gently. **Do not create tension in the stockinette by pulling it tightly. Eliminate any creases or redundancy of material by trimming any overlapping folds with a scissors**. For upper extremity injuries, cut a small hole one-third of the way from the end of one side of the stockinette to allow the thumb to pass through.

The primary layer of padding is provided by the cotton cast padding. **Casts and cast padding should be applied from distal to proximal**. Begin wrapping the cotton cast padding at a point that will be distal to the start of the plaster (**Figure 113-3*C***). This initial band of padding is essential for protection against the cast edge. Keep the roll of padding in contact with the skin so that the material conforms easily to the contours of the extremity as it unrolls. Unroll the padding in a circumferential manner around the extremity. **The cotton cast padding must be laid down neatly and cleanly with no kinks or creases**. Each turn should overlap one-third to one-half of the previous turn. Tear off the extra cotton cast padding to eliminate excess material as you turn angles (e.g., ankle, heel, elbow, or thumb). **Lay the torn edges down by rubbing the padding smoothly**. Continue applying the padding, ensuring that it extends beyond the proximal end of where the cast edge will be to ensure skin protection at the cast edge. Excess length can easily be torn away after the cast is applied and hardened. Typically, two layers of cotton cast padding are adequate for protection between the skin and the casting material.

A

B

C

D

FIGURE 113-3. Preparing to place a cast. ***A.*** Apply an initial layer of tubular stockinette. ***B.*** The stockinette has been unrolled over the extremity. ***C.*** Begin and end the layering of the cotton cast padding at a site distal and proximal to where the casting material will end. Unroll the cotton cast padding in a circumferential manner, covering each preceding layer by one-third to one-half of its width. ***D.*** The cotton cast padding tears easily to provide additional layers of padding over bony prominences.

Pressure points occur over bony prominences or where excess padding has created an unnatural prominence. Palpate the obvious bony prominences to get a sense of whether or not there is adequate padding after the application of the two layers of padding. If the area feels vulnerable, place torn off pieces of cotton cast padding onto the exposed areas (**Figure 113-3*D***). **Do not overpad bony prominences as excess layering can also lead to excess pressure**. Two to three layers of padding are adequate for most pressure points and bony prominences. Rub the torn edges of the padding so that they fuse smoothly to the underlying padding.

Place the rolls of casting material in a bucket of tepid water so that they are standing on end. Let the casting material remain submersed as long as air bubbles rise out from the center of the roll (**Figures 113-2*B* and 113-2*C***). Remove the rolls of casting material when all bubbles stop rising. Hold the casting material in both hands and squeeze some of the water out (**Figure 113-4**). **Do not wring the roll. Do not eliminate all the water from the roll**. Squeeze it gently. The remaining water in the roll is necessary for smoothing and molding the casting material into one solid unit. In general, casts should be applied with "wetter" material and splints with "drier" material as less time is required to apply a splint.

Apply the casting material (**Figure 113-5**). Place the roll of casting material on the extremity (**Figure 113-5*A***). Unroll the casting material in a circumferential fashion around the extremity. **Never lift the roll of casting material off the extremity!** Continue each consecutive wrap around the extremity by overlapping the casting

FIGURE 113-4. Hold the casting material roll in both hands and gently twist each end to squeeze out the excess water. Keeping the free end of the casting material folded over will facilitate access after it has been removed from the water.

material by approximately 50%. The free border of the casting material will have excess material in it as the extremity changes in size. Grasp this excess casting material with the thumb and index finger of the nondominant hand (**Figure 113-5*B***). Pull it outward to create a tuck or a fold. Wrap this fold around the extremity (**Figure 113-5*C***) and smooth the fold down against the extremity. This fold will barely be noticeable in the final product. As one roll of casting material ends, another should begin with a small amount of end-to-end overlap. Do not make consecutive folds at the same site as this will create bumps and add bulk to the cast.

Continuously mold and smooth the casting material with wet hands as each layer is applied. **This action ensures continuity of casting material throughout the cast and forms a smooth cast that conforms to the contours of the extremity. Use only the palmar surface of the hands and proximal digits to mold and smooth the casting material (Figure 113-5*D*). Excessive use of the fingertips will produce irregular indentations and pressure points**. Molding around irregular bony areas is best accomplished with two hands simultaneously rubbing the plaster. **Do not allow excessive time to pass between applying each layer of the casting material, as lamination between layers may not occur. This will weaken the cast considerably**.

A cast thickness of 1/4 inch is felt to be adequate. This usually requires four to five layers of plaster or three to four layers of fiberglass. Allow the cast to set and dry with no further manipulations by the Physician or the patient. The time for drying is variable depending on the casting material used, the water temperature, and the thickness of the cast. Typically, let the casting material set

A

B

C

D

FIGURE 113-5. Apply the casting material to form the cast. ***A.*** Lay the roll of casting material on the extremity and unroll it. ***B.*** As the limb changes in girth there will be excess plaster. Use the nondominant hand to pull on the excess material. ***C.*** Fold the excess material back onto the extremity in a neat tuck. Each tuck should be laid down smoothly with a molding of the hand. ***D.*** The casting material is laminated smooth with a continuous motion of the palmar surface of the hand and the proximal fingers.

FIGURE 113-6. Fold the free ends of the cotton cast padding and the stockinette over the edges of the casting material to finish the cast.

over a period of 10 to 15 minutes. Fold the free ends of the cotton cast padding and the stockinette over the edges of the cast as it sets (**Figure 113-6**). This prevents the rough edges of the casting material from irritating and abrading the skin. Secure the edges of the cotton cast padding neatly with tape or thin strips of casting material.

It is a common practice to bivalve the cast with a cast saw if the potential for increased swelling of the extremity is a concern (**Figure 113-7**). Cut completely through the length of the cast in two spots 180° apart (i.e., medial and lateral or anterior and posterior). This simple maneuver provides some room for edema without compromising the integrity of the reduction or the strength of the cast. **The underlying cast padding must also be split. Splitting the plaster alone will not reduce the pressure sufficiently**.

In the future, three-dimensional (3D) printing may affect how casts are made.[15,16] Recently developed is the Cortex cast. It uses nylon to make a rigid and fishnet-like stocking after an extremity scan. It is thinner and lighter than the traditional cast. The Cortex has other advantages including the following: dressing with clothes over it is easier, it uses less material to make, no waste is produced when making the cast, it is fully ventilated, showering can be done without covering the cast, the underlying skin can be visualized, there is no padding to smell with time, and it can be recycled when taken off.

FIGURE 113-7. Splitting of the cast can be achieved with a cast saw that cuts through the thickness of the plaster. Cut the underlying protective material with a scissors.

UPPER EXTREMITY CASTS AND SPLINTS

COAPTATION SPLINT

A coaptation ("to bring together") splint is used primarily in the acute setting for humeral shaft fractures that are nondisplaced or minimally displaced. This splinting technique allows for motion of the hand and wrist while limiting shoulder and elbow mobility. The cotton cast padding should extend from the nape of the neck to the axilla to avoid skin breakdown. The patient should then be placed in a sitting position to minimize splint displacement during the actual application process. The length of the splint extends from the axilla, around the 90° flexed elbow, along the outer arm, over the deltoid muscle, and over the acromion process (**Figure 113-8**). **It is critical that the splinting material extend over the deltoid muscle and the acromion process**. The shoulder portion can be held down by applying 3 inch wide tape over the portion of the splint that covers the acromion. Secure the splint with an elastic bandage that covers the entire splint. The major pitfall is making the splint too short and having it fall off! A splint that is too long will not provide proper immobilization. Always leave plenty of length over the shoulder. Padding is required to minimize axillary irritation. The disadvantages of this splint include the possibility of fracture displacement and extremity shortening. The splint should be replaced with a functional brace or cast after a short period of immobilization for pain control.[4]

FIGURE 113-8. The coaptation splint. The overwrap has been omitted for easier visualization of the splint.

FIGURE 113-9. The sugar tong splint. Leave the metacarpal joints free for flexion and extension. The overwrap has been omitted for easier visualization of the splint.

FIGURE 113-10. The posterior long arm splint. The forearm and wrist are in a neutral position. The padding and overwrap have been omitted for easier visualization of the splint.

SUGAR TONG SPLINT

Sugar tong splints may be used for mid-forearm fractures, distal forearm fractures, and some wrist fractures. They are most commonly recommended for minimally displaced and distal ulnar and radial fractures (e.g., Colles and Smith). This splint immobilizes the elbow and wrist joints to prevent supination and pronation of the forearm (**Figure 113-9**). The splint begins at the palm, just proximal to the metacarpophalangeal (MCP) joints. It has the distinct advantage of allowing the MCPs to remain free, preventing stiffness in those joints.

Measure the required length of splinting material along the volar surface of the hand (starting just proximal to the MCP joints) and forearm, around the elbow, and back on the dorsal surface of the forearm ending just proximal to the MCP joints. **The MCP joints should be left completely free to prevent stiffness**. Early mobilization of the fingers will help to reduce swelling. Apply cotton cast padding from the MCP joints to just proximal to the elbow. The ulnar styloid process and the olecranon process are two bony prominences that need extra padding for comfort and prevention of pressure sores. The free ends of the splint also need added protection to minimize hand discomfort. Apply the splint. **Mold the splinting material with great caution to prevent closure of the sides of the splint thus forming a closed cast**. Fold and/or cut the volar and dorsal splinting material to ensure that the finger MCP joints are freely mobile (**Figure 113-9**). The splint can be modified to avoid potential skin breakdown at the posterior elbow.[17]

POSTERIOR LONG ARM SPLINT

Distal humeral fractures and proximal forearm fractures can be immobilized in a posterior long arm splint (**Figure 113-10**). It is also useful for fractures of the radial head and neck, olecranon fractures, and severe ligamentous injuries to the elbow. The posterior long arm splint extends from the axillary crease area, behind the elbow, distally to incorporate the wrist joint, and ending at the MCP shafts (**Figure 113-10**). This splint immobilizes the elbow in a range of 45° to 90° with the forearm in supination, pronation, or neutral positioning depending on the type of injury. The wrist can also be in a flexed, extended, or neutral position. **The metacarpals should not be immobilized in this splint unless the distal forearm or wrist fracture is comminuted**. Despite the many possibilities, the posterior long arm splint is usually applied with the elbow flexed 90°, the forearm neutral, and the wrist neutral (**Figure 113-10**).

RADIAL GUTTER SPLINT

The radial gutter splint is used for the treatment of stable phalangeal and metacarpal fractures of the index or middle fingers. The splinting material extends from the pulp of the distal fingers to the proximal forearm (**Figure 113-11**). It is helpful to measure and cut a hole in the middle of the splinting material to allow for the insertion of the thumb prior to wetting the splinting material. Place cotton cast padding between the index and middle fingers prior to applying the splint to prevent skin maceration. Mold the width of the splinting material around the radial aspect of the index finger, middle finger, hand and forearm to create a stabilizing force. The ulnar aspect of the hand and forearm is left entirely free. The hand is immobilized in what is considered a "safe" position with the wrist dorsiflexed 20°, the MCP joints flexed 60° to 90°, and the interphalangeal (IP) joints extended or slightly flexed at 10° (**Figure 113-11**).[18] This is also known as the "position of comfort." It can be described as if the patient were holding a can of soda. The fingertips should be visible to allow for repeat neurovascular examinations.

ULNAR GUTTER SPLINT

This splint is used for the treatment of stable metacarpal and phalangeal fractures of the ring and small fingers (**Figure 113-12**). The long axis of the plaster extends from the pulp of the distal fingers to the proximal forearm. Place cotton cast padding between the

FIGURE 113-11. The radial gutter splint with the hand in the "safe" position.[9]

A B

FIGURE 113-12. The ulnar gutter splint. ***A***. Padding is necessary between any fingers that are immobilized together. ***B***. The final product with the hand in the "safe" position.[9]

ring and little fingers to prevent any maceration (**Figure 113-12*A***). The width of the plaster must wrap around the ulnar aspect of the hand and forearm. Mold the splint around the forearm, hand, fourth finger, and fifth finger. The radial side of the hand is left entirely free. The hand is immobilized in the "safe" position with the wrist dorsiflexed 20°, the MCP joints flexed 60° to 70°, and the IP joints extended or slightly flexed at 10° (**Figure 113-12*B***).[18] The fingertips should remain visible to allow for repeat neurovascular examinations.

VOLAR SPLINT

The volar splint can be used for the treatment of wrist fractures, radial styloid fractures, ulnar styloid fractures, metacarpal fractures, middle phalangeal fractures, and proximal phalangeal fractures. This splint remains only on the volar surface of the hand and forearm as the name suggests. The splint begins at the proximal forearm and ends just proximal to the MCP joints (**Figure 113-13**). The splint can be modified and extended to include the fingers for phalangeal fractures with the wrist dorsiflexed 20°, the MCP joints flexed 60° to 90°, and the IP joints extended or slightly flexed at 10°.

DORSAL ("CLAM DIGGER") SPLINT

The dorsal splint can be used in place of a volar splint for injuries of the distal forearm, wrist, or hand. The splint runs along the dorsal surface of the forearm and hand, from the proximal forearm to the ends of the digits. As with the volar splint, the wrist is extended 15° to 20°, the MCP joints are flexed 60° to 90°, and the IP joints are extended or slightly flexed to a maximum of 10°.

The dorsal splint maintains better control of the MCP and IP joints, assuring that the hand remains in the "safe position" when compared to the volar splint. Pad the splint adequately to prevent pressure sores since the dorsal surface of the hand lacks the intrinsic fat pads of the palm.

THUMB SPICA SPLINT

Scaphoid fractures, navicular fractures, carpometacarpal subluxations and dislocations of the thumb, and collateral ligament injuries of the thumb can all be immobilized in a thumb spica splint (**Figure 113-14**).[19] This splint extends to the proximal forearm. It can be extended proximally to include the elbow joint if required. It is positioned on the forearm, like a radial gutter splint, but only the thumb is immobilized. There are several methods to forming a thumb spica splint. One can simply lay the plaster over the radial aspect of the forearm and thumb. It is useful to cut one side of the

FIGURE 113-13. The volar splint. The overwrap has been omitted for easier visualization of the splint.

A

B

C

FIGURE 113-14. The thumb spica splint. ***A***. One technique of thumb spica application with the splinting material cut to conform to the thumb. ***B***. Cutting the splinting material facilitates this different technique of thumb spica application. ***C***. The final product with the wrist dorsiflexed 20° and the thumb positioned as if a glass were being held in the hand.

splint into a shape that conforms to the thumb to facilitate the placement of the splint material around the thumb (**Figure 113-14*A***). Cutting a wedge out of one side of the plaster will allow for easier splinting of the thumb without excess material collecting in the first web space (**Figure 113-14*B***). Position the thumb as if a glass were being held in the hand with the wrist in 20° of dorsiflexion (**Figure 113-14*C***).

FINGER SPLINTS

Immobilization of the finger lends itself to great creativity in the field of splinting.[20] Finger splints may be adequate for immobilization of stable finger fractures, reduced dislocated joints, or ligamentous strains. Splint the finger in full extension if it involves an extra-articular fracture of the distal phalanx. Splint the finger in slight flexion if it involves the strain of a joint or ligament. The finger can be splinted in isolation or it can be immobilized with the adjacent finger for additional stability. Applying a single-digit or two-digit splint allows neighboring joints to remain mobile. Splinting material is rarely used for finger splints in the modern hospital setting. The creation of foam-padded metal or plastic splints has facilitated immobilization of the affected digit. Nonetheless, small strips of cut splinting material can still be used to stabilize any finger injuries. **The juxtaposition of the affected finger with its neighboring finger requires padding between the digits to prevent skin maceration and breakdown**.

SHORT ARM CAST

The short arm cast is used for stable fractures of the metacarpals, the carpal bones, the distal radius, and the radial or ulnar styloid processes (**Figure 113-15**). The short arm cast begins at the proximal forearm and extends to include the palm and the dorsum of the hand. The metacarpophalangeal and elbow joints are left exposed to allow for full motion at these joints. Flex the patient's elbow 90° (**Figure 113-15*A***). Place the wrist in the desired position. The extent of flexion and ulnar-radial deviation of the wrist is determined by the underlying injury. The forearm can be in a neutral, pronated, or supinated position. Apply bias stockinette (**Figure 113-15*B***). Apply cotton cast padding (**Figure 113-15*C***). Ensure that extra padding is applied to the bony prominences of the base of the thumb and the ulnar styloid.

Apply the casting material. Roll the casting material over the padding from the hand to the forearm. A quick trim of the casting material will allow for a better fit as it passes around the thumb (**Figure 113-15*D***). **Provide adequate space for the thumb so that its motion is not limited**. The application of additional layers to the anteromedial surface will strengthen the cast (**Figure 113-15*E***). Mold the cast with an anterior–posterior force applied to the forearm and not up and down strokes (**Figure 113-15*F***). Use smooth, rapid, and repetitive motions to mold and laminate the casting material. Trim the casting material while it is still wet to even out the thumb opening and the cast ends (**Figure 113-15*G***). Ensure that all protective padding is pulled out from under the casting material to protect the skin from the sharp edges of the splinting material (**Figure 113-15*G***). The patient should be able to touch the tips of the thumb and index fingers when the cast is properly applied (**Figure 113-15*H***).

LONG ARM CAST

A short arm cast can easily be extended into a long arm cast if needed. Simply extend the cast proximally with the elbow in 90° of flexion. Extend the padding and the casting material to the proximal humerus, ending two or three finger breadths distal to the axilla (**Figure 113-16**). Be careful to provide adequate padding around the axilla or the patient will complain about the sharp cast edge.

A

B

C

D

FIGURE 113-15. The short arm cast. ***A.*** Flex the elbow 90°. The patient can help position the wrist and fingers in a position of function. ***B.*** Tubular stockinette is applied to the entire arm in anticipation of a long arm cast. ***C.*** Cotton cast padding is applied to the forearm. Adequate padding is also needed at the thumb as it will remain exposed and mobile. ***D.*** Cut one side of the casting material as it is wrapped around the thumb. Less bunching of excess material occurs with quick cuts of the casting material. ***E.*** An additional length of casting material (four to five layers) can be applied along the ulnar length of the cast. This serves as reinforcement if additional strength is necessary. ***F.*** Mold the cast with the palmar aspect of the hands. ***G.*** The wet casting material and underlying padding are cut and folded back to fully expose the thumb. ***H.*** The "okay" sign of a properly exposed thumb.

E

F

G

H

FIGURE 113-15. (*Continued*)

FIGURE 113-16. The short arm cast is extended into a long arm cast. The axilla needs adequate padding for protection of its sensitive skin.

LOWER EXTREMITY SPLINTS AND CASTS

SHORT LEG SPLINT (ANKLE SPLINT)

The short leg splint helps to immobilize isolated ankle injuries with the joint at a 90° angle (**Figure 113-17**). This splint is commonly used for ankle fractures and sprains. It can also be helpful for certain stable fractures of the foot. This splint can be applied as a posterior splint or a lateral to medial stirrup splint.[21] Combining the two techniques can provide additional support for the ankle and is known as a trilaminar splint. The first part (i.e., the posterior splint) provides posterior support to the foot and ankle. The second part creates a medial-to-lateral stirrup-like splint around the sides of the ankle for additional stability.

Place the patient prone, if there are no contraindications, with their knee flexed 90° and the foot pointing upward (**Figure 113-17*A***). Apply the stockinette (**Figure 113-7*A***) followed by cotton padding (**Figure 113-7*B***). Place the posterior support of splint material from the proximal posterior calf, passing under the heel, and along the plantar surface of the foot (**Figure 113-17*B***). It can be extended distal to the toes to provide protection. Place the foot 90° to the tibia and in neutral rotation.

For a "stirrup" support of the ankle, begin by applying splinting material to the length of the medial aspect of the proximal calf, over the medial malleolus, under the heel, and up the lateral aspect of the ankle and calf (**Figure 113-17*C***). The stirrup portion should be long enough to go from mid-tibia to mid-tibia when wrapped under the affected foot. Fold and smooth the edges of the stirrup around the heel (**Figure 113-17*D***). Keep the ankle flexed 90° with the foot neutral while the splint material sets (**Figure 113-17*E***).

LONG LEG SPLINT

This splint is commonly used for knee and tibial injuries prior to and after surgical fixation. It is basically a longer extension of the short leg splint described above. Posterior, medial, and lateral

A

B

FIGURE 113-17. The short leg or ankle splint. ***A***. Stockinette is applied, and the leg is positioned. ***B***. The splinting material is applied posteriorly. ***C***. A stirrup splint is applied around the medial to lateral ankle. ***D***. Fold the corners and smooth out the splint. ***E***. An elastic wrap is applied over the splint after folding back the stockinette. The foot is maintained in this position until the plaster sets.

C

D

E

FIGURE 113-17. (*Continued*)

lengths of splinting material are used to stabilize the leg while the anterior aspect of the leg is left exposed. Measure the extremity from the gluteal crease distally to the tips of the toes. It is important to add an additional 1 to 2 inches to the measured length so that the splinting material can be folded back on itself to protect the patient from the sharp ends. The application of medial and lateral splinting material begins at the upper thigh just below the level of the gluteal crease and travels down the knee, calf, and under the ankle. The posterior portion of splinting material begins at this same level and travels down the posterior aspect of the leg, behind the knee, curving around the heel, and ending just beyond the ends of the toes. Flex the foot 90° to the tibia, flex the knee 10° to 30°, and mold the cast. Failure to keep the ankle at 90° will allow the Achilles tendon to shorten and stiffen. A pillow may be placed under the knee to maintain 20° to 30° of flexion while the splint hardens.

SPLINT FOR ACHILLES TENDON RUPTURE

Rupture of the Achilles tendon can be managed surgically or conservatively with immobilization. Surgery is often delayed and the patient will require immobilization in the "equines" position. Position the patient as if placing a short leg splint or have the patient sit up with the affected extremity hanging over the table's edge. Position the foot in 20° to 30° of plantarflexion (**Figure 113-18**). This is a position that the foot will naturally relax into. Place a posterior splint on the lower extremity extending from the proximal calf to the distal aspect of the toes. Cut out wedges of plaster in order to minimize buckling at the malleoli as the splint material wraps around the heel.

FIGURE 113-18. Positioning and splinting of the patient with an Achilles tendon injury.

FIGURE 113-19. The short leg cast leaves the knee and tibial tuberosity exposed. The finger is pointing to the tibial tuberosity.

SHORT LEG CAST

The short leg cast is used for stable ankle fractures and stable fractures of the hindfoot, midfoot, and forefoot (**Figure 113-19**). Place the patient either supine or sitting on the edge of the gurney. Place a padded block under the distal thigh if the patient is supine. Instruct an assistant to hold the patient's toes to help keep the lower extremity in a good position. Holding the leg by the toes may allow the force of gravity to disrupt fracture alignment. Care must be taken to prevent this outcome. The patient can sit upright on the edge of the gurney with the affected leg hanging down freely if no assistant is available.

The short leg cast begins at the proximal calf, below the knee, and extends down to the toes (**Figure 113-19**). The knee and tibial tuberosity are left entirely free to allow for full flexion and extension. The toes can be left entirely exposed or the cast can provide a hard sole of support and protection beneath the toes. Apply cotton cast padding with extra attention to the areas of bony prominence such as the fibular head, the lateral malleolus, and the medial malleolus. Apply the casting material from the calf to the toes as if applying a short arm cast. Mold the casting material around the Achilles tendon, away from the malleoli, and to conform to the plantar arch. Cut the wet casting material under the metatarsal heads to expose the toes or leave it long to support the entire toes.

Converting a short leg non–weight-bearing cast to a walking cast requires small adjustments to the existing cast. Supplement the arched foot of the short leg cast with additional casting material to form a flat surface. **Apply a preformed heel after the cast has completely dried to prevent any indentation**. Place the walking heel in the midsagittal plane of the foot with the center aspect lining up with the anterior calf. Secure the heel in place with copious casting material wrapped around the foot and ankle (**Figure 113-20**).

LONG LEG CAST

The long leg cast can be used for immobilization of distal femoral fractures, proximal tibial fractures, or knee injuries (**Figure 113-21**). The cast extends from the metatarsal heads to several finger breadths below the groin. The leg is immobilized with the knee in slight flexion and the foot 90° to the tibia with no inward or external rotation (**Figure 113-21**). Extending the short leg cast up to the groin is a safe

FIGURE 113-20. Converting the short leg cast into a walking cast. A walking heel is secured with additional casting material overwrappings.

and stepwise technique of forming a long leg cast. Be sure to provide sufficient overlap of casting material at the junction between the two casts. Inadequate overlap will weaken the integrity of the cast. Support the knee in slight flexion while the casting material sets. Make sure that there is adequate padding around the proximal free edge of the cast to protect the patient's groin.

KNEE IMMOBILIZERS

Knee immobilizers can be made using splinting material and are indicated for ligamentous injuries. Measure the extremity starting 10 inches above the patella to 10 inches below the patella. Double that length and cut the splinting material. Fold the splinting material end to end. Make a cut in the folded side extending from one edge to the middle of the folded side to create a hinge. Apply cotton cast padding starting 10 inches above the patella to 10 inches below the patella. Place the hinged portion of the splinting material 10 inches below the anterior patella with the cut ends extending proximally up both the medial and lateral leg. Secure the splinting material with a loose fitting elastic bandage.

AFTERCARE

The most feared complication of a splint or cast application is the development of a compartment syndrome. The aftercare is geared toward edema reduction and patient education. Instruct the patient regarding the early signs of a compartment syndrome. This includes increased pain, pain with passive motion, paresthesias, pallor, decreased or altered sensation, as well as delayed capillary refill. The patient should return to the Emergency Department immediately if they develop any of these symptoms, if the digits become cold or blue, or if the patient has other concerns. The extremity should be maintained above the level of the heart for the first 48 to 72 hours after the injury. Ice should be applied to the surface of the cast or splint for at least 15 minutes three times a day. The cold therapy will be transmitted through the cast or splint and result in significant reduction of edema. Active motion of the fingers and toes should be encouraged to help reduce edema in the extremity. **The cast or splint must be kept completely dry**. Should bathing be desired, instruct the patient to place two plastic bags over the extremity and tape the proximal edge to the skin of the extremity. Sufficient pain medication should be supplied to last the patient until their follow-up visit with an Orthopedic Surgeon. This should include nonsteroidal anti-inflammatory drugs supplemented with narcotic analgesics. A sling may facilitate mobilization for some upper extremity injuries.

FIGURE 113-21. The long leg cast.

COMPLICATIONS

The most common complications associated with the application of a cast or splint include plaster sores, compartment syndrome, joint stiffness, thermal injury, infection, and allergic reactions.[4-6,10,14,22] There are also problems with the process of application as described throughout the chapter.[23] The following section focuses on the prevention of these complications.

PLASTER SORES

Plaster sores result from ischemic necrosis of the skin underneath a cast or splint. The skin begins to exhibit necrosis after only 2 hours of continuous pressure. **Great care should be taken in applying a cast or splint and only molding it with the broad surfaces of the hands. Molding with the fingers can result in indentations and localized areas of pressure. The cast or splint should never be allowed to rest on a hard or pointed surface until it is completely dry**. Points of contact on the hard surfaces may cause impressions that result in increased pressure. Extra padding over bony prominences may decrease the incidence of plaster sores.

Complaints of pain should be taken very seriously. The cast or splint should be split or removed immediately and the skin examined. If the pressure point is not addressed rapidly, the pain will often subside as the skin becomes necrotic. This oversight often results in a foul-smelling pressure sore under the cast or splint when the patient returns for follow-up. Cast and splint treatment may be rife with complications for patients with limited sensation from underlying medical conditions (i.e., diabetes, paraplegia, or a myelomeningocele). **Great care should be taken and extra padding used when casting or splinting these individuals**.

Common areas of pressure necrosis also include the proximal and distal ends of the cast or splint. These are areas of stress concentration. **Great care should be taken in padding the ends of the cast or splint during the application. No plaster or fiberglass should ever touch the skin directly. If the edges of the splint are sharp or too long, they should be folded out and away from the patient**.

COMPARTMENT SYNDROME

A compartment syndrome is a significant complication from the application of a cast or, less commonly, a splint. The rigid immobilization prevents soft tissue expansion from edema and decreases the amount of fluid needed to raise compartment pressures.[24]

In cases of acute fractures, casts should be used with caution and always split in the direction perpendicular to the force needed to maintain the reduction**. For example, after casting a distal radius fracture where a dorsal mold is needed to maintain the reduction, split the cast longitudinally on the volar and dorsal surfaces (i.e., bivalved) to allow for mediolateral spread of the plaster. Splinting greatly reduces the chance of iatrogenic-induced compartment syndromes because, unlike casting, splints do not harden circumferentially.**

It is not sufficient to split only the plaster. The plaster and underlying cotton cast padding must be split to visualize the skin underneath. Making a single longitudinal cut (i.e., univalving) in the cast can also decrease the compartment pressure. Univalving the cast can decrease intracompartmental pressures by 30%.[25] Spreading the cast 1 cm after cutting it can lower the pressure 60%.[25] Splitting the cotton cast padding will decrease the pressure by 70%.[25]

JOINT STIFFNESS

Joint stiffness is a significant complication of joint immobilization with casts and splints. Sometimes the immobilization is unavoidable, as with the incorporation of the ankle and the knee in a long leg cast. **Every effort should be made to decrease adjacent joint immobilization as soon as it is safe and practical**. An example would be converting a long leg cast into a short leg cast.

Immobilize the extremity in a position of function as long as this does not interfere with the maintenance of fracture reduction. For example, take great care not to immobilize the ankle in plantarflexion when applying a lower extremity splint or cast. This mistake is commonly seen when a long leg cast is placed. This pitfall may be avoided by the stepwise application of the cast. First apply the cast to the foot and ankle with the ankle held 90° to the tibia. Second, extend the cast proximally to become a short leg cast and mold the reduction. Finally, extend the cast up the thigh as needed. An additional benefit is the reduction of anterior compartment pressures of the leg when the foot is held in up to 37° of dorsiflexion.

Great care should also be taken in splinting the upper extremity. **Every effort should be made to leave the fingers mobile at the metacarpophalangeal joints**. Immobilization of the metacarpophalangeal joints in extension results in shortening of the collateral ligaments and limitation of flexion. **Immobilize the metacarpophalangeal joints in 90° of flexion if they must be immobilized**. This position keeps the collateral ligaments in a lengthened position and allows a rapid return to function.

THERMAL INJURY

Thermal injury may result from the exothermic reaction of plaster or fiberglass as it sets or dries. The heat generated during the setting increases as the number of layers (i.e., thickness) increases as well as the temperature of the water increases. Also important is the ability to dissipate the heat generated by the drying plaster or fiberglass. Placing a cast or splint on a plastic pillow as it dries will result in reflection of the heat and an increased temperature within the cast or splint.[6] The use of cloth pillows or towels under the cast or splint allows for some dissipation of the heat. Optimal heat dissipation occurs by exposing the cast or splint to circulating air.

Sufficient cotton cast padding must be used to protect the skin. Plaster and fiberglass must never touch the skin directly. The incidence of thermal injury can be decreased by using cool water and as thin a layer of plaster or fiberglass as possible to accomplish stable immobilization of the extremity. Great care should be used in the application of a cast or splint to anesthetized patients, insensate limbs, or confused patients.

INFECTION

Infection secondary to a cast or splint is uncommon and is usually related to open wounds or exposed surgical pins underneath the plaster. Fresh water should be used to wet the plaster. Do not use standing or previously used water, as it is an excellent culture medium. All wounds should be dressed with sterile gauze and cotton cast padding prior to applying the cast or splint. Windows can be created over wound sites to allow for regular care and evaluation. Patients should be instructed to keep casts and splints clean and dry so as to prevent skin maceration.[26]

ALLERGIC REACTIONS

Allergic reactions to cotton, fiberglass, and plaster have been reported but are exceedingly rare. Orthopedists and orthopedic technicians may develop a contact dermatitis from continued exposure to plaster over many years. Gloves should be worn for plaster and fiberglass application.

SUMMARY

The initial management of orthopedic trauma is a fundamental aspect of Emergency Medicine. Fractures and dislocations of the extremities are routinely handled in the Emergency Department with prompt Orthopedic follow-up or consultation. The application of external immobilization can be the definitive or temporizing management of the injured extremity. The application of splints accounts for the majority of immobilization of injured extremities. Cast application plays a role in maintaining bony alignment following closed reductions of fractures.

Clear benefits of external immobilization include pain relief and the reduction of further soft tissue injury from bony fragments. Immobilization of the fracture decreases motion and traction on the nerve-rich periosteum.[4,11] The immobilization of fracture ends protects adjacent neurovascular structures from injury and helps prevent bony fragments from penetrating the skin. External immobilization also reduces the area available for hemorrhage and decreases bone bleeding.[4] Early immobilization leading to fracture stabilization is also important in reducing the morbidity associated with long bone fractures.[25] Finally, the closed treatment of fractures facilitates the body's natural processes of repair. External periosteal and internal intramedullary callus formation is optimized in the setting of bony alignment that has been secured by casting or splinting.

The application of splints is an essential skill for any Emergency Physician. The application of a cast in the Emergency Department is appropriate in some select situations. The casting or splinting of an extremity is simple, easy to perform, and relatively quick.

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SECTION 7

Skin and Soft Tissue Procedures

114 General Principles of Wound Management

Ivette Motola and John Sullivan

INTRODUCTION

An acute wound can be defined as a traumatic disruption in the integrity of the skin, including the epidermis and dermis. **The goals of wound management are to restore tissue continuity and function, minimize chances of infection, repair with minimal cosmetic deformity, and distinguish wounds that require special care.** The principles of wound management will be emphasized over specific repair techniques in this chapter. The following chapters will discuss specific details regarding different wounds. **Appropriate management of a wound will result in optimal healing while minimizing the risk of complications.**[1,2] This includes wound cleaning, debridement of the wound edges if needed, and determination of whether a wound requires closure, when wounds are closed, and how wounds will be closed.[3]

PHYSIOLOGY OF WOUND HEALING

THE THREE PHASES OF WOUND HEALING

There are three phases of wound healing (**Figure 114-1**). The first phase involves coagulation and inflammation. The second phase is the proliferative phase. The final phase is the re-epithelialization or remodeling phase.

Phase I consists of coagulation and inflammation, also known as the vascular phase. It occurs in the first 5 days. A fibrin clot forms a transitional matrix that allows for the migration of cells into the wound site over a period of 72 hours. Elevated levels of immunoglobulin G and wound C-reactive protein are found in this phase of wound healing. Inflammatory cells (i.e., macrophages, monocytes, and neutrophils) break down soluble wound debris, kill microbes, prevent microbial colonization, and secrete cytokines.[4]

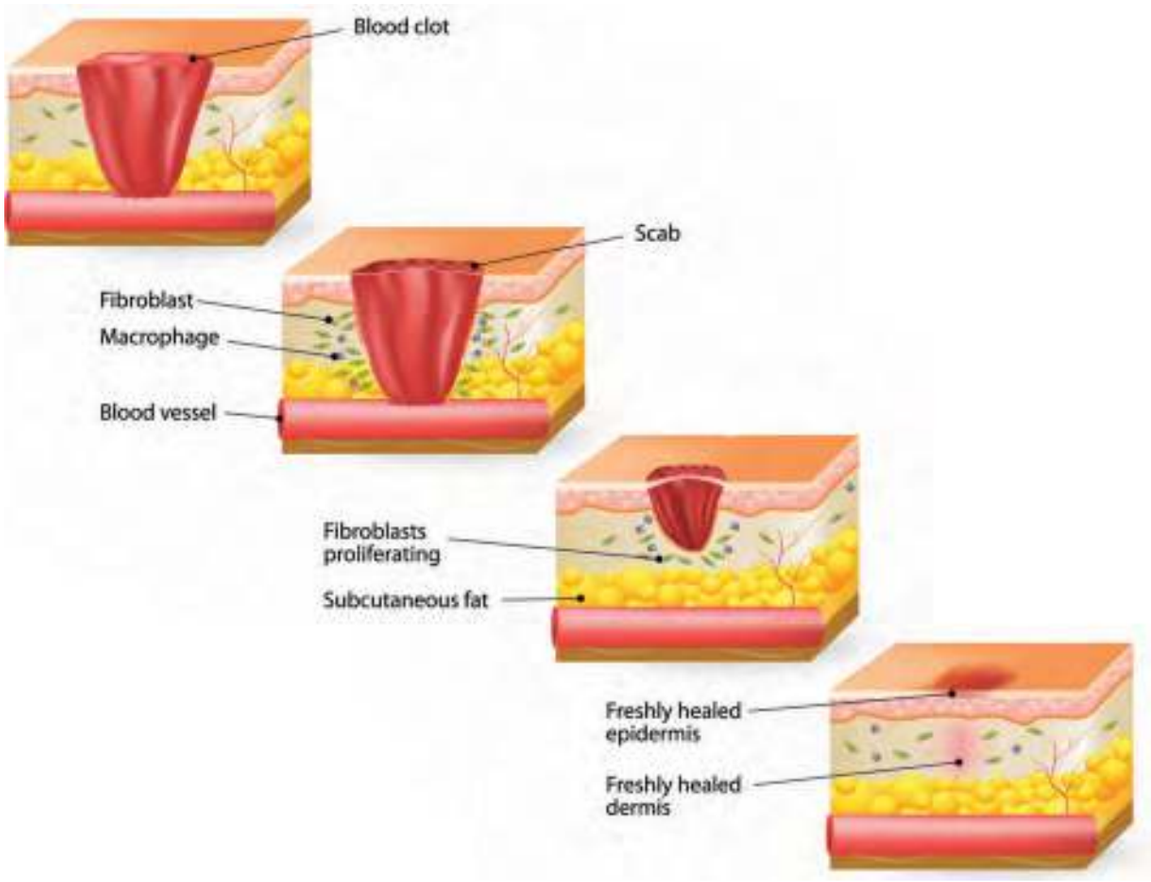

FIGURE 114-1. Wound healing. (Photo courtesy of www.shieldhealthcare.com.)

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The cytokines signal fibroblasts to initiate phase II. Most sutured wounds develop an epithelial covering that is impermeable to water within 24 to 48 hours.

Phase II is the proliferative phase and occurs 5 to 14 days after the injury. Fibroblasts proliferate and synthesize a new connective tissue matrix that replaces the transitional fibrin matrix. Granulation tissue consists of abundant capillaries, epidermal cells that have migrated, fibroblasts, and immature connective tissue within the wound. Fibroblasts release collagen, a protein component of connective tissue. **The tensile strength of the wound at 5 days is 5% that of normal skin. Collagen formation peaks at day 7.**

Phase III is the remodeling, re-epithelialization, or maturation phase. It occurs from day 14 and lasts until there is complete healing of the wound. The new granulation tissue is being converted into a scar. The scar consists of a matrix with lower cell density and lower vascular density compared to unscarred skin, with increased thickness of collagen fiber bundles packed in parallel arrays.[5] **The wound will have 15% to 20% of its full strength at 3 weeks and 60% of its full strength at 4 months**. Tensile strength continues to increase up to 1 year after wounding. The skin will eventually regain only 70% to 90% of its original tensile strength.

SCAR FORMATION

Approximately 6 to 12 months are required to form a mature scar. **This explains why scars should not be revised until 12 months have passed.[6] A wider scar, inadequate wound closure, or a wound dehiscence may occur in areas with increased skin tension or if the wound is in an area of excessive motion (e.g., over joints).** Adequate immobilization of the approximated wound but not necessarily the entire anatomic part is mandatory after wound closure for efficient healing and minimal scar formation**. Contractures can develop when a scar crosses perpendicular to a joint crease. These patients may require physical therapy to prevent the loss of range of motion secondary to contractures.**

Hypertrophic scars result from full-thickness injuries. **Hypertrophic scars are characterized by a thick and raised scar that remains within the boundaries of the original injury (Figure 114-2*A*).** They must often be corrected by surgical intervention.[5]

Keloids are hypertrophic scars (i.e., thick and raised) that exceed the boundaries of the initial injury (Figure 114-2*B*). They can develop from superficial injuries and appear to have a genetic basis. Surgical intervention rarely resolves keloids. They may be prevented or minimized by application of pressure dressings, calcium channel blocker injections, glucocorticoid injections, and silastic dressings.[5]

The repair procedure may result in more scar tissue. Absorbable suture materials contribute to the formation of suture marks because of their increased reactivity whereas nonabsorbable materials do not. Wounds that are approximated too tightly can result in tissue ischemia and more scar tissue formation.

FACTORS ADVERSELY AFFECTING NORMAL REPAIR

It may be useful to divide factors that may negatively impact on optimal wound healing into local and systemic issues. Local causes of improper wound healing include tension on the wound edges and necrosis and/or ischemia of the tissues from local conditions. Crush injuries and contusions decrease blood flow and lymphatic drainage, which alters local defense mechanisms. Retained foreign bodies or contaminated wounds may result in wound infection and poor healing.

Wound infections occur as a result of the patient's resident flora and the environment. Infection is related to wound age, the amount of devitalized tissue, and the tissue concentration of pyogenic bacteria. A wound infection exists when there are bacterial densities of more than 10,000 organisms per gram of tissue.[7] Bacteria slow wound healing by secreting proteases that directly injure the tissue in the wound.[8] Bacteria secrete factors that lead to excess inflammatory cells in the wound which also causes injury to the tissue.[9]

Systemic factors are equally important to recognize when treating an acute wound. Hypovolemia is a major deterrent to wound healing in patients and may occur due to any form of shock. Sepsis originating from the wound or from systemic infection unrelated to the wound may result in a later cause of shock. Other systemic conditions that may result in impaired wound healing include atherosclerosis (i.e., causing peripheral arterial disease resulting in

A

B

FIGURE 114-2. Abnormal scar formation. ***A.*** Hypertrophic scar. ***B.*** Keloid scar. (Photos used from www.commons.wikimedia.org.)

decreased oxygen and nutrient delivery to the wound), collagen vascular disease, immunosuppression, microangiopathy secondary to diabetes, and renal insufficiency. Polymorphonuclear leukocyte function is known to be impaired from cancer, chronic infections, hyperglycemia, jaundice, and uremia.

Medications and nutrition can contribute to good wound healing or affect it adversely. A lack of protein, a lack of vitamins (e.g., vitamins A and C), and malnutrition may inhibit or prolong healing. Zinc deficiency is reversible and may play a role in retarding the healing process.[9] Cytotoxic drugs can delay wound healing. Anti-inflammatory drugs (e.g., aspirin, colchicine, and glucocorticoids) disrupt collagen synthesis, macrophage function, and polymorphonuclear neutrophil concentrations. Glucocorticoids result in delayed wound repair by slowing cell proliferation.[10]

PATIENT EVALUATION AND ASSESSMENT

HISTORY

A thorough and accurate history and physical examination are essential for optimal wound management. Documentation of the patient's age, allergies (e.g., latex or local anesthetics), medications, prior tetanus immunization history, systemic illnesses, and the circumstances of the injury is essential to wound management. These principles are emphasized because the presence of disease processes (e.g., alcoholism, chronic malnutrition, diabetes mellitus, extremes of age, hepatic insufficiency, immune suppression, malignancies, and renal insufficiency) may impair host defenses or complicate wound healing.[11,12]

The wound is often less important than an associated injury to an adjacent structure or cavity. Associated injuries can easily be missed without a specific directed search for their presence. There may be times where wound management is not the immediate concern. Initial trauma management always starts with assessing the patient's airway, breathing, circulation, and any life-threatening conditions. This must take precedence to definitive wound repair. There are times when a laceration may not be closed in the Emergency Department (e.g., a complex facial laceration in the setting of a traumatic intracranial hemorrhage presenting to a community hospital may require immediate transfer to a trauma center). Ask why the trauma occurred. The cause of the wound is straightforward in many instances. Abuse, metabolic abnormalities, or systematic abnormalities may have occurred. Abuse is especially important in elderly and pediatric populations.

Obtain a thorough history concerning the patient's tetanus immunization status. Important factors to consider in assessing the risk of developing tetanus include prior immunization history, the type of wound, the degree of wound contamination, the time from injury to treatment, and the presence of underlying medical disease.

MECHANISM OF INJURY

Severity of injury and associated injuries may be anticipated by determining the precise injury mechanism. This will often suggest whether there is additional soft tissue damage, the presence of a foreign body, or the amount of contamination present.

Document specific components such as how the injury occurred, when the injury occurred, where the injury occurred, and what contaminants were present or involved. Determine the position of the hand at the time of the injury, what kind of work the patient does, and which is the patient's dominant hand if the wound involves the hand. Complicated wounds (e.g., caused by animal or human bites, chemical exposure, or high-pressure injection) may require a more extensive evaluation and consultation with the appropriate specialist.

The mechanism of injury may alter the disposition of a patient. A large but hemodynamically stable machete laceration on the back may be repaired in a community Emergency Department. A 2 mm puncture wound of the hand caused by a high-pressure paint gun may require transfer of care to a Hand Surgeon or to a trauma center depending on the local resources.

TIME OF INJURY

Time of injury must be determined as part of the history. The literature varies regarding if and when to close wounds when presentation to the Emergency Department is delayed. This can often be a challenging decision for the Emergency Physician. Wounds older than 10 hours, or 8 hours for the hand, were found to be at higher risk for infection.[13] Another study performed in an underdeveloped country indicated that wounds might be approximated up to 18 hours after injury.[14] Lacerations that are at low risk of infection (e.g., face, scalp, and torso with minimal contamination) may be closed primarily up to 12 to 24 hours after injury because of the excellent circulation in these areas.[12] Other lacerations may be closed primarily if they are less than 6 to 12 hours old if not heavily contaminated or located in high-risk areas (e.g., foot or hand). One important study concluded that diabetes, lower extremity wounds, wound contamination, and wound length greater than 5 cm were significantly more important than a defined cutoff point for wound closure in terms of risk of infection.[15]

CLASSIFICATION OF WOUNDS

Wounds are described and classified based on their cause and the type of injury (**Figure 114-3**). Abrasions are the result of grinding or abrading forces on the skin. The epidermis and/or dermis is disrupted but not removed in its entirety. Crush injuries are due to compressive forces. The patient sustains a large amount of kinetic energy that results in devitalized tissue, edema, and microvascular disruption. Crush wounds are 100-fold more likely to become infected than lacerations because of the much lower bacterial loads required for infection.[10]

Lacerations are wounds that are caused by shear forces that result in a tearing of the tissue. They are subclassified as avulsion, shear, or tension lacerations. Avulsion lacerations are injuries where there is sharp trauma at an angle that removes the epidermal and possibly the dermal layer of skin. The injury creates a skin flap. Shear lacerations are produced by a sharp force perpendicular to the skin surface that results in a tidy or clean wound. These wounds are usually caused by glass, knives, or sharp metal objects. There is little tissue damage and this type of wound is not prone to infection. Tension or tensile lacerations are injuries with jagged or contused edges that are created by a compressive force. These wounds pose a greater risk for infection than shear lacerations.[16]

Punctures result in a wound that is deeper than it is wide. The skin opening is small and the depth of the wound is often unknown. Puncture wounds are made by discrete and thin objects. **Puncture wounds carry a high risk for infection. Irrigation is mandatory for puncture wounds. The irrigation pressure must not be so high as to drive contaminants deeper into the wound.**

The wound may be clinically classified based on an estimate of microbial contamination and the subsequent risk of infection. Clean wounds are those that occur under aseptic technique. These are usually surgical incisions that are elective and preceded by a thorough skin cleansing and decontamination process. Clean-contaminated wounds are those associated with the usual and normal flora of the region. There is no contamination from foreign

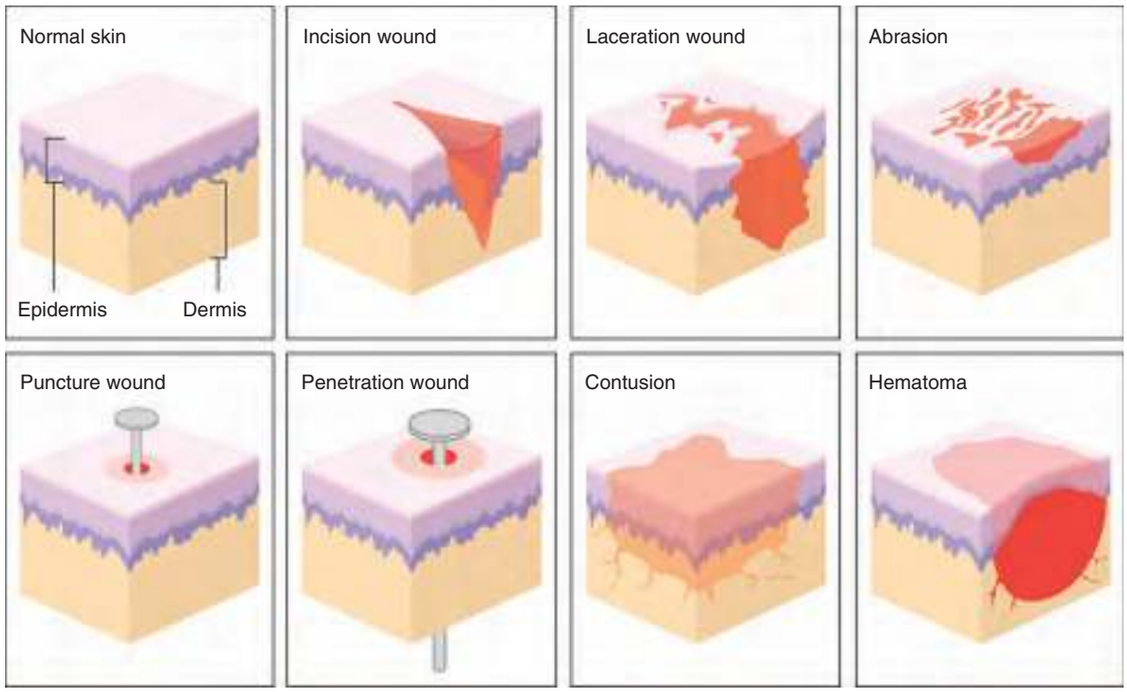


FIGURE 114-3. Some of the common types of wounds. (Photo used with permission under license of shutterstock.com.)

bodies or pus. Contaminated wounds are those that are traumatic (e.g., lacerations and open fractures), less than 12 hours old, or associated with a break in aseptic technique. Most wounds seen in the Emergency Department are classified as contaminated. They may be associated with the introduction of "dirt" or foreign bodies into the wound. Dirty wounds are those that are heavily contaminated (e.g., feces or soil), occur through infected tissue, are over 12 hours old, are associated with retained foreign bodies, or are associated with devitalized tissue.[17]

WOUND ASSESSMENT

A complete examination and documentation of the laceration is necessary. This includes noting the location and depth of the laceration, the presence of any gross contamination, the presence of an obvious foreign body, and any associated injuries. **Measure the wound length and do not estimate the length visually.**[18] Physicians are often wrong in their estimates of wound length. The length affects good documentation and billing.

Assessment of soft tissue wounds involves an examination of the surrounding neurologic structures, surrounding vascular structures, and tendons. Look for bony injuries and foreign bodies. Emergency Physicians must possess a working knowledge of functional anatomy, particularly the distal upper extremity and face.

HEMOSTASIS

Hemostasis must be achieved to adequately visually inspect a wound (Chapter 137). Hemostasis for most simple lacerations can be achieved with direct pressure. Determine the reason when several minutes of direct pressure do not achieve hemostasis. Is there an arterial injury? Is there a foreign body? Does the patient have a coagulopathy, either intrinsic due to hemophilia or medically induced? Use a blood pressure cuff or pneumatic tourniquet proximal to extremity wounds and inflated after raising the extremity to reduce venous blood to allow visualization of the wound and elucidation of the cause of bleeding. Deflate any type of tourniquet after no more than 20 to 30 minutes to restore circulation and to determine if hemostasis has been achieved. Suturing the wound best controls bleeding of the scalp. There are several tourniquets available and hemostatic agents can be applied for more severe bleeding (Chapter 137).[19-21] **Do not clamp vascular structures except small arterioles, small venules, and vessels within muscles. Vascular structures may require special techniques for hemostasis.**

FUNCTIONAL EXAMINATION

It is important to explore the deep structures through a full range of motion to detect partial tendon lacerations or joint capsule disruption. **Tendons can be evaluated by inspection. Test individual muscles for full range of motion and full strength.**

NEUROVASCULAR EXAMINATION

Perform a distal neurologic and vascular examination on extremity injuries. Check capillary refill distally. It should take less than 2 seconds. Neurologic assessment involves checking distal muscle strength and sensation. **Check two-point discrimination prior to the administration of anesthesia for hand and finger lacerations**. Two-point discrimination at 5 mm on the radial and ulnar aspects of the finger pads is the most efficient method of assessing median and ulnar nerve function. Two-point discrimination should be less than 1 cm at the fingertips. A crush injury may be associated with decreased two-point discrimination and may take several months for recovery. Numbness may be an early sign of a developing compartment syndrome (Chapters 93 and 94). Nerve lacerations may or may not be repaired depending on location and functional status of the patient. **Document any nerve lacerations and explain them to the patient and/or family.** A finger laceration resulting in distal sensory loss may be irrelevant in an elderly patient with severe dementia. The same injury may be extremely relevant for a professional pianist and may require referral to a Hand Surgeon.

EVALUATION FOR FOREIGN BODIES

Failure to identify foreign bodies in wounds may lead to complications (e.g., delayed wound healing, increased risk of infection, and loss of function).[12,22] Foreign bodies and foreign matter greatly enhance the infectivity of a given bacterial inoculum.[23] Retained foreign bodies are a common complication of simple wound repair. **Perform a thorough inspection to attempt to diagnose the presence of a foreign body.** Missed foreign bodies have been noted in the literature to be one of the leading causes of lawsuits brought against Emergency Physicians. Many lawsuits brought against Emergency Physicians and up to a quarter of closed claims are related to retained foreign bodies.[22,24,25] Some foreign bodies cause an inflammatory reaction (e.g., cloth, rubber from shoes or foam insoles, splinters, teeth, thorns, and wood), while others do not (e.g., glass, metal, most plastics, and pencil graphite).

Wound exploration, irrigation, and radiography or ultrasound may be needed when the clinical setting suggests a possible foreign body (Chapters 120 and 121). **Spread the tissue during exploration. Do not cut tissue and risk neurovascular injury**. **Puncture wounds have not been proven to benefit by coring or probing to determine the depth of the wound. Imaging may be required to detect retained foreign bodies.** Retained wood, thorns, and plastic are often detectable by wound exploration and ultrasound but may not be visible on plain radiographs. Radiographs will identify retained metallic fragments and more than 90% of glass foreign bodies if the glass does not have a low lead content and the fragments are at least 2 mm long.[26,27] Other studies have shown that the lead content of glass does not actually contribute to visualization on plain radiographs.[28,29] Wound markers can be used during radiography. Radiographs obtained in two planes can help localize the object for recovery. Glass may penetrate at an angle and be buried deeper than it appears. **The use of additional imaging (e.g., computed tomography [CT], magnetic resonance imaging [MRI], or ultrasound) may contribute to identification of foreign bodies when the clinical suspicion is high but suspected foreign bodies are not seen on radiographs or by direct visualization.**[30,31]

Foreign bodies that do not cause an inflammatory reaction are not always removed from lacerations. This is especially true if there are multiple fragments or if excessive tissue disruption will result with attempted removal. **Make the patient aware of any retained foreign bodies at the time of discharge, their benign presence, why removal was not attempted, the possibility of later infection, and the fact that they may eventually self-extrude. This must also be documented in the medical record**. Wounds in a complex area (e.g., the palm) may require specialist consultation for immediate or delayed removal. The wound can be approximated loosely and immobilized for comfort and to avoid further tissue disruption. Prescribe antibiotics and arrange for appropriate outpatient follow-up in 24 to 48 hours.

EVALUATION FOR GROSS CONTAMINATION

Soils have varied levels of contamination potential. Sandy soils present a low risk of wound contamination. Clay-containing soils are pyogenic because they impair host defense mechanisms and promote inflammation. Organic soils contain *Clostridium tetani* and many concentrated bacteria. Soil contaminants can be removed by copious high-pressure irrigation. Leave contaminated wounds open and allow them to heal by secondary or tertiary intention.

Obtain radiographs if there is concern for an underlying fracture or possible bone exposure. Bone injuries require checking the overlying skin to exclude an open fracture. An open fracture is an indication for surgical debridement and operative repair. An exception is the distal phalanx fracture. These can be treated with copious irrigation, oral antibiotics, and detailed discharge instructions.

HIGH-RISK WOUNDS

Many wounds require special consideration in deciding upon the method of closure, the type of suture to use, and the use of antibiotic prophylaxis. These include wounds contaminated by feces, organic material, saliva, soil, and vaginal secretions. **Immunocompromised patients or patients taking immunosuppressive drugs may require antibiotics and longer times for the stitches to remain before removal**. Bite wounds, foot wounds, and hand wounds require special care. Wounds greater than 6 to 12 hours old, other than wounds on the face, may require delayed closure. Puncture wounds may require radiographs, incision, exploration, and antibiotic prophylaxis. Wounds accompanied by excessive tissue damage and devitalization or crush injuries are prone to infection. Wounds with retained foreign bodies may require radiographs, exploration, and removal. Major tissue defects may be closed with advanced wound closure techniques (Chapter 118). Wounds overlying sites of active infection require antibiotics and delayed closure. These topics are covered later in this chapter and in other chapters of this book.

WOUND CLOSURE

OVERVIEW

Most patients presenting to the Emergency Department arrive with the expectation that their wounds will be closed. It is critical that the Emergency Physician understands the various options for wound closure including the decision to delay closure when patients are at high risk of infection. Patient education and specific follow-up are important as always. **Education and follow-up are crucial when patient expectations are not met for medically indicated reasons.** Below are definitions of the three wound closure techniques and information regarding when these techniques may best be used.

PRIMARY INTENTION

Primary intention involves surgically approximating the wound edges shortly after the time of injury. The skin's greatest strength is in the dermal layer. The best repair results when the entire depth of the dermis is accurately approximated to the entire depth of the opposite dermis. **Accurate approximation of the epidermis gives a cosmetically appealing effect to the repair but does not contribute to its strength. Wound eversion and the use of buried stitches can greatly improve healing by primary intention.**

SECONDARY INTENTION

Secondary intention involves allowing the wound to heal without any surgical intervention. The wound is left open and allowed to heal from the inner layer to the outer surface. It is a more prolonged healing process than primary intention. Excessive trauma, imprecise approximation of tissue, infection, or tissue loss may result due to healing by secondary intention. Wound contraction by granulation tissue containing myofibroblasts is the major influence on this type of healing. Wound contraction becomes more significant when the dermis is lost.

Concave skin wounds heal with the best results. These areas may heal better by secondary intention than by primary intention in well-cleaned wounds. This is simply because the skin naturally approximates in these regions (e.g., concave areas of the pinna,

inner ear, nasal alar crease, nasolabial fold, and temple). Flat surfaces (e.g., periorbital areas, side of the nose, and forehead) can heal well by secondary intention, although surgical intervention may be best. Wounds on convex surfaces (e.g., malar cheek, the tip of the nose, and the vermilion border of the lip) are not optimal for healing by secondary intention.[8]

TERTIARY INTENTION (DELAYED PRIMARY CLOSURE)

Tertiary intention or delayed primary closure can often decrease infection rates. Wound closure by tertiary intention is accomplished 3 to 5 days following the initial injury. **It is a combination of allowing the wound to heal secondarily for 3 to 5 days and then primarily closing the wound.** It is the safest method of repair for wounds that are associated with extensive tissue loss, contaminated, dirty, infected, at high risk of infection, and wounds "too old" to close. **The ultimate cosmetic result is the same as that of primary wound closure.** This method may not be ideal for young children having to return a second time for an uncomfortable procedure. This must be clinically weighed against the risk of infection which might result in the need for antibiotics, dehiscence, hospitalization, poor outcomes, or repeated visits. The site of the planned return visit may be the Emergency Department, specialist office (e.g., Plastic Surgeon), or wound care center depending upon the local resources and customs.

Delayed primary closure wounds are all at high risk of infection. This technique involves a thorough cleaning of the wound just as would be done with primary closure. Anesthetize the wound and irrigate it using high pressure. Consider scrubbing this type of wound with saline-moistened gauze, packing it with wet gauze, and then covering it with a surgical dressing. Instruct the patient to apply wet-to-dry dressing changes once or twice a day. Assess the wound for any signs of infection upon the patient's return. Anesthetize and clean the wound again. Scrub the wound base and edges with saline-moistened gauze and irrigate the wound to remove any dirt, debris, and granulation tissue. Stitch the wound to approximate and evert the wound edges. The postprocedural wound care is the same as if the wound was closed primarily.

SKIN AND WOUND PREPARATION

ANESTHESIA

Wounds are anesthetized with local or regional techniques prior to cleansing and repair. Local anesthesia may distort wound edges. Use regional nerve blocks (Chapters 155 to 158) where appropriate (e.g., ear, face, hand, nasal cartilage, palm, and sole). Refer to Chapters 153 through 159 for a complete discussion of local anesthetic agents, nitrous oxide anesthesia, procedural sedation, regional anesthesia, and topical anesthesia.

Lidocaine (Xylocaine) in a dose not to exceed 4.5 mg/kg is an effective and standard local anesthetic agent. Lidocaine anesthesia lasts approximately 60 to 90 minutes. Use bupivacaine if a longer period of anesthesia is required. Bupivacaine provides approximately 120 to 180 minutes of anesthesia. The addition of a 1:100,000 dilution of epinephrine to either will prolong the duration of anesthesia, promote hemostasis, and reduce systemic absorption of locally infiltrated anesthetic solution. Epinephrine is a potent vasoconstrictor and should not be used near end organs (e.g., fingers or toes). It may decrease blood flow and induce ischemia. Avoid epinephrine near the ear, penis, and tip of the nose.

The injection of local anesthetic is often painful. This pain can be reduced by using several techniques. The use of a 27 or 30 gauge needle, addition of bicarbonate to lidocaine (i.e., 9 mL lidocaine to 1 mL of bicarbonate), slower and deeper infiltration into the dermis, and warming the local anesthetic solution may decrease the pain of anesthetic injection.[32-37] Other strategies involve anesthetizing as much tissue as possible through a single site and starting proximally on the extremity and moving distally. Infiltration of the local anesthetic solution through the open wound edges is less painful than through intact skin.

Many reported "allergic" reactions to anesthetics are vasovagal or other adverse responses. True allergies to local anesthetics are rare and are generally seen only with the ester class of local anesthetics. The use of an ester class of local anesthetic is suggested if an allergy to lidocaine (an amide class of local anesthetic) is suspected. An alternative is the use of cardiac lidocaine. The prefilled syringes used in cardiac arrests and codes contain no preservative. It is felt that the preservative in lidocaine is responsible for the most allergic effects. Another alternative is to use a 1% to 2% solution of diphenhydramine (i.e., Benadryl). This provides adequate but not ideal anesthesia. The most common complication of local anesthesia infiltration is hypotension and bradycardia from a vasovagal reaction.

Topical anesthesia is an attractive alternative to injection, particularly in the management of pediatric patients with simple wounds. Lidocaine, epinephrine, and tetracaine (LET) or tetracaine, adrenaline, and cocaine (TAC) are two agents that can be used to provide effective local anesthesia.[38] **These agents contain epinephrine and should not be used on areas involving an end artery or contaminated wounds.** TAC involves expense and incorporates problems with the use and maintenance of a controlled substance. TAC also has the potential for toxicity, especially when applied to mucosal surfaces. EMLA (i.e., eutectic mixture of local anesthetics) cream has been found to provide effective anesthesia for extremity lacerations. EMLA is a combination of 2.5% lidocaine and 2.5% prilocaine suspended in an oil-in-water emulsion. It takes longer to obtain optimal anesthesia with EMLA than with TAC.[39]

SKIN AND WOUND CLEANSING

Meticulous preparation of the skin surrounding the wound, thorough wound irrigation, and wound debridement when needed are critical to good wound healing. The goal of wound cleansing is to remove bacteria, foreign matter, and tissue debris. Anesthetize wounds prior to cleansing and/or local exploration. Use adequate light, anesthesia, and equipment to avoid inadequate debridement, a retained foreign body, or a wound hematoma that can result in a necrotizing soft tissue infection.

Disinfecting the intact skin surrounding the wound and ridding it of foreign bodies, debris, and particulate matter is the initial step in wound preparation. This technique can be accomplished by scrubbing the skin with povidone iodine, chlorhexidine, or poloxamer 188 (Shur-Clens) skin-prep solutions. **Do not expose the wound itself to these solutions.** Povidone iodine and chlorhexidine solution are bactericidal and work as they dry. Their toxicity to wound tissue is controversial. Shur-Clens has no tissue toxicity but also has no antibacterial activity. Prep a wide area surrounding the wound with an antimicrobial agent, preferably povidone iodine or chlorhexidine solution.

HAIR REMOVAL

Hair removal is often unnecessary prior to closing wounds, can be embarrassing for the patient after discharge from the Emergency Department, and may increase the risk of wound infection. Shaving can cause minimal soft tissue trauma and wound infections.[40] **Eyebrows can be shaved if necessary**. It is a myth that they do not

grow back.[41] One meta-analysis of surgical preoperative patients showed that hair removal of any form did not decrease the risk of infection.[42] Simple scalp lacerations can be exposed by using antibiotic ointment or lubricating gel to move the hair away from the wound margins prior to placing sutures or staples. This works in other hair-bearing areas of the body.

WOUND IRRIGATION

Wound cleansing and preparation have been proven to be the foundations of proper wound management and the prevention of wound infections. Irrigation removes contaminants, reduces infection, and improves visualization. There are concerns regarding wound irrigation. What is the pressure required for adequate cleansing of the wound? What means are available to irrigate the wound safely while protecting the health care worker from the threat of human immunodeficiency virus and hepatitis B by contamination?

Irrigation pressures of 5 to 8 pounds per square inch (psi) are felt to be adequate to cleanse a wound that is not heavily contaminated. This surface pressure can be generated by the combination of a 35 mL syringe and a 19-gauge angiocatheter held 2 cm from the wound surface.[43-45] Take care as this process can be quite messy (**Figure 114-4*A***). Use one of the many wound shields to decrease potential exposure and messes (**Figure 114-4*B***). High-pressure irrigation generates peak pressures of 25 to 40 psi and has been a controversial technique in the Emergency Medicine literature. The theory is that high pressures may cause tissue disruption and increase infection rates. Reserve high-pressure irrigation for highly contaminated wounds. High-pressure irrigation may drive contaminants deeper into puncture wounds and should be avoided.

There are a variety of irrigation fluids and the optimal type is unknown. Normal saline is the most commonly used irrigant. Several studies have shown that tap water is as effective with no increased incidence of infection, especially when a large volume of irrigant is required.[46-54] The volume of irrigation fluid to be used has not been well established. The use of 100 to 300 mL has been suggested in the literature. Heavily contaminated wounds require larger amounts of irrigant. Anecdotal recommendations suggest using 50 mL/cm for clean wounds and 100 mL/cm for dirty wounds. Heavily contaminated wounds may have to be scrubbed after adequate anesthesia with fine-mesh gauze or a micropore sponge using a 1% solution of povidone iodine or poloxamer 188. Soaking of wounds is discouraged and is a poor substitute for the preparation of contaminated or clean wounds. **Do not soak wounds in any fluid.** Soaking does not reduce bacterial contamination or decrease infection rates. Soaking may increase infection rates. **Do not use detergents, hydrogen peroxide, or undiluted povidone iodine in the wound as they cause tissue toxicity.**[55]

Numerous commercially available devices are available to irrigate a wound. Use barrier protection to shield the eyes, face, mucosal surfaces, and skin during irrigation. There are several barrier devices on the market that decrease the splatter of irrigation fluid. Some of these devices are preattached to a wound irrigation device. Others can be attached to a wound irrigation device (**Figure 114-4*C***).

WOUND DEBRIDEMENT

Debridement of wounds helps create straight and clean wound edges that are easier to repair by removing tissue that is contaminated by bacteria, contaminated by foreign matter, or devitalized and may impair the ability of the tissue to resist infection. Successful wound closure may require the transformation of a ragged

A

B

C

FIGURE 114-4. Wound irrigation. ***A.*** An angiocatheter on a syringe can be quite messy and can result in an occupational exposure. (Photo courtesy of Zerowet Incorporated.) ***B.*** The Igloo Wound Irrigator. (Photo courtesy of Bionix Medical Technologies, Toledo, OH.) ***C.*** The Igloo attached to a syringe for wound irrigation. (Photo courtesy of Bionix Medical Technologies, Toledo, OH.)

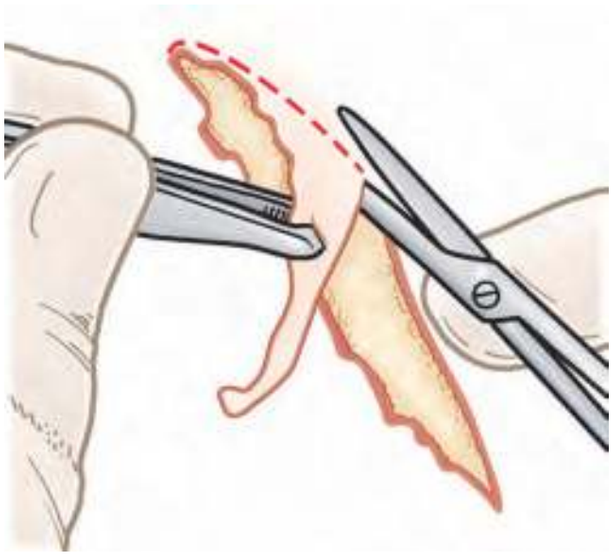


FIGURE 114-5. Wound debridement. Removal of the wound edges with a scissors (or a scalpel).

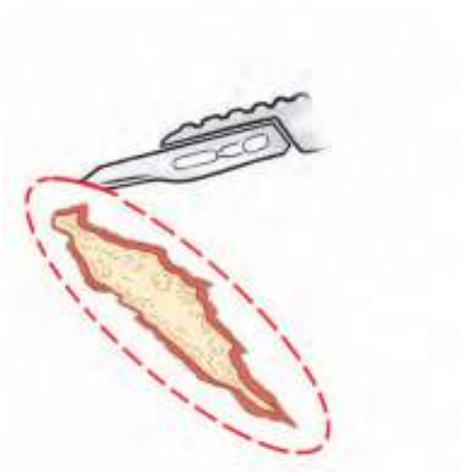


FIGURE 114-6. Wound excision. Removal of an ellipse of tissue that contains the wound results in smooth, clean edges that can be approximated.

laceration, the removal of devitalized tissue, or the removal of contaminated tissue to convert a traumatic wound into a surgical wound (**Figure 114-5**). Devitalized and necrotic tissue must be removed to remove a nidus for bacterial growth and wound infection.[12,56-58]

Close approximation of the wound requires that debridement of jagged edges not be too vigorous to avoid widening the wound and making it difficult to close. Wounds of the face or areas that are devoid of redundant tissue (e.g., feet or hands) require conservative debridement. Debridement to simplify wound closure is not always the answer for a superior cosmetic result in the repair of irregular wound edges. The meticulous repair of complex wound edges can often provide a superior cosmetic result than debridement.

Debridement can be accomplished mechanically, hydrodynamically, or with a combination of both methods. Tissue can be removed mechanically with a #11 or #15 scalpel blade or a scissors (**Figure 114-5**). Superficial debris and contaminants can usually be removed with high-pressure irrigation. **Debridement must be performed using aseptic technique. Scrubbing is not a substitute for debridement of heavily contaminated tissue. Handle wound edges delicately to avoid further soft tissue damage and devitalization of injured tissue.**

WOUND EXCISION

The entire wound may be excised in areas of excess tissue or tissue laxity if no blood vessels, joints, nerves, or tendons lie within or at the base of the wound (Chapter 118 and **Figure 114-6**). The excision of a wound creates smooth, clean edges that may be approximated with suture. This is especially useful in wounds that are heavily contaminated. Most wounds are excised using an elliptical incision (**Figure 114-6**). Other types of wound excision are discussed in Chapter 118.

Carefully plan the excision before removing any tissue. Mark the edges of the proposed incision with a sterile skin marking pen. **Make the long axis of the ellipse two-and-a-half to four times as long as the greatest width of the ellipse.** Removal of too much tissue will produce a large defect that may not be possible to primarily close. **Remove the tissue using aseptic technique to prevent any contamination of the new wound edges.**

WOUND UNDERMINING

The undermining of tissue creates a "flap" that involves the separation of the skin and superficial subcutaneous tissue from the deeper subcutaneous tissue and fascia (Figure 114-7). The process of undermining tissue minimizes skin tension, allows for eversion of the approximated skin edges, and relieves the extrinsic tension from stitches. **Undermining is performed when the wound cannot be closed due to a tissue defect or if a wound is under tension**. **This procedure requires the Emergency Physician to be familiar with the local anatomy so that no blood vessels, nerves, or tendons are injured in the process. Do not undermine contaminated wounds**. Undermining large areas can separate the skin from its underlying blood supply and result in a diminished blood flow that predisposes the area to infection and necrosis.[59] Undermining may be useful on the arm, calf, forearm, forehead, scalp, thigh, and torso. **Never undermine wounds on the face, palms, and soles.**

Undermine tissue at the dermal-epidermal junction or within the subcutaneous adipose tissue. The amount of undermining necessary to close a laceration is approximately double the width of the gap of the laceration at its widest point. Undermine a 1 cm wide laceration for 1 cm on both sides of the wound, including the ends (**Figure 114-7**). The use of a Mayo scissors versus a #15 scalpel blade to undermine tissue is based on experience and preference. A Mayo scissors is generally recommended as it may cause less secondary injury.

EMERGENCY DEPARTMENT VERSUS OPERATING ROOM MANAGEMENT

Laceration repair may have to be performed in the Operating Room in certain circumstances. Indications for Operating Room repair of lacerations include those associated with other injuries (e.g., neurovascular injuries, open fractures, tendon injuries, and visceral injuries), major or complex wounds involving devitalized tissue or extensive amounts of necrotic or ischemic tissue, heavily contaminated wounds, perineal wounds, large or complicated soft tissue injuries, compartment syndromes, or high-pressure injection injuries.[60] Consider Operating Room repair or a form of regional anesthesia if the amounts of local anesthetic solution required to repair a wound would exceed toxic tissue levels (Chapter 153).

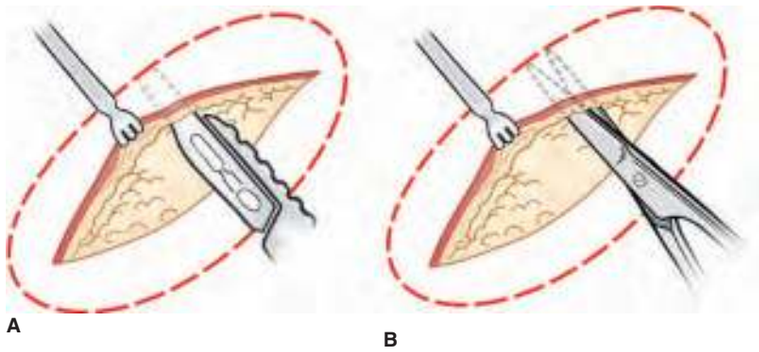


FIGURE 114-7. Wound undermining. ***A.*** Sharp undermining with a #15 scalpel blade. ***B.*** Blunt undermining with a Mayo scissors.

TETANUS PROPHYLAXIS

Worldwide the global incidence of tetanus remains high, although it is comparatively rare in the United States and other developed countries.[61] The reduced incidence in the developed world is due to implementation of large-scale immunization programs, widespread availability of tetanus toxoid and immune globulin, and improved wound care. All traumatic wounds have a potential risk for tetanus infection. Some wounds are more prone to tetanus (**Table 114-1**). The highest risk for developing tetanus in the United States occurs in those with no vaccination, partial vaccination, and patients over 60 years of age (especially if they live in rural areas or are intravenous drug users).

The administration of tetanus prophylaxis is based on the patient's age, immunization history, and the risk of developing tetanus (**Table 114-2**).[62,63] Current guidelines state that tetanus toxoid (Td) may be deferred in patients with "clean, minor" wounds who have completed a primary series or received a booster dose (i.e., Td 0.5 mL intramuscularly [IM]) within 10 years. Consider tetanus immune globulin (i.e., TIG 250 to 500 U IM) in addition to Td for patients at risk of developing tetanus. Elderly patients without documentation of a primary series, patients from nonindustrialized nations, and those from rural or inner-city areas may never have received tetanus immunization and should be considered for TIG.

ANTIBIOTIC PROPHYLAXIS

An important component of optimizing wound healing is the prevention of infection by copious wound irrigation, removal of foreign bodies, debridement of devitalized tissue, and appropriate wound closure. Wound infections still occur with an incidence of up to 6.3% despite the best wound care and management.[64,65] **Most uncomplicated wounds heal without the need for antibiotics.** Antibiotics play an important role in prophylaxis of certain high-risk wounds and in the management of wound infections. Determining which wounds have a higher risk for infection and would benefit from antibiotic prophylaxis is an important component of patient management.

Risk stratification for an infection can be performed based on the patient characteristics, wound location, and wound type.[17] **Wounds that have a high risk of infection include bites, wounds that contain foreign bodies, contaminated wounds (e.g., dirt, feces, and soil), crush injuries, wounds involving deep tissues (e.g., open fractures), wounds with a length > 5 cm, and punctures**.[15] Wounds located in poorly vascularized areas (e.g., the extremities) and those with high concentrations of commensal flora (e.g., axilla, groin, or oral mucosa) are also considered high risk. Wounds in well-vascularized tissue (e.g., head, neck, and scalp) are considered low risk. Patient characteristics associated with a high risk of wound infection include diabetes, immunocompromise, peripheral vascular disease, and age over 65 years.

Recommendations and consensus statements agree that low-risk wounds in immunocompetent patients do not require systemic antibiotics.[12,15,17,66,67] Consider antibiotic prophylaxis when one or more high-risk factors exist (**Table 114-3**). Start antibiotic prophylaxis as early as possible in the Emergency Department. Consider delayed closure to prevent an infection in high-risk wounds.

Topical antibiotics (e.g., bacitracin, mupirocin, neomycin, or polymyxin) are routinely used by many Emergency Physicians. Topical antibiotics in fresh wounds have been shown to decrease the rate of wound infections.[68-70] Some experts recommend topical antibiotics for only crush wounds due to the higher risk of infection.[71]

TABLE 114-1 Characteristics of Tetanus-Prone and Non-Tetanus-Prone Wounds

Clinical feature	Tetanus-prone wounds	Non-tetanus-prone wounds
Contaminated with feces, foreign body, saliva, and soil	Present	Absent
Devitalized tissue	Present	Absent
Infection	Present	Absent
Ischemic or denervated tissue	Present	Absent
Mechanism of injury	Burn, crush, bullet	Sharp and smooth (made with glass or a knife)
Wound age	> 6 hours	< 6 hours
Wound depth	> 1 cm	< 1 cm
Wound type	Abrasion, avulsion, crush, irregular, stellate	Linear or straight

TABLE 114-2 Tetanus Prophylaxis

Immunization history	Tetanus-prone wounds	Non-tetanus-prone wounds
Fully immunized; ≤ 5 years since last dose	None needed	None needed
Fully immunized, > 5 years and < 10 years since last dose	Td	None needed
Fully immunized, ≥ 10 years since last dose	Td and TIG	Td
History of adsorbed Td	Td and TIG	Td and TIG
Unknown or less than 3 doses	Td, TIG, and complete the series	Td and complete the series

Td, tetanus and diphtheria toxoids; TIG, tetanus immune globulin.

TABLE 114-3 Antibiotic Prophylaxis for High-Risk Wounds

Situation	Antibiotic	Treatment days
Dog and cat bites	First dose: parenteral ampicillin-sulbactam, carbapenem, or clindamycin Then: clindamycin plus fluoroquinolone Or Amoxicillin/clavulanate (Augmentin)	3–5
Human bites	First dose: parenteral ampicillin-sulbactam or ertapenem, then Augmentin Or Clindamycin plus fluoroquinolone (sulfamethoxazole-trimethoprim [Bactrim] in children) Or Augmentin	3–5
Intraoral injuries	Penicillin VK or Clindamycin	5
Open fractures	First-generation cephalosporin Add an aminoglycoside for more extensive injuries	1–3

SUTURES

SUTURE TYPES

Suture remains the most commonly used method of approximating open wounds despite the advent of new materials. Properly sized suture material can be summarized as the smallest suture needed to approximate the edges of a wound. Using the smallest size suture that is appropriate for the wound will reduce tissue damage caused by the suture and result in improved cosmesis. The tensile strength of the suture should never exceed the tensile strength of the tissue or it can pull through and damage the tissue (**Table 114-4**). The sutures should be at least as strong as the normal tissue through which they are being placed.

The size of the suture material is related to the diameter of the suture. The diameter of the strand decreases as the number of 0s in the suture size increases. For example, size 5–0 or 00000 is smaller in diameter than size 4–0 or 0000. Smaller sizes of suture have less tensile strength with decreasing size.

Suture can be classified based on several characteristics including the number of strands that make up the suture, the body's ability to break down the material, and whether they are made of naturally occurring or synthetic materials. Monofilament suture is made of a single strand of material. They encounter less resistance passing through tissue, glide easy through the tissue, and are less likely to harbor organisms that may cause suture-line infections. Monofilaments handle easy and elicit a minimal inflammatory response. Multifilament sutures consist of several filaments or strands twisted or braided together. This affords greater flexibility, pliability, and tensile strength. Bacteria can migrate between the filaments and into the wound.

Another classification is based on the ability of the body to break down and absorb the suture material. Sutures that are digested by body enzymes or hydrolyzed in body tissue and lose their tensile strength within 60 days are considered absorbable sutures. Sutures that maintain their tensile strength for more than 60 days are considered nonabsorbable.[59] This distinction between absorbable and nonabsorbable is helpful but not completely accurate. Some sutures that are considered nonabsorbable (e.g., nylon and silk) do lose some tensile strength in the 60-day period.[72]

Absorbable suture can be made of natural or synthetic material. Natural absorbable suture is classified as plain or chromic surgical gut. Plain surgical gut is composed of collagen from bovine or sheep intestine. It is rapidly absorbed, maintains its tensile strength for only 7 to 10 days, and is completely absorbed within 70 days. Chromic gut is plain gut treated with a chromium salt solution to resist body enzymes. It retains its tensile strength for 10 to 14 days and is absorbed over 90 days.

Synthetic absorbable sutures include polyglactin 910 (e.g., Vicryl, Ethicon) and polyglycolic acid (e.g., Dexon). They were developed because of the tissue reaction, suture antigenicity, and unpredictable rates of absorption of natural absorbable sutures. These sutures are braided synthetic materials that retain 50% of their initial strength at 4 weeks. The synthetic absorbable suture retains its tensile strength long enough to ensure the security of the subcutaneous layers after the removal of percutaneous stitches.

Nonabsorbable sutures are made of cotton, linen, metal, nylon, polybutester, polypropylene, or silk. They can be monofilament or

TABLE 114-4 Characteristics of Sutures

Suture	Absorbable	Configuration	Tensile strength	Ease of handling	Knot security	Tissue reactivity	Complete absorption
Glycolide & trimethylene carbonate (Maxon)	Yes	Monofilament	81% at 14 days 59% at 28 days	Fair	Good	Minimal	180 days
Gut, chromic	Yes	Monofilament	Poor at 21–28 days	Poor	Poor	Minimal	90 days
Gut, fast-absorbing	Yes	Monofilament	50% at 3–5 days	Fair	Poor	Low	20–42 days
Gut, plain	Yes	Monofilament	Poor at 7–10 days	Fair	Poor	Moderate	70 days
Poliglecaprone 25 (Monocryl)	Yes	Monofilament	50%–60% at 7 days	Good	Good	Minimal	91–119 days
Polydioxanone (PDS II)	Yes	Monofilament	70% at 14 days 50% at 30 days 25% at 42 days	Poor	Poor	Low	183–238 days
Polyglactin (Vicryl)	Yes	Braided	75% at 14 days 50% at 21 days	Good	Fair	Low	56–70 days
Polyglycolic acid (Dexon)	Yes	Braided	20% at 21 days	Good	Good	Low	60–90 days
Nylon (Dermalon)	No	Monofilament	Good	Good/fair	Poor	Low	
Nylon (Ethilon)	No	Monofilament	Loses 20% per year	Good/fair	Poor	Low	
Nylon (Nurolon)	No	Braided	Good	Good	Fair	Low	
Nylon (Surgilon)	No	Braided	Good	Good	Fair	Low	
Polybutester (Novafil)	No	Monofilament	Extended	Good/fair	Poor	Low	
Polyester (Dacron, Ethibond, Mersilene)	No	Braided	Indefinitely	Very good	Good	Minimal	
Polypropylene (Prolene, Surgilene, Surgipro)	No	Monofilament	Extended	Good/fair	Poor	Minimal	
Silk	No	Braided	None at 365 days	Excellent	Good	Moderate	

FIGURE 114-8. An example of a knotless suture. Note the barbs on the suture; the looped end does not require knotting.

multifilament in construction. Nylon and polypropylene are the most commonly used suture in the Emergency Department. It is used to approximate lacerations at the skin surface. Silk may occasionally be used in the mouth. Silk can cause significant tissue reactions that result in inflammation and granuloma formation as the body "fights off" this natural fiber. The other types of nonabsorbable sutures are generally not used in the Emergency Department.

Several factors must be considered in choosing suture material (e.g., biologic interaction and mechanical performance).[12,59] Choose sutures that match the healing properties of the tissues. Approximate slow-healing tissues (e.g., fascia and tendons) with nonabsorbable suture or a long-lasting absorbable suture. Foreign bodies in potentially contaminated tissues may result in an infection. Multifilament suture can act as a foreign body and may convert a contaminated wound into an infected one. Avoid using multifilament suture. Use monofilament sutures or absorbable sutures that resist harboring infection. Use the smallest inert monofilament suture materials (e.g., nylon or polypropylene), avoid using skin stitches alone (i.e., use subcuticular closure whenever possible), and use sterile skin closure strips for apposition when possible. **Use the smallest possible size of the chosen suture type capable of closing the wound to help minimize scarring.**

Knotless suture is commercially available from many manufacturers (**Figure 114-8**). These are referred to as barbed sutures. The barbs are made by placing nicks along the suture and are all in the same direction (i.e., unidirectional) or in two directions (i.e., bidirectional). The barbs allow the suture to be pulled in one direction. Loops at the distal end allow a fixation point in the tissue. The elimination of knots allows weak spots to be eliminated and quicker insertion.[73] The elimination of knots improves efficiency. Some of the suture has an antibacterial coating.

Surgical zippers are noninvasive and atraumatic when closing superficial wounds. They are mostly used for surgical wounds but can be used in the Emergency Department. These devices are simple to apply using adhesive strips. This results in a quick and safe method to close straight wounds. The adhesive zippers work well in areas that move (e.g., over joints). These devices are rarely used now but more experience will increase their use.

NEEDLES

The surgical needle is used to introduce the suture to the skin and help provide the best approximation of the wound edges. Every surgical needle has three components: the swage, the body, and the point. The swaging process serves to provide a smooth junction between the needle and the suture. Needles come with different curvatures, points, and sizes (**Figure 114-9**). The point of the needle extends from the tip and each type is designed to penetrate specific types of tissue. Needles are usually cutting or tapered (**Figure 114-9**). Cutting needles have sharp ends and sharp edges that act as a cutting instrument (**Figure 114-9*A***). The cutting needle is commonly used for tougher tissues (e.g., cutaneous, intradermal, and subcutaneous). Conventional cutting needles have a third cutting edge on the inside concave curvature of the needle. This needle type may be prone to "cutout" of tissue because the inside cutting edge cuts toward the edges of the incision or wound.

Reverse cutting needles are as sharp as the conventional cutting needle except that the third cutting edge is located on the outer convex curvature of the needle (**Figure 114-9*B***). Reverse cutting needles have more strength than similar-sized conventional cutting needles. The danger of tissue "cutout" is greatly reduced. The hole left by the needle leaves a wide wall of tissue against which the suture is to be tied.

Taper point needles have a pointed end (**Figure 114-9*C***). The rest of the needle is a smooth, rounded tube with no cutting edges. This type of needle is commonly used in surgery to close tissues with minimal trauma. It is used for all tissues except skin.

Two other types of needles are often available but not used in the Emergency Department. The blunt point needle has a smooth tip and tapered body (**Figure 114-9*D***). It is used for suturing friable tissue and blunt dissection. The taper cut needle has a cutting tip and a tapered body (**Figure 114-9*E***). It is a combination of the tapered point and cutting needle. It is used to place sutures through tough tissues. Numerous other needles are available, as are modifications

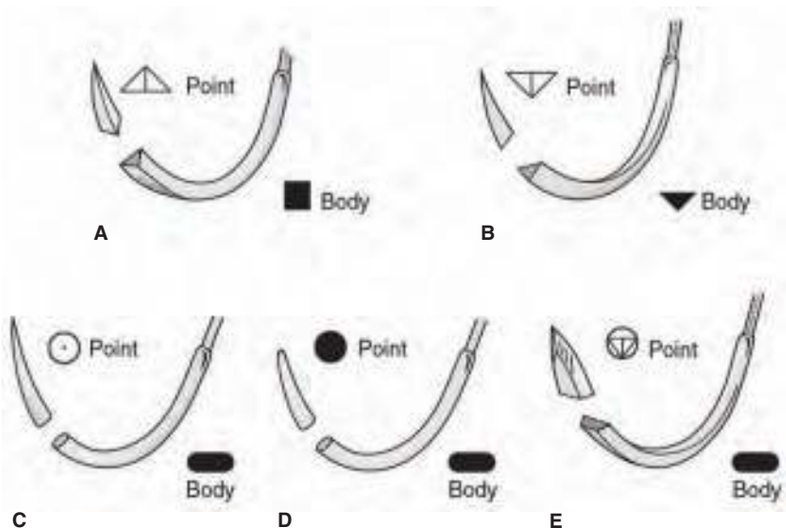


FIGURE 114-9. Common types of suture needles. ***A.*** The cutting needle. ***B.*** The reverse cutting needle. ***C.*** The taper point needle. ***D.*** The blunt point needle. ***E.*** The taper cut needle.

of the five basic needle types. These needles are used by Surgeons for specialized tissues.

Always keep some general principles in mind when suturing. **Pull needles through tissue using a needle driver and never a hemostat**. A hemostat or other clamp can damage the needle. Avoid injury to yourself and others. **Keep all open needles in a place where they will not injure you or your assistant**. **Account for and discard all suture needles in a "sharps" container.** Following these steps will dramatically decrease the chance for a needle-stick injury.

NEEDLE DRIVERS AND HANDLING SUTURES

Always use a needle driver when suturing. The use of a hemostat or other type of "clamp" can damage the needle and cause it to bend or break in the tissue. Needle drivers are generally made of steel with a jaw designed to hold the needle securely without damaging it. They come in numerous sizes and shapes. Choose a needle driver that is an appropriately sized for the needle that is to be grasped. A 4.5 to 6 inch long needle driver is appropriate for Emergency Department use. Grasp and remove a clean needle from its package with the needle driver. Securely grasp the proximal one-third to one-half of the needle with the needle driver (**Figure 114-10*A***). **Do not grasp the distal one-third of the needle**. This can damage its cutting surfaces. **Always use the tips of the needle driver to grasp the needle** (**Figure 114-10*B***). Grasping a needle with the base of the jaws may damage the needle.

Use the needle driver when pushing the needle through the tissue to place a suture (**Figure 114-10*C***). **Apply the force in a direction following the curve of the needle. This will require pronation and supination of the forearm. Do not twist or force the needle to push the point through the tissue and out the other side**. Use a larger needle if the first one is too short or too small. **Do not use a needle that has become dull and difficult to pass through the tissue**. Obtain a new needle and continue the procedure. Grasp the distal tip of the needle with a needle driver when it emerges from the tissues (**Figure 114-10*D***). **Always grasp the needle proximal to its distal third to prevent damage to the cutting edges.**

Always use caution when handing a needle driver armed with a needle to another person. Grasp the needle driver between the thumb, index, and middle fingers (**Figure 114-10*E***). The needle should be facing away to prevent a needle-stick injury. Hand the base of the needle driver to another person. **Do not blindly pass the needle driver. Do not pass the needle driver over to another person without their knowledge of the transfer. Never grasp the distal end of an armed needle driver**.

Typical needle drivers contained within most disposable, commercially available laceration repair trays are not ideal. The suture often snags on the jaws or hinge when performing an instrument tie. Some needle drivers are designed to be snag-free (**Figure 114-11**).

Suturing lacerations can take a significant amount of time. Much of this time is spent tying knots or switching between instruments (i.e., the needle driver and scissors). Some needle drivers are designed to also cut suture. This decreases the total time required to repair a laceration and avoids the constant switching between instruments.

Most laceration repair trays do not contain suture-cutting needle drivers (**Figure 114-11**). A needle driver and scissors can be simultaneously held in the same hand to improve efficiency (**Figure 114-12**). This technique is favored by many but is initially awkward and requires practice to master. Grasp a scissors with the tip pointing ulnarly (**Figure 114-12*A***). Insert the middle finger through the adjacent ring on the handle. Grasp a needle driver in the same hand with the tip pointing radially (**Figure 114-12*A***). Insert the thumb and ring finger through the rings on the handle of the needle driver. Grasp a suture needle with the needle driver. Place a stitch and tie it. Remove the thumb from the ring of the needle driver and place it in the open ring of the scissors. Use the thumb to open and close the scissors. Cut off the excess suture (**Figure 114-12*B***). Place

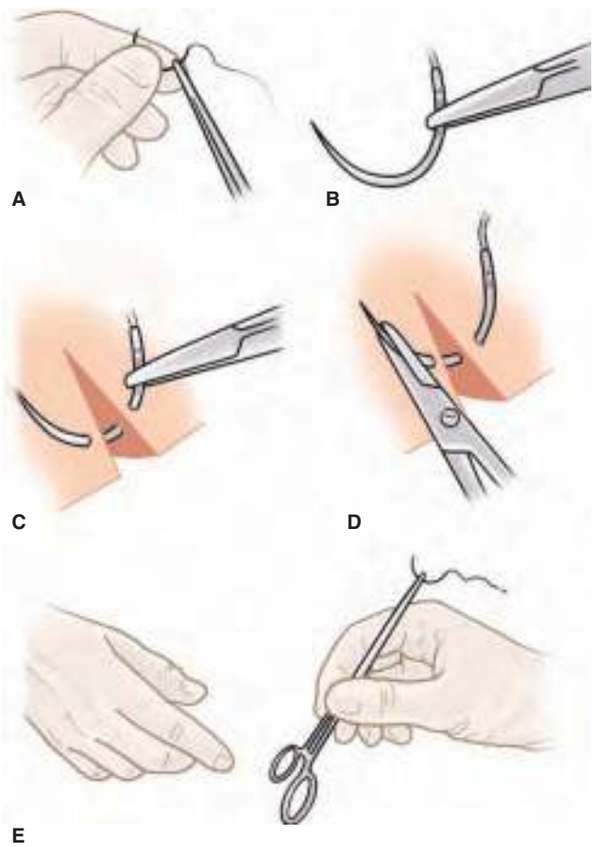
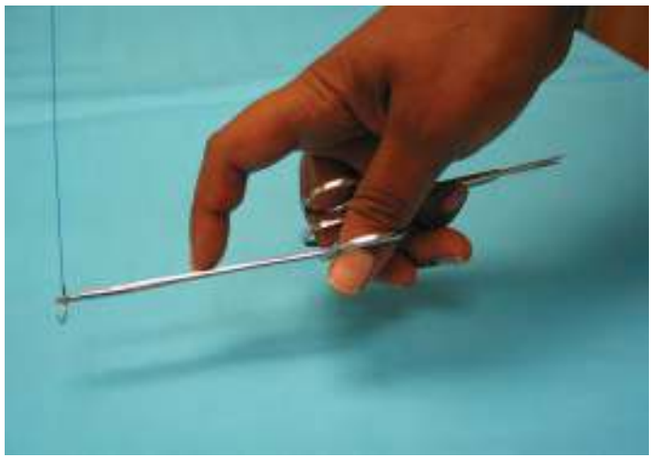
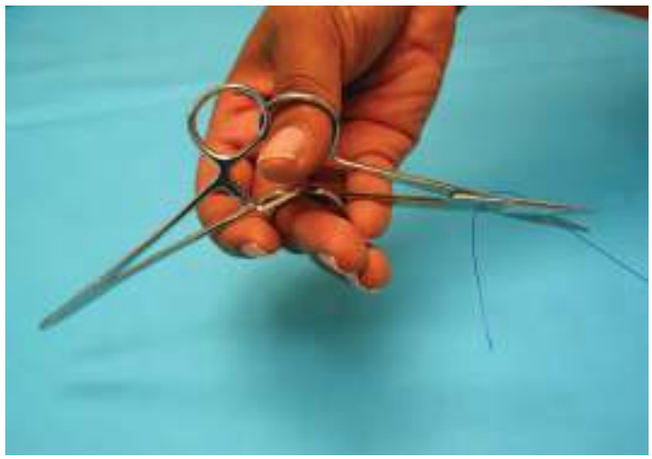


FIGURE 114-10. Using a needle driver. ***A.*** Grasp the proximal one-third to one-half of the needle. ***B.*** Always use the tips of the jaws to grasp the needle. ***C.*** Drive the needle through the tissue following the natural curve of the needle. ***D.*** Grasp the distal needle proximal to the cutting edges. ***E.*** Correct method to pass a needle driver armed with a needle.

FIGURE 114-11. Examples of snag-free needle drivers. From left to right: The Centurion SnagFree (Centurion Healthcare Products, Howell, MI), the SutureCut needle driver (SutureCut LLC, Lexington, KY), and the Olsen-Hegar needle driver (Henry Schein Inc., Port Washington, NY). The SutureCut and Olsen-Hegar needle drivers have a built-in scissors for suture cutting.



A



B

FIGURE 114-12. A one-handed method to simultaneously hold a needle driver and scissors. ***A.*** Placing a stitch. ***B.*** Cutting the suture.

the thumb back into the ring of the needle driver and place the next stitch. Repeat this process until the laceration is closed.

The Alshemari Needle Driver (Alshemari Instruments, Charlotte, NC) holds the needle perpendicular to its length (**Figure 114-13**). This is unlike standard needle drivers. This needle orientation allows for a more comfortable approach and uses vertical, not elliptical, motions to place and tie sutures. The needle driver is used with the hand neutral. The needle driver was designed for otolaryngology procedures in the mouth but can be used anywhere.

WOUND CLOSURE

The goal of wound closure is approximation of the skin under minimal tension while achieving eversion of the wound edges (Chapter 116). Wound eversion slightly raises the wound edges to keep the epidermal cells from migrating into the dermal layers and leaves a flat scar (**Figure 114-14**). **Place stitches closely enough to approximate wound edges but not so tight as to cause tissue necrosis.** The time from the injury to presentation and the mechanism of injury will indicate whether the laceration mandates delayed closure instead of primary closure and whether tetanus prophylaxis is required. Consider high-risk wounds for delayed closure except in patients who are immunocompromised or taking immunosuppressive therapy.

SINGLE-LAYER VERSUS MULTILAYER CLOSURE

The greatest strength of the skin, and of the wound, is contained within the dermis. The narrower the scar, the better will be the coaptation of the dermal edges. The best results occur when the entire depth of the dermis is accurately approximated to the entire depth of the opposite dermis. Dermal closure is best performed with synthetic monofilament absorbable suture that requires enzymatic degradation (e.g., Vicryl). Chromic or plain catgut suture dissolves much more rapidly by means of hydrolysis.

Close the wound in multiple layers to better approximate the edges and improve cosmesis. Close the wound with a minimal number of stitches in a single layer if the goal is a functional result. **Do not suture through fat and muscle.** Fat has no tensile strength. Stitches placed tightly in fat can cause ischemia and necrosis in the wound and increase the risk of a wound infection. Muscle fibers do not support suture. Muscle is best treated by repair of the overlying fascia, immobilization to prevent motion, and natural coaptation of the muscle fibers.

Wounds may cross tissue planes to open them and create potential pockets or dead spaces. Elimination of the dead space has been advocated in the past to decrease the probability of this area becoming a nidus for infection. This once traditional practice of obliteration of dead space to avoid infection of a nonvascularized space or to prevent hematoma formation is now considered controversial. Animal models have found the incidence of infected wounds to be consistently proportional to the number of suture layers.[74,75] Leaving dead space open resulted in lower rates of infection than obliterating

FIGURE 114-13. The Alshemari needle driver (Alshemari Institute, Charlotte, NC).

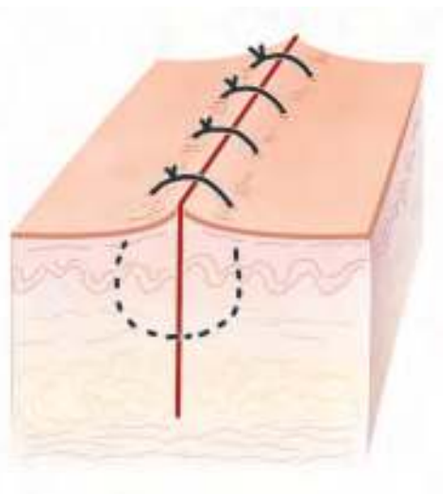


FIGURE 114-14. Eversion of the wound edge signifies proper suture placement and knot tension.

it with sutures.[74,75] Studies in 1994 and 1996 concluded that buried absorbable sutures increase the infection rate, increase the degree of inflammation in contaminated wounds, and do not significantly increase the degree of inflammation in noncontaminated wounds.[76] Stitches placed in fat contribute no strength to the repair and fail to prevent hematoma formation and infection. **Deep absorbable stitches may be placed to repair fascia, muscles, or periosteum. Deep absorbable stitches minimize tension on skin stitches. Use only enough subcutaneous stitches to restore anatomic and functional integrity.** Leaving a potential space may be preferable to attempting to obliterate it.

STERILE GLOVES

It is a common practice to wear sterile gloves when repairing a laceration. The advantages of sterile gloves include a better fit, improved dexterity, and improved tactile sensitivity. The use of sterile gloves for laceration repair costs significantly more than using nonsterile, clean gloves from a box. Boxed, clean, nonsterile, and powder-free examination gloves can be used for uncomplicated wound repair in immunocompetent patients. No clinically important differences in infection rates have been found when comparing sterile gloves to clean gloves in this setting.[77-79]

The use of clean gloves from a box is not always ideal. Clean gloves come in a limited number of sizes (i.e., extra-small, small, medium, large, and extra-large). The fit and feel of clean gloves may not be as comfortable for the Emergency Physician. Clean gloves may have more manufacturing defects when compared to sterile gloves.[80] These defects can result in the loss of personnel protection and the potential to contaminate the wound. Others have shown clean gloves to have comparable quality to sterile gloves.[81-83] A box of clean gloves that has become wet can harbor mold.[84] The decision to wear clean versus sterile gloves is up to each Emergency Physician.

WOUND CLOSURE PROCEDURE

Thoroughly clean any blood, debris, and dirt from the skin. Scrub the skin surrounding the wound with an antiseptic skin cleanser (e.g., povidone iodine or chlorhexidine). Anesthetize the wound with a 27 to 30 gauge hypodermic needle and local anesthetic solution. Irrigate the wound with normal saline. Use a mask with a face shield to prevent exposure to the patient's blood and tissue fluid. Debride and undermine the wound as necessary. Irrigate the wound again to remove exposed debris and devitalized tissue. Repair the wound with sutures or pack it with saline-soaked fine-mesh gauze for delayed closure. Clean the repaired wound with normal saline and apply a dressing for comfort and protection. Consider the application of a splint for wounds across joints or muscle lacerations.

Write a procedure note describing the sterile preparation of the wound, the type and volume of anesthesia administered, the type and number of stitches used in the repair, the layers repaired, the type of repair (i.e., interrupted versus continuous), and how the procedure was tolerated by the patient. Note any complications.

AFTERCARE

Wound care has become a specialty involving sophisticated research in many areas, including dressings and the environment in which wounds heal best. Optimal growth of fibroblasts in tissue culture occurs at low partial pressures of oxygen (e.g., 5 to 10 mmHg). Epidermal cell growth is inhibited at oxygen levels higher than surrounding air. Hydrocolloid dressings can maintain an environment of low oxygen tension independent of the underlying disease process.[85] A wound surface will be cleansed with saline after laceration repair to remove any residual blood and debris before a dressing is applied. The application of an occlusive dressing has been shown to increase the rate of wound healing by approximately 40%, keeps bacteria out of the wound, and prevents environmental trauma. A similar infection rate was found for wounds that were closed by primary intention with and without a dressing.[86] A dressing is generally recommended in wounds left to heal by secondary intention. There is not enough evidence to recommend one dressing over another.[87]

Dressings, regardless of the type used, should produce a moist wound that is free of foreign material, infection, and toxic chemicals. Layered dressings of nonadherent gauze (e.g., Xeroform) can be covered with dry gauze for large sutured lacerations and abrasions. This dressing draws exudate into a layer that can be replaced without disturbing the underlying wound. Shear wounds or hematomas may require gauze that is fluffed and formed into a pressure dressing. Dressings of antibiotic ointment with a standard adhesive bandage (e.g., Band-Aid) provide adequate healing and protection for smaller repaired lacerations. The application of topical antibiotics to the suture line after wound closure may help protect against exogenous bacterial contamination. A Cochrane database systematic review of surgical incisions has shown that topical antibiotic application may reduce wound infection at surgical sites relative to antiseptics or no antibiotics.[87] The preferred topical antibiotic is not clear in this review. There is no evidence that topical antibiotics are harmful and are generally recommended.[88]

DISCHARGE INSTRUCTIONS

Evaluate high-risk wounds (e.g., animal bites, human bites, hand wounds, heavily contaminated wounds, and wounds that require prophylactic antibiotic coverage) within 24 hours. Make patients aware, orally and in writing, that up to one in 10 patients develop a wound infection that can be treated with an oral antibiotic. Puncture wounds are considered high-risk injuries that can result in bone infections and follow-up instructions are essential. Patients should be instructed to immediately return to the Emergency Department or their Primary Physician if a wound becomes red or has a discharge, if redness or red streaks are emanating from the wound, or if they develop a fever. Getting sutures wet in the first 48 hours does not affect the infection rate or tensile strength.[89] Inform the patient that it is alright to wet the stitches but not soak them while the wound is healing.

Discharge instructions should be simple and precise with important points emphasized and reiterated. Patients have been shown to have knowledge deficits regarding home care and return instructions.[90]

Explain briefly the progression of healing. The new scar's appearance is usually worst at 3 to 5 weeks. Most scars remodel within 6 to 12 months. Postpone any revision of the wound for at least 6 to 12 months from the time of injury.

STITCH REMOVAL

The length of time that the stitches remain in place depends on the location of the wound, the amount of tension on the wound, and the healing time of the involved tissue. General recommendations are listed in **Table 114-5**. **Appropriate and timely removal of stitches minimizes scarring.** Full-thickness stitches can be left in place for 2 or more weeks without risk of suture-track formation in areas where sebaceous glands and other adnexal structures are not present (e.g., palmar and plantar surfaces). Leaving stitches in place too long results in epithelialization of the suture tracts, larger scars, and possible infections. Suture removal kits are commercially available. They typically contain a metal or plastic forceps, a scissors,

TABLE 114-5 Suture Removal Recommendations

Location	Days
Back	10–14
Buttocks	10–14
Chest	7–10
Delayed closure	8–12
Foot	10–14
Face	3–4 (child), 3–5 (adult)
Hand	10–14
Legs	8–10
Neck	2–3 (child), 3–4 (adult)
Overlying joints	10–14
Retention sutures	14–30
Upper extremity	7–10

and a few gauze squares. These kits are inexpensive, disposable, and intended for single-patient use.

Remove stitches using aseptic and sterile technique. Clean the wound with saline. Apply hydrogen peroxide to remove any dried blood and serum encrusted around the suture. Grasp the suture strand at the knot with forceps (**Figure 114-15**). Lift the knot off the skin. **Cut the one strand of the suture as close to the skin as possible with a scissors and where the suture enters the skin** (**Figure 114-15**). This will avoid drawing contaminated suture through the depth of the wound. Stitches that are close together, small, or tight may require a #11 scalpel blade to cut them rather than a scissors. Gently pull the suture strand out of the tissue with the forceps and across the wound. Pulling a suture out and away from the wound may result in the wound edges opening (i.e., dehiscing). **Remove one to three stitches and ensure that the wound edges do not dehisce.** Remove the remaining stitches if no dehiscence occurs. Apply skin adhesive strips (e.g., Steri-strips) across the wound to provide support. **Leave the stitches in for a few more days if there is any doubt about adequate tensile strength of the wound and possible dehiscence.** There are few data regarding the effect of advanced age and steroid use on exact timing of removal. Conservative management suggests leaving stitches in these patients longer than in potentially faster healing groups. There is evidence that a select group of patients (e.g., over 19, immunocompetent, simple lacerations) may be willing and capable of removing their own stitches.[91]

MANAGEMENT OF PUNCTURE WOUNDS

Puncture wounds are considered higher risk for infection than simple lacerations. They should be allowed to heal by secondary intention if small or delayed and primary intention if closure is required because they are large or for cosmetic reasons.[92] Local cleansing is the initial step in management. High-pressure irrigation, coring, and probing are generally not recommended.

Infection is most frequently due to *Staphylococcus aureus, Staphylococcus epidermidis*, or streptococcal species. Treatment should be reserved for immunocompromised hosts, dirty wounds, or infected wounds.[93] Puncture wounds of the foot are of special concern due to the risk of *Pseudomonas aeruginosa* infection, particularly with wounds through athletic shoes. A tender wound that is not infected may indicate that there may be a retained foreign body. Persistent infection from a plantar wound suggests an underlying infection and possibly osteomyelitis that requires a further work-up and treatment with antibiotics that may include fluoroquinolones.[94]

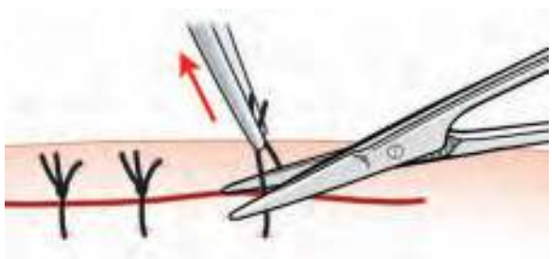


FIGURE 114-15. Stitch removal.

WOUND HEALING IN THE PEDIATRIC PATIENT

Pediatric patients less than 15 years of age experience infection rates of less than 1% for clean surgical wounds.[12] This is less than that seen in adults. Children will sometimes require sedation to make painful or difficult procedures possible. Safe and effective procedural sedation (Chapter 159) for patient comfort or cooperation can facilitate or expedite medical care. Undermining is not useful in most pediatric wounds as they do not usually require advancement of skin over a significant tissue defect. Scalp lacerations account for 30% of pediatric lacerations. Scalp lacerations are well suited for single-layer repair with staples. Cosmetic results are comparable with those of sutured repairs, with no differences in complication and infection rates. Laceration repair with staples is faster, less expensive and can be implanted rapidly and accurately in a moving child.[95]

ALTERNATIVE CLOSURES

Alternative methods of wound closure include skin closure tapes, staples, and tissue adhesives. These are mentioned briefly below. A more complete discussion can be found in Chapters 116 and 117.

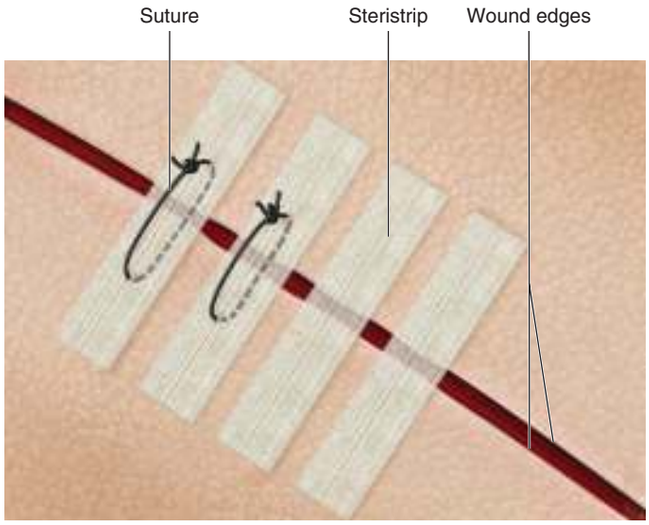
SKIN CLOSURE TAPES

Skin closure tapes are adhesive strips used to approximate wound edges. Adhesive-backed long and narrow strips are used for approximating the edges of lacerations with or without staples or sutures and for closing the skin following many operative procedures. The most common type is the Steri-Strip. Skin closure tapes are felt to develop and increase wound tensile strength faster than sutured wounds because uniformly orienting collagen fibers apply equal stress across the wound. Skin closure tapes are porous which allows for good air inflow and the escape of water vapor during the healing process. Strips are placed perpendicular to the wound in conjunction with an adhesive (e.g., Mastisol or tincture of benzoin). **Take care not to get adhesive in the wound.** There is the option of placing Steri-Strips on thin-skinned individuals and then suturing through both the thin skin and overlying Steri-Strips (**Figure 114-16**). This increases the tensile strength of the skin and minimizes the chance of the stitches ripping through the thin skin.[96-98]

There are numerous advantages and disadvantages to using skin closure tapes. They prevent local skin tension, wound infections, and "railroad track" scarring on the skin. Skin strips maintain equal tension along the length of the laceration if applied correctly. Application reduces time and costs when compared to suturing. The disadvantages include loss of adhesiveness over time, wound dehiscence, difficulty with apposition of skin edges, difficulty in everting the wound edge, and are dependent on the knowledge and skill of the Emergency Physician. Injuries to the skin can occur during placement or removal.

TISSUE ADHESIVES

Tissue adhesives (e.g., butylcyanoacrylate and octylcyanoacrylate) are viable alternatives to standard wound closure techniques using



A

B

FIGURE 114-16. Suturing through Steri-Strips. ***A.*** Artist illustration. (Used with permission from reference 96.) ***B.*** A leg wound with the technique. (Used with permission from reference 98.)

skin tapes, staples, or suture for closure of simple lacerations. It has been clinically proven that there is no difference 1 year after treatment in the cosmetic outcome of wounds repaired with sutures versus those closed with octylcyanoacrylate tissue adhesive.[99] There is no difference in cosmesis when comparing various tissue adhesives for traumatic laceration closure. There is a slight increase in wound dehiscence when comparing tissue adhesives with standard wound closure techniques.[100] The disadvantages are minimal (i.e., cost, proper patient selection, and meticulous technique). They save time when compared to suturing and are easy to use. There is no risk of a needle-stick injury and no need for follow-up visits for removal.

STAPLES

Staple closure is time efficient compared to the suture repair of lacerations.[101] It is primarily used for large wounds that are not on the face, feet, hands, or neck. Stapling is especially useful for closure of incisions in the extremities, hair-bearing skin (i.e., the scalp), and the trunk. The wound edges require manual eversion with forceps prior to placing the staples. Staples are effective for straight wounds and are rapid to place. Staples may cause scarring, skin irritation, and wound contamination and may require removal.

SUMMARY

Expert wound management consists of attention to the details surrounding the wound, gleaning important information concerning the patient's history, and meticulous wound preparation. Attention to the possibility of foreign bodies, underlying injury to anatomic structures of significance, and the risk of subsequent wound infection should all be foremost in the Emergency Physician's mind when managing wounds in the Emergency Department. Educate the patient about the possible outcomes of wounds and lacerations and encourage appropriate follow-up.

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115 Burn Wound Management

Stephen Sandelich and Christopher J. Russo

INTRODUCTION

Injuries secondary to burns represent a significant and prevalent form of injury with major morbidity and mortality. **Prevention must still be in the forefront of burn management.** There has been an increase in the prevalence of a shift to ambulatory or Emergency Department care for burn patients.[1-3] Scald burns make up the majority in children younger than 5 years of age with the balance shifting to flame burns in older children. The rates of hospitalization for burns appear to be decreasing.[4] The annual cost for burn hospitalizations exceeds $200 million per year.[4]

Child abuse must be considered in all pediatric burn patients. Abuse should be suspected in younger patients presenting with burns that are in a bilateral and symmetrical distribution (e.g., a stocking and glove distribution), patterned burns (e.g., a hot grate), or burns on the dorsum of the hands; in cases of delayed medical care; or when there are inconsistent histories.

The Emergency Physician must be familiar with the initial management of burns, the treatment of burns, and the indications for transfer to a regional burn center. This chapter is limited to minor burn management of the skin as an outpatient. It will not cover airway burns, significant burns, fluid and electrolyte management, associated poisoning (e.g., carbon monoxide and cyanide), and inhalation burns.

ANATOMY AND PATHOPHYSIOLOGY

The skin is the largest organ of the body. It consists of the epidermal, dermal, and subcutaneous layers (**Figure 115-1**). The epidermis is the outer layer of stratified squamous epithelium, whereas the dermis is a dense bed of vascular connective tissue. The skin acts as a physical barrier, a sensory organ, and a thermoregulatory organ. The body reacts to a burn with local and systemic responses.

There are three zones of tissue response described after an acute thermal injury.[5] The zone of coagulation occurs at the point of maximum damage. Tissue proteins coagulate and lead to irreversible tissue loss. The next zone is labeled the zone of stasis and is characterized by decreased tissue perfusion. This tissue may be salvaged or completely lost if not properly treated. Surrounding the zone of stasis is a zone of hyperemia. This area sees an increase in tissue perfusion. Tissue in this zone should recover unless there are severe systemic insults (e.g., profound hypotension or sepsis). Severe and major burns can involve a systemic response (e.g., cardiovascular, respiratory, metabolic, and immunologic derangements). Systemic involvement is generally not seen unless a burn is greater than 20% of body surface area (BSA).[6]

Burns are classified by depth, size, and total body surface area (TBSA) involved.[7,8] A superficial or first-degree burn is characterized by dry, blanching, and erythematous skin. This is accompanied by a mild inflammatory response without bulla formation. These burns are usually painful and will heal quickly over the course of 3 to 6 days without scar tissue formation. The outer epithelial layer will peel away from the newly forming layers underneath. It has been compared to a bad sunburn.

Partial-thickness, or second-degree, burns may be either superficial or deep. A superficial partial-thickness burn has destroyed the epidermis but does not penetrate fully through the dermis. A deep partial-thickness burn will extend deeper into the dermis. Superficial burns will form blisters quickly within 24 hours. Edema will be present from increased capillary permeability that occurs from direct thermal injury. These burns are very painful as the nerve endings in the dermal tissue are exposed. They appear erythematous or pink, moist, and blanch with pressure. Deep partial-thickness burns will blister and are often insensate. The burn has destroyed most of the dermal layer and the nerve endings along with it. Deep burns are variable in color (e.g., erythematous to white), appear drier than superficial burns, and do not blanch with pressure.

Full-thickness or third-degree burns have penetrated the entire epidermis and the dermis. They are pale or charred, appear leathery, and are white or black. They are insensate and not painful. There is no capillary refill and the area has red dots from the red blood cells in the capillaries rupturing. The edema will not be as apparent because the tissue has lost much of its elasticity and cannot stretch as readily.[9,10] These burns cannot form new epithelial tissue and will

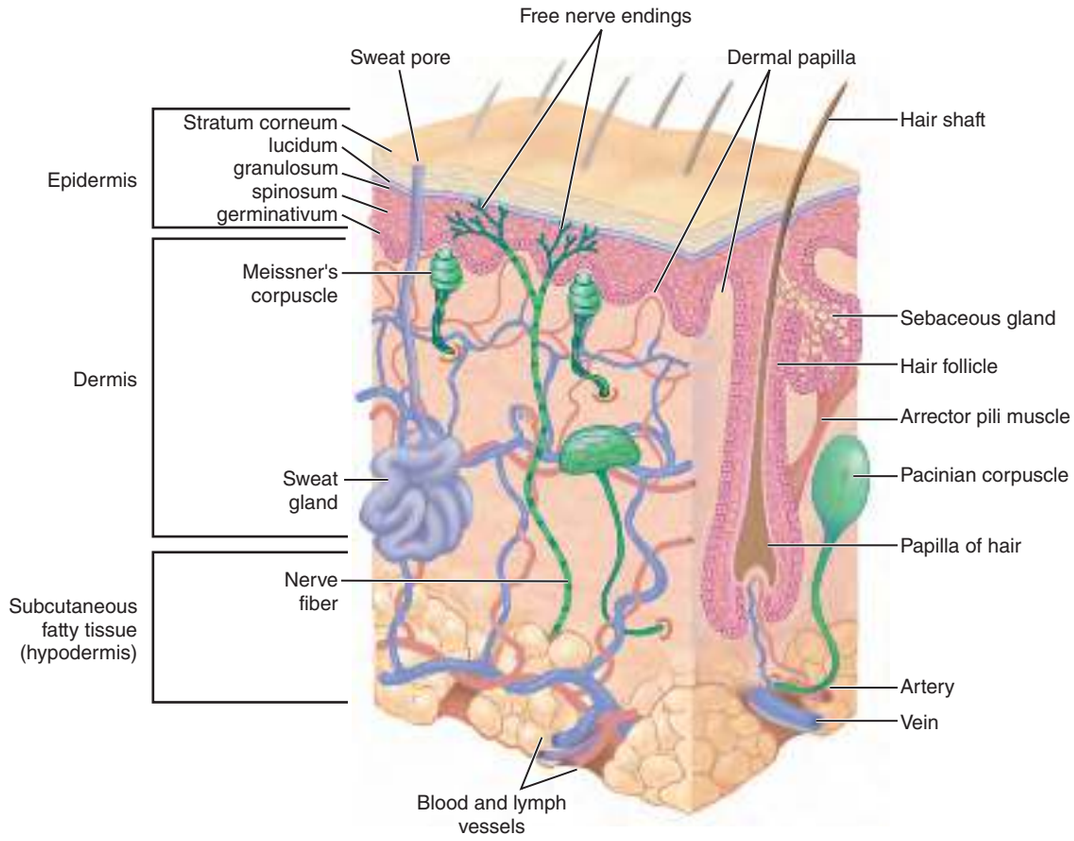


FIGURE 115-1. Cross section of the skin.

most often require skin grafting. It may be difficult to fully classify burns in the acute phase because the full extent of damage may not be apparent until a few days after the original burn.[11]

Accurate estimation of burn size remains important as this can direct management decisions regarding fluid resuscitation and the need for transfer to a burn center.[12] Large burns are often underestimated, resulting in delayed transfer and lack of adequate fluid resuscitation. The two commonly used methods of estimation of burn size are the rule-of-nines (**Figure 115-2**) and the Lund-Browder chart (**Figure 115-3**). The Lund-Browder charts are preferred in children as they account for the relative differences in the body area as the child ages and the proportionately larger heads in children. Smaller burns or burns with patchy areas can also be estimated using the palmar method in which the patient's entire palmar surface is assumed to be equal to approximately 1% TBSA.

INDICATIONS

Minor burns that can be cleaned, dressed, and followed in an outpatient setting include partial-thickness burns less than 5% TBSA in children younger than 10 years old, partial-thickness burns less than 10% TBSA in children older than 10 years old, and full-thickness burns less than 2% TBSA. Burns with areas larger than this should be considered major burns. Consider transfer of more extensive burns to a regional burn center.

CONTRAINDICATIONS

Burns involving the face, hands, perineum, or feet; burns crossing major joint lines; and circumferential burns are considered major burns. Refer these patients to a burn center. **Table 115-1** lists the burns that should be transferred to a burn center.[13] Patients who present with multisystem trauma require stabilization prior to attending to burn care.

EQUIPMENT

- Mild soap
- Warm water
- Topical antibiotics
- Silver impregnated dressing
- Nonadherent dressing (e.g., Adaptic or Xeroform)
- Layered dry gauze (e.g., Karlie)

PATIENT PREPARATION

Explain the procedure to the patient and/or their representative. An informed consent for the care of burns is not required. Obtain an informed consent for any anesthetic technique performed and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. Update the patient's tetanus immune status if required.

Burns are generally exquisitely painful injuries and adequate analgesia should be achieved prior to any painful procedures (e.g., debriding or burn dressing application).[14] Small superficial burns are adequately managed with nonsteroidal anti-inflammatory drugs (NSAIDs) or acetaminophen. The patient will require more aggressive directed pain management with more severe burns. This

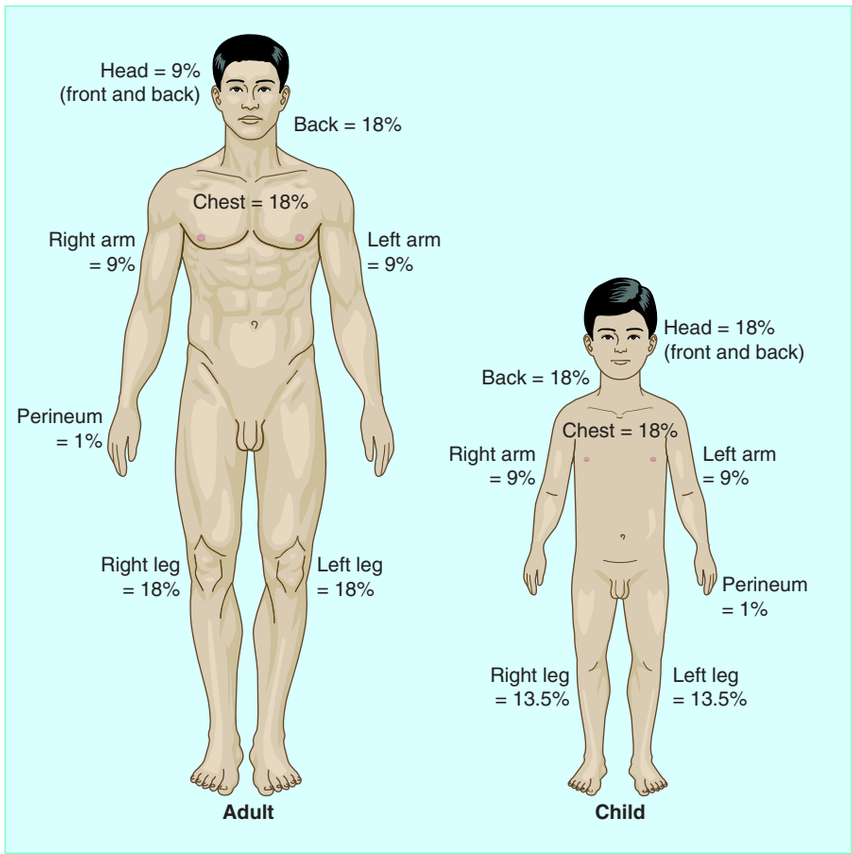


FIGURE 115-2. The rule-of-nines to estimate burn area. (Used with permission from Tauro University.)

can include opioid analgesics (e.g., morphine or fentanyl), nonopioid analgesics, nitrous oxide (Chapter 128), dexmedetomidine, ketamine, or anxiolytics. The patient with significant burns may require procedural sedation and analgesia (Chapter 159) for the initial evaluation and management.

TECHNIQUES

GENERAL MANAGEMENT

Perform an initial assessment. Remove any clothing and jewelry from the affected areas. Remove any rings as these will be difficult to remove once edema occurs. Cool the affected area. Perform cooling within the first 30 minutes after the injury. Early cooling may reduce the ongoing thermal injury and the development of progressive edema. The water should be cool while avoiding ice or ice water.[15,16] Cooling should progress until pain is diminished and not for longer than 5 to 10 minutes. Clean the wounds with a mild antibacterial soap. First-degree burns require no further management. Instruct the patient to continue washing the affected area with mild soap and warm water daily until full healing has occurred.

FLUID RESUSCITATION

Most minor burns treated as an outpatient do not have fluid and electrolyte shifts. Most burns less than 20% BSA do not need fluid resuscitation. Start with crystalloid solutions if fluid is required.[17] The most commonly used formulation is the Parkland formula (**Table 115-2**). Some institutions use the Cincinnati-Shriner or Brooke formula (**Table 115-2**). These fluids represent the volume to be given in 24 hours from the time of the burn and not the clinical presentation. Divide the total volume calculated in half. Administer the first half over the first 8 hours followed by the second half over the next 16 hours. These calculations represent an estimate for resuscitation. They do not include base hydration and other losses. A further discussion is beyond the goals of this chapter.

DEBRIDEMENT

Intact blisters (**Figure 115-4*A***), ruptured blisters, and devitalized tissues represent a nidus for future bacterial infection and should be carefully removed. This is usually a painful procedure and will require some type of pain mitigation strategy. This may include oral analgesics, local or regional nerve blocks (Chapter 156), intravenous regional anesthesia (Chapter 157), nitrous oxide (Chapter 158), or procedural sedation and analgesia (Chapter 159).

Remove the nonviable tissue with gentle traction, scraping, and cutting (**Figures 115-4*B* and 115-4*C***). Blister management is somewhat controversial.[18,19] Remove all ruptured blisters as the dead epithelial layer may increase infection rates and delay wound healing. Burn blisters that have not ruptured may be managed in multiple ways. There is no gold standard to guide practice. One practice leaves the blisters intact in the belief that they represent superficial injury and will heal spontaneously if left undisturbed. The blister protects the wound with its own biological dressing. There is some thought that the burn blister fluid may stimulate the wound-healing process through expression of various growth factors found in the exudate.[20] The opposing camp believes that the

Burn Estimate and Diagram Age and Area

Initial evaluation*

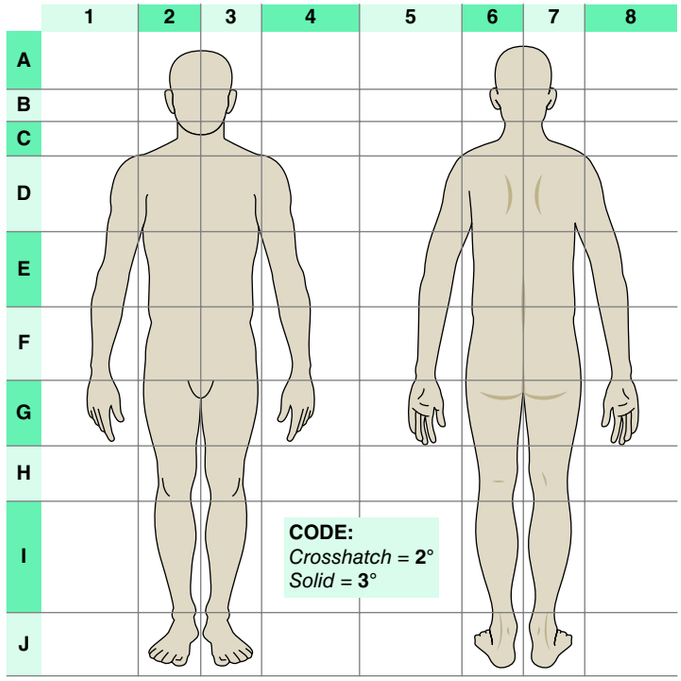
Signature: _____

Date of burn: _____

Date completed: _____

*To be completed by the admitting physician or Licensed Independent Practitioner on admission

This is a working burn estimate diagram only, and is not as accurate as photography.



Area	Birth-1 yr.	1–4 yrs.	5–9 yrs.	10–14 yrs.	15 yrs.	Adult	2°	3°	TOTAL
Head	9	17	13	11	9	7			
Neck	2	2	2	2	2	2			
Anterior trunk	13	13	13	13	13	13			
Posterior trunk	13	13	13	13	13	13			
Right buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Left buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Genitalia	1	1	1	1	1	1			
Right upper arm	4	4	4	4	4	4			
Left upper arm	4	4	4	4	4	4			
Right lower arm	3	3	3	3	3	3			
Left lower arm	3	3	3	3	3	3			
Right hand	2.5	2.5	2.5	2.5	2.5	2.5			
Left hand	2.5	2.5	2.5	2.5	2.5	2.5			
Right thigh	5.5	6.5	8	8.5	9	9.5			
Left thigh	5.5	6.5	8	8.5	9	9.5			
Right lower leg	5	5	5.5	6	6.5	7			
Left lower leg	5	5	5.5	6	6.5	7			
Right foot	3.5	3.5	3.5	3.5	3.5	3.5			
Left foot	3.5	3.5	3.5	3.5	3.5	3.5			
**Only 2° and 3° burns are included in the total TBSA burn percent						TOTAL			

FIGURE 115-3. The Lund-Browder method to estimate burn area. (Used with permission from reference 8.)

blister fluid may depress immunologic functioning of the underlying new epithelial layer, delay total healing time, and provide a culture medium for bacterial growth. Most evidence appears to suggest that best healing results are achieved with leaving blisters intact unless they are large and thin-walled blisters that may rupture during the healing process. **Needle aspiration of a burn blister should generally not be performed as this can increase the risk of infection.** The exception to this is larger thick-walled blisters that are overlying a joint.[9]

TOPICAL ANTIBIOTICS

The decision must be made regarding the need for topical antibiotic coverage. The goal of these dressings is to relieve pain and to promote healing. Superficial burns with an intact dermis have a very small risk for bacterial infection and topical antibiotics are not indicated. These wounds can be effectively managed using a nonperfumed moisturizing cream or aloe vera.[21] Wounds that extend deeper into the dermis are at a higher risk for infection. Apply topical antibiotics before applying the final dressing. The choice of topical antibiotics depends on the patient, the provider, the type of burn, any patient allergies, regional preference, and individual preference.

Silver sulfadiazine (SSD) is one of the most common topical burn treatments available (**Figure 115-4*D***).[22-24] SSD is a nontoxic salt of silver sulfadiazine in a water-based cream. It is painless and has a wide spectrum of antimicrobial action and a long shelf life. It is available as a thick white paste that is applied over the burn and covered with a nonadherent dressing. The SSD dressing can be reapplied up to three times a day until healing occurs. SSD is contraindicated in patients with a sulfa allergy, pregnant women,

TABLE 115-1 The Burn Center Referral Criteria

Burns and concomitant trauma where burns pose a greater risk of morbidity and mortality
Burns in patients who require emotional, rehabilitative, or social care
Burns in patients with preexisting medical problems that could affect mortality
Burns in patients with preexisting medical problems that could complicate management
Burns in patients with preexisting medical problems that could prolong recovery
Burns over major joints
Chemical burns
Children in hospitals without qualified personnel or equipment
Electrical burns
Face burns
Feet burns
Genitalia burns
Hand burns
Inhalation or airway burns
Lightening burns
Partial thickness burns > 10% BSA
Perineal burns
Third-degree burns

Source: Modified from reference 13.

lactating women, and newborns.[25] SSD decreases the amount of bacterial colonization and decreases infection rates. It may prolong wound healing through inhibition of keratinocyte replication and increase the frequency of required wound dressing changes. There are multiple silver-impregnated dressings available for burn care (e.g., Aquacel AG, Mepilex AG, and Acticoat).[24,26] They have been shown to result in faster healing times, decreased pain, decreased dressing changes, and improved patient satisfaction and to reduce overall costs.[27-31]

Some burns are not treated with SSD. Superficial facial wounds are often treated with a clear antibiotic ointment such as bacitracin. The silver preparations can cause cosmetic staining of the skin. Triple antibiotic ointment on deep full-thickness pig skin burns enhanced reepithelization, decreased scar depth, and decreased scar contraction compared to silver preparations.[32] This has not been proven in humans yet. Wounds that are near the eye should be treated with a topical ophthalmic ointment. Systemic prophylactic antibiotics are not required in most burn patients.[33]

WOUND DRESSING

Cover the burn with layered gauze or a nonadherent dressing and secure it (**Figures 115-4*E* and 115-4*F***). No dressings should be applied if the burn is deep or meets burn center transfer criteria (**Table 115-1**). The dressing will need to be removed once the patient reaches the burn center. This will cause the patient more pain and local trauma to the tissues. Cover the burns with dry gauze or a nonadherent dressing and a clean dry sheet. The best outcomes are achieved with a quick transfer to the burn center.

The burn can be covered with ChloraDerm (Entrotech Life Sciences, San Francisco, CA) if the patient is being discharged from the Emergency Department. ChloraDerm was approved for use on burn wounds in early 2016. It is transparent, safe, and can be left on for 7 days. It contains an acid-free chlorhexidine matrix and is cost-effective. It resists the growth of bacteria, fungi, and viruses that can cause infections.

TABLE 115-2 Formulas Commonly Used in Fluid Resuscitation of Burn Patients

Name of formula	Fluid	Total volume (mL in 24 hours)
Parkland	Lactated Ringer's	4 mL/kg × weight (kg) µ %BSA of burns
Cincinnati-Shriner	Lactated Ringer's	(4 mL/kg × weight [kg] × %BSA of burns) + 1500 mL/m^2
Brooke	Lactated Ringer's	1.5 mL/kg × weight (kg) × %BSA of burns
	Colloid	0.5 mL/kg × weight (kg) × %BSA of burns

PEDIATRIC CONSIDERATIONS

Children are often excellent patients for outpatient management of their burns.[34,35] The ability of the parents or caregivers to adequately perform the burn dressing changes must be ascertained. It can be beneficial to select a dressing that can stay in place for 7 days (e.g., Mepilex AG or ChloraDerm) to avoid the parent having to perform a painful dressing change at home.[36,37] **Pay specific attention to pain and anxiety control in the pediatric patient.** Adjuncts to classic pharmacologic treatments (e.g., distraction and child life providers) can significantly improve the initial evaluation and wound dressing.

AFTERCARE

Instruct the patient and their caregivers on the instructions for proper burn dressing change management, pain control, and infection prevention. The primary care physician may be competent to complete adequate follow-up of minor or superficial burns. Follow-up in a certified burn center is more appropriate in deeper burns, burns in younger children, or burns on concerning areas of the body. Superficial burns will heal completely in 3 to 6 days, whereas a superficial partial-thickness burn may take between 7 and 20 days for full healing. Superficial burns do not require dressings and may be managed with daily washing with warm water and gentle soap. Moisturizing lotion and aloe vera may be applied to the burned area to provide comfort. The frequency of dressing changes depends on the type of burn and the type of dressings chosen. Daily changes appear to show no change in morbidity and lead to less pain than multiple changes per day.[22,37] Firm recommendations regarding an optimal number of dressing changes do not exist. Manage these burns as a healing superficial wound with a moisturizing cream or aloe vera once the epithelial layer has been replenished.

COMPLICATIONS

Complications can include inadequate pain control, infections, and/or increased scarring. Most superficial burns will heal quickly and may only need NSAIDs or acetaminophen for adequate pain control. Deeper or more extensive burns may require opiate narcotics to gain adequate pain control. A longer acting analgesic may be needed if the current management is not temporally extending long enough. A dressing requiring less frequent changes may be considered if pain is centered mostly around dressing changes. Anxiety may lead to increased perception of pain in the pediatric population and must be adequately addressed. More than 90% of patients will experience a significant amount of pruritus that can be interpreted as pain, especially in young children. Scheduled antipruritic agents (e.g., diphenhydramine) may be helpful to counteract the pruritus.

Infection is a serious complication. It is a rare complication of most superficial or partial-thickness burns. Infection can greatly delay wound healing or scar formation. Prophylactic oral antibiotic therapy has been shown to be ineffective in preventing infections in the burn patient. A local cellulitis may develop as the patient's normal skin flora recolonizes the area. Cellulitis can be effectively managed as an outpatient with a first-generation cephalosporin if

FIGURE 115-4. Burn wound management. ***A.*** Intact blisters. ***B.*** Blister removal. ***C.*** The blister has been removed. ***D.*** Silver sulfadiazine has been applied to the area. ***E.*** Gauze is placed over the burnt area. ***F.*** The rest of the dressing has been applied. (Used with permission from Roberts JR, Roberts M: Feel the burn. *Emerg Med News*. February 2014.)

there are no signs or symptoms of systemic spread of the infection (e.g., fever, chills, nausea, or emesis).

SUMMARY

Most burn patients can be effectively managed as an outpatient. Most minor burns will heal well with minimal care or intervention. Patients with deep burns, burns that cover a large surface area, burns of the hands or feet, or burns that cross major joint lines should be transferred to a burn center for definitive management (**Table 115-1**). Most superficial burns can be treated with moisturizer and gentle cleansing. Deeper burns should be debrided and a topical antimicrobial dressing applied. Monitor healing for signs of developing infection. Management of pain and pruritus in these patients remains important.

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116 Basic Wound Closure Techniques

Eric F. Reichman

INTRODUCTION

Wound management is crucial to the practice of Emergency Medicine. Emergency Physicians routinely care for wounds ranging from simple lacerations to complex injuries in the trauma patient.[1-6] Wound repair is always secondary to the evaluation and stabilization of any life-threatening and limb-threatening emergencies. However, patients are often legitimately concerned about the outcome of wounds and lacerations. There are several basic suture principles that will help to provide optimal wound healing and ensure a more than acceptable cosmetic result. The previous chapter outlines the essential principles of wound management. This chapter describes the basic methods used to close wounds.

SUTURES

The choice of suture materials is important in wound closure. Sutures are made of a wide variety of materials, both natural and synthetic. Natural substances include gut made from sheep and beef, cotton, and silk. **Natural substance sutures cause more tissue reactions and scarring, which limits their use.** Cotton sutures are not discussed, as they are no longer used in clinical practice. Synthetic sutures can be made of nylon, polyethylene (e.g., Dacron), polyglactin (e.g., Vicryl), polypropylene (e.g., Surgilene and Prolene), polyglycolic acid (e.g., Dexon), poliglecaprone (e.g., Monocryl), polydioxanone (e.g., PDS), polyglyconate (e.g., Maxon), and metal.[6] Metal sutures are used in the Operating Room and not in the Emergency Department as they are difficult to handle, prone to breakage, and indicated in only a few situations. Synthetic sutures tend to have a problem with "memory." That is, they tend to retain the shape of their packaging. This can make it difficult to manipulate the suture during wound closure.

Sutures are constructed as monofilaments or polyfilaments. Polyfilament fibers consist of multiple single filaments braided together to form one suture. They are easier to handle than monofilament sutures, as they tend to be more pliable. Polyfilament sutures have better knot security and therefore reduce the incidence of knot slippage. However, they can be associated with a higher incidence of infection than monofilament sutures. They allow bacteria to migrate (or wick) between the strands of the suture located at the skin surface and into the wound.

Select the smallest diameter suture that can adequately hold the tissue edges together in order to reduce tissue damage and scarring. The largest suture material available is size #5. The suture sizes decrease to zero (i.e., #4, #3, #2, #1, #0) and then are followed by #00 (i.e., 2–0), #000 (i.e., 3–0), and #0000 (i.e., 4–0) in decreasing size. The smallest suture commonly used in the Emergency Department is 6–0 for facial lacerations, nail bed lacerations, and lacerations in cosmetically sensitive areas. The tensile strength of sutures is related to their size. The tensile strength of suture increases as the size increases. For example, 4–0 is stronger than 5–0.

The other main category of suture classification is absorbable versus nonabsorbable. In the past, absorbable sutures were primarily used to close the subcutaneous layers of a wound. More recently, absorbable sutures have also been used for skin closure. Nonabsorbable sutures are primarily used for skin closure.

ABSORBABLE SUTURE MATERIALS

Absorbable sutures are degraded by the body and do not require removal. They usually do not maintain their tensile strength for

TABLE 116-1 Absorbable Suture Materials

Suture type	Source	Tensile strength	Tissue reaction	Knot security	Absorption
Plain surgical gut	Beef or sheep collagen	Poor	Moderate	Poor	1–2 weeks
Chromic surgical gut	Beef or sheep collagen	Poor	Moderate	Fair	2–3 weeks
PolySyn FA	Polyglycolic acid	50% remains at 5 days; 0% at 14 days	Minimal	Good	7 weeks
Monocryl	Polylicaprone 25	20% remains by 3 weeks	Minimal	Good	3 months
Caprosyn	Polyglytone 6211	60% remains at 5 days; 30% at 10 days	Minimal	Good	2 months
Coated Vicryl	Polyglycolic 910, polyglactin 370, and calcium stearate	65% remains at 2 weeks; 40% at 3 weeks	Minimal	Fair	3–6 months
Dexon	Polyglycolic acid	50% remains at 4 weeks	Minimal	Good	3-4 months
Vicryl Rapide	Polyglactin 910	50% remains in 5-6 days	Minimal	Good	42 days
PDS polydioxanone	Polyester polymer	70% remains at 2 weeks; 50% at 4 weeks	Slight	Poor	6 months
Maxon	Polyglyconate	50% remains at 7 weeks	Slight	Fair	6 months
V-Loc 90	Glycolide, diaxanone, and trimethylene carbonate	90% remains at 7 days, 25% at 14 days	Minimal	N/A	90–110 days
V-Loc 180	Glycolic acid and trimethylene carbonate	65% remains at 21 days	Minimal	N/A	180 days

longer than 60 days. Body enzymes dissolve the absorbable sutures with the aid of an inflammatory reaction. The rate of absorption of the sutures varies based upon the tissue where it is placed, the composition of the suture, and the size of the suture. Absorbable sutures placed in mucous membranes absorb faster than those placed in muscle tissue or fascia. Smaller sizes of suture dissolve faster than larger sizes.

There are several types of absorbable sutures, both natural and synthetic (**Table 116-1**). The most commonly used absorbable sutures in the Emergency Department are plain gut, chromic gut, polyglycolic acid (e.g., Dexon), polyglactin (e.g., Vicryl), and Vicryl Rapide. Plain gut and chromic gut are both natural forms of absorbable sutures. They are made from the intestines of sheep and cattle. Gut is a tissue irritant and can cause a substantial tissue reaction while it is being absorbed and degraded by the body. Chromic gut is plain gut that has been soaked in chromic acid salts. This process helps to extend the half-life of the suture and allows it to maintain its tensile strength longer than plain gut. Chromic gut may retain its tensile strength for 2 to 3 weeks, while plain gut retains its tensile strength for 1 to 2 weeks. Both types of gut are packaged wet in order to keep them from drying out and becoming too stiff.

Synthetic absorbable sutures, such as Dexon and Vicryl, are typically used more often than natural absorbable sutures in the Emergency Department. They are degraded by the body more slowly than natural fibers and can therefore help maintain the strength of the wound longer. Vicryl and Dexon maintain their tensile strength at 80 days and 120 days, respectively. They cause less reaction in the tissues as they break down when compared to natural absorbable sutures.

Absorbable sutures have gained some popularity for use in skin closure.[7-12] Absorbable sutures have been shown to yield equal results in their rate of dehiscence, rate of infection, and cosmesis when compared to nonabsorbable sutures.[7] Absorbable sutures have the added benefit for the patient of not having to return to have their sutures removed. Vicryl Rapide is a newer form of Vicryl that is especially suited for this purpose. This type of suture is rapidly absorbed. They begin to fall off in 7 ro 10 days as the wound heals. This can be especially useful for children in whom suture removal can be difficult, for lacerations under casts, or if a patient will not be able to follow up due to travel.

NONABSORBABLE SUTURE MATERIALS

Nonabsorbable sutures are not degraded by the body and must be removed. They maintain their tensile strength for longer than 60 days. They are composed of monofilament or polyfilament strands of organic, synthetic, or metal fibers (**Table 116-2**). Nonabsorbable sutures generally have greater tensile strength and lower tissue reactivity than absorbable sutures. They are used in a variety of applications including skin closure. Nonabsorbable sutures can be used within a body cavity and subcutaneously, where they will eventually become encapsulated in connective tissue.

Nonabsorbable sutures can be classified as organic, synthetic, and wire. Organic sutures include those made of cotton or silk. Cotton is the oldest of the nonabsorbable sutures. It is not discussed here as cotton sutures are no longer used in general medical practice. Silk is a polyfilament suture that has limited use in the practice of Emergency Medicine. There are several advantages to silk suture

TABLE 116-2 Nonabsorbable Suture Materials

Suture type	Source	Tensile strength	Tissue reaction	Knot security	Absorption
Silk (braided)	Organic protein	Gradual loss by progressive degradation	High	Good	Gradual encapsulation by connective tissue
Ethilon	Polyamide (nylon)	Progressive hydrolysis may result in gradual loss of tensile strength	Minimal	Fair	Gradual encapsulation by connective tissue
Nurolon	Polyamide (nylon)	Progressive hydrolysis may result in gradual loss of tensile strength	Minimal	Fair	Gradual encapsulation by connective tissue
Prolene	Polyamide (nylon)	Not subject to degradation	Minimal	Poor	Nonabsorbable
Mersilene	Polyester	No significant change occurs	Minimal	Good	Gradual encapsulation by connective tissue
Ethibond	Coated polyester	No significant change occurs	Minimal	Good	Gradual encapsulation by connective tissue
V-Loc PBT	Polybutanester	Indefinite, high	Minimal	N/A	Nonabsorbable
Tissue adhesive	Synthetic chemicals	Low	Minimal	N/A	Nonabsorbable, falls off
Stainless steel	Stainless steel	Indefinite, high	Minimal	Good	Nonabsorbable
Staples	Inert metals	Indefinite, high	Minimal	N/A	Nonabsorbable

material. Its pliability makes it very easy to handle. It holds knots better than other types of suture. However, as with all natural and/or polyfilament sutures, it has a greater tendency to cause wound infections. The polyfilament braids can provide a place for bacteria to lodge. Silk suture may actually protect the bacteria from attack by the body's defenses if the wound becomes infected. The primary use of silk sutures in the Emergency Department is for the repair of lip, oral cavity, and tongue lacerations.

Synthetic nonabsorbable sutures are available in monofilament and polyfilament forms. Commonly used synthetic sutures include nylon, polypropylene, polybutester, and Dacron. Nylon, polypropylene, and polybutester are monofilament synthetic sutures. Dacron is a polyfilament synthetic suture. The synthetic nonabsorbable sutures have several advantages over the natural nonabsorbable sutures. They are less reactive in tissues, generally stronger than the natural sutures, and retain their tensile strength over many years.

Nylon (e.g., Ethilon and Dermalon) is the most common nonabsorbable suture used in the Emergency Department. It is a monofilament suture, is inert, and does not tend to harbor bacteria. It is primarily used for skin closure. Nylon has good tensile strength and minimal tissue reactivity. However, nylon is difficult to handle and difficult to tie. It requires more knots to achieve good knot security than other types of suture. This is primarily due to the tendency of the suture to return to its packaged shape. This tendency is also known as "memory." Because the knot can unravel or slip, it is important to place at least four or five knots when using nylon suture.

Polypropylene and polybutester are less commonly used synthetic nonabsorbable sutures. Polypropylene (e.g., Prolene) is stronger but more difficult to work with than nylon because it has greater memory. Polybutester (e.g., Novafil) is a newer suture in this category. It is stronger than the other monofilaments and does not have significant memory. Therefore, it is easier to work with than the other monofilament synthetic sutures.

EQUIPMENT

- Needle drivers, 4.5 and 6.0 inch
- Skin hooks
- Scalpel blades (#10, #11, #15)
- Scalpel handles
- Iris scissors, straight 4 inch and curved 4 inch
- Suture scissors, 6 inch
- Forceps, toothed Adson
- Metzenbaum scissors, curved 6 inch
- Hemostats, straight 6 inch, and curved mosquitoes
- Suture material
- Skin closure tapes
- Benzoin solution, swabs or spray
- Gum mastic (e.g., Mastisol)
- Tissue adhesive
- Tissue adhesive forceps
- Skin stapler
- Staple remover
- Gauze, 4×4 squares

Much of the above equipment can be purchased in single-use, sterile, and disposable suture kits from several commercial manufacturers (**Figure 116-1*A***). These kits tend to be expensive and occasionally have a limited amount of equipment. Many hospitals package and sterilize their own wound repair kits (**Figure 116-1*B***). This decreases the cost, as the equipment can be repeatedly sterilized and reused. It also allows the kits to contain a wide variety of instruments for multiple situations (e.g., minor laceration, large laceration, and plastics closure).

Needle drivers come in a variety of sizes. A 4.5 inch needle driver can be used comfortably with most types of needles. A 6 inch needle driver may be required if large needles are used to close a wound. Hold the needle driver with the fingertips to provide greater flexibility. The fingers can also be placed through the finger holes, but this is not as efficient when closing a wound. Grasp the needle one-third of the way from the swag (distal) end with the tip of the needle driver.

The skin must be grasped and manipulated during wound repair to allow for proper suture placement. Forceps are most commonly used to grasp and manipulate the skin. **Smooth (i.e., nontoothed) forceps should never be used to grasp skin.** They require the application of a large amount of force to grasp the tissue. This can crush tissue very easily. An Adson forceps is the forceps of choice. It has fine teeth that grasp tissue securely with minimal force.

A skin hook is a sharp, pointed instrument that is inserted into the wound edge and grasps the tissue from the undersurface. It produces a small puncture wound in the subcutaneous tissues and does not penetrate the skin surface. Skin hooks are preferable to forceps, as they do not crush tissues. A skin hook is awkward to use at first. With proper instruction and experience, the Emergency Physician will most certainly prefer a skin hook to forceps.

A

B

FIGURE 116-1. The equipment required for basic wound closure techniques. ***A.*** The contents of a disposable and commercially available wound closure kit. ***B.*** A hospital packaged kit with reusable instruments.

Several types of scissors are required for proper wound closure. Iris scissors have sharp, delicate tips for making precise cuts in tissue. They should not be used to cut suture material, as this rapidly dulls and nicks the blades. Suture scissors have one blunt tip and one pointed tip. Both blades of the suture scissors are sharp. Suture scissors are used to cut adhesive tape, gauze, rubber drains, and suture material. Metzenbaum scissors should be used to debride heavy tissue, bluntly dissect tissue, and undermine tissue.

Hemostats are used to clamp small vessels that are bleeding, to explore a wound, and to grasp fascia. Hemostats are available in a variety of sizes and styles. A straight 6 inch hemostat is used for most purposes during wound repair. A curved 5 inch mosquito hemostat can be used for small wounds or delicate tissues. **Do not use a hemostat to grasp or drive the suture needle.** The suture needle can bend, rotate, and break as it enters tissue if driven by a hemostat.

Three different scalpel blades should be available when a wound is being repaired. A #11 blade is used to make stab incisions. It is often used for the incision and drainage of abscesses, cricothyroidotomies, and the removal of small or tight sutures. A #10 blade is used to make straight cuts in the skin and debride wound edges. It is rarely used in laceration repair. A #15 blade is small and curved to allow precise incisions. It is used for excising foreign bodies and wound debridement.

SUTURE TECHNIQUES

Proper wound closure requires an understanding of certain basic principles. **The needle should enter and exit the skin at a 90° angle and perpendicular to the wound edges.** By doing so, when the suture loop is closed, the wound edges will be everted. **Sutures should be placed as close to the wound edge as possible (i.e., 2 to 3 mm) in order to avoid excessive tension on the wound.** More force will be required to close the wound if the sutures are placed too far from the wound edge. Edema develops in a wound in the first 48 hours after closure. Sutures placed too far from the wound edge can result in large scars when the edema subsides.

The layers of the wound should be matched evenly and each layer should be closed separately. If a wound involves the deeper layers of skin, fascia should be matched to fascia, dermis should be matched to dermis, and epidermis should be matched to epidermis. **The proper matching of layers avoids an uneven closure, helps to prevent an unnecessarily large scar, and eliminates any dead space.**

The epidermal edges of the wound must be everted to allow for proper healing. Scars contract with time. They will flatten and heal with a better cosmetic result if the wound edge is everted and slightly elevated. If the wound edges are not everted they will contract into a "pit" below the plane of the skin, will be more noticeable, and the final result will be less appealing cosmetically.

Handle the tissues gently and do not squeeze or twist them too tightly with the instruments. This helps to avoid strangulation, which can result in tissue necrosis. **The sutures should be placed carefully and with the proper amount of tension to help promote healing.** Sutures should be snug. Attempts should be made to avoid excessive tension on the wound edges in order to prevent wound dehiscence. The use of the smallest suture size necessary to approximate the wound edges will reduce tissue damage and minimize scarring. **Table 116-3** lists the appropriate suture types and sizes for each body region.

If there will be a temporary delay in the closure of a laceration because of other injuries that may be life-threatening or of greater importance, cover the wound with a saline-soaked gauze in order to keep the tissues from drying.

PRINCIPLE OF HALVING

Large wounds gape open and are difficult to approximate. Closure of the deeper layers will often bring the skin edges into apposition. If not, the principle of halving may be used to approximate the wound (**Figure 116-2**). Identify the midpoint of the laceration. Place the first suture at the midpoint (**Figure 116-2*A***). This stitch is known as the central suture. The next sutures are placed in halves on each side of the central suture (**Figures 116-2*B* and 116-2*C***). Continue the process by placing sutures halfway between previous sutures until the wound is approximated. This results in even closure of the wound edge. This principle can be used for closure of both the deep layers and the skin.

TWO-HANDED SQUARE KNOT

This is the easiest and most reliable method of tying most suture materials. It involves the classic "right-over-left and left-over-right" tie (**Figure 116-3**). The incorrect "right-over-left and right-over-left" is a granny knot which will slip if it is tied in this manner. This square knot is quick and simple to perform. However, it does take significant practice to master this technique.

Place a suture through the skin on both sides of the laceration (**Figure 116-3*A***). Pull the suture through the wound until half is on each side of the laceration. Grasp the right half of the suture with the right thumb and index finger (**Figure 116-3*A***). Grasp the left half of the suture with the left third through fifth fingers and the suture draped over the thumb (**Figure 116-3*A***). Cross the right hand toward the left hand (**Figure 116-3*B***). Continue to move the right hand until the suture is between the left thumb and index finger (**Figure 116-3*C***). Close the left thumb and index finger to entrap the right half of the suture in the pads of the fingers (**Figure 116-3*D***). Pull the right hand down and to the left so that the two halves of the suture form an "X" over the left thumb (**Figure 116-3*E***). Flex the left wrist to slide the "X" off the left thumb and onto the left index finger (**Figure 116-3*F***). Lift the left thumb backward and upward so that the "X" overlies the tip of the left index finger (**Figure 116-3*G***). Reapply the left thumb over the left index finger to entrap the "X" (**Figure 116-3*H***). Extend the left wrist

TABLE 116-3 Typical Suture Choices for Each Body Site

Anatomic site	Deep-layer suture size	Deep-layer suture material	Skin-layer suture size	Skin-layer closure material
Scalp	2–0, 3–0, or 4–0	Absorbable	4–0 or 5–0	Nylon, polypropylene, or staples
Eyelid	5–0, 6–0, or 7–0	Absorbable	6–0 or 7–0	Nylon or polypropylene
Face	4–0 or 5–0	Absorbable	5–0 or 6–0	Nylon or polypropylene
Neck	4–0 or 5–0	Absorbable	4–0 or 5–0	Nylon or polypropylene
Trunk	3–0 or 4–0	Absorbable	3–0 or 4–0	Nylon, polypropylene, or staples
Extremities	3–0 or 4–0	Absorbable	3–0, 4–0, or 5–0	Nylon, polypropylene, or staples
Hands and feet	Not advisable	Not applicable	4–0 or 5–0	Nylon or polypropylene
Sole of foot	3–0 or 4–0	Absorbable	3–0 or 4–0	Nylon or polypropylene

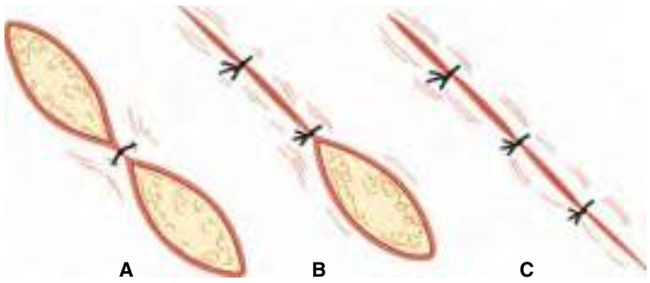


FIGURE 116-2. The principle of halving. ***A.*** The first suture is placed in the middle of the laceration. ***B.*** The second suture is placed halfway between the first suture and the upper end of the laceration. ***C.*** The third suture is placed halfway between the first suture and the lower end of the laceration.

to push the left thumb and the "X" through the loop (**Figure 116-3*I***). Release the suture held with the right hand (**Figure 116-3*J***). Re-grasp the suture with the right hand after it passes through the loop (**Figure 116-3*K***). Pull the suture completely through the loop with the right hand. Simultaneously move the left hand toward the left and move the right hand toward the right (**Figure 116-3*L***). Cross the hands so that the left hand goes toward the right side and the right hand goes toward the left side (**Figure 116-3*M***). Continue to pull both sides of the suture until the knot lies flat and the skin edges are apposed (**Figure 116-3*M***). The first half of the knot is now complete.

Make the second half of the knot to complete the square knot. Raise both hands upward and uncross them until an "X" is formed over the left index finger (**Figure 116-3*N***). Close the left thumb

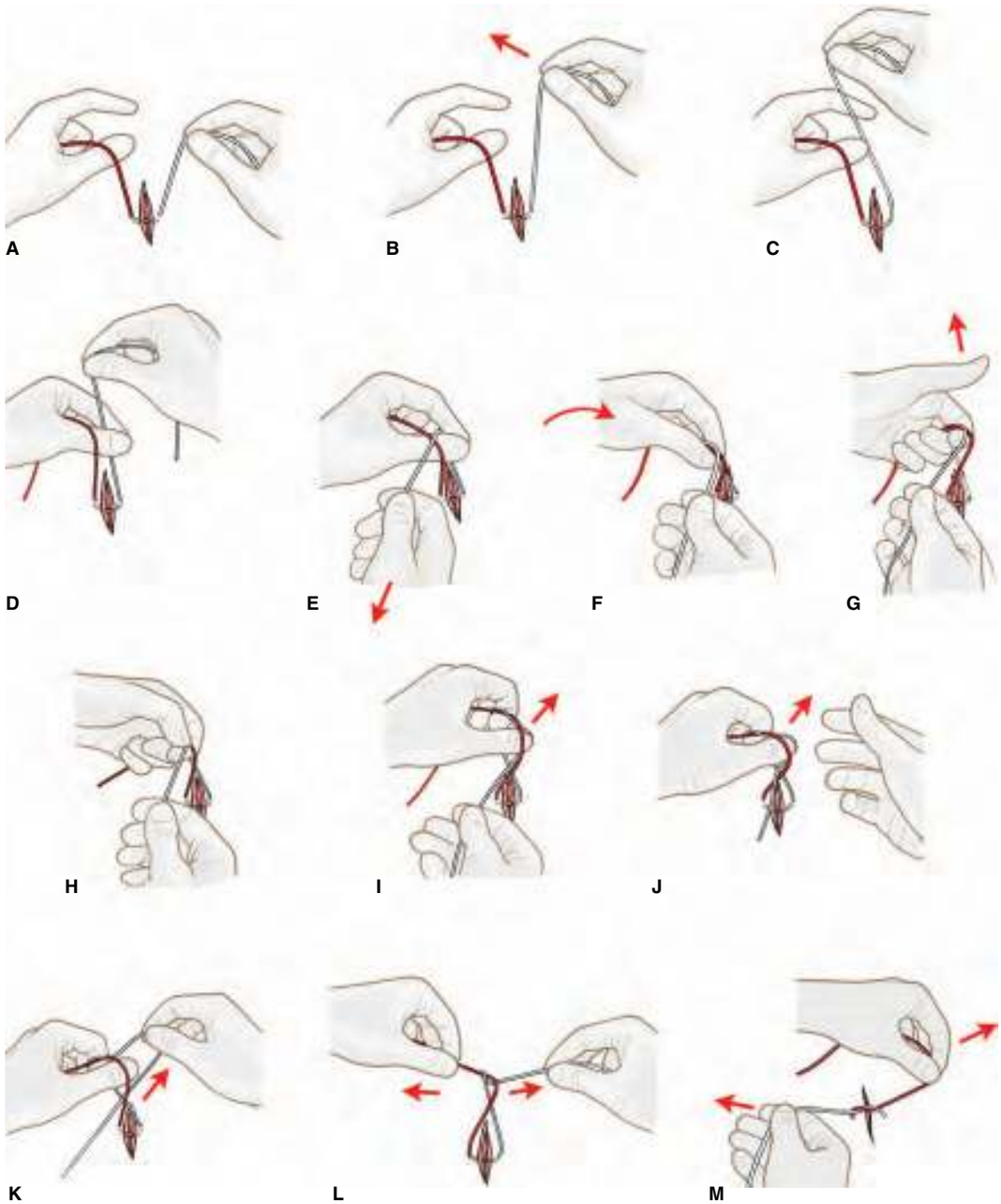


FIGURE 116-3. The two-handed square knot. The two halves of the suture are different colors to make viewing easier.

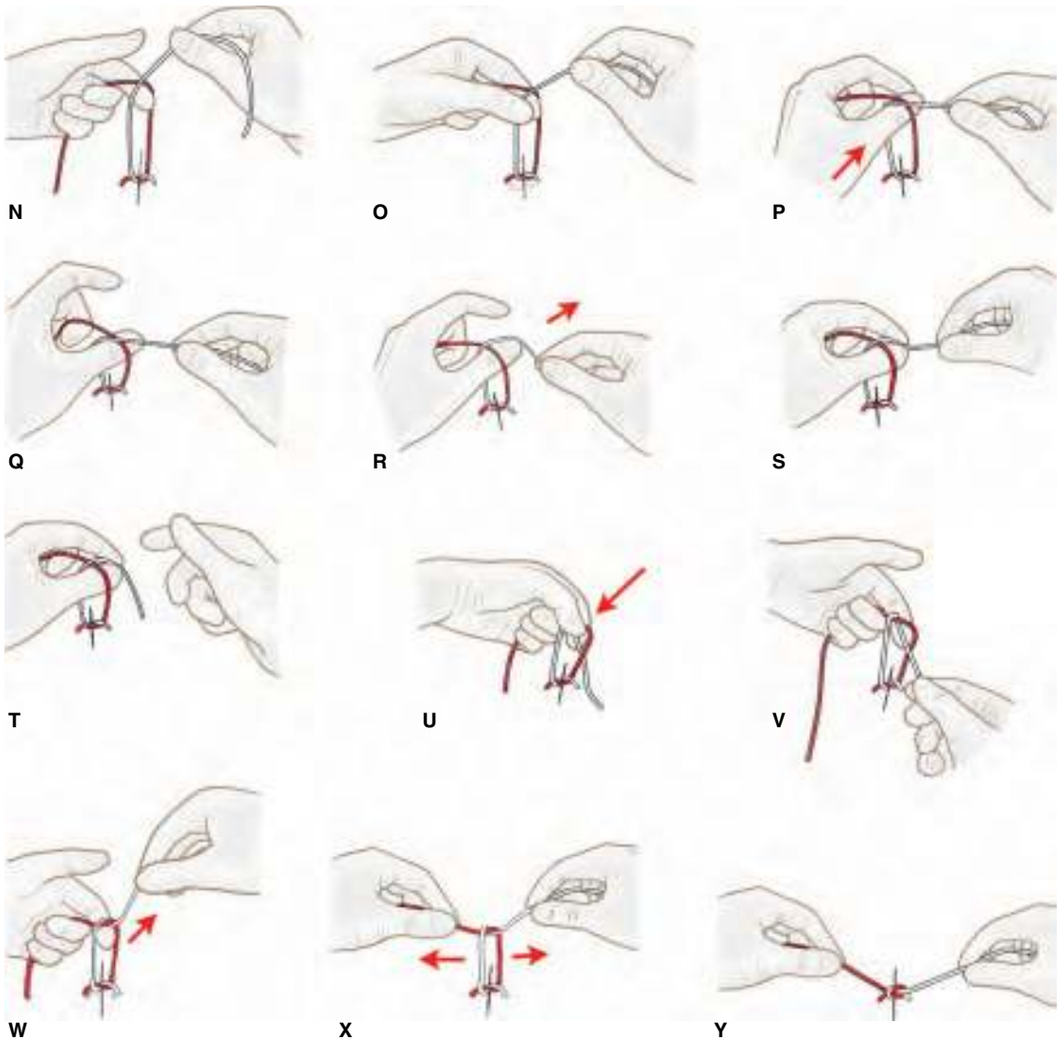


FIGURE 116-3. (*Continued*)

and index finger to entrap the suture being held with the right hand (**Figure 116-3*O***). Extend the left wrist to push the left thumb through the loop (**Figure 116-3*P***). Lift the left index finger upward (**Figure 116-3*Q***). Move the right hand away from you until the suture it holds drapes over the left thumb (**Figure 116-3*R***). Reapply the left index finger onto the thumb to entrap the suture held with the right hand (**Figure 116-3*S***). Release the suture held with the right hand (**Figure 116-3*T***). Flex the left wrist to push the left index finger and suture through the loop (**Figure 116-3*U***). Re-grasp the free suture with the right hand after it passes through the loop (**Figure 116-3*V***). Move the right hand upward and to the right to complete the second half of the knot overlying the left index finger (**Figure 116-3*W***). Simultaneously move the left hand toward the left and move the right hand toward the right (**Figure 116-3*X***). Continue to pull both halves of the suture until both halves of the knot come into contact (**Figure 116-3*Y***). Pull both halves of the suture to secure the knot. The square knot is now complete. Continue the process to add additional knots onto the suture. Cut off excess suture on both sides of the knots.

SURGEON'S KNOT

The physician may choose to use a surgeon's knot instead of a square knot (**Figure 116-4**). The square knot has one loop in the first throw

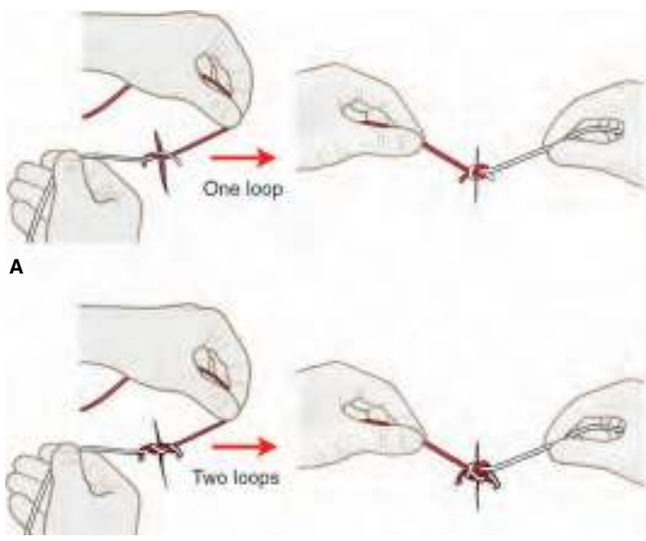


FIGURE 116-4. The square knot (***A***) versus the surgeon's knot (***B***). The first throw of the square knot has one loop (***A***), while that of the surgeon's knot has two loops (***B***). The second throw of both knots are a simple loop.

and one loop in the second throw (**Figure 116-4*A***). The surgeon's knot has two loops in the first throw and one loop in the second throw (**Figure 116-4*B***). The only difference between these two knots is the two loops in the first throw. The second throw and subsequent knots are exactly the same for both knots. The advantage of the surgeon's knot is that the two loops are more secure and stay in place while the second throw is being tied. Thus, the laceration tends to stay closed while the second throw is being formed. The choice to use either knot is dependent on the experience and the training of the physician.

INSTRUMENT TIE

The instrument tie is the most efficient method to complete a simple interrupted suture (**Figure 116-5**). It is the tie that is most commonly used in the Emergency Department. An instrument tie

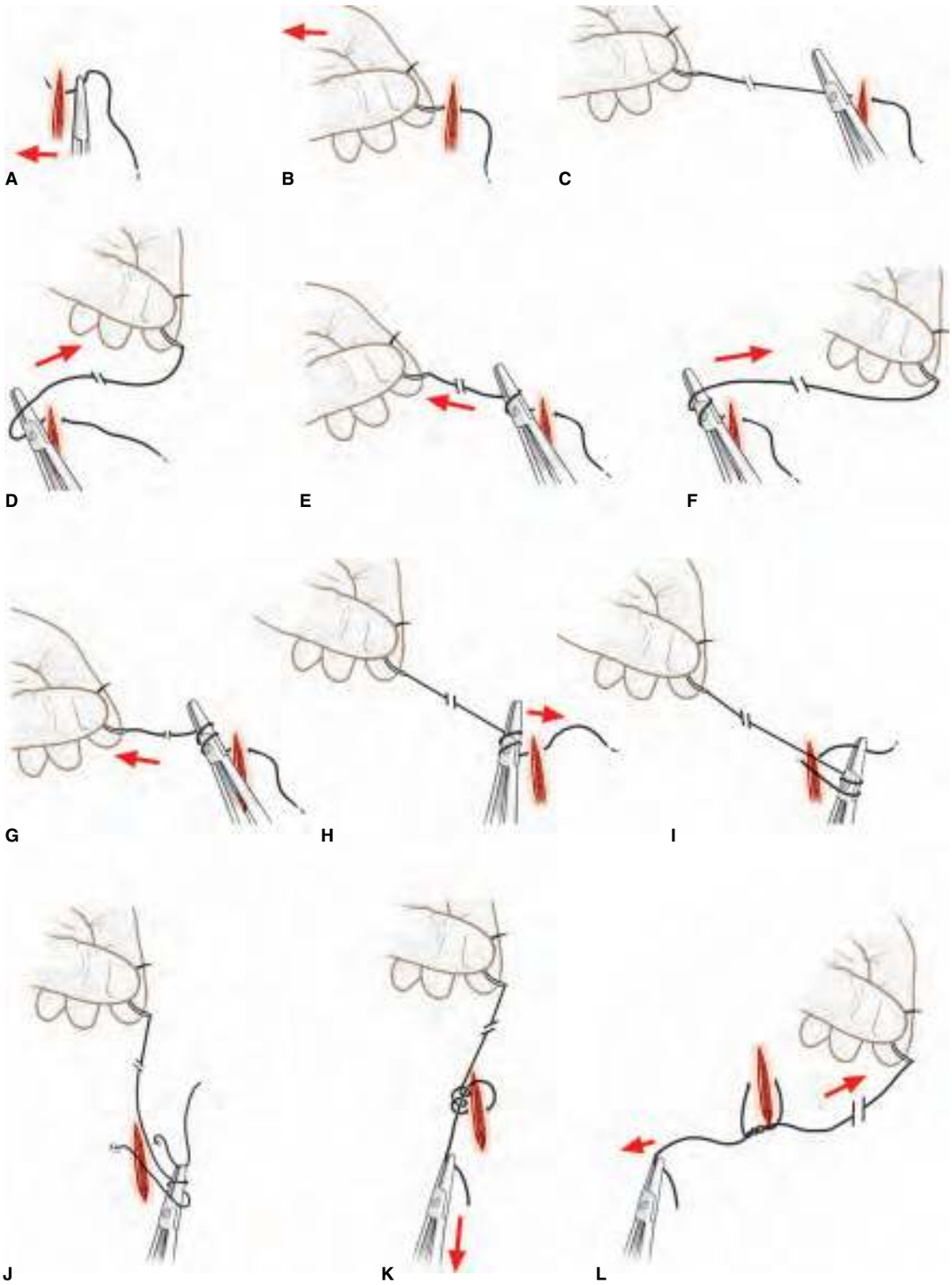


FIGURE 116-5. The instrument tie.

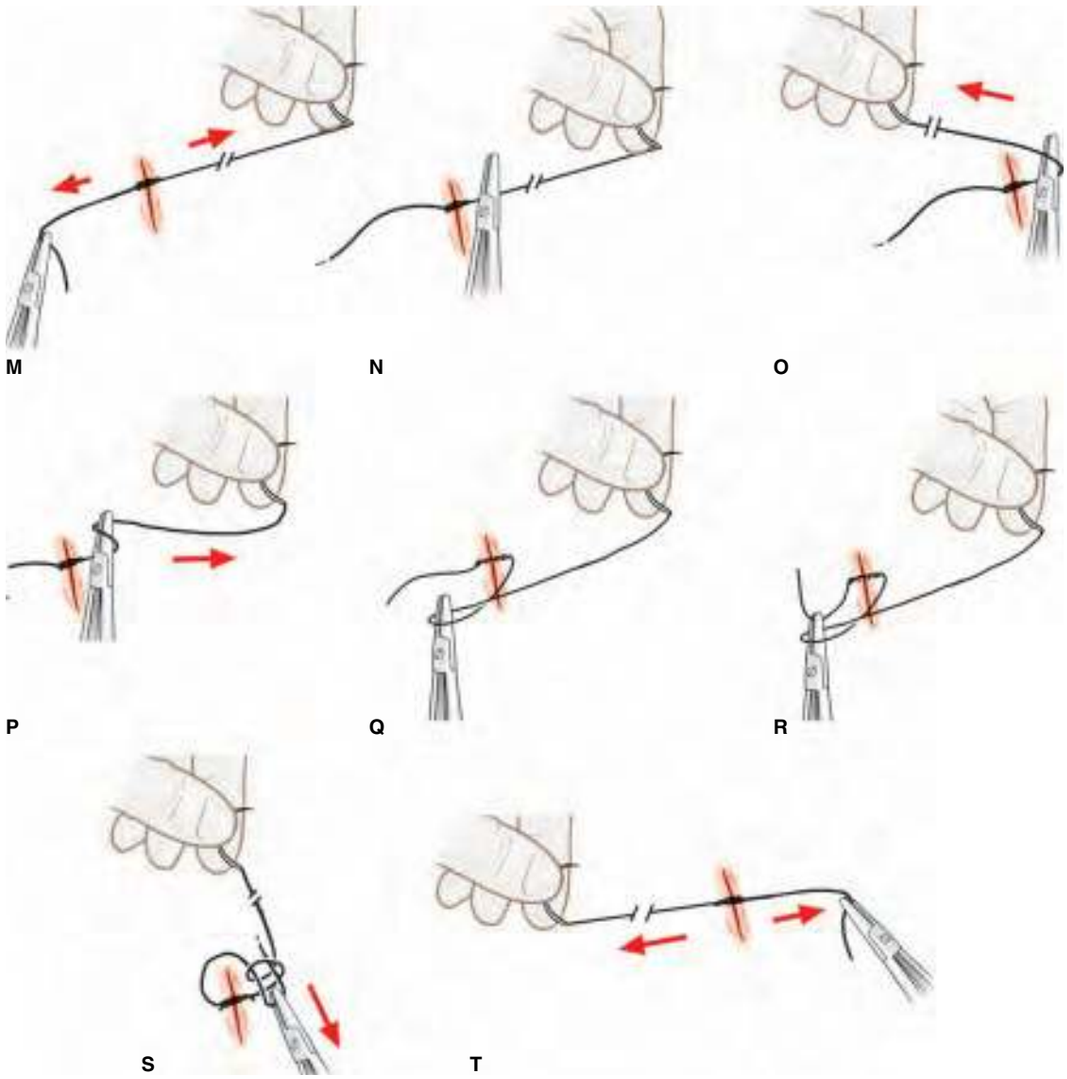


FIGURE 116-5. (*Continued*)

is often quicker, requires less dexterity, and is easier to perform than the two-handed method. It may be used with the square knot or the surgeon's knot.

Place a suture through the skin on both sides of the laceration (**Figure 116-5*A***). Carefully grasp the needle in its midportion and pull it through the laceration (**Figure 116-5*B***). Continue to pull the needle until approximately 1 to 2 cm of suture on the tail end remains outside the laceration (**Figure 116-5*C***). A large amount of suture will be wasted if the tail is left too long, as it will be later cut off and discarded. On first learning the instrument tie, it may be best to leave a tail of 3 to 4 cm until one is proficient with this technique.

Place the needle driver over the laceration but not touching it (**Figure 116-5*C***). Loosely loop the needle end of the suture over (**Figure 116-5*D***) and around (**Figure 116-5*E***) the needle driver. Loosely loop the needle end of the suture over and around the needle driver a second time (**Figures 116-5*F* and 116-5*G***). This will eventually result in the first half of a surgeon's knot. Looping the suture once around the needle driver will result in a simple square knot. Move the tip of the needle driver toward the tail of the suture without letting the loops fall off the needle driver (**Figure 116-5*H***). Grasp the tail of the suture with the needle driver (**Figure 116-5*I***). Pull the tail of the suture through the loop (**Figure 116-5*J***). Pull the tail completely through the loops (**Figure 116-5*K***). Simultaneously move the left hand toward the right and the right hand/needle driver toward the left (**Figure 116-5*L***). Continue to pull both sides of the suture until the hands are opposite each other, the knot lies flat, and the skin edges are apposed (**Figure 116-5*M***). The first half of the knot is now complete.

Make the second half of the knot. Continue to hold the needle and release the tail of the suture from the needle driver (**Figure 116-5*N***). Place the needle driver over the laceration but not touching it (**Figure 116-5*N***). Loosely loop the needle end of the suture over (**Figure 116-5*O***) and around (**Figure 116-5*P***) the needle driver. Move the tip of the needle driver toward the tail of the suture without letting the loop fall off the needle driver (**Figure 116-5*Q***). Grasp the tail of the suture with the needle driver (**Figure 116-5*R***). Pull the tail of the suture completely through the loop (**Figure 116-5*S***). Simultaneously move the left hand toward the left and the right hand/needle driver toward the right (**Figure 116-5*T***). Continue to pull both sides of the suture until both halves of the knot come into contact. Pull both sides of the suture to secure the knot. The knot is

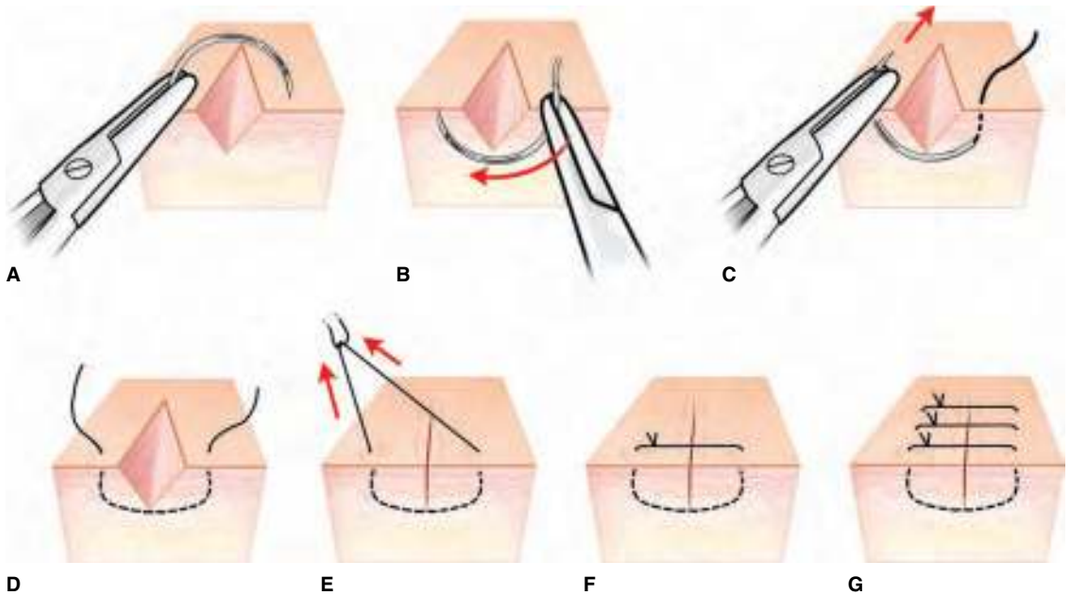


FIGURE 116-6. The simple interrupted stitch.

now complete. Continue this process three or four more times, each in alternative directions, to place additional knots. Cut off the excess suture on both sides of the knots.

SIMPLE INTERRUPTED STITCH

The simple interrupted stitch is the most commonly used suture technique and is useful in many situations (**Figure 116-6**). One major advantage is that each stitch is placed independent of the others. Therefore, tension on each stitch can be adjusted separately. Additionally, the entire repair is not compromised if one suture should happen to come out. The other sutures will remain in place to help assure proper wound healing. **The needle must enter and exit the skin at a 90° angle to help evert the wound edges. Take equal volumes of skin from each side of the area being sutured. Drive the needle equidistantly into and out of the wound edges and incorporate the base of the wound.**

Insert the needle at a 90° angle to the skin (**Figure 116-6*A***). Drive the needle through the tissue until the tip exits the skin (**Figure 116-6*B***). Grasp the needle behind the tip and pull it through the wound (**Figure 116-6*C***). **The suture should enter and exit the skin equidistant from the wound edges (Figure 116-6*D*). If it does not, pull the suture out and repeat the stitch so that it is equidistant from the wound edges.** Make a loop in the suture with the two-handed tie or the instrument tie. Pull the suture to appose the wound edges and cinch down the knot (**Figure 116-6*E***). The tissue at the base of the wound will come into apposition before the tissue at the skin surface and thus evert the wound edge. Complete the knot to one side of the laceration (**Figure 116-6*F***). Just prior to cinching the second throw onto the first, pull the ends so that the knot is not directly over the wound and the edges of the wound remain in apposition. Apply additional sutures equidistant from each other until the wound is closed (**Figure 116-6*G***).

OPEN-LOOP SIMPLE INTERRUPTED STITCH

The open-loop simple interrupted stitch is a variation of the simple interrupted stitch (**Figure 116-7**). The same basic technique is used except that the knot is tied differently. The tie involves laying down the first knot with an instrument tie. However, the second knot placed on the suture is not pulled all the way down. Pull the second knot only until it is just above the first knot (**Figure 116-7*A***). In other words, the second knot is loosely tied, leaving a loop between the first and second knots. Place a third knot as a single knot square to the second knot (**Figure 116-7*B***). Cinch the third knot tightly to the second knot. This "locks" the third knot onto the second knot.

This knot is indicated when there is the possibility of edema forming at the suture site. If edema forms, the first knot will have room to open as it slides toward the second knot. This stitch avoids excessive tension on the wound and prevents the suture from cutting into the skin. This stitch facilitates suture removal when numerous small stitches are placed next to a wound edge. Cutting the open loop

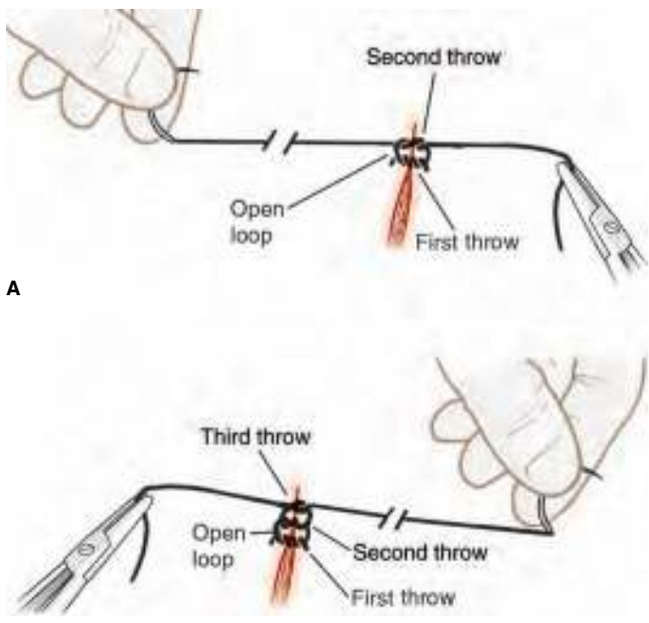


FIGURE 116-7. The open-loop simple interrupted stitch.

unravels the knot and allows for easy removal of the suture. This stitch should not be used in areas where the skin is thin or if there is little subcutaneous tissue (e.g., dorsal hand and foot). In these areas, the wound edges often become unopposed while the knot is being secured.

INTERLOCKING SLIP KNOT

This technique can be used in patients who will be traveling, camping, or otherwise away from their primary source of medical care (**Figure 116-8**). The patient can easily remove the sutures without having to find an unfamiliar or foreign health care provider for routine suture removal. The interlocking slip knot can be removed by hand without the use of a scissors or a scalpel. This can be useful for suture removal in pediatric patients, who may find it hard to sit still for suture removal.

Place a suture through the skin on both sides of the laceration (**Figure 116-8*A***). Loop the tail end of the suture around the tip of the needle driver (**Figure 116-8*A***). Grasp the needle end of the suture with the needle driver (**Figure 116-8*B***). Pull the needle end of the suture through the loop while simultaneously pulling on the tail end of the suture with a second needle driver or your hand (**Figure 116-8*C***). Continue to pull both suture halves in opposite directions until a knot is formed against the skin and the wound edges are apposed (**Figure 116-8*D***).

Release the needle driver holding the now formed loop. Insert the needle driver through the loop and grasp the tail end of the suture (**Figure 116-8*E***). Grasp the needle end of the suture with the second needle driver or your hand (**Figure 116-8*E***). Pull the needle drivers in opposite directions to lock and secure the knot (**Figure 116-8*F***). The knot is now complete (**Figure 116-8*G***). To remove the stitch, pull the free end of the suture to unlock the knot. Continue to pull the suture until it is free from the skin. This stitch can easily become loose or open before the wound is healed. Thus, it is recommended to cover the wound and sutures with skin closure tape (e.g., Steri-Strips).

CONTINUOUS OVER-AND-OVER STITCH (SIMPLE RUNNING STITCH)

Continuous (simple running) sutures minimize the time required for laceration repair. Stitches can be placed very quickly, since each individual stitch does not have to be tied. This stitch provides strength and applies equal tension on all sutures in the repair. This stitch can be used to achieve hemostasis. The wound must be long and straight. Simple running stitches can effectively be used in partial-thickness lacerations and wounds under minimal tension.

However, there are several disadvantages to this stitch. It can be associated with significant epithelialization of the suture track. This is especially true if the suture is not removed early and remains for a prolonged period of time. Inclusion cysts may form within a few weeks after removal of the sutures. **Simple running stitches should not be used on any wound under tension.** If one suture breaks, the entire wound may open. **This stitch should not be used when closing a wound where there is a risk of subsequent hematoma formation.** Hematoma formation would require the removal of all of the sutures in order to drain the hematoma. Although this suture is not commonly used in the Emergency Department, it can be very helpful for closing bleeding scalp wounds, as the injury will be covered with hair and cosmesis is a secondary concern.

Place the initial stitch as a simple interrupted stitch (**Figures 116-9*A***). Do not cut the suture after the knots are securely tied. Place a second stitch 3 to 5 mm from the first stitch as if placing another simple interrupted stitch (**Figures 116-9*B* and 116-9*C***). Place a third stitch 3 to 5 mm from the second stitch (**Figures 116-9*D* and 116-9*E***). Continue to place additional stitches until the end of the laceration is reached. **Use care to ensure that the sutures are all lined up with each other and equidistant from the laceration.** Do not pull the last throw taut against the skin (**Figure 116-9*F***). The loop will act as the tail end of the suture for knot tying. Loop the needle end of the suture twice around the tip of the needle driver (**Figure 116-9*G***). Grasp the last throw with the tips of the needle driver (**Figure 116-9*G***). Pull the last throw through the loops until the knot is against the skin (**Figure 116-9*H***). Perform three to five

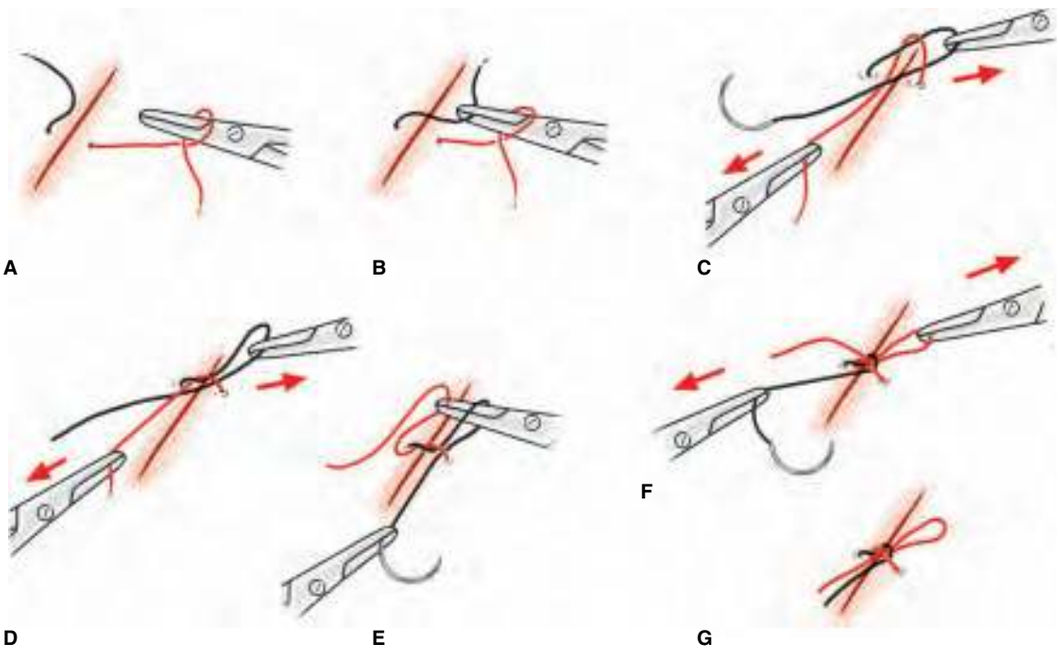


FIGURE 116-8. The interlocking slip knot. The two halves of the suture are different colors to make viewing easier.

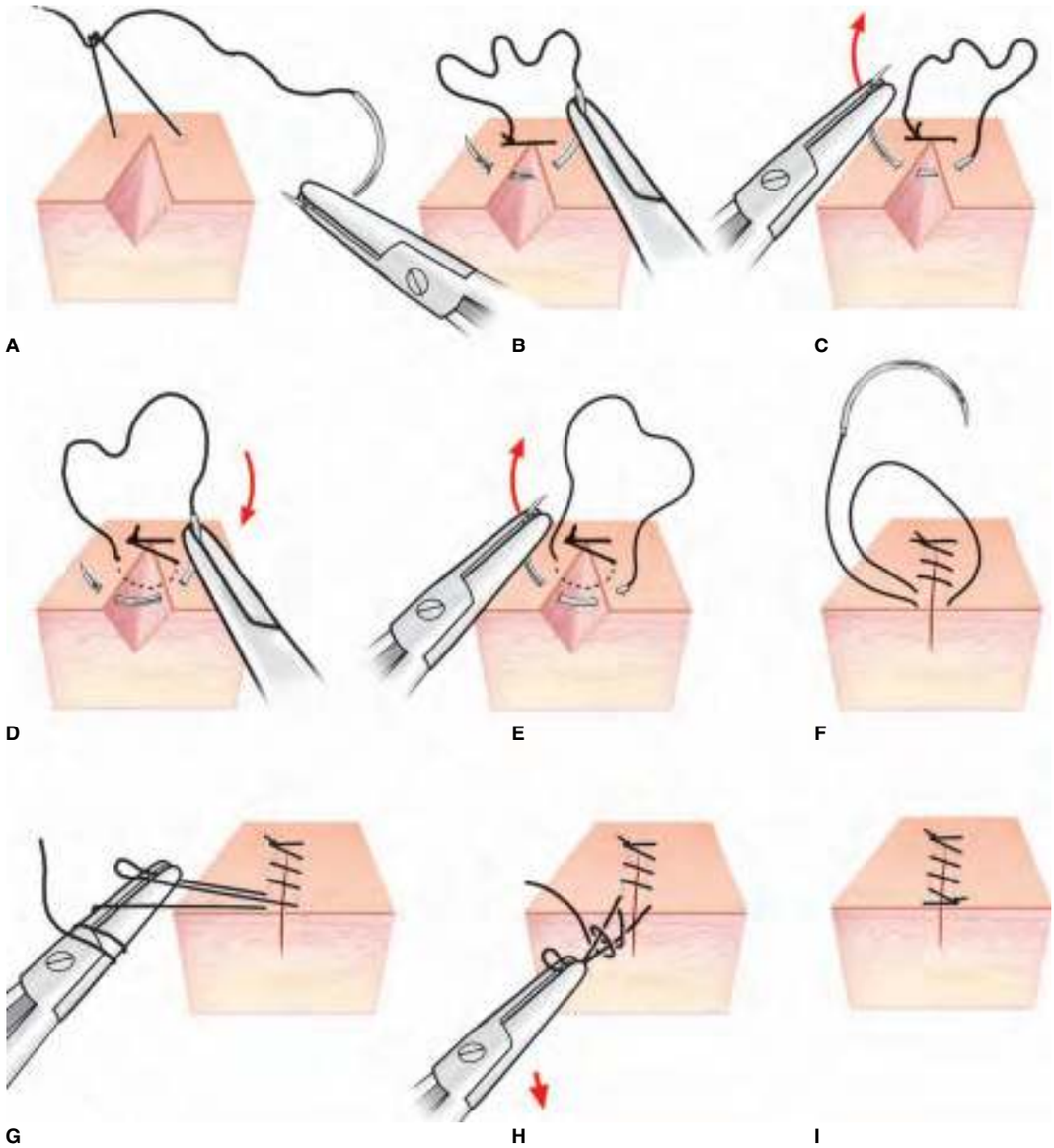


FIGURE 116-9. The continuous over-and-over or simple running stitch.

more instrument ties to secure the knot. Cut off the excess suture (**Figure 116-9*I***).

CONTINUOUS SINGLE-LOCKED STITCH (RUNNING-LOCKED CLOSURE)

This stitch may promote less epithelialization of the suture track than the continuous over-and-over or simple running stitch. It maintains the advantages of a continuous suture. This variation of the simple running closure locks each stitch after it is placed (**Figure 116-10**). It provides a secure apposition of the wound edges while each subsequent stitch is placed. The main disadvantage of this stitch is the time it takes compared to a continuous over-and-over or simple running stitch.

Place the initial stitch as a simple interrupted stitch (**Figure 116-10*A***). Do not cut the suture after the knots are securely tied. Loop the tail end of the suture over the nondominant fifth finger (**Figure 116-10*B***). Apply slight tension on the tail end of the suture while placing the second stitch (**Figures 116-10*C* and 116-*D***). As the needle exits the skin, move the nondominant hand to bring the suture loop down and over the needle (**Figure 116-10*E***). Grasp the needle with the needle driver. Simultaneously pull the needle and suture through the laceration while releasing the loop from the fifth finger (**Figure 116-10*F***). Repeat this procedure until the laceration is closed (**Figure 116-10*G***). **Do not pull the last throw taut against the skin.** The loop will act as the tail end of the suture for knot tying. Loop the needle end of the suture twice around the tip of the needle driver (**Figure 116-10*H***). Grasp the last throw with the tips of the needle driver. Pull the last throw through the loops until the knot is against the skin (**Figure 116-10*I***). Perform three to five more instrument ties to secure the knot. Cut off the excess suture (**Figure 116-10*J***).

VERTICAL MATTRESS STITCH

The vertical mattress stitch is a double stitch that provides for excellent wound eversion and hemostasis (**Figure 116-11**). It optimizes wound closure of lacerations under tension. This stitch is useful in areas where the skin is very lax, such as the elbow and

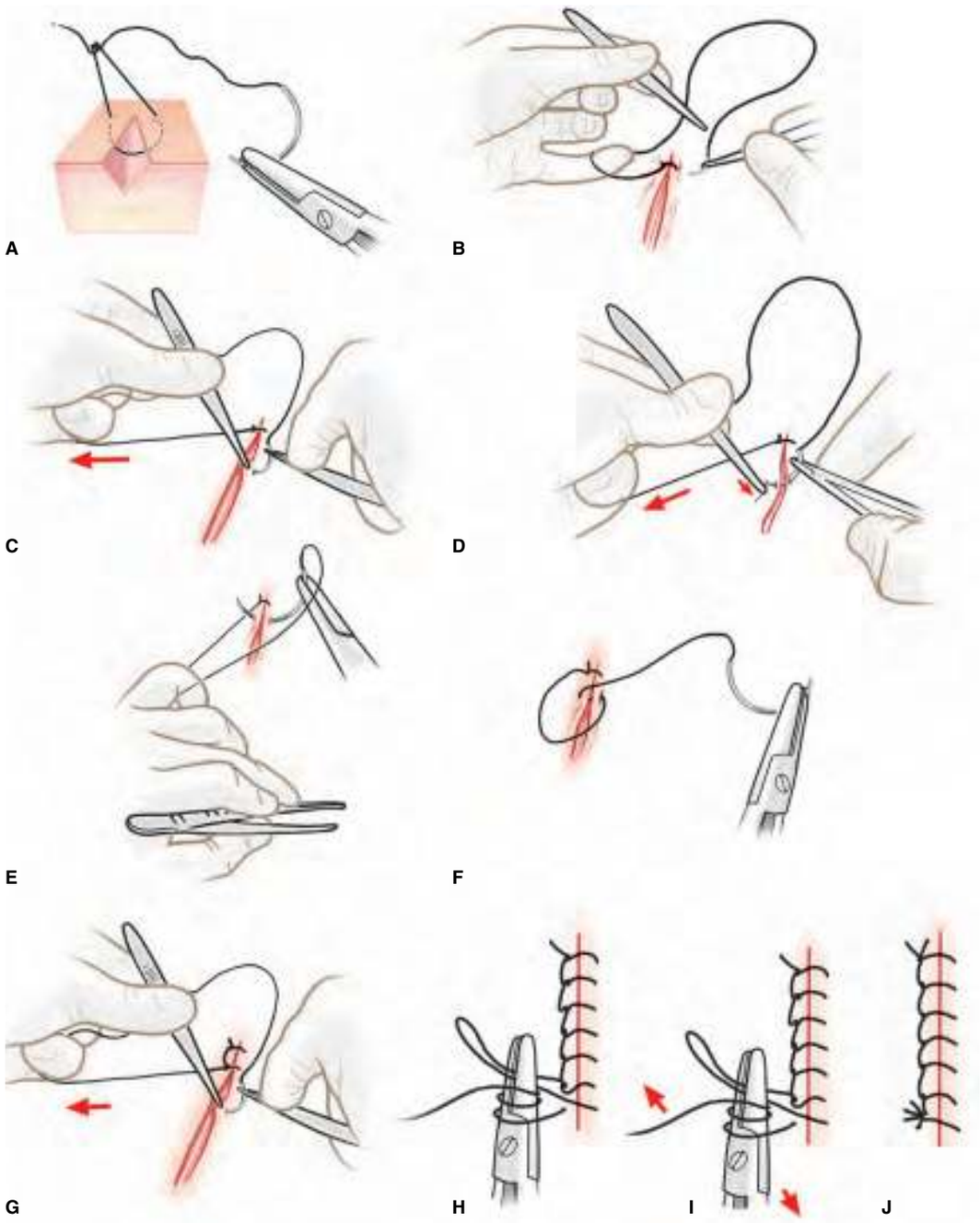


FIGURE 116-10. The continuous single-locked or running-locked stitch.

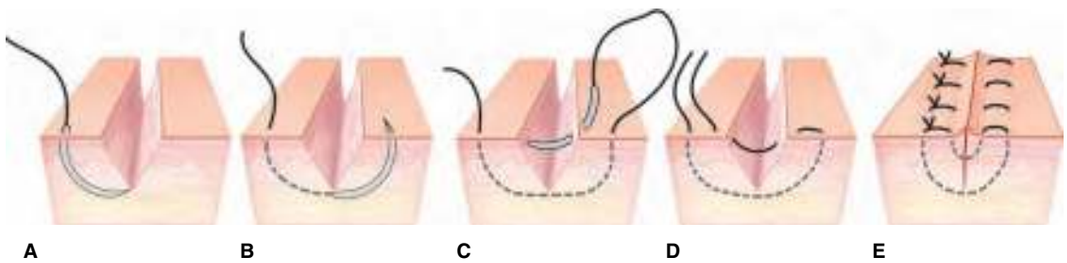


FIGURE 116-11. The vertical mattress stitch.

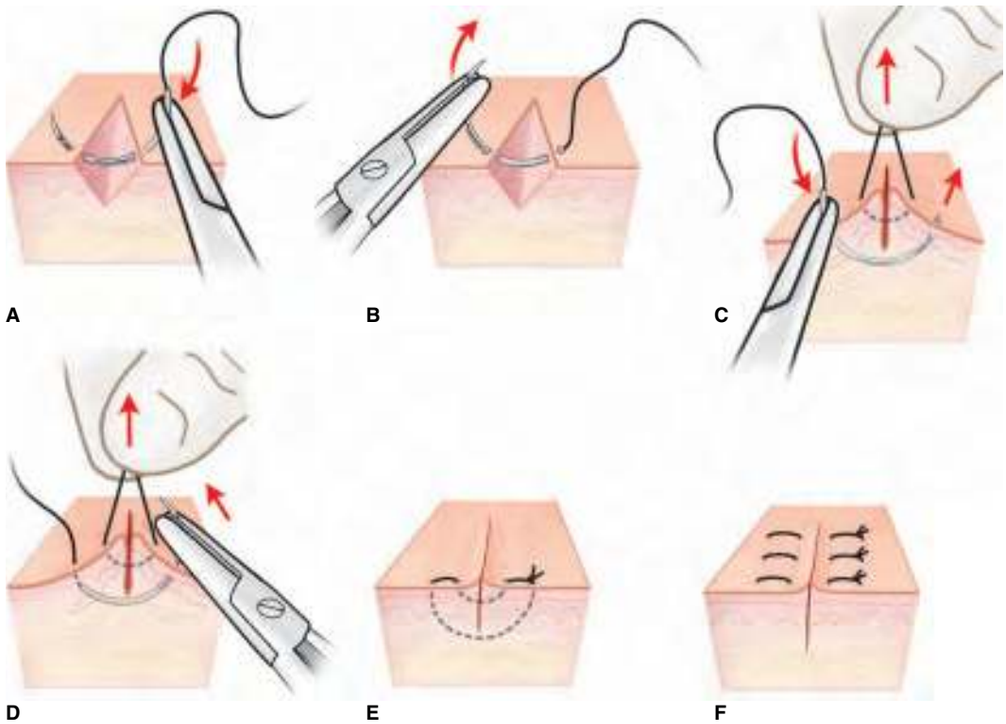


FIGURE 116-12. The "shortcut" vertical mattress stitch. An alternative method to place the vertical mattress stitch.

the dorsum of the hand. This stitch provides for both superficial as well as deep closure of lacerations. **This stitch is contraindicated in lacerations involving the volar aspect of the hands and feet or the face, as it requires the blind placement of a deep suture.** The main disadvantage of the vertical mattress closure is the time it takes to place it.

Place the first throw much like a simple interrupted stitch with a few noted differences. The needle should enter and exit the skin 1.0 to 1.5 cm from the wound edge. The needle should traverse the base of the wound and grasp a large amount of tissue (**Figures 116-11*A* and 116-11*B***). Reverse the needle. The second throw should enter and exit the skin approximately 2 to 3 mm from the wound edge (**Figures 116-11*C* and 116-11*D***). **The first and second throws must be directly over each other and parallel.** Tie the suture to approximate the wound edges (**Figures 116-11*E***). The first throw will close the wound base and relieve the tension at the skin surface. The second throw approximates and everts the skin edges.

The newer version of the classic vertical mattress is referred to as the "shorthand" vertical mattress stitch (**Figure 116-12**). It provides wound eversion in half the time as the traditional method. Place the first throw close to the lacerated wound edge to approximate the skin edges (**Figures 116-12*A* and 116-12*B***). Grasp and pull the suture to elevate the wound edges (**Figure 116-12*C***). This allows the needle to more easily take a large bite of tissue on the second throw. Place the second throw 1.0 to 1.5 cm from the wound edge (**Figures 116-12*C* and 116-12*D***). Release the suture. Tie the suture to approximate the wound edges and evert the skin surface (**Figure 116-12*E***). The final product looks exactly the same as the traditional vertical mattress suture (**Figure 116-12*F***).

LOCKED VERTICAL MATTRESS STITCH

The locked vertical mattress stitch is useful in areas that are widely separated, where deep sutures must be avoided, and in areas that

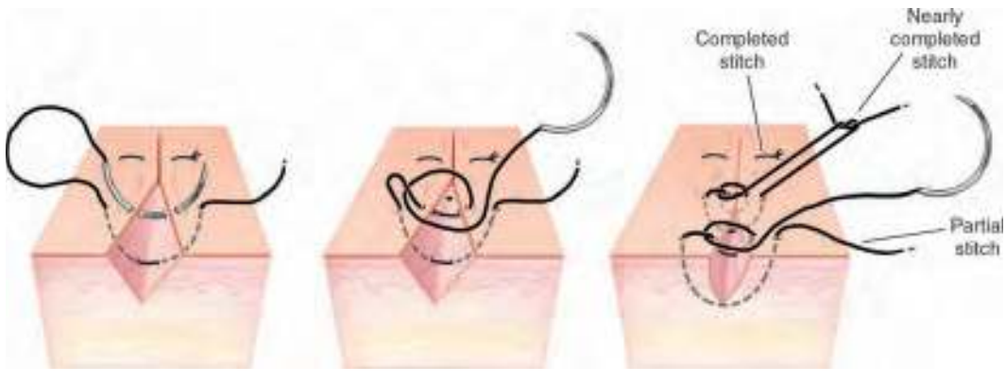


FIGURE 116-13. The locked vertical mattress stitch.

keep bleeding such as the scalp (**Figure 116-13**). This stitch helps to reduce the amount of tension needed to close a wound and provide hemostasis. It helps to avoid the pain and scarring that can result if too much tension is applied to a laceration. It does not put an excessive amount of tension on the deep throw, as does the vertical mattress stitch.

This is a modification of the vertical mattress stitch (**Figures 116-11 and 116-12**). Place the first two throws as if placing a vertical mattress stitch (**Figure 116-13*A***). Leave the suture lax with a loop above the wound surface. Pass the needle end of the suture through the open loop (**Figure 116-13*B***). This step will form the locked portion of the stitch. Pull the needle end of the suture taut to appose the wound edges (**Figure 116-13*C***). Tie and secure the suture ends in the standard manner.

HORIZONTAL MATTRESS STITCH

The horizontal mattress stitch is placed along the axis of the wound and helps to eliminate tension on the wound (**Figure 116-14**). It is a good closure technique for wounds with relatively poor circulation to the wound edges because, theoretically, no suture is placed through the wound edges. This helps to avoid tension on the wound edges from the suture and subsequent local necrosis. This stitch is placed more rapidly than the vertical mattress stitch. It requires fewer stitches to close a wound with horizontal rather than vertical mattress stitches. The throws are side by side rather than on top of each other, as with the vertical mattress, and each stitch closes more tissue. This closure may be used superficially on the volar surfaces of the hands and fingers, as these delicate skin areas may swell and be cut by simple interrupted sutures. The main disadvantage of the horizontal mattress stitch is that it takes more experience to properly place this stitch to achieve wound eversion than with the vertical mattress stitch.

Place the first throw much like a simple interrupted stitch with a few noted differences. The needle should enter and exit the skin 0.5 to 1.0 cm from the wound edge. The needle should traverse the base of the wound (**Figures 116-14*A* and 116-14*B***). Reverse the needle and make a second throw 0.5 cm from the first (**Figure 116-14*C***). **The needle must enter and exit the skin and the wound edges so that the first and second throws are parallel to each other** (**Figures 116-14*C* and 116-14*D***). Pull the free ends of the suture taut to appose and evert the wound edges (**Figures 116-14*E* and 116-14*F***). Tie and secure the suture in the standard manner.

HALF-BURIED HORIZONTAL MATTRESS STITCH

This is the stitch of choice to close complex wounds with multiple flaps in a single-layer closure. This stitch is ideal to close stellate, Y-shaped, V-shaped, and T-shaped lacerations. The half-buried horizontal mattress stitch allows a tissue flap to be reapproximated without tension on the edges of the flap. The vascular supply to a flap is derived from its base. The flaps sometimes have a limited or poor vascular supply. This stitch may be used to approximate a flap-like laceration in which the corner has limited vascularity and/or viability.

The key to this stitch is that the needle and suture pass through the dermis of the flap and not the epidermis (Figure 116-15). Begin by placing the first stitch percutaneously through the skin adjacent to the tip of the flap (**Figure 116-15*A***). Advance the needle through the dermal layer of the flap, through the dermal layer of the skin adjacent to the tip of the flap, and out the skin adjacent to the tip of the flap opposite to where the stitch began (**Figure 116-15*A*). The needle must traverse the dermis of the flap and adjacent tissue at the same level of the dermis to properly approximate the wound edges.** Gently pull on the free ends of the suture to approximate the flap against the adjacent skin edges. Tie and secure the suture in the usual manner. Secure the edges of the flap with half-buried horizontal mattress stitches (**Figure 116-15*A***), simple interrupted stitches, vertical mattress stitches, or horizontal mattress stitches.

Stellate lacerations are often seen in the Emergency Department. They occur due to bursting of the skin from crush injuries. These lacerations are often encountered on the extremities, forehead, and scalp. Begin by inserting the needle through the skin of the largest flap (**Figure 116-15*B***). Advance the needle so that its tip exits the dermis. Continue to advance the needle through the dermis of each flap. The half-buried horizontal mattress stitch should encompass the tips of all the flaps (**Figure 116-15*B***). The remainder of the procedure is as described above.

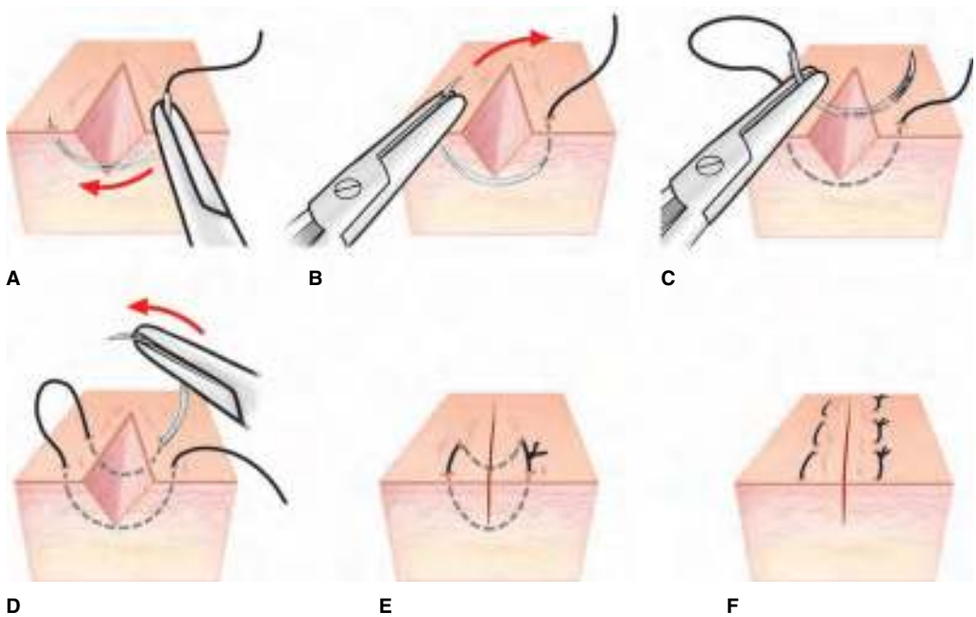


FIGURE 116-14. The horizontal mattress stitch.

FIGURE 116-15. The half-buried horizontal mattress stitch is used to approximate a star-shaped (stellate) laceration (*A*) and a Y-shaped laceration (*B*).

CONTINUOUS (RUNNING) HORIZONTAL MATTRESS STITCH

The running horizontal mattress stitch is indicated in areas of the body where there is loose skin that tends to overlap or invert, such as the skin of the upper eyelids or the dorsum of the hand. This stitch can also be used as the surface closure in a multiple-layer closure if there is a tendency for wound inversion. Like the traditional horizontal mattress stitch, it provides good apposition and can be placed rapidly. The running horizontal mattress stitch is contraindicated in wounds under tension if the goal of wound closure is optimal cosmesis.

This stitch begins with a simple interrupted stitch at one end of a laceration (**Figure 116-16**). The needle is then run along the length of the wound while placing horizontal mattress stitches. The difference between this and the standard horizontal mattress stitch is that the suture is not tied and cut after each individual stitch. Rather, the stitch is continued (running) the length of the laceration. At the end of the laceration, the stitch is tied and secured in the same way as the continuous over-and-over or simple running stitch (**Figure 116-9**).

CONTINUOUS SUBCUTICULAR STITCH

This closure is ideal for lacerations of the face and neck. It provides excellent cosmesis, leaves no suture marks on the skin, and causes minimal scarring. It requires more time and skill to place than other types of stitches. It may be performed for the temporary pull-out (**Figure 116-17**) or permanent placement (**Figure 116-18**) of subcutaneous sutures. Polypropylene or nylon sutures should be used for the pull-out stitch. Polypropylene is preferred for the pull-out stitch as it is stiffer, stronger, and easier to remove than nylon. The use of polypropylene or polyglactin 910 permanently placed as the subcuticular stitch has been shown to be cosmetically equal to polypropylene removed in 14 days.[13]

The use of this stitch is limited to lacerations that are clean and straight, have sharp edges, and are less than 6 cm in length. It may be extremely difficult to remove the suture material for the pull-out technique if the laceration is greater than 6 cm in length. The laceration can be longer if the permanent placement of absorbable (e.g., Vicryl) sutures is being done. The dermis and subcutaneous tissue must be apposed before proceeding with this stitch. If necessary, apply buried absorbable sutures to appose the dermis before applying this stitch. **The superficial wound surface must be tension-free, as this stitch is for cosmesis and not strength.** The wound may require undermining to release the tension from the wound edges. Refer to Chapter 114 for the details regarding wound undermining.

FIGURE 116-16. The continuous or running horizontal mattress stitch.

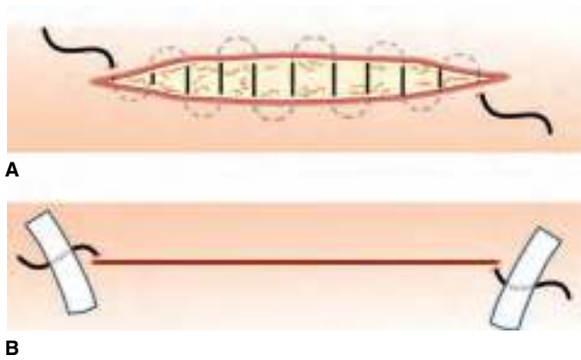


FIGURE 116-17. The continuous subcuticular pull-out stitch.

The pull-out technique allows the subcuticular stitch to be removed after the laceration heals (**Figure 116-17**). The subcuticular suture should enter the intact skin 3 to 4 mm from one end of the laceration and burrow through the dermal-epidermal junction to emerge through the skin at the other end of the laceration (**Figure 116-17*A***). The suture will continuously pass through the subcuticular layer on alternate sides of the laceration. **The point of entry of each stitch should be directly across from or slightly behind the exit point of the previous stitch. It is very important to keep the needle at the same level of depth throughout the wound.**

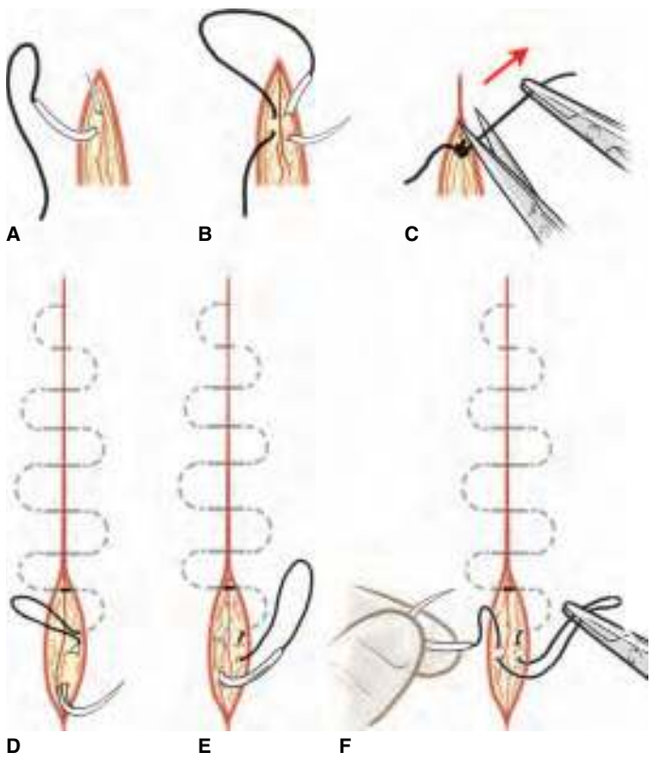


FIGURE 116-18. The continuous subcuticular permanent stitch.

The tension on the suture should be adjusted to ensure that there is no puckering of the skin. Tape the free suture at both ends of the laceration to the skin (**Figure 116-17*B***). Place wound tape (e.g., Steri-Strips) across the laceration to help maintain the apposition of the epidermis. This stitch is easily removed. Remove the wound tape and slowly pull one end of the suture with a needle driver.

As an alternative, the continuous stitch may be placed using absorbable suture material to provide longer-lasting strength to the wound (**Figure 116-18**). Suture material of choice includes Dexon, PDS, or Vicryl. The same indications, preparation, and stitch are used as with the pullout technique. The only difference is in the starting and ending stitch. Place the first stitch into the dermis, just inside the laceration edge, as a buried knot (**Figures 116-18*A*, 116-18*B*, and 116-18*C***). Place the continuous suture until the opposite end of the laceration is reached (**Figure 116-18*D***). The final throw should be left lax with a trailing loop of suture (**Figure 116-18*E***). The loop should be used as the "tail end" to perform an instrument tie (**Figure 116-18*F***). Tie three or four knots in the suture. Lift the free ends of the suture and cut them just above the knot. Apply wound tape across the laceration to help maintain the apposition of the wound.

BURIED (SUBCUTANEOUS) KNOT STITCH

This stitch helps to decrease potential dead space underneath a laceration, decreases the tension on the skin surface, and gives tensile support for up to 4 to 6 weeks while the wound is still weak. The loop is constructed so that the knot lies at the bottom of the wound base (**Figure 116-19**). This helps to keep the skin surface smooth and flat. The buried knot stitch is most useful in closing subcutaneous tissue just under the skin surface. It is preferable to use monofilament absorbable suture for a buried stitch.[5]

This stitch requires practice to master. Insert the needle into one side of the base of the wound (**Figure 116-19*A***). Drive the needle from deep to superficial and exiting at the dermal-epidermal junction (**Figure 116-19*A***). Insert the needle through the dermal-epidermal junction on the opposite side of the wound and drive it through the base of the wound (**Figure 116-19*B***). **The suture should exit the base of the wound across from and level with the entrance site of the first throw.** Pull both free ends of the suture up and out through the laceration (**Figure 116-19*C***). Tie a knot in the suture (**Figure 116-19*D***). Pull both free ends of the suture to lower the knot to the base of the wound and appose the tissue (**Figure 116-19*E***). Tie two additional knots to secure the suture. Cut off any excess suture.

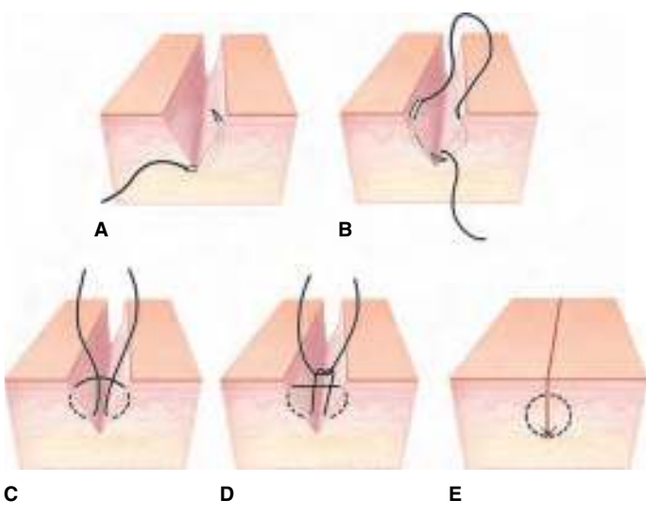


FIGURE 116-19. The buried (subcutaneous) knot stitch.

REINFORCING (RETENTION) SUTURES FOR WOUNDS UNDER TENSION

Reinforcing or retention sutures are particularly useful for wounds in which the edges are widely separated or where the skin is too atrophic to approximate without the suture cutting through the skin. The reinforcing sutures help to decrease the tension on the wound by providing more support for the wound edges. Reinforcing sutures can be placed using sterile buttons, Perchick buttons (Angiodynamics, Latham, NY), or rubber tubing (**Figure 116-20**). Heavy sizes of nonabsorbable suture materials are used for reinforcing sutures. This is not for strength but to avoid the finer suture from cutting through the tissue.

Ideally, a double-swaged (needle) suture should be used to place suture from the inside of the wound toward the outside skin to avoid pulling potentially contaminated epithelial cells through the wound. The stitch is placed like the horizontal mattress stitch and sterile buttons or rubber tubing is used to achieve approximation to a point where the wound edges can be closed without significant tension (**Figures 116-20*B* and 116-20*C***). **Do not attempt to appose the wound edges when using retention sutures.** Appose the remaining skin edges with simple interrupted, vertical mattress, or horizontal mattress stitches. The reinforcing sutures should remain in place after the skin sutures are removed. The reinforcing sutures should be removed after the wound has healed and gained significant tensile strength.

SUTURE REMOVAL TECHNIQUES

Remove sutures as soon as possible to avoid the possibility of infection and prevent the formation of suture marks. However, if they are removed too early, wound dehiscence may occur. Simple interrupted sutures should be cut at the end away from the knot and then pulled out (**Figure 116-21*A***). This helps to prevent the outer contaminated portion of the suture from passing back through the wound. In order to remove a running simple or running-locked stitch, grasp the knot at the end of the closure and cut each loop (**Figures 116-21*B* and 116-21*C***). Pull out each individual suture piece. Vertical and horizontal mattress sutures can be removed in much the same way as the simple interrupted stitch (**Figures 116-21*D* and 116-21*E***).

Patients often take out their own sutures, much to the dismay of Emergency Physicians. This is understandable when you think of the time it takes to go somewhere just to get the sutures removed in 2 minutes. An interesting study looked at sending the patient home with a suture removal kit and suture removal instructions.[14] Most patients were willing to remove their own sutures. The ones that removed the sutures did so without difficulty or complications.

TISSUE ADHESIVE CLOSURE (CYANOACRYLATES)

Tissue adhesives (skin glues) are best used to close low-tension, small, straight-edged, and superficial wounds (**Figure 116-22**). They should not be used for lacerations that are bleeding, lacerations over joints, or lacerations under tension. There must be adequate hemostasis and the tissue must be as dry as possible. The major advantage to the use of tissue adhesives is speed. Wounds can be repaired quickly and without anesthesia. Other contraindications to this type of closure are angled or beveled wounds. Petroleum-based ointments or similar products will dissolve the tissue adhesive and should be avoided on this type of closure. Refer to Chapter 117 for a more complete discussion of tissue adhesives.

Tissue adhesives come in a variety of forms and applicator tips (**Figures 116-22*A* and 116-22*B***). Approximate the wound edges

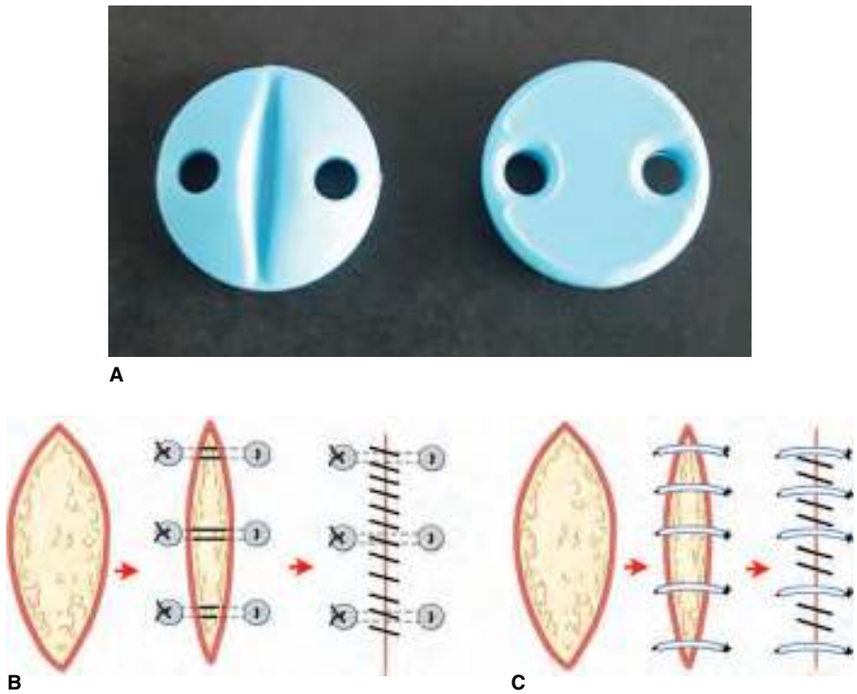


FIGURE 116-20. Reinforcing sutures for wounds under tension. ***A.*** The front and back sides of the Perchick button. Sterile buttons (***B***) or pieces of sterile rubber tubing (***C***) can be used to secure the suture and prevent injury to the soft tissues.

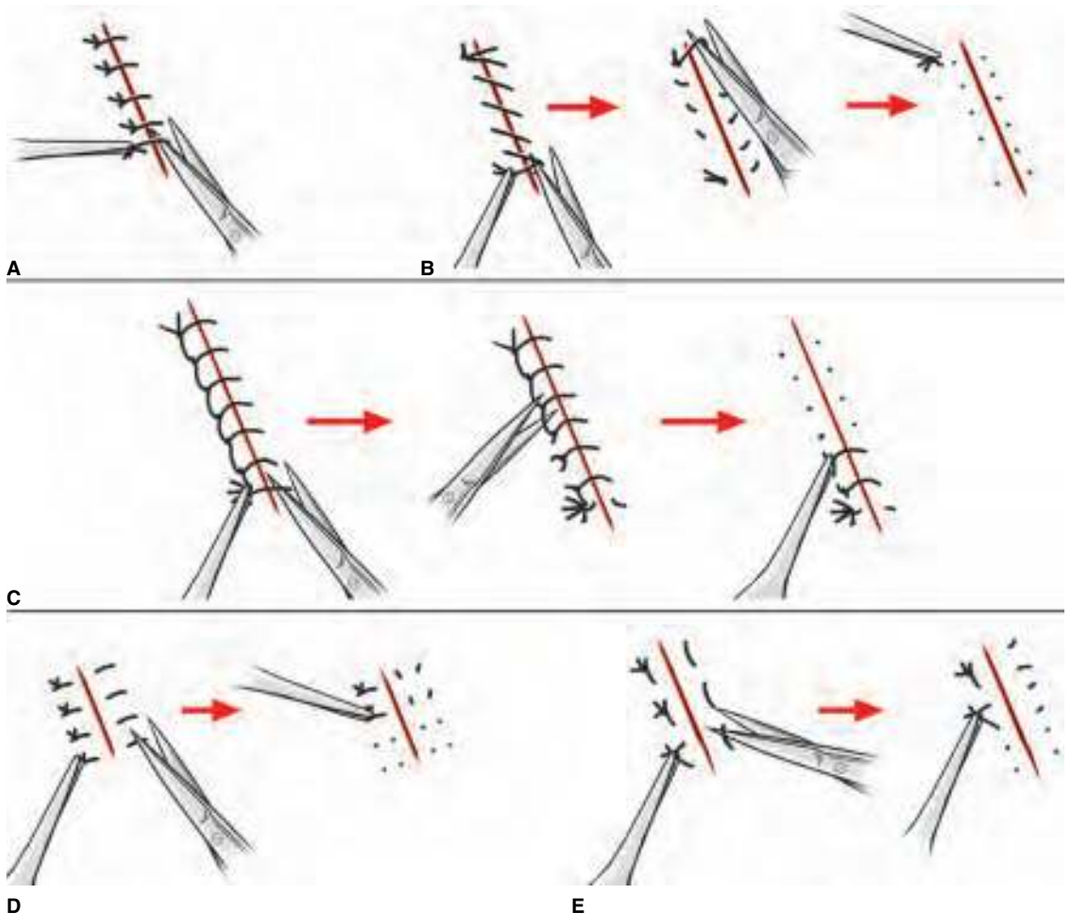
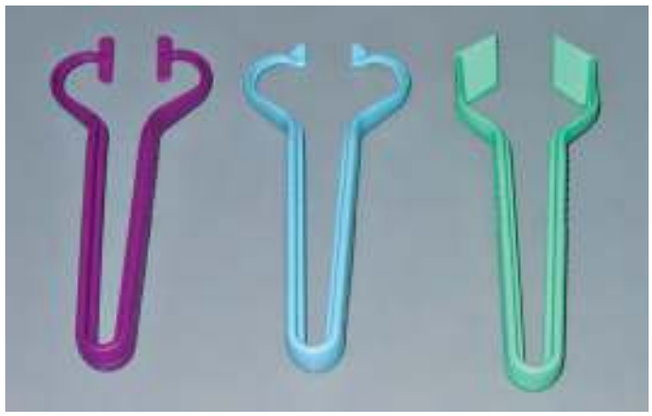


FIGURE 116-21. Suture removal techniques. ***A.*** Simple interrupted stitch. ***B.*** Simple running stitch. ***C.*** Running-locked stitch. ***D.*** Vertical mattress stitch. ***E.*** Horizontal mattress stitch.

A

B



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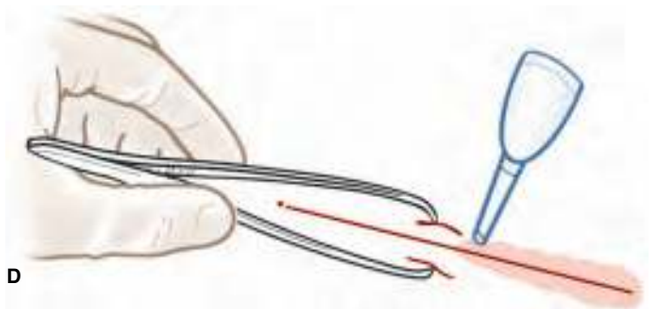

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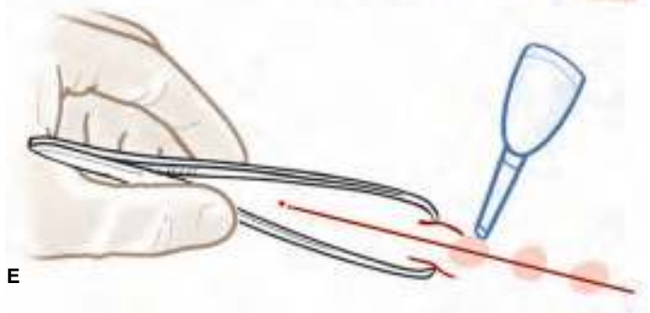

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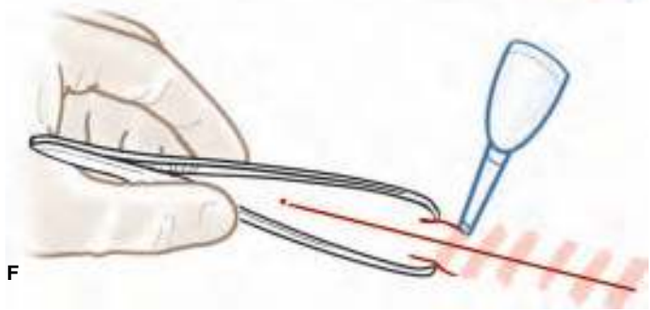


F

FIGURE 116-22. Laceration repair with cyanoacrylate tissue adhesive. ***A.*** Several examples of tissue adhesive. From left to right: Dermabond ProPen, SurgiSeal, Indermil Loctite, and Liquiband Flow Control. ***B.*** Dermabond Prineo System. (© 2016 Ethicon, Inc. Reproduced by permission.) ***C.*** Commercially available wound forceps (Bionix Development Corp., Toledo, OH). ***D.*** Tissue adhesive applied continuously over the laceration. ***E.*** Tissue adhesive applied in spots over the laceration. ***F.*** Tissue adhesive applied across the laceration.

with forceps. Commercially available, disposable, single-patient-use tissue forceps can be used (Bionix Development Corp., Toledo, OH). These are specifically designed to approximate the wound edges prior to using cyanoacrylates (**Figure 116-22*C***). Apply the adhesive in two or three layers along the wound edge (**Figure 116-22*D***). The adhesive may also be applied in spots over the laceration (**Figure 116-22*E***) or across the laceration, like wound tape (**Figure 116-22*F***). Droplets or lines should be placed 0.5 cm from each other. Support the wound for 30 to 60 seconds while the adhesive dries.

The Ethicon Dermabond Prineo (**Figure 116-22*B***) is a modification to the use of tissue adhesives. It is a skin closure system that consists of a flexible, polyester, self-adhering mesh and tissue adhesive pen. This system is supposed to provide a strength greater than suture and staples.

SKIN CLOSURE TAPES

Skin closure tapes (e.g., Steri-Strips) are used to close very-low-tension wounds that are tidy and small.[15] They can be used as the primary closure technique for superficial wounds (**Figure 116-23**) or they can provide reinforcement after sutures have been placed (**Figure 116-24**).[16] Skin tapes are easy to use and can be placed relatively quickly. They do not leave suture marks and have no

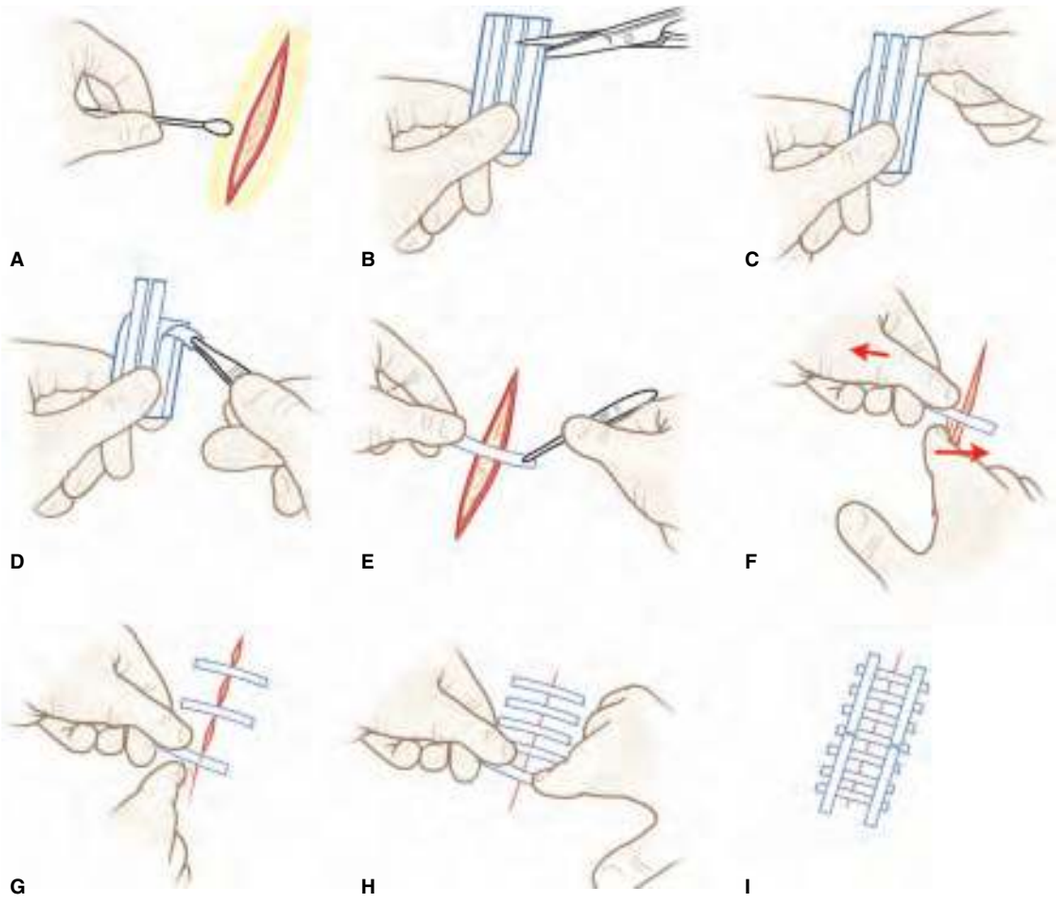


FIGURE 116-23. Skin closure tapes to primarily close a laceration.

skin reactivity. They do not improve cosmesis or decrease scar width.[17]

Skin closure tapes should not be used in wounds where the edges are widely separated or on parts of the body where there is movement or moisture. This technique does not work well on irregularly shaped wounds or wounds where there will be a propensity for swelling of the wound edges. Care should be taken in using these tapes in a child. If they are not secured properly, the child may remove them prematurely.

After the initial cleansing of the skin, clean the skin surface with acetone or alcohol to remove any surface oils. Allow the skin to dry. Apply benzoin solution or gum mastic (e.g., Mastisol) to the skin on both sides of the wound (**Figure 116-23*A***). Allow 60 to 90 seconds for the liquid benzoin to dry and become tacky. Cut the skin closure tapes to the proper length (**Figure 116-23*B***). Gently tear the end-tab off the back of the card to prevent the strips from deforming (**Figure 116-23*C***). Remove a strip from the card (**Figure 116-23*D***). Firmly secure the tape to one side of the wound (**Figure 116-23*E***). Use the nondominant hand to appose the wound edges as the tape is brought over and secured to the skin on the opposite wound edge (**Figure 116-23*F***). Place additional tapes at 2 to 3 mm intervals until the wound edges are apposed (**Figures 116-23*G* and 116-23*H***). Place pieces of tape across the tape edges to prevent premature removal and skin blistering from the tape ends (**Figure 116-23*I***).

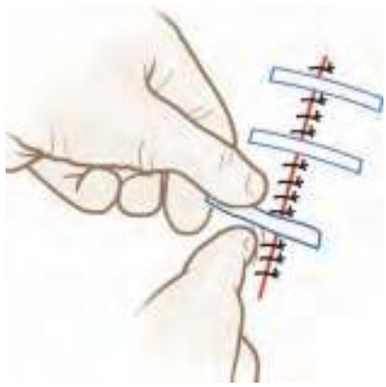


FIGURE 116-24. Skin closure tapes can provide reinforcement for sutures.

Skin closure tapes may be placed over a sutured laceration (**Figure 116-24**). The tapes will provide additional support to the wound edge and help to prevent dehiscence. This technique is especially useful in areas of cosmetic concern, such as the face.

The skin closure tapes should remain in place for at least as long as the sutures. They must be kept dry to prevent them from coming off prematurely and the wound from dehiscing. The wound should be observed daily for signs of infection.

Skin closure tapes may be placed across a wound when sutures or staples are removed. The tapes will maintain the apposition of the epidermis as the wound matures. Apply benzoin solution to the skin before removing the sutures or staples. Remove several sutures or staples and apply the skin closure tapes. Continue this process until all the sutures or staples have been removed and the wound is covered with skin closure tapes. Alternatively, remove all of the sutures or staples and then apply the skin closure tapes.

Suturing lacerations in a thin-skinned individual is often difficult. The skin frequently tears as the wound is approximated and the suture is tied. The use of skin closure tapes can facilitate wound closure by strengthening the skin edges, and allow for a more secure wound closure.[18-20] Clean, prep, and anesthetize the skin. Apply benzoin solution to the skin adjacent to and on both sides of the laceration. Allow the benzoin to sit and become tacky. Apply skin closure tapes over the benzoin on both sides of the laceration. Suture the laceration, ensuring that the needle enters the skin on one side of the laceration and exits the skin on the other side of the laceration, through the skin closure tapes on each side. Remove the skin closure tapes at the time of suture removal.

Two advanced skin closure tape-based systems are the ClozeX (Clozex Medical LLC, Wellesly, MA) and the 3M Steri-Strip S Surgical Skin Closure (3M Healthcare, St. Paul, MN). These are nonlatex, disposable, single-patient-use, transparent, adhesive-based wound closure devices for the primary closure of lacerations and wounds not under tension. They align the wound edges and provide good cosmetic results. They come in a variety of sizes, ranging from 10 to 100 mm.

STAPLE CLOSURE

Stapling is a rapid closure technique that is useful for superficial scalp lacerations and linear lacerations of the trunk and extremities.[21] Staple closure of wounds is cosmetically equal to sutures.[21] Staples should not be used on the face, neck, hands, or feet. These areas have little subcutaneous tissue and the staples can damage underlying structures. Staples should also be avoided, if possible, in any area of the body that will be exposed to computed tomography (CT) or magnetic resonance imaging (MRI) as they will cause artifact interference on the scan. The staples are made of an inert material, which helps to decrease tissue reactivity. Staples should not be used for wounds that are crush wounds, infected, irregular, macerated, over bony prominences, or under tension.

The skin stapler is a simple device (**Figure 116-25**). It is a single-patient-use, sterile, disposable unit that is preloaded with staples. It is grasped and held with one hand. When the handle is squeezed, a staple is inserted into the tissue. The stapler automatically loads the next staple after one staple is discharged. Skin staplers typically have 10 or 35 preloaded staples.

Prepare the wound for stapling. Place deep sutures to close the subcutaneous tissue and, if the wound is gaping, bring the wound edges into apposition. Approximate the skin edges with the dominant hand (**Figure 116-26*A***). Evert the wound edges with a forceps held in the nondominant hand (**Figure 116-26*A***). Grasp the stapler with the dominant hand. Gently place the skin stapler over the laceration (**Figure 116-26*B***). Start at one end of the laceration and work toward the opposite end. **Do not indent the skin with the stapler, as this will cause the staples to be placed too deep.** Align the arrow on the front of the stapler over the laceration (**Figure 95-26*C***). Squeeze the handle of the stapler. A plunger will advance a staple into the wound margins (**Figure 95-26*D***). An anvil will bend the staple into a square or rectangular shape to secure the staple (**Figure 95-26*E***). Continue to evert the wound edges and apply staples every 3 to 5 mm until the wound is approximated and without any gaps (**Figure 95-26*F***). A small space will be visible between the skin surface and the staple if it is properly positioned. If the staple is against the skin, it has been placed too deep. Remove the staple and replace it.

There are a few complications associated with staple use. Their removal can be uncomfortable or difficult. Minor bleeding can occur from the holes after the staples are removed. Staples placed on the face, feet, hands, and neck can damage superficial subcutaneous structures (e.g., blood vessels, muscles, nerves, and tendons). Improper wound eversion can result in wound dehiscence upon staple removal, larger scars, and poor wound healing. Staples can cause larger and more prominent skin marks and subsequent scarring when compared to sutures.

STAPLE REMOVAL

Staples should remain in place for approximately 5 to 10 days, the same amount of time as sutures. They can remain longer if placed over a joint or in cases of slow wound healing. The staple remover is a disposable, sterile, single-patient-use device (**Figure 116-27*A***). It is made of metal or plastic with metal tips. The lower jaw of the stapler has two upwardly angled metal prongs (**Figures 116-27*B* and 116-28**). The upper jaw of the stapler is a flat piece of metal. Insert the prongs of the lower jaw of the staple remover between the staple and the skin (**Figure 116-28*A***). Close the handles of the staple remover. This will cause the upper jaw to compress the center of the staple and the arms of the staple to withdraw from the skin (**Figure 116-28*B***). Lift the staple remover and staple off of the skin. Discard the staple and continue the process until all the staples have been removed. **A patient who plans to follow-up in a clinic or office should be given a staple remover to take with them, as many clinics and offices do not routinely stock these devices.**

A B

FIGURE 116-25. Examples of two styles of skin staplers.

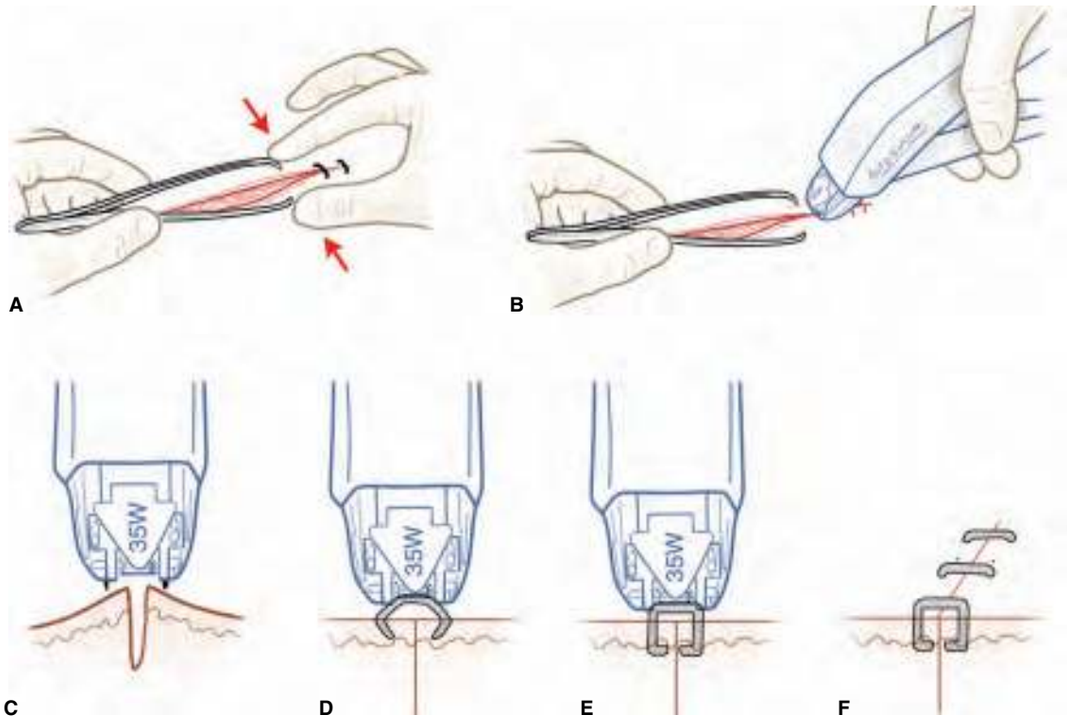
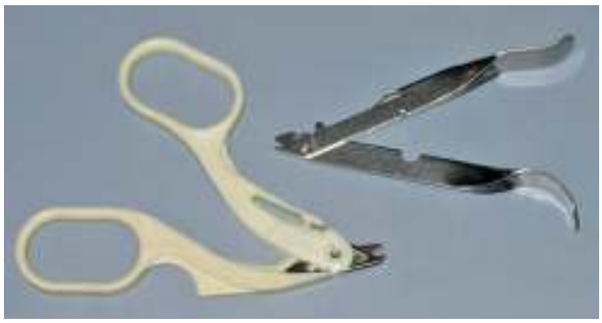


FIGURE 116-26. Laceration repair with staple closure. ***A.*** The wound edges are apposed and everted. ***B.*** The stapler is applied over the laceration. ***C.*** The stapler is applied over the everted wound edges. ***D.*** The plunger advances the staple into the wound margins. ***E.*** The anvil bends the staple into shape. ***F.*** The final product.


A

B

FIGURE 116-27. The staple remover. ***A.*** Overview. ***B.*** The tip with the jaws open.

HAIR APPOSITION

Scalp wounds can be closed using hair-tying, also known as the hair apposition technique (HAT).[22-27] This technique is relatively painless, does not usually require anesthesia, results in a shorter procedure time, eliminates the need for staple or suture removal, is cost-effective, and the wound outcome is similar or superior to sutures.[22,23,26] This technique should not be used on wounds under tension, wounds with ongoing hemorrhage, wounds that are grossly contaminated, or if the hair is less than 3 cm in length.

Clean, prep, anesthetize, and dry the laceration and surrounding skin. Start at one end of the laceration and grasp three to four hairs on each side of the laceration (**Figure 116-29*A***). Twist the

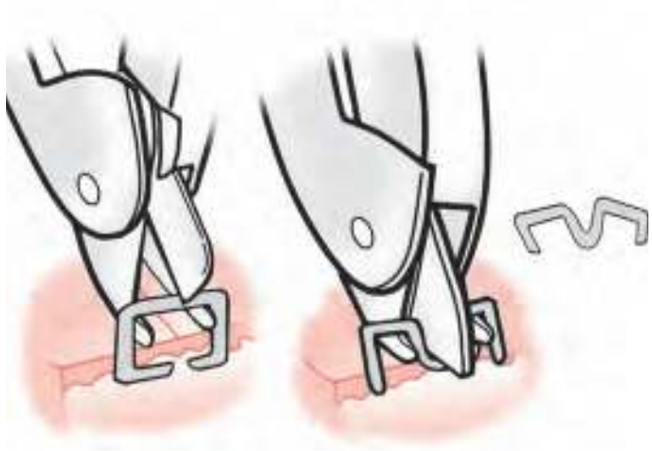

A B

FIGURE 116-28. Staple removal. ***A.*** The lower jaw of the staple remover is placed under the staple. ***B.*** The upper jaw compresses the center of the staple and allows the staple arms to exit the skin.

FIGURE 116-29. The hair apposition technique (HAT) to close a scalp laceration. ***A.*** Grasp hairs on each side of the laceration. Twist the hair strands on each side of the laceration to form a single "rope." ***B.*** Cross the hair ropes to opposite sides of the laceration. ***C.*** Twist the two "ropes" of hair together to close the wound and appose the wound edges. ***D.*** Apply tissue adhesive to the twisted hair and apposed skin segment of the laceration.

hair strands on each side of the laceration to form a single "rope." Tie the two "ropes" of hair together to close the wound and appose the edges (**Figure 116-29*B* and 116-29*C***). Use a hemostat and an instrument tie to make the process simpler and easier. Continue this process until the entire laceration is closed with hair ties. As the laceration heals, the hair knot will grow away from the wound edges. The hair knot can be cut off by a family member, friend, or primary care provider in 2 to 4 weeks.

An alternative HAT uses tissue adhesive instead of making a hair knot (**Figure 116-29**).[26] Clean, prep, anesthetize, and dry the laceration and surrounding skin. Start at one end of the laceration and grasp three to four hairs on each side of the laceration. Twist the hair strands on each side of the laceration to form a single "rope." Twist the two "ropes" of hair together to close the wound and appose the edges. Apply tissue adhesive to the twisted hair and apposed skin segment of the laceration (**Figure 116-29*D***). Allow 30 seconds for the tissue adhesive to dry. Apply a second layer of tissue adhesive and allow it to dry. Continue this process until the entire laceration is closed. This method does not require the later cutting away of the hair knot due to the tissue adhesive breaking down in 7 to 10 days.[26,28] Unfortunately, this technique may not produce as much hemostasis or wound eversion as tying a hair knot.[26] Refer to Chapter 117 for more details regarding tissue adhesives.

MISCELLANEOUS WOUND CLOSURE DEVICES

ABSORBABLE WOUND CLOSURE DEVICES

The V-Loc (Covidien, Norwalk, CT) is a disposable, single-use, unidirectional barbed wound closure device (**Figure 116-30**). It comes in three forms; two are absorbable (**Table 116-1**) and one is nonabsorbable (**Table 116-2**). The proximal end is the suture needle attached to an absorbable barbed suture. The distal end is a fixed loop of the suture material. This self-anchoring loop and barb combination eliminates knot tying when closing wounds. It is placed similar to a running subcuticular stitch. The barbs are circumferentially distributed on the suture strand and spread tension evenly across the wound. This device is mostly used for postoperative skin wounds but may become more popular in the Emergency Department. A similar device is the Stratafix (**Figure 116-31**) (Ethicon, Neenah, WI). Both are modifications of suture.

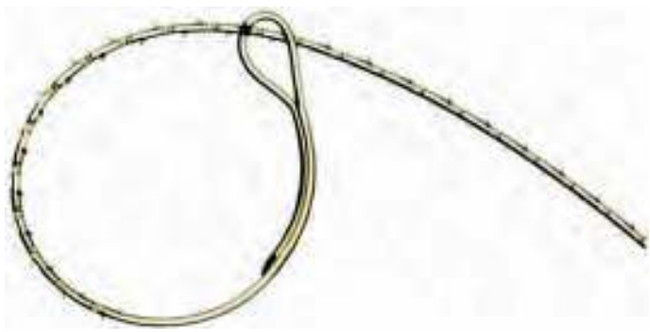


FIGURE 116-30. The V-Loc Wound Closure Device. (Photo courtesy of Covidien Inc.)

INSORB SUBCUTICULAR SKIN STAPLER

The Insorb subcuticular skin stapler (Incisive Surgical, Plymouth, MN) combines absorbable sutures and a device similar to a skin

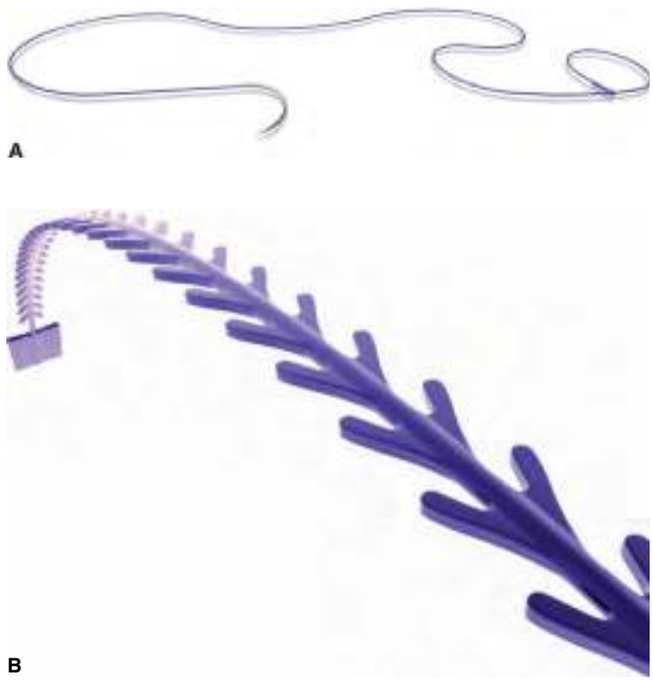


FIGURE 116-31. The Stratafix Wound Closure Device. ***A.*** Overview. ***B.*** Close up of the barbs. (© 2016 Ethicon, Inc. Reproduced by permission.)

A

B

C

D

E

FIGURE 116-32. The Insorb subcuticular skin stapler. ***A.*** The staple unit. ***B.*** The absorbable staple resting on a fingertip. ***C.*** Artist illustration of the unit in action. The inset shows the relationship of the staple to the subcutaneous tissues. ***D.*** The unit closing a laceration. ***E.*** Stapling a laceration with the Insorb Shorty. (Photos courtesy of Incisive Surgical Inc.)

stapler (**Figure 116-32**).[29,30] It has the advantages of a subcutaneous closure with the speed of a stapler. This is a sterile, single-patient-use device that deploys up to 20 absorbable subcutaneous staples (**Figure 116-32*A***). The subcutaneous staples are horseshoe shaped (**Figure 116-32*B***). The device allows wound eversion with no external sutures or metal staples that require later removal (**Figures 116-32*C* and 116-32*D***). It also comes in a Shorty version that is smaller and easier to hold (**Figure 116-32*E***). The final cosmetic results are similar to sutures or skin staples.[31] The company also sells a reusable three-arm proprietary forceps to make wound approximation easier for one person.

DYNACLOSE DYNAMIC TISSUE SYSTEM

Dynaclose Dynamic Tissue System (Southmedic, Ontario, Canada) was developed for use by Surgeons in the Operating Room (**Figure 116-33**). Dynaclose provides an easy, painless, and noninvasive method to close retracted or dehisced wounds up to 5 cm in width. It acts dynamically, moving with skin as it is stretched, while always providing a consistent appositional force. The clear elastomeric strip is anchored by an adhesive fabric tape on either side (**Figure 116-33**). The Dynaclose looks similar to larger Steri-Strips. It can be used in the Emergency Department for wounds

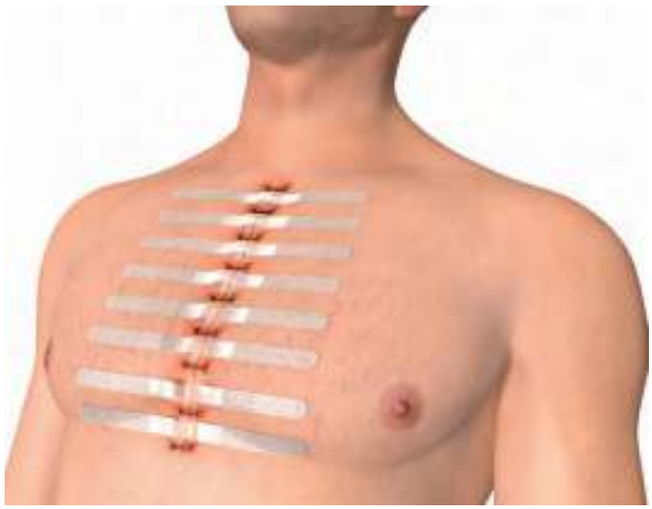


FIGURE 116-33. The Dynaclose Dynamic Tissue System. (Photo courtesy of Southmedic.)

under tension. Apply the Dynaclose strips to approximate the wound edges before suturing. It can also be applied after suturing to reinforce the repair.

ZIPLINE SKIN CLOSURE DEVICE

The Zip Surgical Skin Closure Device (Zipline Medical, Campbell, CA) is a noninvasive alternative to staples, sutures, and glue for the skin (**Figure 116-34*A***). A strong yet gentle hydrocolloid skin adhesive attaches the device on either side of the wound (**Figure 116-34*B***). Integrated adjustable straps are tightened to close the wound. The straps are also reversible if the wound is overtightened during closure. The excess straps are cut with scissors (**Figure 116-34*C***). This prevents patient tampering and disengagement. The device is flexible to move with body movement. It is typically kept on the wound for 7 to 14 days and can be removed at home by the patient. There are several benefits to this device. They include a shortening of the procedure time, a simple application process, no need for injected anesthesia, minimization of needle anxiety, a large force distribution area on the skin, and no need for follow-up visits for suture removal. It is useful for elderly or thin skin that may be challenging to suture. The device can be applied to a curved incision and several devices may be applied to very long incisions. The device may also be cut with scissors for shorter incisions. It is available in four lengths, suitable for closure of incisions up to 4, 8, 16, and 24 cm in length.

FUTURE INNOVATIONS

In the near future, there may be innovations available to the Emergency Physician.[32,33] An automated suturing device being developed by Sutrue streamlines the suturing process. It is available in multiple needle sizes. This device has the advantages of preventing needlestick injuries, speed of suture placement, and no variation in placement, and has a backward option. Another device heat seals straight wounds very quickly.

SUMMARY

There are multiple techniques available for closing wounds. The principles and techniques discussed will help to provide the most appropriate closure for the various types of wounds that are seen in the Emergency Department. Care should be taken to provide the best closure possible to provide good cosmesis and avoid complications.

A

B

C

FIGURE 116-34. The Zip Surgical Skin Closure Device. ***A.*** An example of the 4 cm long device. ***B.*** The device on a skin laceration. ***C.*** The skin wound closed and the device trimmed. (Photos courtesy of Zipline Medical.)

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117 Tissue Adhesives for Wound Repair

Hagop M. Afarian

INTRODUCTION

The year 1942 marked the discovery of cyanoacrylate, the chemical found in adhesives such as Superglue.[1] The use of cyanoacrylates for wound closure has been described since the 1960s when it was first assessed for military use. It was not until 1998 that *N*-2-octylcyanoacrylate (i.e., Dermabond) was approved by the Food and Drug Administration (FDA) for use in the United States. Tissue adhesives have since redefined the overall approach to laceration repair, especially in the Emergency Department. Their ease of use, relative painlessness, and simplicity of aftercare make it an ideal tool for small straight wounds and use in children.[2,3] There has been an increase in companies producing topical tissue adhesives for wound closure (**Table 117-1**).

ANATOMY AND PATHOPHYSIOLOGY

Cyanoacrylates are a monomer liquid. They polymerize when activated by water via an exothermic reaction to form a strong bond. Heat is released by this polymerization reaction and may cause some discomfort. Cyanoacrylates are classified as either butyl or octyl based on the side chain (**Table 117-2**).[4,5] Butyl cyanoacrylates have short and straight side chains. This allows them to form bonds that are poorly flexible, strong, and tight. Unfortunately, these bonds can become brittle and fracture. Butyl cyanoacrylates are best suited for short lacerations under no tension. Examples of butyl cyanoacrylates include Histoacryl (B. Braun, Bethlehem, PA) (**Figure 117-1**) and Indermil (Syneture, Norwalk, CT) (**Figure 117-2**). Octyl cyanoacrylates have longer side chains. This allows them to form bonds that are flexible, less likely to fracture, and strong. Octyl cyanoacrylates can be used on lacerations of any length. Examples of octyl cyanoacrylates include Dermabond (Ethicon Corp., Norwood, MA) (**Figure 117-3**), Exofin (Chemence Medical Inc., Alpharetta, GA) (**Figure 117-4**), and SurgiSeal (Pfizer Inc., New York, NY) (**Figure 117-5**). Newer agents are a combination of the strong and fast-setting butyl cyanoacrylates with the flexibility of the octyl cyanoacrylates. An example is LiquiBand (Advanced Medical Solutions Inc., Plymouth, United Kingdom) (**Figure 117-6**).

The nonmedical and medical adhesives contain similar ingredients. The differences between these two types are sterile production, sterile packaging, and the attached alcohol chain in medical grade tissue adhesives. Converting a methyl to an octyl group reduces the heat produced by polymerization and decreases the amount of direct tissue inflammation caused by the breakdown products of the adhesive.[1,6,7]

TABLE 117-1 Some Commercial Tissue Adhesives

Tissue glue	Name	Manufacturer
Butyl cyanoacrylate	Glubran 2	GEM SRL
	Histoacryl	B. Braun
	Indermil	Syneture
	LiquiBand	Advanced Medical Solutions
	SkinLink	Advanced Medical Solutions
Ethyl cyanoacrylate	EpiGlu	Meyer-Haake
Octyl cyanoacrylate	Dermabond	Ethicon
	Exofin	Clemence Medical
	SurgiSeal	Pfizer

TABLE 117-2 Comparison of Butyl Cyanoacrylate and Octyl Cyanoacrylate

Characteristic	Butyl cyanoacrylate	Octyl cyanoacrylate
Degradation	Slow	Slower
Exothermia	Most	Less
Flexibility	High	Higher
Setting	60–90 seconds	Quicker
Strength	Strong	Strongest

There are many advantages to tissue adhesives.[8-10] The major advantage to the use of tissue adhesives is speed. Wounds can be repaired quickly and without anesthesia. Tissue adhesives have been shown to offer similar wound closure and cosmetic results as adhesive strips (e.g., Steri-Strips) and sutures.[11-14] The initial tensile strength of wounds repaired with tissue adhesives are not equivalent to wounds closed with sutures.[15,16] Within 7 days any differences in tensile strength are no longer present.[15,16] The cost to the patient is less for lacerations repaired with tissue adhesives compared to suturing.[12,17] This accounts for materials, physician time, procedure time, and repeat visits for suture removal.[14,17] There is less need for the painful injection of local anesthetic solution. The risk of a needle-stick injury to the Emergency Physician is decreased when not suturing. An additional benefit of tissue adhesives is that they provide an occlusive covering for wounds, keep wounds moist, keep wounds water tight, and provide protection from invading microbials.[18] Wounds closed with tissue adhesive do not require routine follow-up like those sutured closed for suture removal.

There are also disadvantages to using tissue adhesive.[3,10] There is a variability among Emergency Physicians using the adhesive. Their skill varies as well as their experience. It may be difficult to appose skin edges and evert wound edges. Wounds may dehisce over time because the effectiveness of tissue adhesives over time is lost. There is poor reliability among tissue adhesive when compared to sutures. Placement of and removing topical adhesives can damage the epidermis.

INDICATIONS

Tissue adhesives are best used to close low-tension, small, straight-edged, and superficial wounds. Use precautions when using tissue adhesives near the eye. The liquid adhesive can run into the eye. The tissue adhesive can cause iatrogenic sealing of the eyelids if the eyelid margins are not protected.[19] **Tissue adhesive may be used for wounds that are deep or under tension as a superficial closure layer only after the subcutaneous layer has been repaired to bring the wound edges together and relieve tension.**

FIGURE 117-1. Histoacryl (B. Braun, Bethlehem, PA).

FIGURE 117-2. Indermil (Syneture, Norwalk, CT).

FIGURE 117-3. Dermabond Advanced (Ethicon Corp., Norwood, MA).

FIGURE 117-4. Exofin (Clemence Medical Inc., Alpharetta, GA).

FIGURE 117-5. SurgiSeal (Pfizer Inc., New York, NY). The stylus pen (left), teardrop applicator (right), and twist top (bottom).

Most wounds on the head, neck, proximal extremity, and torso can be closed with tissue adhesive. Flap-type lacerations and lacerations of thin skin can be closed where the use of sutures can compromise the skin. Long lacerations can be divided into segments and each segment closed as if it were a small laceration.[13] Consider using tissue adhesives on abrasions that keep oozing.[20]

CONTRAINDICATIONS

Tissue adhesives should not be used on wounds that are actively infected, heavily contaminated, greater than 6 to 12 hours old, from a crush injury, punctures, on the eyelids or surrounding skin, or from bites. **Tissue adhesives can only be used on the skin surface and not within wounds, on mucous membranes, or on mucocutaneous junctions (e.g., the mouth and lips).** Do not use tissue adhesives on patients with a known hypersensitivity to cyanoacetate and formaldehyde, as cyanoacrylates degrade into these byproducts. It is recommended that tissue adhesives not be used in areas of the body that are exposed to heavy moisture (e.g., the perineum and axilla) and parts of the body prone to frequent movement (e.g., hands, feet, and over joints).[21] **Wounds must be dry.** Do not use tissue adhesives on wounds that are actively bleeding or oozing. Tissue adhesive use in these areas may lead to wound dehiscence as the adhesive cracks and/or peels.[21] Stop the bleeding with direct pressure or the injection of local anesthetic solution with epinephrine prior to the application of tissue adhesive. Tissue adhesive may be difficult to use in areas covered densely with hair (i.e., the scalp and axilla) since the tissue adhesive will not bond adequately to the skin. Tissue adhesives are not recommended for stellate wounds because of the difficulty of adequately approximating the many wound edges. Other contraindications to this type of closure are angled or beveled wounds, unless deep sutures are first placed to approximate the wound edges and relieve any tension.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Gloves
- Tissue adhesive
- Wound cleaning and irrigation supplies (Chapter 114)
- Forceps
- Petroleum jelly
- Acetone or nail polish removal pads
- Gauze squares
- Occlusive dressing (e.g., Tegaderm)

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain a signed consent for the procedure. Anesthetize the wound. Cleanse the wound and surrounding skin of any blood, dirt, and debris. Irrigate the wound with normal saline or tap water. Consider using a wound irrigation device as described in Chapter 114 if the wound is dirty. Injuries that require substantial cleaning may not be good candidates for tissue adhesive closure. Inspect the wound for any retained foreign bodies or injuries to deep structures. **All bleeding must be controlled prior to the application of the wound adhesive.** Repair lacerations with continued or heavy bleeding with sutures to achieve adequate hemostasis. Dry the skin surrounding the laceration with gauze squares.

FIGURE 117-6. LiquiBand (Advanced Medical Solutions Inc., Plymouth, UK).

TECHNIQUES

GENERAL TISSUE ADHESIVE TECHNIQUE

The general technique will be described. There are some differences in the type of applicator. Prepare the tissue adhesive. Some only require the removal of a twist-off plastic cap. Others are supplied in ampules that must be crushed and allowed to soak the foam tip of the applicator. Use the tissue adhesive immediately after opening the container as it dries within minutes and may not continue to flow freely for long. Tissue adhesives dispensers are available with a variety of applicator tips (**Figures 117-1 through 117-6**).

Approximate the wound edges with forceps. Commercially available, disposable, single-patient-use tissue forceps can be used (Bionix Development Corp., Toledo, OH). These are specifically designed to approximate the wound edges prior to using tissue adhesives (**Figure 117-7**). Alternatives to these devices are Adson forceps or using gloved fingers. Place a thin layer of tissue adhesive over the wound and extending 5 to 10 mm beyond the wound margins (**Figure 117-8*A***). The tissue adhesive may also be applied in spots over the laceration (**Figure 117-8*B***) or across the laceration like wound tape (**Figure 117-8*C***). Apply the droplets or lines of tissue adhesive approximately 0.5 cm from each other. **Hold the wound edges together for 30 to 60 seconds following the application of the first layer of tissue adhesive to allow for optimum polymerization.**

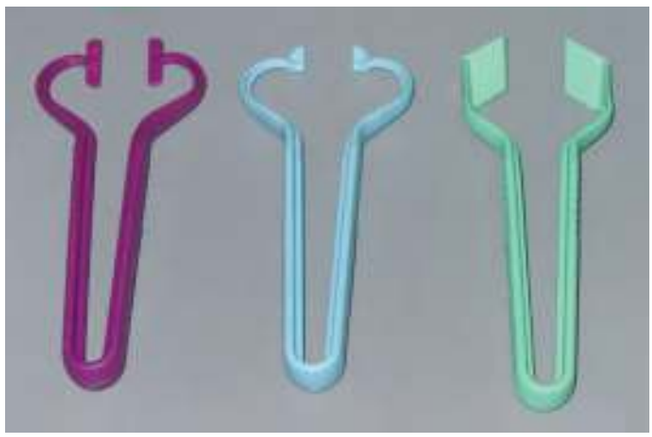


FIGURE 117-7. Commercially available wound forceps (Bionix Development Corp., Toledo, OH).

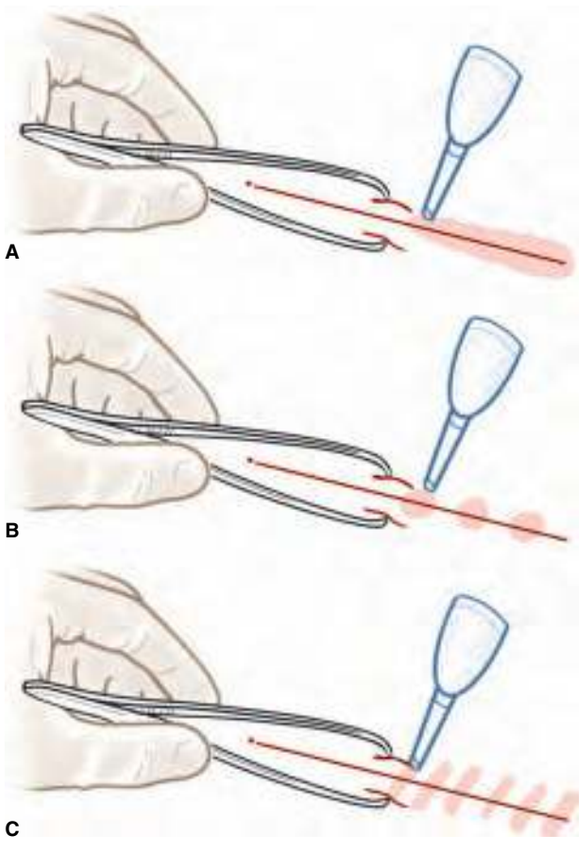


FIGURE 117-8. Laceration repair with tissue adhesive. ***A.*** Tissue adhesive applied continuously over the laceration. ***B.*** Tissue adhesive applied in spots over the laceration. ***C.*** Tissue adhesive applied across the laceration.

Apply a total of three to four thin layers of tissue adhesive. Allow each layer to dry for approximately 30 to 60 seconds before applying the next layer. Each successive layer will take longer to dry as the water available for the polymerization reaction has been covered by the previous layer. **Be careful to use only thin layers and not large drops of adhesive to assist drying time and prevent discomfort from any heat released by the exothermic polymerization reaction. Do not allow the tissue adhesive to flow into the wound**. It can remain in the wound long after the skin has healed leading to delayed healing, prolonged inflammation, and tattooing.[4]

Do not touch the layers of wound glue as they are drying to prevent cracking and inadvertent adhesion of gloves or foreign bodies to the wound. Gently peel gloved fingers away from the most recent layer before it dries if they or other foreign bodies have become attached to the wound. Use poly gloves if available. These are used by food service workers and tissue adhesive does not stick. Wipe away tissue adhesive applied to an area where it does not belong within 10 seconds.

PREVENTING THE TISSUE ADHESIVE FROM RUNNING

Techniques have been described for use when working around areas that must not contact the tissue adhesive (e.g., the eye). The first and easiest precautionary technique is patient positioning. Position the patient so that the tissue adhesive will not run away from the wound. Ideally, the wound surface should be parallel to the floor. Unfortunately, this is not always possible to accomplish. Alternatively, position the patient in a manner such that the wound rests below the sensitive area. Gravity allows any of the liquid tissue adhesive to run away from the area of concern. Avoid squeezing the tissue adhesive container too much to control the amount expressed. This can help to minimize any runoff. Consider placing a piece of gauze or a clear occlusive dressing (i.e., Tegaderm) over the area of concern to protect it from any runoff.

A barrier can be created between the wound and the sensitive area. Place a thin film of petroleum jelly between the wound and the sensitive area. This will ensure that if the tissue adhesive were to run, it will not spread beyond the barrier. Alternatively, apply the petroleum jelly in a wide circle surrounding the wound and create a valley for the tissue adhesive to fill if it runs. It may become difficult to hold the wound edges together in a small space covered with petroleum jelly. The oil-based petroleum jelly may prevent adequate binding of the tissue adhesive if the jelly gets on the skin immediately adjacent to the wound.

A final technique to keep the wound adhesive from running is the use of an occlusive dressing (e.g., a Tegaderm) (**Figure 117-9**).[22] Obtain an occlusive dressing large enough to cover the area in question (**Figure 117-9*A***). Fold the dressing in half (**Figure 117-9*B***). Cut a hemi-ellipse out of the dressing (**Figure 117-9*C***). Unfold the dressing and center the cut-out ellipse over the wound (**Figure 117-9*D***). Make sure that the newly created hole in the center is large enough to include the entire laceration and some surrounding skin. Cut additional material from the dressing if needed. Remove the protective tape from the back of the dressing and apply it to the skin. Approximate the wound edges and apply tissue adhesive (**Figure 117-9*E***). Cover the entire precut hole and some of the surrounding dressing with the tissue adhesive. Allow the layers of tissue adhesive to completely dry. When removing the dressing be careful not to disturb the freshly adhered tissue adhesive. A useful technique is to pull apart the dressing from the center outward, stretching it away from the edge of the adhesive. This allows it to release from the tissue adhesive without causing the adhesive to peel. The result is a well-demarcated, circular film of tissue adhesive overlying the closed laceration (**Figure 117-9*F***).

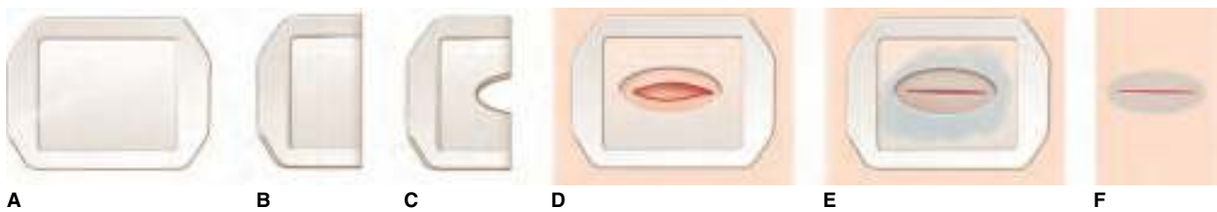


FIGURE 117-9. The use of an occlusive dressing for protection of nearby sensitive structures when using tissue adhesives. ***A.*** The occlusive dressing. ***B.*** The dressing is folded in half. ***C.*** A hemi-ellipse is cut out of the dressing. ***D.*** The dressing is opened with the cut-out ellipse centered over the wound. The protective tape is removed and the dressing adhered to the skin. ***E.*** The wound is approximated and tissue adhesive is applied. ***F.*** The result after the dressing has been removed reveals a well-circumscribed area of tissue adhesive.

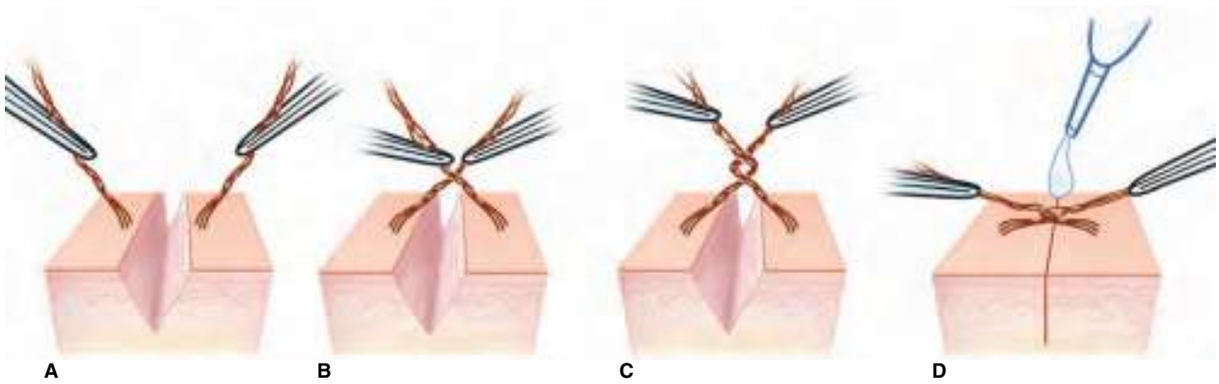


FIGURE 117-10. The hair apposition technique (HAT) to close a scalp laceration. ***A.*** Grasp 5 to 15 hairs on each side of the laceration. Twist the hair strands on each side of the laceration to form a single "rope." ***B.*** Cross the hair ropes to opposite sides of the laceration. ***C.*** Twist the two "ropes" of hair together to close the wound and appose the wound edges. ***D.*** Tissue adhesive is applied to the twisted hair and apposed skin segment of the laceration.

HAIR APPOSITION TECHNIQUE

Scalp wounds can be closed using hair-tying, also known as the hair apposition technique (HAT).[22-27] This technique is relatively painless, does not usually require anesthesia, results in a shorter procedure time, eliminates the need for staple or suture removal, is cost-effective, and the wound outcome is similar or superior to sutures.[23,24,27] Do not use this technique with wounds under tension, wounds with ongoing hemorrhage, wounds that are grossly contaminated, or if the hair is < 3 cm in length.

Clean, prep, anesthetize, and dry the laceration and surrounding skin. Start at one end of the laceration and grasp 5 to 15 hairs on each side of the laceration (**Figure 117-10*A***). Twist the hair strands on each side of the laceration to form a single "rope." Tie the two "ropes" of hair together to close the wound and appose the edges. Use a hemostat and an instrument tie to make the process simpler and easier. Apply tissue adhesive to the hair knot for added security. Continue this process until the entire laceration is closed with hair ties. The hair know will grow away from the wound edges as the laceration heals. The hair knot can be cut off by a family member, friend, or primary care provider in 2 to 4 weeks.

An alternative HAT uses tissue adhesive instead of making a hair knot.[27,28] Clean, prep, anesthetize, and dry the laceration and surrounding skin. Start at one end of the laceration and grasp 5 to 15 hairs on each side of the laceration with gloved fingers or a hemostat (**Figure 117-10*A***). Twist the hair strands on each side of the laceration to form a single "rope." Twist the two "ropes" of hair together to close the wound and appose the edges (**Figures 117-10*B* and 117-10*C***). **Do not wind the strands more than one turn to prevent increased tension and tissue necrosis.** Apply tissue adhesive to the twisted hair and apposed skin segment of the laceration (**Figure 117-10*D***). **Do not allow the tissue adhesive to run into the wound.** Allow 60 to 90 seconds for the tissue adhesive to dry. Apply a second layer of tissue adhesive and allow it to dry. Continue this process until the entire laceration is closed. This method does not require the later cutting away of the hair knot. This technique may not produce as much hemostasis or wound eversion as tying a hair knot.[27]

PRINEO SKIN CLOSURE SYSTEM

The Prineo Skin Closure System (Ethicon Corp., Norwood, MA) is a octyl-2-cyanoacrylate used with a self-adherent mesh (**Figure 117-11**).[8,29] The mesh is applied after the wound margins are apposed. The self-adherent mesh will hold the wound edges together and eliminates the need for manual apposition using fingers. This simplifies wound closure and gets the fingers out of the location of the wound. A single layer of tissue adhesive is needed due the mesh. Use of the Prineo Skin Closure System was faster and equivalent to using suture.[8,29] Patients can have allergic local reactions to the adhesive or the mesh.[30-32]

PEDIATRIC CONSIDERATIONS

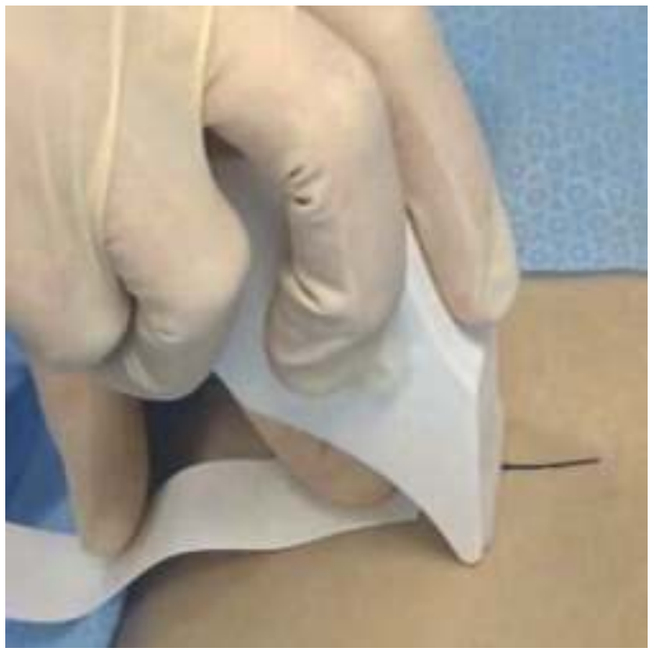
Pediatric patient movement is the factor that causes the greatest difficulties when working with wound adhesives. Tissue adhesives are most useful in the pediatric population, but this population has the greatest risk of movement. Movement will permit running of the adhesive and leakage onto uninvolved areas. Use extra caution when working around sensitive areas. Use a high-viscosity tissue adhesive to limit leakage when working around sensitive areas or on children who have trouble remaining still (**Figure 117-12**). Children may pick at the edges of the newly formed adhesive covering and cause it to become dislodged prematurely. Applying a dressing will help alleviate this risk. Consider using LET (lidocaine, epinephrine, and tetracaine) (Chapter 154) if anesthesia is required.[33]

ASSESSMENT

Assess the closed wound to ensure that the edges are approximated. Tissue adhesive that has adhered and dried on uninvolved areas may be removed using petroleum jelly or topical petroleum-based antibiotic ointment. Apply the oil-based jelly or ointment to the dried tissue adhesive and let it stand for 30 minutes. Gently peel away the tissue adhesive. Use acetone or nail polish remover pads to remove the tissue adhesive more quickly instead of waiting the time for the oil-based products to work.[34] **Be careful to not let acetone drip into the eye or on mucous membranes.**

Tissue adhesive occasionally does enter the eye or the eyelids become glued shut (**Figure 117-13**).[35,36] Tissue adhesives are not toxic to the globe and will not cause damage to the conjunctiva or cornea. **Do not attempt to pry open the eyelids or cut between the eyelid margins to separate the eyelids.** Apply an ophthalmic antibiotic ointment to the eyelids if they are sealed shut. Allow the ointment to sit for 30 minutes. Instruct the patient to open their eyelids. Gently wipe the ointment and adhesive from the eyelids if the patient can open their eyelids. Instruct the patient to apply the ophthalmic ointment 5 to 6 times a day at home and gently attempt

A



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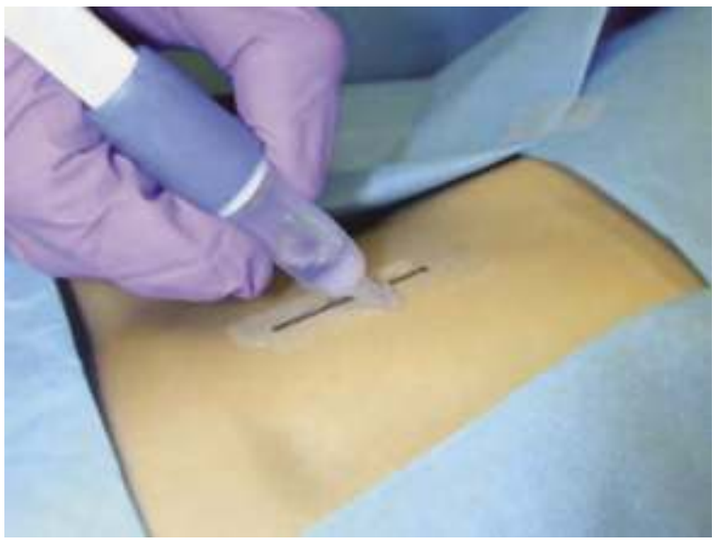

C

FIGURE 117-11. The Prineo Skin Closure System (Ethicon Corp., Norwood, MA). ***A.*** The parts of the system. ***B.*** Wound approximation and placement of the mesh. ***C.*** Application of the tissue adhesive. (Used with permission from reference 8.)

to open their eyelids if they cannot open their eyelids in the Emergency Department. Their eyelids will separate within 2 to 3 days.

AFTERCARE

Instruct the patient not to use oil-based substances on any wound repaired with tissue adhesives.[1] Tissue adhesive does not need to be removed. It will peel away within 5 to 10 days as new epithelial layers are formed below it. Wounds repaired with tissue adhesive may briefly contact water (e.g., showering) but should not be scrubbed clean. Do not soak the wound (e.g., a bath tub, pool, or spa). Instruct the patient to return immediately to the Emergency Department or to their primary care provider for any signs of a wound infection.

COMPLICATIONS

Many of the complications associated with the use of tissue adhesives are preventable. The Emergency Physician must choose the proper patient, the proper wound, and the appropriate wound location. These few things in association with the appropriate tissue adhesive application technique will avoid most complications. Tattooing of the scar can occur from the adhesive, a foreign body in the wound, or not cleansing the wound thoroughly.[37] The details of these have been described previously in the indications, contraindications, and techniques sections.

A certain percentage of repaired wounds will become infected. Minimize any infections by properly preparing the wound

FIGURE 117-12. High-viscosity tissue adhesive can be used routinely to prevent the adhesive from running and in children.

FIGURE 117-13. Iatrogenic eyelid closure after application of tissue glue above the eyelid. (Used with permission from reference 35.)

(Chapter 114). This includes the use of anesthesia if appropriate, wound irrigation, wound debriding, wound undermining, wound exploration for foreign bodies, and the use of deep sutures to release tension.

SUMMARY

Tissue adhesives have changed the face of laceration repair. Wounds are repaired more quickly with tissue adhesives and often at a lower cost compared to suturing. Patient satisfaction is increased. There are very few limitations to the use of tissue adhesives. Proper patient selection, proper wound selection, and proper wound preparation will minimize any complications.

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118 Advanced Wound Closure Techniques

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INTRODUCTION

Traumatic wounds or skin lacerations are among the most common injuries, occurring in people of all ages, that require evaluation and treatment in the Emergency Department. The result of many, if not all, wound closures is scar formation. Most wounds heal with a surprisingly pleasing cosmetic transformation from their initial presentations. It is not uncommon for some wounds to present complications during the healing period as well as to produce an undesirable scar. A systematic approach to wound management serves to help in deciding how to close complicated wounds, reduce the risk for infection, and minimize less favorable outcomes.

Wound management in the Emergency Department includes an assessment of the mechanism and conditions that were present at the time of injury. Initially, one must address the concerns of the patient, family members, or friends with a concise explanation of how the wound will be treated and what can be anticipated for aftercare. Many lawsuits and concerns of poor care evolve from poor cosmetic outcomes. **It is recommended that verbal wound care instructions be offered once wound closure is completed, in addition to giving the patient written discharge instructions.**[1]

Regardless of the severity of the wound or possible inherent complications associated with the injury, many patients are primarily concerned with the potential for scarring or disfigurement. Most patients expect cosmetic and functional perfection as an ultimate result after their wounds are treated and the healing process is completed. These expectations are often not clearly expressed during the evaluation and treatment in the Emergency Department. **The Emergency Physician must openly explain and discuss the fact that virtually no wound heals without a scar following wound closure.**[1,2] **A clear understanding of this is not to be used as an explanation for a poor outcome but to counter any misconception that a wound will heal to look exactly like the previously intact skin. Treatment is rendered to offer the best possible functional and esthetic outcome while reducing the risk of potential soft tissue infection.**

An overall plan of wound site preparation and closure will be needed to provide the greatest likelihood of a pleasing cosmetic result.[1] The mechanism of injury, severity of the wound, location of the wound, and presence or risk of necrotic tissue can all influence the risk of infection. Additionally, the decision of how to approach wound closure will be affected by the patient's skin type, age, gender, occupation, and hobbies.

Wound healing ultimately takes place over at least 6 to 9 months. Any wound presenting with concerns for a poor outcome or an obvious likelihood of wound revision in the future should be evaluated and treated by a Plastic Surgeon when possible.[1,2] All other wounds requiring complex closures should be properly assessed and treated by the Emergency Physician.

ANATOMY AND PATHOPHYSIOLOGY

To have a better understanding of scar tissue formation and the antecedent techniques of wound closure, the Emergency Physician should have knowledge of specific physiologic conditions and anatomic areas that may increase the chances of unfavorable scarring following wound repair.[2,3] The age of the patient and appearance of the patient's intact skin should be taken into consideration. The younger patient tends to heal more rapidly, while the older patient tends to have a more favorable cosmetic outcome with wound closure. Older patients have less overall elastic and subcutaneous tissue and more wrinkling, thus decreasing the tension on the healing wound and making scarring less noticeable.[2,3] Wrinkling, or lines of minimal tension, makes wound repair more technically challenging.

Suturing of asymmetrical, deep, or large wounds requires particular attention to the preexisting lines of minimal tension or lines of facial expression. Without properly addressing such preexistent anomalies, the cosmesis of wound repair can be grossly affected.[2,3] Scars from wounds closed perpendicular to preexisting functionally anatomic lines undergo repetitive physical stress and may result in hypertrophic scar tissue. With markedly less skin elasticity and subcutaneous fat, older patients will often experience more favorable cosmetic results from less complex wound closures. However, younger patients will benefit from more advanced wound closure techniques to properly close large or complicated wounds. Rotational and advancement flaps are frequently performed to make scarring less obvious when suturing across lines of tension.[3]

The type of skin, regardless of age, will affect scar formation.[3,4] Oily or hyperpigmented skin more frequently has poor scar tissue formation. This results in scars that are hypertrophic, deep, and asymmetrical. Consideration of wound outcome should be given to areas of the skin that are rich in sebaceous glands or simply hyperpigmented (e.g., from environmental exposure or ethnicity). Patients with underlying connective disease disorders or conditions with a high likelihood of concomitant vitamin deficiencies should also be scrutinized, as wound closure and healing may be compromised in such cases, resulting in highly variable and less predictable outcomes.

The mechanism of injury, including environmental exposure to underlying tissue, should not be overlooked so that adequate debridement and preparation may be done prior to a complex wound closure. This allows the Emergency Physician to better visualize the anatomic layers of the skin. It can be difficult to determine a clear delineation between the anatomic layers of the skin when the wound was a result of a crush injury, shredding mechanism, or any circumstance resulting in uneven or macerated wound edges. Delineate the pigmented epidermis from the thicker underlying dermis, especially when multilayer wound closures are required, as suturing may then become unnecessarily complicated and affect the overall integrity of the wound closure.

It should be noted that the literature supports a significant underutilization of multilayer closures, although these are often necessary. Single-layer closures and excessively large suture materials are the greatest causes of residual scar tissue.[1] It is recommended to prepare the wound edges by creating a bevel or undercutting of the wound margin to allow subtle epidermal eversion, thus augmenting the natural process of scar formation.[1,2] This allows the natural flattening and depression of the forming scar to occur without excessive depression from the wound margins.[2] This will also help to reduce the thickness of the scar tissue and decrease the refraction of light from the scar, making it less noticeable.

Depending on the presentation of the tissue defect, more than one wound closure technique may be used to adequately close a wound. Using more than one technique will help remove underlying tension and allow better approximation of the epidermis. With the help of specific camouflage techniques used in closing the epidermis, irregularly shaped wounds can heal with less obtrusive scarring. Familiarity with a few of these techniques and their application will allow the Emergency Physician to comfortably close the more challenging wounds encountered with expectantly more favorable cosmetic prognoses.

INDICATIONS

Advanced wound closure techniques are indicated for closing wounds with irregularly shaped defects or defects too large for primary closure. They can be used to close circular, square, elliptical, or asymmetrical skin defects. Advanced wound closure techniques are beneficial when there is a need to reduce skin tension and contracture, which are likely to result in hypertrophic scar formation.[3,4] Rotational and advancement flap techniques are useful in areas where tissue loss must be avoided and the undermining of wound edges must be minimized. This is often encountered with facial wounds in proximity to the eyelids, eyebrows, canthi, nasolabial folds, or lip borders. These techniques allow the initial shape of the wound to be altered such that there is reduced tension on the wound edges, which may then be closed simply.

CONTRAINDICATIONS

Specific wound closure techniques should take into account the potential for scar formation to occur in an undesirable location. This can happen when a wound must be elongated to create parallel lines and to decrease the tension on the wound edges. Elongation of a wound may bring it into proximity of other anatomic positions or landmarks, thus further complicating the healing process. If not planned well, excessively large defects may result, making it more likely that the scar will require later revision. More obvious conditions may exist that compromise complex wound closures. Particular attention must be given to crush injuries with devitalized or contaminated tissues. Severely contaminated wounds, including those with prolonged exposure, generally are at greater risk of infection with multilayer closures. Do not perform these techniques if the patient is at risk for poor wound healing (i.e., diabetes, poor vascular supply to the area, or prior radiation therapy to the area), keloid formation, or coagulopathic. Careful wound assessment may result in a decision to use simple approximation of the wound edges with close follow-up for ongoing wound care. Contraindications to complex wound closures will at times be reliant on temporal factors, such as the need to close a wound prior to the patient receiving surgical intervention for more life-threatening injuries. There must be a commonsense approach in deciding how to close more challenging wounds in the Emergency Department.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Needle drivers, 4.5 and 6.0 inch
- Skin hooks
- Scalpel blades (#10, #11, #15)
- Scalpel handles
- Iris scissors, straight 4 inch and curved 4 inch
- Suture scissors, 6 inch
- Forceps, toothed Adson
- Metzenbaum scissors, curved 6 inch
- Hemostats, straight 6 inch and curved mosquitoes
- Suture material, various sizes and shaped needles
- Skin closure tapes
- Benzoin solution, swabs or spray
- Tissue adhesive
- Tissue adhesive forceps
- Gauze 4×4 squares
- Overhead light source or headlight
- Loupes or magnifying glasses (optional)

The above equipment can be purchased in single-use, sterile, and disposable plastic surgery wound kits from several commercial manufacturers. These kits tend to be expensive and occasionally have a limited amount of equipment. Many hospitals package and sterilize their own wound repair kits. This decreases the cost as the equipment can be repeatedly sterilized and reused. It also allows the kits to contain a wide variety of instruments for multiple situations (e.g., minor lacerations, large lacerations, and plastics closures).

Needle drivers come in a variety of sizes. A 4.5 inch needle driver can be used comfortably with most types of needles. A 6 inch needle driver may be required if large needles are used to close a wound. Hold the needle driver with the fingertips to provide greater flexibility. The fingers can also be placed through the finger holes, but this is not as efficient when closing a wound. Grasp the needle one-third of the way from the swag (distal) end with the tip of the needle driver.

The skin must be grasped and manipulated during wound repair to allow for proper suture placement. Forceps are most commonly used to grasp and manipulate the skin. **Smooth (nontoothed) forceps should never be used to grasp skin.** They require the application of a large amount of force to grasp the tissue. This can crush tissue very easily. An Adson forceps is the forceps of choice. It has fine teeth that grasp tissue securely with minimal force.

A skin hook is a sharp, pointed instrument that is inserted into the wound edge and grasps the tissue from the undersurface. It produces a small puncture wound in the subcutaneous tissues and does not penetrate the skin surface. Skin hooks are preferable to forceps as they do not crush tissues. A skin hook is awkward to use at first. The Emergency Physician will most certainly prefer a skin hook to forceps with proper instruction and experience.

Several types of scissors are required for proper wound closure. Iris scissors have sharp, delicate tips for making precise cuts in tissue. They should not be used to cut suture material as this rapidly dulls and nicks the blades. Suture scissors have one blunt tip and one pointed tip. Both blades of the suture scissors are sharp. Suture scissors are used to cut adhesive tape, gauze, rubber drains, and suture material. Metzenbaum scissors should be used to debride heavy tissue, bluntly dissect tissue, and undermine tissue.

Hemostats are used to clamp small vessels that are bleeding, to explore a wound, and to grasp fascia. Hemostats are available in a variety of sizes and styles. A straight 6 inch hemostat is used for most purposes during wound repair. A curved 5 inch mosquito hemostat can be used for small wounds or delicate tissues.

Three different scalpel blades should be available when a wound is being repaired. A #11 blade is used to make stab incisions. It is often used for the incision and drainage of abscesses, cricothyroidotomies, and the removal of small or tight sutures. A #10 blade is used to make straight cuts in the skin and debride wound edges. It is rarely used in laceration repair. A #15 blade is small and curved to allow precise incisions. It is used for excising foreign bodies and wound debridement.

PATIENT PREPARATION

Explain the risks, benefits, and complications of the wound closure to the patient and/or their representative. The risks include poor healing, wound dehiscence, bleeding, pain, a worse scar, infection, loss of the tissue, and the need for further surgery. The benefits include improved cosmesis and wound healing. Alternatives to advanced wound closure include wet-to-dry dressings and allowing the wound to granulate or follow-up with a Plastic Surgeon for closure or a skin graft. Discuss the presence of a visible scar after the repair which may require subsequent revision. Explain the aftercare and follow-up. Obtained a signed informed consent for the procedure.

Place the patient in a position of comfort that is equally comfortable for the Emergency Physician. This should allow for appropriate stretcher or seat height, lighting, and maneuverability so that physical obstacles are not a complicating variable during the wound repair. Clean the wound and surrounding skin of any dirt and debris. Flush the wound with normal saline. Apply povidone iodine or chlorhexidine solution to the surrounding skin, not the wound, and allow it to dry. Anesthetize the area using local or regional anesthesia (refer to Chapters 153 through 159).

Examine the wound for obvious foreign bodies or contaminants. Remove these with pressure irrigation using sterile saline. Apply sterile drapes to demarcate a sterile field. Apply the drapes so that the wound may be approached easily from different angles and without the risk of contaminating the site or any of the materials being used. **There can be a great degree of variability in sterile techniques. Therefore, it is important to note that the best way to avoid wound infections is to employ and maintain consistency with sterile procedures throughout the wound repair.**

TECHNIQUES

Z-PLASTY

It can be challenging to change the axis of orientation of a wound. **The reason for changing the orientation of a wound is to create a more functionally and cosmetically pleasing scar**. The Z-plasty has been described as a basic technique for scar revision, although its application also proves useful to lengthen and reorient wounds.[5] Wound lengthening reduces the formation of contractures which often occurs when the wound crosses areas of flexion. The Z-plasty should not be used for wounds from burn injuries where normal skin is not present. It also breaks up a linear scar into an accordion-like scar that has some degree of elasticity.

The Z-plasty is generally described to redirect a wound occurring across a flexion crease, over a joint, or on the face. It requires two incisions that create two triangular flaps with approximately 60° of separation between the flaps, although the angle may vary between 30° and 90° (**Figure 118-1**).[5] The greater the angle, the greater is the gain in wound length. Sharper angles increase the risk of necrosis in the tip of the flap. Broader angles result in difficulty in rotating the flaps. Angles of 60° increase the wound length by 75%. Angles of 45° increase the wound length by 50%. Angles of 30° increase the wound length by 25%. **The length of both arms of the incision must be the same length as the wound.** The undermining and separation of the two triangular flaps lengthens the wound and allows it to be reoriented perpendicularly to the original location. Additionally, small Z-plasties may be used in sequence to offset the appearance of straight wounds crossing lines of flexion or where

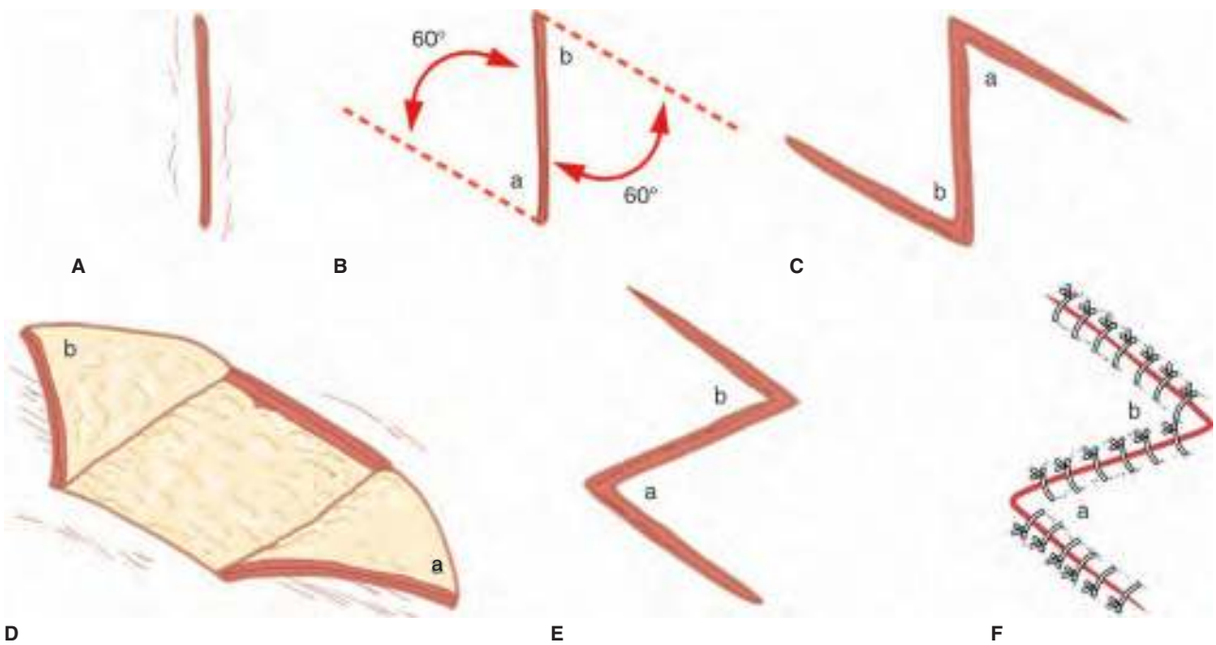

FIGURE 118-1. The Z-plasty. ***A.*** The original laceration. ***B.*** Draw the arms of the Z at a 60° degree angle from the ends of the lacerations. The arms must be the same length as the laceration. ***C.*** The skin has been incised to form the Z. ***D.*** Undermine and elevate the flaps. ***E.*** Transpose the flaps to reorient the wound. ***F.*** Approximate the wound edges with simple interrupted sutures.

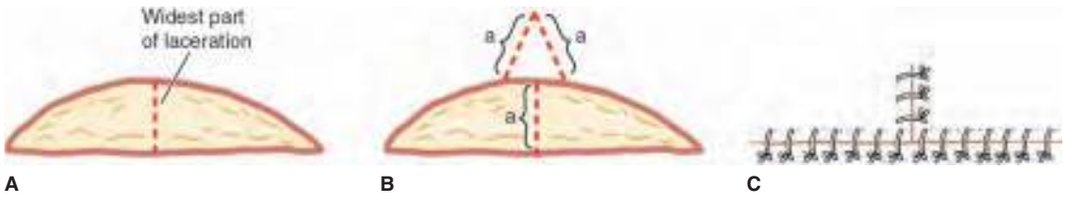

FIGURE 118-2. Approximating edges of grossly unequal lengths in repairing a laceration. ***A.*** The laceration. ***B.*** Draw an equilateral triangle along the longest side and centered about the widest part of the laceration. The sides of the triangle must be equal to the length of the widest part of the original laceration. ***C.*** Approximate the wound edges with simple interrupted sutures.

contractures are likely to occur so that the wound site then becomes parallel to the lines of flexion, further reducing occurrence of contracture formation.[3,4]

Clean, prep, and anesthetize the wound and surrounding skin. If the wound edges are irregular, sharply debride them using a #15 scalpel blade to form straight edges (**Figure 118-1*A***). Measure and draw 60° angles from the ends of the laceration (**Figure 118-1*B***). Draw the arms of the Z on the patient's skin with a skin-marking pen. **The arms must be the same length as the original laceration**. Incise the arms of the Z using a #15 scalpel blade (**Figure 118-1*C***). Undermine the flaps of the Z and the surrounding skin. Elevate the flaps of the Z (**Figure 118-1*D***). Transpose the flaps so that the wound is reoriented (**Figure 118-1*E***). Place simple interrupted sutures to approximate the wound edges (**Figure 118-1*F***).

APPROXIMATING THE EDGES OF A LACERATION WITH GROSSLY UNEQUAL LENGTHS

Creating an equilateral triangle from the midpoint of the longest wound edge allows wound edges of unequal length to be closed easily (**Figure 118-2**).[3-5] Determine the widest point between the two wound edges. Determine which of the two wound edges is longer. Mark the middle of the longest wound edge (**Figure 118-2*A***). Draw an equilateral triangle centered at this mark (**Figure 118-2*B***). **The sides and base of the triangle must all be of equal length and the same length as the widest part of the original wound**. Incise the arms of the equilateral triangle with a #15 scalpel blade and remove the tissue (**Figure 118-2*B***). Undermine the wound edges. Close the wound and the perpendicular incision from the triangle with simple interrupted or running sutures (**Figure 118-2*C***). Closure of the two wounds results in a clean linear wound with a short perpendicular linear wound offsetting the previously unequal edges (**Figure 118-2*C***).

CLOSING A SQUARE-SHAPED DEFECT

Wounds are rarely square-shaped after an injury (**Figure 118-3*A***). Debride the wound to make a square-shaped defect (**Figure 118-3*B***). Square-shaped defects can be difficult to close and require a single pedicle advancement flap.[3] Elongating two sides of the square allows small and moderate-sized defects to be closed primarily. Draw lines to extend two parallel edges of the square by twice their length (**Figure 118-3*C***). Draw Burow's triangles on the ends of the extended lines (**Figure 118-3*C***). These triangles will be removed, allowing the flap to be transposed into the wound and creating a more symmetrical flap.[3-5] Draw the Burow's triangles as equilateral triangles whose sides are half the length of the square defect (**Figure 118-3*C***).

Incise along the extended lines and Burow's triangles with a #15 scalpel blade. Remove the tissue of the Burow's triangles. Undermine the rectangular flap and the area surrounding the base of the flap. **Do not undermine the area lateral to the extended lines**. Advance the tissue flap to close the defect (**Figure 118-3*D***). Place a simple interrupted suture in the center of the short edge of the flap to hold it in position. Place half-buried horizontal mattress sutures to secure the corners of the flap (**Figure 118-3*D***). Approximate the wound edges with simple interrupted sutures along the long arms (**Figure 118-3*D***). Approximate the corner of the Burow's triangles with half-buried horizontal mattress stitches and the rest of the triangle with simple interrupted sutures.[3-5]

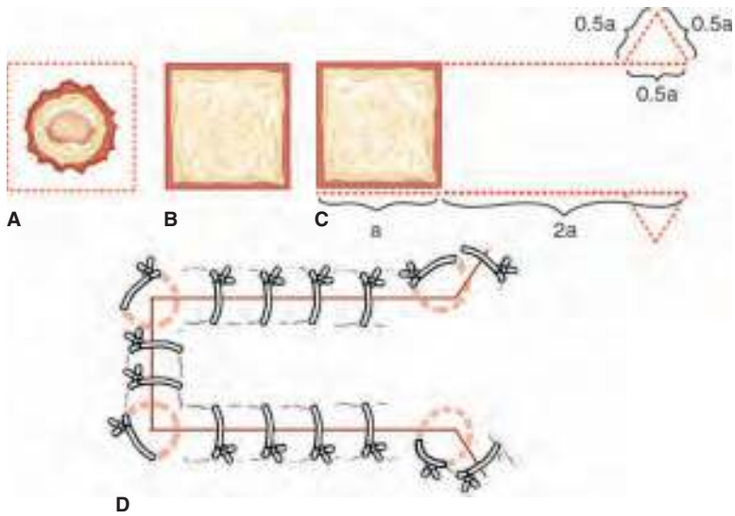

FIGURE 118-3. Closure of a square-shaped defect. ***A.*** The original tissue defect. Draw lines around the defect to form a square. ***B.*** The skin has been incised and the original defect removed to create a square-shaped defect. ***C.*** Draw lines to extend two sides of the square into a rectangle. Draw Burow's triangles at the ends of the rectangular lines. ***D.*** Advance the flap and approximate the wound edges.

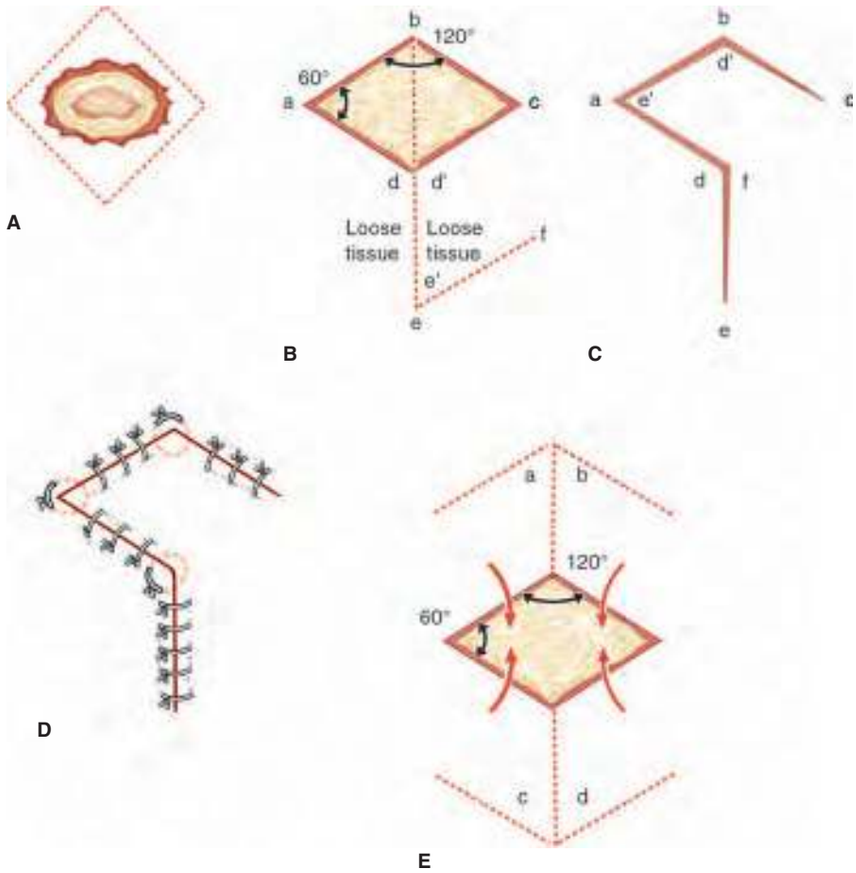

FIGURE 118-4. Closure of a diamond- or rhomboid-shaped defect. ***A.*** The original tissue defect. Draw lines around the defect to form a diamond or rhomboid. ***B.*** The skin has been incised to create a diamond-shaped defect. Draw lines to form the flap. Extend the short diagonal (BD) by one times its length to form line DE. Draw line EF parallel to line CD and the same length as line CD. ***C.*** Transpose the flap to close the defect. ***D.*** Approximate the wound edges. ***E.*** The four available Limberg flaps that can be created to fill the defect.

CLOSURE OF A DIAMOND-SHAPED DEFECT

Diamond- or rhomboid-shaped defects require the rotation of a flap referred to as a Limberg flap. **The Limberg flap is a transposition flap suitable only for closing a diamond- or rhomboid-shaped defect. It requires the formation of two adjacent angles of the rhomboid that must be 60° and 120° for an optimal flap**.

Wounds are rarely diamond-shaped after an injury (**Figure 118-4*A***). Debride the wound to make a diamond-shaped defect (**Figure 118-4*A***). Draw a line to extend the distance of the short diagonal of the defect to double its total length (**Figure 118-4*B***). Draw a line from the extended line and parallel (back cut) to the adjacent wound edge that is equal to the length of the extended line (**Figure 118-4*B***). Incise along the extended lines with a #15 scalpel blade. Undermine the flap and adjacent skin. Rotate the flap into the diamond-shaped defect (**Figure 118-4*C***). Approximate the wound edges with simple interrupted sutures along the linear edges and half-buried horizontal mattress sutures at the intersection or angles of the wound edges (**Figure 118-4*D***).[3-5] Depending on the location of loose tissue and adjacent structures, one of four Limberg flaps can be created to close the defect (**Figure 118-4*E***).

CLOSURE OF AN ELLIPTICAL DEFECT

Wounds are often irregular and elliptical (**Figure 118-5*A***). Creating an ellipse from a wound allows more even closure of asymmetrical wounds. This is also referred to as an S-plasty. This technique may be used when there is concern of significant scarring and contracture formation from an associated thermal burn injury and Z-plasties are not recommended.[4] The less acute and more rounded edges of the ellipse tend to result in less tissue necrosis.[3,4]

Draw lines to debride the wound and form an S-shaped defect (**Figure 118-5*A***). Incise the extended lines with a #15 scalpel blade to form the S-shaped defect and excise the wound (**Figure 118-5*B***). Undermine the wound edges. Place buried sutures to close the wound and prevent tension on the wound edges. Approximate the wound edges by placing simple interrupted sutures alternating at each end of the S-shaped defect and ending in the middle (**Figure 118-5*C***).

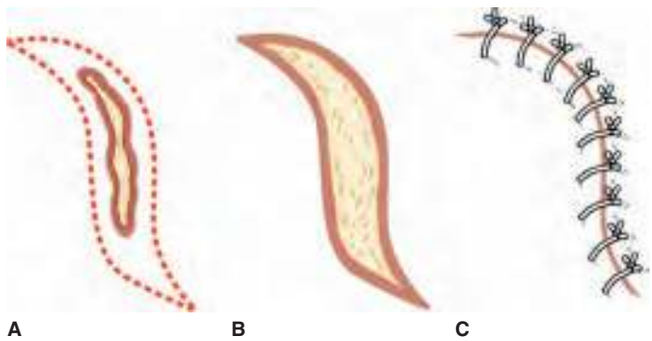

FIGURE 118-5. Closure of an elliptical defect. ***A.*** The original tissue defect. Draw lines around the defect to form an S-shaped defect. ***B.*** The skin has been incised and the original defect removed to create an S-shaped defect. ***C.*** Approximation of the wound edges with simple interrupted sutures.

Suturing from the ends and moving inward reduces the tension on the wound edges.[3,4]

CLOSURE OF A V-Y ADVANCEMENT FLAP

The V-Y flap is not a rotational flap but rather a V-shaped flap created away from the wound site which then allows the skin to be advanced into the defect (**Figure 118-6**).[3-6] These advancement flaps may be used to avoid defects from lacerations of the fingertips, lips, or face.

Wounds may be oval and elliptical (**Figure 118-6*A***). Creating an oval from a wound allows more even closure of asymmetrical wounds. Draw lines to debride the wound and form an oval-shaped defect (**Figure 118-6*A***). Incise along the lines with a #15 scalpel blade to form the oval-shaped defect and excise the wound (**Figure 118-6*B***). Draw a V-shaped line adjacent to the oval-shaped defect (**Figure 118-6*C***). **The line should be the length of the oval-shaped defect and approximately the width of the original wound away from the defect along its entire length.** Although the two sites do not directly communicate with each other, the V-shaped incision allows the original wound to be closed primarily and without tension. Undermine the wound and the V-shaped incision. Approximate the oval-shaped defect with buried sutures, if necessary, and simple interrupted sutures (**Figure 118-6*D***). This will result in an opening of the V-shaped defect (**Figure 118-6*D***). Draw and incise a line perpendicular to the apex of the V-shaped defect with a #15 scalpel blade (**Figure 118-6*E***). The line should be as long as the width of the V-shaped defect. Approximate the three arms of the defect with simple interrupted sutures to form a Y (**Figure 118-6*F***).

An alternative V-Y advancement flap can be used to close the injury without making a second wound (**Figure 118-7**). Draw lines to debride the wound and form a V-shaped defect (**Figure 118-7*A***). Incise along the lines with a #15 scalpel blade to form the V-shaped defect and excise the wound (**Figure 118-7*B***). Draw and incise a line perpendicular to the apex of the V-shaped defect with a #15 scalpel blade (**Figure 118-7*C***). The line should be as long as the width of the V-shaped defect. Undermine the V-shaped defect and the perpendicular line. Place a half-buried horizontal mattress suture to close the center of the Y (**Figure 118-7*D***). Approximate the edges of the defect with simple interrupted stitches (**Figure 118-7*D***).

CLOSURE OF A RECTANGULAR DEFECT

Wounds may be in the form of an ellipse or oblong (**Figure 118-8*A***). These can be converted into a rectangular defect to allow primary closure.[3] Draw lines to debride the wound and form a rectangular defect (**Figure 118-8*A***). Incise along the lines with a #15 scalpel blade to form the rectangular defect and excise the wound (**Figure 118-8*B***). Draw lines to convert the short ends of the rectangle into triangles (**Figure 118-8*C***). **The width of the rectangle should serve as the measurement to create an equal distance between the base and the apex of the triangle.** Excising triangles from the ends of the rectangular defect reconfigures the ends of the wound. Incise along the lines with a #15 scalpel blade and remove the triangles (**Figure 118-8*D***). Undermine the skin surrounding the defect. Approximate the wound edges with simple interrupted sutures to form a straight line (**Figure 118-8*E***).

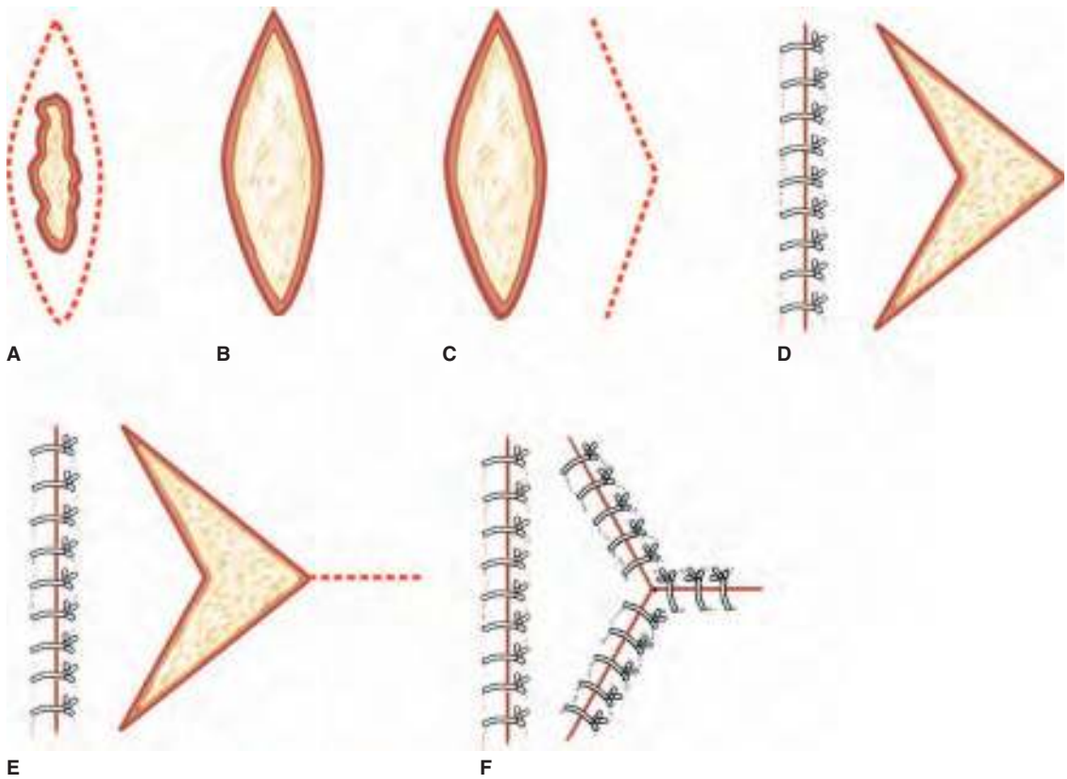

FIGURE 118-6. Closure of a V-Y advancement flap. ***A.*** The original tissue defect. Draw lines around the defect to form an oval. ***B.*** The skin has been incised and the original defect removed to form an oval-shaped defect. ***C.*** Draw a V-shaped line adjacent to the oval defect. It should be positioned the maximum width of the oval defect from the wound edge. ***D.*** Incise the V and undermine the skin edges. Approximate the oval-shaped defect with buried sutures and simple interrupted sutures. ***E.*** Draw and incise a line perpendicular to the apex of the V and equal to the maximum width of the V-shaped defect. This forms the V into a Y. ***F.*** Approximation of the Y-shaped defect.

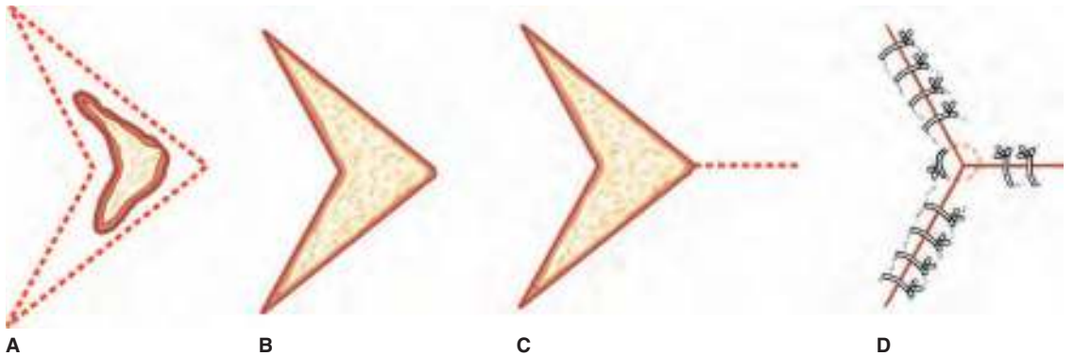

FIGURE 118-7. An alternative V-Y advancement flap closure. ***A.*** The original tissue defect. Draw lines around the defect to form a V. ***B.*** The skin has been incised and the original defect removed to form a V-shaped defect. ***C.*** Draw and incise a line perpendicular to the apex of the V and equal to the maximum width of the V-shaped incision. ***D.*** Approximate the center of the Y with a half-buried horizontal mattress suture. Approximate the arms of the Y with simple interrupted sutures.

CLOSURE OF A TRIANGULAR DEFECT (ROTATION FLAP)

Closing of a triangular defect can be accomplished with the use of a rotational flap (**Figure 118-9**). These flaps can be turned on a pivot point. **The flap must be planned carefully so that the direction of rotation coincides with the geometry of the defect.**[3-5] **Always plan and draw the arch of the flap carefully to visualize the pivot point and direction of rotation prior to making the incision. The creation of a rotation flap is a significant procedure. It can result in a vascular disaster and leave a deformity greater than the original defect it was supposed to correct. Do not create a rotation flap unless you have experience with this technique and know that the flap has an adequate vascular supply.**

Wounds are often irregular and elliptical (**Figure 118-9*A***). Draw lines to debride the wound and form an isosceles triangular defect (**Figure 118-9*A***). Incise along the lines with a #15 scalpel blade to form the triangular defect and excise the wound (**Figure 118-9*B***). **Draw the rotation flap very carefully.** The edge of the flap is an arch from the base of the triangle and three to four times longer than the actual base of the triangle (**Figures 118-9*C* and 118-9*D***).[3] Draw a second triangle as a Burow's triangle in the area next to the pivot point of the flap (**Figure 118-9*C***).[3-5] **The base of the Burow's triangle should be half the length of the base of the triangular defect, with one corner of the base formed by the end of the arch.** Incise along the lines with a #15 scalpel blade. Remove the Burow's triangle. Undermine the rotation flap and the surrounding skin. Rotate the flap to close the defect and approximate the wound edges with interrupted sutures (**Figure 118-9*C***). Place half-buried horizontal mattress sutures to approximate the triangular defect and any corners. Place simple interrupted sutures to approximate the remaining wound edges.

A triangular defect may be closed with a modification to the above technique when there is minimal room to form and excise the Burow's triangle or if lines of skin tension limit the location of the pivot point.[3] **This modified technique should be considered only when necessary, which may occur from poor planning of the initial flap or in areas where fascia can be separated from the subcutaneous layer (e.g., scalp wounds and areas involving the trunk).**[3-5] Form the triangle to debride the wound and draw the arch as described previously. **Do not draw the area beyond the point perpendicular to the apex of the triangle (Figure 118-9*D*).** Rather than drawing a Burow's triangle for excision, draw a line to make a back-cut from the pivot point (i.e., the end of the arc) and along the base of the flap (**Figure 118-9*D***).[3,5] **This line should be three-fourths the length of the base of the triangular defect.** Incise along the lines with a #15 scalpel blade. Undermine the defect and the rotation flap. Rotate the flap to close the defect and approximate the entire wound edge with half-buried horizontal mattress sutures (**Figure 118-9*D***). Suture the back-cut prior to closing the triangular defect or the arch.[3] **This alternative technique carries the risk of having a poor blood supply to the flap due to its small base.**

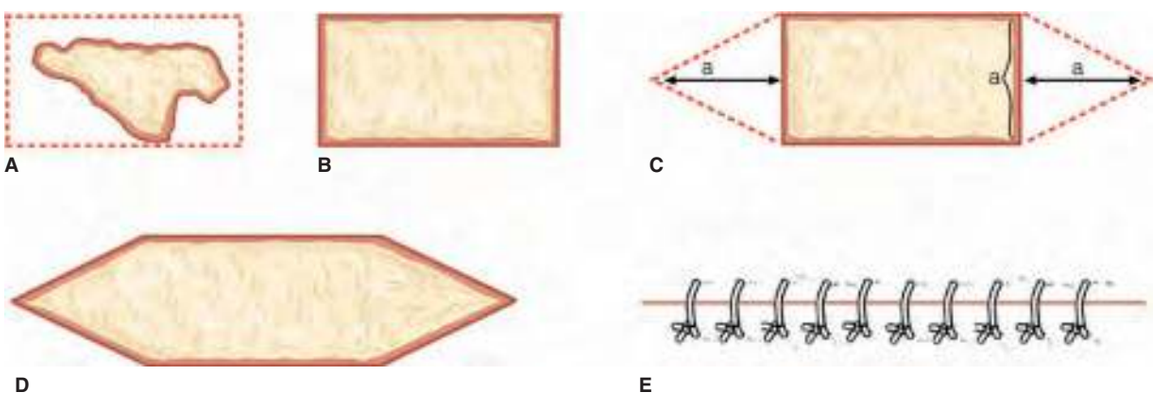

FIGURE 118-8. Closure of a rectangular defect. ***A.*** The original defect. Draw lines around the defect to form a rectangle. ***B.*** The skin has been incised and the original defect removed to form a rectangle. ***C.*** Draw triangles along the short sides of the rectangle. The length from the apex to the base of the triangle must be equal to the width of the rectangle. ***D.*** The resulting defect after incising and removing the triangles. ***E.*** Approximation of the wound edges to form a linear scar.

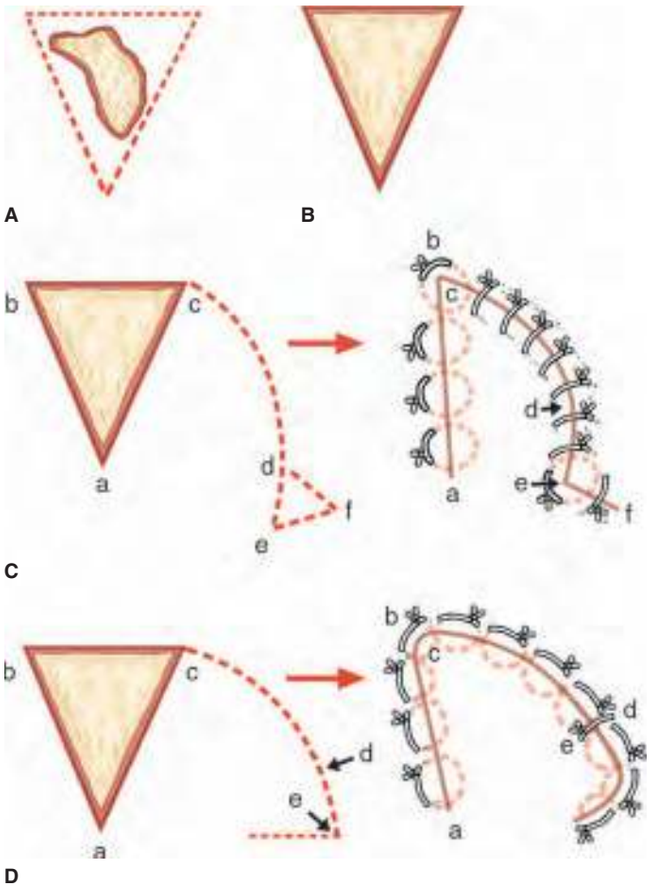

FIGURE 118-9. Closure of a triangular defect. ***A.*** The original defect. Draw lines around the defect to form a triangle. ***B.*** The skin has been incised and the original defect removed to form a triangle. ***C.*** Draw a line to extend the base of the triangle in a wide arc that is three to four times the length of the base of the triangle. Make sure that the arc is drawn beyond the line from point a to point d. Draw a Burow's triangle at the end of the arc. The base of the Burow's triangle should be half the length of the base of the triangular defect. Approximate the triangular defect and any corners with half-buried horizontal mattress sutures. ***D.*** An alternative technique. Draw a wide arc from the base of the triangle similar to that in C but that ends opposite the apex of the triangle (point a). Draw a line to make a back-cut that is three-fourths the length of the base of the triangular defect. Approximate the entire wound edge with half-buried horizontal mattress sutures.

CLOSURE OF AN OVAL DEFECT

Oval defects can be closed by creating a rotational or interpolation flap from the intact adjacent tissue (**Figure 118-10**).[3] Wounds are often irregular and elliptical (**Figure 118-10*A***). Choose a side of the oval defect to form the flap. The adjacent skin used to make the flap must be vascularly intact to avoid the risk of tissue necrosis once the flap is sutured into position. Draw lines to debride the wound and form an oval defect (**Figure 118-10*A***). Incise along the lines with a #15 scalpel blade to form the oval defect and excise the wound (**Figure 118-10*B***). **Draw the rotation flap very carefully. Draw a mirror image of the defect abutting the original wound (Figure 118-10*C*).** Incise only along the lines noted with a #15 scalpel blade. Undermine the rotation flap and surrounding skin. Rotate the flap to close the defect (**Figure 118-10*D***). Approximate the edges of the flap with half-buried horizontal mattress sutures (**Figure 118-10*D***). Approximate the wound from where the flap originated with simple interrupted sutures, if it is not excessively large or under tension, or with half-buried horizontal mattress sutures.

CLOSURE OF A CIRCULAR DEFECT

Some defects can be closed primarily. Examples include circular defects or triangular defects that are converted to ellipses (**Figure 118-11**). Excise the tissue defect to form a circle. Excise a surrounding ellipse of tissue centered about the circle (**Figure 118-11*A***). **The ellipse must be 2½ to 3 times as long as its greatest width.** Undermine the edges of the ellipse. Close the resulting defect with deep sutures if required and cutaneous sutures (**Figure 118-11*B***).

Larger defects require the use of a double V-Y closure (**Figure 118-12**). Draw two sliding pedicle flaps of equal length, one opposite or 180° from each other, and centered about the defect (**Figure 118-12*A***). Create the two sliding pedicle flaps with a #15 scalpel blade. **Incise the skin and dermis but not the underlying subcutaneous tissue to form an ellipse centered about the tissue defect** (**Figure 118-12*A***). Remove the tissue defect and debride the tissues at the base of the flaps to form two straight edges (**Figure 118-12*A***). Gently undermine the edges of the ellipse. **Do not undermine the triangular tissue flaps so that their vascular supply is preserved.** Slide (i.e., advance) the flaps on their subcutaneous pedicles until the bases are touching. Approximate the base of one flap to the other using simple

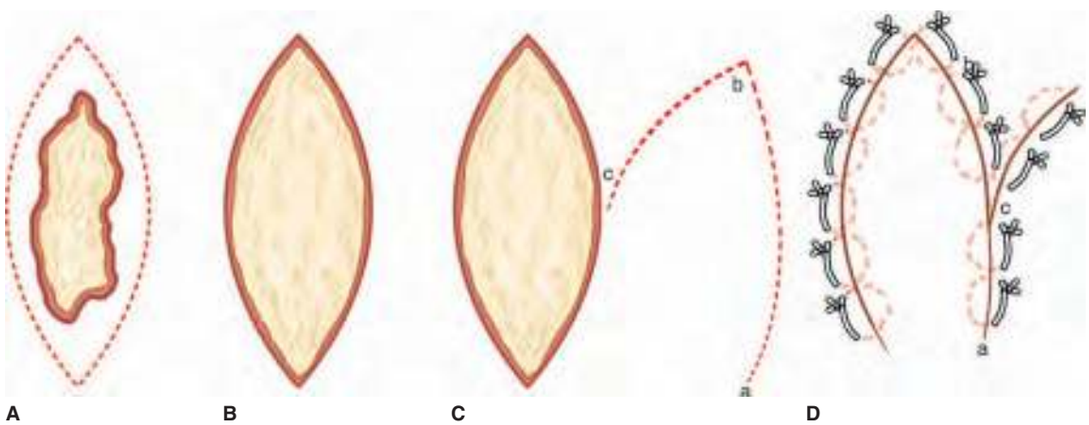

FIGURE 118-10. Closure of an oval-shaped defect. ***A.*** The original defect. Draw lines around the defect to form a rectangle. ***B.*** The skin has been incised and the original defect removed to form an oval. ***C.*** Draw a mirror image of the defect so that it is abutting the defect. ***D.*** The flap has been rotated and the wound margins approximated with half-buried horizontal mattress sutures.

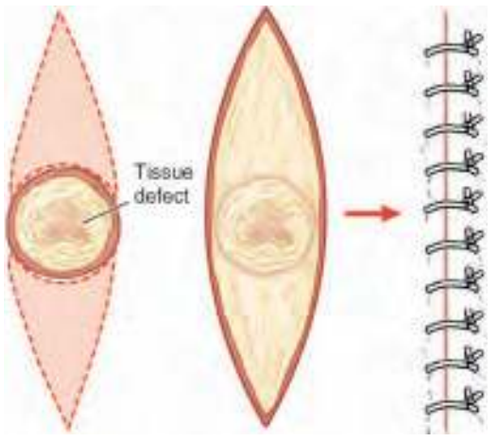

FIGURE 118-11. Closure of a circular tissue defect. ***A.*** Draw lines around the defect to form an ellipse. ***B.*** The skin has been incised and the original defect removed to form an ellipse. The wound edges are approximated with deep and cutaneous sutures.

interrupted sutures (**Figure 118-12*B***). Approximate the arms and bases of the Ys using simple interrupted sutures.

ASSESSMENT

Inspect the wound edges carefully for adequate approximation. Observe the wound for a period of time to make sure that it remains viable and is not compromised due to a poor blood supply or tight sutures. **A nonviable repair requires immediate removal of the sutures and consultation with a Plastic Surgeon.**

AFTERCARE

Pull the suture knots lying directly over the wound margin to one side so that all the knots lie on the same side. Wipe off any residual povidone iodine or chlorhexidine solution with sterile saline. Apply a topical antibiotic ointment to the wound followed by a nonadherent dressing. Arrange follow-up in 24 hours with the patient's Primary Care Provider, a Plastic Surgeon, or the Emergency Department. Consider the use of prophylactic antibiotics even though there is no evidence to support or refute this practice.

Instruct the patient and/or their representative regarding wound care and dressing changes. Provide clear instructions of what to look for regarding possible signs of early infection, both localized around the wound site as well as systemic symptoms. Any patient who experiences excessive swelling, erythema, a purulent or foul-smelling discharge, significant pain from the wound site, or fever should return to the Emergency Department immediately.

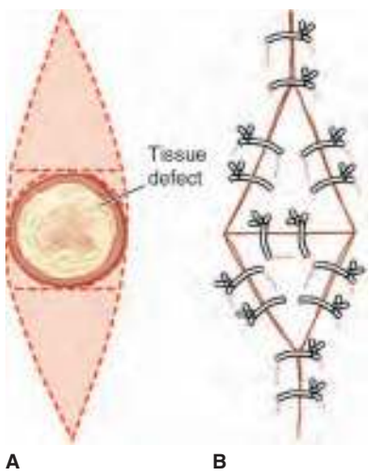

FIGURE 118-12. The double V-Y closure to repair a tissue defect. ***A.*** Create an ellipse centered about the tissue defect. Remove the tissue defect and form straight edges at the bases of the triangular flaps. ***B.*** Approximate the bases of the triangular flaps, followed by the arms and bases of the "Ys," using simple interrupted sutures.

COMPLICATIONS

A brief discussion of the complications of wound closure is presented below. Refer to Chapter 114 for a more complete discussion. The complications of any wound can be greatly affected by the preparation of the wound prior to wound closure. The maintenance of sterile technique throughout wound closure and adequate irrigation of the wound will limit the risk of infection. It is unrealistic to expect any wound site to be bacteria-free.

Wounds may show poor scar formation or delayed healing due to several factors. Poor aftercare without adequate dressing changes or neglect (e.g., premature exposure to environmental irritants such as dirty water, direct and excessive sun exposure, or chemicals) will likely result in a less favorable and unpredictable healing process. The wound site should be protected from excessive contact or use during the initial healing period. Mechanical trauma or overuse can increase the chance of edema or hematoma formation, leading to wound dehiscence or atypical scar formation.

Proper follow-up should be arranged and stressed within the initial 24 to 48 hours following the treatment and thereafter as may be warranted. Awareness that wound healing takes place in sequential physiologic steps is needed to properly direct patients so that the risk of complications or the need for antibiotics will be minimal.

SUMMARY

Patients present to the Emergency Department with a wide variety of wound types. The use of local flap techniques allows the Emergency Physician to close difficult and complex wounds. If primary closure is not possible, these techniques decrease the tension on a wound and allow for appropriate cosmesis. They require close follow-up and appropriate patient selection if complications are to be prevented.

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119 Management of Specific Soft Tissue Injuries

Thomas M. Kennedy and Christopher Russo

INTRODUCTION

Blunt and penetrating trauma can lead to a myriad of soft tissue injuries. The management of many of these injuries is discussed elsewhere in this text. Some specific soft tissue injuries require detailed explanations for their repair. These injuries are discussed below. Administer immunoprophylaxis (e.g., tetanus and tetanus immune globulin) when indicated. Consider the use of procedural sedation (Chapter 159) in children.

MULTIPLE FOREHEAD LACERATIONS

Forehead lacerations are common in all age groups. They occur most frequently during early childhood. Their location demands a complete head and neck evaluation.[1] **Their visibility requires meticulous attention to detail.** Knowledge of the principles regarding their repair allows for good cosmesis. The repair of forehead lacerations differs from other soft tissue injuries due to the role of skin tension lines, the lack of extra tissue, and scarring promoted by too many deep dermal sutures.[2-4] They are broadly categorized into superficial and deep in relation to involvement of the frontalis muscle.

Forehead injury repair is governed by several principles. Skin tension lines run parallel to the skin creases and play a major role in the outcome of any forehead laceration. Lacerations angled more than 35° from lines of tension are more likely to heal with a poor result.[5] Consider counteracting skin tension with deep stitches and undermining. Lacerations running perpendicular to skin tension lines are more likely to result in a noticeable scar.[2,3] There is little excess tissue on the forehead to allow for later wound revisions. **Resist the temptation to excise ragged wounds.**[4] This leaves enough tissue for the Surgeon to manipulate if further revision is required. **Deep lacerations are closed with a layered repair to maintain muscle function, eliminate dead space, and reduce skin tension. Place as few deep sutures as possible.** They tend to promote more tissue reaction and more noticeable scar formation.

Most forehead lacerations require repair to promote cosmesis and provide hemostasis. The rich blood supply to the area allows primary repair up to 24 hours after the initial insult.[1] This allows referral to a consultant if there is any question about the ability to achieve satisfactory cosmesis or if the wounds are so extensive as to take the Emergency Physician away from their departmental responsibilities for an unacceptably long time. Primary closure beyond 24 hours may be considered after the risk of infection is weighed against the cosmetic benefit and discussed with the patient.[1]

Laceration repair is dependent on the type of laceration. Small uncomplicated lacerations and flaps smaller than 5 mm can be closed with 6–0 nonabsorbable suture (e.g., nylon or Prolene) or 6–0 absorbable suture (e.g., fast-absorbing gut) using simple interrupted stitches.[1] Multiple studies in pediatrics have shown absorbable suture to have similar outcomes to nonabsorbable suture in cosmesis and adverse events.[6] Close larger flaps using the half-buried horizontal mattress stitch. Allow partial-thickness abrasions and gouges less than 1 cm wide and 2 mm deep to heal by secondary intention.[2,3,7] Bunched up, small flap lacerations can be excised together and the resulting defect repaired primarily.[7] This technique is described under "Multiple Small Skin Flaps" in this chapter.

Close deeper transverse lacerations that involve the deep fascia or the frontalis muscle in layers with buried stitches using 5–0 absorbable suture (e.g., Monocryl or Vicryl).[6] Repair the periosteum using 5–0 absorbable suture (e.g., nylon or Prolene).[1] Close the epidermal layer with simple interrupted stitches using 6–0 nonabsorbable suture (e.g., nylon or Prolene) or absorbable suture (e.g., gut), skin closure strips (Chapter 116), or tissue adhesive (Chapter 117). The latter two are attractive alternative techniques of wound closure if the patient is at risk to develop keloids or hypertrophic scars. **Do not use these alternative techniques on vertically oriented lacerations as they are under tension.**[5] **Approximate skin folds, skin creases, and the hairline with great precision.**

EYELID LACERATIONS

The objective of eyelid repair is the restoration of normal alignment and anatomic function.[8] Eyelid lacerations are classified as marginal if they cross the eyelid margin or extramarginal if they do not cross the eyelid margin.[7] **Consult an Ophthalmologist to close marginal lacerations (Figure 119-1).** Repair requires considerable experience, the use of magnification, and the placement of deep sutures.

Repair of marginal eyelid lacerations demands a thorough understanding of anatomy and a high degree of suspicion for injury to critical structures. The eyelid consists of five layers of tissue (from superficial to deep): skin, subcutaneous tissue, orbicularis oculi muscle, tarsal plate, and conjunctiva. The skin is thin and does not protect the eye from penetrating injuries. The orbicularis oculi muscle controls eyelid closure. The tarsal plate is composed of dense elastic and connective tissue, contains the meibomian glands, and contains the eyelashes.[9] The levator palpebrae superioris muscle elevates the upper eyelid. Its aponeurosis inserts into the superior tarsal plate and skin of the eyelid. The orbital septum is a membranous sheet that acts as the anterior boundary of the orbit. It extends from the orbital rim, blends with the levator aponeurosis in the upper eyelid, and blends with the tarsal plate in the lower eyelid.[10] On the medial aspect of the eyelids are the superior and inferior lacrimal papillae and puncta which communicate with the canalicular system. The nerve supply to the eyelids arises from the temporal and zygomatic branches of the facial nerve.

A careful history and a thorough physical examination looking for concealed injury or foreign bodies is imperative.[11,12] The examination must specifically address and exclude canthal injuries, lacrimal apparatus (e.g., most commonly canalicular) injuries, and/or injuries to deep structures. Injuries medial to the lacrimal

FIGURE 119-1. Eyelid laceration involving the margin. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2016. Photo contributor: Lawrence B. Stack, MD.)

punctum must prompt the possibility of a lacrimal apparatus or canalicular injury.[13] Consult an Ophthalmologist for a probe evaluation and irrigation of the lacrimal apparatus.[14] **Failure to identify canalicular interruptions can compromise outcome.** Canalicular repair delayed beyond several days is less successful than primary repair.[13] A delay of 2 to 3 days may be advantageous, as the cut medial ends may become edematous, whitened, and easier to locate.[15,16]

A thorough examination is necessary prior to performing a repair. The assessment should include testing visual acuity, visual fields, extraocular muscles, and for the presence of foreign bodies, globe rupture, and corneal abrasions.[9] Lacerations that initially appear superficial may be associated with penetration into the eye or intracranial cavity. Carefully evert the eyelid to determine the depth of penetration. Gently pull open one edge of the wound.[13] Full-thickness lacerations warrant consultation with an Ophthalmologist because improper repair of the inside surface of the eyelid can result in scar formation with persistent corneal irritation.[17] Consider obtaining a computed tomographic scan of the brain, orbits, and midface in these situations to evaluate for a retained foreign body, ruptured globe, or intracranial injury. Ptosis in the presence of an eyelid laceration is concerning for injury to the levator palpebrae muscle or its aponeurosis and requires consultation.[14] Consult an Ophthalmologist for globe rupture, avulsion of eyelid tissue, and marginal lacerations for proper repair to prevent deformity and disability (e.g., ectropion or epiphora). Transmarginal lacerations can be complicated by medial and lateral canthal tendon injury.[18]

Recognition and management of injuries to deep structures (e.g., lacrimal apparatus, orbicularis oculi muscle, levator palpebrae muscle, tarsal plates, and medial or lateral canthal tendons) are necessary to prevent dysfunctional tearing, lid malalignment, ptosis, functional abnormalities, and cosmetic defects. Awareness of the eyelid's complex anatomy is essential to the proper recognition and repair of less evident impairment due to injury. Certain key features are reviewed in this section as they are necessary to prevent overlooked injury and understand proper repair.

The eyelid's skin is the thinnest in the body, with that of the upper eyelid being thinner than that of the lower eyelid. The skin moves freely over the deeper tissues and is easily mobilized with forceps. Surface landmarks include, from posterior (nearest the globe) to anterior (nearest the skin), the mucocutaneous junction, the orifices of the meibomian glands, the gray line, and the lash line. **The gray line is a key landmark.** It is located on the palpebral edge and consists of an isolated strip of pretarsal orbicularis oculi muscle just anterior to the tarsus (i.e., Riolan's muscle). Lacerations through the gray line require diligent reapproximation and should be referred to an Ophthalmologist.[19] There are three or four irregular rows containing approximately 100 lashes on the upper eyelid and two or three rows of approximately 50 lashes on the lower eyelid.

The levator palpebrae muscle arises from the roof of the orbit. It inserts into the midtarsus and overlying skin, intimately associating with the orbicularis oculi muscle. Suspect levator injuries with any horizontal laceration of the upper eyelid. Improper repair or failure to repair a laceration may result in ptosis or a deformity of the supratarsal fold.

Canthal injuries must be actively sought. Determine whether the medial or lateral canthal tendon is injured. Apply lateral traction on the eyelid. Displacement of the punctum laterally may be due to a disruption of the medial canthal tendon. Such a disruption is likely to be associated with nasal fractures, orbital fractures, ethmoid fractures, and canalicular injuries.[20] Apply medial traction on the eyelid. Displacement of the lateral canthus medially is due to a disruption of the lateral canthal tendon. Inspection and/or probing through the wound may confirm a canalicular interruption. Early repair is preferred as the tissue becomes more difficult to identify and repair when swollen. The value of early repair of canalicular lacerations is debated.[21] Early repair may exacerbate the degree of canalicular damage, increase the risk of stenosis, and may result in damage to other parts of the canalicular system.[21] Consult an Ophthalmologist as meticulous repair is mandatory to avoid damaging the lacrimal apparatus.

The orbicularis oculi muscle closes the eyelids tightly. Failure to properly repair the levator muscle, the tarsal fascia, or the orbicularis muscle in a deep upper eyelid laceration may result in ptosis. Consult an Ophthalmologist for all deep upper eyelid lacerations.

There are at least six published variations on the techniques described for repair of the eyelid margin with less than one-third of tissue loss measured horizontally, yet there is no literature comparing outcomes.[7,16,17,20,22-24] The experience and skill of the Emergency Physician are vital. Each minor variation aims for precise apposition to avoid malalignment or notching of the eyelid margin. Each technique varies the sequence, number, type, or order of margin stitches (i.e., final or temporary) versus tarsal suture placement and closure (if required).

Eyelid lacerations with tissue loss require individualization.[15,20] Wound edge loss of less than 25% of the horizontal length of the eyelid can often be closed primarily. Approximate the wound edges with toothed forceps to evaluate wound tension. Freshen (i.e., debride) ragged edges without loss of tissue. **Avoid penetration to the conjunctival surface to avoid contact with the cornea. Accurate repair of the tarsus is vital because it forms the skeleton of the eyelid.** Remove the sutures in 5 days. Tissue loss of greater than 25% may require a canthotomy, cantholysis, or a tissue flap. Refer these injuries to an Ophthalmologist or Oculoplastic Surgeon. Older patients with skin laxity may be able to tolerate a greater than 25% loss with adequate cosmesis.[15,20]

Some eyelid lacerations without eyelid margin involvement may involve the levator muscle. Evaluate horizontal lacerations of the upper eyelid for levator interruption. Visible fat indicates orbital septum penetration and raises the suspicion of levator involvement (**Figure 119-2**). Repair these lacerations with fine absorbable suture to avoid ptosis.[19,20] **An eyelid crease and/or minimal levator function suggests an intact levator.[20]**

The orbital septum lies deep to the orbicularis oculi muscle. The levator palpebrae muscle lies deep to the orbital septum. The septum

FIGURE 119-2. Eyelid laceration with the underlying fat pad protruding. (Used with permission from Schafermayer R, et al: *Strange and Schafermeyer's Pediatric Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2015.)

is rigidly attached to the orbit and does not move on traction with forceps. Grasp the levator muscle with forceps and instruct the patient to look up. A brief pull will be felt as the muscle contracts. It is crucial to distinguish between the levator apparatus and the orbital septum. Injuries involving the orbital septum (**Figure 119-2**) carry a higher risk of globe injury, intraorbital foreign body, and orbital cellulitis. **The septum must not be included in a repair of the levator, as it will restrict movement of the levator.** Consult an Ophthalmologist or an Oculoplastic Surgeon for deep extramarginal lacerations with suspected levator muscle involvement. Obtain a computed tomographic scan to evaluate for potential transorbital fascia/septal involvement.[25]

Complications associated with eyelid repair include malalignment, missed canalicular lacerations with attendant tearing dysfunction, missed foreign bodies, corneal abrasions, missed globe injury, missed canthal interruption, and missed fractures with entrapment. Referral of all patients upon discharge from the Emergency Department to an Ophthalmologist or Oculoplastic Surgeon is recommended.

Many lid lacerations can be treated conservatively without sutured repair. Wounds that can be allowed to heal by secondary intension are lacerations that comprise less than 25% of the eyelid and are superficial. A time to primary closure of up to 36 hours is considered acceptable.[14]

Repair any laceration that comprises over 25% of the eyelid. Instill a drop of topical anesthetic into the eye followed by a protective sclera shell over the eye to prevent injury.[13] A Morgan Lens can also be used for this purpose. **Extreme care is needed to avoid deep penetration of the needle into the eye if the laceration is repaired without a scleral shell.**[8] Administer local subcutaneous anesthetic (e.g., 1% lidocaine with epinephrine) or perform field blocks (e.g., supraorbital, infraorbital, and/or anterior ethmoidal nerve blocks). Perform gentle irrigation. Consult an Ophthalmologist if foreign bodies are unexpectedly encountered and appear to penetrate the globe or orbital tissues. Close the eyelid skin with simple interrupted stitches using 6–0 absorbable suture (e.g., fast-absorbing gut) (**Figure 119-3**).[13] An alternative is using 6–0 or 7–0 nonabsorbable suture (e.g., nylon or Prolene) with the plan for removal in 3 to 5 days.[20] Take only small bites through the skin layer.[8] **Do not grasp deep tissue within the eyelid with the suture to prevent an increased vertical tension and ectropion.**[26]

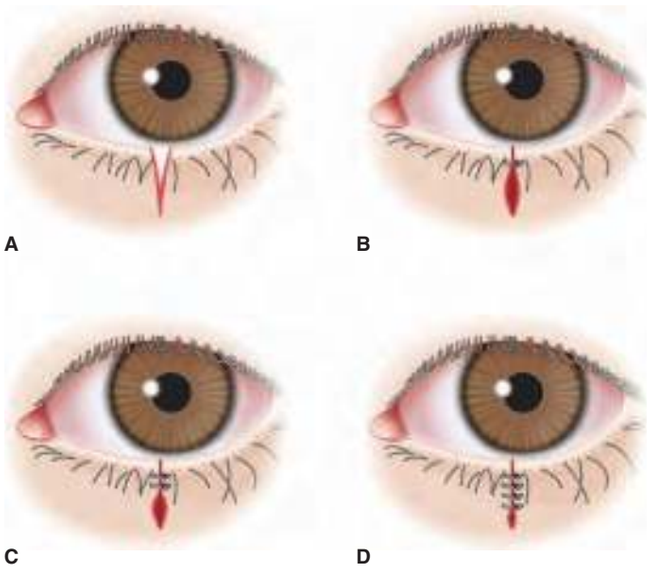


FIGURE 119-3. Fixing an eyelid laceration involving the margin. ***A.*** The laceration. ***B.*** The first suture is placed to approximate the margin. ***C and D.*** The remainder of the laceration is closed. (Used with permission from Tintinalli JE, et al: *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th ed. New York: McGraw-Hill; 2016.)

EAR LACERATIONS

Ear lacerations can result from blunt or sharp trauma to the auricle (**Figure 119-4**).[27] The primary goals are to preserve the normal contours of the auricle, to prevent chondritis, and to prevent hematoma formation.[28,29] The skin of the ear is extremely vascular. **A potential space exists between the auricular cartilage and the adherent perichondrium.** The underlying auricular cartilage is avascular and receives its nourishment from the overlying perichondrium and skin. **Minimize any debridement of the auricular soft tissues to ensure that the repair covers all exposed cartilage.** Auricular laceration repair follows the same principles as other laceration repair techniques. Differences to be appreciated include the importance of debriding as little soft tissue as possible, always covering exposed cartilage, splinting the ear appropriately after the repair, and recognizing the indications for consulting a Plastic Surgeon (e.g., cartilage defects > 5 mm, inability to cover exposed cartilage, devitalized tissue, and complete or near-complete amputations). A subperichondrial hematoma or seroma after repair will cause the cartilage to become infected or necrotic, leading to abscess development or the formation of fibrocartilage causing the deformity (i.e., cauliflower ear).

Examine the area for signs of an acute hematoma or other associated traumatic injuries (e.g., hemotympanum or Battle's sign).[19] Clean, prepare, and anesthetize the auricle (Chapters 156 and 200).[30] Consider local infiltration for small and isolated wounds without cartilage involvement or an auricular block for complicated or extensive lesions. **An auricular block is often the best means of providing anesthesia to avoid distortion of the anatomy.** Local infiltration of 1% lidocaine without epinephrine is required if the laceration involves the posterior wall of the external auditory canal or the concha because this area is innervated by the auricular branches of the vagus nerve.[17] Some recommend surgical consultation for lacerations involving the external auditory canal because stenosis of the canal is possible without appropriate management.[30] Use only a local anesthetic agent without epinephrine to prevent potential complications. Close simple lacerations primarily with interrupted 6–0 nonabsorbable suture (e.g., nylon or Prolene).

A cotton plug can be inserted into the ear canal during irrigation for patient comfort. **Do not irrigate with such force as to further dissect the cartilage from the perichondrium.**[14] Skin debridement is not recommended in most cases as there is little excess skin available to cover the cartilage.[17] **Close the wound without suturing the cartilage to decrease the infection risk caused by placement of internal sutures if the injury to the cartilage is small and the edges are well opposed.**[5] Use 6–0 nonabsorbable suture (e.g., nylon or Prolene) in a simple interrupted stitch. Repair the anterior aspect of the auricle first to allow for greater accuracy aligning the more cosmetically important anterior aspect.[6,14] **Use care to assure all cartilage is covered with skin to prevent the development of chondritis.**[27]

Close wounds involving soft tissue and cartilage loss of less than 5 mm with a wedge excision and repair technique (**Figure 119-5**).[14] **The auricular skin does not stretch to allow coverage of defects.** The wedge excision technique allows a primary closure that would otherwise have been difficult to achieve without distortion or buckling the anatomy of the auricle due to the underlying cartilage. Debridement performed by the Emergency Physician is controversial. Saving as much tissue as possible is best and leaves more for the surgeon to manipulate if revision is necessary.[14,17] Consult a Plastic

A B

FIGURE 119-4. Ear laceration. ***A.*** Before repair. ***B.*** After repair. (Photo courtesy of Brian Lin, MD.)

Surgeon or Otolaryngologist prior to performing any debridement, if possible.

Excise a full-thickness triangle of tissue in the antihelix with a #15 scalpel blade (**Figure 119-5*A***).[31] First mark the area with a surgical marking pen. The angle at the apex should be no more than 30°.[32] Carefully undermine the skin adhering to the cartilage along the wound margins anteriorly and posteriorly, allowing for elevation of the skin off the cartilage to ensure a tension-free closure.[31] The resultant smooth wound edges can then be closed in a layered fashion after hemostasis has been achieved. **Approximate the skin on the anterolateral surface followed by the posterior surface** with simple interrupted stitches using 6–0 nonabsorbable suture (e.g., nylon or Prolene). **Carefully approximate the ridges and valleys to minimize the cosmetic defect. Place the sutures through the skin and perichondrium and not the cartilage.** The skin and underlying cartilage are so adherent to each other that it is not necessary to close the cartilage separately. A preferred technique by some is to place numerous interrupted stitches through the skin and perichondrium on either side of the wound (**Figure 119-5*B***) and then approximate the wound edges by tying the sutures (**Figure 119-5*C***). It is believed that the cartilage fragments will be drawn together and heal much better.

Auricular lacerations can involve one or all layers without any loss of tissue. The cartilage may protrude into the wound further than the overlying skin (**Figure 119-6*A***). This type of wound is difficult to close primarily without debridement as the skin does not stretch to cover the cartilage. Use a #15 scalpel blade to carefully trim the cartilage back to the level of the skin or so that the skin overhangs the cartilage by 1 mm (**Figure 119-6*B***). This allows the skin edges to be everted when closed. Approximate the skin and perichondrium with simple interrupted stitches using 6–0 nonabsorbable suture (e.g., nylon or Prolene).

Extensive lacerations of the auricle are managed in a similar manner. Trim any protruding cartilage as described above. Place simple interrupted stitches using 6–0 absorbable suture (e.g., Vicryl or Dexon) through the perichondrium to approximate the cartilage at important landmarks and remove tension from the wound edges. Approximate the skin and perichondrium with simple interrupted stitches using 6–0 nonabsorbable suture (e.g., nylon or Prolene).

A B C

FIGURE 119-5. The wedge excision and repair technique for auricular lacerations. ***A.*** Excise a full-thickness triangle of tissue. ***B.*** Place interrupted nonabsorbable sutures through the skin and perichondrium. ***C.*** Approximate the wound edges by tying the sutures.

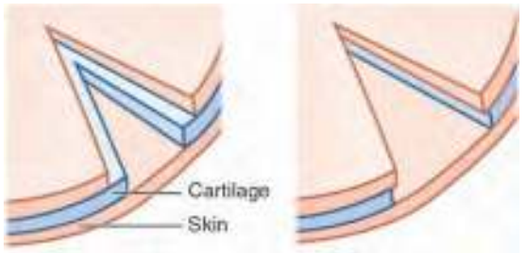


FIGURE 119-6. Repair of an auricular laceration. ***A.*** The skin has retracted and the cartilage protrudes into the wound. ***B.*** Trim the cartilage so that it is level with the skin or so that the skin overhangs the cartilage by 1 mm.

Lacerations of the external auditory canal require repair only if the underlying cartilage is exposed. This is done to prevent a chondritis. Pack the external auditory canal with a nonabsorbent wick (e.g., petrolatum gauze wrapped around a cotton ball) to approximate the wound edges and speed the healing process.

Consult a Plastic Surgeon for wounds with tissue loss of greater than 5 mm, wounds with exposed cartilage that cannot be covered without sacrificing greater than 5 mm of cartilage, complete or almost complete ear avulsion injuries, and injuries with devitalization of the auricle. **Care for the avulsed auricle as an "amputated part" to preserve viability should the consultant desire to pursue reimplantation.**

Uncomplicated wounds not involving the auricular cartilage require local wound care and suture removal in 4 to 5 days. Larger wounds and those involving the auricular cartilage require oral antibiotics to cover skin flora and a dressing that conforms to the anatomic configuration of the auricle (Chapter 200). The dressing will provide support and prevent an auricular hematoma from forming. Remove the sutures in 3 to 5 days in children and 4 to 5 days in adults.[28] Removal in 7 days has also been advocated.[31] The decision when to remove the suture is up to the discretion of the Emergency Physician and depends on the clinical situation.

The complications following ear laceration repair are similar to those occurring after all wound repairs. Specific problems include the development of a chondritis, which is much more likely if the auricular cartilage is left exposed. Deformities can be due to the injury itself, poor repair techniques, or the development of an auricular hematoma secondary to poor ear splinting. Antistaphylococcal antibiotic coverage is recommended in cases where cartilage has been exposed or a hematoma has been drained. Recheck drained hematomas in 24 hours to evaluate for reaccumulation.[28] Refer to Chapter 200 for a complete discussion of auricular hematoma complications.

NASAL LACERATIONS

The structure of the nose is composed of osseous, cartilaginous, and fibrofatty tissue. Lacerations of the nose are often caused by blunt trauma. Inspection for associated injuries (e.g., facial fractures, cerebrospinal fluid rhinorrhea, or a nasal septal hematoma) is critical. A hematoma can develop between the cartilage and perichondrium of the septum and deprive it of nourishment with consequent septal perforation or saddle-nose deformity.

Important points to note in dealing with nasal lacerations are the extent of the laceration and the structures involved. Cartilage involvement increases the likelihood of developing an infection. Lacerations are difficult to close because the skin is inflexible and lacks redundancy. Associated injuries (e.g., nasal fractures and septal hematomas) must be managed. Suturing back a totally avulsed nose is unnecessary as it will most often fail.[33]

Direct injection of local anesthetic is reasonable for simple lacerations. This can distort the anatomy and be painful. The repair of nasal lacerations requires local anesthesia with an infraorbital nerve block (Chapter 156) or a nasal block (Chapter 203). The nasal mucosa can be anesthetized using cocaine-soaked pledgets (Chapter 203). The location of the injury on the nose determines which nerve blocks are necessary.[34] It is safe to use epinephrine-containing anesthetics on the nose.[17] **Significant defects can occur from a small amount of nasal cartilage debridement.[14] Debridement is not recommended.**

Lacerations limited to the outer aspect of the nose (i.e., skin lacerations and not through-and-through lacerations) can be repaired as simple lacerations (**Figure 119-7**). **Minimize any debridement as the lack of redundancy of nasal skin can result in disfiguring scarring.[35]**

Lacerations involving the nasal cartilages require a thorough cleansing, minimal if any debridement, and a multilayered closure.[36] **Do not place suture through the avascular nasal cartilages as this increases the chances of a postrepair infection.** Approximation of the nasal mucosa, subcutaneous tissues, and skin will oppose the cartilage edges. **It is crucial to have proper alignment of the alar rim and columella to achieve good cosmetic results and to avoid the postrepair complication known as "notching."[33]** The repair proceeds from inside the nose to outward.

The depth of the nasal laceration determines the method of repair.[36] Simple, nongaping injuries are straightforward. Repair these with 6–0 nonabsorbable suture (e.g., nylon or Prolene) or 6–0 absorbable suture (e.g., fast-absorbing gut) using simple interrupted stitches. The latter is preferred in pediatric patients. Gaping lacerations require the placement of absorbable subcutaneous suture before skin closure to relieve tension. The skin overlying the nose is taut and sutures can easily tear through the wound edges.[1,2]

Through-and-through lacerations require layered closure with meticulous alignment of the internal and external alar rim for optimal cosmetic outcome.[36] Begin the repair with a 5–0 nonabsorbable suture (e.g., nylon or Prolene) that aligns the skin surrounding the entrance to the nasal canal at the alar margin. Leave the ends of the suture untied and long. Use this suture to apply gentle traction to facilitate alignment of the mucosa and cartilage layers while placing additional sutures.[4,7] Close the mucosal layer with 5–0 chromic gut suture on a tapered needle with simple interrupted stitches. Suture placement in the cartilage is rarely needed as repair of the overlying skin usually approximates the cartilage. Tie the initial stitch. Close the overlying skin with simple interrupted stitches using 6–0 nonabsorbable suture (e.g., nylon or Prolene), or 6–0 absorbable suture (e.g., fast-absorbing gut), or a continuous subcuticular stitch.[2,14]

ORAL MUCOSAL LACERATIONS

The two common types of oral mucosal lacerations are those involving the buccal mucosa and those at the mucosal reflections (i.e., labiogingival groove or buccolabial sulcus) located at the transition point between the buccolabial mucosa and the alveolar mucosa.[17] The latter are easily missed without manipulation of the lips to allow proper visualization and their depth can appear deceptively shallow without appropriate wound exploration. Recognizing the presence of any foreign material in the wound (e.g., debris or tooth fragments) remains essential. Inspect the teeth for fractures and consider the need for obtaining a radiograph or bedside ultrasound prior to repairing any wounds.[37] Recognizing the possibility of a Stensen duct injury, located adjacent to the upper second molar, is

A

B

C

FIGURE 119-7. Repair of an external nasal laceration. ***A.*** The laceration. ***B.*** The laceration 1 day postinjury. ***C.*** The laceration 7 days postinjury. (Photo courtesy of Benjamin C. Paul, MD.)

critical in the management of a buccal laceration and requires an Oral Surgeon.

Most oral mucosal lacerations are small, heal without intervention, and do not require repair. Wounds requiring repair are those large enough to trap food particles (e.g., greater than 2 to 3 cm) and wounds with a tissue flap that falls between the occlusive surfaces of the teeth.[6] Consider the age of the wound prior to preceding with primary repair. Lacerations in the oral cavity more than 6 hours old may be at higher risk for infection.[37]

Coated 4–0 Vicryl suture on a tapered needle is preferred by some when closing the oral mucosa.[37,38] Vicryl suture is less irritating to the child and is easier to work with than plain gut suture or chromic gut suture. Tapered needles are less traumatizing to the tissue.[37,38] Some prefer to place 4–0 silk suture as it is not irritating to the patient and does not tempt the patient to "play" with the suture with their tongue or bite through them. The disadvantage of silk sutures is that they require a return visit for removal. The lacerations can be closed with a continuous stitch or simple interrupted stitch.[38]

Only include the mucosa in the stitch to prevent an external pucker if the underlying muscle is included.[17] Close through-and-through lacerations of the cheek in layers. Irrigate the injury prior to closure through the external open wound. Leave mucosal injuries open if they are less than 2 cm.[38] Close the mucosal layer, if it requires closure, before the muscle and skin. Close the muscle layer and external skin of large flaps.[15] Close the muscle layer first with 5–0 Vicryl suture using simple interrupted stitches and then the skin with simple interrupted stitches using 6–0 nonabsorbable suture (e.g., nylon or Prolene) or absorbable suture (e.g., fast-absorbing gut). Tissue flaps that fall between the occlusal surfaces of the teeth may be approximated or excised.

The role of prophylactic antibiotics for intraoral lacerations is controversial.[39] These wounds are considered contaminated because of the high bacteria count within the mouth.[38] Evidence to date is inconclusive regarding the benefit of prophylactic antibiotic therapy. Administration is guided by Emergency Physician discretion. Make the patient aware that it is normal for buccal mucosa lacerations to often develop a white ridge during the healing process and it is not a sign of infection.[6] A diet of soft foods and liquids is recommended for the first 2 to 3 days after the repair. Rinse the mouth gently two or three times a day and after meals with chlorhexidine solution.

LIP LACERATIONS

The lip has a layered anatomy and requires a layered repair when injured to achieve an optimal cosmetic outcome, maintain proper function, and avoid complications. The skin of the face meets the dry vermilion at the junction between them known as the vermilion border (**Figure 119-8**). This border is further divided into the white line and red line. Its relative pallor compared to the lip and skin makes it easily identifiable.[6] The dry vermilion is composed of a specialized epithelium that is translucent, allowing the vascular papillae below it to give the lips their pink color.[14] The dry vermilion then transitions into the wet vermilion at the wet-dry junction as the mouth is entered. The wet vermilion is composed of the oral mucosal epithelium. The internal layered structure of the lip from the oral mucosal epithelium to the epithelium of the face is the mucosal

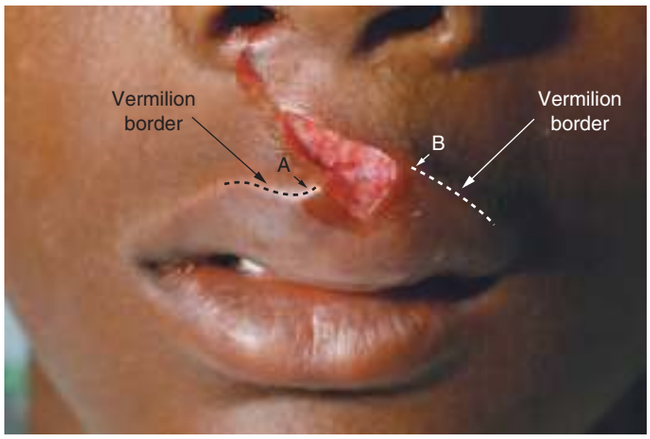


FIGURE 119-8. A lip laceration that crosses the vermilion border. Points "A" and "B" are connected to realign the vermillion border (*dotted lines*). (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2016. Photo contributor: Kevin J. Knoop, MD, MS.)

epithelium, the submucosa that contains minor salivary glands, inner fibrofatty junction, muscle (i.e., orbicularis oris), outer fibrofatty junction, subcutaneous fat, dermis, and facial epithelium.[38] The orbicularis oris muscle has multiple functions including participation in facial expression, speech, and retaining oral secretions.[14]

Attention to detail is essential for attaining a good cosmetic result in repairing lip lacerations, as they can result in devastating cosmetic defects if not repaired properly. Malalignment of the vermilion border or the "white line" (i.e., the border between the skin of the face and the red part of the lip) by as little as 0.5 to 1 mm will be easily noticeable.

Lip lacerations can be safely closed primarily within 24 hours of the injury.[38] Lacerations presenting beyond this time frame may still be closed after consultation with a Plastic Surgeon as irrigation in the Operating Room or healing by secondary intention may be indicated. **The lips are best anesthetized with regional nerve blocks (Chapter 156 and 209) to not distort the landmarks that demand precise alignment.** This can be accomplished by blocking the infraorbital nerve and mental nerve for upper and lower lip lacerations, respectively. Avoid using epinephrine with anesthesia, as this will blunt the vermilion border landmark.[40] Inspect the teeth for fractures and explore the wound for foreign bodies. An embedded tooth fragment or other debris can act as a nidus for infection.[40] A unique aspect of the lip is that any traumatized minor salivary glands should be removed to prevent delayed formation of a mucocele.[38] **Be sure not to debride any tissue containing the vermilion border.** Irrigate the wound adequately prior to closure.

Discussing the repair of a through-and-through laceration of the lip that includes the vermilion border provides explanations for repair of each layer. The points made are applicable to all less severe lacerations of the lip. Consider debridement of irregular borders.[40] It is recommended to start the repair of any vermilion border laceration with a tacking stitch and align the red and white lines precisely (**Figures 119-8 and 119-9*A***).[1,14,17,38] **Misalignment of this border by as little as 1 mm can result in a cosmetically displeasing outcome.**[14] Use 6–0 nonabsorbable suture (e.g., nylon or Prolene). This stitch can be tied immediately or left untied with long tails, which is helpful for through-and-through lacerations (**Figure 119-9*A***). Apply gentle traction on this initial alignment suture when needed to help approximate the underlying tissues as the remainder of the repair is performed. The repair proceeds from the inside out with the oral mucosa first to the wet-dry junction using buried interrupted stitches. Use 5–0 chromic gut suture or coated 4–0 Vicryl suture on a tapered needle. Repair the orbicularis oris muscle to include the inner and outer fibrofatty layers. Use buried interrupted stitches with 4–0 or 5–0 Vicryl suture, plain gut suture, or chromic gut suture (**Figure 119-9*B***). **The muscle must be accurately approximated anteriorly and posteriorly to prevent contraction away from the wound edge and produce a scar with obvious ridging or depression when the lip is in function (i.e., not at rest).**[38] Close the dry vermilion and facial skin by placing simple interrupted stitches with 6–0 nonabsorbable suture (e.g., nylon or Prolene) or fast-absorbing gut suture (**Figure 119-9*C***).[6]

The aftercare for a sutured lip laceration is much the same as for oral mucosal lacerations. Instruct the patient to avoid bringing excessive pressure to bear on the suture line. Remove nonabsorbable sutures in 4 to 5 days to avoid scarring. Warn parents that a child may bite the stitches while the lip is still anesthetized and advise them to distract the child from doing so during this time.[6,40] Administration of prophylactic antibiotics is likely beneficial for through-and-through lip lacerations.[17,41]

TONGUE LACERATIONS

Most tongue lacerations are the result of oral trauma and tongue biting and usually heal well without intervention (**Figure 119-10**).[42] There is controversy associated with the indications for laceration repair of the tongue.[14] Lacerations that do not require repair are generally small, linear, nongaping, and superficial lacerations located in the central tongue region or small flaps on the edge of the tongue that can be excised (**Figure 119-10*A***). Repair full-thickness

FIGURE 119-9. Lip laceration repair. ***A.*** Always approximate the vermillion border first. ***B.*** Approximation of the orbicularis oris muscle. ***C.*** Approximation of the mucosal surface and the skin.

A

B

FIGURE 119-10. Tongue lacerations. ***A.*** Nongaping. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2016. Part A: Photo contributor: James F. Steiner, DDS.) ***B.*** Gaping. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2016. Part B: Photo contributor: Lawrence B. Stack, MD.)

lacerations, large lacerations of a free edge, large flaps, lacerations that continue to bleed, those that are likely to entrap food particles, and lacerations that bisect the tongue (**Figure 119-10*B***).[6,14,43] Tongue amputations and large avulsions require microsurgical repair by an Oral Surgeon or an Otolaryngologist.[43] The tongue's generous blood supply allows wounds to heal well yet can cause serious hemorrhage and potential airway compromise.[6,42] Evaluate patients who have tongue lacerations that require repair for potential airway problems, the need for procedural sedation, or the need for general anesthesia.[40] Some wounds requiring closure can be very challenging to repair.[2,3,7,44] These may include large lacerations over 1 cm, gaping wounds, actively bleeding lacerations, U-shaped lacerations, wounds that bisect the tongue, and large flaps on the tongue edge (**Figure 119-11**). The main problem is not the repair but achieving control of the area to facilitate the repair.

Anesthetize the tongue via local wound infiltration or a lingual nerve block for the anterior two-thirds of the tongue (Chapter 209). These can be supplemented with procedural sedation techniques (Chapter 159). Keep the mouth open during the repair by using a bite block, padded tongue depressor, or a Denhardt-Dingman side mouth gag.[6] Instruct an assistant to gain control of the tongue. Grasp the anesthetized tip of the tongue with a towel clip, a suture through the tip of the tongue, or a gauze square (**Figure 119-11**).[38] Thoroughly irrigate the wound. Close the laceration using absorbable 4–0 plain gut, chromic gut, or Vicryl sutures. Take large bites that include the mucosal and muscular layers of the tongue. Take full-thickness bites to include the two mucosal surfaces and the muscle between or half-thickness bites with one suture from above and another from below.[43] **Buried or deep sutures are not required when taking large bites.** Multiple well-secured sutures are preferred to prevent the untying of suture material with tongue motion.[42] An alternative is inverted sutures tied loosely to prevent untying or tearing of the muscle fibers as the tongue swells.[6,43] Tissue adhesive may be used in the future to close tongue lacerations.[45]

FIGURE 119-11. Repair of a tongue laceration involving the margin. Note the stitch placed at the tip of the tongue to provide control. (Used with permission from Shah BR, et al: *Atlas of Pediatric Emergency Medicine*, 2nd ed. New York: McGraw-Hill; 2013.)

The infection rate of tongue lacerations is low. Some recommend that all patients be discharged home with a prescription to use an antibiotic mouthwash (e.g., chlorhexidine gluconate 0.12%) twice per day for 1 week.[14,43] Extensive complex tongue lacerations are at a greater risk for infection and should be treated with prophylactic antibiotics that cover normal oropharyngeal flora.[42] Children with tongue lacerations may chew off the stitches. Inform parents of this and instruct them to distract the child until the local anesthesia wears off.[40] Aftercare instructions are similar to those for oral mucosal lacerations.

GINGIVAL LACERATIONS

Gingival lacerations differ from other anatomic sites in that there is often no subcutaneous tissue available to anchor the flap. Small gingival lacerations tend to heal well without intervention because of the extensive blood supply in this area. Repair wounds that are large, actively bleeding, gaping open, or that fall onto the occlusive surface of the teeth.

Flaps retract after injury and initially appear too small to cover the wound. They give the appearance of an avulsion. However, the flap can usually be spread to cover the wound.[46] A topical anesthetic (e.g., 20% benzocaine) can be used prior to primary repair. Alternatively, perform a regional nerve block (Chapters 156 and 209). The anterior maxillary gingiva as far posterior as the maxillary molars can be anesthetized by performing a regional block of the infraorbital nerve.[47]

A B

FIGURE 119-12. Gingival laceration repair. ***A.*** Front view. ***B.*** Superior view.

Flap lacerations of the teeth (i.e., gingival degloving) expose the alveolar ridge and tooth roots pose a special problem, as there is no subcutaneous support to anchor the mucosa. A flossing technique can be performed using 4–0 or 5–0 chromic gut or Vicryl suture to hold the flap in place.[46] The suture can be passed between the teeth or the needle can be passed through the gingiva. The technique requires the placement of a 4–0 or 5–0 absorbable suture that first runs circumferentially around and then is tied posterior to the tooth.[48] Place the suture 2 to 3 mm proximal to the gingival margin (**Figure 119-12*A***). Place the suture through the gingiva so that it passes circumferentially around the tooth to secure the flap (**Figure 119-12*B***). Loosely tie the suture on the inner aspect of the tooth so that the knot does not irritate the lip or strangulate the tissues (**Figure 119-4*B***). The aftercare is the same as described under "Tongue Lacerations" in this chapter. Consider a prescription for an antibiotic mouthwash (e.g., chlorhexidine gluconate 0.12%) twice per day for 1 week.[43,46]

SUTURING THROUGH HAIR

It is inadvisable to shave hair bordering the edges of a laceration, as this only serves to increase the likelihood of a wound infection.[47,49] Instead of shaving the hair, brush it aside or mat it down using petroleum jelly or antibiotic ointment prior to wound repair. **It is important to never to shave eyebrows or eyelashes as they will take months to grow back.**[50] Always debride an eyebrow laceration oblique to the hair follicles and not perpendicular to the skin edge (**Figure 119-13**). This will reduce the loss of hair follicles and prevent ingrown hairs.

MUSCLE LACERATIONS

The repair of muscle lacerations begins with a careful assessment of the extent of injury and the level of contamination prior to repair. Large and/or grossly contaminated muscle injuries require operative management. All other muscle injuries may be managed in the Emergency Department. Treatment should focus on repair or reconstruction of a muscle using its long tendons of origin and insertion to anchor the repair, as the muscle tissue alone is inadequate for suture repair.[51]

Lacerations of the fascia surrounding a muscle are common. Thoroughly cleanse the area and debride any devitalized tissue. Approximate small violations of the muscle fascia with simple interrupted stitches using 3–0 or 4–0 absorbable suture (e.g., Vicryl or Dexon). Closing small rents will prevent symptomatic herniation of muscle tissue through them in the future. Do not repair large violations of the muscle fascia. Anecdotal reports of muscle compression and compartment syndromes after such repairs abound.

Lacerations through the muscle require a thorough cleansing and debridement of any devitalized tissue. Repair the laceration with 3–0 or 4–0 absorbable sutures (e.g., Vicryl or Dexon). **Place modified horizontal mattress stitches similar to repairing an extensor tendon (Chapter 96) to close the laceration in a muscle.** Interrupted sutures are not effective, as they tend to pull through the muscle fibers and not hold.

INCOMPLETE LACERATIONS

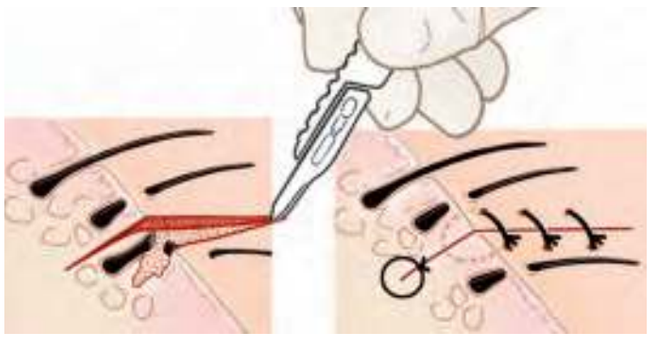
Incomplete lacerations involve the epidermal and superficial papillary layers of the skin but spare the deep papillary layer. Excise the loose epidermal pieces of skin. Cover the wound with petrolatum gauze and a pressure dressing. The wound can heal by second intention. Alternatively, use Steri-Strips (Chapter 116) or tissue adhesive (Chapter 117) to close the laceration.

WOUNDS OF UNEQUAL THICKNESS

Wounds of unequal thickness are not suited for repair with simple interrupted sutures. Unequal tissue loss on each edge of a wound creates a thick edge–thin edge wound. **The depressed edge must be elevated to the level of the nondepressed edge to attain proper wound apposition and cosmesis.**

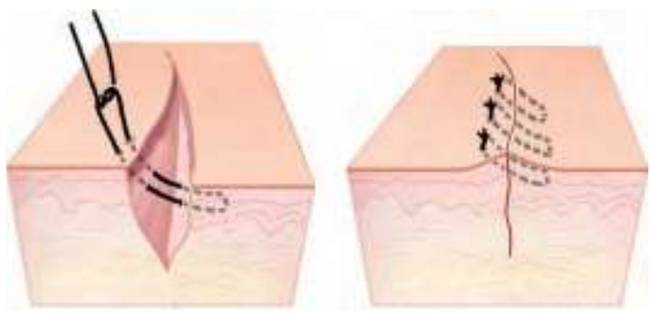
There are two techniques to repair wounds with edges of unequal thickness. The first technique uses a half-buried horizontal mattress stitch (**Figure 119-14**).[1,3] Place the suture through the thick edge of the wound, across the wound and buried into the subcutaneous tissue of the thin edge, and back out the skin of the thick edge (**Figure 119-14*A***). Apply traction to the suture and tie it to approximate the wound (**Figure 119-14*B***). Apply an ointment-based compressive dressing.

The second technique requires undermining both wound edges at the same depth in the subcutaneous tissue plane (**Figure 119-15**).[7] Make an incision in the subcutaneous tissues of both wound edges and at the same level (**Figure 119-15*A***). Undermine the area to free



A B

FIGURE 119-13. Repair of eyebrow lacerations. ***A.*** Debride the wound edges obliquely and parallel to the hair follicles and not perpendicular to the wound edges. ***B.*** Approximate the wound edges.



A B

FIGURE 119-14. Closing a wound with edges of unequal thickness using half-buried horizontal mattress stitches.

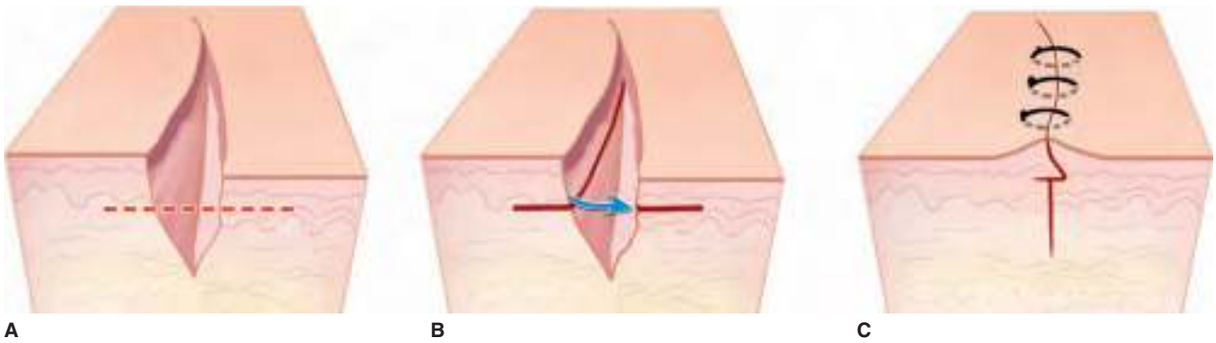


FIGURE 119-15. An alternative technique to close a wound with edges of unequal thickness. ***A.*** Make an incision in the subcutaneous tissue of both wound edges. ***B.*** Undermine the edges. Transpose the subcutaneous tissue of the thicker side into the undermined area of the thinner side (*arrow*). ***C.*** Approximate the wound edges using interrupted stitches.

the tissue flaps (**Figure 119-15*B***). Grasp the subcutaneous tissue flap from the thicker side and insert it under the thinner side beneath the undermined area (**Figures 119-15*B* and 119-15*C***). Place a buried horizontal mattress stitch to maintain the flaps in position. This "flap" elevates the depressed wound edge and facilitates appropriate wound approximation (**Figure 119-15*C***).[7] Place interrupted stitches to approximate the wound edges (**Figure 119-7*C***).

Tangential flap lacerations over thin skin (e.g., the dorsum of the hand or the pretibial area) where there is very little subcutaneous tissue can be approximated with a specially placed simple interrupted suture. Insert the needle through the tip of the thin edge, across the wound, into the dermis of the thick edge, and out the skin of the thick edge. Apply traction to the suture to pull the thick edge up to meet the thin edge, producing good approximation rather than overlap of the edges. Tie the sutures to oppose the wound. Apply an ointment-based compressive dressing.

An alternative technique uses Steri-Strips to strengthen the skin so the suture does not pull through the thin skin. Apply benzoin to the intact skin on both edges of the laceration. Do not get the benzoin in the wound. Allow 1 to 2 minutes for the benzoin solution to dry and become tacky. Place stitches through the skin and Steri-Strips.

FLAP LACERATIONS

Flap lacerations occur when shearing forces to the skin tear the dermis from the underlying subcutaneous tissues. This type of laceration is problematic because the flap is separated from its blood supply except for the blood entering through the base. This makes the flap susceptible to necrosis and infection. A flap will remain viable if the base of the flap is three times its length.[53] Flaps with a ratio of less than 3:1 are less likely to survive intact.

A flap can easily be repaired primarily if it has viable edges and meets or exceeds the 3:1 ratio of base to length. Anesthetize, clean, and prepare the area. Trim the excess fatty tissue from the underside of the flap. This will improve the chances of healing. Place a half-buried horizontal mattress stitch to close the corner or the tip of the flap (**Figure 119-16*C***). Approximate the sides of the flap with simple interrupted stitches or half-buried mattress stitches (**Figure 119-16*C***).

Some viable flaps have nonviable edges that must be debrided prior to closure to ensure proper cosmesis and survival of the tissue. Debride the nonviable edges sharply with a #15 scalpel blade or an iris scissors (**Figures 119-16*A* and 119-16*B***). Place a half-buried horizontal mattress stitch to close the corner (**Figure 119-16*C***). This is necessary to close the flap and relieve pressure. Approximate the sides of the flap with simple interrupted stitches, mattress stitches, or half-buried mattress stitches (**Figure 119-16*C***).

Some flaps may be too small after debridement or be under too much tension to stretch across the wound. These flaps can be closed by forming a Y-shaped closure instead of a V-shaped closure (**Figure 119-16*C***). Place a half-buried horizontal mattress stitch to close the tip of the flap. Approximate the base and arms of the Y with interrupted stitches, mattress stitches, or half-buried mattress sutures.

A flap laceration may present difficulties. It may not be possible to revise and repair it, or it may be nonviable. It is often possible to

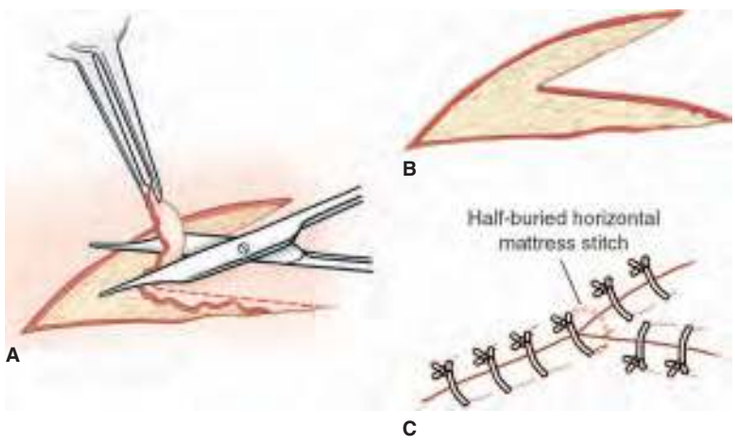


FIGURE 119-16. Repair of a flap laceration. ***A.*** Sharply debride the edges. ***B.*** The resultant defect. ***C.*** Approximation of the wound edges. First place a half-buried horizontal mattress stitch to close the tip of the flap. Approximate the remaining wound edges with interrupted stitches.

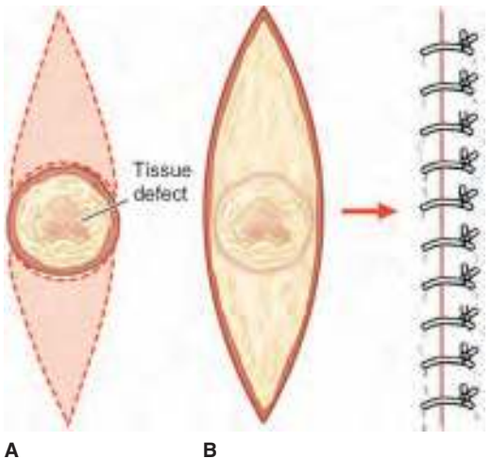


FIGURE 119-17. Closure of a tissue defect. ***A.*** Excise the defect and a surrounding ellipse. ***B.*** Approximation of the wound edges with deep and cutaneous stitches.

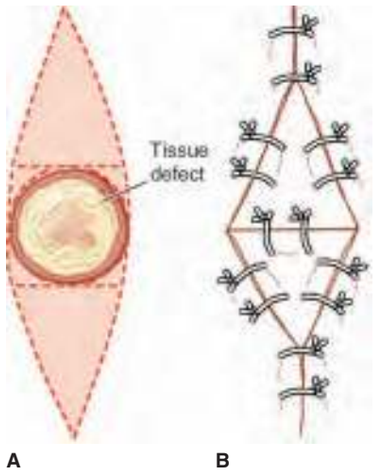


FIGURE 119-18. The double V-Y closure to repair a tissue defect. ***A.*** Create an ellipse centered about the tissue defect. Remove the tissue defect and form straight edges at the bases of the triangular flaps. ***B.*** Approximate the bases of the triangular flaps followed by the arms and bases of the Y's using interrupted stitches.

"ellipse" the flap (**Figure 119-17**). Excise the tissue defect to form a circle. Excise a surrounding ellipse of tissue centered about the circle (**Figures 119-17*A* and 119-17*B***). The ellipse must be 2½ to 3 times as long as its greatest width. Undermine the edges of the ellipse. Approximate the center of the resulting defect using a locked vertical mattress stitch, which allows approximation of the remainder of the wound with minimal extrinsic tension. Approximate the remainder of the resulting defect with deep sutures, if required, and cutaneous sutures (**Figure 119-17*B***).

There is not enough tissue in some cases to cover the defect no matter what technique is attempted. These wounds may be left to heal by secondary intention if small or referred to a Plastic Surgeon for skin grafting. The only complication unique to the repair of flap lacerations is the possibility that the flap may not survive and require referral, revision, or skin grafting.

AVULSION INJURIES

The treatment of tissue avulsion injuries varies depending on the amount of tissue lost and its depth. Allow full-thickness defects less than 1 to 2 cm^2 to heal by secondary intention after debridement. Full-thickness defects greater than 2 cm^2 require a different approach using skin grafts or flaps or converting the wound to one that can be closed primarily. Treat avulsed tissue as an "amputated part" that may be used in the repair process. Consider consulting a Plastic Surgeon for a large avulsion that cannot be closed primarily or to convert it into a wound that can be closed primarily.

Some defects can be closed primarily. Examples include circular defects or triangular defects that are converted to ellipses (**Figure 119-17**).[3] This technique is described above under "Flap Lacerations."

Larger defects require the use of a double V-Y closure. This converts the flap to a different shape prior to the repair and thus increases the chances of successful primary closure (**Figure 119-18**).[3] Create two sliding pedicle flaps with a #15 scalpel blade. **Incise the skin and dermis but not the underling subcutaneous tissue to form an ellipse centered about the tissue defect** (**Figure 119-18*A***). Remove the tissue defect and debride the tissues at the base of the flaps to form two straight edges (**Figure 119-18*A***). Gently undermine the edges of the ellipse. **Do not undermine the triangular tissue flaps so their vascular supply is preserved.** Slide (i.e., advance) the flaps on their subcutaneous pedicles until the bases are touching (**Figure 119-18*B***). Approximate the base of one flap to the other using simple interrupted stitches using nonabsorbable suture (e.g., nylon or Prolene) (**Figure 119-18*B***). Approximate the arms and bases of the Y using simple interrupted stitches using nonabsorbable suture (e.g., nylon or Prolene).

MULTIPLE SMALL SKIN FLAPS

Numerous small skin flaps that are bunched up, as commonly seen when a patient's forehead shatters a windshield, are difficult to repair individually. Excise them as a group to form a single laceration that can be repaired primarily (**Figure 119-19**). Larger and more "spread out" groups of flap lacerations can be repaired in a manner similar to that described under "Multiple Forehead Lacerations," above.

DISTAL FINGERTIP AMPUTATIONS

Injuries of the distal fingertip rank among the most frequently injured parts of the hand.[54] Amputations of the fingertip are defined as loss of the tissues distal to the insertions of the extrinsic flexor

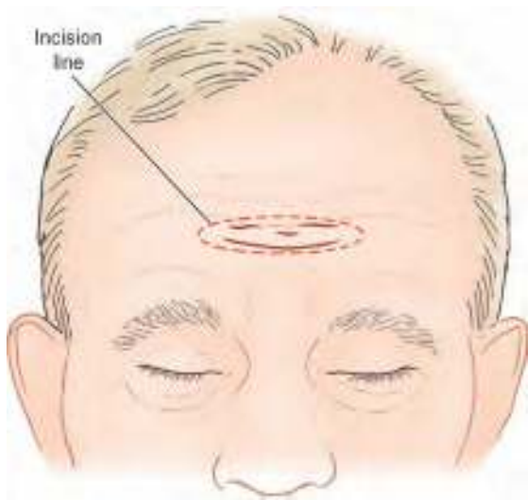


FIGURE 119-19. Repair of multiple small lacerations. Excise the lacerations as a group to form a single wound that can be closed primarily.

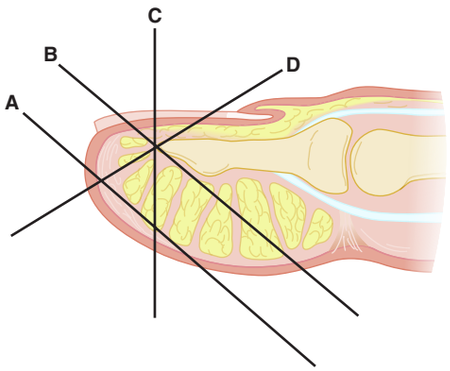


FIGURE 119-20. Fingertip amputations. ***A.*** Volar angulation without bone exposure. ***B.*** Volar angulation with bone exposure. ***C.*** Transverse or perpendicular angulation with bone exposure. ***D.*** Dorsal angulation with bone exposure. (Used with permission from Tintinalli JE, et al: *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th ed. New York: McGraw-Hill; 2016.)

FIGURE 119-21. A fingertip degloving. (Photo courtesy of www.lifeinthefastlane.com.)

and extensor tendons on the distal phalanx, distal to the level of the lunula. These injuries may include skin, pulp, bone, the nail matrix, and/or the nail plate (**Figure 119-20**).[54] Multiple classification systems to describe the pattern of injury and facilitate communication have been developed (e.g., Allen, Fassler, and Tamai classification schemes).[55] The Allen classification is based on the transverse level of amputation. The Fassler system is based on the geometry of pulp loss and whether there is exposed bone. The Tamai classification system better defines vascular injury and facilitates treatment planning. Anatomic classification schemes for the level of injury are multiple and make literature comparisons problematic. It is best to describe injuries related to anatomic landmarks.

The goals of managing fingertip amputations include optimizing healing time and cosmesis, preserving sensory function and length, limiting nail deformity, and minimizing time lost from work.[56] Treatment options have evolved remarkably over the past 30 years, although change in clinical practice has lagged. Multiple treatment modalities exist and depend on whether the amputated part was retrieved, its condition, the character of the injury, the patient's preference and underlying health status, availability of consultants, and sophistication of consultants. Current recommendation is to place the amputated part in saline-moistened gauze in a bag that is kept cool in an ice-water mixture.[57]

Perform a physical examination of the injured finger. Obtain radiographs of the finger and the amputated part. Perform a digital block (Chapter 156). Obtain a bloodless field by applying a digital tourniquet. Thoroughly irrigate the wound and debride any devitalized tissue.

Amputations with pad loss of less than 1 cm, with and without bone exposure, can be treated with conservative nonsurgical management with serial dressing changes of nonadherent gauze (**Figures 119-21 and 119-22**).[58] A petrolatum gauze dressing is best used to cover the wound. It is effective at optimizing healing and minimizing discomfort when removed.[57] Attempt to place this dressing only on the wound itself to avoid maceration of the intact skin. Cover this dressing with standard gauze. Instruct the patient to soak the wound in warm water mixed with antibacterial soap daily for 10 minutes followed by irrigation with tap water and reapplication of petrolatum gauze dressing covered with gauze for the first 10 to 15 days.[59] Apply a finger splint for protection. Advise the patient to follow-up with a Hand Surgeon within 48 hours.

Nonoperative open management has been shown to be at least as effective as primary closure, local and distant tissue-transfer flap procedures, and grafts.[59-68] Conservative management is advocated in children less than 12 years of age, as they have greater regenerative potential than adults.[54,69]

Amputations proximal to the lunula are less common. Those who address the issue specifically recommend shortening and primary closure without discussion or data. These injuries require emergent consultation for possible reimplantation. Injuries from the flexor digitorum profundus insertion to approximately the level of the mid-nail can be replanted if the amputated part is available.[70-72] The purpose of replantation is the restoration of cosmesis and function. The patient's perception may be that replantation will restore functional and cosmetic normality. Inform the patient that this is doubtful. Consultation with a Hand Surgeon is recommended for all patients with a complete amputation proximal to the lunula to determine if replantation is indicated.[59]

Conservative therapy usually provides acceptable preservation of contour, better salvage of two-point discrimination than grafts, is cheaper than replantation, is associated with a virtual absence of postsurgical infections, and limits joint stiffness. It is equivalent to other techniques in terms of functional use, preservation of length, and time absent from work. Eventual patient satisfaction may be even greater than with surgical techniques. Patients may have to be convinced that closure by secondary intention is both rational and optimal. A set of photographs demonstrating this is often helpful.

FIGURE 119-22. A fingertip amputation. (Photo courtesy of ww.lifeinthefastlane.com.)

Clean, prep, and anesthetize the finger. Apply a tourniquet if not done previously. Fingertip amputations tend to bleed profusely. Achieve hemostasis with direct pressure using a nonadherent dressing while the hand is raised above heart level for several minutes. Do not remove the dressing if this is successful. Consider the application of a sterile compression sponge covered with a nonadherent dressing and the application of pressure if the bleeding continues. Attempts at cauterizing bleeding vessels are usually unsuccessful because of the tissue's vascularity.

The presence of protruding bone poses several treatment options. Some physicians rongeur any protruding bone exposure less than 0.5 cm in length with soft tissue loss of less than 1 cm^2 until it is flush with the wound surface and thereafter continue open treatment.[59] There is no published evidence supporting rongeuring of protruding bone in adults. Anecdotal statements supporting this technique exist in texts.[62,68] Published reports support not shortening protruding bone in children under the age of 12.[66,73] Healing may be prolonged if protruding bone is not debrided or if it is debrided later. Consider trimming the nail bed back to the bone margin if a remnant overhangs or is not supported by adequate bone.

Dress the wound with either a bulky hand dressing or tube gauze and a disposable splint (e.g., metal or plastic).[74] The use of prophylactic antibiotics is controversial in fingertip amputations, including those with open fractures.[57,75] Consider antibiotics in contaminated wounds or those with significantly devitalized tissue. Refer all patients to a Hand Surgeon or primary care provider in 24 to 48 hours. Inform all patients of the 30% to 50% incidence of sensory impairment (e.g., diminished sensation, cold intolerance, dysesthesia), impaired function, or cosmetic deformity.[61,63,67,68,76] **Document this discussion in the medical record.** Additional indications for consultation with a Hand Surgeon include specific occupational concerns or when the thumb or index finger is affected.[59] The possibility of a wound infection is very small with conservative treatment.[65]

ANIMAL BITES

Approximately 1% of annual Emergency Department visits in the United States are for the management of bite wounds.[77] These bites are from dogs (80% to 90%), cats (5% to 10%), humans (2% or 3%), and rodents (2% to 3%).[78,79] A thorough understanding of the management of these injuries is essential. The goals of management include identification of injuries to underlying structures (e.g., tendon, bone, vasculature or joint space), retained foreign bodies (e.g., teeth), signs of infection, appropriate irrigation and debridement of devitalized tissue, determination of primary versus secondary closure, and administration of prophylactic therapies for infection (e.g., local bacterial infection, tetanus, rabies, hepatitis, syphilis, and HIV).

Obtain plain radiographs if there is concern for a fracture, joint space violation, or retained foreign body. Any injury to tendons or vasculature requires appropriate surgical consultation. A bite wound of the dorsal hand (e.g., clenched fist injury) may cause a tendon injury that may not be visible in the wound field when the fingers are extended. **Examine all hand wounds through a full range of motion of the fingers for tendon injury or penetration of the joint capsule.**[80] Pain with finger motion is suspicious for a deep compartmental infection or tendonitis.[79]

Signs of local wound infection warrant immediate initiation of antibiotic therapy. The microbiology of wound infections from bites is directly related to the oral flora of the offending species. The most common bacteria involved in dog and cat bites include *Staphylococcus, Streptococcus*, and *Pasteurella*.[81] *Pasteurella multocida*, found most commonly in cat bite wounds, can cause a wound infection within hours of injury. An important pathogen is *Capnocytophaga canimorsus*, which can cause fulminant sepsis in immunocompromised patients bitten by dogs. Primary closure of bite wounds is avoided in patients with immunodeficiencies.[82] Human bite wounds are usually infected by *Staphylococcus*, *Streptococcus*, and *Eikenella*.[83] Patients with systemic symptoms warrant hospitalization for intravenous antibiotic therapy.[79] Appropriate treatment is with ampicillin-sulbactam or clindamycin plus a fluoroquinolone, if penicillin allergic, for adults and clindamycin plus trimethoprim-sulfamethoxazole for children.[83]

The importance of proper local wound care with irrigation and debridement cannot be overemphasized. It prevents infection more effectively than prophylactic antibiotics.[79] **Copiously irrigate the wound. Antibiotic coverage is no substitute for meticulous wound cleansing. Debridement of devitalized tissue decreases infection risk.**[84] Cleaning the wound with soap and water will decrease the transmission rate for rabies.[80] The infection rate is further decreased 20-fold by irrigating with sufficient pressure by using a 19 gauge needle or angiocatheter attached to a 30 mL syringe.[79]

Determining whether to close the wound primarily is based on numerous factors (**Table 119-1**). **Wounds at high risk for infection should not be closed primarily. These include wounds sustained by a cat or human bite, puncture wounds, wounds < 2 cm in size, hand and foot wounds, wounds more than 12 hours old, and wounds in immunosuppressed patients.**[79] Do not close wounds less than 2 cm in size in cosmetically insignificant areas. They can

TABLE 119-1 The Repair of Laceration-Type Bite Wounds

Location	Dog bites	Cat bites	Human bites
Face	PC	PC	PC
Scalp	PC	PC	PC
Neck	PC	PC	PC
Trunk	PC if > 2 cm DPC if < 2 cm	PC if > 2 cm DPC if < 2 cm	PC or DPC
Arm	PC if > 2 cm DPC if < 2 cm	PC if > 2 cm DPC if < 2 cm	PC if > 2 cm DPC if < 2 cm
Hand with foreign body, extensor tendon injury, joint capsule injury, or bone involvement	Hand Surgeon consultation, exploration, and intravenous antibiotics	Hand Surgeon consultation, exploration, and intravenous antibiotics	Hand Surgeon consultation, exploration, and intravenous antibiotics
Hand with only soft tissues involved	DPC or secondary closure	DPC or secondary closure	DPC or secondary closure
Leg	PC if > 2 cm DPC if < 2 cm	PC if > 2 cm DPC if < 2 cm	PC if > 2 cm DPC if < 2 cm
Foot	DPC	DPC	DPC

DPC, delayed primary closure; PC, primary closure.

TABLE 119-2 Indications for Prophylactic Antibiotic Therapy for Bite Wounds

	Dog bites	Cat bites	Human bites
Puncture wound	Yes	Yes	Yes
Hand bites	Yes	Yes	Yes
Facial bites	No	Yes	Yes
Non-hand-laceration-type bites	No	Yes	Yes
Immunocompromised patients*	Yes	Yes	Yes
Surgical closure	Yes	Yes	Yes
Severe crush	Yes	Yes	Yes

*Immunocompromised includes patients with age > 50, diabetes, alcoholism, or asplenia, and patients with any other illness associated with an impaired immune status.

be closed with delayed primary closure or allowed to heal by secondary intention. An exception to this general rule is cat and human bite wounds on the face and neck.[5,82] The excellent vasculature supply and the importance of an optimal cosmetic outcome in these areas may outweigh the risk of infection. Primary closure of dog bites resulted in a similar infection rate to unclosed bites but better cosmesis.[85] Primary closure of a human bite wound to the face is associated with an infection rate of approximately 10%.[85] Have a thorough discussion with the patient regarding this risk and document this discussion in the medical record. Some experts suggest closing the subcutaneous dead space of large hand wounds with a minimal amount of absorbable stitches and closing the skin 3 to 5 days later if there is no evidence of infection.[79] **Limit the use of buried sutures as they can increase the infection rate of a repaired bite wound.**

Determining whether to administer prophylactic antibiotics is based on numerous factors (**Table 119-2**).[77] The data demonstrating their effectiveness for bite wounds are limited. A Cochrane review suggests that prophylactic antibiotics significantly decrease the risk of infection after primary closure in hand wounds sustained by dog and human bites.[86] The review did not include studies on hand injuries from cat bites. It found no benefit of prophylactic antibiotics for wounds sustained to other areas of the body. Wounds sustained from cat bites have a higher rate of infection than those sustained by dog and human bites likely due to the deeper penetration of bacteria resulting from their long and thin teeth. Human bites have a higher rate of infection than dog bites. Experts recommend prophylactic antibiotics for all cat and human bite wounds.[77,79,82] Prophylactic antibiotics are indicated for bites more than 8 hours old with significant crush injury or edema or potential damage to adjacent structures (e.g., bones, joints, or tendons), for deep puncture wounds, in older patients, in immunocompromised patients, and for bites near or in a prosthetic joint.[79,83] Administer the first dose intravenously to obtain effective tissue levels rapidly followed by an oral course.[83] Bite wounds not involving a joint or tendon can be cleaned, debrided, splinted, watched expectantly on an outpatient basis, and treated with oral antibiotics.[87] Instruct the patient to keep the hand elevated and return in 12 to 24 hours for a reevaluation.

Treatment time depends on structures involved.[88] Treat for 5 to 7 days if a tendon or joint is involved. Treat for 3 to 5 days if a tendon or joint is not involved. Antibiotics include amoxicillin-clavulanate (875 mg orally twice daily), doxycycline (100 mg orally twice daily), moxifloxacin (400 mg orally or intravenously [IV] once daily), ampicillin-sulbactam (3 gm IV four times daily), or imipenem (1 gm IV four times daily).

Dress bite wounds allowed to heal by secondary intention with a nonadherent dressing (e.g., petrolatum gauze) to maintain a moist environment for epithelial regeneration.[82] Immobilize extremities with extensive wounds. Apply bulky mitten dressings and support the hand with an arm sling.[79] **Always consider the need for immunoprophylaxis for tetanus, rabies, viral hepatitis, syphilis, and HIV.** Consult the local public health department for guidelines and the incidence of rabies in your community.

The discharge instructions are just as important as the Emergency Department management. Provide the patient with a written copy of a wound care sheet, regardless of whether the wound was closed primarily. Instruct the patient to elevate any involved extremity, even with antibiotic treatment.[89] Arrange follow-up within 24 to 48 hours for a reevaluation of the wound. Physical therapy following a hand infection may be required and initiated 3 to 5 days after any infection resolves.[90]

DEBRIDEMENT OF GUNSHOT WOUNDS

The lack of primary literature on this subject makes it a controversial area. Wounds created by low-velocity bullets tend to cause damage only along the bullet track. Debridement is unnecessary for wounds created by bullets with muzzle energy of less than 400 foot-pounds, as many consider bullets to be sterile.[91] Devitalized and contaminated tissue is more likely to result from shotgun wounds and high-velocity bullets.[92] The shock wave created by the bullet damages tissue distant from the track of the bullet.[92] Consider these wounds for debridement.[92] The debridement of gunshot wounds requires exposure of the entire bullet track and treatment as a delayed closure followed by referral for skin grafting if needed.

Clean, prep, and anesthetize the skin overlying the path of the bullet or shotgun blast for tangential and superficial wounds. Consult a surgeon for other wounds as they need to be debrided in the Operating Room. Incise the wound with a #11 scalpel blade to expose the area. Sharply debride any devitalized and contaminated tissue. Cover the wound and refer the patient to a surgeon for follow-up. Repeat staged exploration at 24 hours and 48 hours to remove necrotic or devitalized tissues.[93] Treat the patient with prophylactic antibiotics.[93] Consider delayed closure techniques, giving priority to reestablishment of bony relationships followed by soft tissue coverage.[93] Consider skin grafting for large areas of tissue destruction for adequate closure.

ABRASIONS

An abrasion is a skin wound created by tangential trauma to the epidermis and dermis. The skin is forced against an abrasive surface in a rubbing fashion and the resultant injury resembles a thermal burn. The goals of managing an abrasion include the prevention of infection, promotion of healing, and prevention of "tattooing" with retained foreign bodies. Large and heavily contaminated abrasions are best managed in the Operating Room. The volume of local anesthetic required to achieve anesthesia would likely exceed toxic limits.

Prepare the wound. Anesthesia may be required prior to wound management. EMLA cream, which contains lidocaine and prilocaine, produces anesthesia of the intact skin but must be in place for about 60 minutes to provide significant benefit.[94] Perform a field block or regional nerve block (Chapter 156) as appropriate. Large abrasions may be anesthetized by applying 5% lidocaine gel topically for 5 to 10 minutes. Remove any dirt or debris using a sterile scrub brush and surgical soap or saline. Consider radiographic imaging to evaluate for potential foreign bodies.[40] Use the tip of a #11 scalpel blade to remove deeply embedded and larger particles from the wound. Apply petroleum jelly or antibiotic ointment to remove embedded tar.[95] Apply an antibacterial ointment to the wound and a dressing.

Instruct the patient to cleanse the wound three to four times a day. Cover the wound with petrolatum gauze and sterile gauze. Instruct the patient on how to properly cleanse the wound and reapply the bandage. Provide the patient with wound care supplies prior to discharge from the Emergency Department.

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120 Subcutaneous Foreign Body Identification and Removal

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INTRODUCTION

Wounds with a retained foreign body (FB) are a frequent presenting complaint to Emergency Departments. It is important to identify and remove debris and FBs to promote optimal healing. **The presence of a FB may not be obvious.** Up to 38% of embedded objects are missed on the initial assessment and many of these are frequently missed because imaging was not performed at that time.[1] **The presence of an unrecognized FB can lead to infection, joint injury, loss of function, osteomyelitis, pain, tendon rupture, tenosynovitis, and vascular injury.**[2-7] Chronic FBs may lead to the formation of pyogenic granulomas.[8] Certain soft tissue FBs have the capacity to migrate great distances months to years after the original injury, leading to increased morbidity and mortality from what originally was a benign FB.[9] **Assess patients presenting with chronic, recurrent, or delayed skin infections for the presence of an unrecognized FB. Failure to diagnose and treat a FB is a common cause of litigation against Emergency Physicians. A high index of suspicion and careful methodical examination, including appropriate imaging, must be undertaken to identify a FB.**

It is important to be familiar with the characteristics of different types of FBs because they may have various pathologic consequences. This information is crucial in determining the urgency or necessity for removal, the imaging techniques required to identify the object, the approach to removal, and determination of whether specialty referral is needed. **The successful removal of a FB requires a directed history and physical examination, appropriate imaging, adequate light, anesthesia, exposure, hemostasis, proper equipment, and patient cooperation. The removal of FBs from subcutaneous tissue can be a frustrating and time-consuming endeavor. The Emergency Physician will need to dedicate a reasonable uninterrupted time to attempt removal, provide wound care, and assure appropriate follow-up.**

It is important to note that a new type of self-injury known as self-embedding behavior has recently been described in adults and adolescents.[10] It involves the self-insertion of a FB into soft tissues. This behavior is relatively rare and is usually not a suicide attempt. Patients who self-embed most often have behavioral health diagnoses. This behavior indicates a much higher risk for this population to commit accidental or intentional suicide.[11] **It is the responsibility**

of the Emergency Physician to treat the injury and to provide appropriate referral for prompt psychiatric evaluation.

ANATOMY AND PATHOPHYSIOLOGY

Only a small percentage of wounds contain concealed FBs.[12] The mechanism of injury may give some idea of the likelihood of a retained object.[1] Crush wounds and puncture wounds, especially those involving the sole of the foot, as well as wounds deeper than 5 mm involving adipose tissue, are associated with a higher incidence of FBs that are often difficult to find.[12] Motor vehicle accidents have a higher incidence of retained FBs compared to other injuries.[13] Wounds caused by objects that shatter, splinter, or break in the process have a higher risk of a retained FB.[9] **Glass is the most commonly found retained FB.**[9,14] Lip or facial lacerations associated with dental fractures must be explored for pieces of teeth. Thorns, spines, or slivers tend to penetrate deeply and break. Broken-off needles are common FBs in injection drug users. Objects greater than 4.5 mm in diameter that penetrate the skin may push fragments of epidermis deep into the wound, producing an epidermal inclusion cyst, which can act as a FB.[15]

Depending on the type of material retained and the physical form of the foreign object, excess inflammation may delay healing or destroy surrounding soft tissues. Wood, thorns, spines, and other organic FBs are considered highly inflammatory and can lead to chronic granulomatous reactions, osteolytic lesions, periosteal reactions, or severe infections (e.g., necrotizing fasciitis).[15-17] The presence of soil in wounds markedly lowers the concentration of bacteria required to cause an infection by its interaction and interference with white blood cells.[18] Wounds tend to be resistant to antibiotics when a FB is present and it may be impossible to eradicate the infection until the FB is removed.[2,3,19] Glass, metal, and plastic are relatively inert materials.[20] Metals that oxidize may cause mild to moderate inflammation. Some retained FBs (e.g., lead) have the potential to produce systemic effects when in contact with pleural, peritoneal, cerebrospinal, or joint fluid.[18] Retained FBs that are not dissolved or extruded by the body's defenses become encapsulated, after which the inflammation will subside.[21]

HISTORICAL AND PHYSICAL ASSESSMENT

Patients presenting after an injury require a focused history including the details of the incident, the wounding agent, and the mechanism of injury. This information may suggest the presence of a retained FB and direct which imaging study is required.[22,23] Patients presenting with a FB sensation are more likely to have FBs identified on exploration or with imaging. **Using the patient complaint as a sole determinant would miss most FBs.**[13] Accurate diagnosis may be made more challenging. A history of skin puncture is often not recognized or reported by the patient.[24] Historical features (e.g., diabetes, immunosuppression, lymphedema, peripheral vascular disease, or renal failure) may cause difficult wound management and healing.[25] Assess past anesthetic history and the potential for aspiration if procedural sedation is to be considered.[26] Review the patient's medications, allergies, and tetanus immunization status. Provide booster doses and/or tetanus immunoglobulin as indicated.

A directed physical examination should begin with a brief inspection and documentation of the distal neurovascular status and function. Consider vascular injuries and address them before continuing with the rest of the examination. An injured extremity must be carefully examined through a full range of motion to ensure the integrity of the tendons. Discoloration of the skin may suggest a FB.[15] Palpation may reveal superficial FBs. Sharp localized pain with palpation over a puncture wound may suggest a retained FB. Adequate anesthesia, lighting, and good hemostasis are required to allow a thorough examination of the wound. **The examiner must avoid probing only superficially since the subcutaneous tissue can reapproximate and give the appearance of a superficial wound**. Extend the wound edges with a scalpel to facilitate inspection if there is concern regarding a retained FB and direct visualization is difficult.

A retained FB can be ruled out with a negative predictive value of 96% for wounds less than 5 mm deep if the bottom of the wound is visible.[12] **Never insert a gloved finger to probe the wound cavity. This can result in injury from sharp FBs.** Gentle blind probing with a hemostat is an acceptable and preferred alternative. A grating sensation that can be appreciated by the examiner is produced if the probe strikes a metallic or glass FB. **Avoid blind grasping within a wound with a hemostat**. Direct visualization is preferable when examining wounds of the face, feet, or hands.

RADIOLOGIC ASSESSMENT

Imaging is indicated in most cases where a retained FB is suspected but not found during wound exploration, when thorough exploration of the entire wound cavity is not possible, if the mechanism suggests a higher risk for retained FB, or if the patient feels that there is a retained FB. An Emergency Physician should have a low threshold for using appropriate diagnostic imaging while understanding the limitations of each of these tools (**Table 120-1**).

PLAIN RADIOGRAPHY

Most FBs missed during the initial clinical examination can be seen on plain radiographs.[16,27] Plain radiographs are readily available. Some authors suggest radiographic evaluation of nearly any penetrating wound involving an extremity.[1,4,28] Digital radiography has been proven superior to plain film radiography for the detection of subcutaneous FBs.[29] Perform standard anteroposterior and lateral radiographs using an underpenetrated "soft tissue technique" to increase the contrast between the FB and the surrounding tissue.[3] Oblique and other views can be added to avoid superimposition of the object over bony structures. Visibility of foreign material in soft tissues is dependent upon its composition, configuration, orientation, relative density, and size.[30,31] Radiopacity should not be considered an all-or-nothing concept. The radiographic appearance of a FB depends on its characteristics and the characteristics of the surrounding tissues. Certain FBs may be visible radiographically in one part of the body but not another.[32] The terms "radiopaque" and "radiolucent" will be used to describe the characteristic radiographic appearance when visualized within the subcutaneous tissues. Aluminum, bone, certain plastics, gravel, metal, pencil graphite, sand, and teeth are routinely visible on plain radiographs.[3,31,33] **Glass fragments were once thought to require lead or heavy metal to be visible on radiographs. Glass fragments as small as 0.5 mm appear on two-view plain radiographs if not obscured by bone; fragments of glass as small as 2 mm appear in the presence of overlying bone regardless of lead content.**[34,35]

TABLE 120-1 Comparison of Radiology Tests of Foreign Bodies

FB	Plain Radiograph	US Scan	CT Scan	MRI Scan
Glass	Good	Good	Good	Good
Metal	Good	Good	Good	Poor due to interference
Organic material	Poor	Good	Good	Good
Plastic	Moderate	Good	Good	Good
Wood	Poor	Good	Good	Good

Organic materials and plastics are not reliably detected on plain radiographs. They may be indirectly shown as a radiolucent filling defect when the object is less dense than the surrounding tissue, making plain radiography worthwhile even in cases of suspected nonradiopaque FBs.[36,37] This allows consideration of radiographs while understanding this imaging modality's poor overall sensitivity in these situations.

Examine the entire radiograph for the appearance and location of any FBs.[38] Other imaging modalities are required if there is a strong suspicion of a retained FB that usually does not show up on plain radiographs.

The advantages of plain radiographs include their universal availability, low cost, and familiarity to most Emergency Physicians. The disadvantages include the inability to resolve objects with densities similar to body tissues and poor sensitivity for detecting organic matter. Plain films do not demonstrate anatomic structures that may be intervening between the skin and FB along the planned surgical approach. It may be difficult to accurately judge the depth of a FB using two-dimensional radiographs.[15] Standard radiographs remain a clinically practical screening modality for FBs despite these drawbacks.[30]

ULTRASOUND (US)

Radiography alone is not sufficient to identify a nonradiopaque FB. Bedside US should be the next diagnostic tool to be considered. US can help determine the depth, size, shape, and relationship of the FB to nearby structures.[39,40] These factors may influence the timing and technique of attempted extraction while potentially preventing the patient from being exposed to additional radiation.[41] The lack of radiation and its ease of use dictates an assessment with bedside US as a screening tool when FBs are suspected or not found on plain radiography.

Studies have shown ultrasonography to have a higher sensitivity for radiolucent FBs than plain radiography.[42,43] **High-resolution real-time US using a 7.5 MHz or greater linear array transducer can detect radiolucent superficial FBs with a similar radiographic density as the surrounding tissue.** It should be the first choice in the imaging of suspected radiolucent FBs.[15,44-48] This includes fish bones, sea urchin spines, small glass fragments, vegetative material, and wood.[15,48-52] Wood FBs can cause severe inflammatory reactions and act as a source for infection. They should be evaluated with US in patients in whom radiographs are negative.[49] This is especially important in the Emergency Department where wood FBs make up as much as 34% of all FBs and plain radiographs have a sensitivity as low as 7.4% for organic material.[50] One study showed that the accuracy of detection of radiolucent objects was above 80% when performed by US Technologists, Radiologists, and Emergency Physicians.[52]

US can localize a FB within three dimensions. This can help to establish its relation to adjacent bone, muscle, tendons, and tendon sheaths.[46] Preoperative US results in less damage to the surrounding tissues, reduced operative time, and reduced postoperative morbidity.[46,53]

The procedure for locating a subcutaneous FB with US begins with selecting the highest available transducer frequency.[54] Higher frequency transducers have better resolution for superficial structures. The use of a standoff pad or gel-filled glove with the air and talc carefully removed can act as a dead zone for objects that would normally be too superficial to fall within the focal zone of the transducer (**Figure 120-1*A***).[55-57] The water-basin technique can be used in a similar manner. Place the wounded extremity in a basin of water and align the transducer above the skin (**Figure 120-1*B***).[58] Adjust the focus to the required depth and proceed to scan the field in two orthogonal planes.[54] Refer to Chapter 121 for more details regarding US-guided FB identification and removal.

The US appearance of FBs is related to the surface characteristics and not its composition.[59,60] Metals and needles are often hyperechoic with reverberation artifact or comet tails. Gravel is usually hyperechoic and shows dense posterior acoustic shadowing. Organic materials have a characteristic appearance and are ideal for US evaluation. Wood is most often hyperechoic with a posterior acoustic shadow (**Figure 120-2**). Wood FBs lose echogenicity over time.[61] Furthermore, FBs are often surrounded by a hypoechoic halo (**Figures 120-3 and 120-4**).[35,37,51] This frequently represents a zone of edema, granulation tissue, or abscess formation.[52] The hypoechoic halo can represent an inflammatory response and may not be visible for the first 24 hours.[49] Doppler US may reveal a hypoechoic or black ring around a hyperechoic or white FB. Doppler shows marked hyperemia around the periphery of a FB which is considered a reliable secondary sign.[62] It has been hypothesized that injection of saline into the interstitial space around a suspected FB may make bedside US more sensitive. A cadaveric study failed to show a significant improvement.[63]

A

B

FIGURE 120-1. US identification of superficial FBs. ***A.*** A glove used as a stand-off pad. (Used with permission from reference 57.) ***B.*** A water bath. (Used with permission from reference 58.)

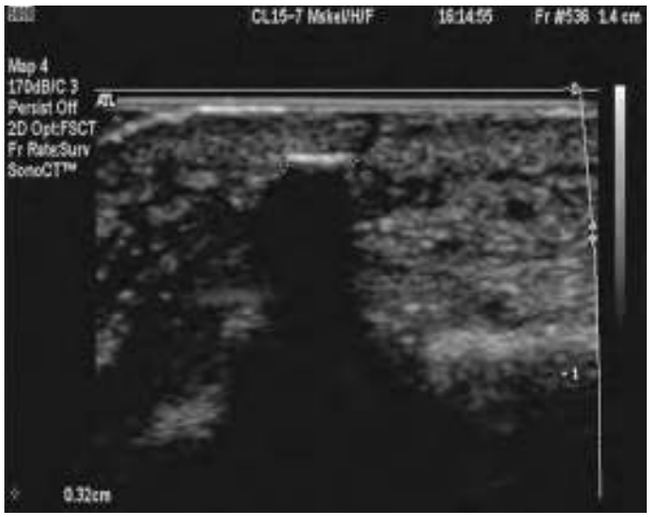

FIGURE 120-2. An US image of a subcutaneous thorn. The FB is hyperechoic (*white*) with an acoustic shadow deep to it.

Mark both ends of the FB on the patient once a FB has been located on US.[64] This can be done by inserting paper clips between the skin and the transducer and connecting both ends with a marked dotted line. **Meticulously scan along the entire length of the suspected FB and visualize the FB perpendicular to the transducer.**

There are several disadvantages when using US, including the level of skill required for its use in diagnosing musculoskeletal FBs.[53-56,58-65] Studies assessing the accuracy of US have shown a sensitivity of 30% to 100% and a specificity of 70% to 90%.[66] These values have been mostly demonstrated in referred patients studied by Radiologists and certified technicians.[67,68] US performed by Emergency Physicians has a limited specificity ranging from 47% to 59% under conditions that replicate a typical Emergency Department situation.[68-70] Small wood FBs 1 cm or less in size may fail to be identified by bedside US, further limiting its utility.[66] Another disadvantage is that false positives can occur with air in wounds, calcifications, fresh bleeding, keratin plugs, old scar tissue, ossified cartilage, sesamoid bones, sutures, and tendons.[15,49,56] Prior exploration for FBs will place patients at higher risk of having false-positive findings from scar tissue.[71] US localization may be difficult if the object is close to bone or deep to subcutaneous gas.[59] **A high suspicion for a retained subcutaneous FB not identified by bedside US should follow with a request for a formal diagnostic imaging study.**

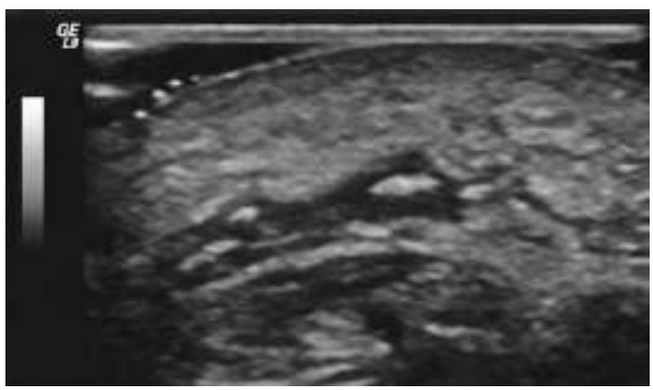

FIGURE 120-3. An US image of several shards of glass in the palm. Note the hyperechoic (*white*) FBs surrounded by an echo-poor (*black*) signal consistent with either a hematoma or edema.

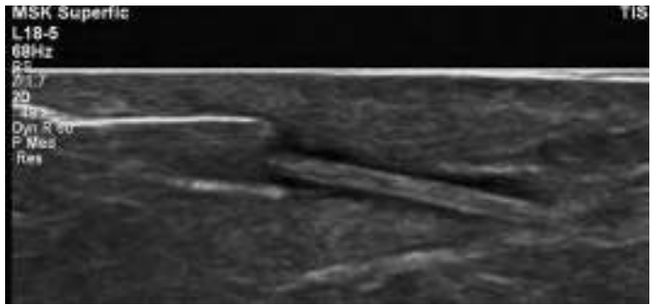

FIGURE 120-4. US-guided removal of a wooden splinter using crocodile forceps. The echogenic jaws of the forceps are seen grasping the FB. The hypoechoic "halo" around the splinter is demonstrated. (Image courtesy of Dr. Mike Bradley.)

COMPUTED TOMOGRAPHY (CT) SCAN

Consider CT imaging in cases where radiography and US have failed to demonstrate suspected FBs (e.g., radiolucent material or objects that cannot technically be located due to overlying bone or gas).[72-75] CT is sensitive in differentiating densities and can detect more types of FBs.[22,76-79] It is helpful for high-risk wounds when there is potential for infection or joint involvement. CT images can be created in multiple planes and can demonstrate the relationship of a FB to anatomic structures.[76,80] Three-dimensional reconstruction images are accurate in identification of complex FBs and their associated complications.[81] CT-guided percutaneous placement of a catheter or needle can guide surgical dissection to a FB and aid in the subsequent removal.[76,80,81] The disadvantages of CT scanning include its higher cost, radiation dosage, availability, and the need for patient cooperation. Organic FBs become less visible over time due to absorption of body fluids.[42]

MAGNETIC RESONANCE IMAGING (MRI)

MRI imaging may be considered for chronic or complicated FBs.[41] MRI appears to be more accurate than any other modality for identifying wood, glass, plastic, spines, and thorns.[22,76] It is superior to CT scanning in detecting the presence and extent of edema, hemorrhage, or infection surrounding FBs.[22,76] MRI's other advantages include the high resolution and contrast between adjacent tissues. It allows the precise localization of FBs in three dimensions to aid in surgical planning and assessment of the need for advanced exploration, debridement, and irrigation. MRI does not expose the patient to radiation.[82] Magnetic resonance angiography (MRA) is the diagnostic modality of choice when there is clinical suspicion of a FB associated with vascular injury.[41]

The disadvantages of MRI include its lack of availability and high cost. Patients must be assessed to rule out a magnetic FB due to the risk of movement of the FB during MRI scanning.[83] Prior history of an ocular or other metallic FBs must be sought prior to MRI scanning to prevent iatrogenic injury. This requires a thorough screening history and plain radiographs prior to MRI scanning. Gravel and ferromagnetic substances produce significant artifact so that differentiating scar tissue, tendons, and calcifications from an actual FB can be challenging.[22,84]

INDICATIONS

Make every effort to identify FBs, even if they are not likely to be removed. It must be decided whether an identified FB needs to be removed immediately, electively, or if at all (**Table 120-2**). Factors that influence the decision to proceed with attempted removal include the size and reactivity of the FB, its proximity to vital

TABLE 120-2 The Indications for Removal of a FB

Allergic reaction
Chemically reactive
Contaminated wounds (e.g., feces, saliva, or soil)
Cosmesis
Heavy metals
Impairs gait
Impinging on structures (e.g., nerves, tendons, or vessels)
Infection in area
Intraarticular
Intravascular
Migration potential
Persistent pain
Proximity to fractured bone
Psychological stress of patient
Vegetative material
Venomous spines

structures, and associated injuries. These must be weighed against the potential for further tissue damage and contaminating the wound. An extraction attempt will depend on the amount of time an Emergency Physician can safely allocate while providing appropriate care to the other patients in the department. Superficial FBs in adults can be removed using local anesthesia. Pediatric FBs may be removed in the Emergency Department under procedural sedation or in the Operating Room under general anesthesia. Consider delaying the removal of deep-seated FBs in adults as the procedure will be more successful in the Operating Room.[85]

A FB requires urgent removal if it is likely to provoke significant tissue inflammation or injury.[3,86,87] Immediately remove contaminated objects (e.g., FBs located in the presence of an established infection, soil, and teeth).[3,18] Immediately remove allergenic FBs, those causing hemorrhage, those causing ischemia, or toxic FBs.[3,87] Remove FBs having the potential for migration, interfering with motor function, or interfering with sensory function.[3] FBs in the hands and feet usually require removal as they may cause persistent pain and have the potential to sever nerves or tendons.[5] The FB may require removal for cosmetic or psychological reasons.[1]

CONTRAINDICATIONS

A FB should be urgently removed under ideal operative conditions by an appropriate Surgeon if it is associated with a neurovascular injury or is located near blood vessels, nerves, or tendons.[1,3] Avoid deep exploration of the hands and feet to prevent injuring intricate structures. Large, deep, and impaled FBs are assumed to help tamponade an active hemorrhage and should be left in place until removed in the Operating Room.[38] FBs associated with fractures or located within a joint require prompt surgical debridement to prevent osteomyelitis or septic arthritis.[87] Urgent surgical intervention is required for all high-pressure injection injuries. These injuries may initially appear innocuous but often cause extensive damage and carry a significantly high risk of complications.[87] Consider referral based on experience, anticipated problems related to the FB's location or depth of penetration, the duration of retention, or other patient factors likely to complicate the procedure or follow-up.

A deeply embedded, inert object not near any vital structure can be left in place. The difficulty of removal is usually not worth the potential tissue damage. These patients can be referred for elective removal if necessary.[1,87] **The patient must be informed and possible complications must be discussed if the decision is made not to remove a FB. This includes migration of the FB and infection. Document this discussion in the medical record.**

EQUIPMENT

- 18 gauge needles
- 27 gauge needles
- 10 mL syringes
- Povidone iodine or chlorhexidine solution
- Lidocaine with and without epinephrine
- Topical 4% liposomal lidocaine
- Depilatory wax, rubber cement, or hardening facial gel
- #11 scalpel blade on a handle
- #15 scalpel blade on a handle
- Nylon suture, 1–0 or 2–0
- Methylene blue
- Paper clips
- Wire grid
- Eye magnet
- Hemoclips
- Hemoclip applier
- Fluoroscopy unit
- Hemostats, two sizes
- Forceps, Crocodile and Mosquito
- Sharp scissors
- 22 gauge needles for localization
- Magnification eye loupes
- Normal saline
- 19 gauge blunt-tip needle or angiocatheter
- 35 mL syringe
- Blood pressure cuff or Penrose drains
- 4×4 gauze squares
- Adhesive tape
- Bedside US with high-frequency transducer
- Standoff pad, gel-filled glove, or small basin of water

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. FB removal can be frustrating and time consuming, even when performed under ideal conditions. It is appropriate to set a time limit that is reasonable based on the staffing and volume of the Emergency Department. Inform the patient of the planned time limit at the outset. The search should be discontinued and the patient referred to a Surgeon after expiration of the predetermined time.

Apply povidone iodine or chlorhexidine to the skin site surrounding the wound. Avoid spilling the solution into the wound cavity as both are toxic to wound defenses and may increase the incidence of a subsequent wound infection.[19] Obtain a bloodless field to adequately examine a wound for a FB. Place a blood pressure cuff proximal to the injury. Elevate the extremity for at least 1 minute and then inflate the cuff to a pressure greater than the patient's systolic blood pressure. This is safe for up to 2 hours but will cause some discomfort.[88] A local vasoconstrictor can be used with the anesthetic to control localized capillary bleeding if no contraindications exist. A 0.25 to 0.50 inch Penrose drain can be wrapped tightly around a finger or toe and secured in place with a hemostat.

An alternative method for achieving hemostasis of a finger involves cutting out a "finger" from a glove. A cut is made across the tip of the "finger." Place the cut finger from the glove on the affected finger. Roll the latex finger from distal to proximal to create a "ring tourniquet" at the base of the proximal phalanx. Consider using Trendelenburg position for removing FBs in the foot. Elevation above the level of the heart can easily be achieved by placing the patient in a steep Trendelenburg position. This position can vastly reduce bleeding at the site of incision without the use of a tourniquet.[89]

Adequate anesthesia is crucial to the procedure. A field block or regional nerve block (Chapter 156) does not distort the wound.[90] Consider using topical anesthetics (Chapter 154) in the pediatric population. Liposomal lidocaine has a very fast onset and is associated with higher success rates, reduced procedure time, and less pain.[91,92] Procedural sedation (Chapter 159) or general anesthesia may be required if cooperation is not possible.

TECHNIQUES

SUPERFICIAL WOOD OR ORGANIC SPLINTER REMOVAL

Very fine FBs can be difficult to visualize. One method to help localize them is to spread soft soap very lightly over the skin.[93] Only superficial organic splinters and FBs should be pulled out with forceps as they often come apart and leave a fragment that is more difficult to remove.[3,94] Cactus spines or wood splinters occasionally lie superficial and parallel to the skin surface. Make an incision parallel to the long axis of the FB and then lift it out of the wound. Enlarge the skin entrance wound with a scalpel so that the FB can be grasped and withdrawn with a hemostat under direct visualization if it is lodged in the subcutaneous tissue. The splinter can be removed by picking it out with an 18 gauge needle, using light feathering strokes to de-roof the skin over the splinter. Subungual splinters may be removed using a "V-cut," in which sharp scissors are used to remove a V-shaped piece of nail to visualize the underlying splinter and remove it using forceps (Chapter 128). **Caution must be exercised not to disturb the nail matrix for more proximal subungual splinters because this may result in failure of the nail to grow back normally.[20]**

Small cactus spines may be difficult to locate and remove directly. Application of a depilatory wax, rubber cement, or a water-soluble facial gel with a brush can successfully aid in the removal of small fine spines.[86] Apply the wax, rubber cement, or facial gel over the skin containing the protruding spines. Apply a layer of gauze over the wet substance. Allow the substance to dry onto the skin and the gauze. Remove the gauze to lift off the dried substance and attached spines. Repeat this process as required to remove the remaining spines.

PUNCTURE WOUNDS

Puncture wounds of the foot are commonly seen in Emergency Departments. Punctures not infrequently drive pieces of clothing, shoes, or other debris deep into the wound and result in an infection rate of approximately 10%.[95-97] Puncture wounds in the distal foot may profit from debridement and irrigation.

Puncture wounds can be trimmed or ellipsed to remove contaminated and/or embedded FBs (**Figure 120-5**).[3] This technique works well for FBs that are difficult to localize. Make an ellipse in the skin surrounding the FB with a #15 scalpel blade (**Figure 120-5*A***). Lift the ellipse of skin and separate it from the underlying dermis (**Figure 120-5*B***). The FB may be visible. Grasp it with hemostats or forceps and remove it from the tissue.

The FB and the ellipsed puncture wound can both be extracted in a block of tissue if unable to exactly localize the object (**Figure 120-5*B*, dotted line**).[90] **First ensure that no blood vessels, nerves, or tendons will be injured**. Alternatively, make the skin ellipse and extract the block of tissue containing the FB without dissecting the skin from the subcutaneous tissue (**Figure 120-5*C***). Both techniques allow for the removal of FBs and better cleaning of the wound. Puncture wounds of the foot require careful follow-up due to the risk of infection.

There are several alternative techniques if removing an ellipse of skin and/or tissue is contraindicated or if the Emergency Physician is not comfortable with this technique (**Figures 120-6 and 120-7**). A FB may be embedded perpendicular to the skin (**Figure 120-6*A***). Make a linear incision that passes 1 mm to the side of the puncture wound with a #15 scalpel blade (**Figures 120-6*A* and 120-6*B***). Spread the incision open. Visualize the FB (**Figure 120-6*C***), grasp it with a hemostat, and remove it.

Another technique involves making a superficial skin incision and manually expressing the FB (**Figure 120-7**). Make an elliptical incision surrounding the FB or a linear incision over the FB (**Figure 120-7*A***). Remove the ellipse of skin or spread the linear incision. Undermine the subcutaneous tissues surrounding the FB (**Figure 120-7*B***). Apply digital pressure over the undermined areas to displace the FB into the center of the wound and upward (**Figure 120-7*B***). Grasp the FB with a hemostat and remove it.

GEOMETRIC APPROACH FOR A NEEDLE IN THE FOOT

A technique that is useful for the removal of a needle in the plantar surface of the foot involves the use of standard anterior, posterior, and lateral radiographs to identify the cutaneous site corresponding to the location of the needle.[98] The incision site is determined by bisecting the midpoint of the needle, as seen in each projection, by a line drawn at right angles to the long axis of the needle. The ideal plane of dissection is perpendicular to the needle's midpoint, which is correlated with the surface anatomy of the foot.

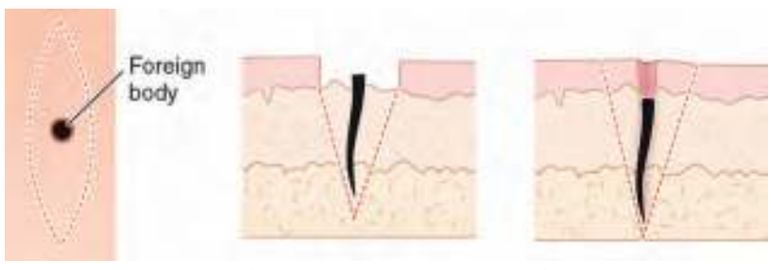

FIGURE 120-5. Creating an ellipse to remove a contaminated or embedded FB. ***A.*** An ellipse is made in the skin surrounding the FB. ***B.*** The epidermis is removed to expose the FB. A block of subcutaneous tissue (*dotted line*) can be removed if the FB is difficult to visualize. ***C.*** The skin, the subcutaneous tissue, and the FB can be removed en bloc.

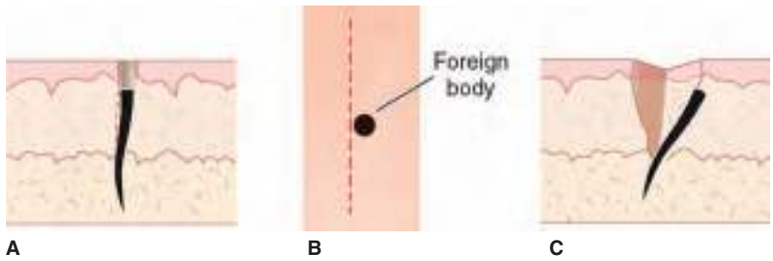

FIGURE 120-6. Removal of a FB embedded perpendicular to the skin. ***A.*** The embedded FB. The *dotted line* represents the proposed incision line. ***B.*** A linear incision is made 1 mm lateral to the FB. ***C.*** The incision is spread open to visualize and remove the FB.

Prepare and anesthetize the skin after determining the dissection plane. Make a 0.5 to 1.0 cm skin incision in the plane perpendicular to the needle in its midpoint. Advance an iris scissors into the incision and along the dissection plane with the blades slightly open. Advance the scissors 1 to 2 mm and close the blades. Withdraw the scissors slightly. Open the blades of the scissors, advance them 1 to 2 mm, and close the blades. **Care must be taken to avoid cutting the flexor tendons, although they are located deep and in close association to the bones of the foot**. Continue this process of advancing and closing the blades until the needle prevents closure of the scissors. Advance a hemostat into the wound and over the blades of the scissors. Grasp and secure the needle. Pull the hemostat out of the wound to simply back the needle out of its entry tract. This procedure is reported to have a 100% success rate and takes approximately 10 minutes.[98]

DYE TECHNIQUE FOR LOCALIZING A NEEDLE

Methylene blue may be used to track the location of a FB.[99] After identification of a FB by plain radiography or US, clean and prepare the skin. Sterilely inject 0.1 to 0.2 mL of methylene blue very gently through the entrance wound of the FB. The dye will travel along the path of least resistance, that is, the path of the FB. Make an A-, U-, V-, or Y-shaped incision from the point of entry and raise a flap of tissue. The blue dot of dye may serve as a guide to the location of the FB. This procedure is often complicated by seepage of the dye and is of limited value.

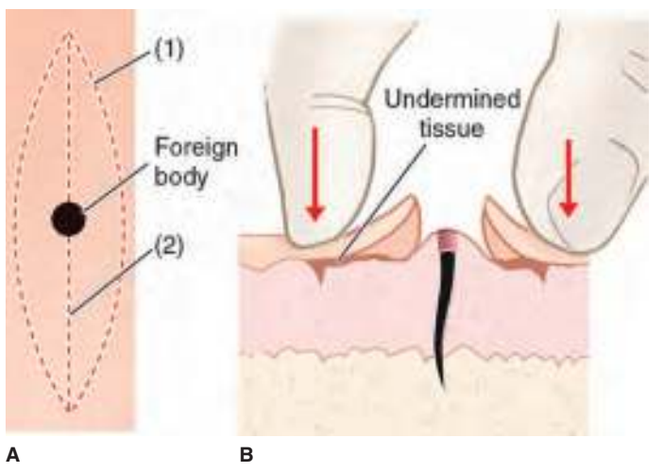

FIGURE 120-7. An alternative technique to remove a FB from a puncture wound. ***A.*** Incise and remove an ellipse of epidermis (1). Alternatively, make a linear incision centered over the FB (2). ***B.*** Undermine the subcutaneous tissue surrounding the FB. Apply digital pressure (*arrows*) to visualize and express the FB.

NYLON SUTURE TECHNIQUE

One technique described for use in fresh wounds involves the localization of a FB by marking its entry tract with a nylon suture.[100] Grasp a piece of 1–0 or 2–0 nylon suture between the thumb and index finger. Rotate the suture while pushing it into the wound so that it follows the tract made by the FB. It is reported that the FB is easily felt when the nylon contacts the FB.[101] Leave the suture in the wound tract. Open the wound tract by cutting alongside the nylon suture until the FB is reached, at which time it is removed. A 92% success rate up to 48 hours after the time of injury has been reported with this technique.[100]

PAPER CLIP X-RAY LOCALIZATION

Simple paper clips may be used to locate a FB (**Figure 120-8**).[101,102] Obtain plain radiographs to demonstrate the presence of a radiopaque FB. Bend two or more paper clips into different shapes. Place the ends over the wound entry site. Secure the paper clips in position with tape. Obtain two plain radiographs or an US taken at right angles to each other. Examine the radiographs or US to determine the cutaneous location of the FB in relation to the paper clips and note its depth from the skin surface. Mark the exact cutaneous location of the FB on the patient's skin with a permanent ink pen. Remove the paper clips.

Clean, prepare, and anesthetize the skin. Make a stab incision at a 90° angle to the middle of the FB, taking the shortest distance between the skin and the FB and following the method discussed for the geometric approach for a needle in the foot. Insert a small hemostat into the incision. Advance the hemostat allowing localization of the object with minimal probing. Grasp and remove the FB.

TAGGED HEMOCLIPS

Even with the most elaborate marking system using grids or needles, it can be difficult to find FBs once the dissection begins since the tissues may be distorted by retraction, edema, or local anesthesia. The tagged Hemoclip method was developed to address this problem.[103] It requires a skin incision and dissection down to where the Emergency Physician believes the FB to be based on plain radiographs. Prepare two or three Hemoclips with a long silk suture attached to each one. Place them into the Hemoclip applier. Dissect down to where the FB is believed to be located. Place two or three Hemoclips into the depths of the wound. Obtain repeat radiographs to show the relationship of the FB to the Hemoclips. Remove all but the closest Hemoclip. Dissect toward the Hemoclip and the FB. Identify and remove the FB. Remove the Hemoclip. Repeat the procedure after placing two or three additional Hemoclips if unable to exactly localize the object. Follow the trail of Hemoclips to the FB.

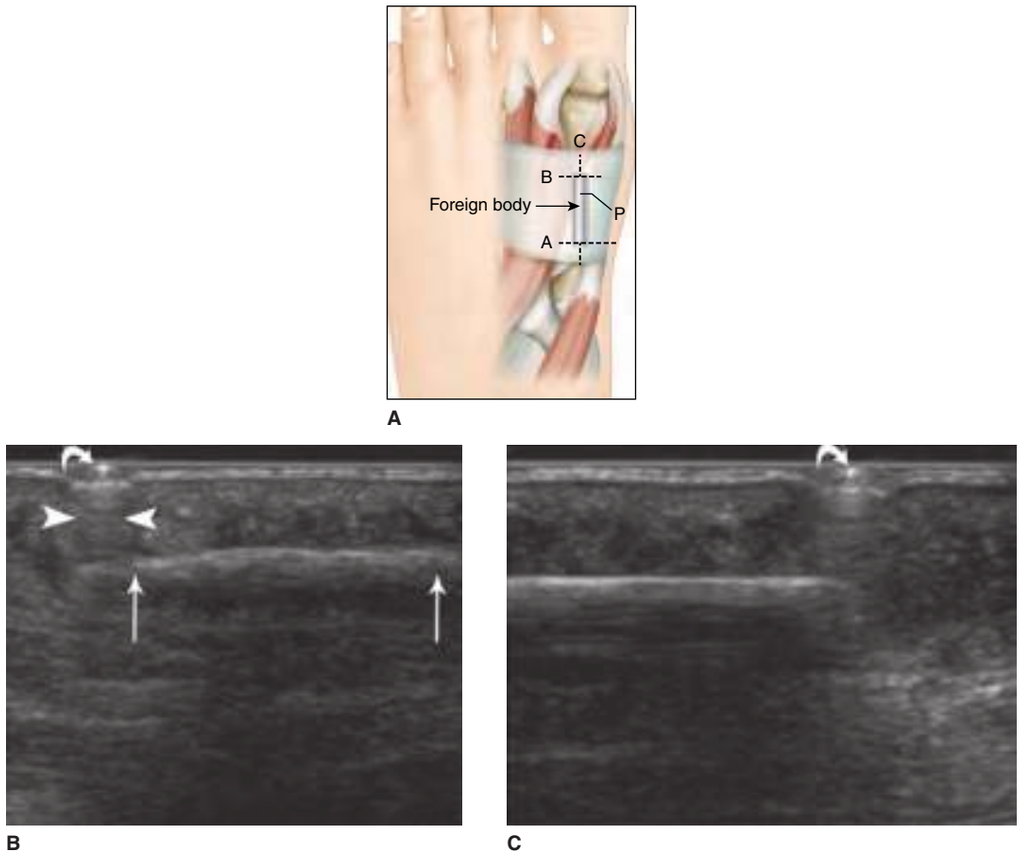

FIGURE 120-8. Paper clip localization of a FB. ***A.*** The transducer (P) is placed over the FB. (A = proximal end of FB, B = distal end of FB). ***B.*** US of the FB (*arrows*). The paper clip (*curved arrow*) is seen at the proximal end of the FB with a comet tail artifact (*arrowheads*). ***C.*** US through the distal end of the FB. The paper clip is again seen (*curved arrow*). (Used with permission from Creel SA, Girish G, Jamadar DA, et al: Sonographic surface localization of subcutaneous foreign bodies and masses. *J Clin Ultrasound* 2009; 37:158–160.)

NEEDLE GRID LOCALIZATION

The grid principle may be used to remove superficial and nonlinear radiopaque FBs after they have been identified on plain radiographs. Clean, prep, and anesthetize the skin. Insert three 25 gauge, 1.5 inch needles into the skin near the estimated location of the FB and at right angles to each other. Obtain two orthogonal plain radiographs. This process can be repeated, each time moving the needles slightly until one is superimposed on the FB. Determine the cutaneous position of the FB by noting its position within the needle grid. Mark this position on the skin. It must be remembered that this technique does not provide a true three-dimensional image since divergence and parallax distortion of images occur on the radiographs.[3]

The Emergency Physician must recall that tendons and other structures may block the planned path to the object.[15] Make a 0.5 to 1.0 cm incision in a plane perpendicular to the long axis of the FB to an appropriate depth or dissect along the path of the closest needle.[38] Identify and remove the FB. A drawback of this technique is the potential for dislodging of the needles with attendant repeated trips to the Radiology Department. The use of fluoroscopy will prevent this problem.

CROSS-NEEDLE GUIDED TECHNIQUE

A cross-needle guided technique can be used if the FB is superficial. Precise identification of the superficial FB is necessary with this technique. Localization of a small FB requires meticulous insertion of the first needle. Apply topical local anesthetic directly over the FB site or perform a regional nerve block. Injecting local anesthesia in the tissue site before inserting the first needle may lead to tumescence and loss of the FB. Therefore, using topical anesthesia or a regional nerve block is recommended. Insert a 23 gauge needle tip into the skin 5 to 10 mm away from the FB site, advance the needle below the FB, and put upward and forward pressure on the needle until it emerges out the other side. Inject the surgical site with lidocaine. Insert a second 23 gauge needle in the same manner and below the FB perpendicular to the first needle to help localize the FB and propel it toward the surface of the skin. Use a #15 scalpel to bluntly dissect directly over the fixed FB and remove it.[104]

MAGNET REMOVAL

Another technique reported to have a good success rate in removing metallic FBs uses a hand-held magnet.[105,106] Confirm the presence of a suspected metallic FB using radiographs or US. Prepare, drape, and anesthetize the area. Slightly enlarge the entry wound to permit entrance of the magnet tip. Apply a sterile US transducer cover or glove over the magnet. Gently probe the wound track with the magnet until a "click" is appreciated. Withdraw the magnet with the FB attached to the magnet. Perform further and more directed wound exploration with the magnet if resistance is met. Magnets have been used to localize a metallic FB as the attraction will often create a characteristic "hilliness" in the soft tissue. This localization leads to smaller incisions and reduced scar tissue formation.[107]

FLUOROSCOPIC TECHNIQUES

Fluoroscopy offers an excellent aid in the removal of radiopaque FBs.[105] Make an incision over the FB as judged from plain

radiographs. Localize the FB under fluoroscopy. Guide a curved hemostat to the FB with brief intermittent exposures of the fluoroscopy unit.[108] Grasp and remove the FB. This procedure exposes the Emergency Physician's hands to radiation and may risk damaging structures in the wound by blind manipulation of the hemostat.[109]

The paper clip and needle localization techniques described previously can be applied under fluoroscopy. Rotating the site of injury under direct fluoroscopy may improve the ability to judge the location of the FB in three dimensions and aid in its removal. Intermittent exposure with a fluoroscope can allow repositioning of the needles until the FB is localized between two needles or at the tip of a single needle. Make a small incision carried down to the FB and remove it.[109,110]

A modified "paired" needle technique involves placing two pairs of needles under fluoroscopy. The initial needle is placed in between the c-arm and the FB so that the FB falls directly in the "shadow" of the needle. The second needle is then placed in the same line as the first, but distal to the FB. This process is repeated in a second plane perpendicular to the first view. The pairs of needles create two vectors, the intersection point of which will provide an accurate incision leading to precise dissection and removal.[111]

Additional techniques using fluoroscopy and neurosurgical stereotactic devices have been described.[3] The cost, availability, and practicality make these approaches unattractive in the Emergency Department and therefore, they are not discussed further.

ULTRASONOGRAPHIC REMOVAL

There are a multitude of different approaches to the US-guided removal of subcutaneous FBs.[51,54-56,64,112] A detailed working knowledge of the pertinent anatomy is essential. Locate and mark the location of the FB. Clean, prep, and anesthetize the skin. Insert a needle under US guidance. The US monitor screen will display a hyperechoic linear object with posterior reverberation effect. Guide the needle under US until it touches the FB. Make a skin incision down to the FB. The addition of a second needle in a perpendicular plane can enhance the object's localization.[55] Insert closed hemostats or forceps to approach the FB.[55,112] Insert the hemostat or forceps along the plane of the FB. Open the jaws of the instrument under US guidance (**Figure 120-3**). The FB can be removed under US guidance once within the grasp of the hemostat or forceps. **Always rescan the area after the FB is removed to ensure that no additional fragments remain.**

One of the challenges associated with this technique occurs when the FB is pushed away by the forceps. This difficulty may be avoided by using the US transducer or a free finger to apply pressure to "fix" the FB in one spot to facilitate its removal.[113,114] Certain materials (e.g., glass) may be difficult to grasp with the technique described above.

One adjunct to US-guided FB extraction is hydrodissection. This involves guiding a small needle under real-time US to the FB. Lidocaine is injected to dissect away the surrounding tissues. This allows for more precise visualization and removal of the FB.[10,109] A more detailed description of US-guided FB removal is available in Chapter 121.

AFTERCARE

It is often prudent to take postextraction radiographs to ensure complete removal of the FB, especially if multiple objects are involved or if there is concern about the object fragmenting.[115] Carefully irrigate and debride the wound of all epidermal fragments.[3] The most effective form of wound preparation and cleansing is jet lavage irrigation with normal saline to decrease the bacterial load in the wound and the risk of developing infection.[116] This can be performed with a commercially available device or a 35 mL syringe armed with a 19 gauge angiocatheter or blunt needle.[19] Studies of simple pediatric wounds have found that using drinkable tap water to irrigate wounds is a good alternative to saline, as it shows similarly low infection rates.[117] The recommended volume for irrigation is 50 to 100 mL/cm of wound, although gross contamination should be taken into account.[118] Soaking wounds is not recommended. Soaking may adversely affect wound repair and healing.[118] The quality of mechanical cleaning is important to wound prognosis.[1] Cleaned, thoroughly irrigated, and debrided wounds with a good blood supply do not require antibiotics and may be sutured closed after removal of the FB. Arrange follow-up in 5 to 7 days for suture removal.

Consider delayed primary closure or healing by secondary intention if complete cleansing of the wound is not assured or if the wound is at significant risk for infection for other reasons.[19,119] Delayed primary closure involves packing the wound open for 4 to 5 days. Reassess it and close it primarily if the edema has resolved, no infection is present, and exudate has been removed. This technique results in minimal tissue damage. It is especially useful in clean contaminated and contaminated wounds with a 90% success rate in appropriate patients.

Antibiotic therapy may be considered for those with wounds that are at significant risk for infection. **Antibiotics are not a replacement for proper wound care**. Infection rates for traumatic wounds range from 4.5% to 6.3%. High-risk wounds are bites, burns, wounds associated with cartilage injury, contaminated wounds (e.g., feces, saliva, or soil), crush injuries, wounds associated with frostbite, wounds associated with open fractures, puncture wounds, wounds associated with tendon injuries, and wounds in immunocompromised patients; these wounds should be considered for antibiotic therapy.[120,121] Close follow-up is especially important in these patients. Prophylaxis for endocarditis is not recommended unless FB removal is undertaken through an area of an established infection.[122] Splint the involved extremity if the FB is near a joint, a highly mobile region, or a vital structure to prevent injury and/or migration of the FB if the patient is referred for delayed removal.[15] A more complete discussion on wound cleansing, wound irrigation, and the general principles of wound management can be found in Chapter 114.

TETANUS PROPHYLAXIS

Tetanus prophylaxis must be considered for wounds involving FBs. Wounds that are avulsions, burns, contaminated (e.g., feces, saliva, or soil), crushing, frostbite, punctures, or resulting from missiles are considered tetanus prone. Risk factors such as age over 65 years, diabetes, and intravenous drug abuse place patients at higher risk to develop tetanus. Special attention should be placed on these patient groups presenting with minor wounds.[123] Studies have found that up to 26% of patients who reported they were up to date with tetanus vaccination were confirmed not to be, while a further 30% were indeterminate.[124] **It is vital to investigate a patient's tetanus status and vaccinate according to the accepted guidelines.**[54,125] For patients who have had three or more doses, those who received their previous dose within 5 to 10 years presenting with a nonclean and minor wound should receive the Tdap booster (preferred over the Td booster). Patients who have gone more than 10 years since their last dose should also receive the booster regardless of wound type. All patients in whom there have been fewer than three doses or an uncertain vaccination history should receive the vaccine booster. Of these patients, those with complicated or contaminated wounds should receive the tetanus immunoglobulin (TIG) in addition to the vaccine booster.[125] Emergency Physicians continue to fail to offer appropriate postexposure prophylaxis even in patients who able to describe their vaccination history accurately.[126] Refer to Chapter 114 for more complete details regarding tetanus prophylaxis.

COMPLICATIONS

The removal of embedded FBs is relatively free of complications if properly conceived. **Care must be taken to document the functional and neurovascular status prior to and after any significant manipulations**. Appropriate referral of complicated cases (e.g., FBs located deep in the hands, the feet, or near vital structures) will lessen the risk of unfavorable outcomes. Infection remains the most common complication of a retained FB, even when the object itself is not contaminated.[127] Aseptic technique and avoidance of excessively prolonged manipulation and searching for embedded FBs are important to prevent introducing infection into a previously sterile area. In cases where the FB cannot be found, it may be necessary, although less than ideal, to wait for abscess formation to pinpoint the location of an object.[3] Referral for additional imaging and removal may be appropriate. The patient must be advised in detail of the likely course, and follow-up must be assured and documented.

SUMMARY

Virtually all embedded subcutaneous FBs should be identified and located through rigorous assessment of every traumatic wound and by maintaining a high index of suspicion. The history and physical examination should guide the search for retained FBs and the approach to locating them. Most FBs are visible on plain radiographs. Wounds in which a radiopaque FB may be retained should be imaged with standard radiographs. Bedside US should be used to assist in the identification and removal of subcutaneous FBs. Additional diagnostic imaging with CT or MRI may be indicated if a FB is still suspected but not identified by radiographs or bedside US. Several techniques are available to aid in localization and removal of the FB. A decision must be made regarding the necessity of immediate or urgent removal in the Emergency Department or by referral to a specialist. Inert FBs that are unlikely to cause long-term complications may be left in situ. Provide verbal and written information to the patient who explains the reasoning behind this conservative approach if a FB is left in the tissue. Appropriate wound care is crucial to satisfactory healing. Postprocedure follow-up must be addressed.

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121 Ultrasound-Guided Foreign Body Identification and Removal

Daniel S. Morrison and Chirag N. Shah

INTRODUCTION

Retained foreign bodies are associated with up to 1.9% of wounds.[1] **The presence of a foreign body in a wound increases the incidence of a wound infection.[2] Retained foreign bodies are a major cause of litigation against Emergency Physicians.[3,4] A high index of suspicion for a retained foreign body must be maintained whenever there is the potential for a foreign body in a wound.[5-7]** This chapter focuses on the identification and removal of subcutaneous foreign bodies using bedside Emergency Department ultrasound (US).

Foreign bodies may be small, leave no skin entry marks, and consist of many different types of material. Almost any object (i.e., solid, liquid, or gas) can become a foreign body and present itself in a location where it should not reside. Standard plain radiographs and fluoroscopy are good at identifying radiopaque objects (e.g., metal, gravel, and glass).[8-14] Glass accounts for up to 50% of missed foreign bodies using the physical examination and radiographs.[15] The ability of techniques to identify a foreign body vary by the size and composition of the foreign body.[10] Magnetic resonance imaging (MRI) and computed tomography (CT) are useful to detect foreign bodies.[13,16,17] These are expensive imaging modalities and the images vary depending on the length of time the foreign body has been present.[17] Certain foreign bodies may produce artifacts that diminish the MRI and CT image qualities.[13] Plastic, hair, vegetative material, and rubber are not radiopaque and are not routinely identified on plain radiographs.[6,8,9,11-14,18-20] US identifies radiopaque foreign bodies. **US can detect foreign bodies that are not radiopaque due to their different echotexture in relation to surrounding structures.[6,9,16-33]** The sensitivity and specificity of US in detecting retained soft tissue foreign bodies have been reported as 72% and 92%, respectively.[15] Subgroup analysis of radiolucent or wooden foreign bodies demonstrated a sensitivity of 96.7% and specificity of 84.2%.[15] US can detect foreign bodies that are very small.[34]

ANATOMY AND PATHOPHYSIOLOGY

Foreign bodies can be present in almost any part of the body. Foreign bodies can be detected with US in nontraditional places. They have been identified in the eye, esophagus, and tongue using US.[32,35,36] Foreign bodies isolated to small spaces (e.g., the web spaces of the hand) may not be identified due to the size of the US transducer footprint and an anatomic location that is difficult to scan.[29]

Not diagnosing foreign bodies can be dangerous for the patient and lead to an increased risk of complications. Legislation to introduce an aluminum penny into circulation in the United States in 1973 was defeated in part by Pediatricians' and Pediatric Radiologists' concern that these new coins would not be easily identified on plain radiographs.[37] Patients with retained lead foreign bodies can have statistically significant elevated blood lead levels as compared with matched controls.[38]

Foreign bodies have specific US characteristics. Smooth and flat surfaces typically produce a dirty shadowing or reverberation artifact.[39,40] Irregular surfaces with a small radius or curvature produce a cleaner shadow (**Figure 121-1**). Glass and metal typically produce a ring-down or reverberation artifact (**Figure 121-2**).[12,26,31,32,39,41] A wood foreign body produces a bright echogenic reflection with a strong acoustic shadow (**Figure 121-3**).[6,12,32,42] Foreign bodies residing in soft tissue longer than 24 hours can cause an inflammatory reaction that creates a hypoechoic rim around the echogenic foreign body (**Figure 121-4**).[39] Secondary signs (e.g., edema and inflammation) may not be seen in cases of retained foreign bodies.[43]

INDICATIONS

Patients with a foreign body sensation or soft tissue mass should be considered to have a foreign body until ruled out.[5,18] Foreign bodies can migrate great distances from the original insertion site. This requires the Emergency Physician to maintain a high index of suspicion for a foreign body.[44] The ideal treatment window for

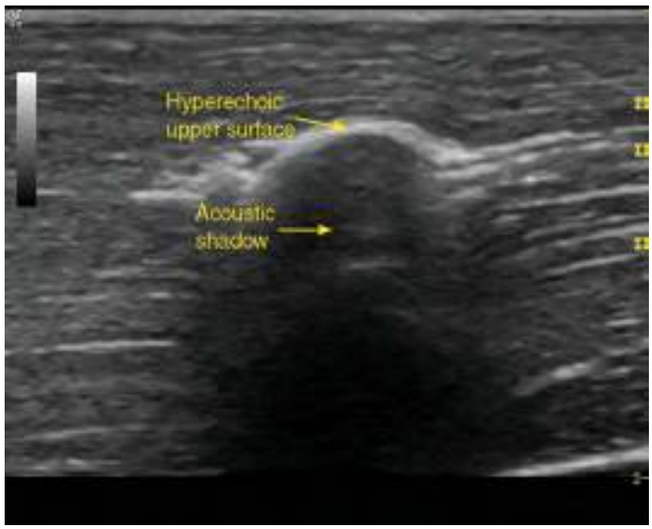


FIGURE 121-1. US image of a subcutaneous pebble. Notice the hyperechoic short radius and tight curvature. The pebble casts a strong acoustic shadow as it blocks transmission of the US beam.

retained foreign body removal is within 24 hours.[15] This time frame allows for visualization of the entry and exit wounds, decreased inflammation, decreased induration, and decreased scarring.[15] Any patient in whom the Emergency Physician has any index of suspicion for a foreign body should have additional investigations. Wood, thorns, spines, vegetative foreign bodies, dirt, clothing, and heavily contaminated foreign bodies should be removed immediately. Remove foreign bodies immediately with the potential for migration or entrance into the systemic circulation. It is generally believed that glass, metal, and plastic are inert and can be removed electively as they tend to be encysted by scar tissue.

Foreign body removal is dependent on the specific situation, physician preference, and patient characteristics. Remove the foreign body in patients who have persistent pain, neurapraxia, impairment of function, limitation of range of motion of a joint, or psychological distress. Remove foreign bodies adjacent to bone within 96 hours because they can cause osteomyelitis.[45] **Any patient from whom a foreign body, especially wood, was removed should be considered for further investigation to ensure there are no additional foreign bodies or a fragment of the removed foreign body present.[20]**

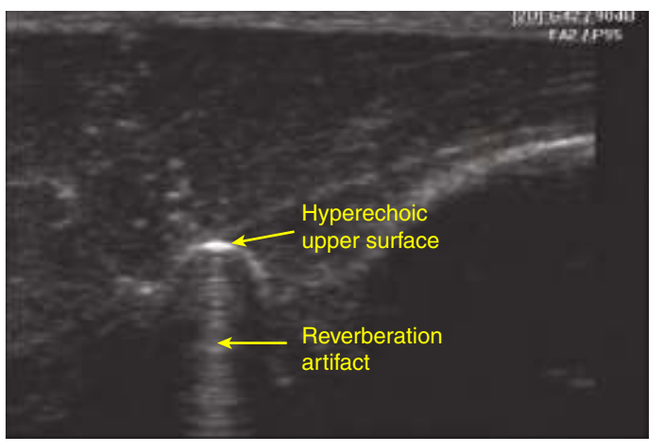
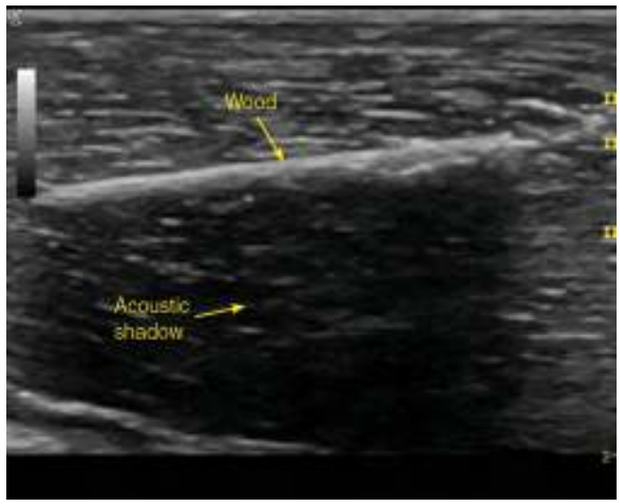


FIGURE 121-2. US image of a subcutaneous metallic BB. The BB is hyperechoic and produces a reverberation artefact.



A

B

FIGURE 121-3. US image of a subcutaneous wood foreign body. The wood produces a strong hyperechoic reflection and a hypoechoic acoustic shadow. ***A.*** Long axis US view. ***B.*** Short axis US view.

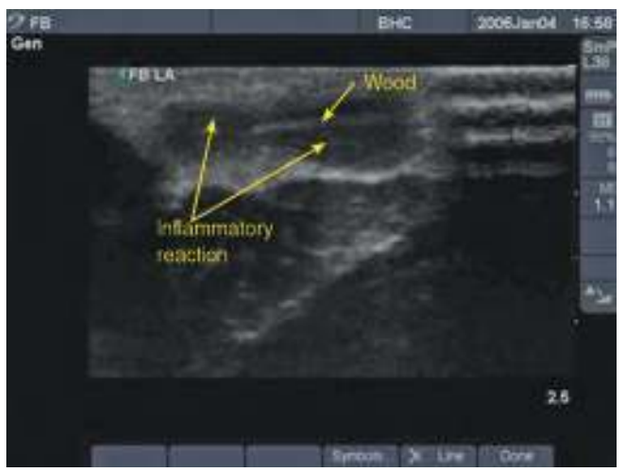


FIGURE 121-4. US image of a subcutaneous wood foreign body that has been in place for over 24 hours. The inflammatory reaction forms an anechoic rim around the foreign body. (Courtesy of James W. Tsung, MD, MPH.)

CONTRAINDICATIONS

US is a safe imaging modality without any known ionizing radiation. There are no known contraindications to using US to identify and assist in the removal of a foreign body.

EQUIPMENT

- US machine
- High-frequency linear US transducer, 7.5 MHz or greater
- Sterile US transducer cover or glove
- Skin cleansing solution (e.g., chlorhexidine or povidone iodine)
- Sterile US gel or other type of sterile gel (e.g., Surgilube)
- #11 scalpel blade
- Suture kit or individual components (i.e., sterile drape, gauze pads, hemostat, and forceps)
- Local anesthetic solution, usually lidocaine
- 5 mL syringes
- 27 gauge needles
- 18 gauge needle to draw up the local anesthetic solution
- Skin marking pen
- Paper clips
- Stand-off pad or a small (e.g., 50 or 100 mL) saline bag

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Cleanse the skin of any dirt and debris. Apply a liberal quantity of US gel over the area in which the foreign body is suspected. Use a high-frequency linear US transducer to search for the foreign body. Look for signs of the foreign body such as reverberation artifact, comet-tail artifact, acoustic shadowing, a hypoechoic rim surrounding an echogenic structure, or any other signs associated with foreign bodies. **It is recommended that a combination of axial, sagittal, coronal, and oblique scans are obtained due to the various ways a foreign body may be oriented.**[46]

Foreign bodies may be very superficial. It may be necessary to change the focal zone on the US machine. An acoustic interface may assist in locating a superficial foreign body. Apply a stand-off pad over the site of the foreign body. This can be either a commercially available stand-off pad, a small saline bag in which the air is removed, or a glove filled with saline or US gel.[19,47] The water bath technique works well for the hands and feet.[48] Place the body part in a water-filled container. Place the US transducer in the water-filled container without contacting the patient's skin. Identify the presence of a foreign body. Identify and localize the foreign body to make the removal easier for the patient and the Emergency Physician.

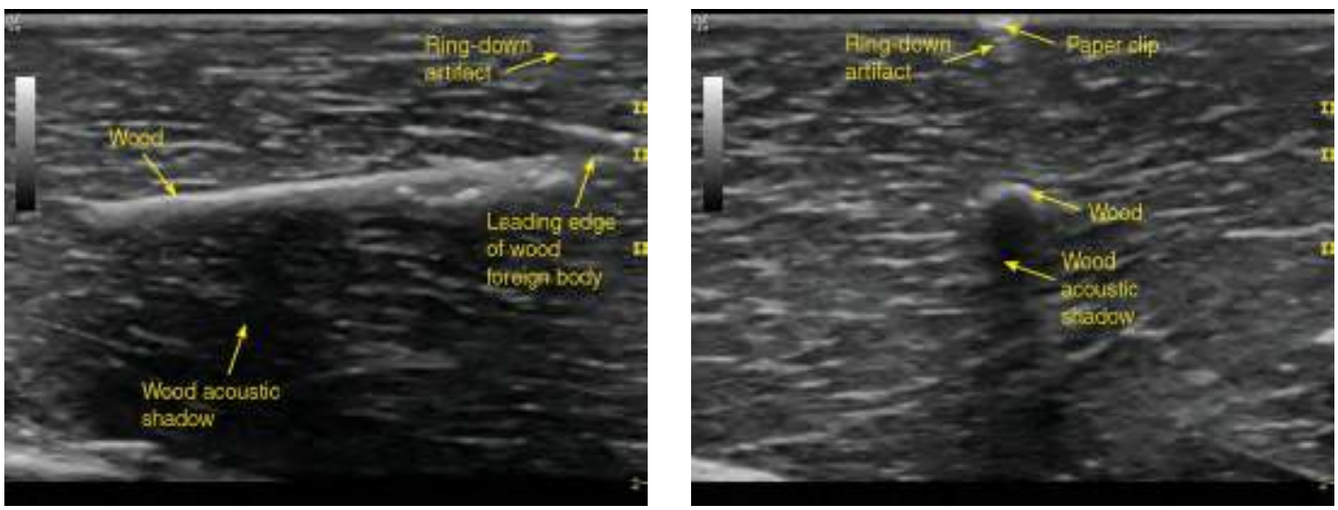
TECHNIQUES

PAPER CLIP LOCALIZATION AND REMOVAL

Mark the location of the foreign body on the patient's skin. It is helpful to use a metal foreign body (i.e., the paper clip) to create a shadow to locate the foreign body. The metal paper clip will cast a distinct shadow.[49]

Identify the foreign body on US. Place the US transducer on the patient's skin above the foreign body. Turn the US transducer so its long axis is aligned with the long axis of the foreign body. Insert the tip of an unfolded paper clip between the US transducer and the skin surface. Slowly advance the paper clip until the shadow it casts aligns with the leading edge (i.e., the most superficial aspect) of the foreign body (**Figure 121-5**). Use a skin marker to mark this location on the patient's skin. Rotate the US transducer 90° to visualize the short axis or cross-sectional view of the foreign body. Insert the tip of the unfolded paper clip between the US transducer and the patient's skin. Mark the location on the patient's skin where the shadow cast by the paper clip aligns with the leading edge of the foreign body. **The intersection of these two lines is where the leading edge of the foreign body resides under the skin.**

Prepare to remove the foreign body. Prepare a 5 mL syringe with a 27 gauge needle and local anesthetic solution. Cleanse the patient's skin with chlorhexidine or povidone iodine solution and allow it to dry. Apply sterile drapes to form a sterile field. Inject local anesthetic solution subcutaneously around the skin markings over the leading edge of the foreign body.



A B

FIGURE 121-5. Paper clip localization of a foreign body. The wood foreign body has a bright echogenic reflection and casts an acoustic shadow. The paper clip casts a ring-down or reverberation artifact that is lined up over the leading edge or most superficial most aspect of the foreign body. ***A.*** Long axis US view. ***B.*** Short axis US view.

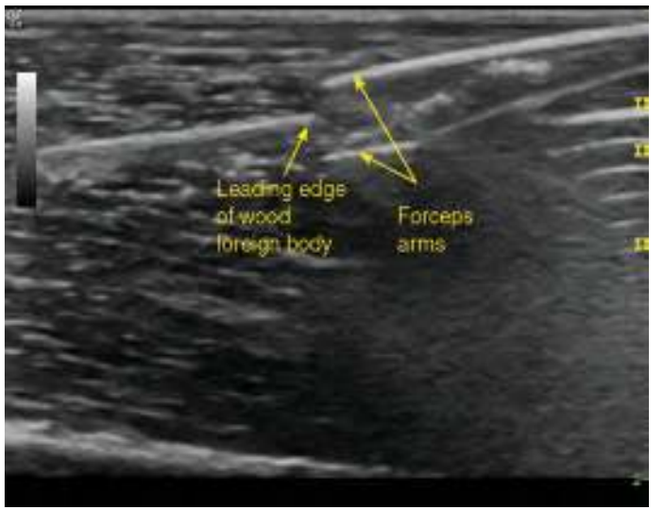


FIGURE 121-6. Retrieval of the foreign body. US image of the forceps approaching a wood foreign body. Note that the arms of the forceps are open and in line with the foreign body.

Prepare the US transducer. Apply US gel to the footprint of the US transducer. Apply a sterile transducer cover or a sterile glove over the US transducer. Squeeze any air out of the space between the US transducer and the cover. Apply sterile US gel on the transducer cover.

The Emergency Physician should don sterile gloves. The use of a hat, face mask, and sterile gown is not necessary for this procedure. Grasp the US transducer with the nondominant hand. Align the long axis of the US transducer along the long axis of the foreign body and approximately 3 to 4 mm proximal to the skin markings. Aim the syringe containing the local anesthetic solution downward and in the direction of the foreign body. Slowly insert and advance the needle through the skin mark under US guidance while slowly injecting a small volume of local anesthetic solution. The injection of local anesthetic solution around the foreign body creates a hypoechoic halo that helps with the identification of the foreign body.[50] Stop advancing the needle when the tip reaches the foreign body. Remove the needle without moving the US transducer.

Use a #11 scalpel blade to make a small skin incision just proximal to the skin mark and in the previously anesthetized area. Insert the jaws of a closed hemostat or the arms of a closed alligator forceps into the skin incision. Advance the instrument under US guidance and bluntly dissect down to the foreign body. Align the tip of the instrument with the leading edge of the foreign body (**Figure 121-6**). Open the instrument, grasp the foreign body, and remove it.

NEEDLE LOCALIZATION AND REMOVAL

Identify the foreign body using US. Cleanse the skin, apply sterile drapes, prepare the syringe, and prepare the US transducer as described above. Infiltrate local anesthetic solution subcutaneously in the area overlying the foreign body. Align the long axis of the US transducer with the long axis of the foreign body. Insert a 27 gauge needle in front of the US transducer. Slowly advance the needle under US guidance until its tip is against the foreign body. Injecting local anesthetic adjacent foreign body will often detach the foreign body from the adjacent tissue and facilitate removal.[51] Release the needle with the dominant hand and grasp a #11 scalpel. Insert the #11 scalpel blade along the needle until the tip of the blade is at the tip of the needle. Remove the scalpel blade and needle without moving the US transducer. Insert a forceps, hemostat, or alligator forceps along the incision track under US guidance. Open the instrument, grasp the foreign body, and remove it.

ASSESSMENT

It is important to rescan the area after removal of the foreign body to ensure there is no additional foreign body or a retained piece of the original foreign body that needs to be retrieved. Foreign bodies may not be removed in their entirety. A second foreign body may be present in an injury and not visible on the initial US scans.

AFTERCARE

Ensure that the foreign body is completely removed. There are many different thoughts on the care of wounds. Copiously irrigate the area. There is no evidence that using tap water to cleanse acute wounds in adults increases infection, and some evidence suggests that it reduces infection rates.[52] There is no strong evidence that cleansing wounds increases healing or reduces infection.[52] Close any lacerations from the wound itself or from the procedure to remove the foreign body. There is no strong evidence to support the use of antibiotics in simple nonbite wounds.[53] Wounds associated with foreign bodies are at an increased risk for infection. **Instruct the patient of the signs and symptoms of an infection.** In one review, 44.4% of all subjects with a foreign body removed were prescribed antibiotics.[54] When consultants were used in the care of these patients, the number rose to 84.4%.[54] A short course of antibiotics may be appropriate to prevent iatrogenic septic complications or sequelae caused by mobilization of the foreign body.[51]

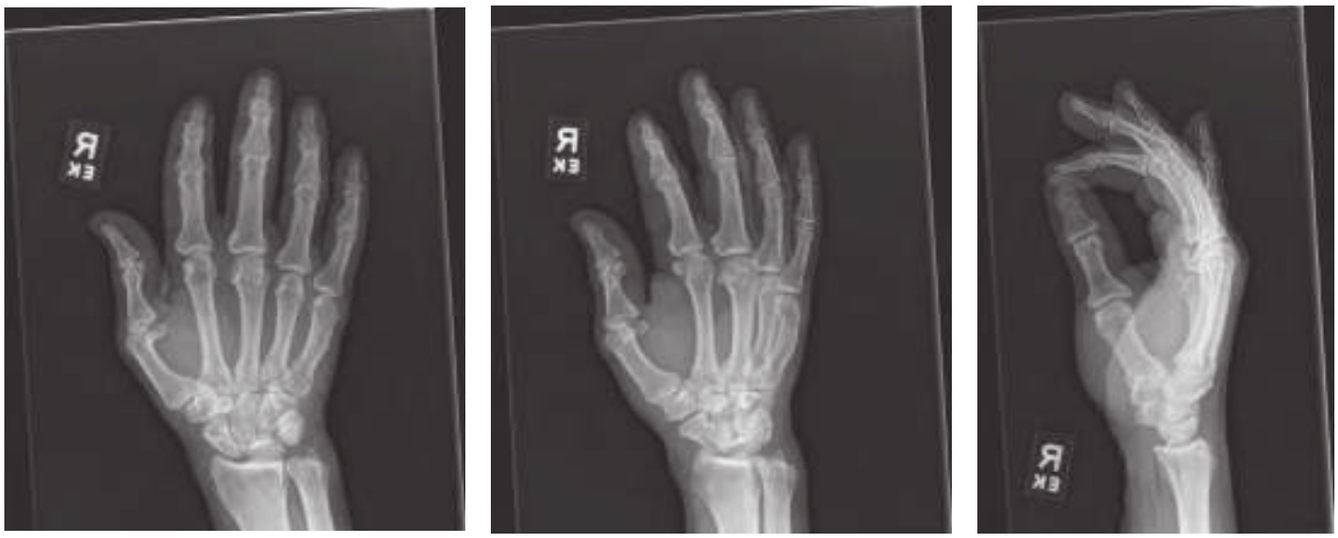
COMPLICATIONS

Foreign bodies and the procedures to remove them have been associated with infections (e.g., flexor synovitis, cellulitis, gas gangrene, abscesses, osteomyelitis, fungal infections, and lymphangitis), laceration of adjacent structures (e.g., nerves and tendons), neurapraxia, inclusion cysts, fractures, arthritis, and even an angiosarcoma.[14,26,45,55,56] Attempts are made to identify and remove all foreign bodies, but some of them may be missed. A retained foreign body can later lead to similar signs and symptoms.[57,58] **It is important to investigate and remove foreign bodies in a timely manner.**

CLINICAL EXAMPLES

Cases will be used to illustrate many of the complexities of removing a foreign body.

An elderly male presented with the complaint of a piece of wood lodged in his hand while working with a piece of bark. The patient had no imaging but did have local exploration that revealed a foreign body, which was removed. The patient left the Emergency Department prior to receiving discharge instructions or a prescription for antibiotics. The patient presented the next day with increased pain. He had a hand series of plain films which were interpreted as negative (**Figure 121-7**). The patient presented to the Emergency Department 69 days after the original injury. By this time, the patient had multiple courses of antibiotics, saw a Hand Surgeon the day before who indicated he could not help him, and was referred to the Emergency Department. The patient's physical examination revealed induration and tenderness on the palmar aspect of the wrist (**Figure 121-8**). The bedside US revealed a subcutaneous collection of fluid (**Figure 121-9*A***) and a foreign body (**Figures 121-9*B* and 121-9*C***). A Hand Surgeon was consulted due



A B C

FIGURE 121-7. Plain radiographs of the hand. ***A.*** Anteroposterior view. ***B.*** Oblique view. ***C.*** Lateral view.

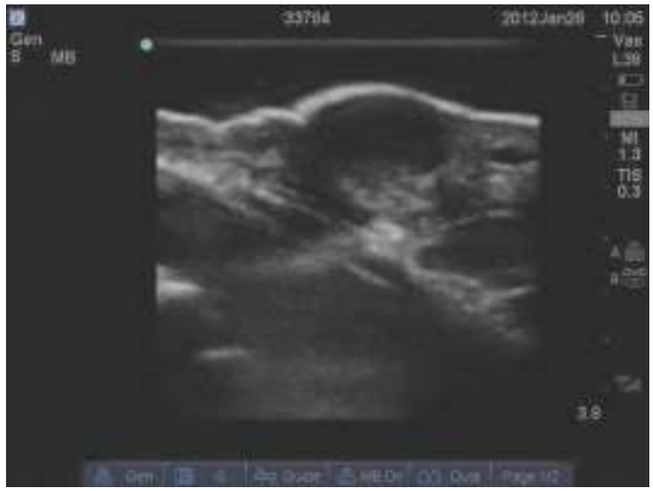
to the duration the foreign body was in place and the associated US findings (i.e., an abscess). The foreign body was removed and the abscess drained in the Operating Room.

Another patient fell on a glass table and broke the top with her upper extremity. She presented to the Emergency Department by paramedics soon after the injury. The patient had numerous small lacerations on her upper extremity. Plain radiographs were negative for any foreign bodies. Bedside US was performed by the Emergency Physician. A glass foreign body was seen (**Figure 121-10**). The glass was removed in the Emergency Department without difficulty.

SUMMARY

Foreign bodies are a major source of litigation. They are often not detected on physical examination or standard plain radiographs and can be associated with complications. A high index of suspicion must be maintained when the patient has a laceration, soft tissue mass, or foreign body sensation. US can be used to identify and remove foreign bodies in the Emergency Department.

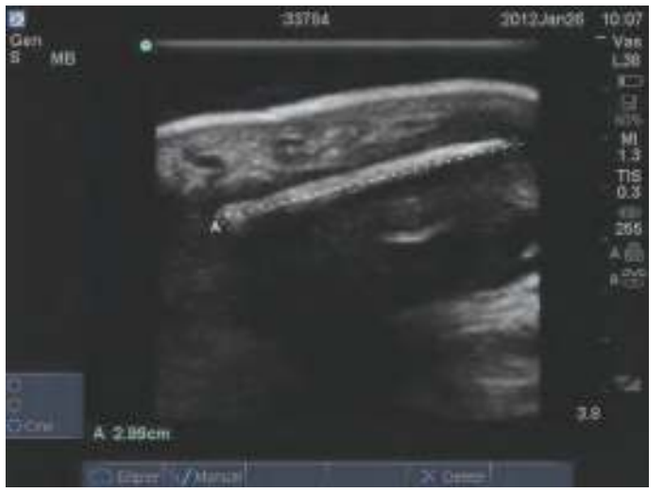
FIGURE 121-8. The patient's hand 69 days after the original injury.



A

B

FIGURE 121-9. The Emergency Department bedside US. ***A.*** A subcutaneous fluid collection at the top. ***B.*** The wood foreign body as seen without a water bath. ***C.*** The wood foreign body as seen with a water bath.



C

FIGURE 121-9. (*Continued*)

FIGURE 121-10. An US image of a glass foreign body. (Courtesy of Lloyd Gordon, MD.)

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122 Hair Tourniquet Management

Asim A. Abbasi

INTRODUCTION

Hair tourniquet syndrome or hair-thread tourniquet syndrome refers to the circumferential constriction of a finger, a toe, or the genitalia by hair or cloth fibers (**Figure 122-1**). Contraction of the tourniquet can lead to pain, tissue injury, and tissue necrosis if untreated. Infants and young children under 2 years of age are predominantly affected.[1-3] Genital hair tourniquets that affect the clitoris or labia are not as common and tend to occur in older children.[3-5] Hair tourniquets can rarely affect oral structures.[6-8]

There can be various clinical presentations. Fussiness and parental perception of pain are the common presenting signs.

FIGURE 122-1. A toe tourniquet and the distal edema. (Used from www.upload.wikimedia.org/wikipedia/commons/d/d3/Hairtournequet.JPG.)

Always consider a hair tourniquet in the differential for an inconsolable infant. The tourniquet may often be missed during a routine examination as the constriction may be mistaken for a physiologic anatomic crease on the infant's digit. Risk factors include increased maternal shedding of hair postpartum (e.g., one cause of telogen effluvium), co-bathing of the mother and infant, and co-laundering of clothes of others with those of the infant.[9,10]

Prompt diagnosis allows for treatment and prevention of further pain and morbidity. Mechanical release has been the mainstay of tourniquet release. Recent studies have proposed chemical depilatory agents as the primary modality to release a hair tourniquet.[4]

ANATOMY AND PHYSIOLOGY

The hair fiber is an elastic structure that can stretch when dry to 20% to 30% of its original length.[11] It can stretch up to 50% of its original length when the hair fiber becomes wet.[11] **A single strand of hair can be almost transparent, especially when wet.**[12] The presumed process of the hair tourniquet syndrome begins when moist or wet hair becomes entrapped within the confines of the infant's mitten or socks. Repetitive movement of the infant's flexed digit and contraction of the hair as it dries lead to constriction of the affected digit. The hair may cut into and become embedded within the epidermis. There may be reepithelialization of skin over the embedded hair.

Restriction of lymphatic drainage leads to local tissue edema distal to the constriction. Compromise of the vascular supply can lead to ischemia and necrosis. Erosion of the underlying bone with tendon involvement has been demonstrated in several cases.[9,13] The urethra can be involved with a penile tourniquet.[14]

A knowledge of the neurovascular supply of the affected appendage will avoid injury in the mechanical release of a hair tourniquet with an incision. Each finger and toe has four neurovascular bundles with each consisting of a digital artery and nerve. One bundle is located on each of the medial and lateral sides of the ventral and dorsal aspects of the digit. The neurovascular bundles of the penis and clitoris are located on their respective dorsal surfaces at the 11 o'clock to 1 o'clock positions. The location of the incision will depend on whether the hair tourniquet is on the finger, toe, or genitalia.

INDICATIONS

All hair tourniquets should be removed urgently to prevent further underlying tissue injury. Removal of hair tourniquets from the fingers and toes can be performed in the Emergency Department. Any concern for an underlying tendon injury or lack of improvement of tissue edema after removal of the hair tourniquet warrants an urgent referral to an Orthopedic Surgeon. Hair tourniquets involving the genitalia can often be removed with simple manipulation or a superficial incision. Those that are difficult to remove require an emergent consultation with a Gynecologist or Urologist.

CONTRAINDICATIONS

There are no proposed or studied absolute contraindications to the removal of hair tourniquets. The removal will immediately prevent or decrease the risk of tissue necrosis and morbidity. The use of chemical depilatory agents is not recommended on or near mucous membranes due to concerns for chemical irritation.

EQUIPMENT

- Sucrose solution for soothing the infant
- Gloves
- Topical analgesia (e.g., EMLA or LMX)
- Local analgesia without epinephrine
- Povidone iodine solution or chlorhexidine
- Needles, 25 or 27 gauge
- Syringes, 3 and 5 mL
- #11 scalpel blade
- Blunt probe
- Fine-tipped forceps
- Fine-tipped scissors
- Commercial depilatory cream (optional)

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the removal procedure and the anesthesia to the patient and/or their representative. Obtain an informed consent for the anesthesia and the removal of the hair tourniquet. Place the patient on a gurney. Secure infants and young children with a papoose and the affected extremity exposed (Chapter 232).

Clean the skin of any dirt and debris. Apply povidone iodine or chlorhexidine and allow it to dry. Provide anesthesia if needed. Perform a digital block or a block of the penis or clitoris (Chapter 156). Draw up local anesthetic solution into a syringe armed with a 25 or 27 gauge needle. Perform the regional block (Chapter 156). Perform a procedural sedation if necessary to control the patient (Chapter 159). This will prevent movement of the patient and accidental injury to underlying structures.

TECHNIQUES

MECHANICAL REMOVAL ON THE DIGITS

Grasp hair tourniquets that are easily visible, superficial, and not deeply embedded with a fine-tipped forceps. Unwind the hair tourniquet. Tourniquets that are tight or flush against the skin make grasping difficult. Insert a blunt probe underneath the hair tourniquet if possible (**Figure 122-2*A***). Make an incision with the scalpel blade using the metal probe as a cutting board (**Figure 122-2*B***). Cutting on the probe prevents accidentally cutting into the underlying skin.

Sometimes the hair tourniquet cannot be released using the techniques described above. This may be due to the hair tourniquet being deeply embedded within the skin, the skin reepithelializing over the hair thread, or worsening edema of the finger preventing visualization. Anesthetize the digit or provide procedural sedation. Make a direct incision into the digit longitudinally (**Figure 122-3*A***). **Incise from the proximal to the distal end and at the 3 o'clock or 9 o'clock position.** Incising at these positions decreases the risk of

A B

FIGURE 122-2. The use of a probe to remove a hair tourniquet. ***A.*** Probe insertion. ***B.*** Cutting the tourniquet.

A B

FIGURE 122-3. The incision remove a hair tourniquet. ***A.*** The lateral incision. ***B.*** The dorsal incision.

damage to the digital nerves and arteries. Some make the incision at the 12 o'clock position as an alternative (**Figure 122-3*B***). The incision should be made to the depth of the bone to ensure that the constricted hair thread is released completely.[15] The incision will be clean, have well-approximated edges, and be allowed to heal by secondary intention.

DEPILATORY REMOVAL ON THE DIGITS

There are several studies that have employed a chemical depilatory agent (e.g., Nair or Veet).[4,16] This approach was initially proposed as an adjunct to mechanical removal and has transitioned to a primary modality. The thioglycolate-based depilatory agents break the disulfide bonds in keratin and allow the hair strand to weaken. The advantage of this technique is that it is nearly painless except stinging and needs no incisions. **The chemical depilatory agents will have no effect on cloth or synthetic fibers (e.g., cotton, polyester, and rayon).**[17,18] They are not recommended for areas in proximity to mucous membranes.

Apply the depilatory agent to the area around the hair tourniquet. Allow it to work for 20 to 30 minutes. Wash off the depilatory under running warm water in the sink. Attempt to manually remove the remnants of the hair tourniquet. The depilatory agent can be applied for a second time if necessary.

GENITAL TOURNIQUET REMOVAL

Remove hair tourniquets on the penis or clitoris. Grasp hair tourniquets that are easily visible, superficial, and not deeply embedded with a fine-tipped forceps. Unwind the hair tourniquet. Tourniquets that are tight or flush against the skin make grasping difficult. Insert a blunt probe underneath the hair tourniquet if possible. Make an incision with the scalpel blade using the metal probe as a cutting board. Cutting on the probe prevents accidentally cutting into the underlying skin.

Hair tourniquets embedded within the epidermis or with epithelialization over it are harder to remove. **Concern for ischemia requires emergent Gynecology or Urology consultation. Prepare for a direct incision if the consultant is not immediately available.** Apply topical anesthesia to the area. A delay in the topical anesthetic taking effect may require the performance of regional anesthesia or procedural sedation. Make a superficial incision on the inferior surface on the penis or clitoris at the 4 o'clock or 8 o'clock position (**Figure 122-4**). Incising at these positions decreases the chance of damaging any neurovascular structures. **Stay superficial to the fascia when making the incision.** Make successive strokes with the scalpel blade deeper through the tissue until the constriction is released. **Do not cut into the corpora to prevent massive bleeding that is difficult to control.**

An alternative to incising is to initiate chemical depilatory treatment to the penis as described above.[4] The burning of these depilatory agents may require topical anesthesia, regional anesthesia, or procedural sedation**. Chemical depilatory agents are not recommended for clitoral hair tourniquets due to the proximity of mucous membranes.** Consult a Gynecologist urgently if simple isolation and incision of a clitoral hair tourniquet is not possible.

ASSESSMENT

Assess the patient's skin after the release of a hair tourniquet. Note and document any lacerations or abrasions from the hair tourniquet or the removal process. Check the neurovascular status of the affected digit after the procedure to ensure there is no compromise.

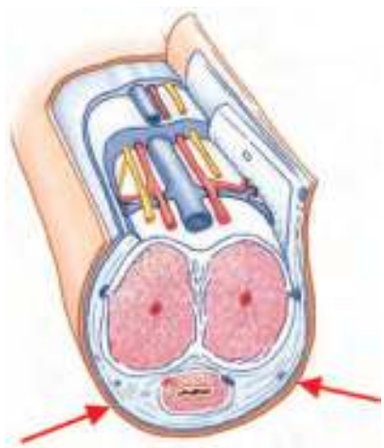

FIGURE 122-4. The penile incisions (*arrows*) to remove a hair tourniquet.

AFTERCARE

Reassess the patient after the release of a hair tourniquet and they have had time to sit calmly. There should be a noticeable improvement in the swelling. There may not be complete resolution of the edema. Recheck and document the neurovascular status of the affected digit after the procedure to ensure there is no compromise.

Verify the patient's tetanus status if an incision is made into the skin. Topical antibiotic ointment over the incision site can be used to prevent an infection. Prescribe antibiotics for 3 to 5 days if there is concern for contamination of the wound, contamination during the release of the hair tourniquet, or a laceration to the skin. Arrange follow-up with the primary physician to ensure that the affected tissue has returned to its normal caliber and there is no evidence of infection.

Provide anticipatory guidance to the caregivers to prevent a recurrence. Examine the baby's toes, fingers, and genitalia after every bath. Launder mittens and socks inside-out to prevent accumulation of hair fibers.[19] Launder the infant's clothes separately. Remove and inspect the infant's socks and mittens with diaper changes.

COMPLICATIONS

Compromise of the neurovascular status of the appendage may occur from the hair tourniquet or iatrogenic injury during the removal. Tenosynovitis of the digit may result if the tendon sheath was affected. Infection and cellulitis may also occur. Transient chemical irritation from the depilatory agent has been reported with its increasing use.

SUMMARY

The hair tourniquet syndrome is a rare diagnosis in infants. It can involve a finger, a toe, or the genitalia. The "hair" can be a hair or cloth fibers. Untreated hair tourniquets can lead to edema, strangulation, and ischemia. Release of the hair tourniquet can often be performed with a simple unwinding of the hair, a direct incision into the underlying tissue, or the application of a topical chemical depilatory agent. Close follow-up is required to ensure complete resolution of the edema and a normal neurovascular status.

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123 Tick Removal

Laurie Krass and Dhara Amin

INTRODUCTION

Ticks are blood-feeding external parasites that pose a significant infectious disease risk to humans and animals worldwide (**Figure 123-1**). They have been implicated as vectors in the transmission of more than 200 pathogens (e.g., babesiosis, ehrlichiosis, Lyme disease, Rocky Mountain spotted fever, tick-borne relapsing fever, tick paralysis, and tularemia).[1-3] Disease transmission occurs when stomach contents and saliva from the tick are introduced into the host during the blood-feeding process. Transmission of infectious agents by ticks is closely related to the duration of tick attachment and blood-feeding. Lyme disease is the most common tick-borne illness in the United States.[4] The risk of contracting it increases significantly once a tick has remained attached for more than 24 to 36 hours.[5,6] This is the time required for bacteria to migrate from the midgut of a tick to the salivary glands.[5-7] **The prevention of disease transmission relies on early and effective removal of attached ticks.**[8-11]

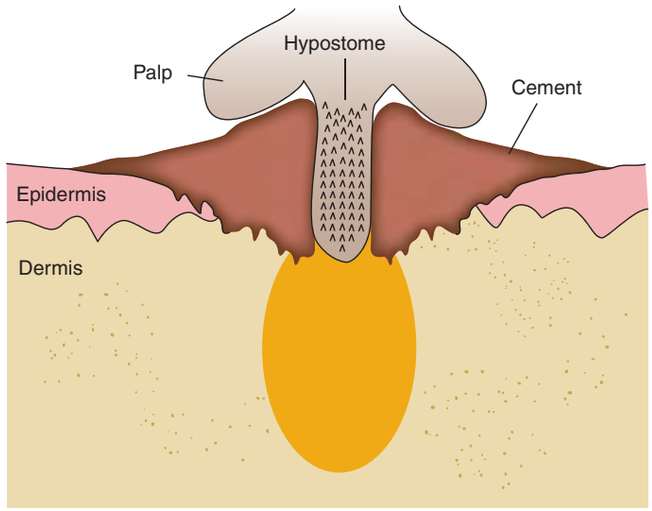
ANATOMY AND PATHOPHYSIOLOGY

Ticks are arachnids and divided into Argasidae (i.e., soft ticks) and Ixodidae (i.e., hard ticks).[12] Hard body ticks are responsible for the transmission of most human diseases and will be the focus of this chapter. Hard ticks pass through four life cycle stages from birth (i.e., egg, larva, nymph, and adult). They require a blood meal to progress to the next stage of their development.

A B

FIGURE 123-1. Examples of a tick. (Used from www.commons.wikimedia.org.)

A



B

FIGURE 123-2. The specialized mouthparts of the tick. ***A.*** Photograph. ***B.*** Artist illustration of the attachment of a tick.

Ticks are often encountered in the late spring, summer, and early fall. Ticks are more prevalent in rural and wooded areas. They like to feed on dark (i.e., covered) and moist areas of the body (e.g., axilla, groin, and scalp). The bite of a tick is painless, often goes unnoticed, and the tick is found attached to the skin.

Ixodes ticks have a shield-like scutum on the dorsum of their body with mouthparts that protrude forward. Nymphs and adults have eight legs. They vary in size depending on life cycle stage. Nymphal ticks are approximately 1.5 mm long. Adult unfed ticks are about 3 mm long, although once fed they can enlarge to 11 mm in length.[13] Tick coloration varies based on species and stage of feeding. Unfed ticks often appear black or brown. Engorged ticks during or after feeding can appear pink or dark red.[14] Ticks have specialized mouthparts that make their removal difficult (**Figure 123-2**).[12] They screw their mouthparts into the skin in a clockwise direction. Mouthparts include the palps, the chelicerae, and the hypostome. Ticks cut through skin using the chelicerae. Ticks then insert their hypostome, a tube-like structure through which the feeding takes place.[13] The hypostome has many backward-facing sharp barbs called denticles that prevent the tick from being dislodged. The tick can secrete a cement-like material around the hypostome to secure its attachment to the host while it feeds.[13] **The longer the tick is attached, the more difficult it becomes to remove it intact. The tick releases its mouthparts from the host after the meal is complete.** It can take anywhere from hours to up to 1 week for an adult tick to finish its blood meal and detach from the host.[15] The difficulty of removing a tick with all parts intact increases the longer the tick remains attached.

INDICATIONS

Remove any tick attached to the skin. Transmission of bacteria, spirochetes, viruses, or other infectious agents is directly related to length of time of attachment. Ticks attached less than 24 hours have a lower risk for transmission of disease.[16]

CONTRAINDICATIONS

There are no absolute or relative contraindications to the removal of a tick.

EQUIPMENT

REQUIRED EQUIPMENT

- Povidone iodine solution, chlorhexidine solution, or isopropyl alcohol swabs
- Gloves
- Fine forceps or mosquito hemostat

OPTIONAL EQUIPMENT

- Magnifying headlamp
- 18 gauge needle
- Local anesthetic solution
- 3 mL syringe with a 27 gauge needle
- Skin biopsy punches
- #15 surgical scalpel blade on a handle
- 5–0 or 6–0 nonabsorbable suture
- Specimen container with isopropyl alcohol

PATIENT PREPARATION

Explain the risks and benefits of the procedure to the patient and/or their representative. Risks include failure to remove all tick parts and infection. Obtain an informed consent for the procedure. Clean any dirt and debris from the skin. Apply povidone iodine or chlorhexidine solution to the skin surrounding the tick and allow it to dry. An alcohol swab can be used as a substitute.

A B

FIGURE 123-3. Removal of the tick. ***A.*** The tick is grasped as close to the skin as possible. ***B.*** Firm upward pressure is applied to remove the tick.

TECHNIQUE

Direct mechanical removal of the tick is the only recommended technique.[15] Grasp the tick with a fine forceps or mosquito hemostat at the tick's head or mouthparts, as close to the patient's skin as possible (**Figure 123-3*A***). **Do not crush, puncture, squeeze, or tear the tick's abdomen**. Apply firm and upward pressure to remove the tick (**Figure 123-3*B***). **Carefully examine the skin for any remaining portions of the tick's mouthparts.** Gently grasp and remove any retained parts of the tick with fine forceps or the tip of a sterile needle.

Direct upward traction is the preferred method of removal.[15] Some have suggested that a rotary counterclockwise movement combined with a firm pull may be more effective.[17] Another technique that has been described suggests first rotating the tick two complete revolutions around its axis to loosen its attachment prior to pulling it away from the skin.[18] The premise is that the twisting action will disengage the tick's mouthparts. There is not enough information to recommend or refute these methods. **It is not advised to rotate the tick during removal as this may break the mouthparts.**[19,20]

ALTERNATIVE TECHNIQUES

Some Emergency Physicians prefer a surgical technique to remove the tick and its attachment to the skin. This ensures that no mouthparts are accidentally left in the skin and avoids all contact with the tick. It theoretically eliminates the risk of increasing salivation, regurgitation of midgut contents into the bite, and the increase in disease transmission. Surgical removal may be the preferred method when attempting to remove smaller ticks or nymphs, or when removing a crushed tick from a previous failed attempt at conventional removal.[21]

Clean and prep the skin with povidone iodine solution or chlorhexidine solution and allow it to dry. Inject 0.25 to 0.50 mL of local anesthetic solution subcutaneously immediately underneath the tick's mouthparts. Apply povidone iodine or chlorhexidine solution to the skin again and allow it to dry. Stretch the skin on each side of the tick with the nondominant hand. Apply the skin biopsy punch perpendicular to the skin. **Make sure that the tick is centered within the skin biopsy punch.** Advance the skin biopsy punch downward with a twisting from clockwise to counterclockwise until a loss of resistance is felt. The loss of resistance indicates that the skin biopsy punch is through the epidermis and at the level of the dermis. Remove the skin biopsy punch. Grasp and lift the punched skin plug with forceps. Cut the plug at the dermal-epidermal junction with an iris scissors or a #15 scalpel blade. Alternatively, remove the tick with a limited skin excision using a #15 surgical scalpel blade by making an incision around the attachment site 2 to 3 mm in diameter and depth.[21] The skin can be closed with a single nonabsorbable suture, closed with Steri-strips, or left open to granulate and heal.

There are several chemically based techniques that have been used to stimulate passive self-detachment of a tick secondary to a lack of oxygen that patients may attempt at home to avoid an Emergency Department visit. These involve coating the tick with noxious materials (e.g., kerosene, fingernail polish, liquid nitrogen, petroleum jelly, rubbing alcohol, vegetable oil, or vinegar) to cause the tick to voluntarily withdraw its attachment.[22-24] These techniques have not been shown to be successful.[15,19,25-27] Techniques that attempt to suffocate the tick are not felt to be useful due to the low respiratory rate of 3 to 10 breaths per hour in a feeding tick.[15,18,19,23,26,27] The use of a heated object (e.g., a match tip or piece of metal) applied to the abdominal surface of the tick will not induce detachment, can cause the tick to burst and release infectious fluids, and can burn the patient's skin.[13,28] **Avoid all techniques that prolong the duration of attachment of a tick.** Subcutaneous injection of local anesthesia is ineffective at stimulating the tick from detachment.

Many devices have often been used to remove ticks. There are specific devices that are commercially available and advertised to aid in manual tick removal (**Figure 123-4**). This includes radiofrequency electrodes set to low power.[29] Some of these techniques and devices have been tested and are not felt to offer any advantage over forceps removal.[22,23,27,30]

ASSESSMENT

Carefully inspect the bite site to ensure no foreign material has been retained. Discard the removed tick as testing of the tick is usually not indicated. Place the tick in a specimen container with isopropyl alcohol until testing can be performed by a laboratory or the local public health department if it is desired.

AFTERCARE

Cleanse the area with a mild disinfectant or soap and water. Administer tetanus prophylaxis if the immunization history is not up to date. Instruct the patient that the appearance of any rash or the occurrence of any febrile illness 2 to 12 days after a documented tick exposure requires further medical follow-up. Provide the patient with information regarding the signs and symptoms of Lyme disease and other tick-borne diseases based on geographical area. Instruct the patient to inspect the site twice a day for signs of an infection and return to the Emergency Department or their Primary Care Physician if they develop discharge, drainage, erythema, a rash, swelling, or tenderness at the bite site.

A detailed discussion identifying the types of ticks or the indications for prophylactic antibiotic therapy are beyond the scope of this chapter. Consult the current medical literature or an Infectious Disease Specialist regarding these questions. The decision to prescribe antibiotics for a patient after tick removal should be made on an individual basis based on exam findings concerning for infection. The use of prophylactic oral antibiotics to cover typical skin flora or Lyme disease is not routinely recommended.

Educate the patient about ticks and preventative measures to avoid tick bites. **Prevention is the best protection.** Advise the patient to wear clothes to cover their arms, legs, and torso when outside. Tuck the cuffs of their pants into boots or socks. Apply a repellent (i.e., chemical or botanical) or an insecticide to clothes and exposed skin of people and pets. Avoid high grass and piles of leaves. Avoid contact with animals. Treat any pet dogs and cats with tick collars or liquid treatments monthly as per Veterinarian recommendations. Avoid placing any clothes on the ground outside. Wear light-colored clothing whenever possible. Physically check for the presence of ticks at the end of the activity or the end of the day.

A B C D

FIGURE 123-4. Examples of some of the commercially available tick removal devices. ***A.*** The Tick Key. ***B.*** The Poly-Ject Tick Remover. ***C.*** The Pro-Tick Remedy. ***D.*** The Tick Remover.

COMPLICATIONS

The major complication of the direct removal technique is the separation of the tick body from the embedded head. Leaving foreign material in the wound can serve as a site for a subsequent infection.[8,18] Any remaining pieces of the tick should be removed using the tip of a sterile needle, a skin biopsy punch, or sharp dissection with a #15 scalpel blade.

Inadvertent crushing of the tick may allow stomach contents or saliva from the tick to enter the wound. It is theorized that grasping the tick too distal to the head or across the thorax/abdomen could induce regurgitation of midgut contents into the wound and increase the risk of disease transmission.[8,13,18]

Local complications of tick removal include bleeding and infection. The application of direct pressure will control any bleeding. A cellulitis presents a few days after the bite and is often due to the ticks feeding process, skin contamination entering the bite site, or retained mouthparts. Carefully inspect the bite site to ensure there are no retained mouthparts. Use a skin biopsy punch to remove the bite site if unsure. Treat the cellulitis with oral antibiotics that cover typical skin flora.

SUMMARY

Ticks are a vector for several diseases with potentially severe complications. They should be removed from the skin as soon as possible. Disease transmission is directly related to the amount of time a tick is attached. The prompt removal is essential to prevention. The only recommended technique for tick removal that has been studied and proven effective is manual detachment using forceps.

Surgical excision is an option for smaller or crushed ticks. Efforts should be made to remove all parts of a tick from the bite site. Any mouthparts left in the skin increase the risk of subsequent infection.

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124 Fishhook Removal

Eric F. Reichman

INTRODUCTION

The presentation of a fishhook embedded in the subcutaneous tissue can be common depending on practice location and season of the year. The patient, or a well-meaning bystander, will often have already attempted removal that was prevented by the hook's barb. The ensuing tissue trauma and patient anxiety can complicate the task for the Emergency Physician. Removal can be difficult because a fishhook is designed not to pull out of a fish's mouth. Several methods of removal have been described.[1-19] The method chosen depends on the type and size of the hook, the depth of penetration, and the anatomic location of injury.

ANATOMY AND PATHOPHYSIOLOGY

Most fishhooks become embedded in the skin and subcutaneous soft tissue. The anatomy of a fishhook is simple (**Figure 124-1**). The long, straight section is known as the shaft. The proximal end of the shaft has a closed circle, the eyelet, where the fishing wire attaches. The distal end of the shaft curves in a semicircle known as the belly of the fishhook. The belly tapers into a sharp point with a barb. The barb is usually located on the inner surface of the hook, pointing away from the tip. It can also be on outer surface of some hooks. The barb, once pierced through the skin, becomes embedded within the tissue and prevents removal of the fishhook. Additional barbs may be located along the shaft of the fishhook.

INDICATIONS

Any embedded fishhook must be removed from the body. There is no reason a fishhook should not be removed by the Emergency Physician if no contraindication exists.

CONTRAINDICATIONS

There are no absolute contraindications to fishhook removal. Occasionally, the procedure should be referred to a consultant. Globe perforation, globe laceration, and eyelid perforation require emergent consultation with an Ophthalmologist.[10,11,13,14,18] Place the patient supine with a shield, not a patch, over the eye. Please see Chapter 193 for the complete details regarding eye patching and eye shields. **Penetration of, or near, vital structures (e.g., the neck, groin, or major neurovascular structures) should be given consideration for the appropriate surgical consultation prior to removal of the fishhook.**

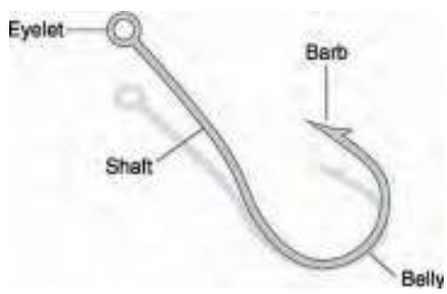

FIGURE 124-1. Anatomy of a fishhook.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Local anesthetic solution without epinephrine
- 3 mL syringe armed with a 25 gauge needle
- Wire cutter
- Needle driver
- Hemostat
- 18 gauge needle
- #11 scalpel blade on a handle
- String, fishing line, or a strong silk tie, at least 50 cm in length
- Tongue depressors
- Safety glasses/goggles or a face mask with an eye shield

The wire cutter and string need to be "clean" and not sterile. The patient's skin as well as the fishhook were probably dirty and contaminated when the hook penetrated the skin. Wipe the wire cutters with alcohol before and after using it to remove the fishhook.

PATIENT PREPARATION

Explain the risks and benefits of the procedure to the patient and/or their representative. Ask the patient to describe the type of fishhook embedded, especially in regard to the number and location of the barbs. Ask the patient to draw a picture of the fishhook or provide you with a similar one to examine. The number and location of the barbs will help in determining the appropriate removal technique. Obtain a signed consent form prior to beginning the procedure. Cleanse the skin of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the skin surrounding the embedded fishhook and allow it to dry. **The Emergency Physician should wear eye protection when removing a fishhook to prevent injury to their eyes.** At minimum, wear safety glasses or goggles. If available, a facemask with an eye shield is preferred.

TECHNIQUES

PULL-THROUGH TECHNIQUE

This is the traditional method that is used for larger sized hooks embedded in the soft tissue with the barb near the skin surface. This is the preferred technique for fishhooks embedded in the ear, a joint, or the nasal cartilages. Experienced fishermen often perform this technique in the field, as they would hate to lose prime fishing time to go to the Emergency Department.

Identify the barbed end of the fishhook located under the skin surface. Inject 0.5 to 1.0 mL of local anesthetic solution into the subcutaneous tissue overlying the barbed end of the fishhook to raise a skin wheal (**Figure 124-2*A***). Allow 3 to 4 minutes for the local anesthetic solution to take effect. Grasp the shaft of the fishhook with a needle driver (**Figure 124-2*B***). Advance the fishhook until the barbed end protrudes through the anesthetized skin (**Figure 124-2*B***). **Securely clamp a hemostat over the barb.** This will prevent it from becoming a projectile when cut and injuring someone. Cut the belly of the fishhook just proximal to the barbed end with a wire cutter (**Figure 124-2*C***). Grasp the shaft of the fishhook with the needle driver and withdraw it along its direction of entry (**Figure 124-2*D***).

Occasionally, fishhooks may have additional barbs on the shaft or the belly (**Figure 124-3**). Inject 0.5 to 1.0 mL of local anesthetic solution into the subcutaneous tissue overlying the barbed end of the fishhook (**Figure 124-3*A***). Allow 3 to 4 minutes for the local anesthetic solution to take effect. Grasp the shaft of the fishhook with a needle driver (**Figure 124-3*B***). Advance the fishhook until the barbed end protrudes through the anesthetized skin (**Figure 124-3*B***). Continue to advance the fishhook until all the barbs on the belly and shaft are below the skin surface (**Figure 124-3*B***). Securely clamp a hemostat over the proximal shaft of the fishhook (**Figure 124-3*C***). Cut the shaft of the fishhook at the level of the skin with a wire cutter (**Figure 124-3*C***). Grasp the fishhook just proximal to the barbed end with the needle driver and pull the remainder of the fishhook out of the tissues (**Figure 124-3*D***).

BARB-SHEATH TECHNIQUE

This method is reserved for small fishhooks embedded near the skin surface.[15] This technique should not be used for fishhooks in or near the eye, eyelids, ear, nose, or a joint cavity. Inject 0.5 to 1.0 mL of local anesthetic solution to form a wheal subcutaneously around the area where the fishhook enters the skin. Insert an 18 gauge needle along the entrance wound and aimed toward the barb (**Figure 124-4*A***). The bevel of the needle should face the barb with the goal being to engage and cover the barb. Advance the needle and engage the barb in the core of the needle (**Figure 124-4*B***). Gently twist and pull the hook back through the entrance wound while the needle covers the barb (**Figure 124-4*C***).

An alternative method is to insert a #11 scalpel blade through the anesthetized skin and parallel to the shaft of the fishhook at the site

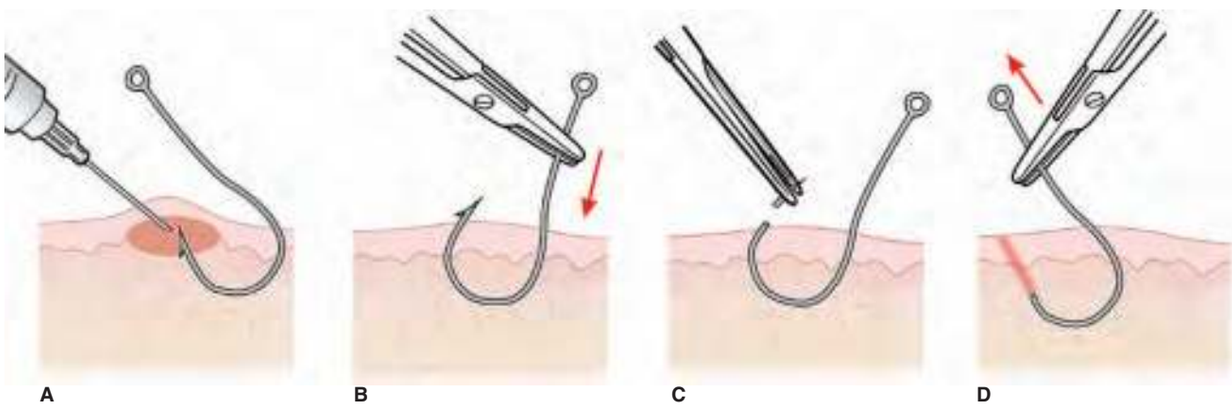

FIGURE 124-2. The pull-through technique for fishhook removal. ***A.*** Subcutaneous anesthetic is placed over the barb. ***B.*** The fishhook is advanced until the barb protrudes from the skin. ***C.*** A hemostat is placed over the barb before it is cut off with wire cutters. ***D.*** The fishhook is removed.

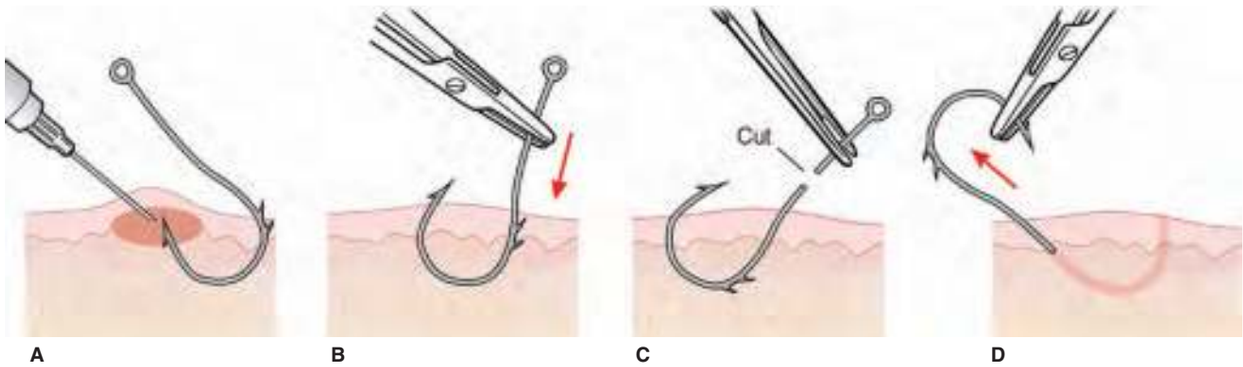

FIGURE 124-3. An alternative pull-through technique for the removal of a multibarbed fishhook. ***A.*** Subcutaneous anesthetic is placed over the barb. ***B.*** The fishhook is advanced, the distal barb has protruded through the skin, and additional barbs are under the skin. ***C.*** The shaft is cut. ***D.*** The fishhook is removed.

it enters the skin. Advance the scalpel blade until it is adjacent to the barb. Withdraw the fishhook and scalpel through the tract. The barb will be resting against the scalpel blade and not get embedded in the subcutaneous tissues. This method is not recommended due to the blind insertion of a scalpel blade into the soft tissues and the potential for secondary injury.

STRING-YANK TECHNIQUE

This method has been extensively described and is often performed by experienced fishermen in the field. It is rapid, effective, and easy to perform; requires fewer repeat attempts; and is relatively painless.[12] This technique should not be used for fishhooks in or near the eye, eyelid, ear, nose, or a joint cavity. **The Emergency Physician should take caution for themselves, the patient, and bystanders. The hook often forcibly flies out of the patient. Ensure the suspected path of the fishhook is clear. Eye protection is recommended with this technique for both the Emergency Physician and the patient.**

Place the body part that the fishhook entered firmly on a flat surface. Local anesthetic solution can be infiltrated, at the Emergency Physician's discretion, into the area where the fishhook enters the skin. Wrap the midpoint of a long string around the belly of the fishhook at the site it enters the skin (**Figure 124-5*A***). The string ends should be firmly wrapped around and secured to the index and middle fingers of the Emergency Physician's dominant hand. Use the gloved nondominant thumb or index finger to firmly depress the shaft of the fishhook against the skin until slight resistance is met (**Figure 124-5*B***). The shaft should be parallel to the skin and touching the skin. This will disengage the barb from the soft tissues. Quickly and firmly jerk the string (**Figure 124-5*C***). This maneuver will release the fishhook from the subcutaneous tissues and pulls it out through the entry wound.

Alternatively, the Emergency Physician may use two tongue depressors to provide secure traction to the string. Wrap each end of the string around one tongue depressor. Instruct an assistant to gently depress the fishhook shaft against the skin while the Emergency Physician jerks the string.[6]

AFTERCARE

The wound should be cleaned of any blood and a dry dressing applied. Administer tetanus prophylaxis if the immunization history is not up-to-date. A radiograph is indicated if there is any suspicion of a retained foreign body. Acetaminophen or nonsteroidal anti-inflammatory drugs will provide any required analgesia. Instruct the patient to clean the area with warm soapy water three times a day and to keep the wound covered until healed. Instruct the patient as to the signs of infection. They should return to the Emergency Department, or their physician, if signs of an infection develop. Routine follow-up is often not required unless the fishhook penetrated the ear, nose, or a joint.

Antibiotic prophylaxis remains controversial.[1,2,16,17] The use of antibiotics is left to the discretion of the Emergency Physician and should include consideration for the anatomic site of injury, depth of penetration, evidence of gross contamination, and factors that may compromise the patient's immunity (e.g., diabetes mellitus, HIV infection, steroid use, or malignancy). If antibiotic prophylaxis is chosen, prescribe a broad-spectrum antibiotic such as trimethoprim-sulfamethoxazole or doxycycline. These will provide good coverage against gram-negative organisms typically associated with water recreation injuries, as well as some coverage against methicillin-resistant *Staphylococcus aureus* and other community-acquired infections.[7] Adding ciprofloxacin for *Pseudomonas* coverage may also be considered, as *Pseudomonas* and its subtypes (i.e., specifically *Aeromonas*) are commonly found in soil and freshwater.[6,16]

FIGURE 124-4. The barb-sheath technique for fishhook removal. ***A.*** Insert the needle through the entrance wound and aimed toward the barb. ***B.*** Advance the needle through the entry site to catch the barb in the core of the needle. ***C.*** The needle and fishhook are removed as a unit.

FIGURE 124-5. The string-yank technique for fishhook removal. ***A.*** A string is wrapped around the belly of the fishhook. ***B.*** The shaft of the fishhook is depressed until resistance is encountered. The shaft should be parallel to the skin and touching the skin. ***C.*** A quick tug on the string will remove the fishhook.

COMPLICATIONS

Complications include infection or damage to the surrounding tissue. Infection is often due to either the contaminated fishhook inoculating the tissues or the fishhook penetrating contaminated skin. Using proper removal techniques will minimize, but not eliminate, any damage to the soft tissues.

Use caution when extracting fishhooks to avoid secondary injury to bystanders, the patient, or the Emergency Physician. Protective eyewear should be worn with all the techniques of fishhook extraction. Ocular injury has been reported to occur during this procedure.[2] Fishhooks have been surgically extracted from the hypopharynx and the intestine.[8,9,19] A surgical face mask with a face shield is another mode of protection that the author recommends. When using the pull-through technique, clamping a hemostat to the exposed portion of the fishhook that is to be cut off will prevent flying shrapnel.

SUMMARY

Fishhook removal can be accomplished by one of several simple techniques. It can be performed in the Emergency Department, the office, or the field with minimal supplies. Almost painless removal is possible in most cases without anesthesia. This procedure is gratifying both for the patient and the Emergency Physician.

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125 TASER Probe Removal

Myles McClelland, Alfred Coats III, and Thuy Nguyen

INTRODUCTION

Projective electronic control devices or conductive electrical weapons are commonly known as "TASER" (Thomas A. Swift's Electric Rifle) devices (**Figure 125-1**). They are often used by law enforcement and civilians as a less than lethal alternative to subdue individuals. These devices are deployed at an estimated rate of 904 times per day or approximately one every 2 minutes (personal communication, Steve Tuttle, TASER Int.). TASER devices are estimated to have saved individuals from serious injuries and death from guns and hand-to-hand combat. The devices prevent injury and death of law enforcement officers.[1] These devices are currently used in 107 countries throughout the world. They work by delivering a high-voltage, low-amperage electric shock that affects the motor and sensory functions of the nervous system.[2]

There are currently no U.S. Occupational Safety and Health Administration (OSHA) guidelines for TASER probe removal. This leaves protocols to be established by individual agencies, Emergency Departments (EDs), Emergency Physicians, and hospitals. The protocols vary. **Treat all probes that have penetrated the skin as biohazards. Follow OSHA standard universal precautions when handling and removing the probes.**

A

B

C

D

FIGURE 125-1. Different models of conducted electrical weapons. (Photos courtesy of TASER Int.)

ANATOMY AND PATHOPHYSIOLOGY

The electric shock is delivered through barbed electrodes that attach to the weapon by conductive wires, penetrates skin and clothing, and disrupts voluntary muscle control. The probe is like a fishhook (**Figure 125-2**). A wire is attached to the proximal end to conduct the electrical charge from the weapon to the probe. The body of the probe is smooth and aerodynamic. The distal end of the probe contains the barb with a reverse hook to maintain itself in a person and not fall out. The barb end contains one or two barbs (**Figures 125-2*A* and 125-2*B***). A new probe was released at the end of 2016 and is available as of 2017.

Blast doors protect the undeployed cartridge. The cartridge contains the probes that are angled away from the center, puncture pin, primer, nitrogen capsule, and the anti-felon identification system (AFID) (**Figure 125-3**). The probes are loaded into the cartridge such that the top probe goes straight and the other probe is at a fixed angle when deployed. The cartridges snap into the weapon. The TASER cartridges are deployed by an electrical arc. The electricity fires a small primer which forces a nitrogen capsule back into a hollow puncture pin. The pin releases the nitrogen into the probe chambers which force the probes out of the cartridge (**Figure 125-4**). The AFID consists of many small pieces of paper with the identity of the cartridge they came from. The pieces of paper are released when the cartridge discharges. The number on the pieces of paper can be tracked by the company to the cartridge purchaser. Inadvertent deployment can occur from static electricity. There are numerous cartridges that project the probe up to 35 feet accurately (**Table 125-1**). Cartridges for consumers (i.e., not law enforcement) are only available in the 15 foot projection.

A

B

FIGURE 125-2. The TASER probes. ***A.*** The XP and the standard probe. ***B.*** The new probe released at the end of 2016. (Photos courtesy of TASER Int.)

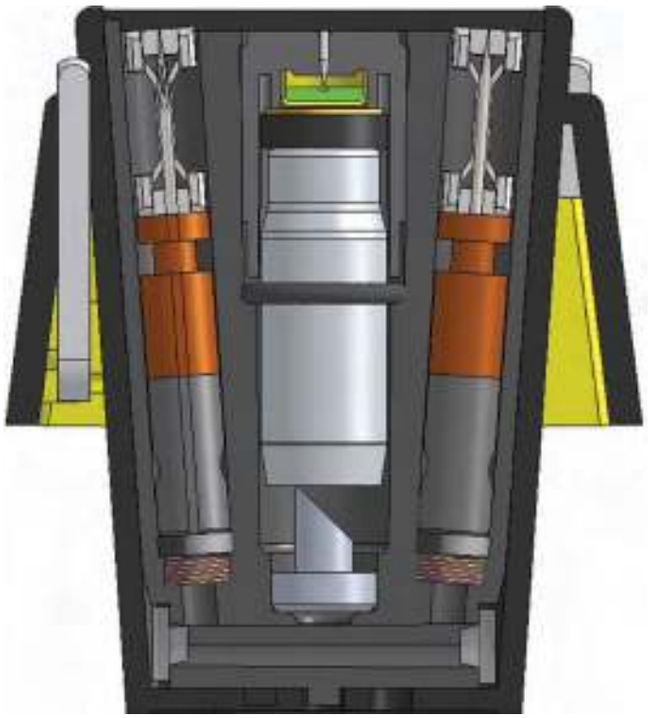

FIGURE 125-3. The internal components of the TASER cartridge. (Photo courtesy of TASER Int.)

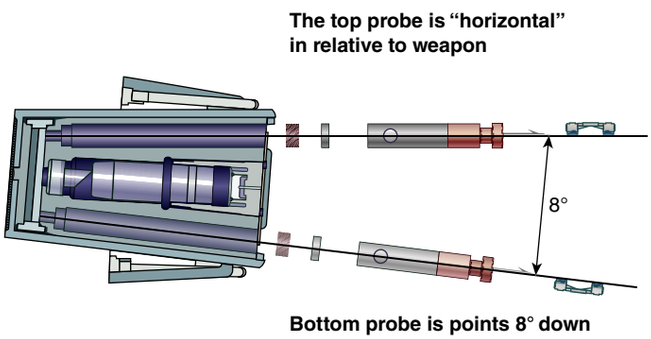

FIGURE 125-4. The firing of the TASER probes from the cartridge. (Photo courtesy of TASER Int.)

FIGURE 125-5. The firing of an actual TASER weapon. The AFID is seen as colored paper leaving the cartridge. (Photo courtesy of TASER Int.)

Squeezing the trigger on the weapon discharges the electricity into the patient (**Figure 125-5**). The wires from the weapon to the probes are live when the trigger is squeezed and can result in a shock if touched. The average power output of most devices is approximately 26 W, a current of 2 mA, and a maximum of 50,000 V.[3,4] The electric current is delivered in 100 millisecond pulses.

The nervous system is an intricate network that communicates via electrical impulses. The peripheral nervous system contains sensory and motor nerves that work in conjunction to communicate information between the brain and musculoskeletal system. This complex circuitry works to control voluntary and involuntary movements. TASER technology mimics these electrical impulses by inhibiting alpha motor neurons and causes involuntary contractions of skeletal muscle. This leads to neuromuscular incapacitation (NMI). There are different levels of NMI ranging from limited area effects to significant body lockup.

The TASER can be used to cause pain in addition to NMI. The pain results from the weapon touching the skin and pressing the trigger. The cartridge must not be in the weapon or it must be deployed for the weapon to be used to cause pain. The local pain that results from touch does not affect the central nervous system and does not cause NMI.

INDICATIONS

All TASER probes require removal. The indications for removal of TASER probes in the ED include patients transported for their removal and probes that are unable to be removed in the field by qualified personnel. Failure to remove the probe in the field may be due to either safety concerns, technical difficulties, or both.

TABLE 125-1 The TASER Probe Spread Varies by the Cartridge Used and the Distance It Travels

15, 21, and 25 foot cartridges		15 and 25 foot smart cartridges		35 foot smart cartridges	
General: ~1 (0.3 m) feet spread for every 7 feet (2.1 m) of probe travel		General: ~1 (0.3 m) feet spread for every 9 feet (2.7 m) of probe travel		General: ~1 (0.3 m) feet spread for every 14 feet (4.3 m) of probe travel	
Target distance (feet/meters)	**Probe spread (in/cm)**	**Target distance (feet/meters)**	**Probe spread (in/cm)**	**Target distance (feet/meters)**	**Probe spread (in/cm)**
2/0.6	4/10	9/2.7	12/31	14/4.3	12/30
5/1.5	9/23	18/5.4	25/64	28/8.5	24/61
7/2.1	13/33	25/7.6	36/92	35/10.6	30/76
10/3.0	18/46				
15/4.5	26/66				
21/6.4	36/91				
25/7.6	38/109				

Source: Modified from information received from TASER Int.

CONTRAINDICATIONS

Contraindications for immediate removal in the field include penetration to critical or vulnerable areas such as the eye, neck, head, female breast, penis, scrotum, and groin.[5-13] Injuries to these areas may need further evaluation and consultation prior to removal. Consultation with specialists (e.g., Neurosurgeons, Ophthalmologists, Urologists, or Vascular Surgeons) may be indicated for complicated injuries depending on the location.

EQUIPMENT

- Personal protective equipment
- Povidone iodine or chlorhexidine solution
- Local anesthetic solution (e.g., 1% lidocaine)
- #11 scalpel
- Wound dressings
- Medical pliers
- 3 or 5 mL syringe
- 25 or 27 gauge needle
- 16 or 18 gauge needle

PATIENT PREPARATION

Thoroughly assess the individual for direct and indirect injuries that occurred during and after incapacitation. Examine the entire patient and obtain diagnostic studies if indicated

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. Check the patient's tetanus immune status and update it as necessary.

Place the patient in a position of comfort on a gurney. Expose the area where the TASER probe is embedded. Prepare the area surrounding the probe. Remove any dirt and debris from the skin and the probe. Apply povidone iodine or chlorhexidine solution onto the skin and the probe. Allow this to dry. Don personal protective equipment (i.e., gloves, a gown, a face mask, and eye protection) to prevent exposure to the patient's blood or tissue fluids.

Some patients will tolerate the removal of the probe without anesthetic. This is not recommended. Draw up the local anesthetic agent using sterile technique into the syringe with a large (i.e., 16 or 18 gauge) needle. Switch the needle to a smaller one for patient comfort when administrating the local anesthetic solution. Infiltrate the area where the probe enters the skin with 1 to 2 mL of the local anesthetic solution.

TECHNIQUE

The area has been previously cleaned, prepared, and anesthetized. Cut the wires attached to the probe if they are still attached (**Figure 125-6**). Place the nondominant hand on the patient's skin surrounding the probe. Hold the skin taut. Grip the probe firmly with the dominant hand. Medical pliers can be used to grip the probe if unable to grasp it tightly or there are concerns about the possibility of barb injury. Pull the probe perpendicular to the skin in one swift motion. **Take care not to injure yourself or anyone assisting you with the barb.** Hold pressure to site to ensure hemostasis.

The barb may be difficult to remove by manual traction if significant resistance is met. Make a small incision with an #11 scalpel blade. Make the incision down to the level of the barb. **Do not cut any nerves, tendons, or vascular structures.** Most TASER probes have a channel on the base of the projectile that orients the direction of the barb. Remove the probe from the skin as described above. Clean the area with antiseptic. Irrigate the wound and close the incision with sutures.

FIGURE 125-6. The TASER probes with attached wires in a person. (Photo courtesy of TASER Int.)

ALTERNATIVE TECHNIQUE

An alternative method is like that used for the removal of a fishhook (Chapter 124). Use a 3 mL syringe armed with a 16 or 18 gauge needle. Insert the needle along the channel of the probe with the bevel of the needle facing inward. Cover the barb of the probe with the bevel of the needle. Slowly remove the probe and needle together to prevent the barb from catching in the surrounding tissue. Hold pressure to the area to ensure hemostasis.

ASSESSMENT

The barbed electrodes from a TASER probe are designed to penetrate at a depth of no greater than 4 mm, reducing risks for serious injuries and complications. Assess the patient for any neurologic or vascular damage that occurred from the probe removal. Consult the appropriate specialist if any damage is found.

AFTERCARE

Hold pressure to ensure hemostasis. Clean the area with antiseptic and apply a dressing. Standard wound care should be applied to the affected site. Have the patient monitor the probe site for signs and symptoms of an infection (e.g., erythema, fever, purulence, or tenderness). A wound check in 48 hours is advised for contaminated wounds, difficulty during the removal process, or if there is concern for a retained foreign body. Prophylactic antibiotics are not indicated due to the low risk for significant wound infection.[14]

COMPLICATIONS

Rare and significant injuries that have been reported in association with electronic control devices include bone lodgement of the probe, cutaneous burns, lacerations, rhabdomyolysis, testicular torsion, ocular injury, miscarriage, and death.[6-9,13,15-21]

TASER injuries around the orbits should raise the concern for the possibility of penetrating ocular injury.[5,7,9,12] Removal of the probe in such areas should not be attempted in the ED due to the likelihood of vitreous loss which may cause the globe to collapse.[7] A computed tomography (CT) scan of the orbits may be necessary to evaluate the extent of the injury and to identify the location of the barb. Consult an Ophthalmologist for removal of the probe in the Operating Room under general anesthesia.

Treat probes in the neck as penetrating trauma. Perform a careful and prompt assessment to the airway and surrounding vascular structures. Emergent treatment should include airway establishment, hemodynamic monitoring and stabilization, and clarification of the extent of injury per standard advanced trauma life support (ATLS) protocol. Standard treatment protocol remains controversial, but surgical intervention to determine the extent of injury caused by penetrating trauma to the neck remains the gold standard.[22] Use angiography in the stable patient to assess any suspicion of vascular injury. Consult a Vascular Surgeon if any signs of injury are noted.

Probes lodged in anatomically sensitive areas (i.e., the female breast, penis, scrotum, and groin) are normally not removed in the field by most prehospital protocols. Injuries to these areas may require local anesthesia, evaluation of surrounding structures, radiographic studies, and a more appropriate setting due to the sensitive location of the injury.

Probe penetration to the head and skull will require further evaluation, radiographic studies, and appropriate consultation in the ED.[10,11,23] A physical assessment, including a thorough neurologic exam, is necessary to determine if the individual is neurologically intact. Perform a CT scan to determine the extent of penetration and to rule out the possibility of intracranial hemorrhage. Removal of probes that are embedded in the skull or head is not recommended at the bedside in the ED. The removal process should be performed in the Operating Room to ensure that all parts of the probe are adequately removed.

SUMMARY

Law enforcement agencies continue to evolve into using less than lethal means of subduing suspects. There is a growing need for Emergency Physicians to understand the possible complications of these methods. The use of conductive electrical weapons (e.g., the TASER) is becoming more common. These weapons are meant to subdue an individual without causing death, serious injuries, and complications. Health care personnel involved in prehospital care as well as those in the ED should be able to recognize these possible complications in order to manage and provide appropriate care.

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126 Ring Removal

Abraham Berhane, Abdoulie Njie, Robert Needleman, and Steven H. Bowman

INTRODUCTION

The need to remove a ring is not uncommon in the Emergency Department.[1] Patients may present with an initial primary complaint that they can no longer remove a ring or that a ring has become painful. A variety of conditions may necessitate the urgent removal of a ring (e.g., allergic reactions, burns, fractures, increases in total volume status, infection, and swelling from extremity trauma). Swelling of the digit can rapidly progress and cause the ring to become a constricting band and compromise blood flow to the digit (**Figure 126-1*A***). Critically ill patients undergoing admission to intensive care settings or emergency surgery may need to have rings removed urgently. **The Emergency Physician's goal is to remove the ring in a timely manner and not cause additional injury.** The information in this chapter applies to rings on the fingers and toes.

ANATOMY AND PATHOPHYSIOLOGY

The second through fourth digits receive their blood supply through four vessels (i.e., palmar radial digital arteries, palmar ulnar digital arteries, dorsal radial digital arteries, and dorsal ulnar digital arteries). The thumb receives its blood supply from the dorsalis pollicis and princeps pollicis arteries. Blood returns from the digits via the dorsal digital veins. Venous return is impeded and swelling ensues when the digit is compressed for prolonged periods by a tight-fitting

A

B

FIGURE 126-1. The tight ring that cannot be removed. ***A.*** The tight ring with distal finger swelling in an unconscious patient. ***B.*** After ring removal with a ring cutter. The finger is macerated with an iatrogenic laceration from the ring cutter. (Courtesy of Brian Lin, MD, and www.lacerationrepair.com.)

ring. The swelling results in greater compression and further propagation of this cycle. The increased swelling will eventually impede the arterial supply to the digit in theory.

The greatest circumference of the finger is at the proximal interphalangeal (PIP) joint. **Rings usually become entrapped proximal to the PIP joint.** Skin breakdown and tissue necrosis occur if the constricting ring is not removed. The untreated digit is at risk for infections (e.g., cellulitis, osteomyelitis, and tenosynovitis). The digit viability may be threatened in severe cases. There are several case reports of rings that have become embedded in the soft tissue of the digits.[2-6]

Most patients will experience pain and seek medical attention prior to the development of severe complications. Patients with an altered mental status, peripheral neuropathies, peripheral vascular disease, psychiatric illness, or other chronic disability may present later with complications.[2,3,5,6]

INDICATIONS

Rings are usually removed to prevent ischemia of a digit. Remove a ring if the digit shows any signs of neurovascular compromise (e.g., decreased capillary refill, decreased pulse wave on pulse oximetry, decreased sensation, mottling, paleness, or paresthesias). Remove rings whenever the patient complains that a ring is causing pain. Rings must be removed from any injured digit where edema is possible (e.g., burns, contusions, crush injuries, fractures, lacerations, and sprains). Remove all rings from the digits on the involved side for any extremity injury above the digits and where edema of the distal extremity is possible. Other nontraumatic conditions that may necessitate emergent ring removal include acute increases in volume status, allergic reactions, and infections of the upper or lower extremity. A patient may present and request a ring be removed that is tight and can no longer be taken off. Consider the need for urgent ring removal with markedly decreased levels of consciousness and in all critically ill patients, particularly those being admitted to intensive care settings or undergoing emergent surgery.

CONTRAINDICATIONS

There are no absolute contraindications to removing a ring. It is important to note that certain techniques may be more applicable than others depending on the individual patient, provider experience, and the ring material.

EQUIPMENT

METACARPAL BLOCK

- Povidone iodine or chlorhexidine solution, or an isopropyl alcohol swab
- 3 mL syringe
- 25 or 27 gauge needle, 1 inch long
- 3 to 5 mL local anesthetic solution without epinephrine

LUBRICANT AND CATERPILLAR TECHNIQUES

- Lubricant (e.g., K-Y jelly, liquid soap, mineral oil, petrolatum, or Surgilube)

STRING TECHNIQUE

- String (1–0 silk suture, cotton umbilical string, intravenous tourniquet, Penrose drain, or tracheal tape)
- Mosquito hemostat

RUBBER BAND TECHNIQUE

- 3 to 4 mm wide rubber band
- Lubricant (e.g., K-Y jelly, liquid soap, mineral oil, petrolatum, or Surgilube)
- Mosquito hemostat

GLOVE TECHNIQUE

- Rubber gloves
- Lubricant (e.g., K-Y jelly, liquid soap, mineral oil, petrolatum, or Surgilube)
- Mosquito hemostat
- Scissors

RING CUTTER TECHNIQUE

- Ring cutter, manually operated or battery-powered
- Steinman pin cutter if a ring cutter is not available
- Two large hemostats or needle drivers
- Pliers, optional

VICE-GRIP PLIERS TECHNIQUE

- Medium size vice-grip pliers

MISCELLANEOUS SUPPLIES

- Ice water
- Penrose drain or intravenous (IV) tourniquet
- Basin or zip-lock bag for ice water

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Document this in the medical record. A signed informed consent is not required for the removal of a ring. Place the patient sitting upright or in a semi-recumbent position. Position their hand on a bedside procedure table. Provide analgesia in the form of a metacarpal block for patients complaining of pain (Chapter 156).

The removal of a tight ring is considered a two-step process. Reduce the edema in the finger and then remove the ring.[7] Instruct the patient to elevate the affected hand or foot to reduce edema prior to any attempts at ring removal. Use a finger trap to elevate the affected extremity if the patient has difficulty or cannot keep the extremity elevated (**Figure 126-2**). Consider soaking the hand or foot in ice water for 5 to 10 minutes to reduce edema. An elastic bandage, gauze, Penrose drain, or piece of tape can be wrapped around the finger from distal to proximal to further reduce swelling.[8]

The simplest and most effective way to remove a ring from a finger is to cut the ring using a ring cutter. Reassure the patient that most cut rings can be repaired by a jeweler. Patients may still be reluctant to have expensive rings or rings with sentimental value removed with a ring cutter. The Emergency Physician must consider time needed to remove the ring, the individual circumstances regarding the entrapment, the equipment available, which alternative techniques may be effective, and the status of the Emergency Department.[1,9]

TECHNIQUES

LUBRICANT TECHNIQUE

This technique works for mild cases of ring entrapment with minimal swelling and without significant trauma. Many patients will attempt to remove the ring using some type of lubricant at home prior to presenting to the Emergency Department (**Figure 126-3**). The same technique may be tried in the Emergency Department prior to attempting other techniques. Liberally apply a lubricant (e.g., K-Y jelly, liquid soap, mineral oil, petrolatum, or Surgilube) to the digit and beneath the ring. Attempt to advance the ring over the PIP joint with steady traction.

CATERPILLAR TECHNIQUE

This technique is useful in that it requires no additional materials other than lubricant (**Figure 126-4**).[10] Liberally apply a lubricant to the finger (**Figure 126-4*A***). Slide the ring down the proximal phalanx and just proximal to the PIP joint. Apply and maintain upward pressure on the bottom of the ring (**Figure 126-4*B***). Simultaneously swing the upper portion of the ring distally (**Figure 126-4*C***). Pull the top of the ring over the PIP joint. Apply and maintain downward pressure on the top of the ring (**Figure 126-4*D***). Simultaneously swing the lower portion of the ring distally to free it (**Figure 126-4*E***).

FIGURE 126-2. The hand and arm suspended in a finger trap. (Courtesy of Instrument Specialists Inc., Boerne, TX.)

FIGURE 126-3. The use of butter as a lubricant to remove an entrapped ring.

Continue the technique motions to remove the ring from the finger. This "caterpillar motion" allows the ring to be slowly advanced distally and removed. A Kelly clamp can be used to push the ring and gain a mechanical advantage.[11]

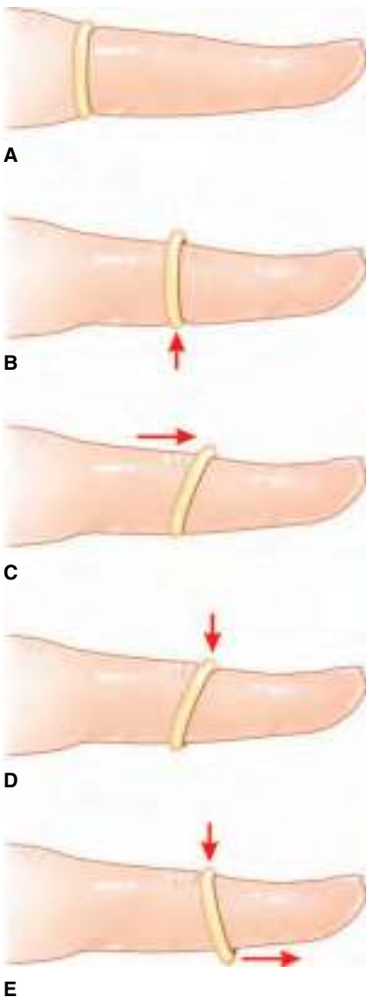

FIGURE 126-4. The caterpillar technique. ***A.*** The entire finger is lubricated. ***B.*** The ring has been pushed down the proximal phalanx until it reaches the PIP joint. Push the bottom of the ring up firmly while maintaining pressure. ***C.*** Swing the top portion of the ring distally over the joint while maintaining upward pressure. ***D.*** Push down firmly while maintaining downward pressure. ***E.*** Swing the bottom portion of the ring proximally over the joint and remove the ring.

STRING TECHNIQUE

Several authors have extensively described the string technique and its modifications (**Figure 126-5**).[5,7,12-14] This technique consists of using a "string" to compress the edematous tissue, exsanguinate the digit, and facilitate the passage of the ring over the PIP joint. Avoid this technique if the patient has an embedded ring, an associated finger fracture, or an associated finger laceration.[12,15]

Pass a length of 1–0 silk suture or a piece of string underneath the ring (**Figure 126-5*A***). Avoid monofilament sutures and smaller-size sutures as they may break or inadvertently cut the patient if wound too tightly. Passage of the string or suture may be facilitated with the use of a mosquito hemostat.[12,15,16] Wind the distal portion of the suture tightly around the digit in a closed spiral (**Figure 126-5*B***). **There should be no interposition of skin between the turns of the suture material to ensure even compression of the skin and soft tissue**. Continue the spiral distally to just beyond the PIP joint. Grasp the proximal end of the suture. Unwind the suture while maintaining traction in the distal direction (**Figure 126-5*C***). The ring will be pushed distally as the suture unwinds. The ring is easily removed once it clears the PIP joint (**Figure 126-5*D***).

Other materials (e.g., cotton gauze, oxygen mask straps, Penrose drains, rubber intravenous tourniquets, surgical mask ties, or umbilical tape) have been used instead of string.[7,13,14,17-20] These materials have certain practical advantages. They require fewer turns and shorter lengths to encircle the finger because they are wider than suture. It is easier to wind these materials around the digit without interposition of the skin between the turns (**Figure 126-6**).

Insert the umbilical tape under the ring from distal to proximal with the aid of a mosquito hemostat (**Figure 126-6*A***). Pull 6 to 7 cm of the umbilical tape proximal to the ring (**Figure 126-6*B***). Wind

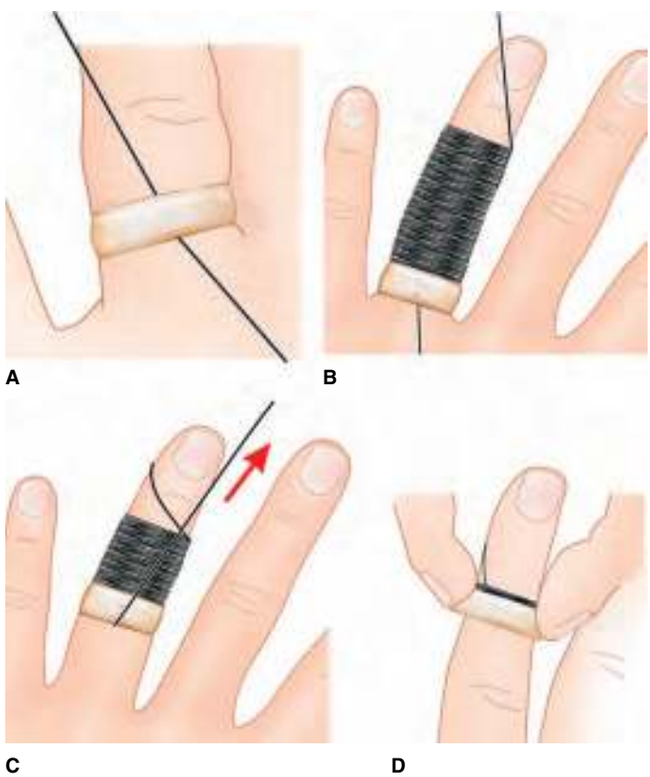

FIGURE 126-5. The string technique. ***A.*** A string is passed between the ring and the finger. ***B.*** The string distal to the ring is wound tightly around the finger and continued distally to the level just below the PIP joint. ***C.*** The string proximal to the ring is slowly unwound and moves the ring distally. ***D.*** When the ring passes the PIP joint, it usually comes off without effort.

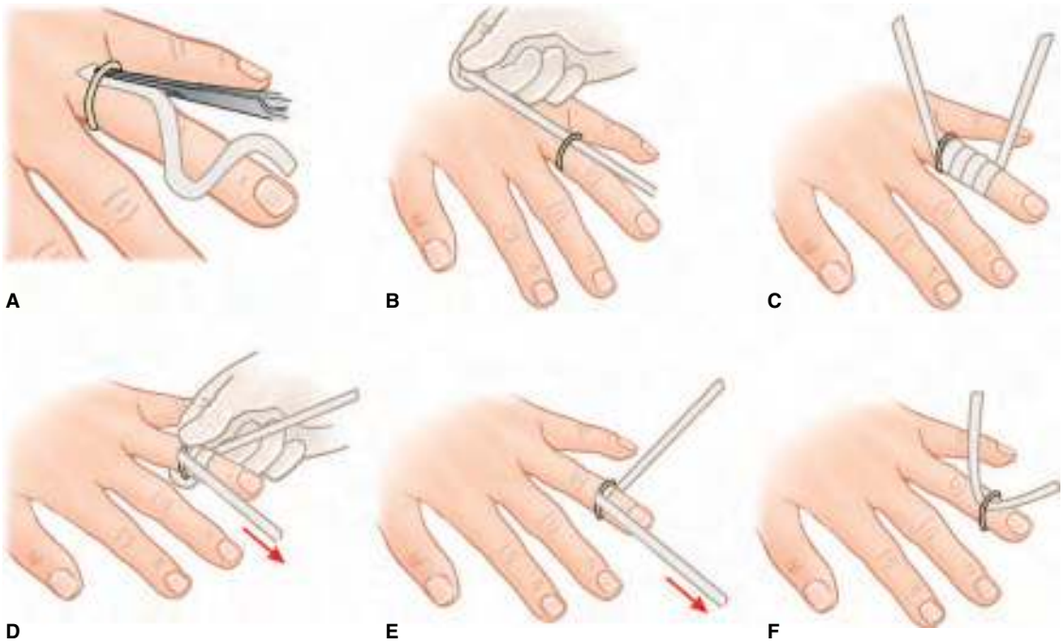

FIGURE 126-6. The string technique with umbilical tape. ***A.*** The umbilical tape is inserted under the ring with a mosquito hemostat. ***B.*** The umbilical tape is pulled through to the other side of the ring. ***C.*** The umbilical tape distal to the ring is wound tightly around the finger to the level just below the PIP joint. ***D.*** The umbilical tape proximal to the ring is slowly unwound and moves the ring distally. ***E.*** The ring passes the PIP joint. ***F.*** The ring is free and usually comes off without effort.

the distal portion of the umbilical tape tightly around the digit in a closed spiral (**Figure 126-6*C***). There should be no interposition of skin between the turns of the umbilical tape. Continue to spiral distally to just beyond the PIP joint (**Figure 126-6*C***). Grasp the proximal end of the umbilical tape. Unwind the proximal end of the umbilical tape while maintaining traction in a distal direction (**Figure 126-6*D***). Continue to unwind the umbilical tape until the ring passes the PIP joint (**Figure 126-6*E***). The ring is easily removed once it clears the PIP joint (**Figure 126-6*F***).

This technique may be slower than using a ring cutter. It does assure the integrity of the ring and minimizes the potential risk of patient injury that may occur with use of certain commercial devices (e.g., the locking pliers for removal of tungsten carbide rings).[13]

RUBBER BAND TECHNIQUE

This technique uses a 3 to 4 mm wide rubber band to apply traction on the ring and facilitate its passage over the PIP joint in a safe

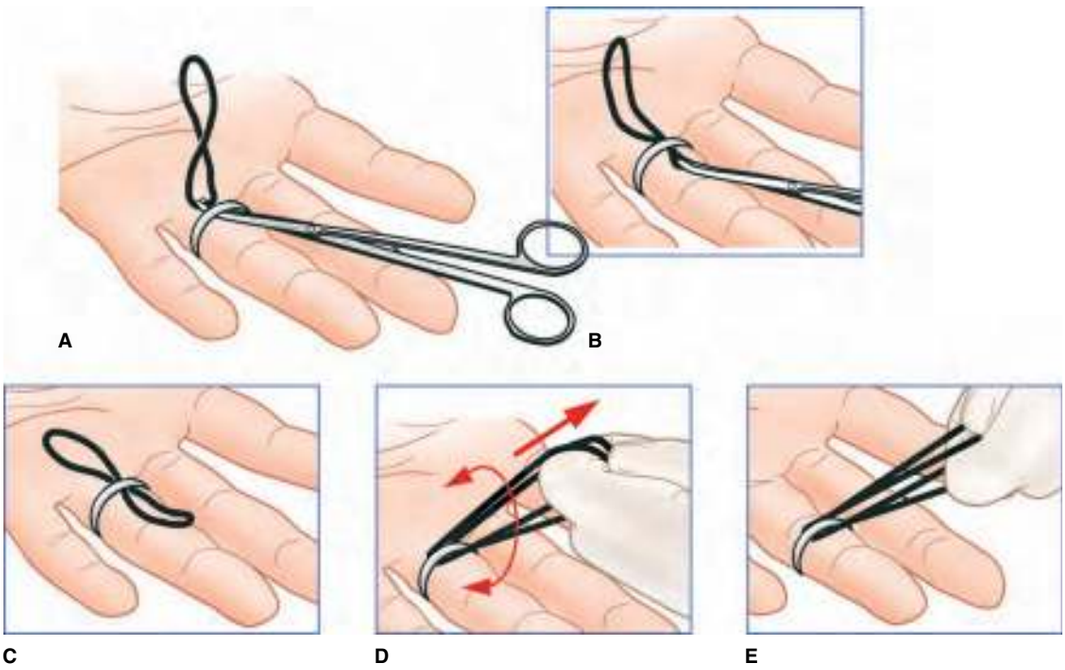

FIGURE 126-7. The rubber band technique. ***A.*** A mosquito hemostat is passed below the ring and grasps the rubber band. ***B.*** The rubber band is pulled beneath the ring and out the other side. ***C.*** The rubber band is positioned with equal lengths of loop on each side of the ring. ***D.*** Both loops of the rubber band are grasped and pulled distally while it is simultaneously rotated circumferentially. ***E.*** When the ring passes the PIP joint, it usually comes off without effort.

and efficient manner (**Figure 126-7**).[16,21] Success has been reported using rubber bands. Reserve this technique for less severe ring entrapments. Avoid this technique if the patient has an embedded ring, a fracture, or a laceration.

Lubricate the finger liberally. Pass the rubber band beneath the ring using a mosquito hemostat (**Figures 126-7*A* and 126-7*B***). Position the rubber band so that equal lengths are on each side of the ring (**Figure 126-7*C***). Insert a finger through both loops of the rubber band (**Figure 126-7*D***). Pull the loops of the rubber band distally while simultaneously moving them circumferentially between the ring and the finger (**Figure 126-7*D***). Continue the motion until the ring is removed (**Figure 126-7*E***).

GLOVE TECHNIQUE

This technique has been advocated for use in patients with underlying soft tissue injury to the finger.[22,23] Its success is anecdotal. The glove technique uses a finger cut from an appropriately sized rubber glove (**Figure 126-8**). The glove finger acts as a barrier to protect damaged soft tissue, provides a "leading edge" to guide the ring over the damaged tissues, and provides mild compression. The glove technique may be no more effective than any other in cases of severe finger edema despite these theoretical advantages.

Choose a glove that fits the patient snugly. Use the patient's other hand to aid in choosing the right size glove. Cut the finger from a glove to match the finger of the patient (**Figure 126-8*A***). Cut off the tip of the finger of the glove to create a cylinder (**Figure 126-8*A***). Slide the cylinder onto the patient's finger. Advance the proximal portion of the cylinder beneath the ring using a mosquito hemostat (**Figure 126-8*B***). Lubricate the cylinder and the ring (e.g., K-Y jelly, liquid soap, mineral oil, petrolatum, or Surgilube). Pull the proximal edges of the latex cylinder distally with fingers or mosquito hemostats to advance the ring distally (**Figure 126-8*C***). Continue to pull on the proximal cylinder until the ring moves past the PIP joint and falls off the finger.

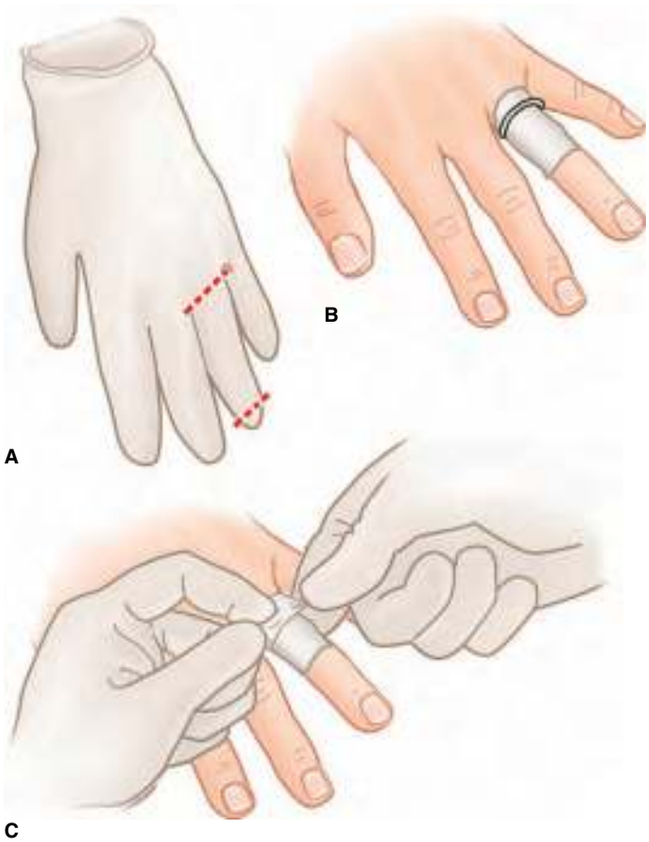

FIGURE 126-8. The latex glove technique. ***A.*** The finger matching the one on which the ring is lodged is cut from an examination glove. ***B.*** The latex cylinder is put on the finger. The proximal edges of the cylinder are pulled under and proximal to the ring using a mosquito hemostat. ***C.*** The proximal edges of the latex cylinder are slowly pulled distally to roll the ring off the finger.

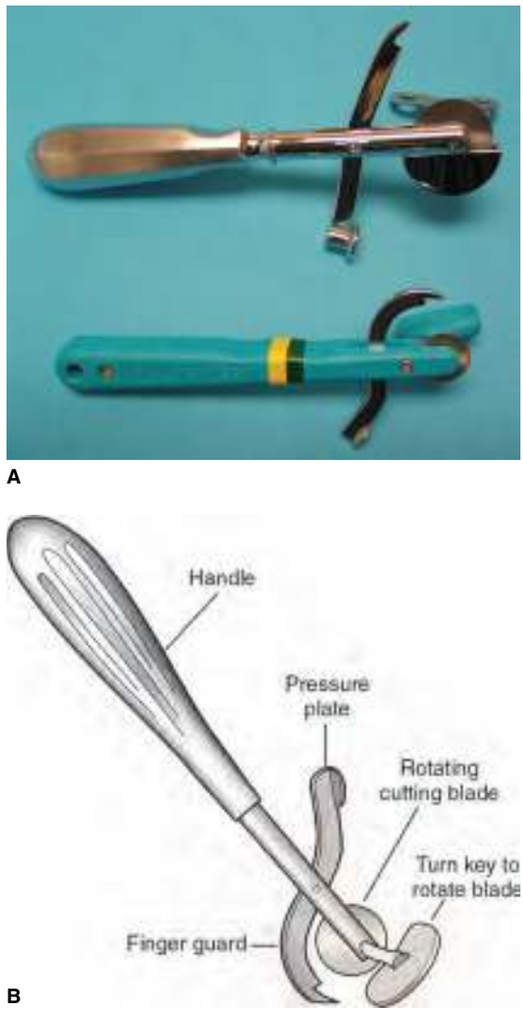

FIGURE 126-9. Manually operated ring cutters. ***A.*** Examples of manually operated ring cutters. ***B.*** The anatomy of a ring cutter.

RING CUTTER TECHNIQUE

The definitive method for ring removal is cutting the ring. Cut the ring if the patient presents with an embedded ring, entrapment with nonjewelry items, severe swelling, or an underlying injury. Cutting a ring is generally rapid and safe. A ring cutter can be used to cut rings made of gold, plastic, platinum, silver, stainless steel, and titanium.

Many devices will cut rings. The standard rotary ring cutter should be available in every Emergency Department (**Figure 126-9**). Inspect the ring cutter blade periodically to ensure that it is sharp. Battery-powered ring cutters are available (**Figure 126-10**). The advantages of the powered ring cutter are that they can easily cut nonjewelry items, do not rely on strength, and are easy to use, fast, lightweight, and powerful. The Steinman pin cutter has been used successfully in the absence of a ring cutter to cut rings (**Figure 126-11**).[24] A sharp pair of diagonal pliers can be effective to cut small rings.

FIGURE 126-10. The GEM Ring Cutting System (Mooney & Co., Ashland, OR). A battery-powered and motorized ring cutter.

Pass the finger guard of the ring cutter between the ring and the digit at the thinnest part of the ring (**Figure 126-12**). **Use care to place the ring cutter correctly and avoid additional injury.** Squeeze the ring using a heavy needle driver, hemostat, or pliers to change its shape from round to oval to facilitate passage of the finger guard (**Figure 126-13**). This additional distortion of the ring will not further exacerbate the entrapment.[24] Lower the cutting blade onto the ring. Turn the turn key to rotate the blade while maintaining pressure on the ring (**Figure 126-14**). Continue turning the turn key until the blade completely cuts through the ring. Pry the ring open with two hemostats, needle drivers, or pliers once it is completely cut through and remove the ring (**Figure 126-15**).[25] An alternative to one cut and prying the ring open is to make two cuts on opposite sides of the ring so that it falls apart in two pieces.

Irrigate any open wound (**Figure 126-1*B***) with normal saline to remove any small metal fragments that may result from the cutting process. There is a case report of a foreign body granuloma caused by metal particles left in a finger wound following ring removal.[26]

VICE-GRIP PLIERS TECHNIQUE

Many rings cannot be removed with the previously described techniques.[27] These rings can be of a material that cannot be cut with a carbide, diamond, or metal cutting disk on a ring cutter. This includes rings made of ceramics, natural stone (e.g., jade or onyx), and tungsten carbide. These rings can be removed by cracking them into pieces in a controlled fashion using a vice-grip pliers.[13,28]

A medium-sized vice-grip pliers is usually appropriate for most ring sizes. Open the jaws of the vice-grip pliers. Adjust the tightening screw so that the closed jaws of the vice-grip fits over the ring (**Figure 126-16**). Close and clamp the vice grip so that the jaws close lightly on the ring. Release the jaws, turn the tightening screw one-quarter of a turn, and reclamp the jaws on the ring. Continue this process of releasing the jaws, turning the tightening screw one-quarter of a turn, and reclamping the jaws on different places of the ring each time until a crack is heard. Keep continuing this process of tightening the jaws and reclamping different areas of the ring until the ring breaks into pieces and falls off.

FIGURE 126-12. Ring removal using a manual ring cutter.

Tungsten carbide rings are an important consideration due to their increasing popularity. These rings are inexpensive and bought from all over (e.g., Internet). **It is difficult to remove these rings with traditional ring-cutting devices due to inorganic compounds that make them extremely hard.** Tungsten carbide is the hardest material used to make jewelry. It cannot be cut or even scratched by instruments routinely in the Emergency Department. **The brittle nature of the material makes tungsten carbide amenable to removable with the vice-grip pliers.**[13,29] Removal techniques using string, umbilical tape, or vice-grip pliers are effective at removing tungsten rings.[13] Use of a vice-grip plier was more efficient (i.e., 23.1 vs. 135.4 seconds) at the expense of destroying the ring.[13] Potential complications to consider include superficial lacerations and retained debris within any potential wound.[29]

FIGURE 126-11. The Steinmann or orthopedic pin cutter.

FIGURE 126-13. A pair of pliers is used to make the ring "oval" in shape. This may facilitate the use of a ring cutter.

FIGURE 126-14. Cutting a ring with the manual ring cutter (left). The ring is pried off (upper right) and the finger is released (lower right).

ALTERNATIVE TECHNIQUES

Patients may rarely present with other heavy circular objects on their digits (e.g., nuts, steel rings, or washers) that cannot be removed using a manually operated ring cutter. Powered cutting tools such as heavy-duty saws and bolt cutters may be needed to remove these objects. Battery-powered ring cutters may be ideal in these situations. Power saws and Dremel tools with carbon blades have been used to successfully remove hardened steel rings from fingers.[6,30,31] Case reports exist of the use of dental saws or dental drills for the same purpose.[32-35] **Use care if it becomes necessary to use a powered metal cutting device to protect the patient from secondary injury from the cutting element and from thermal injury associated with the heat generated by these powered devices**. Keep the finger wet to reduce friction-associated heat and thermal injury from electric saw equipment.[34]

Another technique uses the elastic strap from a nonbreather mask.[14] Wrap the elastic strap around the finger from distal to proximal until reaching the ring. Push the tail of the strap under the ring toward the hand. Allow time for the edematous tissue to be compressed by the elastic band. Unwind the elastic band starting at the top of the hand toward the tip of the finger, moving the ring over the PIP. The pain from compression can be mitigated by a digital block.

ASSESSMENT

Thoroughly examine the finger for any injuries after the ring is removed (**Figure 126-1*B***). Reassess perfusion to the digit by noting the capillary refill time, color, and pulse oximeter readings on the affected digit compared to adjacent fingers.

FIGURE 126-15. Prying open a cut ring.

FIGURE 126-16. A vice-grip pliers can be used to remove hard or brittle rings.

AFTERCARE

No specific aftercare is required following the ring removal process. Elevation, nonsteroidal anti-inflammatory drugs (NSAIDS), and local wound care are all that is necessary. Consultation with a Hand Surgeon, Orthopedic Surgeon, Plastic Surgeon, or Podiatrist is recommended in severe cases that include embedded rings, infections, neurologic compromise, or vascular compromise. The aftercare is based on any lacerations and/or fractures of the digit. Determine the patient's tetanus immune status and administer the appropriate prophylaxis if the skin is broken. Instruct the patient not to place any rings on the digit until the edema has completely resolved. Place the ring, and any pieces, in a specimen container and return it to the patient. A jeweler can later fix most rings.

COMPLICATIONS

The direct complications of ring removal are minor compared to the complications that may occur from failure to remove a ring. Direct complications include secondary injury (i.e., to nerves, soft tissues, and vascular structures) and granuloma formation. This can be due to passing objects and instruments under an extremely tight ring or from improperly used instruments to remove the ring. Ring fracture compression techniques (i.e., vice-grip pliers) that require large amounts of pressure for removal have been found to be a safe methodology when indicated.[13]

Ring cutters have their own specific issues. It may cut too slowly. Ensure that the proper cutting technique is being used. The cutting disk may be dulled or the wrong cutting disk for the material. Check that the proper cutting disk is on the ring cutter and that it is not dull or worn. Install fresh batteries or recharge them to maximize the power of the electric ring cutter. The ring and the patient's finger can become quite hot and burnt.[36] Periodically check the progress and make sure that the ring has not been cut and is now cutting the finger guard. Using a cutting disk that is dull, worn, or incorrect can generate significant heat. Correct these issues. Submerge the finger in ice water for a few minutes and then resume cutting the ring.

SUMMARY

Ring removal is a relatively straightforward and simple procedure. A variety of potential approaches may assure success. Use of the ring cutter is the most reliable and quickest technique. Base the decision to use a ring cutter upon the urgency with which the ring must be removed and not upon the monetary or sentimental value of the ring. Direct compression techniques may increase in implementation as the popularity of titanium and tungsten rings grow. Formal ring removal algorithms exist.[34] Implement the techniques that are most familiar to achieve safe and timely results.

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127 Subungual Hematoma Evacuation

Steven H. Bowman, Neera Khattar, and Natasha Thomas

INTRODUCTION

The fingers and toes are two of the most commonly injured areas of the body. The distal ends of these digits (i.e., the nail apparatus) are especially prone to injury. Digital injuries include amputations, avulsions, contusions, crush injuries, fractures, and lacerations.

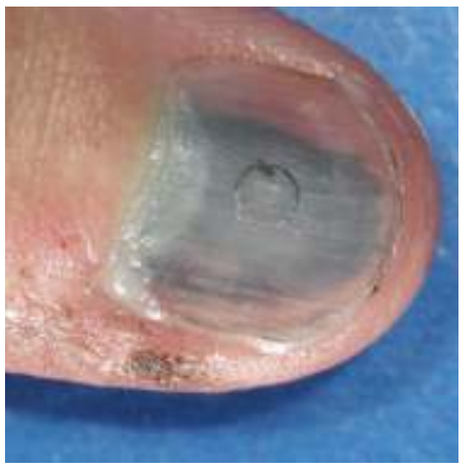


FIGURE 127-1. A subungual hematoma. (Courtesy of Path Scientific LLC, Carlisle, MA.)

The most common injuries to the distal fingers and toes are crush injuries (e.g., closure of a door and dropped objects). Digital injuries occur when the distal aspect of the finger or toe is caught between two objects and typically involves a large amount of force.

Subungual hematomas often develop following blunt or crush injuries to the distal fingers or toes (**Figure 127-1**).[1-3] Hematomas can form from repetitive mild trauma to the digits (e.g., running while wearing tight shoes). These injuries cause accumulation of blood between the nail and the nail bed and result in a subungual hematoma (**Figure 127-2**). Treatment of a subungual hematoma is relatively straightforward, yet in some cases, it is still controversial. It is important to understand the structure of the distal finger or toe (**Figure 127-3**) to determine whether drainage alone will be sufficient management and to also consider how initial management may affect outcome.

ANATOMY AND PATHOPHYSIOLOGY

The nail apparatus is a complex structure composed of multiple components (**Figure 127-3**). The nail plate is a keratinized structure that overlies the nail bed and matrix.[4] It is curved on three edges to allow it to embed into the surrounding soft tissues. The perionychium is composed of the nail bed and the surrounding soft tissue. The hyponychium is the junction of the nail bed at the sterile matrix and the fingertip skin beneath the distal margin of the nail. The eponychium is the distal portion of the nail fold where it attaches to the proximal surface of the nail plate. The matrix is visible in the proximal portion of the nail as a white half-moon structure known as the lunula.

The nail bed consists of the germinal matrix on the proximal ventral floor of the nail fold and the sterile matrix extending from the

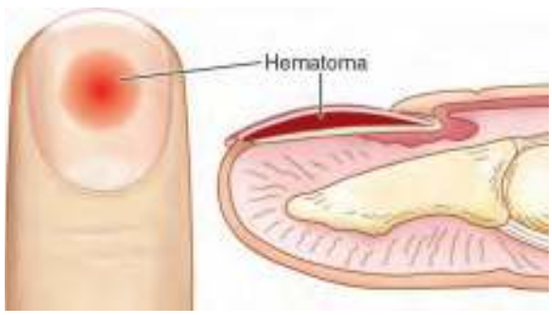


FIGURE 127-2. The subungual hematoma. ***A.*** Surface view. ***B.*** Sagittal view.

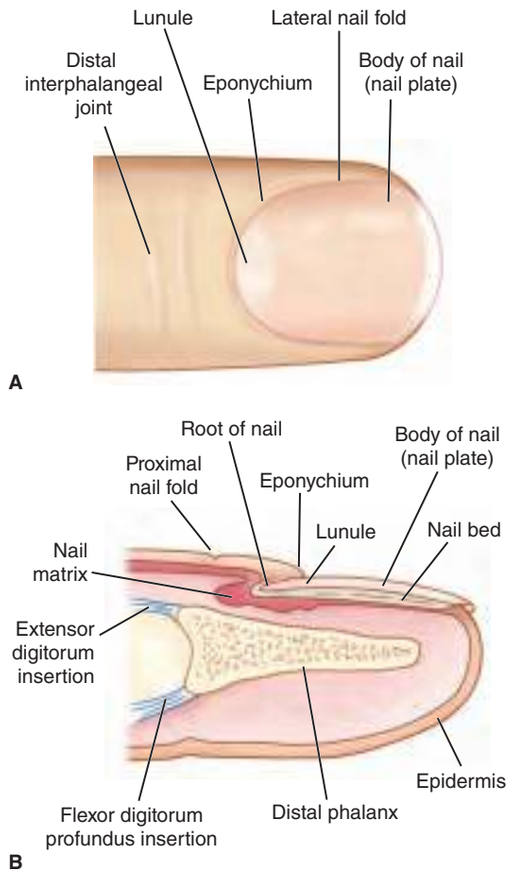


FIGURE 127-3. The anatomy of the distal fingertip and nail bed. ***A.*** Surface anatomy. ***B.*** Midsagittal view.

lunula to the hyponychium.[4] It was originally thought that the germinal matrix is primarily responsible for the growth of the nail, with a significant contribution from the sterile matrix.[5,6] It is now known that the entire nail bed contributes to the growth and migration of the nail plate.[4] The nail bed must be smooth for normal nail growth. A nail matrix that has not been well approximated to minimize scar formation may develop a deformed nail.[3-9] Trauma to the matrix can cause nail deformities, leading to poor cosmetic and functional outcomes.

The nail bed receives its blood supply from terminal branches of the proper palmar or volar digital artery, which communicate to form blood sinuses.[4] Venous drainage involves a combination of the superficial and deep venous systems that begin at the proximal portion of the nail bed and the skin proximal to the nail fold.[5] Injuries to the distal ends of the fingers and toes disrupt the vascular structures and cause compression of the capillaries located between the distal phalanx and the nail plate (**Figures 127-1 and 127-2**). The blood collection that results is known as a subungual hematoma. **The hematoma appears as a black-and-blue or black-and-purple area under the nail that is extremely tender to palpation.**

Nailbed injuries may be classified as avulsions, crush injuries, lacerations, and stellate lacerations.[1,6] Each of these types of nail bed injuries may result in a subungual hematoma. It is important to recognize how painful subungual hematomas are due to the pressure from the hematoma. The injury also causes pain.

The management of subungual hematomas is still somewhat controversial. The approach to management was initially very aggressive, since subungual hematomas are often seen with fractures of the distal phalanx, damage to the nail, and damage to the

nail apparatus.[1,2,5-9] The surgical literature generally recommends removal of the nail plate, inspection of the nail bed, and repair of any nail bed injury if the subungual hematoma involves 25% or more of the nail surface.[1,2,5-10] This practice has been questioned by controlled studies that demonstrated excellent outcomes in patients with large (i.e., greater than 25%) subungual hematomas treated by trephination alone, regardless of the presence of fractures.[6,10-13] Larger hematomas involving over 50% of the nail surface may be treated successfully with trephination. Many authors, primarily Hand Surgeons, still advocate the removal of the nail plate to thoroughly inspect the nail bed and effect repair in all patients who present with a subungual hematoma. Although this approach is time-honored, studies have demonstrated that nail plate removal is not necessary if it is still attached to the matrix, even in the presence of a distal phalanx fracture.[12,13] Trephination alone results in significant cost savings.[13,14]

PHYSICAL EXAMINATION

Thoroughly examine the injured digit prior to drainage of a subungual hematoma. Examination involves inspecting the hematoma to assess the approximate size or percentage of the nail involved. Assess the nail plate for any other injuries. Perform a complete neurologic exam including motor and sensory function. Assess extensor and flexor tendon function. Some patients with a nail bed injury may require a digital nerve block prior to examination. This will blunt the motor and sensory assessments. Capillary refill time can be used to determine vascular function in the injured digit.

IMAGING

The utility of imaging in patients with subungual hematomas is controversial. Some studies advise a complete set of radiographs to rule out foreign bodies and fractures. Other studies have shown that imaging can be avoided in patients with complete resolution of their pain with trephination alone. Most distal phalanx fractures have a low rate of complications, have a low yield for positive imaging, are nondisplaced, and are simple distal phalanx fractures. Using ultrasound to assess for significant soft tissue injuries or distal phalanx fractures is becoming more popular in the Emergency Department (Chapter 100). The use of ultrasound may become a recommended form of imaging in the future.[15,16]

INDICATIONS

Patients who present to the Emergency Department after sustaining an injury with a resultant subungual hematoma will generally complain of severe pain. Trephination (i.e., the process of making a small hole in the nail plate to allow collected blood to drain) will result in significant pain relief for most patients.[2,10] Use trephination when the patient presents with a painful subungual hematoma and an intact (i.e., not fractured or avulsed) nail plate that is still attached to the matrix.[13,14,17,18] Treatment may be more complicated than simple trephination if the nail plate is partially or completely avulsed from the matrix.[13,16,18] Trephination can be avoided in favor of conservative management with a protective splint and analgesia if the hematoma is not causing the patient significant pain or discomfort.

CONTRAINDICATIONS

Simple trephination is reserved for patients with intact nails and nondisplaced distal phalanx fractures. **Patients who present with nail plate fractures, avulsions of the nail plate, disruption of the nail margin, or partial amputations may require more extensive therapy with removal of the nail plate and repair of the nail bed (Chapter 129).**[13] Avoid trephination using heat-based methods in patients wearing artificial nails due to the potential for igniting the nail or nail adhesive.[20] **Subungual hematomas that extend proximal to the nail bed often represent proximal nail plate avulsions or injuries that require nail plate removal, nail plate repair or reinsertion, and nail bed repair.**

The lack of trauma is a concern if the patient has no recollection of trauma to the digit. The subungual hematoma is usually the result of a traumatic injury. A spontaneous subungual hematoma can be seen with subungual Kaposi's sarcoma, subungual melanoma, and other systemic diseases. Refer all spontaneous subungual hematomas to a Dermatologist.

EQUIPMENT

DIGITAL OR METACARPAL BLOCK

- Povidone iodine or chlorhexidine solution, or an isopropyl alcohol pad
- 3 mL syringe
- 25 or 27 gauge needle, 1 inch long
- Local anesthetic solution without epinephrine

ELECTROCAUTERY

- Povidone iodine or chlorhexidine solution, or an isopropyl alcohol pad
- Battery-powered electrocautery device

PAPER CLIP TECHNIQUE

- Povidone iodine or chlorhexidine solution, or an isopropyl alcohol pad
- Heat source (i.e., open flame)
- Paper clip or safety pin
- Hemostat

DRILL TECHNIQUE

- Povidone iodine or chlorhexidine solution, or an isopropyl alcohol pad
- 18 gauge needle
- Cotton-tipped applicators

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Consider obtaining radiographs for significant traumatic injuries, suspected associated fractures, or any injury where the nail plate is damaged or avulsed. Place the patient in a sitting or semirecumbent position on a gurney or multipositional procedure chair. Sit facing the patient.

Place the hand with the injured digit palm side down on a flat surface. Cleanse the injured digit of any blood, dirt, and debris. Apply povidone iodine or chlorhexidine solution over the nail plate and allow it to dry. An alcohol swab is an alternative to these solutions. **Always allow the alcohol to dry before touching the nail plate with a hot object so that the alcohol does not ignite.** Copiously irrigate the affected digit to remove any debris and reduce the likelihood of an infection. Studies have shown that copious irrigation has more success in preventing infection than prophylactic antibiotics.[21]

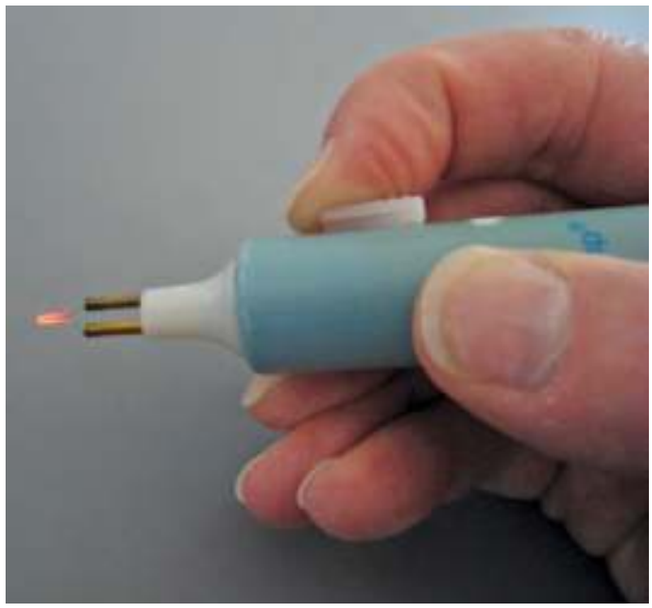


FIGURE 127-4. The battery-operated electrocautery device.

A digital block is generally not needed if using heat-based methods to penetrate the nail plate.[2,6,10,13,14,20,22] The nail plate is not innervated and the hematoma prevents contact with the nail bed. A digital block may be required if the patient is excessively anxious, if additional injury is present, or if a drill technique is to be performed (Chapter 156). An alternative to the digital block is to soak the affected finger in ice water for a few minutes prior to the procedure. The techniques described below may be used on fingernails and toenails.

TECHNIQUES

ELECTROCAUTERY TECHNIQUE

Electrocautery is an efficient technique to drain a subungual hematoma. Battery-operated microcautery devices are generally available in the Emergency Department (**Figure 127-4**). **Assure the patient that they will not be burned.** Stabilize the injured digit proximally with the nondominant hand. Grasp the cautery unit like a pencil with the dominant hand. Press the button on the cautery unit to heat the tip. Release the button and allow the tip to cool for 1 to 2 seconds. This may prevent penetrating the nail plate too quickly and damaging the underlying nail bed. Place the hot tip on the nail plate, centered over the hematoma (**Figure 127-5**). **Tap the nail plate several times with the cautery pen tip. Do not constantly hold the hot tip against the nail plate.** The cautery tip will easily penetrate the nail plate and a "give" is typically felt. **Do not plunge the cautery pen tip into the subungual hematoma to avoid injuring the nail bed and/or causing additional pain.**

Darkened blood will flow out of the hole when the hematoma is entered. The nail will regain its normal color after the hematoma is drained. Apply slight digital pressure to the nail plate to ensure complete drainage of the hematoma. "Milk" the blood out the hole(s) made in the nail plate. The patient will usually begin to feel pain relief at this point.

The tips of some microcautery devices are shaped in such a way that they will not make a hole that is wide enough to allow adequate drainage. Slightly rotate the cautery unit as it traverses the nail plate to ensure an adequate sized drainage hole of 3 to 4 mm. Some physicians prefer to make an additional hole in the nail plate to ensure drainage if the first hole becomes occluded. As an alternative to the single large hole, place three to four smaller drainage holes in the nail plate.

A

B

FIGURE 127-5. The electrocautery technique. The hot tip of the unit is centered over the subungual hematoma and allowed to penetrate the nail plate. ***A.*** Artist illustration. ***B.*** Using the device on a patient. (Used with permission from Shah BR, et al: *Atlas of Pediatric Emergency Medicine*, 2nd ed. New York: McGraw-Hill; 2013.)

PAPER CLIP TECHNIQUE

This technique is similar to using the electrocautery unit. Unfold and heat the tip of a paper clip or safety pin with a flame from a lighter or alcohol lamp. **Ensure that any alcohol used to clean the nail plate completely dries before attempting the paper clip technique.** Place the heated tip of the paper clip against the nail plate and centered over the subungual hematoma (**Figure 127-6**). Apply slight downward pressure to allow the heated paper clip to perforate the nail plate. **Do not plunge the cautery pen tip into the subungual hematoma to avoid injuring the nail bed and/or causing additional pain to the patient.** A drop of blood is seen when the paper clip penetrates the nail plate and enters the hematoma. Place at least one additional hole in the nail plate to ensure drainage if the first hole becomes occluded.

The paper clip will not get as hot as a cautery device. More than one attempt may be necessary to penetrate a thick nail. This will generally require reheating of the paper clip. Another disadvantage of this technique is the possibility of introducing carbonaceous

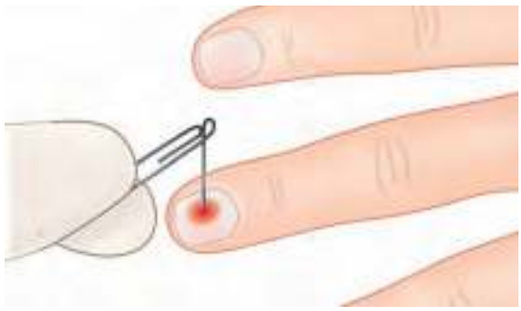


FIGURE 127-6. The paper clip technique. The hot tip of the paper clip is centered over the subungual hematoma and allowed to penetrate the nail plate.

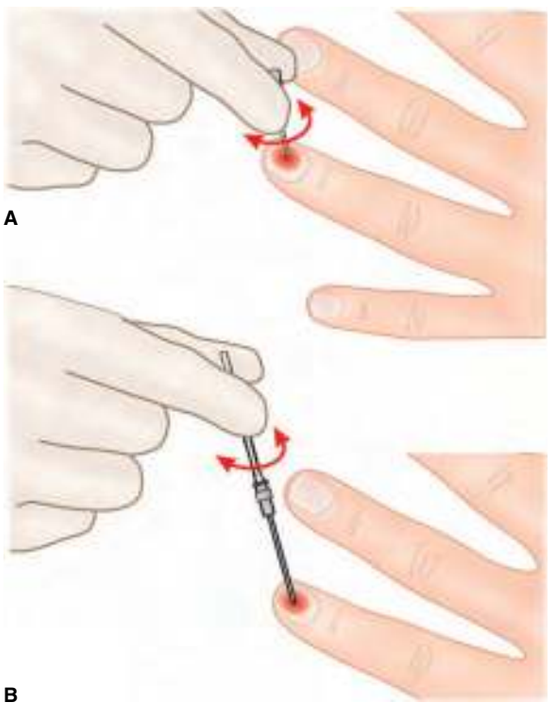
material into the hole. The use of an open flame may be potentially dangerous or prohibited in the Emergency Department.

DRILL TECHNIQUE

This technique uses a needle as a small drill to penetrate the nail plate (**Figure 127-7**). Small electric nail drills are available that greatly simplify this procedure, although they may not be readily available in the Emergency Department. Drilling through the nail plate may require a digital block. This is particularly true if there is a fracture, another associated injury, or the nail plate is very thick (i.e., toenails). Needles of various sizes can be used for this technique. There is typically a trade-off between the gauge of the needle and the bevel length. This can affect the ease with which the nail plate is penetrated. Most studies used an 18 gauge needle.

Grasp an 18 gauge needle by its hub with the dominant thumb and forefinger (**Figure 127-7*A***). Place the tip of the needle over the nail plate and centered over the subungual hematoma. Spin the needle back and forth while applying gentle downward pressure. Small shavings will appear as the needle begins to drill through the nail plate. A loss of resistance will be felt as the hematoma is entered and darkened blood will flow from the hole. **Do not plunge as the nail bed can be injured in addition to causing additional pain.** Place at least one additional hole in the nail plate to ensure drainage if the first hole becomes occluded.

The editor uses a modified version of this technique (Reichman, personal communication). A cotton-tipped applicator may be wedged into the needle hub to facilitate the drilling (**Figure 127-7*B***). This method makes the drilling more efficient. The cotton-tipped applicator is easier to hold and offers a mechanical advantage when it is twisted. The drill technique is very useful in the absence of a heat-generating device.



A

B

FIGURE 127-7. Drill techniques. ***A.*** A twisting motion of the 18 gauge needle is used to penetrate the nail plate. ***B.*** A cotton-tipped applicator has been inserted into the hub of the needle. A twisting motion is used to penetrate the nail plate.

ALTERNATIVE TECHNIQUES

The PathFormer (Path Scientific, Carlisle, MA) is a U.S. Food and Drug Administration (FDA)-approved device for nail plate trephination (**Figure 127-8**).[18] It allows for the precise control of the depth of penetration while making a 400 μm diameter hole in the nail plate. The device is a battery-powered drill that automatically retracts the drill bit after the nail plate is penetrated. The sensitive and vascular nail bed is not contacted or injured. The electrical impedance triggers a feedback and the rapid retraction of the cutter when it penetrates the nail plate and enters the hematoma. The need for anesthesia is eliminated. The PathFormer device is not often available in Emergency Departments.

A

B

FIGURE 127-8. The PathFormer. ***A.*** The device. ***B.*** Three holes made in the nail plate with the device. (Courtesy of Path Scientific LLC, Carlisle, MA.)

Insulin needles can be used to drain a subungual hematoma.[15] Insert the insulin needle underneath the nail plate and parallel to the hyponychium. Move the needle in a back-and-forth motion to evacuate the hematoma. This technique has been shown to cause little to no pain when compared with more traditional methods. It is limited to subungual hematomas that are near the free edge of the nail plate. This technique requires further studies of its safety and utility before it can be used in the Emergency Department.

Other devices have been used to drain a subungual hematoma. Battery-operated FDA-approved nail trephination devices are available for commercial use. Carbon dioxide lasers can be used to drain subungual hematomas. The laser beam is directed toward the center of the hematoma and results in quick and efficient drainage. The laser can be used without anesthesia because there is no contact with the nail bed itself.[24] These devices are not generally available in Emergency Departments.

ASSESSMENT

Squeeze the nail plate to evacuate the hematoma. Evaluate and manage any underlying injuries.

AFTERCARE

Inform the patient to keep the wound clean and monitor drainage. The nail plate may be covered with a nonadherent dressing. The hematoma may continue to drain for several hours or up to 1 to 2 days. The nail can be soaked in warm water and pressure applied to express the hematoma if there is a reaccumulation denoted by the reappearance of darkened blood beneath the nail plate. Inform the patient that the discoloration under the nail plate can persist for several weeks. Inform the patient that the nail plate may fall off and it can take up to 3 months for another nail plate to completely form. Apply a splint if a distal phalanx fracture is present. No studies have demonstrated that prophylactic antibiotics are beneficial in the management of a subungual hematoma.[10,25-27] The patient should immediately return to the Emergency Department or their primary physician for fever, increased pain, purulent drainage from the nail, or any redness or swelling of the digit.

COMPLICATIONS

Direct complications from nail trephination are rare.[12-14,17,20-22,25,28] Complications will more likely result from the original injury and include cosmetic deformities, infection, nail deformities, and nail loss.[1] Warn patients that loss of the nail is a possibility as the nail grows out. The rate of nail plate growth significantly slows for approximately 3 weeks after trauma before rapidly accelerating. This can cause a hypertrophic line to form along the nail plate.[1] Inform the patients that this is a possibility. Hypertrophic lines typically grow out with no residual cosmetic deformities. The hematoma may reaccumulate if the hole in the nail is too small and becomes occluded. Reaccumulation can be prevented by making a large hole or multiple holes in the nail plate. Plunging through the nail plate with a cautery unit or needle will cause the patient pain and injury to the nail bed that may be permanent and result in a deformed nail plate.

SUMMARY

Distal digit injuries are common. Patients will often present to the Emergency Department with a subungual hematoma and a complaint of pain. Rapid relief of pain, good cosmetic outcomes, and good functional outcomes can be obtained in most cases by simply performing a nail trephination. It is important to distinguish when trephination alone will not be adequate therapy. Patients presenting with nail plate fractures, avulsions of the nail plate, or partial finger amputations will require removal of the nail plate and repair of the nail bed.

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128 Subungual Foreign Body Removal

Ginger Clinton, Ameera Haamid, and Steven H. Bowman

INTRODUCTION

Subungual foreign bodies are often difficult to treat. Foreign bodies (e.g., hair, metal splinters, pencil lead, spines, thorns, and wood) may become lodged beneath the fingernail.[1-3] Tradesmen (e.g., auto mechanics, carpenters, landscapers, and metal workers) who work without hand protection with materials that produce small splinters are at risk for this type of injury. Subungual foreign bodies may present less commonly under the toenails.

Patients generally present for medical intervention with pain under the nail after unsuccessfully attempting to remove the foreign body. Prior removal attempts often result in breakage of the foreign body or pushing it further beneath the nail. This can complicate the next extraction attempt. Retained subungual foreign bodies if untreated can become infected, cause tissue reactions, and cause granuloma formation. Subungual foreign bodies can be treated rapidly with complete removal of the foreign body and without causing additional patient discomfort.[4]

ANATOMY AND PATHOPHYSIOLOGY

The distal fingertip and nail apparatus are complex structures (**Figure 128-1**). The perionychium is composed of the nail bed and the surrounding soft tissue. The hyponychium is the junction

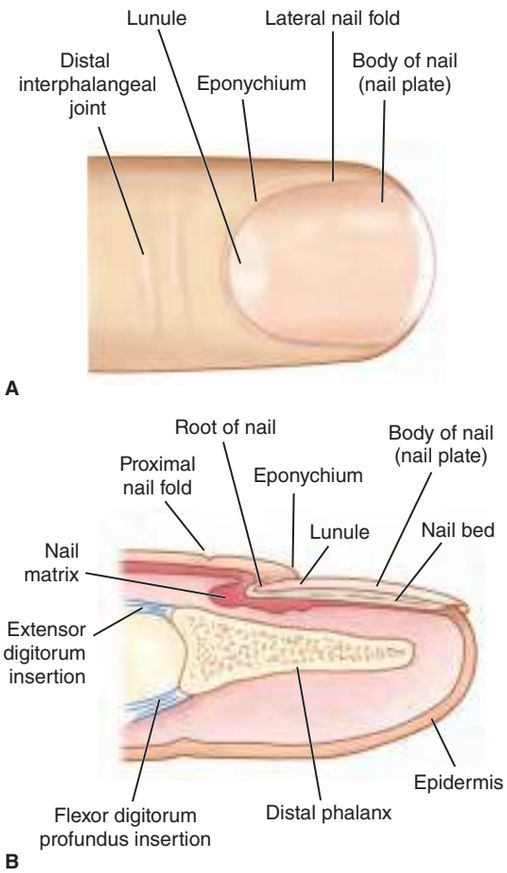

FIGURE 128-1. The anatomy of the distal fingertip and nail bed. ***A.*** Surface anatomy. ***B.*** Midsagittal view.

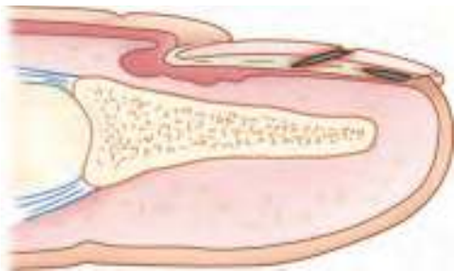

FIGURE 128-2. Subungual foreign bodies can enter from under the distal nail plate or through the nail plate.

of the nail bed at the sterile matrix and the fingertip skin beneath the distal margin of the nail plate. The eponychium is the distal portion of the nail fold where it attaches to the proximal surface of the nail plate. The lunula is the white arc seen on the proximal portion of the nail plate. The nail bed consists of the germinal matrix on the proximal ventral floor of the nail fold and the sterile matrix that extends from the lunula to the hyponychium. The germinal matrix is primarily responsible for the growth of the nail. The subungual space is the area immediately beneath the nail plate.

Foreign bodies may enter the subungual space at the distal fingertip beneath the nail or may penetrate the nail plate (**Figure 128-2**). Separation of the nail from the nail bed results in severe pain. Patients frequently attempt to remove the foreign body immediately because of this intense discomfort. An infection or foreign body reaction will often ensue if the foreign body is not removed in its entirety.

INDICATIONS

Subungual foreign bodies should be removed to prevent the complications of foreign body reactions, infection, and possible nail deformity. Deeply embedded foreign bodies, splintered foreign bodies, those that traverse the nail plate, or contaminated foreign bodies may require the removal of the nail plate to extract the foreign body (Chapter 129). Consult a Hand Surgeon if the foreign body cannot be removed, if the site is infected, if the foreign body is "chronic," if an osteomyelitis is present on radiographs, or if significant injury to the digit is present.

CONTRAINDICATIONS

There are no absolute contraindications to the removal of a subungual foreign body.

EQUIPMENT

GENERAL SUPPLIES

- Povidone iodine or chlorhexidine solution
- Alcohol swabs
- Sterile saline solution
- Topical antibiotic ointment
- Nonadherent dressing (e.g., petrolatum gauze)
- 4×4 gauze squares
- Adhesive tape

DIGITAL OR METACARPAL BLOCK

- 3 mL syringe
- 25 or 27 gauge needle, 1 inch long
- 3 to 5 mL local anesthetic solution without epinephrine

SCRAPE TECHNIQUE

- Splinter forceps
- #11 or #15 scalpel blade on a handle

WEDGE TECHNIQUE

- Splinter forceps
- Tissue scissors or nail clippers

NEEDLE TECHNIQUE

- Needles, 19 and 25 or 27 gauges
- Splinter forceps
- Hemostat

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Ascertain the patient's tetanus immune status and administer the appropriate tetanus prophylaxis if needed. Gain the patient's cooperation to make them more comfortable. Place the patient sitting or in a semi-recumbent position with their hand on a bedside procedure table.

Prep the finger. Clean any blood, dirt, or debris from the affected finger. Do this gently as any manipulation of the nail bed will result in additional patient discomfort. Apply povidone iodine or chlorhexidine solution and allow it to dry. Determine the need for a digital or metacarpal block based on the type of the foreign body, degree of embedment, and the chosen removal technique (Chapter 156).

Radiographs are not required unless a metallic foreign body cannot be visualized, the clinical suspicion exists for an osteomyelitis, or there is the suspicion for a gas forming finger infection. Ultrasound has been useful in visualizing the location and orientation of radiolucent foreign bodies.[5,6] It has been described for confirmation of complete foreign body removal.[5,6]

TECHNIQUES

The removal technique is chosen based on the location of the foreign body and Emergency Physician comfort. Foreign bodies protruding through or from underneath the nail plate can usually be grasped with forceps and removed whereas deeply located objects may require special techniques. A scalpel blade or 18 gauge needle may be used to entrap a small protruding tip of the foreign body against the nail plate and draw it out. **Do not attempt to remove the foreign body through a puncture wound or small incision.** Enlarging the access site allows for easier removal, not breaking or fragmenting the foreign body, and complete removal. Subungual foreign bodies may be removed by one of the following techniques.[4]

SCRAPE TECHNIQUE

This technique works well for foreign bodies that have traversed the nail plate, lodged beneath the distal nail, or lodged beneath the middle portion of the nail. This technique has been described anecdotally.[7,8] It appears promising as it does not require the administration of a digital block and causes less trauma to the nail and the nail bed compared to other techniques.

Support the patient's hand on a procedure table. Sit on a chair facing the patient. Place a #11 or #15 scalpel blade on the nail plate directly over the foreign body (**Figure 128-3*A***). Hold the blade perpendicular to the surface of the nail plate. Draw the scalpel blade

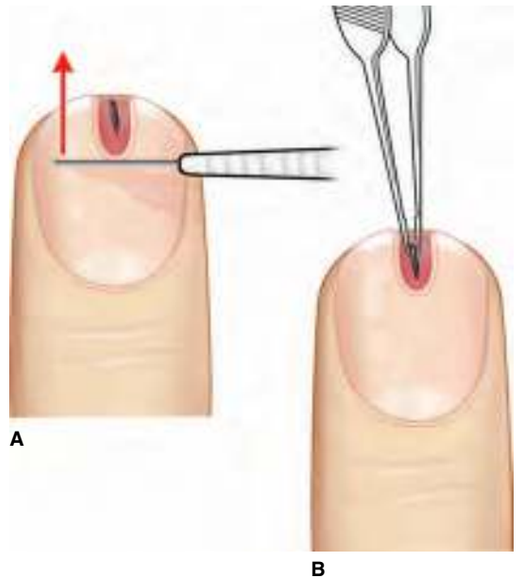

FIGURE 128-3. The scrape technique. ***A.*** Strokes are made in a proximal to distal direction with the scalpel blade held 90° to the nailbed. A U-shaped defect will be created to expose the foreign body. ***B.*** The foreign body is grasped and removed with forceps.

from proximal to distal using short strokes and gentle pressure over the foreign body (**Figure 128-3*A***). A small shaving of the nail plate is removed with each stroke of the scalpel blade. Soak the finger in lukewarm water for 15 to 20 minutes to soften the nail plate if it is thick and difficult to shave. Continue to remove successive slivers of the nail plate to eventually create a U-shaped defect and expose the foreign body (**Figure 128-3*B***). Grasp the foreign body with a splinter forceps and remove it once a significant portion protrudes (**Figure 128-3*B***). The defect created in the nail plate will move distally and eventually be replaced as the nail plate continues to grow.

WEDGE TECHNIQUE

The wedge technique works well for subungual foreign bodies lodged beneath the distal portion of the nail plate.[1,9] Patients will require a digital or metacarpal block prior to attempting this technique since it involves manipulation of the nail bed.

Sit on a chair facing the patient. Cut a triangular wedge from the distal portion of the nail plate overlying the foreign body with a small pair of tissue scissors or nail clippers (**Figure 128-4*A***). **Prevent iatrogenic injury to the nail bed by ensuring that the tip of the scissors under the nail plate is aimed upward and against the nail plate.** Remove the wedge of nail. This will provide enough exposure to grasp and remove the foreign body with splinter forceps (**Figure 128-4*B***).

NEEDLE TECHNIQUES

The needle technique works well for subungual foreign bodies located beneath the distal portion of the nail plate.[1] Patients will require a digital or metacarpal block prior to attempting this technique. Insertion of a needle into the nail bed is extremely painful. The major drawback of this technique is the potential for leaving fragments of the foreign body beneath the nail plate.

Sit in a chair facing the patient. Introduce a 19-gauge needle beneath the nail plate and along the track of the foreign body (**Figure 128-5*A***).[10] Use the tip of the needle to touch the foreign body. Lower the hub of the needle to raise its tip and trap the foreign body between it and the nail plate. Withdraw the needle

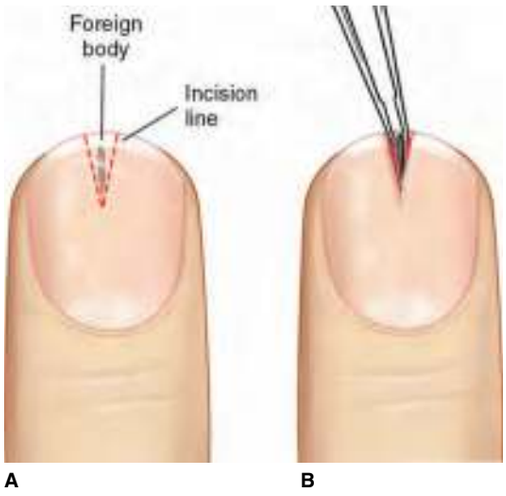

FIGURE 128-4. The wedge technique. ***A.*** A triangular incision is made in the nail plate overlying the foreign body. ***B.*** The cut section of the nail plate has been removed. The foreign body is grasped with forceps and removed.

while dragging the foreign body out along the nail plate. Alternatively, tease out and move the foreign body distally until it can be grasped with a splinter forceps.

Two alternate techniques have also been described for the needle extraction of a subungual foreign body.[10,11] The first is a modification of the needle technique.[7] Place a hook in the distal end of a 25 or 27 gauge needle with a hemostat or needle driver (**Figure 128-5*B***). Pass the bent needle along the foreign body tract. Grasp the foreign body with the tip of the bent needle. Withdraw the needle to move the foreign body distally so that it may be grasped with a splinter forceps. The second technique involves the excision of a small portion of the nail plate overlying the foreign body with an 18 gauge needle.[11] This is similar to the shave technique with the exception of an 18 gauge needle being used instead of a scalpel blade.

FIGURE 128-5. Needle techniques. ***A.*** A 19 gauge needle is inserted along the tract of the foreign body. The tip is used to tease the foreign body out of the tissues. ***B.*** The tip of a 25 or 27 gauge needle has been formed into a hook. The needle is inserted along the tract of the foreign body. The tip is used to grasp the foreign body and pull it out of the tissues.

ASSESSMENT

Inspect the subungual area for any remaining fragments of the foreign body that may have broken off in the nail bed. Ultrasound may aid in this evaluation. **Any remaining fragments of the foreign body must be removed as to not cause complications as previously described.** Refer the patient to a Hand Surgeon if the foreign body fragments cannot be removed.

AFTERCARE

Local wound care and the application of a topical antibiotic ointment are all that is required in most cases. Irrigate the foreign body tract and excision site with sterile saline. Apply topical antibiotic ointment to the area. Apply a nonadherent dressing over the nail. Follow-up with a Hand Surgeon and systemic antibiotics may be necessary in severe cases (e.g., the presence of a nail deformity, chronic foreign bodies with an infection, or inability of the Emergency Medicine physician to remove the entire foreign body). Manage postprocedural pain with acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs). The use of prophylactic antibiotics is not recommended unless the foreign body was contaminated or deeply penetrated the soft tissue of the digit. Instruct the patient to return immediately to the Emergency Department if they develop any signs of an infection (i.e., fever, increased tenderness, purulent drainage, redness of the digit, or swelling of the digit).

COMPLICATIONS

There are a few potential complications from subungual foreign body removal. Damage to the nail bed can result in a residual nail deformity. Failure to completely remove a subungual foreign body may result in a nail deformity, abnormal nail growth, an infection, or a foreign body reaction with granuloma formation. An infection can result from a contaminated foreign body, flora on the nail plate, skin driven into the soft tissues by the foreign body, or if sterile technique is not followed.

SUMMARY

Patients with subungual foreign bodies often present with severe pain within the nail after prior unsuccessful attempts at removal or after complications develop. It is important that the Emergency Physician completely remove the foreign material in the subungual space to prevent further complications. Providing adequate anesthesia, using appropriate instruments, and using appropriate techniques allow the successful removal of most subungual foreign bodies.

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129 Nail Bed Repair

Dayle Davenport

INTRODUCTION

The fingertip, the most sensitive area of the hand, is often our first contact with the environment. It has important functional roles in grasping and pinching, in addition to its sensory and cosmetic functions.[1,2] The fingernail protects the fingertip and provides increased sensation to the volar pulp.[3] However, it also conceals the true extent of fingertip injuries. Hand injuries, with the fingertips most frequently involved, are second only to back injuries as the most common occupational injuries resulting in loss of work days.[4] Hence, it is important for the Emergency Physician to evaluate the full extent of the injury, to assess potential disabilities, and to recognize the need for prompt referral to a Hand Surgeon.

The fingernails are frequently injured due to their anatomic location and their functional role. **Immediate primary repair is the ideal management when these injuries involve the nail bed and surrounding skin fold structures.[3,5,6] Careful repair is necessary to avoid functional impairment and cosmetic derangement of the nail plate.**[7] The following discussion will refer primarily to the fingernail. The toenail has less importance, both cosmetically and functionally, as grasp and pinch are not needed. However, all the principles and recommendations made also apply to the toenail.[8]

ANATOMY AND PATHOPHYSIOLOGY

Knowledge of the anatomy of the nail unit enables the Emergency Physician to recognize the types of injuries and provide anticipatory guidance of the consequences of these injuries to the patients. The "perionychium" or the nail unit consists of the nail fold, nail plate, nail bed, and hyponychium (**Figure 129-1**).[1,2]

The nail plate enhances the sensibility of the fingertip by applying a counterforce to the pulp space nerve endings.[3] The digital tip and the nail plate also function in unison to smoothly coordinate normal pinch and grasp which are important for picking up fine objects such as coins and pins.[3,9]

The nail plate is comprised of compacted, flattened, and elongated anucleated cells that originate from cornified epithelial cells.[10] There are three anatomic sites where these cells exist.[5,9] The nail bed contains two of the sites: the sterile matrix and the germinal matrix (**Figure 129-1*A***).[1,9] The other location is the dorsal roof matrix. Of these, the germinal matrix is the most important for normal nail growth.[5] The germinal matrix is responsible for approximately 90% of the nail plate by volume.[9] The sterile matrix is responsible for a small percentage of the nail plate by volume and varies in each individual. This cell production accounts for the nail plate being thicker at its distal tip compared to its proximal origin.[9] The nail cells from the dorsal roof matrix are small in number and form a very thin layer on the surface of the nail plate. These cells are responsible for the shine of the nail. The nail will lose its shine and become dull if the dorsal roof matrix cells are destroyed.

Skin overlies the nail plate proximally and laterally (**Figure 129-1*B***).[10] The proximal skin fold is referred to as the eponychium.

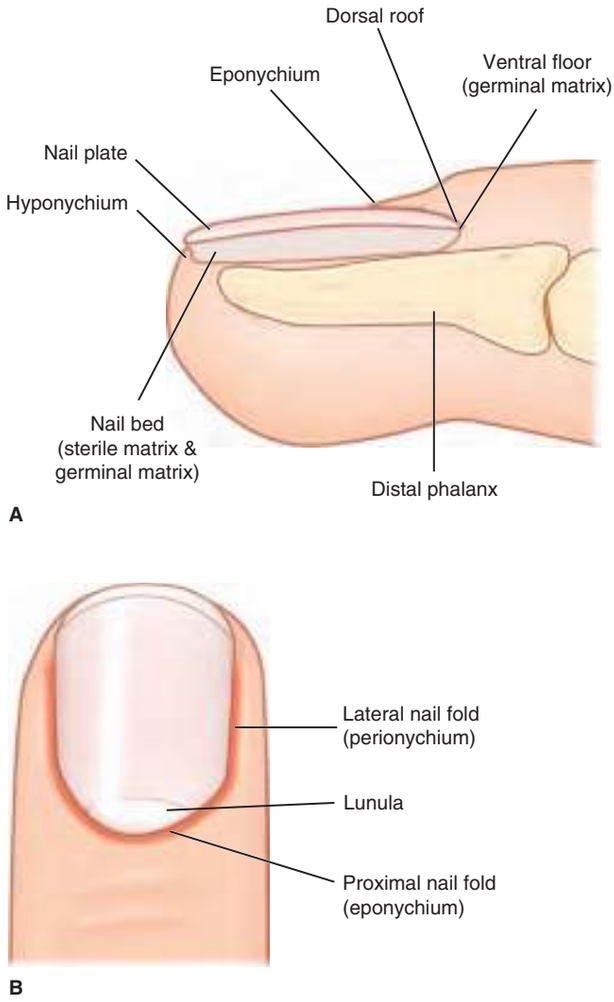

FIGURE 129-1. Anatomy of the fingernail. ***A.*** Lateral view. ***B.*** Top view. The colored area represents the perionychium.

The eponychium protects the germinal matrix located in the proximal nail bed and is the home of the dorsal roof matrix. The skin immediately over the dorsal roof is called the nail wall.[9] The lateral skin folds, the adjacent cutaneous areas, and the adjacent nail bed (i.e., germinal matrix and sterile matrix) are collectively referred to as the perionychium (**Figure 129-1*B***).[7,9,10] The lunula is the pale arc just distal to the eponychium and roughly corresponds to the location of the germinal matrix.[1,2,9,10]

The nail bed is composed of the germinal matrix proximally and the sterile matrix distally, with the junction of these two at the level of the lunula.[1,9] The borders of the nail bed are the proximal nail fold, the lateral nail folds, and the hyponychium distally. The hyponychium is the thick layer of cells at the junction of the distal nail bed (i.e., the sterile matrix) and the fingertip skin.[9] It is located just under the distal free margin of the nail plate (**Figure 129-1*A***). The hyponychium serves as a barrier preventing the delicate nail bed from exposure to bacteria and fungi.[9]

The rate of nail growth varies from finger to finger, from individual to individual, and varies with age.[3,9] Nail growth is fastest between 4 and 30 years of age and after 80 years of age. Fingernails grow four times faster than the toenails.[1] A new nail takes a minimum of 4 months to grow and even longer following an injury. Progression of the distal nail occurs at a rate of 0.1 mm/day or 0.5 to 1 mm/week.[3,10] This rate varies and is usually faster in fingers than

in toes and faster in the summer months.[3] The pressure of new cells being formed leads to the flattening and elongation of the older cells as well as their progression distally.[9]

The sterile matrix is more adherent to the nail plate than to the adjacent germinal matrix. Therefore, avulsions involving nail bed tissue are more likely to occur at the sterile matrix.[9,11,12] Conversely, the nail plate is loosely held to the germinal matrix. This accounts for the peculiar injury of avulsions of the proximal nail plate from the proximal nail fold while the distal nail plate remains attached (**Figure 129-2**).

The digital artery supplies the volar radial and ulnar sides of the finger. It consistently sends even smaller branches to the proximal nail fold and to the nail bed, producing a rich capillary network.[1] The small veins of the nail bed, pulp, and lateral nail folds coalesce proximally to form a larger commissural vein that runs lateral to the distal phalanx and a terminal vein that runs dorsal to the distal phalanx. The digital nerve accompanies the digital artery and sends branches beyond the distal interphalangeal joint and into the nail fold, nail bed, and finger pulp. The extensor tendon attaches itself to the distal phalanx just proximal to the germinal matrix.[9,12] The periosteum of the distal phalanx, in turn, closely adheres to the sterile matrix.[9]

Certain patterns emerge as a result of an injury. Avulsions of the nail bed tend to occur in the sterile matrix rather than the less adherent germinal matrix. Another pattern of injury seen is the avulsion of the proximal nail plate from the proximal nail fold while the distal part remains attached to the nail bed (**Figure 129-2**). Because the sterile matrix closely adheres to the periosteum of the distal phalanx, fractures of the distal phalanx might injure the nail bed, producing a subungual hematoma.[7] Due to the close proximity of the germinal matrix and the attachment of the extensor tendon, injuries to or repair of these structures might involve the other. A subungual hematoma is a collection of blood between the nail bed and the nail plate. Although controversial, a large subungual hematoma may require repair of the underlying nail bed.

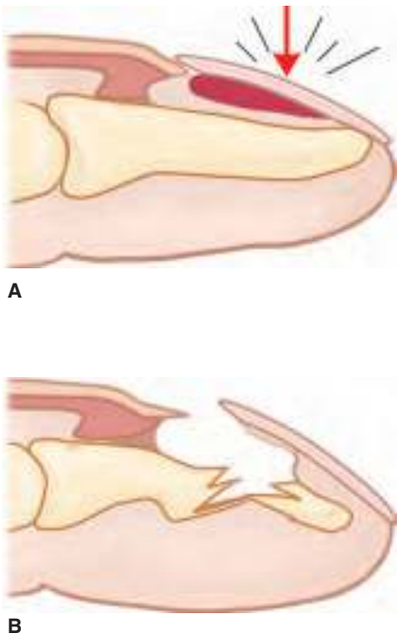

FIGURE 129-2. A crush injury to the distal fingertip. ***A.*** Proximal nail bed avulsion. ***B.*** A more severe crush injury can result in a proximal nail bed avulsion and fracture of the distal phalanx. Note that the distal nail plate remains attached to the sterile matrix in both of these injuries.

Emergency Physicians must consider in each case whether there exists enough damage to require surgical nail bed repair. It is important to note that significant force is required to break, penetrate, or avulse the nail plate.[7] Therefore, the fragile nail bed is most likely disrupted if the nail plate is disrupted.

INDICATIONS

Injuries to the fingertip and nail, if not initially managed correctly, have long-lasting functional and cosmetic consequences. The most important consideration is functional.[10] Normal nail growth after an injury requires a smooth nail bed. Therefore, nail bed injuries must be meticulously repaired to prevent cosmetic deformities and functional impairment.[10-12] Scar tissue may form between the wound edges if the nail bed is not accurately approximated. This scar tissue will not form the intermediate nail cells responsible for nail adherence.[9] Loss of the germinal matrix alone will result in permanent loss of the nail plate.[5,13] **The skin folds surrounding the nail margins must also be preserved.[5,12]** Failure to do so will result in the painful complication of adhesion formation between the skin fold and the nail bed.[5,12] Secondary repair of these spaces, or of the nail bed, requires more complex procedures and is usually associated with a poor outcome.[4,11] **Therefore, every effort should be made to primarily repair all significant nail bed injuries**.

CONTRAINDICATIONS

No absolute contraindications exist for primary nail bed repair. Any life-threatening injuries, limb-threatening injuries, and/or uncontrolled hemorrhage must be addressed prior to nail bed repair.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Restraining device, as necessary
- 10 mL syringe armed with a 27 gauge needle
- Local anesthetic solution without epinephrine (1% lidocaine or 0.25% bupivacaine)
- Digital tourniquet
- #15 scalpel blade on a handle
- Magnification device
- Sterile prefabricated nail, if available
- Nonadherent petrolatum gauze
- 5–0, 6–0, and 7–0 chromic gut (or polyglactin 910 or irradiated polyglactin 910) on a P-3 cutting needle
- 6–0 monofilament nylon sutures
- 2-Otylcyanoacrylate (e.g., Dermabond) or another tissue adhesive
- Needle driver
- Curved hemostat
- Fine scissors or periosteal elevator
- Forceps
- Sterile towels or drapes
- Sterile gloves
- Dry gauze or tube gauze
- Splinting material
- Battery-powered electrocautery device

The use of a digital tourniquet is essential to properly repair a nail bed. Its use provides a bloodless field. This allows for a more meticulous and precise wound approximation. Complications associated

FIGURE 129-3. A sterile Penrose drain or IV tourniquet used as a digital tourniquet.

FIGURE 129-5. The Tourni-cot digital tourniquet.

with their use includes neurovascular injury secondary to excessive tourniquet pressure and digital ischemia caused by a forgotten tourniquet. Numerous options are available to use as a tourniquet in the Emergency Department. These include the use of a sterile Penrose drain, rubber intravenous (IV) tourniquet, or sterile glove. A sterile Penrose drain or IV tourniquet secured with a hemostat is often used as these items are readily available (**Figure 129-3**). A sterile glove may be used as a tourniquet (**Figure 129-4**). Apply a sterile glove onto the patient's hand that is snug or a size one-half smaller than their actual glove size. Cut just the tip off the glove over the affected finger(s). Roll the cut finger back and onto the proximal phalanx to form the tourniquet (**Figure 129-4**).

Several commercially produced digital tourniquets are often available in the Emergency Department. The Tourni-cot (Mar Med Co., Grand Rapids, MI) is a sterile, single-use, disposable, rubber ring that slides over the fingertip and is rolled backward until it rests over the proximal phalanx (**Figure 129-5**). These come in several sizes to fit the appropriate finger size. The advantage of the Tourni-cot is that it exsanguinates the digit as it is applied. The T-Ring (Precision Medical Devices LLC, San Clemente, CA) is a "one size fits all" digital tourniquet (**Figure 129-6**). It is a sterile, single-use, disposable, rubber diaphragm with a central hole. It slides over the fingertip and is pushed backward until it rests over the proximal phalanx. The T-Ring also exsanguinates the digit as it is applied. The Tourni-cot and T-ring are superior to the glove and Penrose drain in providing hemostasis with the least amount of pressure.[14]

PATIENT PREPARATION

The first step in the evaluation of fingertip injuries is obtaining a thorough history.[15] The mechanism of injury can provide some clues to the type and extent of injuries as well as amount of contamination to be expected. Other pertinent information includes the patient's age, hand dominance, occupation, hobbies, comorbidities, and tetanus vaccination status. All significant injuries to the fingertip should be evaluated radiographically for fractures. The management of fingertip injuries may differ in the presence of a fracture. Thoroughly evaluate and document a complete neurovascular examination, tendon function and intactness, and ligamentous stability of the joints. Administer tetanus immune prophylaxis if required.

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. **It is important to explain both the risks of doing the procedure and of not doing the procedure.** Document this conversation and obtain an informed consent. Place the patient on a gurney with the hand on a bedside procedure table in a well-lit area. Be prepared to use age-appropriate

FIGURE 129-4. A sterile glove used as a digital tourniquet.

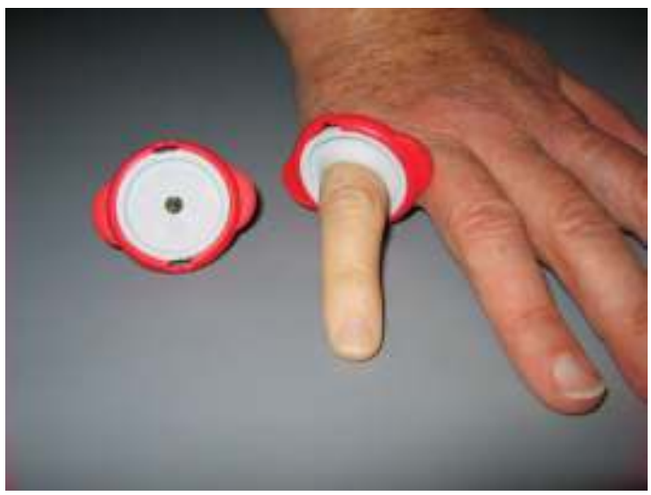

FIGURE 129-6. The T-Ring digital tourniquet.

sedation or to apply appropriate restraints for children.[12] Procedural sedation may be necessary in patients who are agitated, uncooperative, or require extensive repair. Apply a type of magnification device if available. This may consist of a head-strap device, a swing arm device, magnification glasses, or loupe magnification glasses.

Anesthetize the injured digit(s) involved (Chapter 156). Anesthesia may be achieved with a digital block or a metacarpal block when only a single digit is involved.[7,15] Lidocaine (1% or 2%) can provide pain relief of up to 3 hours. For longer analgesia, use 0.5% bupivacaine or lidocaine with epinephrine, with multiple studies showing that the risk of digital necrosis is minimal.[16-19] Metacarpal blocks or an axillary nerve block may be performed if multiple digits are involved.[7]

Thoroughly clean the hand of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry.[20] Irrigate the wound with sterile saline. If the nail plate has been avulsed, irrigate it with a dilute povidone iodine or chlorhexidine solution followed by a gentle rinse with sterile saline. **It is important not to scrub the undersurface of the avulsed nail plate because adherent squamous tissue may be destroyed.**[7]

A bloodless field is desired and, in many cases, necessary. Apply a digital tourniquet as described in the equipment section above.[7] If available, a pneumatic tourniquet may be placed on the arm instead of using the digital tourniquet. The pneumatic tourniquet is especially helpful when the patient has fractures or lacerations of the proximal digit that preclude the use of a digital tourniquet. **Avoid using a blood pressure cuff as these tend to deflate during use. As with all tourniquets, limit the amount of time in which the tourniquet is in place**. Create a sterile field by applying sterile towels.

TECHNIQUES

Adhering to certain principles will improve the outcome when repairing nail beds. A smooth and flat nail bed is necessary to the normal growth of the nail and should be the primary goal in any repair. Avoid or severely limit the amount of debridement.[5] The germinal matrix must be meticulously repaired and the proximal nail fold (i.e., the eponychium) preserved or that space is obliterated within a few days and can result in adhesions or abnormal nail growth.[5] Thoroughly clean and replace the nail plate whenever possible. This will preserve the nail folds surrounding the nail bed, allow the nail plate to serve as a splint for fractures, and act as a protective cover for the healing nail bed.[7] Treatment goals also include preservation of length and sensation of the fingertip, early mobilization, prevention of joint contractures, and attention to cosmesis.[4,21]

The technique of nail bed repair depends on the type of injury as well as which structures are involved. Various classification schemes, such as the nature of injury or anatomic location, have been developed to categorize fingertip injuries and guide treatment. Nail bed injuries are classified as simple lacerations, crushing lacerations, avulsion-lacerations, lacerations with associated fractures, lacerations with loss of skin and pulp, and fingertip amputations.[2,12]

NAIL PLATE REMOVAL

A significant force is required to break the nail plate. **The nail plate should be removed to visualize the underlying nail bed if the nail plate is damaged or avulsed or if the lateral skin folds are lacerated, as this makes an associated nail bed injury highly likely.** Removal of the nail plate is unnecessary in minor injuries where the nail plate is not damaged and still attached to the nail folds.

Remove the nail plate to repair nail bed injuries or to inspect the nail bed for potential injuries (**Figure 129-7**). Insert the closed tips of a fine scissors between the nail bed and the nail plate. A periosteal elevator, if available, can be substituted for the scissors. Hold the scissors parallel to the long axis of the finger. Slightly angle the tips of the scissors toward the nail plate to prevent any damage to the nail bed.[12] Advance the tips of the scissors in 1 to 2 mm increments. Open the blades of the scissors to separate the nail bed from the nail plate. Close the blades of the scissors. Continue to advance the tips of the scissors in 1 to 2 mm increments and separate the nail bed from the nail plate. Stop advancing the scissors when the tips of the blades are at the level of the eponychium. Firmly grasp the nail plate with a hemostat. Pull the nail plate parallel to the long axis of the finger to completely remove it from the finger.

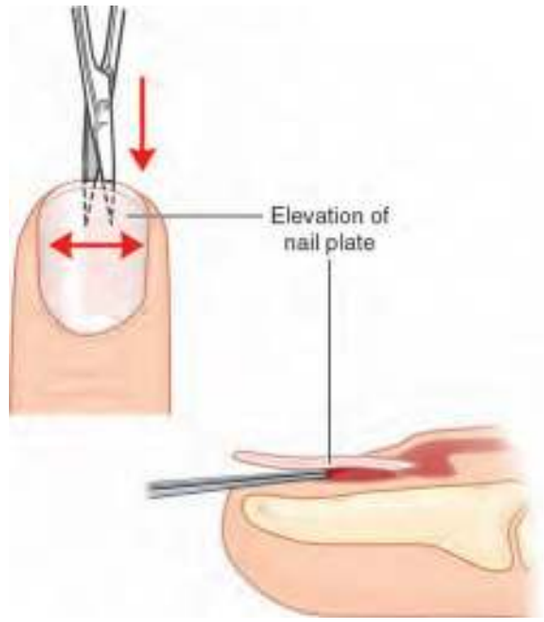

FIGURE 129-7. Removal of the nail plate. Dissect along the plane between the nail plate and the nail bed using a pair of fine scissors or periosteal elevator.

Making two linear incisions with a scalpel at 90° from the eponychium edge will allow for greater exposure to the germinal matrix for the repair (**Figure 129-8**).[8] This allows the eponychium to be folded or sutured back and therefore increases the exposure of the germinal matrix.[2,21]

SIMPLE LACERATIONS

Simple lacerations are caused by localized blows to the nail plate. After removing the nail plate and exposing the nail bed, repair the laceration meticulously using 6–0 or 7–0 chromic gut or irradiated polyglactin 910 sutures.[2,7,21] Alternatively, 2-otylcyanoacrylate (e.g., Dermabond) can be used and has no difference in cosmesis, pain, or functional ability and was significantly faster than suturing.[22,23] **Minimize debridement as much as possible to avoid scarring that**

FIGURE 129-8. Incisions can be made at 90° to the corners of the eponychium for better exposure of the germinal matrix.

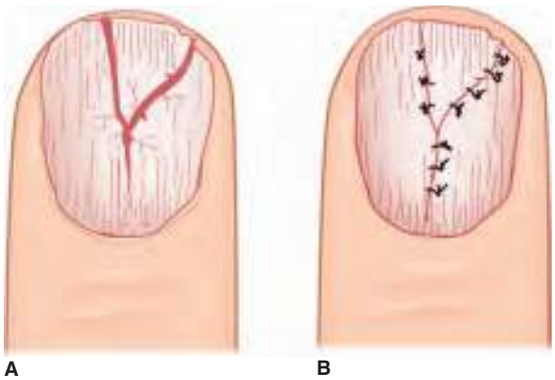

FIGURE 129-9. Crushing lacerations to the nail bed. ***A.*** Stellate or complex lacerations of the nail bed can occur after a crush injury. ***B.*** Approximation of the nail bed with fine absorbable sutures.

will result in nonadherence or splitting of the nail plate. Repair any skin lacerations adjacent to the nail bed using 6–0 or 5–0 monofilament nylon sutures.[5] Nail fold lacerations may require repair in layers in order to preserve these spaces.[10]

CRUSHING LACERATIONS

The second type of injury is a crush injury with resultant lacerations. Crushing injuries may produce stellate lacerations and fragmentation of the nail bed (**Figure 129-9*A***). Attempt to meticulously repair the fragmented nail bed using 6–0 or 7–0 chromic gut, irradiated polyglactin 910 sutures, or tissue adhesive to achieve precise approximation and the smoothest result possible (**Figure 129-9*B***).[7,22,23]

AVULSION-LACERATIONS

The third type of nail bed injury is the avulsion-laceration (**Figure 129-10*A***). These injuries are more complex and sometimes the fingernail is too damaged to be repositioned. In these cases, a nail substitute should be used to protect the fingernail during the healing process and to avoid adherences along the proximal nail bed and nail fold.[24] Distal nail bed avulsions simply require petrolatum gauze to be placed over the injury followed by sterile gauze. Suture the petrolatum gauze and sterile gauze in place using 5–0 or 6–0 nylon suture for 10 days to allow the wound to heal by second intention (**Figure 129-10*B***).[5,21] Avulsion-laceration injuries may require consultation with a Hand Surgeon depending on the amount of tissue involved and whether the germinal matrix is involved.[7,12,13] More severely damaged nail beds with a large amount of avulsed tissue usually require dermal grafts or split-thickness matrix grafts.[7]

Small fragments of avulsed nail bed may remain attached to the nail plate. If only the sterile matrix is damaged, the nail should be left attached proximally in the nail fold.[25] These may be simply treated by carefully replacing the nail plate in its anatomic position. Larger segments of avulsed nail bed that remain attached to the nail plate should be repaired (**Figures 129-11*A* and 129-11*B***). Gently shave away the nail bed from the nail plate with a #15 scalpel blade (**Figure 129-11*C***). Replace the avulsed nail bed and suture it in place with 5–0 or 6–0 chromic gut, irradiated polyglactin 910 sutures, or tissue adhesive (**Figure 129-11*D***).[11,22,23] Apply a petrolatum gauze dressing and replace the nail plate.

A special type of avulsion injury occurs when there is a crush injury to the distal fingernail. This results in an avulsion of the proximal nail plate with or without involvement of the germinal matrix (**Figure 129-2**). The proximal nail plate is less adherent to the nail bed and can be pulled out from under the eponychium (**Figure 129-2**). Remove the nail plate if the proximal nail plate is avulsed without

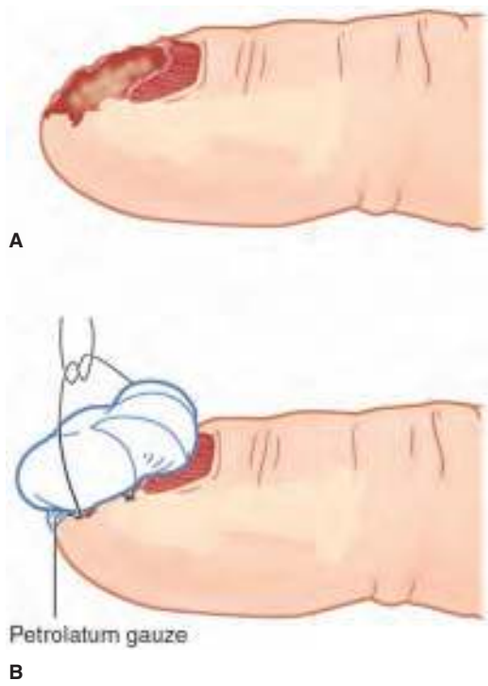

FIGURE 129-10. Avulsion-laceration of the nail bed. ***A.*** The dorsal aspect of the fingertip is avulsed with the nail plate. ***B.*** Petrolatum gauze is placed over the injury. This is subsequently covered with sterile gauze and sutured into place for 10 days.

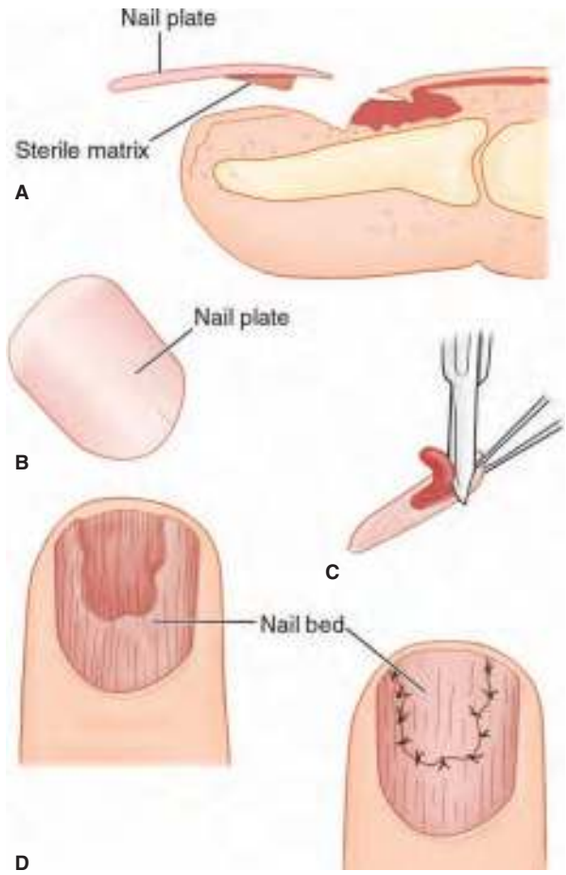

FIGURE 129-11. Large avulsion of the nail bed that is adherent to the nail plate. ***A.*** Lateral view of injury. ***B.*** Top view of injury. ***C.*** Gently shave away the avulsed segment. ***D.*** Repair the nail bed using the avulsed segment.

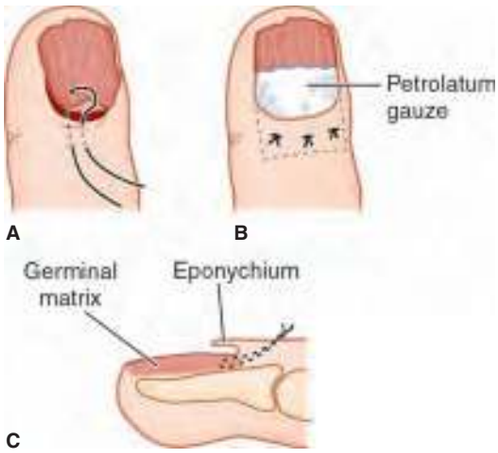

FIGURE 129-12. Repair of the germinal matrix after an avulsion. ***A.*** A series of horizontal mattress sutures are placed. ***B.*** The nail bed is returned to its proper location and the sutures tied. ***C.*** Lateral view of the repair.

involvement of the germinal matrix. Clean the nail plate and nail bed with sterile saline. Replace and secure the nail plate.

More often with these crush injuries, the germinal matrix is avulsed as well. In these cases, the germinal matrix should be replaced with a series, usually three, of 6–0 nylon simple interrupted or horizontal mattress sutures (**Figure 129-12**).[5,7,11] Place all three sutures before returning the nail bed to its proper location. Making two linear incisions with a scalpel at 90° from the eponychial edge will allow exposure to the germinal matrix for repair (**Figure 129-8**).[8] This allows the eponychium to be folded or sutured back and therefore increases the exposure of the germinal matrix.[2,21] Place a piece of petrolatum gauze between the eponychium and the germinal matrix after the repair (**Figure 129-12*B***). Larger germinal matrix avulsions require consultation with a Hand Surgeon for grafting.[2,7,12,20]

LACERATIONS WITH ASSOCIATED FRACTURES

The fourth type of nail bed injury are lacerations associated with fracture(s). Approximately 50% of nail bed injuries have an associated phalangeal tuft fracture.[24] The nail bed laceration should be repaired as previously described and the fracture addressed as a separate entity.[5] It is important to remember that the sterile matrix is closely adherent to the dorsal periosteum of the distal phalanx. Therefore, fractures require precise anatomic reduction in order for normal nail bed healing and nail plate growth to take place.[7,11,13] Replacing the nail plate and splinting the finger after repairing the nail bed laceration is often enough to reduce these fractures. Occasionally, fixation may be employed by a Hand Surgeon using Kirschner wires to prevent rotation of the bony fragments.[2,7,24]

LACERATIONS WITH SKIN LOSS AND FINGERTIP AMPUTATIONS

The final two types of injury are lacerations with loss of skin and pulp and fingertip amputations.[26] They can be further classified according to zones based upon the anatomic level of amputation (**Figure 129-13**).[3] Zone I injuries occur distal to the bony phalanx. These do not result in loss of function and rarely result in a cosmetic deformity. Management consists of cleansing, placing topical antibiotic ointment over the injured area, and then applying a layer of petrolatum gauze. This should be followed by a sterile dressing and a splint. Zone II injuries occur distal to the lunula and over the bony phalanx. These injuries often have exposed bone. Zone III injuries occur proximal to the distal end of the lunula. Zone II and zone III

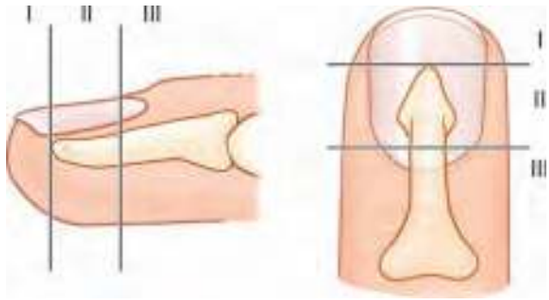

FIGURE 129-13. Classification of fingertip injuries. Zone I is distal to the bony phalanx. Zone II is distal to the lunula and over the bony phalanx. Zone III is proximal to the distal end of the lunula.

injuries should be managed in consultation with a Hand Surgeon. Either type of injury may require reconstruction with a pedicle flap and/or skin grafting.[3]

TREPHINATION FOR A SUBUNGUAL HEMATOMA

A subungual hematoma is a collection of blood under the nail plate caused by blunt trauma to the fingertip. The nail bed is usually crushed or lacerated with resultant extravasation of blood into the plane between the nail plate and the nail bed.[12] Compression of nail bed nerves occurs and often causes significant pain as pressure builds up. Usually this pain is what causes the patient to seek medical attention. The management of subungual hematomas (Chapter 127) is discussed separately because these injuries are typically minor and treated by simple trephination.[27]

It is generally accepted that subungual hematomas less than 50% of the nail plate may be managed conservatively by simple trephination (i.e., puncture) of the nail plate.[2,5,7,12,21,28,29] This allows for drainage of blood and immediate pain relief. No anesthesia is usually necessary for this procedure. This is accomplished with the use of an electrocautery device creating a 3 to 4 mm hole in the nail plate overlying the hematoma. The nail plate should be clean and dry for this procedure. A heated paperclip may be used if an electrocautery device is not available.[5]

Some controversy exists over the management of subungual hematomas greater than 50% of the nail plate.[30] The concern is that a larger hematoma may hide an occult laceration that requires repair in order to avoid the complication of step-off with subsequent ridging as the new nail grows back. Some authors feel that it is impossible to accurately assess the amount of damage beneath a subungual hematoma unless the nail plate is removed to directly inspect the nail bed.[7,29] These authors noted that subungual hematomas that have separated greater than 50% usually have lacerations that require repair. Another study found that a subungual hematoma with more than 50% separation had a 60% incidence of having a nail bed laceration that required repair and up to a 95% incidence when there was an associated phalanx fracture.[29] In contrast to these studies, a prospective study found no complications at 6 months of follow-up for subungual hematomas treated by electrocautery trephination alone.[2,28] This was regardless of the size of the subungual hematoma or the presence of a fracture. These authors feel that removing the nail plate and attempting repair may actually cause further trauma to the nail bed.[28] These results were also duplicated in the pediatric population, with trephination alone having results that were equal to or better than removal of the nail.[31] Nail removal and primary care of the laceration are recommended in the case of disruption of the nail plate or nail fold or with displaced fracture fragments.[32]

It was previously recommended that radiographs be obtained to rule out a fracture for all hematomas larger than 50% of the nail plate. A newer study found no correlation between the size of the hematoma and the presence of fractures.[28] **Have a low threshold**

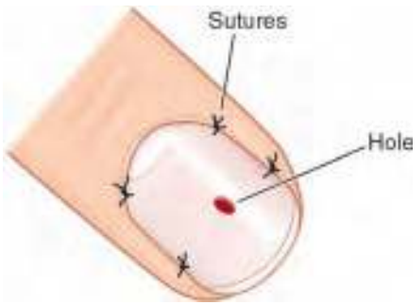

FIGURE 129-14. A hole is placed in the nail plate before it is sutured in place.

FIGURE 129-15. Figure-of-eight suture for securing the nail plate.

to obtain plain radiographs prior to evacuating the hematoma if a fracture is suspected. This is especially important in children, where proper bony alignment and stability are necessary for normal finger growth.[32] Bedside ultrasound is a promising tool for detecting nail bed injuries and tuft fractures.[33,34]

In summary, most subungual hematomas may be treated by simple nail trephination using an electrocautery device. This procedure will lead to beneficial drainage only if done before 36 to 48 hours from the time of the injury.[12] The clinical benefit of nail bed repair with larger subungual hematomas is controversial but may not be necessary if the nail plate is adherent to the nail bed and is without laceration. Nail plate removal and nail bed repair are favored in cases where a proximal fracture involves the germinal matrix, a displaced fracture of the distal phalanx, or laceration with injury to the nail plate.[35] **It may be prudent to maintain a lower threshold for nail bed repair with a larger subungual hematoma, particularly if an ideal cosmetic outcome is desired.**

AFTERCARE

Whenever possible, the nail plate should be replaced after repairing any of the previously mentioned injuries. The nail plate covers the sensitive nail bed and protects it from injury, maintains the proximal nail fold (i.e., eponychium) to allow for growth of a new nail, and splints the nail bed. Place a hole in the center of the nail plate to allow for fluid drainage to accomplish the best outcome.[12] Make one or two holes in the lateral aspects of the nail plate. Suture the nail plate in place using 5–0 nylon sutures through the lateral skin folds (**Figure 129-14**).[7,11,12] A figure-of-eight suture technique to attach the nail does not require an intact eponychial fold and minimizes iatrogenic trauma (**Figure 129-15**).[36] Place an absorbable suture (4–0 in adult, 5–0 in children) into one side of the paronychium in a distal to proximal fashion. Run the suture through the notches in the distal aspect of the nail plate and then place them from distal to proximal in the opposite paronychium. The suture is then tied across the nail plate to create a figure-of-eight pattern (**Figure 129-15**). Remove these sutures securing the nail plate after 3 weeks, at which time the nail will be adherent or slough to allow for new nail growth.[37] An alternative to suturing the nail plate in position is to use tissue adhesive. Replace the nail plate and apply tissue adhesive along the eponychium and lateral nail folds where they overlap the nail plate.

A nail alternative should be used if the patient's nail is not available, too fragmented, or too dirty to use as a natural splint. Numerous materials have been suggested for nail bed covers, including the reservoir of an infusion set, acrylic nails, a nasogastric catheter splint, and the aluminum suture package material.[24,37-39] An artificial nail may be used if the original nail plate is not available. The potential problem with sterile prefabricated nails is that there exists an increased risk for infection and a risk of erosion into the nail bed or nail folds.[7] The editor recommends petrolatum gauze be used to maintain the nail fold structures when the original nail plate is not available or significantly damaged (**Figure 129-16**).

After repair of the nail bed, the digit(s) involved will need to be bandaged in order to protect it from infection, moisture, and trauma. The application of becaplermin (i.e., recombinant human platelet-derived growth factor) has been shown to improve outcomes.[40] This substance is not routinely used in the Emergency Department and should be applied after consultation with a Hand Surgeon. Place petrolatum gauze over the nail bed to avoid adherence from secretions. Apply a tube gauze dressing followed by a digital aluminum splint.[3] Movement of the distal interphalangeal joint should be restricted for 7 to 10 days with a splint.[7] The entire hand should be dressed for infants and young children. Remove any petrolatum gauze used to keep the proximal nail fold (i.e., eponychium) open in 5 to 10 days.[7] Petrolatum gauze used to keep open the lateral nail folds (i.e., perionychium) should remain in place for 10 days.[5] Any sutures placed in the skin structures or nail folds should be removed in 7 to 10 days.[5]

Prophylactic antibiotics are not routinely required. They are recommended for large avulsions, amputations, animal or human

FIGURE 129-16. Petrolatum gauze may be placed between the dorsal roof and germinal matrix (*A*) or between the lateral skin fold and nail bed (*B*) to preserve the skin fold spaces.

bites, and associated fractures when there is contamination with organic material.[7] Topical antibiotics may be applied to zone I fingertip amputations. All injuries require a wound check in 24 to 72 hours.[3,5,7] The patient's tetanus immunization status should be determined and prophylaxis administered if required. The hand should be elevated whenever possible. Nonsteroidal anti-inflammatory drugs supplemented with 1 or 2 days of narcotic analgesics may be prescribed as needed for pain control.

It is important to explain to the patient that regeneration of a new nail may take up to 6 to 12 months.[7] Warn the patient that the new nail may look irregular and snag on cloth or string objects.[7] The patient should trim and file the leading edge of the new nail once it extends past the hyponychium in order to avoid snagging.

COMPLICATIONS

The complications associated with failure to repair or improper repair of a nail bed can be functional, cosmetic, or both. The more serious complications are those that impair function or cause pain. There are basically seven complications that may arise. These exclude the infectious complications (e.g., abscess formation, cellulitis, and lymphangitis) that are inherent to all wounds. The occurrence of osteomyelitis from these injuries is rare, even with open fractures.[25] The seven complications are loss of the nail, a split nail, a nonadherent nail, an ingrown nail, a malaligned nail, wide nails, and narrow nails.[13]

The complete loss of a nail could result in significant functional impairment to the fingertip as well as an abnormal-looking fingertip. Complete nail loss occurs when there is complete disruption of the germinal matrix, either by significant avulsion of the matrix or amputation. Remember that if the germinal or roof matrices are not repaired, the pouch deep to the proximal skin fold (i.e., eponychium) is obliterated within a few days.[28]

A split nail occurs when the germinal matrix is improperly approximated.[5,7,13,24] A wide scar will result that will not form a new nail. Subsequently, a split nail develops. This can be avoided by the careful approximation of the nail bed with sutures.

A nonadherent nail occurs when the nail bed is not repaired and granulation tissue forms secondarily. The nail plate will not adhere at the site of the granulation tissue as well as distal to the granulation tissue.[13] The nail can snag and be exposed to repeated tears once the nail plate loses adhesion to the nail bed.

The nail plate may ingrow if the lateral skin folds or sulci are not maintained and kept open.[13] Adhesions that form between the skin and nail bed can be very painful when the new nail tries to grow through that space.[5] Ingrown nails also have the long-term problem of a higher rate of infections (e.g., a paronychia). This can be avoided by the replacement of the nail plate or the placement of petrolatum gauze to elevate the lateral nail fold from the nail bed.

The nail plate will grow in a malaligned direction if the matrix is displaced or repaired in such a manner that it is improperly aligned.[13] Functional impairment and cosmetic deformity may ensue depending upon the degree of this misdirected growth.

Wide nails often result from a crush injury with a tuft fracture.[13] Separated bony fragments leave the nail bed flatter and wider. Narrow nails occur when a central avulsion-laceration is not repaired. Scar tissue forms in the center and allows the intact lateral portions to contract toward each other.[13] The new nail subsequently becomes narrow and thick.

Tubular gauze dressings are commonly applied after the repair of a nail bed. **Tubular gauze dressings can result in significant injury when improperly placed.**[25,41] This includes digital ischemia that may be permanent and require an amputation. **Tubular gauze dressings should not be placed by personnel not trained in their proper application.**

SUMMARY

Nail bed injuries are common and may result in a cosmetically deformed or functionally impaired fingertip. Complications may occur even with precise repair. The treatment of choice is immediate primary repair of the nail bed and surrounding structures. Remember to minimize any debridement and to replace the nail plate whenever possible. Injuries with associated fractures and simple subungual hematomas are managed separately from the nail bed injury. Always be thorough and meticulous when repairing nail bed injuries to provide the best possible outcome. Finally, know when to consult with a Hand Surgeon.

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130 Ganglion Cyst Aspiration and Injection

Thomas P. Graham

INTRODUCTION

Ganglion cysts (i.e., synovial cysts or ganglia) are the most common soft tissue tumors of the wrist and hand.[1] They are a common reason for patients to present to the Emergency Department. The chief complaint is usually a mild pain or ache, exacerbated by movement, and localized to a 1 to 2 cm mass on the wrist or hand (**Figures 130-1, 130-2, and 130-3**). Patients may also present with concerns about a painless "lump." Acute trauma prior to presentation is uncommon. Patients often give a history of repetitive motion at the site. The mass usually increases in size progressively over time or occasionally may grow rapidly over a short period. Patients presenting to the Emergency Department with ganglia may have already attempted one of several popular home remedies (e.g., homeopathic medications or striking the cyst firmly with a large book or hammer).

Ganglion cyst aspiration is a relatively simple procedure that may be performed by the Emergency Physician. The practice of cyst aspiration has been challenged because of the high recurrence rates of up to and even greater than 50%.[2-6] The procedure usually alleviates presenting symptoms, is occasionally curative, and is more cost-effective than referring all patients for surgical treatment.[7]

ANATOMY AND PATHOPHYSIOLOGY

Ganglia are synovial cysts that originate from a joint capsule or tendon sheath.[8] They have no malignant potential. It is unclear whether ganglia are formed by herniation of the tendon sheath, myxomatous degeneration of connective tissue, or some other mechanism. Contained within the cyst is a viscous, jelly-like fluid. Ganglia often connect with the underlying synovial cavity or tendon sheath by a stalk (**Figure 130-2**). Hyaluronic acid makes up all or part of the mucoid fluid.[9]

FIGURE 130-1. Oblique view of the wrist demonstrating a ganglion cyst overlying the scapholunate joint. (Used from James Heilman, MD at www.commons.wikimedia.org.)

Ganglia are usually encountered on the dorsum of the wrist, in particular over the scapholunate ligament (**Figures 130-1, 130-2, and 130-3**). They may also be found on the palmar surface of the wrist, the lateral surface of the wrist, or on the hand itself. Ganglia of the foot and ankle are less commonly seen.[10] Ganglia are less commonly encountered in other areas (e.g., shoulder, hip, elbow, knee, lumbar spine, temporomandibular joint, and the odontoid process of the cervical spine).[11-13]

Ganglia present as fixed or slightly movable masses that are usually solitary. The cyst is frequently characterized as smooth and "rubbery." Cysts may become more noticeable with wrist flexion. They vary in size from barely palpable to 3 cm in diameter, with smaller than 1.5 cm being the norm. Tenderness is sometimes but not invariably present. Ganglion cysts will transilluminate since they are fluid-filled. They may "disappear" over time by spontaneously rupturing or due to resorption.

Diagnosing a ganglion cyst is usually not difficult. Ganglia of the foot and other uncommon locations may be difficult to palpate despite causing significant discomfort. The differential diagnosis of ganglia includes epidermoid cysts, xanthomas, rheumatoid nodules,

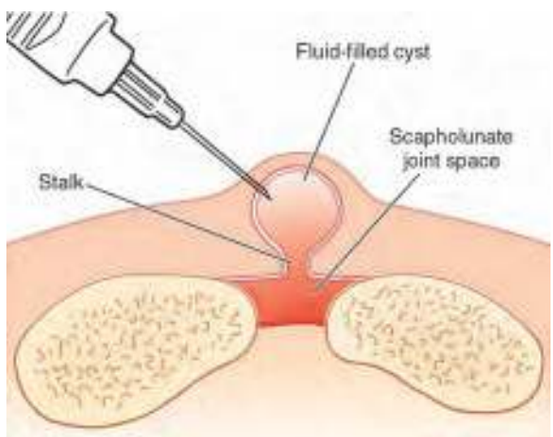

FIGURE 130-2. Cross-section through the scapholunate joint demonstrating a ganglion cyst. The needle is inserted into the cyst cavity to aspirate its contents.

A

B

C

FIGURE 130-3. Aspiration of a ganglion cyst. ***A.*** The needle is inserted. ***B.*** Aspiration of the contents flattens the ganglion cyst. ***C.*** The aspirated ganglion cyst with the aspirated material on the slide.

benign tumors (e.g., lipomas and neuromas), joint capsulitis, and other soft tissue neoplasms (e.g., sarcomas and chondrosarcomas). Ultrasonography can be used to aid in the diagnosis, particularly of occult ganglia or ganglia presenting atypically.[14-16] Magnetic resonance imaging (MRI) is frequently used to confirm the diagnosis and plan its operative removal.[15,17,18] Plain radiographs and laboratory tests are not helpful in the work-up of a ganglion cyst.

INDICATIONS

The most common indication for ganglion aspiration is worsening pain and swelling, in particular when normal range of motion is restricted or occupational disability is present. Failure of conservative measures (e.g., rest, splinting, and the use of nonsteroidal anti-inflammatory drugs) to resolve symptoms may prompt ganglion aspiration in the Emergency Department.

CONTRAINDICATIONS

There are few contraindications to ganglion cyst aspiration. Avoid introducing a needle through an area of cellulitis. Cellulitis overlying a ganglion is uncommon and should raise suspicion for an alternate diagnosis (e.g., a skin abscess). The procedure can be safely deferred and the patient referred to a Hand Surgeon if the diagnosis of a ganglion is uncertain.

The location of a ganglion is generally not a contraindication to aspiration. Defer the procedure if there is a concern that the aspirating needle could damage an adjacent structure, cause neurologic injury, or cause vascular injury. Aspiration of palpable lower extremity ganglia may be performed in a similar fashion to hand and wrist lesions with similar results.[10,19] Some literature suggests that volar wrist ganglion cysts recur at an even higher rate than those of the dorsal wrist and lower extremity. This had led some authors to recommend surgical excision as the primary therapy for this subset of ganglia.[20]

EQUIPMENT

- Sterile gloves
- Povidone iodine solution or chlorhexidine solution
- Local anesthetic solution without epinephrine
- 25 or 27 gauge needle on a 3 mL syringe for local anesthesia
- 16 or 18 gauge needle on a 5 or 10 mL syringe for aspiration
- 10 to 15 mg methylprednisolone acetate (20 mg/mL) or prednisolone tebutate (20 mg/mL)

PATIENT PREPARATION

Explain the risks and benefits of the procedure to the patient and/or their representative. Obtain an informed consent, either signed or verbal, with adequate documentation to support the latter method. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Ultrasonography can be used to confirm the diagnosis and to facilitate needle entry into the cyst. Ultrasound can help to avoid neurologic or vascular structures when ganglia are located near them.[21] Place the patient on a gurney with the hand on a bedside procedure table. Clean the skin of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. It is recommended to provide local anesthesia for patient comfort, although the cyst can be aspirated and/or injected without anesthesia. Place a subcutaneous wheal of local anesthetic solution immediately over or adjacent to the periphery of the ganglion.

TECHNIQUE

GANGLION ASPIRATION

Manipulate the area to expose more of the cyst and facilitate needle entry into the cavity. Ultrasound may be used to delineate the cyst but is usually not necessary for the palpated ganglions.

Use sterile technique. Insert a 16 or 18 gauge needle on a 5 or 10 mL syringe through the anesthetized tissue and into the ganglion cyst cavity (**Figures 130-2 and 130-3*A***). Apply negative pressure to the syringe to aspirate the cyst contents once the tip of the needle is within the cyst cavity (**Figure 130-3*B***). Aspiration may require more force than anticipated as the mucinous contents of the cyst are quite viscous. The cyst can be manipulated and compressed to express more of the contents into the syringe once the very viscous clear or yellow material begins to flow into the syringe. Approximately 1 to 2 mL of fluid can be aspirated from a typical ganglion. Withdraw the needle when fluid can no longer be aspirated (**Figure 130-3*C***). Apply a simple dressing. A pressure dressing may also be temporarily applied. **Take care not to compromise neurologic or vascular function with the pressure dressing.**

GLUCOCORTICOID INJECTION

The injection of glucocorticoids into a ganglion cyst immediately after aspiration is commonly recommended. One small study showed glucocorticoid injections decreased recurrence rates compared to aspiration alone.[22] The literature has not yet shown a clear benefit.[23] Securely hold the aspirating needle in place after the cyst contents have been aspirated into the syringe. Remove the syringe. Do not move the needle. Attach a second syringe containing 10 to 15 mg of the glucocorticoid solution onto the needle. Aspirate to confirm the tip of the needle is not within a vascular structure. Inject the glucocorticoid solution into the cyst cavity. Withdraw the needle and apply a simple dressing.

ALTERNATIVE TECHNIQUES

Other variations of ganglion cyst aspiration and injection have been described in the literature. The injection of hyaluronidase into the cyst, with or without corticosteroids, has shown favorable results.[24,25] Puncturing the ganglion wall at multiple separate locations has not been proven to decrease the recurrence rate when compared to aspiration at a single point alone.[26] Injection of hypertonic saline and other sclerosing agents (e.g., 1 mL of 3% sodium tetradecyl sulfate) has been suggested. Applying blunt force trauma to the cyst with a large book or other heavy object is a common home remedy with many anecdotal reports of success without recurrence.[27] Further study is necessary before these techniques can be recommended for widespread use in the Emergency Department.

ASSESSMENT

Patients usually report total or near-total relief of their symptoms immediately after aspiration. Obtaining highly viscous clear or yellow fluid from the cyst virtually confirms the diagnosis. Obtaining purulent fluid suggests a skin abscess and not a ganglion cyst. Failure to obtain fluid does not rule out the diagnosis of a ganglion. It should prompt an evaluation for alternative diagnoses.

AFTERCARE

Reassure patients that ganglia are not malignant tumors. Temporary immobilization (i.e., < 24 hours) of the affected limb may be performed for patient comfort. Splinting limits the ability of patients to function normally and does not appear to affect recurrence rates.[28] Instruct the patient to return to the Emergency Department for any significant increase in pain, erythema at the site or up the extremity, swelling beyond the ganglion's original size, purulent discharge, continued bleeding, or the development of a fever. Instruct the patient to elevate the extremity, avoid strenuous activity of the affected limb, and rewrap the pressure dressing if one is applied to make it snug but not uncomfortable. Recommend acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs) for relief of mild postprocedure pain. Narcotic analgesics are not required nor recommended. Oral corticosteroids have not been demonstrated to play a role in ganglion therapy.

Inform all patients with ganglion cysts aspirated in the Emergency Department of the potential for recurrence and the possibility of definitive ganglion treatment by surgical excision.[8,29] Provide a referral to a Hand Surgeon if desired by the patient. The most effective therapy for ganglion cysts is believed to be open excision, although reports of postsurgical recurrence rates vary widely.[30] The Hand Surgeon removes the cyst and the stalk or pedicle that connects it to the normal synovium. Arthroscopic removal of wrist ganglia has also been described and performed successfully.[6,31]

COMPLICATIONS

Complications of ganglion cyst aspiration are uncommon. They include bleeding and infection. Bleeding is self-limited and easily controlled with manual pressure. The use of sterile technique will minimize any infectious complications (e.g., abscess, cellulitis, or septic arthritis). Rare complications of corticosteroid injection include localized depigmentation that is due to injection outside of the cyst capsule or leakage out of the cyst capsule through the needle tract.[32] Subcutaneous infiltration of glucocorticoids can result in fat atrophy, skin atrophy, and skin dimpling from fat atrophy.

SUMMARY

Ganglion cysts are common growths of the wrist and hand. They frequently enlarge to cause pain, sometimes to the point of disability. Contraindications to the aspiration of a ganglion cyst in the Emergency Department are few. The procedure is often warranted due to debilitating pain, deformity, or inability on the patient's part to promptly seek the care of a surgical specialist. The procedure is simple, quick, and only slightly uncomfortable. The injection of steroids into the cyst is commonly advocated to prevent recurrence. The efficacy of steroid injection has not been conclusively demonstrated. Ganglia recurrence is very common after aspiration and may occur even after surgical excision. **This fact must be clearly relayed to the patient.** All patients should be offered referral to a Hand Surgeon on a nonemergent basis to discuss further intervention.

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131 Subcutaneous Abscess Incision and Drainage

Priya Perumalsamy

INTRODUCTION

Subcutaneous abscesses are commonly seen in the Emergency Department. Approximately 3% of patients present with this chief complaint.[1,2] Abscesses occur in numerous anatomic areas with varied etiology and bacteriology. **An abscess is a tender and fluctuant mass located in the dermal or subdermal tissue.** It usually demonstrates the classic inflammatory responses of rubor, tumor, dolor, and calor. The abscess is usually tender. **The surrounding and underlying tissue should not be tender.**[3-5]

Incision and drainage is the definitive treatment of a simple soft tissue abscess.[5-7] This procedure results in significant improvement in symptoms and a rapid resolution of the infection in uncomplicated cases.[8] The addition of antimicrobial therapy is needed for systemic signs of sepsis, immunocompromised patients, if source control is incomplete, and for abscesses with significant cellulitis.[5] Premature incision before localization of pus will not be curative and may be deleterious. Oral antibiotics and warm compresses may be of value in helping the infection to coalesce in cases of immature abscesses or cellulitis. These methods are not a substitute for incision and drainage and should not be continued for more than 24 to 36 hours without a reassessment of the patient. The role for ancillary antibiotic use has come into question with the emergence of methicillin-resistant *Staphylococcus aureus* (MRSA).

ANATOMY AND PATHOPHYSIOLOGY

PATHOGENESIS

An abscess is a localized collection of pus caused by suppuration buried in a tissue, organ, or confined space.[9] Intact skin is very resistant to bacterial invasion. Localized pyogenic infections are usually initiated by a breakdown in the normal epithelial defense mechanisms in the normal host. Plugging of the ducts of a superficial exocrine gland may initiate the process. Occlusion prevents desquamation and provides a moist environment for organisms to proliferate. The combination of a high concentration of organisms, the presence of nutrients, and sufficient damage to the corneal skin layer to allow organisms to penetrate the skin defenses results in abscess formation.[10,11]

Subcutaneous abscesses typically begin as a cellulitis with organisms that cause necrosis, liquefaction, and accumulation of leukocytes and debris. Early stages appear as an area of hyperemia and tender inflammation that later becomes fluctuant as an exudate of leukocytes, necrotic material, and cellular debris accumulates. This is followed by loculation and walling off of the pus. This progresses and the area of liquefaction increases until it "points" and eventually ruptures through the area of least resistance.[4] **A simple abscess should not extend into deeper tissues or have multiloculated extension.** The induration and erythema are limited to a defined area of the abscess. An abscess with extensive overlying cellulitis, multiloculated extensions, or systemic signs of illness can be considered complex in comparison to a simple abscess.[5]

The body area involved depends upon host factors (e.g., drug use, employment-related exposures, or minor trauma).[5,11-13] Areas with a compromised blood supply are more prone to infection as normal host cell-mediated immunity is not as available.[11] Soft tissue infections most commonly affect the lower extremities, perineum, and abdominal wall.[5]

BACTERIOLOGY

Most abscesses are polymicrobial with the isolated organisms usually representing the normal resident flora associated with the body area on which the abscess is found.[10,13-15] Nonresident bacteria are found in abscesses that occur from direct inoculation of extraneous organisms (e.g., following human bite wounds, intravenous drug use, or bacterial seeding of embedded foreign bodies).[16] Most infections involving healthy skin are caused by gram-positive cocci, most commonly *S. aureus* and group A *Streptococcus*.[5,13,15] Anaerobes are found in all areas of the body but predominate in abscesses of the buttocks and perirectal regions.[14,17]

S. aureus is present as a single pathogen in only 25% of cutaneous abscesses.[13] Community-acquired MRSA (CA-MRSA) is presently considered to be the most common identifiable pathogen causing abscess at major centers.[18,19] CA-MRSA is defined as infection with MRSA acquired in the community lacking the traditional risk factors of hospital-acquired MRSA (e.g., a recent stay in a long-term care facility or in a day care setting, recent health care contacts

or surgery, an indwelling device, dialysis, immunosuppression, chronic illness, or recent antibiotic use).[13,20-22] Patients at a higher risk of acquiring MRSA infections include those with recent household or daycare contacts, children, men who have sex with men, those in the military, incarcerated patients, athletes who participate in contact sports or share equipment, Native Americans, Pacific Islanders, patients with previous MRSA infections, and intravenous drug users.[13,20-24] It is essential not to exclude MRSA in the absence of risk factors.[18] Investigate patients presenting with a complaint of a "spider bite" for MRSA.[20-22,25] MRSA was isolated from abscesses in 51% of patients.[26] An American study of academic centers in 2006 found MRSA incidence rates as high as 60% in patients presenting with skin and soft tissue infections.[27] Up to 77% of MRSA-related illnesses are skin and soft tissue infections.[28]

In the United States, 98% of CA-MRSA strains carry the *mec*A gene, which is thought to impart the antimicrobial resistance. This is carried on the staphylococcal cassette chromosome type-IV which is distinct from other forms of MRSA.[20-22,29] Most have the gene for Panton-Valentine leukocidin (PVL), an exotoxin that predisposes patients to cutaneous infections.[21,22,29] PVL has been identified in up to 95% of CA-MRSA cases.[29] PVL-positive strains are associated with tissue necrosis and abscess formation.[20]

Up to 17% of abscesses are sterile.[8,10,17] Nearly 40% of these are secondary to intravenous drug use and most likely result from injection of necrotizing chemical irritants.[30] Viruses (e.g., herpes), autoimmune mechanisms, or systemic illnesses (e.g., benign tumors, metastatic tumors, and granulomatous disease) may cause sterile abscesses.[3,31] These atypical etiologies may present with an exacerbation of the underlying disease process in the absence of local inflammatory signs and symptoms.

SPECIFIC CLINICAL ENTITIES

Furuncles, or boils, are acute circumscribed abscesses of the skin and subcutaneous tissue that most commonly occur on the axilla, breast, buttocks, face, neck, perineum, and thigh. Carbuncles are aggregates of interconnected furuncles that frequently occur on the back of the neck where the thick skin causes lateral extension of the infection rather than pointing toward the skin surface. These occur with a higher frequency in diabetics. They can be large and cause systemic signs, symptoms, and complications. Carbuncles often require surgical consultation and treatment in the Operating Room.

Hidradenitis suppurativa is a chronic relapsing inflammatory disease process affecting the apocrine glands primarily in the axilla, the inguinal region, or both.[32] This process initially will appear like a typical abscess and is only identifiable in its chronic scarring phase when there are multiple lesions with tender areas of induration and inflammation in various stages of healing. The chronic process leads to draining fistulous tracts that require ongoing surgical management. Emergency Department management involves the usual incision and drainage procedure of any area of fluctuance. Patients should be informed that the intervention is not curative and that the problem is chronic. Arrange a referral to a General Surgeon, Dermatologist, or Plastic Surgeon for long-term follow-up.

Up to 80% of breast abscesses occur in nonlactating women.[3] Peripheral and superficial lesions are similar to abscesses elsewhere on the body. They respond to conservative incision and drainage with an incision that radiates out centripetally from the nipple.[33] Deeper and periareolar abscesses are often complex and require surgical referral and general anesthesia to properly treat. Postpartum mastitis is common and precipitated by milk stasis and bacterial invasion through a cracked nipple. The offending organism is commonly *S. aureus* or *Streptococcus* species. Treatment includes the application of heat, oral antibiotics, and continued breast emptying with a breast pump or feeding of the baby. The mastitis may evolve into an abscess and is often associated with systemic symptoms. Appropriate antibiotic therapy and follow-up in 24 to 48 hours are required.

Sebaceous cysts are a common cause of a subcutaneous abscess. They can persist for long periods as nontender subcutaneous swellings before becoming infected. They appear like most other abscesses. Sebaceous cysts can be identified by a small punctate sinus tract near the center of the fluctuant area. The initial treatment is incision and drainage. The contents are usually thick cheesy material that needs to be manually expressed. A sebaceous cyst has a definite shiny white capsule that must be excised at the time of incision and drainage or at the first follow-up visit to prevent recurrence. The area is then treated as any other healing abscess cavity.

The recurrence of an abscess that has been previously drained should suggest the possibility of underlying osteomyelitis, a retained foreign body, or the presence of unusual organisms such as mycobacteria or fungi. Recurrent abscesses should prompt further investigation including an assessment of the patient's immune status.

SPECIAL CONSIDERATIONS

The precise risk for endocarditis associated with subcutaneous abscesses is unknown. Up to 5% of patients with abscesses have bacteremia at the time of presentation.[3,34] Incision and drainage of cutaneous abscesses can result in transient bacteremia with the organism causing the abscess.[8,35,36] The clinical relevance of this bacteremia has become controversial.[34] **Only patients considered to be at high risk for endocarditis are recommended to receive antimicrobial prophylaxis before incision and drainage (Table 131-1).**[37] Direct bacterial endocarditis prophylaxis at the most likely pathogen causing the infection. An antistaphylococcal penicillin or a first-generation cephalosporin is an appropriate choice for most soft tissue infections (**Table 131-2**). Clindamycin is an acceptable alternative for patients allergic to penicillin. Patients with immunodeficiency and localized soft tissue abscesses may be at higher risk for developing septicemia secondary to bacteremia induced by incision and drainage, but it is unclear if they are at higher risk of complications and death.[38,39] These patients may benefit from prophylactic antibiotics prior to incision and drainage, but only indirect evidence is available and no controlled studies have been done.[40] **High-risk patients with known or suspected MRSA infection should receive intravenous vancomycin or clindamycin when undergoing incision and drainage.**[37]

PATIENT ASSESSMENT

Take a focused history and assess for trauma, intravenous drug use, history of fever, and past medical history. Inquire about diabetes, renal failure, steroid use or other immune suppression, peripheral vascular disease, and valvular heart disease. It is important to note any risk factors for CA-MRSA as this may alter management of certain patient populations.[41] Past anesthetic history and

TABLE 131-1 Cardiac Conditions at Risk for Endocarditis That Require Antibiotic Prophylaxis for Incision and Drainage[32]

Cardiac transplant with cardiac valvulopathy
Prosthetic cardiac valves, including bioprosthetic and homograft valves
Previous bacterial endocarditis
Repaired congenital heart disease (CHD) with prosthetic material or device within 6 months after the procedure (regardless of method of placement)
Repaired CHD with residual defects at or adjacent to site of prosthetic patch or device
Unrepaired cyanotic CHD, including palliative shunts and conduits
Except for the conditions listed above, antibiotic prophylaxis is no longer recommended for any other form of CHD

TABLE 131-2 Prophylactic Antibiotic Regimens[32]

Clinical situation	Agent	Dose*	Timing
Standard general prophylaxis	Amoxicillin	Adults: 2.0 g Child: 50 mg/kg	PO, 30–60 min prior to the procedure
Unable to take oral medications	Ampicillin	Adults: 2.0 g Child: 50 mg/kg	IV or IM, 30 min prior to the procedure
Unable to take oral medications	Cefazolin or ceftriaxone	Adults: 1.0 g Child 50 mg/kg	IV or IM, 30–60 min before procedure
Allergic to penicillin or ampicillin	Cephalexin†	Adults: 2.0 g Child: 50 mg/kg	PO, 30–60 min prior to procedure
Allergic to penicillin or ampicillin	Clindamycin	Adults: 600 mg Child: 20 mg/kg	PO, 30–60 min prior to procedure
Allergic to penicillin or ampicillin	Azithromycin or clarithromycin	Adults: 500 mg Child: 15 mg/kg	PO, 30–60 min prior to procedure
Allergic to penicillin and unable to take oral medications	Cefazolin† OR Ceftriaxone	Adults: 1.0 g Child: 25 mg/kg	IV or IM, 30–60 min prior to procedure
Allergic to penicillin and unable to take oral medications	Clindamycin	Adults: 600 mg Child: 20 mg/kg	IV or IM, 30–60 min prior to procedure
Known or suspected MRSA	Vancomycin	Adults: 1 g Child 20 mg/kg	IV, 30 minutes prior to procedure

IM, intramuscular; IV, intravenous; PO, oral.

*Total child dose should not exceed adult dose.

†Cephalosporins should not be used in patients with immediate-type hypersensitivity reactions (e.g., urticaria, angioedema, or anaphylaxis) to penicillin.

the potential for aspiration should be assessed if procedural sedation (Chapter 159) is to be considered.[42] Ask about medications and allergies. The patient's tetanus status must be confirmed and booster doses provided as required.

A brief physical examination documenting function and intact distal neurovascular status of extremities involved is required. Evidence of pain on passive or active movement of fingers may suggest a deep space infection.[3] A high index of suspicion is required, especially in injection drug users, to identify those seemingly simple cutaneous infections that unpredictably evolve into extensive necrotizing soft tissue infections.[41,43] General assessment of the airway and cardiopulmonary system, vital signs, and mental status is indicated if procedural sedation (Chapter 159) is to be employed.[42]

Routine laboratory studies are not indicated in otherwise healthy individuals. Consider a complete blood count looking for leukopenia or toxic granulations in immunocompromised patients. Diabetics should have electrolytes, blood urea nitrogen, creatinine, and glucose assessed. Elevated potassium in diabetics may indicate myonecrosis.[44] Consider a urinalysis for myoglobinuria. Consider obtaining radiographs of the affected areas if there is a history of trauma, drug use, or concern regarding a deep infection. Abscesses in injection drug users may be contaminated with foreign bodies (e.g., broken needles). Foreign bodies and fractures may not be easily identifiable because of the edema and tenderness caused by the infection.[5,45] Gas or osteolytic lesions on plain radiographs may indicate severe deeper infection, the need for urgent surgical consultation, and prompt antibiotic therapy.[45] Vascular complications should also be considered based on the site and evaluated with duplex sonography if needed. A further evaluation for endocarditis is necessary if there are persistent signs of sepsis.[5]

Culturing the purulent material from a drained abscess was previously considered to be of little value. Controversy exists today as to the value of culture in abscess management. Although many experts suggest it is of little value because it has no bearing on acute management,[7,46-48] others suggest it is vital to help determine antimicrobial susceptibility and outbreak patterns.[5,25,41] **Obtain cultures because they may alter later management in cases of recurrent or refractory infections, patients who are seriously ill, immunocompromised, patients on an antimicrobial with variable activity, patients who have failed surgical treatment, and any patient being admitted to the hospital.**[5,25,41,49] Gram's stain and cultures for both aerobic and anaerobic bacteria may be helpful in patients who are febrile, systemically unwell, or immunocompromised, or who present atypically.[5,41,50,51]

ULTRASONOGRAPHIC EVALUATION

Bedside ultrasound (US) use in the Emergency Department is of great value in evaluating skin and soft tissue infections.[52] US can be used to differentiate a cellulitis from an abscess in difficult cases. There are numerous advantages to using US (e.g., portability, inexpensive, comfortable for patients, and no radiation exposure). It is fast and easy to gain proficiency for specific applications.[53] US is indicated for ambiguous physical findings (e.g., widespread cellulitis), localizing an incision site, and ruling out dangerous masses (e.g., a pseudoaneurysm that masquerades as an abscess).[54] US is helpful in identifying small or early abscesses, deep abscesses, abscesses under previous scars, and for localizing adjacent major vessels or nerves.[54-59] US has a sensitivity of 98% and specificity of 88% in detecting an abscess.[53] Another study noted a sensitivity of 87% and a specificity of 72% overall.[60] The numbers were better when the clinical examination was sure there was an abscess present and worse if not. Studies of presumed cellulitis in the Emergency Department found that US changed the case management because swelling thought to be due to cellulitis can hide an abscess.[52,57,61] US is useful in evaluating cellulitis in children and determining if an incision and drainage is necessary.[62] US can be used to guide an aspiration, to confirm the presence of an abscess, and to obtain material for culture and Gram's stain.[63]

It is important that the Emergency Physician has an appreciation of the anatomic structures of the area. The location and sonographic appearance of arteries, veins, nerves, and tendons should be known before the incision and drainage procedure. A 5 to 7.5 MHz linear array transducer is generally used. Some find a standoff pad or gel-filled glove to be useful for superficial structures.[54,64]

Scan the area of infection in two orthogonal planes.[54,61] Lower frequency transducers are useful for deeper abscesses, whereas higher frequency transducers are more effective for superficial abscesses.

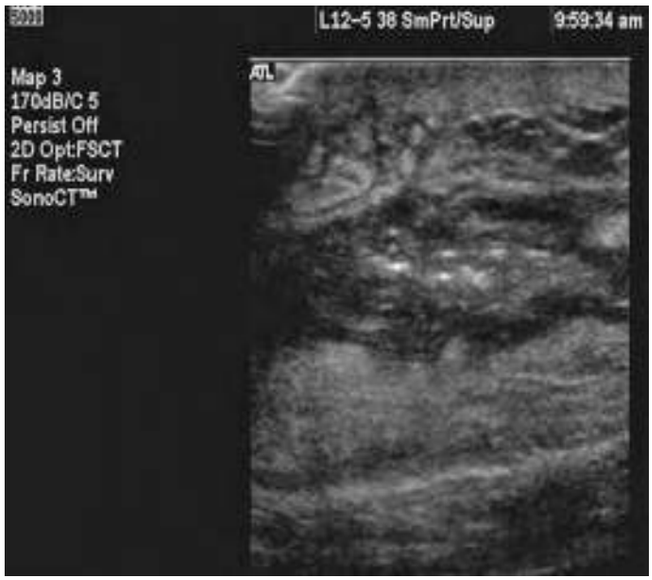

FIGURE 131-1. Ultrasound image of a breast showing cobblestoning of the subcutaneous tissue indicative of edema secondary to cellulitis with echogenic material surrounded by echo-poor areas consistent with early abscess formation.

It is important to use transducer covers as well as proper cleaning and disinfecting supplies to avoid cross-contamination.

Differentiation between a cellulitis and an abscess is based on several findings. Cellulitis shows diffuse hyperechogenicity and thickening of the skin and subcutaneous fat. Echo-poor strands between hyperechoic fatty lobules in the subcutaneous tissue is known as cobblestoning and is indicative of cellulitis (**Figure 131-1**).[53,54,59,63] An abscess has a wider array of presentations on US. An abscess is most frequently a spherical- or elliptical-shaped anechoic or echo-poor region with sharper echogenic borders (**Figure 131-2**).[54,65] Within the echo-poor area there may be gas, lobulations, septations, and echogenic debris. There are some cases where the abscess can appear to be isoechoic or hyperechoic. There may be a role for US-guided aspiration in these cases.[65] Gentle digital palpation or pressure may induce motion of the purulent material within the abscess if unsure if an abscess is present on US.[65] Posterior acoustic enhancement can be seen in the presence of an abscess.[54] This technique can help rule out similarly presenting hematomas, necrotic tumors, vascular lesions, or schwannomas.[63] There is an emerging role for Doppler US in abscess evaluation. The findings of increased

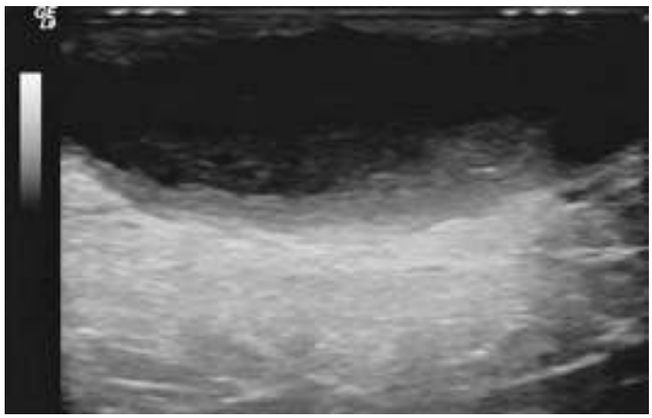

FIGURE 131-2. Ultrasound image showing a large hypoechoic area representing a large superficial abscess.

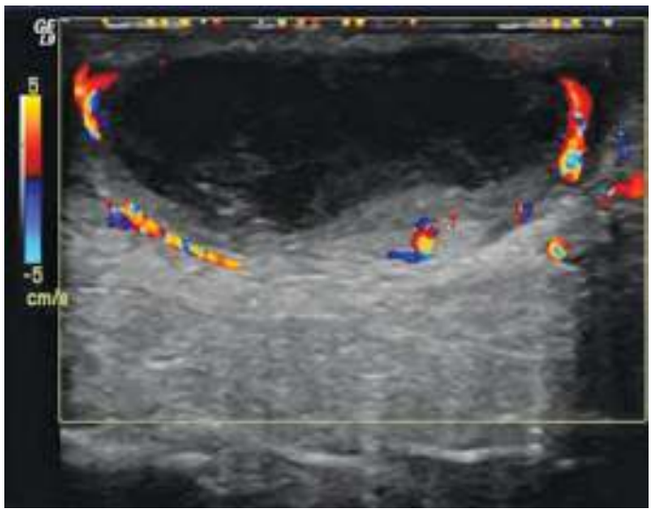

FIGURE 131-3. Color Doppler ultrasound image showing peripheral hyperemia around a large abscess cavity.

vasculature, peripheral blushing and hyperemia, and increased large vessel flow around the abscess are common (**Figure 131-3**).[66]

INDICATIONS

The presence of a fluctuant mass in an area of induration, erythema, and tenderness is clinical evidence that an abscess requires incision and drainage. Antimicrobial treatment without incision and drainage can lead to treatment failures in patients with abscesses.[67] Examination alone may not definitively indicate an abscess, especially if it is deep. This can be further confirmed by bedside US, with or without needle aspiration.[3] Obtaining purulent material on aspiration identifies an abscess and is an indication for incision and drainage.[3] Oral antibiotics, warm compresses, and follow-up in 24 hours must be arranged for a reassessment if no pus is aspirated.

CONTRAINDICATIONS

The only absolute contraindication to the incision and drainage of an abscess is the possible association with a mycotic aneurysm.[12,44] Abscesses commonly overlie large vessels (e.g., the anterior triangle of the neck, the supraclavicular fossa, the deep space of the axilla, the antecubital fossa, the groin, and the popliteal space).[68] Fine-needle aspiration for blood, diagnostic imaging, and/or angiography are indicated prior to incision and drainage in these locations or if the abscess is pulsatile.

Relative contraindications to incision and drainage are common. It includes an inability to achieve adequate anesthesia. An abscess associated with a deep foreign body that requires additional real-time imaging (e.g., fluoroscopy or US) may require surgical referral.[69] Proximity of an abscess to important neurologic, tendinous, or vascular structures may require specialty consultation and magnification in the Operating Room. Deep space infections or involvement of any joint requires admission for parenteral antibiotic therapy and possible operative debridement. Patients presenting with soft tissue infections exhibiting pain out of proportion to physical examination findings or deep anesthesia of the involved or distal area should raise the possibility of deeper infections (e.g., necrotizing fasciitis or myonecrosis).[45] Perirectal and periurethral abscesses are often larger and deeper than they appear and may be complicated by sinus tracts that require exploration under general anesthesia. Manipulation of abscesses in the "danger triangle" of the face (i.e., the corners of the

mouth to glabella) can lead to septic thrombosis of the cavernous sinus. Periorbital or orbital abscesses require a consult from an Ophthalmologist for an assessment and treatment.

EQUIPMENT

ANESTHESIA

- 18 and 27 gauge needles, 1½ inches long
- 10 mL syringes
- Povidone iodine or chlorhexidine solution
- Local anesthetic solution with epinephrine
- Local anesthetic solution without epinephrine if abscess is near an end arteriolar system
- Ethyl chloride spray
- Ice pack

PROCEDURE

- #11 and #15 scalpel blades on a handle
- Hemostats in two sizes for breaking up loculations and probing the cavity
- Scissors
- Normal saline
- 10 or 20 mL syringe with a 20 or 22 gauge angiocatheter
- Suction source, tubing, and catheter for larger abscesses
- 4×4 gauze squares
- Iodoform gauze packing
- Adhesive tape
- Culture swabs and vials
- US machine
- A 5 to 7.5 MHz transducer
- US transducer covers
- US gel
- Standoff pad
- US transducer cleaning and disinfecting solutions

PATIENT PREPARATION

Explain the procedure, its risks, and its benefits to the patient and/or their representative. **Patients should be warned of potential cosmetic complications prior to proceeding.** Obtain an informed consent to perform the procedure. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. Oral regimens should be given 1 hour prior to the procedure and parenteral regimens within 30 minutes of the procedure if endocarditis prophylaxis is indicated (**Table 131-1**).

Clean the skin of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the skin and allow it to dry. Apply drapes to delineate a sterile field. Many Emergency Physicians regard this last step as optional. **The procedure of cutting into a contaminated abscess is done cleanly and not sterile.**

ANESTHESIA

It is usually possible to achieve adequate anesthesia for the skin incision, but any additional manipulation may be painful.[70] Local anesthetic infiltration is often less effective than in other procedures. The pH of infected tissue is often low and retards the diffusion of the local anesthetic solution into nerve axons.[71] A regional field block can be instituted by injecting a ring of 1% lidocaine or 0.5% bupivacaine subcutaneously approximately 1 cm away from the perimeter of the erythematous border of the abscess. The onset of anesthesia occurs after about 5 minutes. A small amount of local anesthetic solution can be injected intradermally into the roof of the abscess in a linear fashion along the line of the planned incision (**Figure 131-4*A***).[3] **Be careful during this portion of the procedure as the abscess may be under pressure. The inadvertent injection of local anesthetic solution into the abscess cavity may cause fluid to be forcibly ejected toward the Emergency Physician. Appropriate universal precautions should be employed including a face mask and eye protection.** The use of a transdermal lidocaine-tetracaine patch (Synera; Chapter 154) provides equal analgesia to injection of lidocaine.[72] Consider using this patch for the incision and drainage of an abscess.

Topical ethyl chloride spray can be used to provide anesthesia for superficial abscesses or furuncles which are unlikely to require significant exploration. Invert the bottle and compress the spray nozzle to begin the flow of fluid. Direct the spray toward the planned site of incision. The pain relief from ethyl chloride is variable and fleeting. It is also highly flammable.[4] The application of an ice pack over the planned incision site secured with an elastic bandage for 15 minutes can also be effective. This may be especially useful for children, patients with severe needle phobias, or in cases of a true anesthetic allergy.

Nitrous oxide is safe and was previously thought to be effective as an adjunct to the incision and drainage of abscesses.[73] More recently, it has been shown to cause no significant reduction in pain and to only be marginally effective as an anxiolytic.[74] Procedural sedation (Chapter 159) can be useful in deep abscesses that require extensive probing.[75] It is difficult to obtain adequate pain control with a field block.

TECHNIQUES

ASPIRATION

Aspiration is performed as a diagnostic procedure in cases of soft tissue infections where the presence of an abscess is unclear, a mycotic aneurysm must be ruled out, or samples for Gram's stain and culture are required. **Aspiration is not a therapeutic procedure.** Anesthetize the skin. If US is available, locate the abscess cavity and determine the shortest needle path. Insert an 18 gauge needle attached to a 10 mL syringe into the skin. The needle appears as a bright, hyperechoic line with posterior reverberation artifact.[63] The needle will become increasingly oblique as it penetrates deeper. Apply negative pressure to the syringe.

Advance the needle into the area where pus or blood is presumed to be loculated. Terminate the US-guided aspiration and perform an incision and drainage if pus is obtained. Inoculate anaerobic and aerobic culture bottles directly from the syringe if cultures are indicated. Simple swabbing of the purulent material after incision and drainage is inadequate for growth of anaerobic organisms. Blood aspirated signifies that the needle tip is in a vascular structure. **Terminate the procedure and apply firm pressure to the area to prevent a hematoma from forming if blood is aspirated.** Angiography with surgical consultation should immediately follow. Redirect the needle in several directions to confirm the absence of an abscess if no pus or blood is aspirated. Discharge the patient with oral antibiotics, warm compresses, and instructions to follow-up in 24 hours for a reassessment.

INCISION AND DRAINAGE

Make an incision spanning the entire area of fluctuance and parallel to the relaxed skin tension lines to reduce scarring.[44,76] A straight incision with a #11 scalpel blade is usually performed

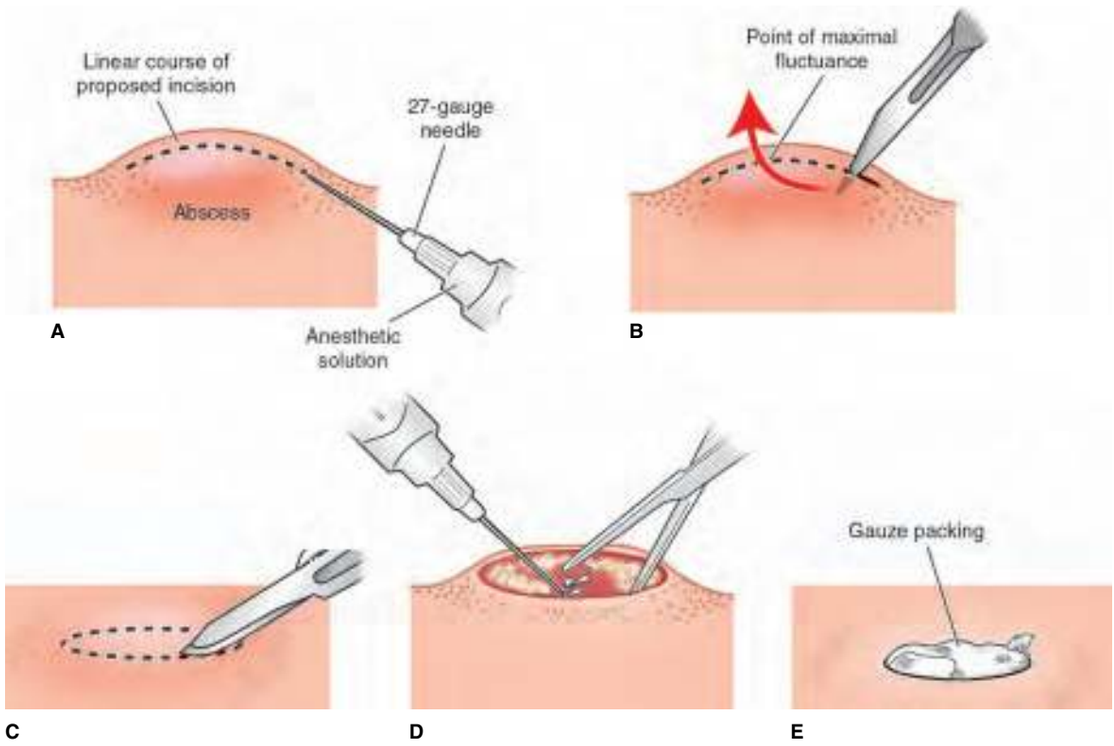

FIGURE 131-4. Incision and drainage of a subcutaneous abscess. ***A.*** Infiltration of local anesthetic solution over the abscess. ***B.*** A straight incision to drain the abscess. ***C.*** An elliptical incision to drain the abscess. ***D.*** The wound is irrigated with sterile saline. Any pockets of pus are opened by blunt dissection with the hemostat. ***E.*** The wound is packed open.

(**Figure 131-4*B***). An elliptical incision with a #15 scalpel blade is an alternative and often results in a similar-appearing final scar (**Figure 131-4*C***). The purpose of the elliptical incision is to remove a full-thickness wedge of tissue so that the wound will remain open. **Do not make an elliptical incision in cosmetically sensitive areas (e.g., face, neck, or breasts) or in areas with minimal subcutaneous tissue (e.g., hands and feet). Ensure a large enough incision is made to promote adequate drainage except for cosmetically sensitive areas where a stab incision may be initially attempted to limit scar formation.**[4]

Debride any necrotic or devitalized tissue. Probe the cavity by inserting a hemostat. Gently spread the jaws open to break up any loculations and to release any further pockets of purulent material (**Figure 131-4*D***).[6] Rotate the hemostat around the entire abscess to break any loculations. **Do not use a scalpel for the blunt dissection of an abscess cavity as it may cause additional tissue damage and bacteremia.**[77] Remove the tough shiny capsule by grasping the edges with a hemostat and applying firm traction if the abscess is due to an infected sebaceous cyst. The capsule can often be removed intact.

Irrigate the abscess cavity with an 18 gauge angiocatheter attached to a 10 mL syringe containing sterile saline (**Figure 131-4*D***). An alternative is the Splash Cap (BSN Medical Inc., Charlotte, NC). The advantages of this device are that it attaches to saline bottles, is disposable, and shields from contaminated spray when irrigating (**Figure 131-5**). This will flush away all loosened purulent and necrotic material.

Loosely pack iodoform gauze into the abscess cavity (**Figure 131-4*E***).[76] Leave 1 to 2 cm of gauze exiting from the cavity to prevent the incision from sealing over and ensure an adequate drainage tract for the cavity. The value of antiseptic impregnated gauze over plain gauze is uncertain. Do not overpack the abscess cavity as this may interfere with the inflammatory hyperemia necessary for healing, retard drainage, and reproduce "abscess-like" conditions.[6] Apply an absorbent dressing of 4×4 gauze over the wound. Splinting and elevation of the affected area may be beneficial in select patients (e.g., young patients and confused patients).

FIGURE 131-5. A Splash Cap on a saline bottle. (Photo courtesy of Joseph Schultz, MD.)

LOOP DRAINAGE TECHNIQUE

The loop drain technique confers several advantages over the traditional incision and drainage. Smaller incisions result in better cosmesis. The incision drains while the loop is in place and eliminates packing changes. This is especially beneficial in the pediatric population for whom packing changes and wound care may be difficult. A drain will be needed in addition to the usual equipment. This can be a silicone vessel loop, a small Penrose drain, a sterile rubber band, sterile suture, or a piece of a sterile glove.[78]

The skin is cleaned, draped, and anesthetized in the usual fashion. Make an initial 0.5 to 1 cm incision at one end of the abscess and if possible where the abscess is pointing. Use a hemostat to break up any loculations (**Figure 131-6*A***). Express as much pus as possible. Obtain cultures if clinically indicated. Use the hemostat to probe the abscess and tunnel through until the opposite end of the cavity is reached. Continue to express any pus present.

Tent the skin and make a second incision over the tip of the hemostat (**Figure 131-6*B***). The distance between the two incisions should only be a few centimeters. Irrigate the wound with sterile saline. A syringe and plastic catheter will provide adequate pressure for irrigation. An alternate is to use a Splash Cap (**Figure 131-5**).

Insert the drain. Use the hemostat to pull the drain through the second incision and exit the first incision (**Figures 131-6*C* and 131-6*D***). The drain should be under the skin and traverse the length of the abscess cavity. Tie the two loose exposed ends of the drain in a loose loop. Use a finger or a syringe as a placeholder to avoid excessive skin tension when the tying the loop (**Figure 131-6*E***). Tie multiple knots to secure the drain (**Figure 131-6*F***). Remove the syringe, if used, and trim the excess free ends of the drain. Large or irregularly shaped abscesses will need multiple loops for adequate drainage (**Figure 131-7**). Cover the skin puncture site with gauze.

AFTERCARE

Most available evidence suggests that incision and drainage of subcutaneous abscesses alone is adequate treatment and additional antimicrobial therapy is unnecessary in healthy patients with no major comorbidities.[7,47,48,79-81] Many recent studies involving abscesses, including those caused by MRSA, have demonstrated that patients have similar outcomes regardless of whether appropriate antibiotics were used after an abscess incision and drainage.[18,28,82-84] Pediatric studies have shown no difference in treatment failure based on the size of an abscess or surrounding cellulitis.

A

B

C

D

FIGURE 131-6. The loop drainage technique. ***A.*** A hemostat is used to break loculations. ***B.*** The second skin incision is made. ***C.*** The hemostat grabs the loop material. ***D.*** The loop material is pulled through the incisions. ***E.*** The loop material is loosely tied. ***F.*** The appearance of the completed loop. (Photos used with permission from reference 101.)

E

F

FIGURE 131-6. (*Continued.*)

Treatment failure was higher in those presenting with fever and age less than 1 year.[85-87]

Antimicrobial therapy should be considered if systemic signs of sepsis are present, in immunocompromised patients, if the source control is poor, in patients with multiple abscesses, in patients at the extremes of age, in patients with a lack of response to incision and drainage, or if there is significant cellulitis.[5,22,50,51,85] Consider MRSA-specific antibiotics for patients at risk for MRSA.[5,50,51] Recommendations from the Centers for Disease Control and Prevention suggest antibiotics after incision and drainage in patients with rapid progression, comorbidities (e.g., diabetes mellitus), human immunodeficiency virus, or neoplastic disease; patients at extremes of age; patients with abscesses in locations that are difficult to drain or that have a risk of septic phlebitis associated with it; and in patients who fail to respond to incision and drainage alone.[22,88] Pediatric patients with abscesses greater than 5 cm should also receive empiric antimicrobial therapy.[82]

Many oral antibiotics (e.g., cephalexin, dicloxacillin, and trimethoprim-sulfamethoxazole) can be a good first choice for healthy individuals in areas where the prevalence of MRSA is extremely low.[22,51,89] **Knowledge of local MRSA prevalence and antimicrobial susceptibility patterns is paramount when selecting the appropriate antibiotic.**[22,44,50,51] Empiric treatment with agents that show activity against MRSA is warranted if more than 10% to 15% of community *S. aureus* isolates are MRSA.[90-92] US may be used to identify those abscesses that are caused by MRSA (e.g., ill-defined edges, indistinct, irregular shaped, or small).[93,94] Current guidelines for outpatient therapy suggest using trimethoprim-sulfamethoxazole, clindamycin, doxycycline, minocycline, or linezolid.[51,89,92] Intravenous therapy for patients with significant comorbidities and signs of systemic infection should include clindamycin, ceftaroline, daptomycin, linezolid, tigecycline, or vancomycin.[50,51,95] Clindamycin, doxycycline (if over the age of 7 years), or trimethoprim-sulfamethoxazole is recommended in healthy children.[96] Guide antibiotic selection using the current literature and consideration of the local antibiotic resistance patterns.[22,51,97] Administer antimicrobial therapy for at least 5 days. Consider extended treatment or a different regimen if the infection has not improved in 5 days.[51]

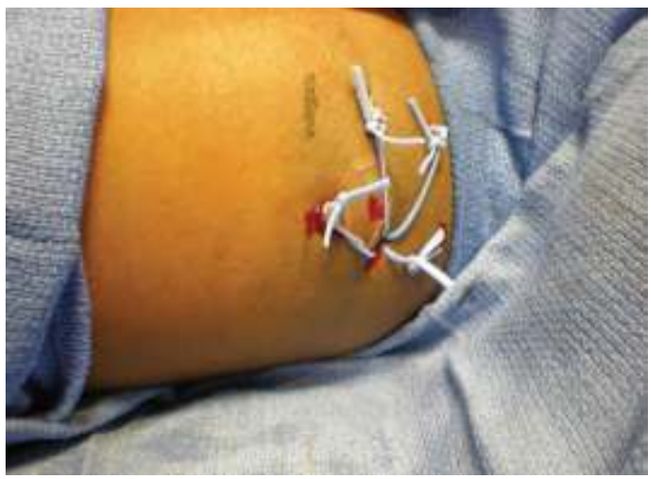

FIGURE 131-7. Large or irregular abscesses may require more than one loop. (Photo used with permission from reference 101.)

The patient should follow up in 24 to 48 hours to remove the packing and assess the response to therapy. Pain can be adequately controlled with the use of acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDS). Narcotic analgesics are rarely required. Instruct the patient to immediately return to the Emergency Department if they develop fever, chills, increased pain, increased swelling, or increased redness to the surrounding skin.

Repack the cavity approximately every 48 hours until granulation tissue develops throughout the wound and the drainage tract is well established if a large amount of drainage continues.[98,99] At that time, the remaining packing is removed and the patient is instructed to soak the area in warm water three to four times per day.[30] Healing occurs in 5 to 9 days in most cases.[3,6,30] The patient may be discharged from medical care when all signs of infection (e.g., erythema, drainage, pain, and induration) have resolved.

The aftercare with a loop drainage is easier than with the traditional incision and drainage. The patient can bathe and shower as usual. Continue warm compresses over the area. Change the dressing one to two times a day, or more frequently if saturated. Gently pull the loop daily to help keep the wound open. Remove the drain by cutting the loop and pulling it out when the drainage stops. The small incision sites will heal with better cosmesis than a large incision by secondary intention.[100,101] The loop technique does not involve the pain of packing.[70] The abscess resolution and complications are similar to those of the traditional technique.[102]

FUTURE ADVANCES IN ABSCESS MANAGEMENT

It is currently standard practice to pack an abscess cavity after an incision and drainage.[51] Packing is used for hemostasis, to keep the abscess cavity from closing prematurely, and to debride the abscess cavity. **Packing an abscess cavity can be painful and requires multiple follow-up visits.** These visits are at an additional cost to the patient. Several small studies of noncomplicated abscesses found that not packing an abscess cavity resulted in less pain, less analgesic use, and no increased morbidity.[103-106] To pack or not to pack an abscess cavity is up to the Emergency Physician and the informed patient. An elliptical incision or the loop technique does not require packing.

Incision and drainage of abscesses usually requires flushing of the abscess cavity. Irrigation of abscesses after incision and drainage did not improve outcomes.[107] The irrigation group in the small study was from a single site, younger, more often treated with packing, and treated with antibiotics, confounding results. Approximately only half of Emergency Physicians irrigate the abscess cavity even though it is recommended by guidelines.[108]

Abscesses are currently allowed to heal by granulation. This process can take weeks and results in large scars. Primary suture closure after incision and drainage has been used around the world except in the United States.[109,110] This treatment can reduce scarring, reduce pain, and promote faster healing. Most of the studies involved a small number of patients, administered preprocedural antibiotics, and were performed in the Operating Room. Further studies are required before this change in practice can be recommended for the Emergency Department management of abscesses.

DECOLONIZATION AND PREVENTION

Decolonization regimens after incision and drainage do not have established effectiveness.[20,22,48] A decolonization regimen may play a role in patients with recurrent infections, patients not responding to therapy, or patients with a closely associated cohort at risk (e.g., infirm family, sports teams, or the institutionalized).[49] Colonization rates vary by geography and colonization is considered a risk factor for MRSA infection. Decolonization is not routinely recommended due to limited supporting evidence, poor patient compliance, and the risk of increasing antibiotic resistance.[25,48,111]

Patient education is the most effective way to reduce the spread of infection and recurrent infections from MRSA.[22,25,111] Maintaining a clean and dry wound, frequent handwashing and bathing, avoiding sharing any personal items, laundering clothing that has come into contact with the wound, avoiding skin to skin contact, and disinfecting equipment and other surfaces are essential recommendations that should be made to outpatients with MRSA skin and soft tissue infections.[22,111]

COMPLICATIONS

Complications resulting from the incision and drainage of an abscess are uncommon. Scarring will result from deliberate open packing and secondary intention granulation of the wound. Infectious complications (e.g., inciting bacterial endocarditis) is possible. **Endocarditis can be avoided with appropriate screening of patients and the administration of prophylactic antibiotic therapy in patients at risk.** Precipitation of septicemia because of transient bacteremia in an immunodeficient patient must be considered prior to the procedure. Incision and drainage of a mycotic aneurysm should not occur if an appropriate assessment is completed prior to making the incision. The use of US with Doppler can determine the difference between an abscess and a vascular structure (**Figure 131-8**).

A

B

FIGURE 131-8. Ultrasound of an abscess. ***A.*** The abscess. ***B.*** Doppler showing lack of vascularity of an abscess. (Courtesy of Lloyd Gordon, MD.)

SUMMARY

Simple incision and drainage under local or regional anesthesia in the Emergency Department can effectively treat most subcutaneous abscesses. Adjunctive procedural sedation may be required to adequately probe a deep cavity. A directed history and physical examination will identify patients who may require additional lab work, imaging, specialty consultation, and follow-up. Most patients will not require antibiotics, although there may be a role for ancillary treatment in selected patients with MRSA infections. Attention must be paid to requirements for endocarditis prophylaxis and consideration given to the possibility of inducing bacteremia in any given patient.

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132 Paronychia or Eponychia Incision and Drainage

Lisa R. Palivos and Tim Richardson

INTRODUCTION

A paronychia is inflammation of the soft tissue surrounding the nail plate, commonly caused by an infection or an abscess (**Figure 132-1**). It is the most common infection of the hand.[1,2] A paronychia can be located on the fingers or the toes. It occurs in all age groups. It can cause significant pain and discomfort leading to a visit to the Emergency Department.

A paronychia initially presents with redness, swelling, and tenderness along the edges of the nail plate (**Figure 132-1**). This can progress to an abscess that requires drainage. An infection that extends to the overlying proximal cuticle is termed an eponychia. This chapter discusses the treatments which vary with the extent and the location of the infection.

ANATOMY AND PATHOPHYSIOLOGY

The dorsal aspect of the distal digit consists of the nail plate, the nail bed (i.e., matrix), and the perionychium (**Figure 132-2**). The nail bed is situated beneath the nail plate and is responsible for growth of the nail. The perionychium is the soft tissue surrounding the nail plate. It is composed of the eponychium proximal to the nail plate and the lateral nail folds.

A paronychia is usually the result of minor trauma to the seal formed by the nail plate and nail fold.[3] This may occur from tight-fitting apparel (e.g., gloves, pantyhose, and shoes), aggressive manicures, the use of artificial nails, hangnails or ingrown nails, foreign bodies, or nail biting.[3,4] The disruption of this seal allows bacteria to enter causing a localized cellulitis that may progress to abscess formation in the potential space between the nail plate and nail fold (**Figures 132-1 and 132-2**).[1,5] A paronychia can grow and spread under the nail plate causing a subungual abscess. The infection may advance to the volar soft tissues and deep structures resulting in a felon, osteomyelitis, or tenosynovitis.[6]

Most paronychia are polymicrobial. They often contain a mixture of aerobic and anaerobic organisms.[7,8] The most common organism to cause a paronychia is *Staphylococcus aureus*.[9] Paronychia are often caused by anaerobes secondary to finger sucking or nail biting in children and nail biters.[10] Gram-negative organisms should be considered in immunocompromised hosts. The etiology of a chronic paronychia is complex and likely multifactorial. Patients are often colonized with *Candida albicans* but the infection is not likely the cause.[11] Chronic paronychia refractory to steroid and pathogen-directed therapies must be considered as result of a neoplasm (e.g., subungual melanoma or squamous cell carcinoma).[12]

FIGURE 132-1. Paronychia of the middle finger. (Used from Chris Craig at www.commons.wikimedia.org.)

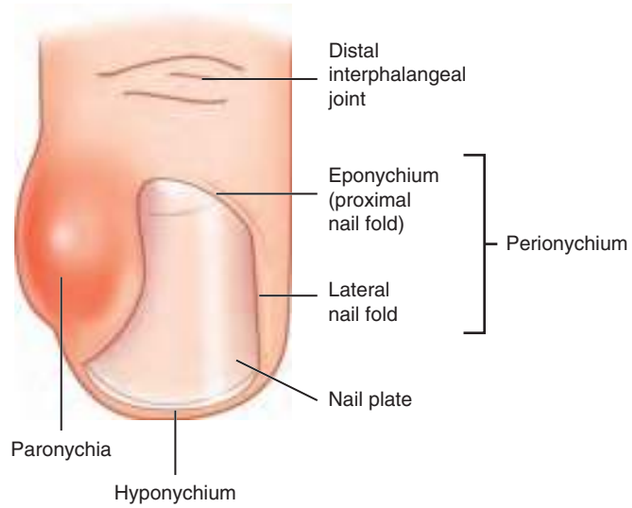

FIGURE 132-2. The distal finger illustrating a paronychia and the surface anatomy.

INDICATIONS

An early paronychia will present with signs of cellulitis, redness, and tenderness without fluctuance of the nail fold. This may be treated nonsurgically with warm water soaks six to eight times per day using chlorhexidine or povidone solutions.[13] The addition of oral antibiotics to cover *Staphylococcus*, plus anaerobic coverage if nail biting or finger sucking were causative, is recommended if symptoms persist.[14,15] Amoxicillin-clavulanate, clindamycin, and trimethoprim-sulfamethoxazole are commonly used antibiotics. **The prevalence of methicillin-resistant *S. aureus* (MRSA) in your community will help decide the choice of antibiotics.**

A progression of the infection results in fluctuance and the formation of an abscess. The digital pressure test may be used to diagnose the presence of an abscess when the exam is otherwise equivocal. Instruct the patient to oppose the thumb and affected finger.[16] Apply light pressure to the distal volar aspect of the affected digit. The skin under the nail plate will blanch if an abscess is present.[17] **The presence of an abscess, fluctuance, or pus beneath the nail plate requires an incision and drainage procedure.** This procedure will relieve the patient's pain, promote healing, and prevent complications from local extension into surrounding bone and soft tissues.

CONTRAINDICATIONS

A herpetic whitlow is a herpes simplex virus infection of the distal phalanx that can be confused with an early paronychia or felon.[13,18] The presence of multiple clear vesicles that coalesce suggests a herpetic whitlow. The herpetic whitlow is a nonsurgical and self-limited infection. Treatment consists of a dry dressing to the affected finger to prevent autoinoculation and transmission of the infection, oral antiviral agents, and analgesics. Incision and drainage is not recommended as it will prolong the recovery and may lead to secondary

bacterial infection.[19] A chronic paronychia should be referred to a Hand Surgeon or Dermatologist for treatment.[20]

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile gloves
- #11 scalpel blade or an 18 gauge needle
- Local anesthetic solution without epinephrine
- 18 and 27 gauge needles
- 5 to 10 mL syringe
- Ethyl chloride spray
- Forceps
- Mosquito hemostat
- Ribbon gauze packing, ½ inch wide
- Petrolatum gauze, ½ inch wide
- Scissors
- 4×4 gauze squares
- 18 gauge angiocatheter
- 20 mL syringe
- Sterile saline
- Adhesive tape

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Include in the informed consent any anesthetic technique used in the procedure. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Prepare the patient. Assess and update the patient's tetanus immune status if indicated. Place the patient on a gurney with the extremity on a bedside procedure table in a well-lit room. Soak the digit in warm water for 5 minutes to soften the skin. Perform a digital nerve block (Chapter 130) if the patient is apprehensive, has significant tenderness, or if it is not a simple paronychia or eponychia. Yellow or white overlying tissue may indicate that local sensory nerves have infarcted which makes anesthesia unnecessary.[12] Alternatively, apply ethyl chloride spray to the area until the skin turns frosty and pale (Chapter 154). Apply povidone iodine or chlorhexidine solution circumferentially to the digit and allow it to dry. The digit can be secured to a sterile tongue depressor for better control, especially in uncooperative children.

TECHNIQUES

SIMPLE PARONYCHIA OR EPONYCHIA

There is no need for a skin incision in an uncomplicated paronychia or eponychia. Simply lifting the eponychium off the nail plate at the point of maximal tenderness and/or fluctuance is usually curative. Slide the tip of a #11 scalpel blade or an 18 gauge needle under the paronychia, or eponychia, at the site of maximal fluctuance (**Figure 132-3**). Advance the scalpel blade to lift the soft tissue from the nail plate until there is an efflux of purulent fluid (**Figure 132-4*A***). Apply digital pressure to the area to express the pus. Gently place a hemostat under the soft tissue to break any loculations (**Figure 132-4*B***). Irrigate the pocket with an angiocatheter on a syringe containing sterile saline.

FIGURE 132-3. Drainage of a simple paronychia or eponychia. The eponychial fold is elevated from the nail plate with a #11 scalpel blade. Note that the blade is parallel to the nail plate, thereby avoiding injury to the nail matrix and not incising the skin.

Packing a paronychia is controversial. Place a small piece of ribbon gauze or petrolatum gauze under the elevated soft tissue (**Figure 132-4*C***). Apply an antibiotic ointment (**Figure 132-4*D***) followed by a simple dressing or bandage (**Figure 132-4*E***). Oral antibiotics are not recommended for an uncomplicated paronychia or eponychia.[21]

PARONYCHIA WITH EXTENSION UNDER THE LATERAL NAIL PLATE

A more extensive incision and drainage is required when pus accumulates laterally and beneath the nail plate as a subungual abscess. Remove the lateral nail plate (Chapter 218) to allow adequate drainage (**Figure 132-5*A***). Use scissors to cut the nail plate longitudinally. **Aim the point of the scissors upward and against the undersurface of the nail plate to prevent injuring the nail bed.** Remove the lateral nail plate with a hemostat. A small (i.e., 2 to 3 mm) incision may be required in the corner of the nail fold to remove the nail plate (**Figure 132-5*A***). This will result in the egress of pus. Irrigate the area with saline. Insert a small piece of petrolatum gauze under the nail fold (**Figure 132-5*B***). This will prevent the nail fold from fusing to the nail bed.

PARONYCHIA WITH EXTENSION UNDER THE PROXIMAL NAIL PLATE

A paronychia with extension under the proximal nail plate forming a subungual abscess requires removal of a portion of the proximal nail plate (**Figure 132-6**). Make two 3 to 4 mm long incisions at the corners of the nail folds to access the proximal edge of the nail plate (**Figure 132-6*A***). Alternatively, bluntly raise the eponychium taking care not to damage the nail matrix.[22] Cut the proximal one-third of the nail plate with scissors (**Figure 132-6*B***). **Aim the point of the scissors upward and against the undersurface of the nail plate to prevent injuring the nail bed.** Grasp and remove the proximal segment of the nail plate with a hemostat. This will result in the egress of pus. Irrigate the area with saline. Insert a piece of petrolatum gauze under the nail fold to prevent it from fusing to the nail bed (**Figure 132-6*C***).

Removal of the entire nail is rarely necessary except in the case of an extensive subungual abscess. An alternative to nail removal is trephination (Chapter 129) with a heated paper clip, a heat pen, or a microcautery unit.[22] A large opening or multiple holes are required with this technique to eliminate the pus.

CHRONIC PARONYCHIA

Chronic paronychia result from inflammation secondary to prolonged exposure to finger biting, finger sucking, chemical irritants, cuticle trimming, moisture, a neoplasm, or psoriatic conditions.[20,22,23]

A

B

C

D

E

FIGURE 132-4. Management of a paronychia. ***A.*** The skin fold is elevated with a #11 blade. ***B.*** A hemostat is inserted to break loculations. ***C.*** The paronychia is packed. ***D.*** Antibiotic ointment is applied. ***E.*** A dressing has been applied. (Used with permission from reference 5.)

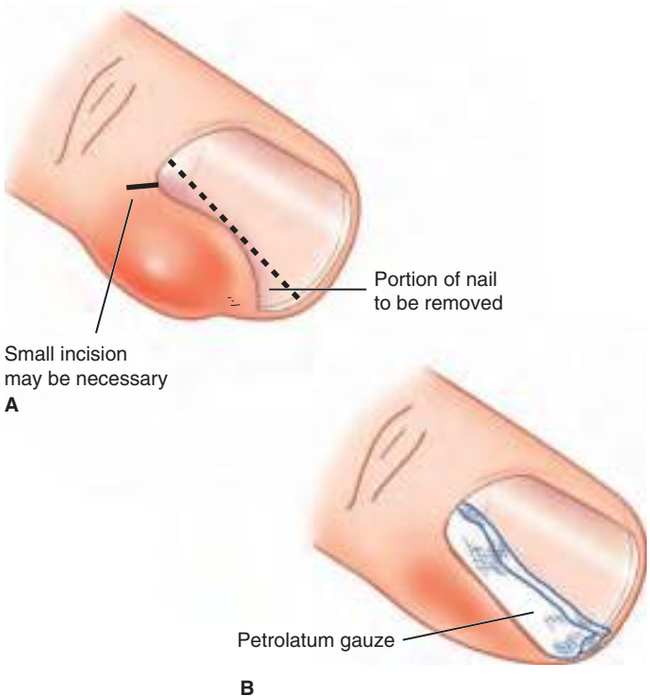

FIGURE 132-5. Removal of the lateral nail plate is required when pus extends laterally below the nail plate. ***A.*** The dotted line over the nail represents the incision required to remove the nail plate. An additional incision may be required on the eponychium. ***B.*** The lateral portion of the nail plate has been removed and petroleum gauze packing has been inserted to keep the nail fold elevated from the nail bed.

C. albicans has long been implicated in the pathogenesis. Studies suggest that chronic paronychia may not result from a primary mycotic infection but rather secondary colonization. Patients with diabetes or immunosuppression are more susceptible. Excessive hand washing is the likely culprit in occupational exposures of dish washers and health care workers. Treatment is likely not definitive in the Emergency Department. It is much more difficult to treat and eradicate than an acute infection. Removal of the causal irritant and topical steroids are first-line therapies. Favorable outcomes have been shown using topical steroids versus systemic antifungals.[11,13] Patients who are not responsive to common interventions must be evaluated for a malignancy.

Definitive treatment for refractory chronic paronychia is eponychial marsupialization. This involves removal of a crescent-shaped piece of skin proximal to the nail fold and parallel to the eponychium. Complete or partial nail removal may be necessary if nail ridging is present.[24] A chronic paronychia may be confused with another condition that looks similar and mimics a chronic paronychia such as cysts, foreign body reactions, malignancies, psoriasis, and verrucae.[23,25] Refer all chronic paronychia to a Hand Surgeon due to the higher rate of recurrence, the complexity in management, and where follow-up care can be more consistent.

AFTERCARE

Immobilize and elevate the digit. Instruct the patient to avoid nail biting or sucking. Any discomfort can be treated with acetaminophen or nonsteroidal anti-inflammatory medications. Follow-up care in 24 hours is important as complications can occur despite proper Emergency Department management. Packing of a simple paronychia should be removed in 24 hours. Warm soaks can begin immediately if packing is not placed. Delay warm soaks until the packing is removed in 24 hours. **There is no evidence that oral antibiotics improve outcome after the incision and drainage of a simple and uncomplicated paronychia.**[21] Oral antibiotics are not necessary unless the nail bed is involved, there is apparent cellulitis of the surrounding tissue, or there are systemic signs of infection such as lymphangitis and fever.[26] Patients should return to the Emergency Department if they develop a fever, reaccumulation of pus, redness extending up the finger and hand, or increased tenderness to the digit.

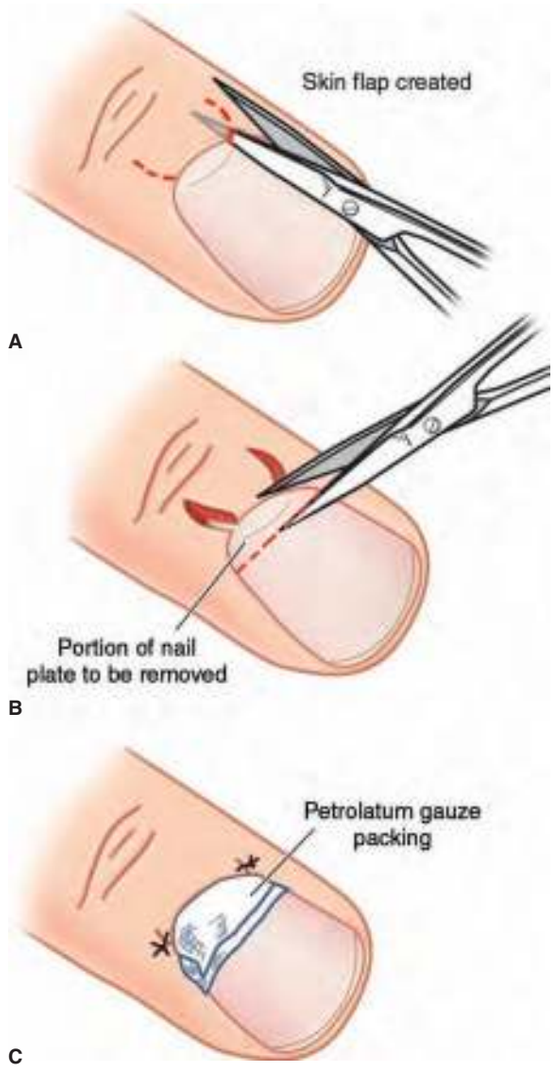

FIGURE 132-6. Removal of the proximal nail plate is required when pus extends proximally below the nail plate. ***A.*** Two incisions are required through the eponychium, represented by dotted lines. ***B.*** The dotted line over the nail plate represents the incision required to remove the nail plate. ***C.*** The proximal portion of the nail plate has been removed and petrolatum gauze packing has been inserted to keep the nail fold elevated from the nail bed.

Most simple paronychias resolve within a few days. If they persist longer or recur, consult a Hand Surgeon for more aggressive management (e.g., eponychial marsupialization and nail plate removal).

Paronychia or eponychia that extend under the nail plate require follow-up in 24 hours. The packing must be maintained between the nail fold and the nail bed for at least 5 to 7 days. **The nail fold will fuse to the nail bed if the packing is removed too soon and a new nail plate will not form.** These infections require a 5 to 7 day course of oral antistaphylococcal antibiotics. Nonsteroidal anti-inflammatory medications supplemented with occasional narcotic analgesics will provide adequate pain control for these patients.

COMPLICATIONS

Complications occur in even a properly drained paronychia (e.g., a felon, osteomyelitis of the distal phalanx, or a tenosynovitis). Superinfection with *C. albicans* or other fungi can also occur. These complications can be due to the paronychia itself or result from an inadequate incision and drainage procedure.

Complications from the incision and drainage procedure are rare if it is properly performed. Care must be taken if the lateral or proximal nail plate is removed to avoid damaging the underlying matrix so that a nail deformity does not result. Fusion of the nail fold to the nail bed will result in a new nail not being formed. Inadequate drainage can result in the infection spreading to adjacent bone and soft tissues. Incision of the skin instead of elevating it off the nail plate can result in prolonged healing.

SUMMARY

A paronychia is one of the most common hand infections. The treatment depends on the extent and location of the infection. The incision and drainage procedure is quick, simple, and easy to perform. Simple paronychias require elevation of the nail fold with no incision. A more extensive incision and drainage is required along with nail excision when pus accumulates below the nail plate. Follow-up is critical as complications may occur, even when the Emergency Department treatment is optimal.

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133 Felon Incision and Drainage

Lisa R. Palivos and Sonali Gandhi

INTRODUCTION

A felon is a subcutaneous infection or abscess in the pulp space on the palmar aspect of the fingertip (**Figure 133-1**). It is usually caused by penetrating trauma, an abrasion, spread from adjacent tissues (e.g., eponychium, osteomyelitis, or paronychia), or a minor cut that leads to the invasion of bacteria.[1] A felon can develop in the presence of a foreign body (e.g., wood splinter or thorn).[2] It can be iatrogenic from multiple fingersticks for blood glucose measurement.[3,4] The offending organism is usually *Staphylococcus aureus*.[5] Mixed infections and gram-negative infections may occur in the immunocompromised patient. A felon can less commonly occur on the toes. The information in this chapter can be applied to a felon of the finger or the toe.

Felons initially present with a gradual onset of pain and erythema of the distal volar finger consistent with symptoms of cellulitis.[1] Intense throbbing pain, warmth, and swelling develop with the formation of an abscess as the infection progresses. **The definitive management for a felon is incision and drainage.** There are multiple techniques to incise and drain a felon. The patient requires digital elevation, immobilization, oral antistaphylococcal antibiotics, oral analgesics, and close follow-up to prevent complications following the incision and drainage.[6-10]

FIGURE 133-1. An example of a felon. (Used with permission from Knoop KJ, et al: *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2016. Photo contributor: Daniel L. Savitt, MD.)

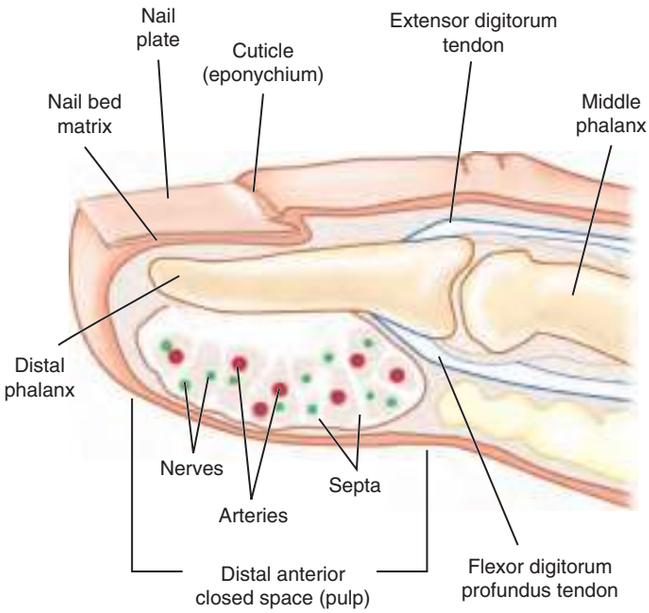

FIGURE 133-2. Midsagittal section demonstrating the anatomy of the distal finger.

ANATOMY AND PATHOPHYSIOLOGY

The distal finger consists of a closed compartment that is bound by the nail plate dorsally, the skin ventrally and distally, and the flexion crease proximally (**Figure 133-2**). The fingertip pulp region is divided by 15 to 20 vertical fibrous septa.[11] These septa extend from the volar surface of the fat pad to the periosteum of the distal phalanx. They divide and compartmentalize the pulp area. An abscess in this enclosed space is confined by the septa which limits the proximal spread of an infection.[12] The septa inhibit the abscess from reaching the surface and inhibit drainage after the incision and drainage procedure. Blood is supplied by branches of the digital arteries that run parallel and lateral to the phalanx and terminate in the pulp region. The terminal branches of the digital nerves lie palmer and superficial to the arteries. The flexor digitorum profundus tendon inserts on the volar surface of the proximal distal phalanx.

INDICATIONS

All felons that are fluctuant should be incised and drained. Volar digital pads that are tense, tender, painful, and suspected of containing a felon should be evaluated for incision and drainage regardless if fluctuance is palpated or not.

CONTRAINDICATIONS

Felons that are not yet fluctuant, as in an early infection, may be treated with warm soaks, elevation, oral antibiotics, and follow-up in 24 hours.[9,10] A herpetic whitlow can sometimes be confused with a felon.[5,13] A herpetic whitlow can be clinically distinguished by the presence of multiple vesicles and a history of recurrence or simultaneous lesions (e.g., genital or oral). Treatment of a herpetic whitlow is nonsurgical and consists of a protective dry dressing, oral antiviral agents, and analgesics. Incision and drainage of a herpetic whitlow may spread the virus and predispose the patient to secondary bacterial infection.[13]

Consult a Hand Surgeon for complicated felons. This includes a felon that is associated with lymphangitis, osteomyelitis, a flexor tenosynovitis, an infection that has spread proximal to the distal interphalangeal joint, or if the patient is immunocompromised. These patients require hospital admission, intravenous antibiotics, and possible incision and drainage in the Operating Room.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile gloves
- #11 scalpel blade on a handle
- Local anesthetic solution without epinephrine
- 18 and 27 gauge needles
- 5 or 10 mL syringe
- 20 mL syringe
- Ethyl chloride spray
- Sterile saline
- 18 gauge angiocatheter
- Mosquito hemostat
- Ribbon gauze, ½ inch wide
- Bandage material
- Digital splint (e.g., plaster, preformed, or tongue depressor)
- Sling
- Digital tourniquet, optional
- Aerobic and anaerobic culture bottles, optional

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. Assess and update the patient's tetanus immune status if indicated.

Some physicians prefer to obtain anteroposterior and lateral radiographs of the digit to rule out an osteomyelitis or foreign body prior to performing the procedure. A positive radiograph for osteomyelitis will alter the time course for antibiotic therapy and require follow-up with a Hand Surgeon.

Place the patient on a gurney with the extremity on a bedside procedure table in a well-lit room. Soak the digit in warm water for 5 minutes to soften the skin. Perform a digital nerve block (Chapter 156) if the patient is apprehensive, has significant tenderness, or if it is not a simple felon. Alternatively, apply ethyl chloride topical spray (Chapter 154) to the area until the skin turns frosty and pale. Apply povidone iodine or chlorhexidine solution circumferentially to the digit and allow it to dry. Apply sterile drapes to delineate a sterile field. The digit can be secured to a sterile tongue depressor for better control, especially in uncooperative children. The application of a digital tourniquet (Chapter 129) to create a bloodless field is optional.

TECHNIQUES

Multiple incisions can be employed. Make an incision in the area of greatest fluctuance or tenderness with a #11 scalpel blade.[6-9] Make a longitudinal incision if the maximal tenderness is in the center of the pulp of the distal fingertip (**Figure 133-3*A***).[1,5] **The incision should not come within 4 mm of or cross the crease of the distal interphalangeal joint as this can lead to injury of the flexor digitorum longus tendon or the joint, flexor tenosynovitis, and flexion contractures.**[10] Make the incision along the lateral surface of

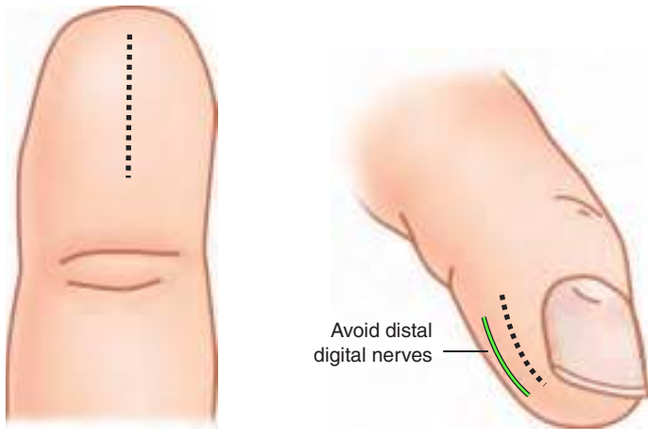

FIGURE 133-3. Recommended incisions for the incision and drainage of a felon. A felon should be incised and drained in the area of maximal fluctuance. ***A.*** The longitudinal fat pad incision over the area of maximum fluctuance. ***B.*** The unilateral longitudinal incision is high, lateral, and just below the level of the nail.

the finger if the felon has maximal tenderness on the radial or ulnar aspect of the finger (**Figure 133-3*B***).[1] Purulent and/or bloody pink fluid will exit from the incision. Consider obtaining aerobic and anaerobic cultures of the purulent material. Bluntly and gently dissect the loculations with a mosquito hemostat. Irrigate the wound with an 18 gauge angiocatheter on a 5 or 10 mL syringe containing sterile saline. Place a piece of ribbon gauze into the wound. Apply a dry bulky dressing.

ALTERNATIVE TECHNIQUES

Alternative incisions have been advocated but are not recommended because they have higher complication rates (Figure 133-4).[1,5] These incisions can result in neurovascular injury, painful scars, and altered fingertip sensation. The hockey stick incision can result in digital nerve injury and produce numbness to the fingertip (**Figure 133-4*A***).[8] The through-and-through or bilateral longitudinal incision can result in bilateral digital nerve injury and complete anesthesia of the fingertip (**Figure 133-4*B***).[9] The transverse palmar incision may transect the digital neurovascular bundles (**Figure 133-4*C***). The fishmouth or horseshoe incision is very extensive, can take a long time to heal, and produces a large scar and an unstable pulp (**Figure 133-4*D***).[8]

AFTERCARE

Splint the involved digit. Provide the patient a sling to keep the hand elevated. The incidence of community-acquired methicillin-resistant *S. aureus* (CA-MRSA) skin and soft tissue infections presenting to the Emergency Department are increasing.[1,4] An oral antibiotic effective against CA-MRSA is recommended.[1,4,14,15] Sulfamethoxazole-trimethoprim, doxycycline, and clindamycin are appropriate choices for a 7 to 10 day course depending on local resistance patterns. Nonsteroidal anti-inflammatory medications supplemented with narcotic analgesics will control any postprocedural pain. Instruct the patient to soak the digit in warm water several times a day to speed healing. Patients should immediately return to the Emergency Department if they experience fever, increased pain, difficulty using the finger, redness of the finger or hand or arm, or a discharge from the wound.

The patient should be reevaluated in 24 to 48 hours for removal of the gauze and inspection of the digit. Remove the gauze during the follow-up visit. Perform a digital or metacarpal block for patient comfort. Irrigate the wound with sterile saline and break up any further loculations, if needed. Replace the gauze for another 24 to 48 hours if there is continued drainage.

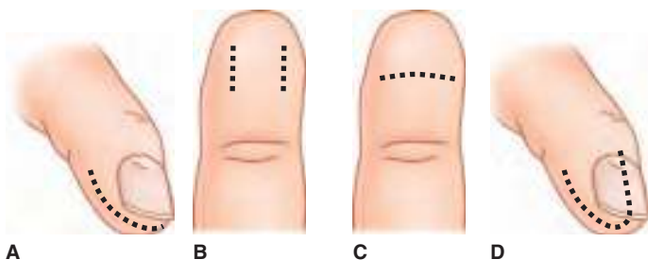

FIGURE 133-4. Incisions not recommended for the drainage of a felon. ***A.*** The hockey stick incision. ***B.*** The through-and-through or bilateral longitudinal incision. ***C.*** The transverse palmar incision. ***D.*** The fishmouth incision.

COMPLICATIONS

Untreated or mistreated felons may cause skin necrosis, osteitis or osteomyelitis of the distal phalanx, septic arthritis of the distal interphalangeal joint, extension of the infection into the palm and adjacent fingers, suppurative tenosynovitis, and lymphangitis.[16] Flexor tenosynovitis can occur if the incision is extended too far proximally and too deep. Improperly placed incisions can result in injury to the flexor digitorum profundus tendon or the distal interphalangeal joint, mobility of the pad of the finger, neurologic compromise, and/or vascular compromise. The lack of improvement within 24 to 48 hours requires consultation with a Hand Surgeon.

SUMMARY

Felons require incision and drainage. The procedure is quick, simple, and easy to perform. The incision should be made at the point of maximal fluctuance while avoiding injury to the digital arteries, digital nerves, the flexor digitorum profundus tendon, and the distal interphalangeal joint. Close follow-up is mandatory to prevent any complications. Consult a Hand Surgeon for any complications associated with the felon.

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134 Pilonidal Abscess or Cyst Incision and Drainage

Carolyn Chooljian

INTRODUCTION

Controversy surrounds pilonidal disease, from who first described it, to the etiology, and how to manage it surgically. Some believe that pilonidal disease was first described in 1880 by Hodges.[1] However, others say it was first described in 1833 by Mayo.[2] Hodges used the term "pilonidal sinus" to describe a chronic infection that contained hair and was usually found between the buttocks. The word "pilonidal" comes from "pilus" or hair and "nidus" or nest. It literally means "nest of hair." The condition did not receive much attention until it became a significant problem in the armed services around the time of World War II. In 1940, in the United States Navy, the number of sick days caused by pilonidal disease and its complications exceeded those of either syphilis or hernias.[3] This condition was coined as "jeep disease" by Buie in 1944 because of the high occurrence rate in those who drove or were frequent passengers in military vehicles.[4] There is an increased prevalence in drivers and others with occupations requiring long periods of sitting.[5-7]

Pilonidal sinus disease primarily affects Caucasian males. Blacks are infrequently affected and the condition is rare in Asians and Indians. Males are affected three to four times more frequently than females. The affected females tend to be younger than males.[8] The condition is prevalent from the onset of puberty to young adulthood and is rare after the age of 40. The peak age of incidence is 21 years. The increased incidence in adolescents and young adults is attributed to hormonal effects of increased hair on the torso, increased activity of sebaceous and sweat glands, fat deposition on the buttocks, and deepening of the gluteal cleft. Other risk factors may include hirsutism, obesity, and poor personal hygiene. Repeated trauma to the area may also contribute to the formation of pilonidal disease. There is an increased prevalence in drivers and others with occupations requiring long periods of sitting.[5,6]

Patients with pilonidal sinus disease may present with three different clinical pictures: asymptomatic disease, an acute abscess, or chronic disease. Asymptomatic disease patients have a painless sinus pit at the top of the natal cleft. Patients with chronic disease may have mild discomfort and a chronically draining sinus in the upper gluteal region. Approximately half of patients with symptomatic pilonidal disease will present acutely with severe pain and swelling that is indicative of a pilonidal abscess that necessitates incision and drainage (**Figure 134-1**).[9,10] Inspection will reveal one or more midline sinus tract openings, often with protruding tufts of hair. The area will be tender, erythematous, and indurated when an abscess is present. Fluctuance and swelling may not be readily appreciated. The sinuses may be quite extensive depending upon the chronicity of the disease process prior to presentation. Patients with chronic disease may have mild discomfort and a chronically draining sinus in the upper gluteal region.

FIGURE 134-1. *A pilonidal abscess. (Used with permission from Knoop KJ, et al:* The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2016. Photo contributor: Louis La Vopa, MD.)*

ANATOMY AND PHYSIOLOGY

A pilonidal sinus consists of a characteristic midline opening, or series of openings, in the upper aspect of the gluteal cleft approximately 4 to 5 cm from the anus (**Figure 134-2).** The skin enters the sinus giving the opening a smooth edge. This primary tract leads into a subcutaneous cavity that contains granulation tissue and often a nest of hairs (**Figure 134-3**). The hairs may be seen projecting through the skin opening. Many sinuses have lateral or secondary openings (i.e., fistulas) extending from the pilonidal abscess (**Figure 134-3**).

Loose hairs from the adjacent gluteal region are thought to form a bristly tuft and penetrate into the skin, perhaps in an area of skin irregularity. This process may be aided by pressure on the region in persons with occupations that require long hours of driving or sitting. The hairs may also be pulled in by a suction effect between the moving buttocks. The hair penetrates the skin and causes a foreign body reaction and secondary inflammation with the potential for infection and abscess formation. The sinuses spread cranially and laterally. They rarely approach the anus and generally remain superficial to the presacral fascia.[10-12]

There have been various opinions as to the etiology of the condition since the first description of the disease. In the first half of the twentieth century, it was generally attributed to a congenital lesion. Some authors believed that the pilonidal sinus originated from a

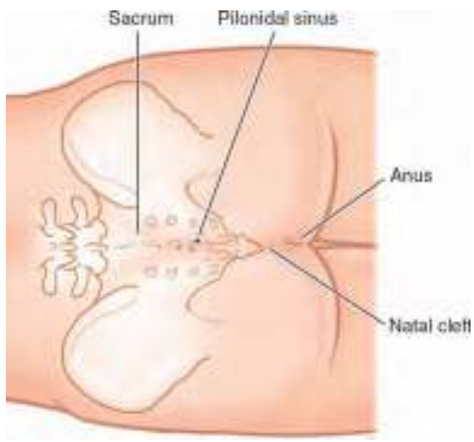

FIGURE 134-2. Pilonidal sinuses occur in the midline, approximately 4 to 5 cm above the anus and in the natal cleft.

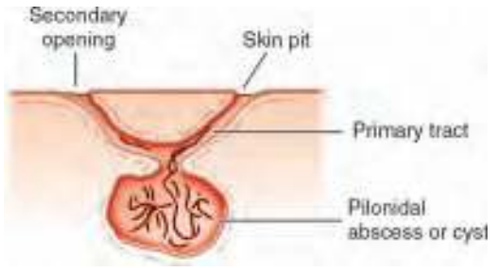

FIGURE 134-3. Cross-section through a pilonidal abscess and sinus. A primary tract and skin pit lead to the subcutaneous abscess. There may be secondary or lateral openings (fistulas).

remnant of the medullary canal that subsequently became infected. However, pilonidal disease can form in other areas of the body that lack hair, as some barbers have experienced in interdigital spaces.[11,13] Currently, it is widely accepted that pilonidal disease is acquired.[14] Some of the contributing factors to this belief are the rate of recurrence after excision, occurrence at other sites than the gluteal cleft, and the frequency seen among certain populations (e.g., barbers, armed forces).[11,13]

INDICATIONS

Incision and drainage is indicated whenever a patient presents with a pilonidal abscess.[15] Antibiotics alone are ineffective in treating a pilonidal abscess, although they are indicated when cellulitis is present.[16,17] Rarely, systemic signs and symptoms may ensue. There are reported cases of necrotizing fasciitis from neglected pilonidal sinus disease. Thus it is preferable to treat pilonidal abscesses expeditiously with an incision and drainage procedure.

CONTRAINDICATIONS

Patients who are asymptomatic do not require an incision and drainage and can be referred to a Surgeon for removal. Most pilonidal abscesses may be drained in the Emergency Department. Patients with fever, systemic signs and symptoms, and/or toxicity should be admitted to the hospital for parenteral antibiotics, incision and drainage, and observation. This is particularly true if the patient has diabetes or is immunocompromised. Consult a Surgeon to manage these patients. Extensive abscesses should be incised and drained in the Operating Room under general anesthesia. The procedure should be conducted under general anesthesia in the Operating Room if adequate anesthesia cannot be obtained and pain limits the procedure.

EQUIPMENT

- Gown, face mask, and gloves
- Benzoin solution
- Povidone iodine or chlorhexidine solution
- Skin razor
- 10 mL syringe
- 25 or 27 gauge needle, 2 inches long
- Local anesthetic solution with epinephrine, lidocaine or bupivacaine
- #11 scalpel blade on a handle
- #15 scalpel blade on a handle
- Curved hemostat
- 4×4 gauze squares
- Ribbon gauze, plain or iodoform
- Adhesive tape

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. The postprocedure care should be explained as well. Document the discussion of the risks and benefits of the procedure. Obtain a signed informed consent for the procedure.

The best visualization of the sacral region, particularly in obese patients, occurs with the use of a proctoscopic examination table, if available (**Figure 134-4*A***). Place the patient prone on a gurney or on the proctoscopy table. Alternatively, place the patient in the lateral knee-chest position to expose the affected area (**Figure 134-4*B***). Apply benzoin solution to the buttocks and allow it to dry. Apply adhesive tape to the buttocks and tape them open (**Figure 134-5**). Clean any dirt and debris from the skin overlying the abscess or

A B

FIGURE 134-4. Patient placement. ***A.*** Prone on a proctoscopy table. The patient may also be placed prone on a gurney. ***B.*** The lateral knee-chest position.

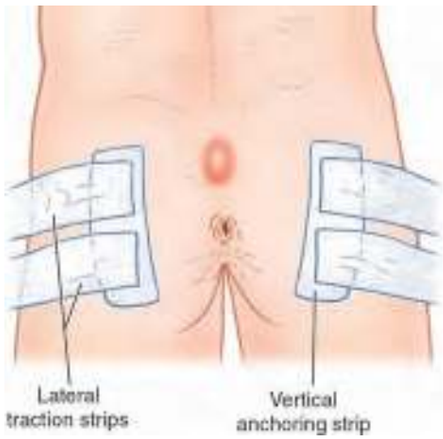

FIGURE 134-5. Exposure of the abscess.

cyst. Apply povidone iodine or chlorhexidine solution and allow it to dry. Shave the surrounding area, if the patient is hirsute, to aid in the application of the dressing after the procedure. Some authors advocate shaving the sacral region to prevent recurrence as well, although this has not been proven to be effective. A gown, face mask, and nonsterile gloves are recommended to be worn for this procedure.

ANESTHESIA

Local anesthesia should be administered, recognizing that it is often difficult to obtain complete anesthesia by direct infiltration of an abscess.[18] Local anesthetics are weak acids and are less effective in the acidic environment of an abscess. The skin over the abscess cavity usually becomes insensate, but anesthesia of the abscess cavity itself is not possible. The pain caused by injection of the local anesthetic solution is related to the rate that it is injected and the force necessary to inject it.

Inject the local anesthetic solution slowly through a small-bore needle (25 or 27 gauge) as the needle is withdrawn through the dermis. The needle bore will create a passage through the subcutaneous tissue as it is inserted that enables the local anesthetic solution to be infiltrated slowly and with less discomfort. Hold the syringe horizontal in reference to the skin surface. Inject 3 to 4 mL of local anesthetic solution intradermally over the dome of the abscess (**Figure 134-6**). The skin will blanch if the injection is given properly. **Do not inject the local anesthetic solution into the abscess cavity.** The increased pressure within the cavity will cause more discomfort to the patient and may cause the local anesthetic

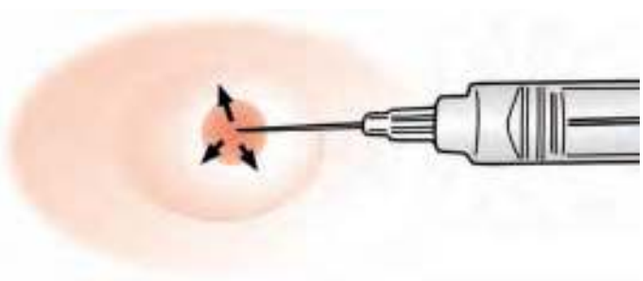

FIGURE 134-6. Subcutaneous infiltration of local anesthetic solution. The needle and syringe are held parallel to the skin. The needle is inserted into the subcutaneous tissue overlying the pilonidal abscess. Infiltrate the local anesthetic solution as the needle is withdrawn. The skin should blanch (shaded area) if injected properly.

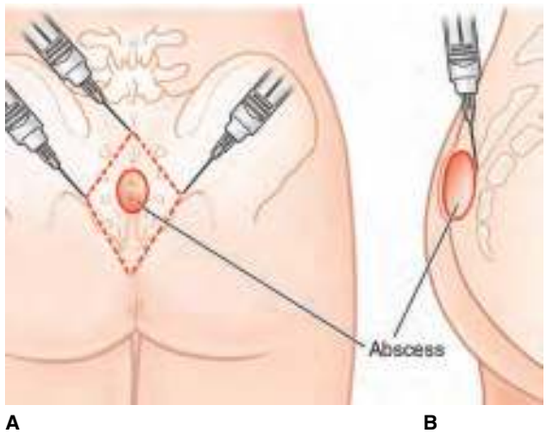

FIGURE 134-7. Field block anesthesia for a pilonidal abscess. ***A.*** Local anesthetic solution is infiltrated subcutaneously on all four sides of the abscess. ***B.*** The local anesthetic solution is infiltrated deep to the abscess cavity in a fan-like pattern.

solution or the abscess contents to be forcefully expelled if there is an opening in the skin.

Additional anesthesia is accomplished by performing a field block (**Figure 134-7**).[19] Inject local anesthetic solution subcutaneously around the periphery of the abscess (**Figure 134-7*A***). Inject local anesthetic solution deep to the abscess in a fan-like pattern (**Figure 134-7*B***).

Systemic analgesia (i.e., procedural sedation and analgesia, Chapter 159) may be required since it is quite difficult to obtain adequate anesthesia of an abscess locally.[18] Patient-administered nitrous oxide (Chapter 158), with or without supplemental narcotic analgesics, is an alternative to procedural sedation. Obtain an additional signed informed consent for the procedural sedation or the nitrous oxide administration. **The procedure should be conducted under general anesthesia in the Operating Room if adequate anesthesia cannot be obtained in the Emergency Department and pain limits the procedure.**

A novel technique was used to anesthetize the pilonidal abscess.[18] It does not require multiple infiltrations of the abscess or surrounding tissues. It involves the needle aspiration of the abscess followed by injection of local anesthetic slowly, in a volume to replace the purulent material aspirated, into the abscess cavity to anesthetize the area before incision and drainage. More information is needed before this technique of anesthesia can be recommended.

TECHNIQUE

Incise the skin over the area of maximum fluctuance with a scalpel blade. A 10% recurrence rate after drainage of chronic abscesses through a vertical incision lateral to the midline has been reported.[20] This may be due to better healing of wounds that are off the midline. Thus, some authors and Colorectal Surgeons recommend that the incision for an acute abscess be off the midline if the abscess can be drained adequately through the incision (**Figure 134-8*A***). Extend the incision the length of the abscess to allow for proper drainage. A full-thickness, thin ellipse of skin can be removed to prevent premature closure of the skin edges. Approximately 40% of pilonidal abscesses will be cured from simple incision and drainage alone.[21] It is not necessary to perform more radical excision procedures in the Emergency Department.

It is important that loculations be lysed and the area thoroughly drained to minimize recurrence. Several methods can be used to lyse adhesions within the cavity. A gloved finger may be used to bluntly break up the adhesions. Hemostats can be inserted and

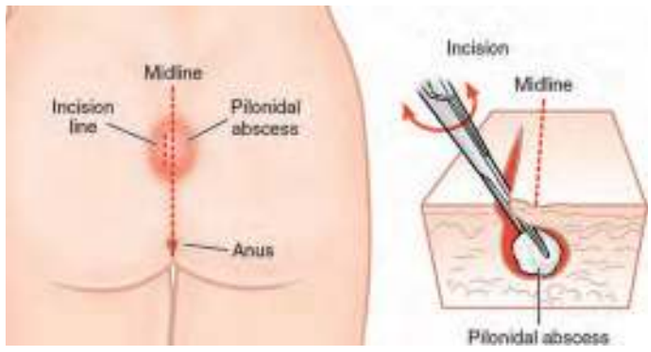

FIGURE 134-8. Incision and drainage of a pilonidal abscess. ***A.*** An incision is made lateral to the midline and overlying the abscess cavity. ***B.*** A hemostat with gauze clamped in the jaws is inserted into the abscess cavity and rotated to break loculations and remove debris.

spread within the abscess cavity. A useful technique employs a 4×4 gauze square clamped in a hemostat and swirled inside the abscess cavity to break adhesions and remove debris (**Figure 134-8*B***). This technique aids in removing hair and the infected lining of the cyst. Irrigation of the abscess cavity with normal saline is left to the Emergency Physician's discretion but may not be necessary.[22]

Loosely pack the cavity with ribbon gauze. Packing the cavity too tightly may cause ischemia to the surrounding tissue, delays healing, and is uncomfortable for the patient. The purpose of the packing is to keep the skin edges from adhering before the cavity closes. Cover the incision with a simple bandage composed of gauze and adhesive tape. A thick layer of absorbent gauze will soak up any continued drainage.

ALTERNATIVE TECHNIQUE

A loop (Chapter 131) can be used to drain the pilonidal abscess.[23] A similar technique has been used in China for more than 40 years.[23] They refer to this technique as suture-dragging.

AFTERCARE

Antibiotics are generally unnecessary to treat a simple abscess when there is no cellulitis surrounding the wound.[5,6,16,17,24] No data could be found on the optimal duration of antibiotic treatment if the overlying skin is cellulitic. The conventional 7 to 10 day course of antibiotics is probably adequate. Patients with diabetes, patients with cardiac valve disease, those who have hardware in their body, or those who are immunocompromised and have a pilonidal abscess are at risk for infectious complications and it is advised that they be treated with oral antibiotics.[15,16]

There is a disparity in the literature regarding the bacteriology of pilonidal abscesses. *Staphylococcus aureus* is the most commonly found bacteria, and surprisingly, *Escherichia coli* is rarely found.[25] A report in children recovered primarily anaerobes from pilonidal cysts.[26] *E. coli* was the most common aerobe cultured from this series. **In light of these conflicting results and in the event that antibiotics are deemed necessary, coverage for skin flora as well as aerobes and gram-negative organisms would be advised.** A combination of a first-generation cephalosporin or penicillinase-resistant penicillin along with metronidazole or clindamycin is recommended. **Keep in mind the ever increasing rates of community-acquired methicillin-resistant *S. aureus* (CA-MRSA) infections.** Consider prescribing clindamycin, doxycycline, or trimethoprim-sulfamethoxazole if CA-MRSA is a potential pathogen.[8]

Instruct the patient to change the gauze dressing as often as necessary to keep the outside of the dressing dry. The patient should return for follow-up in 48 hours for a wound check and removal of the packing. If the wound is large or there is not a clear open tract for continued drainage, reinsert new packing upon follow-up. Incisions that remain open do not require repacking. Advise the patient to have the packing changed every 24 to 48 hours depending upon the amount of drainage. Decrease the amount of packing each time to allow the wound to heal from the base outward. The patient should thoroughly wash the wound with soap and water in the shower or take a sitz bath each time the packing is removed. It is helpful to let the stream of shower water run inside the wound to aid in wound irrigation. After showering, the patient should dry the area thoroughly. Discontinue the packing once the wound is well granulated and there is no concern that the skin edges will adhere to each other. The patient must continue to clean the wound thoroughly every day until it is fully healed. Healing may take several weeks depending upon the size of the abscess cavity. Instruct the patient that they must return to the Emergency Department if they develop a fever, increased pain, or increased redness of the skin surrounding the abscess. Patients often have pain in the first 2 to 3 days after the incision and drainage. This can be controlled with nonsteroidal anti-inflammatory drugs and supplemented with occasional narcotic analgesics.

Inform the patient that incision and drainage in the acute care setting is not definitive treatment and that the condition may recur. Arrange for follow-up with a Surgeon who can provide wound care as well as definitive therapy if surgery is required. Definitive treatment of a chronic pilonidal abscess or sinus still remains controversial among Colorectal Surgeons. Options include de-roofing and curettage, excision with primary closure, wide excision with secondary closure, or marsupialization.[2,22,27-33]

Educate the patient about their role in the prevention of a recurrence. Recurrence may be prevented with meticulous hygiene and periodic shaving or hair removal to the area.[11,19,34,35] The application of phenol on follow-up has shown promise to prevent recurrence.[36-38] Instruct the patient on the methods for meticulous hygiene in the area, even after the wound has healed. Repeated trauma to the area should be avoided. This includes exercises such as sit-ups and leg lifts and prolonged periods of sitting.

COMPLICATIONS

Pilonidal disease may return, even with radical and extensive surgical excision procedures. **Thus, recurrence is to be expected, and the patient should be alerted to this possibility.** Rarely, pilonidal lesions progress to necrotizing fasciitis. Proceed cautiously with patients who are diabetic or otherwise immunocompromised as they are at risk for widespread infections. Those with systemic signs and symptoms are best admitted for treatment. Necrotizing fasciitis is a surgical emergency and requires extensive operative debridement, systemic antibiotics, and intensive supportive care.

Alternative diagnoses should be considered when evaluating patients with pilonidal disease including hidradenitis suppurativa, furuncles, Crohn's disease, perianal fistula, and infections such as tuberculosis, syphilis, and actinomycosis.[15] Rarely, a nonhealing pilonidal infection may be a pilonidal sinus malignancy.[39] Squamous cell carcinoma has been described to arise from chronic sinus tracts. This emphasizes the importance of follow-up for all patients with pilonidal sinus disease.

Other complications include infection and tissue injury. The incision and drainage procedure can result in a subsequent cellulitis, endocarditis, fasciitis, meningitis, myositis, sacrococcygeal osteomyelitis, or septicemia. The sharp and blunt dissection can injure underlying or adjacent structures including blood vessels, the coccyx, muscles, nerves, and tendons.

SUMMARY

Pilonidal disease is common in the young adult population and more prevalent in males. It is now widely accepted as being an acquired condition caused by hair that penetrates an irregular area of skin in the sacral area. Pits occur in the skin that in turn lead to cysts in the subcutaneous tissue. Patients may present with asymptomatic pits, chronically draining sinuses, or acute painful abscesses. No treatment is necessary for asymptomatic patients. Nontender sinuses may be referred for surgical treatment. Abscesses should be drained expeditiously under adequate analgesia and anesthesia. Antibiotics are not indicated unless the patient has surrounding cellulitis or is immunocompromised. Recurrences are common and patients must be referred for follow-up with a Surgeon who can provide wound care as well as surgical treatment for chronic cases.

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135 Perianal Abscess Incision and Drainage

John Ramos and Deena Bengiamin

INTRODUCTION

Anorectal infections are common problems presenting to the Emergency Department. Understanding anorectal anatomy is essential to make a diagnosis, institute proper treatment, and anticipate complications. Failure to diagnose and treat an extensive abscess may be life threatening. Surgical consultation is imperative if the extent of an abscess is unclear.

Anorectal infections occur mostly in the third or fourth decade of life. Perianal abscesses (**Figure 135-1**) are two to three times more common in men than women.[1,2] Male predominance is even more pronounced in the pediatric population.[3] In one series, all patients under 2 years of age and 60% greater than 2 years were males.[3] The increased incidence of perianal infection in males may be related to androgen conversion in the anal glands.[4] Deep anal crypts are associated with perianal abscesses in infants.[5]

Abscesses may completely resolve after proper incision and drainage. Approximately 50% recur or develop a chronic epithelialized tract or fistula-in-ano. Abscesses and fistulas are different sequelae of the same process.[6]

ANATOMY AND PATHOPHYSIOLOGY

Knowledge of the anatomy of the region is important to understand the pathophysiology of anorectal infections (**Figures 135-2 and 135-3**). Columnar epithelium transitions to squamous epithelium at the columns of Morgagni at the level of the dentate line. Semilunar folds of epithelium called anal valves connect the inferior borders of the anal columns. At the base of each anal valve is an anal crypt into which the ducts of the anal glands drain. The anal glands are

FIGURE 135-1. A perianal abscess. (Used with permission from Hornez E, et al: Surgical proctologic emergency in isolated sea-based environment: how is it performed in the French navy. *Mil Med* 2013; 178(4):e498-e502.)

positioned circumferentially in the anal canal and secrete mucus to aid in the evacuation of feces. The anal glands are in the space between the internal and external anal sphincter muscles. Most anorectal infections begin in this intersphincteric space due to blockage and resultant infection of the anal glands.[7]

The spread of an infection is determined by the anatomy of the anorectal region. There are five anatomic spaces into which an infection can spread (**Figure 135-4**).[6] The perianal space is located at the area of the anal verge. The ischiorectal space is continuous with the perianal space and extends from the levator ani muscle to the perineum. The submucosal space can be visualized with the use of anoscope. The intersphincteric space lies between the internal and external anal sphincter muscles. It connects inferiorly with the perianal space and superiorly with the rectal wall. The supralevator (i.e., pelvirectal) space is located superior to the levator ani muscle. It is bounded superiorly by the peritoneum, laterally by the pelvic wall, and medially by the rectum. The deep postanal space is located between the tip of the coccyx and the anus. It courses through the superficial external anal sphincter and the levator ani. The superficial postanal space lies posterior to the anal verge and is subcutaneous. The retrorectal space is high in the pelvis. It occupies the area between the distal rectum and the sacrum.

Most anorectal infections begin in the intersphincteric space. Natural barriers are broken down by the formation of an abscess and the infection can spread to contiguous spaces. Abscesses are classified by their location.[8] Perianal abscesses are common and make up 60% of abscesses. Ischiorectal abscesses occur less frequently at 22%, and supralevator abscesses are the least common type at 9%. Bilateral involvement may occur when an infection spreads circumferentially via the deep postanal space and results in a horseshoe abscess.

Patients with anorectal abscesses present with buttock pain and swelling. There is purulent drainage at the abscess site if it spontaneously drains. Symptoms depend upon the location of the abscess. Patients with perianal infections have anal pain that increases with defecation or sitting. Pain associated with deeper infections may be atypical. Patients with supralevator abscesses may have deep rectal pain, gluteal pain, dysuria, or other urinary symptoms.

Erythema, swelling, and fluctuance are often present on examination at the abscess site. The location of the swelling and fluctuance of a perianal abscess is at the anal verge. Ischiorectal space infections track farther from the anus onto the buttock. Supralevator and intersphincteric abscesses may have minimal or no external signs.[9] **The diagnosis of perianal cellulitis is highly suspect. These patients have either an anorectal abscess or Fournier's gangrene until proven otherwise. Patients with gluteal pain and a small amount of erythema in the perianal area have a deep-seated abscess until proven otherwise.**

A digital rectal examination is necessary as intersphincteric, deep postanal, and submucosal abscesses may be palpated but often cannot be appreciated on external examination. **A superficial digital**

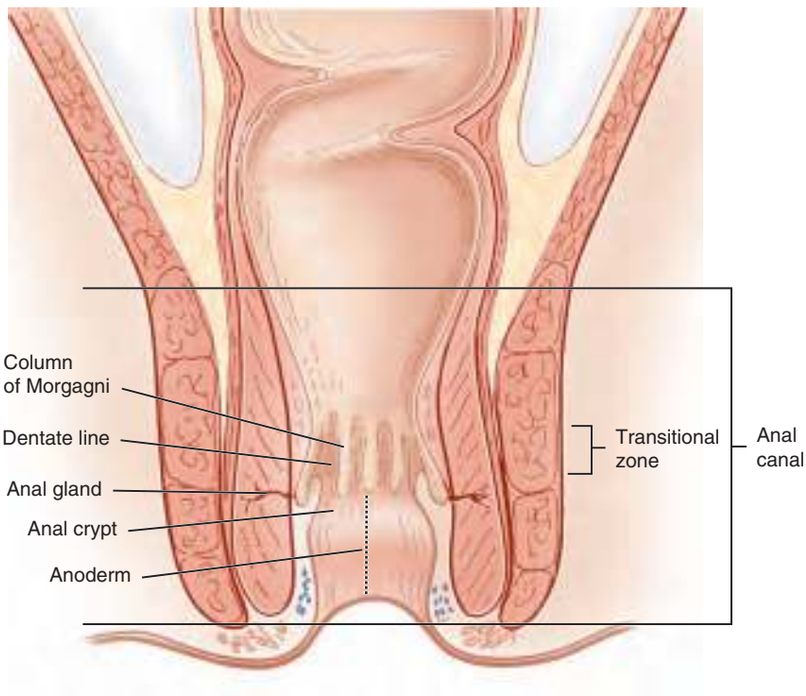

FIGURE 135-2. The anatomy of the anal canal.

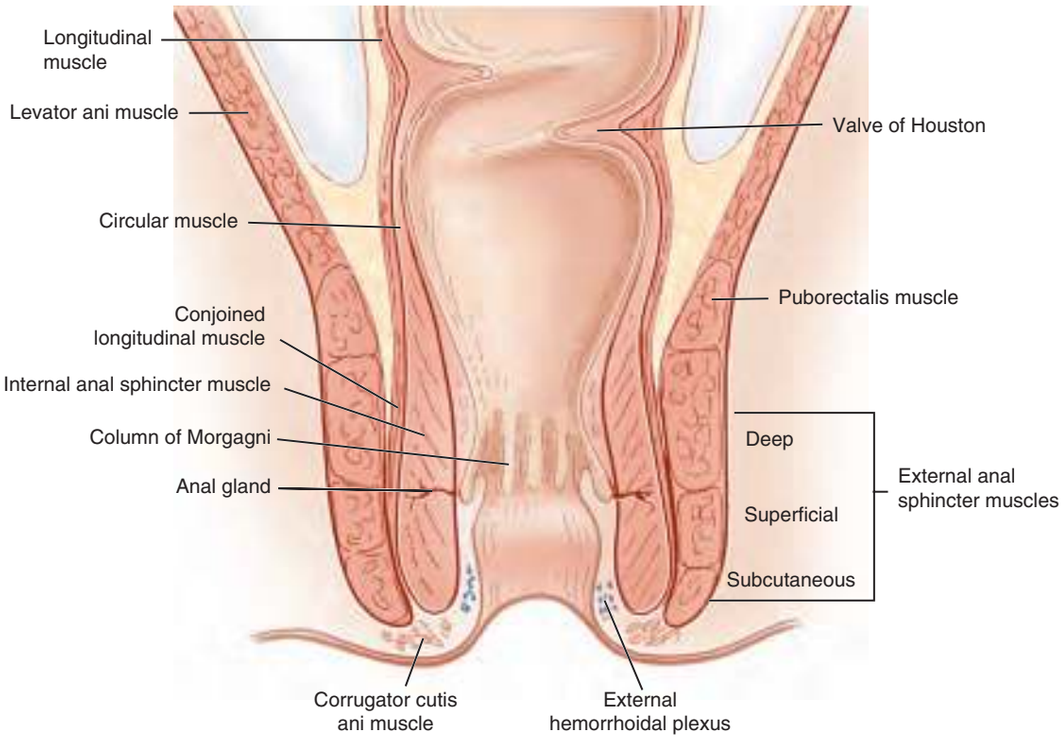

FIGURE 135-3. The major structures of the anal canal.

examination of the anal canal alone is inadequate for detection of some abscesses. The gloved finger must extend into the rectum seeking tenderness and a mass. The digital rectal examination may fail to detect some deep abscesses due to patient discomfort.

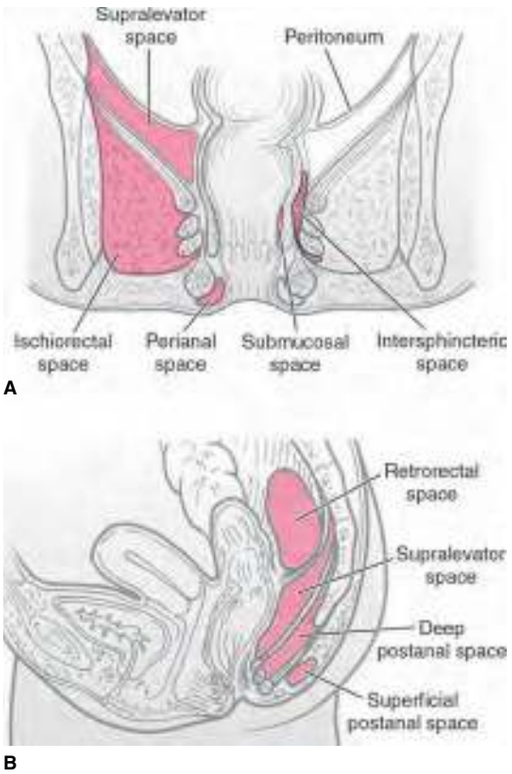

FIGURE 135-4. The anorectal spaces. ***A.*** Coronal section through the pelvis. ***B.*** Sagittal section through the pelvis.

Obtain a computed tomography (CT) scan of the pelvis with rectal and intravenous contrast if unable to obtain an adequate examination due to pain or if a deep abscess is suspected.[10]

A fistula-in-ano represents the chronic phase of an unhealed perianal abscess. Fistulas may form due to persistent obstruction of the anal gland, inadequate drainage of a perirectal abscess, or rupture of a perianal abscess.[11] The tract eventually becomes epithelialized with glandular tissue. Fistulas may also form due to epithelialization by cells derived from the transitional zone of the anal canal and thus may be unrelated to persistent anal gland disease.[12] Patients with a fistula-in-ano will often give a history of a previous abscess. Developing a fistula-in-ano does not appear to be related to whether the abscess drained spontaneously or surgically.[13] Patients may complain of chronic drainage from the site or subacute pain.

Physical examination of a fistula-in-ano reveals an external opening with scant drainage and visible surrounding granulation tissue. Digital rectal examination may reveal an indurated cord-like structure beneath the skin within the anal canal. The internal opening may be palpable along the dentate line. Pus may be expressed externally or from within the anus upon palpation of the fistulous tract.

The greater the distance from the anus that the external opening is located, the more complex is the fistulous tract. Goodsall's rule describes the likelihood of the location of fistulous tracts and the internal opening based upon the location of the external opening (**Figure 135-5**).[14] Anterior external openings tend to communicate in a linear fashion with the internal opening in the anal canal. Fistulas with posterior external openings tend to communicate in a curvilinear fashion with the internal opening.

Not all perianal bumps are an abscess. An abscess must be distinguished from other diseases that affect the area. This includes actinomycosis, Crohn's disease, Fournier's gangrene, a furuncle, herpes simplex, hidradenitis suppurativa, human immunodeficiency virus (HIV)–related complications and diseases, malignancies, radiation fibrosis, sexually transmitted diseases, syphilis, and tuberculosis.[15-18] Obtain a CT scan and consult a surgeon if unclear of the diagnosis.

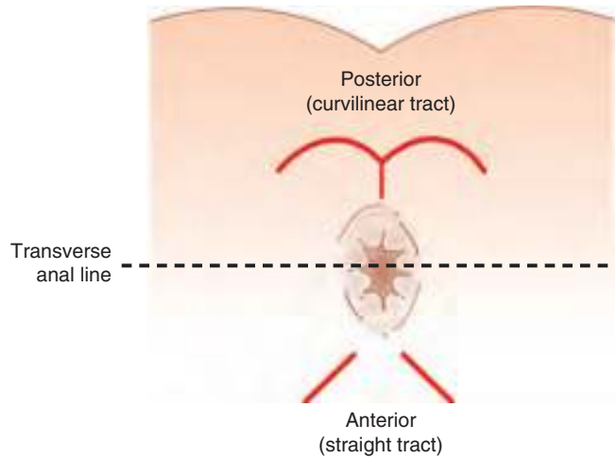

FIGURE 135-5. Goodsall's rule.

Patients with multiple or recurrent fistulas require evaluation for Crohn's disease. This is particularly true if associated with chronic diarrhea or cramping, both of which suggest inflammatory bowel disease. Recurrent fistulas may be indicative of tuberculosis or a sexually transmitted disease (e.g., lymphogranuloma venereum). **It is imperative that these patients be referred to a Surgeon who is experienced in managing anorectal disease.**

INDICATIONS

Incision and drainage is the treatment for an anorectal abscess.[15,18] Uncomplicated perianal abscesses and submucosal abscesses may be drained in the Emergency Department. Management of deeper or more extensive abscesses should be in consultation with a Surgeon. **Use caution in draining abscesses.** The ischiorectal space is quite large, particularly in the obese patient, and adequacy of drainage is not assured except under general anesthesia. It is not uncommon for an abscess to have a small erythematous and swollen area on the buttock overlying an extensive and deep abscess. Attempts at drainage in the Emergency Department may be inadequate.

Perianal disease is commonly encountered in the HIV-infected patient. Complication rates were noted to be high in the past and a hands-off approach was espoused. More recent data suggest that patients with a relatively preserved immune system may safely undergo standard surgical drainage. Small perianal abscesses may be drained in the Emergency Department. Consult a General or Colorectal Surgeon for patients with late-stage HIV disease. Infections are more likely to be extensive and anorectal sepsis is more common because these patients have poor wound healing.[19]

CONTRAINDICATIONS

Most small, uncomplicated perianal, and submucosal abscesses can be drained in the Emergency Department. **A General or Colorectal Surgeon should drain all other types of anorectal abscesses. Caution is urged when an abscess is on the buttock because it may manifest an ischiorectal abscess.** Patients with bleeding disorders, taking anticoagulants, or with thrombocytopenia should be managed by a Surgeon. Consult a Surgeon if the infection is extensive or if the extent of the abscess cannot be determined. Surgical consultation is also necessary for patients with purulent drainage from inside the anus. Internal findings may indicate that the patient has an intersphincteric or a supralevator abscess, the full extent of which can be determined only under general anesthesia. The procedure should be conducted by a Surgeon under general anesthesia in the Operating Room if adequate anesthesia cannot be obtained or pain limits the procedure in the Emergency Department.

Patients with fever or toxicity should be admitted to the hospital for parenteral antibiotics, incision and drainage in the Operating Room, and observation. **Anorectal infections occasionally progress to necrotizing fasciitis which is a true surgical emergency.** Physical examination findings in such cases may initially be minimal except for systemic signs or symptoms. Patients who are immunocompromised or with late-stage HIV infection should be referred to an experienced Surgeon due to the higher complication rate and increased risk of extensive infection.

Chronically draining fistulas without an acute infection should be referred to a General or Colorectal Surgeon for care. Patients with purulent drainage from the anus, even if there is no significant tenderness, should be examined under general anesthesia as they may still have an internal abscess along with a fistulous tract.

EQUIPMENT

- Gown, face mask, and gloves
- Povidone iodine or chlorhexidine solution
- 10 mL syringe
- 25 or 27 gauge needle, 2 inches long
- Local anesthetic solution with epinephrine
- #11 scalpel blade on a handle
- #15 scalpel blade on a handle
- Curved hemostat
- 4×4 gauze squares
- 10 to 16 French mushroom (de Pezzer) catheter (optional)
- Adhesive tape
- Feminine napkin (optional)
- Normal saline
- 18 gauge angiocatheter
- 20 mL syringe
- 2–0 nylon suture
- Needle driver
- Scissors

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure along with alternatives to the patient and/or their representative. The postprocedure care should be explained as well. Document the discussion of the risks and benefits of the procedure. Obtain an informed consent for the procedure. Wear a face mask, gown, and gloves for the entire procedure. The injection of local anesthetic solution can force abscess contents to shoot out. Incision of a tense abscess can also result in contamination.

The best visualization of the sacral region, particularly in obese patients, occurs with the use of a proctoscopic examination table (**Figure 135-6*A***). Place the patient prone on a gurney or on the proctoscopy table. Alternatively, place the patient in the prone or lateral knee-chest position on a gurney or examination table to expose the affected area (**Figure 135-6*B***). Apply benzoin solution to the buttocks and allow it to dry. Apply adhesive tape to the buttocks and tape them open (**Figure 135-7**). Clean any dirt, stool, and debris from the skin overlying the abscess or cyst. Apply povidone iodine or chlorhexidine solution and allow it to dry. Shave the surrounding area, if the patient is hirsute, to aid in the application of the dressing after the procedure.

FIGURE 135-6. Patient placement. ***A.*** Prone on a proctoscopy table. The patient may also be placed prone on a gurney. ***B.*** The lateral knee-chest position.

ANESTHESIA

Local anesthesia must be administered, recognizing that it is often difficult to obtain complete anesthesia by direct infiltration of an abscess. Local anesthetics are weak acids and are less effective in the acidic environment of an abscess. The skin over the abscess cavity usually becomes insensate, but anesthesia of the abscess cavity itself is not possible. The pain caused by injection of the local anesthetic solution is related to the rate that the anesthetic is injected and the force necessary to inject it. **Inject the local anesthetic solution slowly through a small-bore needle (e.g., 25 or 27 gauge) as it is withdrawn through the dermis**. The needle bore will create a passage through the subcutaneous tissue as it is inserted that enables the local anesthetic solution to be infiltrated slowly and with less discomfort.

Hold the syringe horizontal to the skin surface. Inject 3 to 4 mL of local anesthetic solution intradermally over the dome of the abscess (**Figure 135-8**). The skin will blanch if the injection is given properly. **Do not inject the local anesthetic solution into the abscess cavity**. The increased pressure within the cavity will cause more discomfort to the patient and may cause the solution to be forcefully expelled if there is an opening in the skin.

Additional anesthesia is accomplished by performing a field block (**Figure 135-9**). Inject local anesthetic solution subcutaneously around the periphery of the abscess (**Figure 135-9*A***).[20] Inject local anesthetic solution deep to the abscess in a fan-like pattern (**Figure 135-9*B***).

Systemic analgesia in the form of procedural sedation (Chapter 159) is usually required since it is difficult to obtain adequate anesthesia of an abscess locally. Self-administered nitrous oxide (Chapter 158) with or without opioid supplementation is an alternative. Obtain an additional informed consent for the procedural sedation or nitrous oxide administration. **The procedure should be conducted by a Surgeon under general anesthesia in the Operating Room if adequate anesthesia cannot be obtained or pain limits the procedure**.

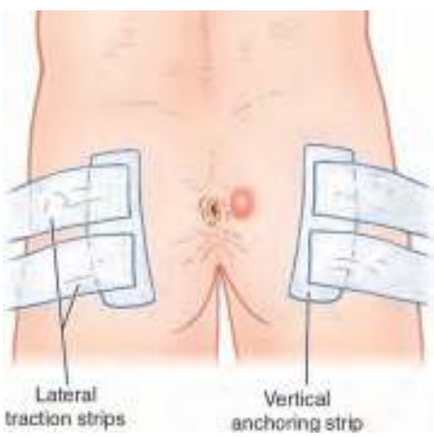

FIGURE 135-7. Exposing the abscess.

TECHNIQUES

INCISION AND DRAINAGE OF PERIANAL ABSCESSES

Make a stab incision with a #11 scalpel blade in the skin overlying the area of fluctuance to decompress the abscess (**Figure 135-10**). **Make the incision as close to the anus as possible so that if a fistula forms, its size will be limited**. This maneuver will minimize the length of a fistulotomy in the future should it become necessary. Extend the incision with the #11 scalpel blade or a #15 scalpel blade in a full-thickness elliptical pattern along the length of the abscess cavity or the area of fluctuance (**Figure 135-11*A***). Remove the ellipse of skin (**Figure 135-11*B***). A full-thickness ellipse of skin is excised to prevent premature closure of the skin edges. Some prefer

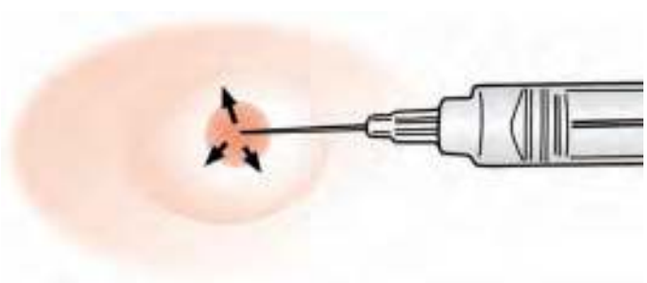

FIGURE 135-8. Subcutaneous infiltration of local anesthetic solution. The needle and syringe are held parallel to the skin. The needle is inserted into the subcutaneous tissue overlying the abscess. Infiltrate the local anesthetic solution as the needle is withdrawn. The skin should blanch (shaded area) if injected properly.

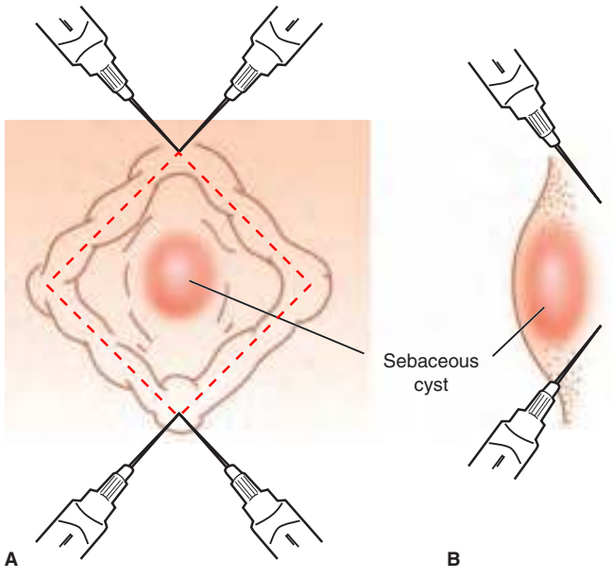

FIGURE 135-9. Field block anesthesia. ***A.*** Local anesthetic solution is infiltrated subcutaneously on all four sides of the abscess. ***B.*** The local anesthetic solution is infiltrated deep to the abscess cavity in a fan-like pattern.

to make a cruciate incision over the abscess and excise the edges (**Figure 135-12**).[6] These techniques delay cutaneous healing while the abscess is decompressing and allow it to drain freely without the need for packing.

FIGURE 135-10. The initial stab incision into the perirectal abscess. (Used with permission from Hornez E, et al: Surgical proctologic emergency in isolated sea-based environment: how is it performed in the French navy. *Mil Med* 2013; 178(4):e498-e502.)

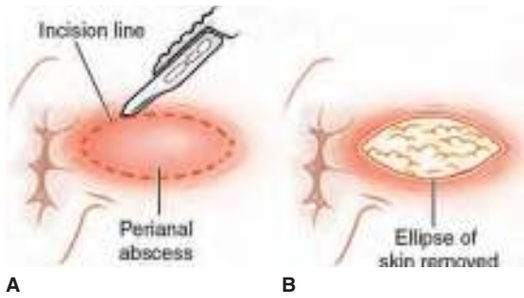

FIGURE 135-11. Drainage of a perianal abscess. ***A.*** An elliptical incision is made in the skin. ***B.*** The ellipse of skin is removed to prevent premature closure of the skin edges.

A linear incision is acceptable but is not recommended for a perianal abscess. The body location and excessive skin often result in premature closure before the cavity has healed. A linear incision requires repeated packing at 24 to 48 hour intervals to prevent premature closure. The area is difficult to access by the patient to pack the cavity. The pain of repeated packing often results in noncompliance. It is also difficult for the patient to wash out the cavity in the shower or bathtub.

It is important that loculations be lysed and the area thoroughly drained to minimize recurrence. Several methods can be used to lyse adhesions within the abscess cavity. A gloved finger may be used to bluntly break up the adhesions. Hemostats can be inserted and spread within the cavity. A useful technique employs a gauze 4×4 square clamped in a hemostat and swirled inside the abscess cavity to break adhesions and remove debris. Irrigate the cavity with normal saline.

It is not necessary to pack an incised and drained perianal abscess that has been incised by removing a full-thickness skin ellipse or a cruciate incision. Consider temporarily packing the abscess cavity in the Emergency Department to control any hemorrhage. Remove the packing before discharging the patient and to reassess the need for further hemorrhage control. Cover the wound with a thick layer of absorbent gauze to soak up continued drainage. A feminine napkin may also be used to absorb drainage and obviates the need for taping the dressing in place.

CATHETER DRAINAGE OF PERIANAL ABSCESSES

Another method used to drain perianal abscesses, and preferred by some Colorectal Surgeons, is catheter drainage.[15] The mushroom or de Pezzer catheter has a tunnel through a solid mushroom-shaped tip (**Figure 135-13*A***).[21] Make a stab incision with a #11 scalpel blade over the anal side of the area of fluctuance (**Figures 135-10 and 135-13*B***). Lyse any adhesions and express the pus. Flush the abscess cavity with normal saline using an 18 gauge angiocatheter on a 20 mL syringe.

Insert a 10 to 16 French latex mushroom catheter using a hemostat to stretch the tip so that it will fit through the incision (**Figure 135-13*C***). Place the tip of the hemostat through the hole in the mushroom catheter. Holding the hemostat with one hand,

FIGURE 135-12. Drainage of perianal abscess employing a cruciate incision. ***A.*** The cruciate incision is made over the abscess. ***B.*** Excision of the skin flap edges. ***C.*** The final appearance.

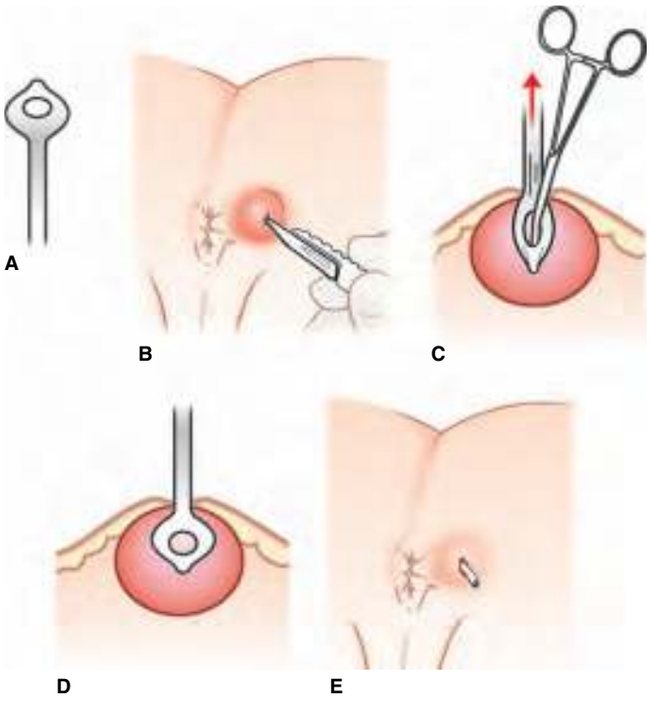

FIGURE 135-13. Catheter drainage of an abscess. ***A.*** The mushroom (de Pezzer) catheter. ***B.*** A stab incision is made over the area of maximal fluctuance. ***C.*** Place the tip of the hemostat through the side hole to stretch the tip of the catheter. Insert the stretched catheter through the stab incision. ***D.*** Remove the hemostat to expand the head of the catheter so that it remains within the abscess cavity. ***E.*** The catheter is cut so that it protrudes 2 to 3 cm from the skin incision.

use the other hand to pull on the tubing to stretch the mushroom tip and enable it to fit into the abscess cavity (**Figure 135-13*C***). Insert the stretched mushroom tip into the abscess cavity (**Figure 135-13*C***). Release the traction on the hemostat once the catheter tip is within the abscess cavity and the mushroom shape will be restored (**Figure 135-13*D***). Remove the hemostat from the abscess cavity.

Suture the catheter in place. Place a single simple interrupted stitch using 2–0 nylon adjacent to the stab incision. Leave both ends long, tie the suture knots, and do not cut the suture. Pass the needle through the catheter as it exits the skin incision. Tie the needle end of the suture to the tail end of the suture to secure the catheter. Cut off the excess suture. Cut the catheter so that it protrudes only 2 to 3 cm from the incision (**Figure 135-13*E***). Apply a dressing of gauze squares or a feminine napkin.

Many Colorectal Surgeons prefer the catheter method. Subsequent visits can assess the wound for the presence of a fistula without removing the catheter. Hydrogen peroxide can be infused through the catheter. Bubbles seen escaping from an opening within the anal canal are diagnostic for a fistula. Hydrogen peroxide is also used to produce an ultrasound interface that facilitates the definition of a fistulous tract and the internal opening.[6] The smaller stab incision takes less time to heal than the larger incision and drainage wound.

SUBMUCOSAL ABSCESSES

Most submucosal abscesses may be drained in the Emergency Department. The procedure requires the use of an anoscope (Chapter 88) to visualize the abscess. Make a superficial stab incision in the abscess with a #11 scalpel blade. Gently insert a hemostat and lyse any adhesions. Remove a small ellipse of the mucosa to allow the abscess to drain. Arrange follow-up with a Colorectal Surgeon within 24 hours.

AFTERCARE

The questions of bacterial cultures and antibiotic administration always come up in relation to these abscesses. Bacterial cultures are not helpful.[22] Antibiotics are generally unnecessary to treat a simple abscess when there is no cellulitis surrounding the wound.[15,23] No data could be found on the optimal duration of antibiotic treatment if the overlying skin is cellulitic. Antibiotic treatment should not delay surgery. The conventional 7 to 10 day course of antibiotics is likely adequate. There are minimal data in the literature regarding the treatment of patients with diabetes, patients with cardiac valve disease, those who have hardware in their body, or those who are immunocompromised with antibiotics for a perianal abscess.[24] These patients are at risk for infectious complications. It is advised that they be treated with antibiotics.[25] Bacteriology of anorectal abscesses is polymicrobial with coliforms and anaerobes predominating.[18,26] Recommended antibiotics include an extended-spectrum β-lactam, a second- or third-generation cephalosporin with metronidazole or clindamycin, or a newer fluoroquinolone with metronidazole or clindamycin.

The patient may change the gauze dressing as often as necessary to keep the outside of the dressing dry. Instruct the patient to return for follow-up in 48 hours for removal of packing if placed and a wound check. The patient may begin sitz baths or showers 24 hours after the procedure. They should thoroughly clean the wound with soap and water at least once a day until the wound is fully healed. It is helpful to let the stream of shower water run inside the wound to aid in irrigation. Healing may take several weeks depending upon the size of the abscess. Additional measures to aid in healing and comfort include stool bulking agents and stool softeners. Pain can be controlled with nonsteroidal anti-inflammatory drugs supplemented with occasional narcotic analgesics.

Advise the patient that the condition is likely to recur. They must be referred to a General or Colorectal Surgeon who is experienced in the management of anorectal infections. Inform the patient that they may require an operation to prevent future recurrences. Instruct the patient to immediately return to the Emergency Department if they develop a fever, increased pain or redness to the area, or a foul smell to the discharge.

COMPLICATIONS

Perianal abscesses may recur or a fistula may form. Recurrence is more likely in children or if the patient has had abscesses in the same location in the past.[27] Anorectal infections progress to necrotizing fasciitis in less than 1% of patients.[28] Any systemic signs and symptoms require surgical consultation, hospital admission, parenteral antibiotics, and a drainage procedure.[29]

Complications associated with the incision and drainage procedure are rare. A linear incision or too small of an incision can result in premature skin closure, incomplete healing of the cavity, and recurrence. Too large of an incision can result in delayed healing. An overly aggressive incision can damage adjacent structures. Postprocedural bleeding is rare.

SUMMARY

Anorectal infections are commonly seen in the Emergency Department. They are thought to be due to obstruction and subsequent infection of the anal glands that form an abscess. Small perianal abscesses and submucosal abscesses can safely be drained in the Emergency Department. A digital rectal examination may aid in determining the extent of an abscess. A General or Colorectal Surgeon should manage patients with fever, signs of toxicity, or

evidence of a deep or extensive infection beyond the perianal area or patients who are immunocompromised. Be alert that abscesses with maximum fluctuance on the buttock are more likely to have ischiorectal extension, are more complex, and are greater in size. Perianal abscesses must be drained expeditiously to prevent their spread. Approximately 50% of anorectal abscesses will develop a fistulous tract. Patients require referral for follow-up with a General or Colorectal Surgeon who can provide wound care as well as manage chronic cases.

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136 Sebaceous Cyst Incision and Drainage

Carlos J. Roldan

INTRODUCTION

Sebaceous cysts are common, present with a very benign evolution, may be located anywhere on the body, and frequently become infected. They are most commonly found on the face, neck, and trunk. Sebaceous cysts are usually asymptomatic unless they become infected, cause compression, or cause a deformity over sensitive areas of the body (**Figure 136-1**). The Emergency Physician must be acquainted with treating infected sebaceous cysts, particularly if they are located on cosmetically important areas such as the face.

ANATOMY AND PATHOPHYSIOLOGY

Sebaceous cysts are the result of obstruction of sebaceous gland ducts. They are freely mobile, slow growing, round shaped, painless, and located in the subcutaneous tissues.[1] The cysts are made of a thin white capsule filled with a thick, cheesy, and keratinous material. Their size is variable and ranges from less than a quarter of an inch to more than 2 inches. These keratin-containing lesions are usually seen in young and middle-aged adults in relation to a pilosebaceous follicle.[2] Sebaceous cysts may be present for many years before infection occurs. Physical examination often reveals a subcutaneous mass that is fluctuant and tender (**Figure 136-1**). The overlying skin may appear normal or erythematous.

The initial treatment of choice of an infected sebaceous cyst is incision and drainage. The sebaceous material is too thick to allow for spontaneous drainage and it must be expressed. **The sebaceous cyst will likely recur unless the capsule of the cyst is removed.** Patients may have the initial incision and drainage performed in the Emergency Department with follow-up at some later date to remove the cyst capsule. Alternatively, the cyst capsule may be removed at the time of the initial incision and drainage (**Figure 136-2**).

Ultrasound is a useful tool for the diagnosis of a sebaceous cyst.[3-5] It allows for the differential between it and its mimics (e.g., cutaneous masses, hernia, lipoma, lymph node, subcutaneous malignancies, or a vascular abnormality). It can be difficult to distinguish between a sebaceous cyst and another entity. A sebaceous cyst may appear as a small, well-defined, and superficial mass. The inside appearance can vary with the contents.

FIGURE 136-1. An example of an infected sebaceous cyst located on the posterior neck. (Used from Steven Fruitsmack, www.commons.wikimedia.org.)

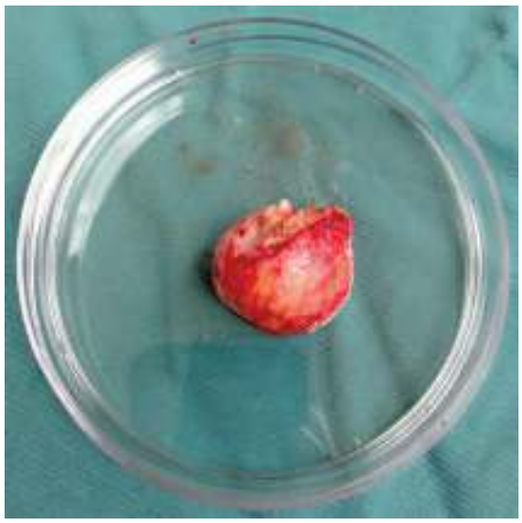

FIGURE 136-2. The removed sebaceous cyst. Note that it is enclosed in a connective tissue capsule. (Used from Steven Fruitsmack, www.commons.wikimedia.org.)

INDICATIONS

Incision and drainage in the Emergency Department is indicated whenever a patient presents with a tender sebaceous cyst consistent with an abscess. The procedure will relieve the patient's pain. Antibiotics without drainage are ineffective in treating abscesses.[6] The vast majority of infected sebaceous cysts may be drained in the Emergency Department, Urgent Care setting, clinic, or office setting. A noninfected sebaceous cyst may be removed electively and for cosmetic purposes in the clinic or office setting by a Primary Care Provider or a Surgeon (**Figure 136-2**).

CONTRAINDICATIONS

There are no absolute contraindications to the incision and drainage or removal of an infected sebaceous cyst. Caution is advised in patients with bleeding disorders, taking anticoagulants, or with thrombocytopenia. Incision and drainage is preferred if the overlying skin is cellulitic. The capsule can be removed at a later time. Manage patients with extremely large abscesses, those with cosmetic concerns, or those in which adequate anesthesia is not possible in the Operating Room by a General Surgeon or Plastic Surgeon. The procedure is to be conducted under general anesthesia in the Operating Room if adequate anesthesia cannot be obtained and pain limits the procedure. Refer patients with noninfected sebaceous cysts to their Primary Care Physician, a General Surgeon, or a Plastic Surgeon for removal.

EQUIPMENT

- Gown, face mask, and gloves
- Povidone iodine or chlorhexidine solution
- 10 mL syringe
- 25 or 27 gauge needle, 2 inches long
- Local anesthetic solution, with or without epinephrine
- #11 scalpel blade on a handle
- #15 scalpel blade on a handle
- Curved hemostat
- Iris scissors
- Ribbon gauze, plain or iodinated
- 2×2 gauze squares
- Adhesive tape
- Sterile saline
- Nylon sutures for skin closure, various sizes
- 3–0 Vicryl suture
- Needle driver

PATIENT PREPARATION

Explain and document the risks, benefits, and potential complications of the procedure to the patient and/or their representative. Explain the postprocedure care. Obtain an informed consent for the procedure. Obtain an additional informed consent for the procedural sedation or nitrous oxide procedure if it applies. Wear a face mask, gown, and gloves for the entire procedure. The injection of local anesthetic solution can force abscess contents to shoot out. Incision of a tense abscess can also result in contamination of the Emergency Physician.

Antibiotic resistance is a growing concern. The incision and drainage of a simple and noncomplicated skin abscess does not require antibiotic therapy.[7,8] The incision and drainage procedure, especially when forceful digital pressure is required, may release bacteria into the circulation. Consider the use of preprocedural intravenous antibiotics in patients suspected or known to be immunocompromised, with a history of prosthetic heart valve replacement, with a history of artificial joint replacement, or with signs of systemic toxicity.

Clean any dirt and debris from the skin overlying the abscess or cyst. Apply povidone iodine or chlorhexidine solution and allow it to dry. Apply drapes to delineate a procedural field and absorb any material or blood that escapes from the abscess cavity.

ANESTHESIA

Anesthesia is often difficult to obtain. Local anesthesia or specific nerve blocks should be considered the first choice. Direct infiltration of the skin and soft tissues in a fan-like pattern surrounding an abscess or "field block" provides sufficient anesthesia to tolerate the procedure (**Figure 136-3**).[9] **Local anesthetics are weak acids,**

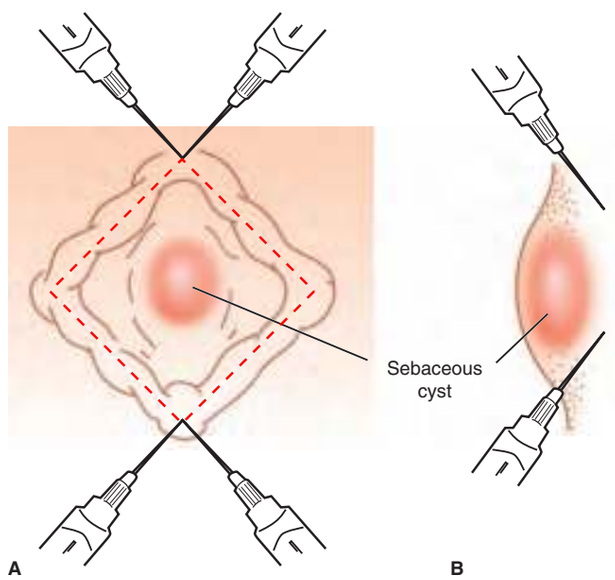

FIGURE 136-3. Field block anesthesia. ***A.*** Local anesthetic solution is infiltrated subcutaneously on all four sides of the infected sebaceous cyst. ***B.*** Local anesthetic solution is infiltrated deep to the infected sebaceous cyst in a fan-like pattern.

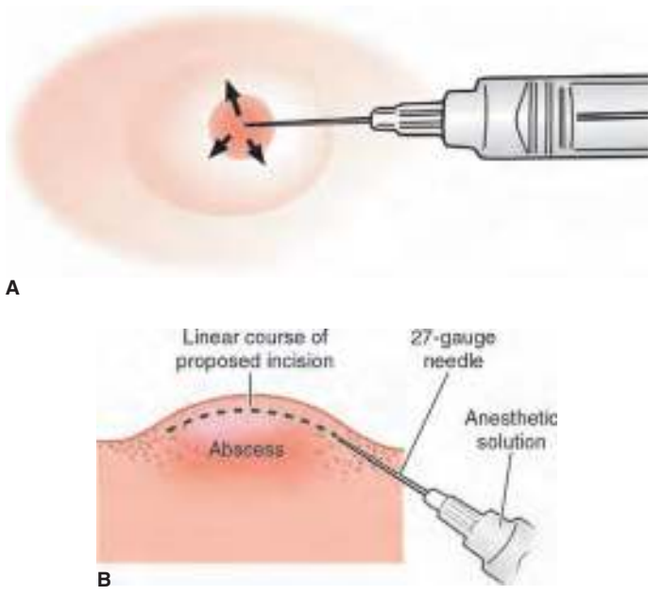

FIGURE 136-4. Subcutaneous infiltration of local anesthetic solution. The needle and syringe are held parallel to the skin. The needle is inserted into the subcutaneous tissue overlying the infected sebaceous cyst. Infiltrate the local anesthetic solution as the needle is withdrawn. The skin should blanch (shaded area) if injected properly. ***A.*** Superior view. ***B.*** Lateral view.

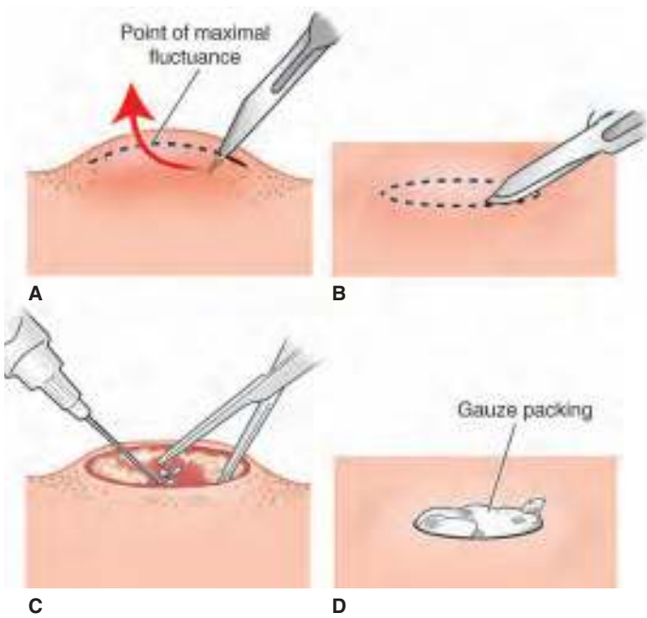

FIGURE 136-5. Incision and drainage of an infected sebaceous cyst. ***A.*** A straight incision to drain the abscess. ***B.*** An elliptical incision to drain the abscess. ***C.*** The wound is irrigated with sterile saline. Pockets of purulent material are opened with the hemostat. ***D.*** The wound is packed open.

less effective in the acidic environment of an abscess, and should not be directly injected in the abscess cavity. The pain caused by injection of the local anesthetic solution is related to the rate that the anesthetic is injected and the force necessary to inject it. Inject the local anesthetic solution slowly through a small-bore needle (e.g., 25 to 30 gauge) as the needle is withdrawn through the dermis. The needle bore will create a passage through the subcutaneous tissue as it is inserted that enables the local anesthetic solution to be infiltrated slowly and with less discomfort.

Hold the syringe horizontally in reference to the skin surface. Inject 3 to 4 mL of local anesthetic solution intradermally over the dome of the abscess (**Figure 136-4**). The skin will blanch if the injection is given properly. The increased pressure within the cavity will cause more discomfort to the patient and may cause the solution to be forcefully expelled if there is an opening in the skin; therefore, some Emergency Physicians prefer to skip this step. Alternative anesthesia could be accomplished with regional or individual nerve blocks (Chapter 156) when the abscess is located in an anatomic area of innervation.

Systemic analgesia (i.e., procedural sedation, Chapter 159) is strongly recommended in the pediatric population and it may occasionally be required as well in adults when a field anesthesia has suboptimal results. In rare occasions, the procedure should be conducted under general anesthesia in the Operating Room if adequate anesthesia cannot be obtained and pain limits the procedure.

TECHNIQUES

INCISION AND DRAINAGE

Several techniques have been described, such as the squeeze technique (i.e., cyst squeezed out through an elliptical or linear incision) and an oval incision at the top or around the punctum of the cyst to dissect it from the surrounding tissue to decrease scar tissue.[10,11] A traditional technique is practical and effective when used in the Emergency Department.

Make a stab incision with a #11 scalpel blade in the skin overlying the area of fluctuance (**Figure 136-5*A***). The incision should be parallel to any lines of tension to produce the least conspicuous scar, particularly in cosmetically important areas such as the face. Extend the incision the length of the fluctuant area with a #11 or #15 scalpel blade unless the abscess is in a cosmetically important area. A linear incision is adequate, although some advocate a cruciate incision. The cruciate incision results in greater scarring and is probably is not necessary. An elliptical incision can be performed in non–cosmetically important areas (**Figure 136-5*B***). **The purpose of the elliptical incision is to remove a full-thickness wedge of tissue so that the wound will remain open. Limit the length of the incision on cosmetically important areas to 3 to 4 mm.** This is just large enough to drain the abscess.

Express the pus and the sebaceous material. It is too thick to drain spontaneously. It is important that loculations be lysed and the area be thoroughly drained to minimize recurrence. Insert a hemostat and spread the jaws within the cavity (**Figure 136-5*C***). Useful technique employs gauze clamped in the jaws of a hemostat and swirled inside the abscess cavity to break adhesions and remove debris. Irrigate the cavity with normal saline solution (**Figure 136-5*C***).

Loosely pack the wound cavity with ribbon gauze or gauze squares to prevent the skin edges from closing prematurely if a linear incision was made (**Figure 136-5*D***). Cruciate and elliptical incisions do not require packing of the wound. Cover the wound with a bulky gauze dressing to soak up continued drainage.

INSERTION OF A de Pezzer CATHETER

A de Pezzer catheter is inserted by some to drain the infected sebaceous cyst.[12] Prepare the skin, incise the cavity with a stab incision, and insert a hemostat to break up the loculations and/or remove the capsule. Insert the de Pezzer catheter into the cavity. Use a size 10 to 16 French de Pezzer catheter depending on the size of the cavity. The de Pezzer catheter keeps the wound open while it heals without the use of packing. Place a safety pin or umbilical clamp on the catheter to prevent it from falling into the cavity. Trim the catheter just past the safety pin or umbilical clamp. Apply a dressing over the wound.

INCISION AND DRAINAGE WITH PRIMARY CYST REMOVAL

The entire sebaceous cyst, including the capsule, can be removed at the time of the incision and drainage with a simplified technique (**Figure 136-2**).[13] Make an incision in the skin overlying the center of the sebaceous cyst. Extend the incision to be slightly longer than the diameter of the sebaceous cyst. **Do not cut into the dermis or subcutaneous tissues.** Sharply dissect the sebaceous cyst free of the surrounding subcutaneous tissues with an iris scissors. The delineation between the thin, shiny, white capsule and the surrounding tissues is very obvious. **Do not puncture the capsule of the sebaceous cyst.** Doing so and spilling some of the contents sets up a nidus for subsequent infection or reformation of the sebaceous cyst. Start at both ends of the incision and free the cyst circumferentially. Free the sides of the cyst from the surrounding adipose tissue. Gently grasp the top of the cyst with forceps or a hemostat. Elevate the cyst. Dissect the inferior border of the cyst free until it can be removed. Irrigate the wound with at least 200 mL of normal saline solution. Allow the cavity to heal by granulation. Alternatively, close the pocket with loosely applied 3–0 Vicryl deep sutures and approximate the skin edges with nylon sutures.

The cyst capsule can often rupture when attempting to remove it intact. If this occurs, express the contents as if incising and draining an abscess. Gently flush the cyst cavity with normal saline. Grasp the shiny, cut edges of the capsule with a hemostat. Gently elevate the cyst capsule edges and dissect the complete cyst capsule free from the surrounding adipose tissue. Irrigate the wound with at least 200 mL of normal saline solution. Allow the cavity to heal by granulation. Alternatively, close the pocket loosely with 3–0 Vicryl deep sutures and approximate the skin edges with nylon sutures.

Submit the complete cyst and capsule or the ruptured capsule for pathologic diagnosis in a sterile container. Several pathologic conditions can mimic a sebaceous cyst. This includes adenomas, adenocarcinomas, dermoids in children, and melanomas. Preauricular tender masses can be parotid gland tumors.

According to one study, this technique results in fewer days to heal, less pain for the patient, and less scarring than with incision and drainage alone.[13] However, this was not a blinded study and no other studies could be found to verify the results. The researchers noted that primary resection (average of 50 minutes) takes longer than simple incision and drainage (average of 10 minutes). This may limit its use in the Emergency Department.

AFTERCARE

Antibiotics are generally unnecessary to treat a simple abscess unless there is cellulitis of the skin surrounding the wound.[6] No data could be found regarding patients with an abscess who are diabetic, have cardiac valve disease, who have hardware in their body, or who are immunocompromised. These patients are at risk for infectious complications. It is advised to cover these patients with antibiotics. There are no data on the optimal duration of antibiotic treatment. The conventional 7 to 10 day course of antibiotic coverage is probably adequate.

Bacteriology of cutaneous abscesses remote from the rectum usually shows aerobic skin flora, with *Staphylococcus* and *Streptococcus* being the most common etiologies.[8] Antibiotics recommended are a first-generation cephalosporin, a penicillinase-resistant penicillin, or a newer fluoroquinolone. Prescribe clindamycin, doxycycline, or trimethoprim-sulfamethoxazole if methicillin-resistant *Staphylococcus aureus* is suspected as the etiology.[14]

Instruct the patient to change the gauze dressing as often as necessary to keep the outside of the dressing dry. Patients should have scheduled follow-up in 48 hours for a wound check and removal of the packing. The packing should be removed in 24 hours if the wound is on the face. Reinsert the packing upon follow-up if the skin closes on its own. Incisions that remain open or that have an elliptical or cruciate incision do not require packing. Advise the patient to change the packing every 24 to 48 hours depending on the amount of drainage. Decrease the amount of packing each time to allow the wound to heal from the base outward. The patient should thoroughly wash the wound with soap and water in the shower each time the packing is removed. It is helpful to let the stream of shower water run inside the wound to aid in wound irrigation. Discontinue the packing once the wound is well granulated and there is no concern that the skin edges will adhere to each other. The patient must continue to clean the wound thoroughly every day until it is fully healed.

Healing may take 1 to several weeks depending on the size of the abscess cavity, the patient's age, and any comorbidities. Instruct the patient to return to the Emergency Department immediately if they develop a fever, increased pain, or worsening redness of the skin surrounding the abscess. Pain relief can be provided with nonsteroidal anti-inflammatory drugs. Narcotic analgesics may occasionally be required in the first 24 hours after the procedure.

Inform the patient that incision and drainage in the acute care setting is not definitive treatment and that the condition is likely to recur unless the cyst is removed. Refer the patient to a physician who can provide wound care as well as remove the cyst capsule.

COMPLICATIONS

Sebaceous cyst infections may spread if the wound is inadequately drained. Attention must be paid to underlying anatomic structures (e.g., branches of cranial nerve VII) when draining facial abscesses to avoid complications caused by inadvertently incising these structures. Incomplete removal of the cyst wall or spillage of the cyst contents sets up a nidus for future infection and/or recurrence of the sebaceous cyst. A linear incision or too small of an incision can result in premature skin closure, incomplete healing of the cavity, and recurrence. Too large of an incision can result in delayed healing and significant scarring. Postprocedural bleeding is rare.

SUMMARY

Infected sebaceous cysts are commonly seen in the Emergency Department. They are thought to be due to blockage of the ducts of sebaceous glands that subsequently become infected and form an abscess. Most cutaneous abscesses can be drained in the Emergency Department. Consult a Surgeon for patients with large abscesses who require drainage in the Operating Room. Patients with signs of fever or toxicity should be admitted for parenteral antibiotics, incision and drainage, and observation. Refer patients to a physician who can provide wound care as well as definitive excision of the sebaceous cyst.

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137 Hemorrhage Control

Christopher Freeman and Ariana Wilkinson

INTRODUCTION

Control of external hemorrhage from an injury is a priority of basic first aid. It begins with first responders before arrival at the hospital and continues with Physicians in the resuscitation suite (**Figure 137-1**). Bleeding from extremity wounds is common. Most bleeding is a minor inconvenience for the busy Emergency Physician in the crowded Emergency Department, prolonging wound closure and complicating wound healing. Major exsanguinating hemorrhage can be life-threatening. Hemorrhage from extremity injuries was a leading cause of death in the Vietnam War and Operation Desert Storm.[1,2] Hemorrhage remained the leading cause of death in Operation Iraqi Freedom and Operation Enduring Freedom, with torso hemorrhage leading the deaths.[3] **Methods for rapid and effective control of bleeding are essential in managing traumatic injuries and optimizing wound management.**

ANATOMY AND PATHOPHYSIOLOGY

Hemostasis is the first biological response to injury.[4-6] Hemostatic platelet plugs form at the ends of transected vessels within seconds of traumatic disruption of the skin. Fibrin fibers gather about the platelet plug within minutes. This fibrin mesh becomes part of an early matrix that initiates wound healing.[4]

Hemostasis is the priority in wound management for the Emergency Physician caring for traumatic wounds.[7] Control of bleeding is necessary to establish hemodynamic stability and prevent further blood loss (Figure 137-1). Hemostasis is the first step in preparing for wound closure. Inadequate hemostasis with hematoma formation impairs wound healing, increases the risk of wound infection, leads to tissue ischemia, and results in hypertrophic scars.[8,9] Large hematomas may cause delayed wound dehiscence.

Bleeding from wounds may be superficial or deep. Superficial wounds (e.g., abrasions, avulsions, or simple lacerations) involve damage to the epidermis, dermis, and subcutaneous tissue. Bleeding from most superficial wounds is predominantly from capillaries, small veins, or arterioles. Wounds deep to the fascia involve larger vessels and are typical of deep punctures, gunshot wounds, major crush injuries, and stab wounds. The approach to the bleeding wound will depend upon the nature of bleeding (e.g., large vessel versus small, discrete source versus diffuse), the site of injury, and its association with other major organ injury (**Figure 137-2**).

FIGURE 137-1. A traumatic amputation with a SWAT-T Tourniquet. (Courtesy of TEMS Solutions LLC, Abingdon, VA.)

INDICATIONS

The immediate control of excessive bleeding is always a priority during the first contact with the patient. **All bleeding must be controlled (Figure 137-2). Exsanguinating hemorrhage must be immediately controlled.** All other bleeding can wait until the ABCs (airway, breathing, and circulation) and life-threatening conditions are addressed. The timing and selection of specific measures to isolate and treat the bleeding source will depend upon the management priorities for each patient. A simple compressive dressing or tourniquet may be used as a first-line measure to control bleeding in the multiple trauma patient. Definitive measures may be taken early to identify and treat isolated specific injuries.

CONTRAINDICATIONS

There are no absolute contraindications to any technique of bleeding control.[7] Choose the technique best suited to the individual situation. **An impressive wound should not distract or divert attention away from other injuries that may be less dramatic but more immediately life-threatening. Use the simplest and most effective techniques to control hemorrhage when faced with multiple injuries.**

EQUIPMENT

PRESSURE CONTROL

- Blood pressure cuff
- Sterile 4×4 gauze pads
- Elastic bandage
- Tourniquet, limb and junctional

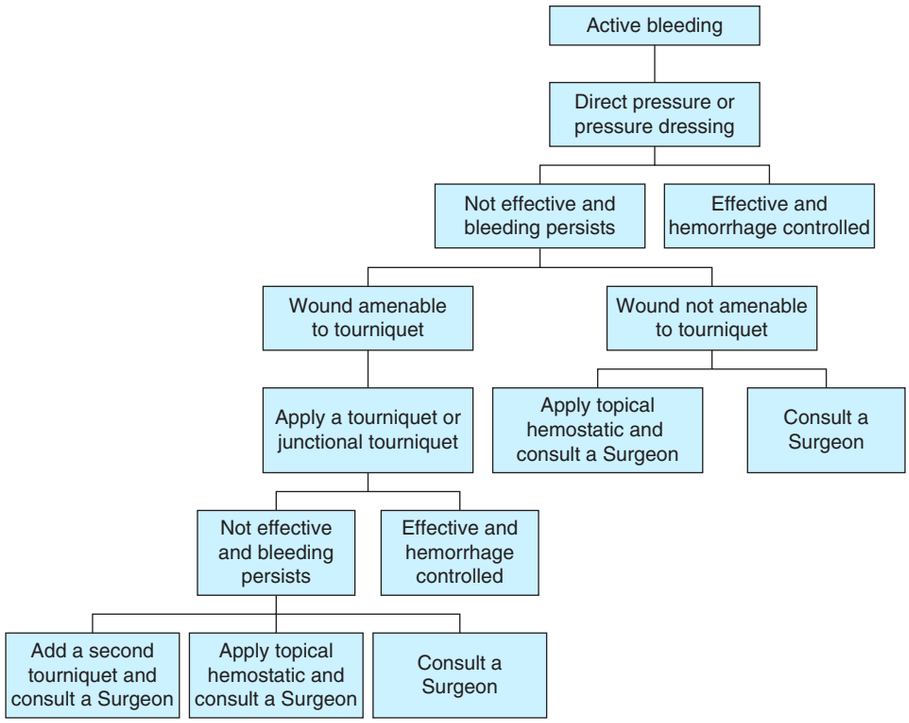

FIGURE 137-2. An algorithm for hemorrhage control.

WOUND MANIPULATION

- Hemostats
- Needle driver
- Assorted suture
- Scissors
- Sterile saline
- 20 mL syringes
- 10 mL syringes
- 18 gauge needles
- 27 gauge needles

ANESTHETICS

- Lidocaine, with and without epinephrine
- Bupivacaine, with and without epinephrine
- 18 gauge needles
- 27 gauge needles
- 10 mL syringes

WOUND CAUTERY

- Silver nitrate ($AgNO_3$)
- Electrocautery unit
- Monsel's solution (i.e., 20% ferric subsulfate solution)
- Drysol (i.e., 30% aluminum chloride solution)

VASOCONSTRICTORS

- 1:1000 epinephrine
- 1% to 4% cocaine
- Tetracaine, epinephrine, and cocaine (i.e., TEC or TAC) solution

TOPICAL HEMOSTATIC AGENTS

- Gelfoam
- Surgicel
- Cellulose
- Dry gelatin
- Thrombin
- Microfibrillar collagen
- Cyanoacrylate
- Various agents as listed in the techniques section

MISCELLANEOUS SUPPLIES

- Povidone iodine or chlorhexidine solution
- Penrose drain
- Finger tourniquet
- Hemoclips
- Hemoclip applicator
- Bone wax
- Raney scalp clips and applier
- Resuscitative endovascular balloon occlusion of the aorta supplies (Chapter 74)
- Tranexamic acid

PATIENT PREPARATION

Control of hemorrhage is the priority. Attention to wound preparation should not delay definitive action to control heavy bleeding. Obtain intravenous access and a type and crossmatch for blood products in any patient with active bleeding and hemodynamic compromise while applying direct pressure to the bleeding site. Explain the procedures to the heavily bleeding patient while

preparing for and performing the procedures. There is no need to obtain an informed consent for the procedure if the bleeding is heavy. Document this in the medical record. Explain the risks, benefits, and alternative procedures to the patient and/or their representative if the bleeding is minimal. Obtain an informed consent for the procedure and place this in the medical record.

Administer a local anesthetic prior to significant wound manipulation if the injury is minor and the patient is stable. Irrigate contaminated wounds free of foreign bodies and debris. Clean the surrounding area with an antiseptic solution (e.g., povidone iodine or chlorhexidine). Refer to Chapter 114 for the complete details of wound cleansing and preparation.

TECHNIQUES

DIRECT PRESSURE

The quickest and easiest method to stop bleeding is the application of direct pressure to the bleeding site.[10-12] Poor lighting may prevent exposure and visualization necessary to identify discrete bleeding sites before hospital arrival. A compressive dressing may be the best option to initially control bleeding. **Many compressive bandages apply too little pressure over too wide an area and act more like a sponge than a pressure dressing. Significant blood loss can be hidden within a bulky pressure dressing.**

Explore a bleeding wound as soon as lighting is sufficient and circumstances allow. **Brisk bleeding frequently has a few discrete sources that can be easily managed once identified.** Direct pressure over bleeding vessels allows time for a platelet plug to form and gives a chance for the body's natural mechanisms of hemostasis. Apply pressure over arterial wounds for 10 to 15 minutes to control most bleeding. Apply pressure to a proximal artery to impede arterial inflow and control, or slow, the bleeding when wound exploration is not practical.[13,14]

TOURNIQUETS

The use of tourniquets for extremity hemorrhage has received a great deal of attention throughout history. **Tourniquets are seldom necessary to control hemorrhage.**[15] **Direct pressure is more effective and causes less tissue ischemia.** Tourniquets may be required to control bleeding and free personnel to attend to other concerns if there is significant bleeding. Use tourniquets as a last resort when other methods fail and the patient's life is in jeopardy.[13,15] The correct application of a tourniquet causes the patient pain. **Do not remove the tourniquet.** It is placed to prevent exsanguination. Some of the currently available tourniquets include the Combat Application Tourniquet (Composite Resources, Rock Hill, SC), the Special Operations Forces Tactical Tourniquet (Tactical Medical Solutions, Anderson, SC), and the Emergency and Military Tourniquet (Delfi Medical, Vancouver, Canada).

Many mistakes are made with tourniquets (**Table 137-1**). This includes not taking off the tourniquet within 2 hours, not tightening the tourniquet enough, taking the tourniquet off too soon, and restoring blood flow to the limb before tourniquet damage. The complications associated with a tourniquet are listed in **Table 137-2**.

There is no definitive time to safely apply a tourniquet. There is a risk of limb ischemia and eventual limb loss any time a tourniquet is used.[7] It is generally accepted that 2 hours is a safe time.[16-18] It is believed that beyond this time permanent muscular or neurologic injury may occur. A tourniquet has been safely applied for up to 6 hours without permanent complications in some cases.[18-20] **Use the minimal tourniquet pressure necessary to maintain hemostasis. Release the tourniquet periodically and reassess the extremity.**

TABLE 137-1 Common Errors of Using a Tourniquet

Not making the tourniquet tight enough
Not taking the tourniquet off when not needed
Not using a second tourniquet if needed
Not using a tourniquet when indicated
Periodically loosening the tourniquet
Placing a tourniquet too proximally with distal bleeds
Removal of the tourniquet by emergency medical services
Removing the tourniquet if the patient complains of pain
Removal of the tourniquet if the extremity is amputated
Removing the tourniquet if other hemorrhage control methods are contraindicated
Removing the tourniquet within 2 hours if bleeding is not controlled
Removing the tourniquet if the wound cannot be reexamined
Removing the tourniquet when the patient is in shock
Using a tourniquet for minimal bleeding
Waiting too long to place the tourniquet

It is best to limit the use of a tourniquet for the minimum time required to temporarily be lifesaving and until definitive care can be received.

JUNCTIONAL TOURNIQUETS

Junction hemorrhage or proximal extremity hemorrhage is the most common cause of death from compressible hemorrhage. **Junctional hemorrhage is defined as hemorrhage from the junction of the torso and extremity or proximal extremity where a traditional tourniquet is unable to be applied.**[21] Topical hemostatic agents, direct pressure, or a combination were used for junctional hemorrhage without much success.

There are now four devices that have been approved by the Food and Drug Administration (FDA) for junctional hemorrhage control in the military and prehospital setting.[22-24] These devices are easy and safe to use, effective, low in cost, lightweight, quickly applied, and small. **These devices, like extremity tourniquets, are not meant as an alternative to surgical intervention but are a bridge to definitive treatment.**

The Abdominal Aortic and Junctional Tourniquet (AAJT) is a pneumatic belt that allows for prolonged delivery of pressure on

TABLE 137-2 Complications of Using a Tourniquet

Local
Bone injury
Compartment syndrome
Delayed recovery
Neurapraxia from compression
Pain
Soft tissue injury
Stiffness
Swelling
Vascular injury
Wound hematomas
Wound infections
Systemic
Acid-base disturbances
Cardiac decompensation
Deep venous thrombosis
Elevated central venous pressure
Fibrinolysis
Hypertension
Respiratory decompensation
Rhabdomyolysis
Stroke

FIGURE 137-3. The Abdominal Aortic Junctional Tourniquet (AAJT). (Courtesy of Compression Works LLC, Birmingham, AL.)

one area (Compression Works LLC, Birmingham, AL).[25,26] The circumferential device uses a belt, windlass, and pneumatic pressure to compress the aorta (**Figure 137-3**). It can be used up to 4 hours. The wedge-shaped bladder provides pressure over a large surface area and allows a lower overall tissue pressure than other junctional tourniquets. A case report from Afghanistan showed safe and effective cessation of severe internal bleeding from an unknown battlefield injury after the patient became bradycardic with impending cardiac arrest.[24] The AAJT effectively tamponades the abdominal aorta. This device may show some utility in the management of severe pelvic bleeding as the internal iliac artery branches off the aorta distal to the area of occlusion and can stabilize the pelvis.

The AAJT is simple to apply. Place the belt of the of the AAJT around the abdomen with the inflatable cuff overlying the umbilicus. Manually tighten the buckle of the belt. Further tighten it with a windlass device located on the front of the belt. Inflate the cuff. The AAJT can be used for upper extremity junctional hemorrhage by placing it in the axilla.

The Combat Ready Clamp (CroC) is designed for control of junctional hemorrhage in the inguinal and axillary area (Combat Medical Systems, Harrisburg, NC).[26-30] This device was created to exert mechanical pressure with plastic circular discs directly over a junctional wound or indirectly over proximal vessels to occlude blood flow to the wound (**Figure 137-4**). The disc is tightened down onto the desired area by turning a handle attached to a metal vertical rod. There is currently limited evidence on its use. A study

FIGURE 137-4. The Combat Ready Clamp (CroC). (Courtesy of Combat Medical Systems, Harrisburg, NC.)

FIGURE 137-5. The Junctional Emergency Treatment Tool (JETT). (Courtesy of North American Rescue LLC, Greer, SC.)

showed the CroC had 100% efficacy for stopping axillary junctional hemorrhage.[31]

The Junctional Emergency Treatment Tool (JETT) was developed by the University of Texas Health Science Center for Translational Injury Research and North American Rescue Products LLC (Greer, SC). The JETT is for use only with inguinal junctional hemorrhage.[26] It is unique in that it has a pelvic binder component and has the capability to compress one or both femoral arteries (**Figure 137-5**). The JETT uses a windlass mechanism to cinch down the device and mechanically provide direct pressure.

The SAM Junctional Tourniquet was approved by the FDA for axillary hemorrhage, lower extremity junctional hemorrhage, and unstable pelvic fractures (SAM Medical Products, Wilsonville, OR).[26,28,32] It is effective at controlling bleeding in proximal limbs or other high-level amputations.[27] It contains a belt that is fastened around the abdomen or pelvis and two inflatable cuffs known as Target Compression Devices (TCDs) (**Figure 137-6**). The TCDs are placed proximal to the injury and inflated to effectively occlude blood flow. A case report showed the efficacy of the SAM Junctional Tourniquet in the battlefield for severe inguinal hemorrhage.[33]

Junctional tourniquets have advantages and disadvantages. Additional benefits of junctional tourniquets are that they can be used as pelvic binders and may aid in tamponade of severe pelvic bleeding from pelvic fractures. Complications of junctional

FIGURE 137-6. SAM Junctional Tourniquet. (Courtesy of SAM Medical, Wilsonville, OR.)

tourniquets include ischemic injury if left on too long and continued hemorrhage if applied incorrectly. There are otherwise very few risks to applying these devices. Junctional tourniquets have safe application times between 1 and 4 hours and are used only as a temporizing measure to get patients definitive care in the Operating Room.[34] None of these devices have been studied in prospective or randomized controlled trials. The only studies available are animal studies, manikin bleeding models, or case reports. The implementation by trained professionals in the correct setting could be lifesaving.[35]

EXTREMITY TOURNIQUETS

The use of extremity tourniquets is not new. Much of the literature supporting the use of extremity tourniquets has been produced by the military.[36-38] Civilians have close access to prehospital providers who can focus on hemorrhage control. Civilian extremity injuries are often less severe than those seen in war. Despite this, the application of an extremity tourniquet can be lifesaving for civilians. Below is a representative sample of extremity tourniquets.

The Basic Windlass Tourniquet (TBWT) is a reusable tourniquet (rthInnovations LLC, Gloucester, VA). It is made of a heavy-duty polypropylene weave with a breaking strength of 1200 pounds (**Figure 137-7**). The TBWT does not require clips, knots, pins, or ties to secure the device.

The SWAT-T tourniquet is a single-use device (TEMS Solutions, Abingdon, VA). It is composed of elastic that wraps around an extremity (**Figure 137-8**). The SWAT-T is easy to apply in seconds with little prior training. It is effective in wet conditions. Application requires two hands.

The Combat Application Tourniquet (C-A-T) was designed for one-handed use (North American Rescue LLC, Greer, SC). It has been used by the United States Army in the field. It applies quickly with minimal prior training. It has a Velcro retention strap and windlass mechanism that locks in place (**Figure 137-9**).

The SAM XT is a single-use tourniquet (SAM Medical, Wilsonville, OR). It uses a buckle to clamp on the extremity and a windlass mechanism to tighten (**Figure 137-10**). The device requires simple training and is easy and quick to apply. It is made of nylon and autolocks upon tightening to eliminate slack.

BANDAGES

These devices include the Emergency Bandage (PerSys Medical Co., Houston, TX), H-Bandage (H and H Medical Corp., Williamsburg, VA), and the WoundStop (PerSys Medical Co., Houston, TX) (**Figure 137-11**). These consist of an elastic-containing compression bandage, similar to an Ace wrap, and a nonadherent pad. The single-use bandages are quick and easy to apply with two hands. The bandages are sterile and single-use. Place the pad over the bleeding site and tightly wrap the bandage around the extremity. Secure the end of the bandage.

BALLOON CATHETERS

Physicians have been using balloon catheters to tamponade exsanguinating bleeding from deep wounds or wound tracks.[39,40] Devices used include Fogarty catheters, Foley catheters, and Sengstaken-Blakemore tubes. Balloon catheters are blindly placed in the wound and inflated to tamponade bleeding. These devices are not specifically designed for this purpose. **They are used as an improvised technique to temporarily tamponade the hemorrhage.** Reviews have shown that balloon tamponade was useful in the initial control of bleeding from limbs and intraabdominal sources.[41]

A

B

C

FIGURE 137-7. The Basic Windlass Tourniquet (TBWT). ***A.*** Device. ***B.*** Use on a lower extremity. ***C.*** Use on an upper extremity. (Courtesy of rthInnovations LLC, Gloucester, VA.)

Tourniquets specific for deep wound hemorrhage are being developed. These devices may be used when conventional external methods (e.g., clotting agents, direct pressure, and tourniquets) do not control the hemorrhage. This group of devices are inserted into the wound or wound track and inflated to tamponade the bleeding. One of these devices is the TourniCath (CardioCommand Inc., Tampa, FL). It uses a double-walled cylindrical balloon to cause wound track compression

A

B

FIGURE 137-8. The SWAT-T Tourniquet. ***A.*** The SWAT-T. ***B.*** Use on a lower extremity. (Courtesy of TEMS Solutions LLC, Abingdon, VA.)

FIGURE 137-9. Combat Application Tourniquet (C-A-T). (Courtesy of North American Rescue LLC, Greer, SC.)

FIGURE 137-10. The SAM XT Extremity Tourniquet. (Courtesy of SAM Medical, Wilsonville, OR.)

A

B

C

FIGURE 137-11. Wound bandages. ***A.*** H-Bandage. (Courtesy of H and H Medical Corp., Williamsburg, VA.) ***B.*** Emergency Bandage. (Courtesy of PerSys Medical Co., Houston, TX.) ***C.*** WoundStop. (Courtesy of PerSys Medical Co., Houston, TX.)

FIGURE 137-12. The TourniCath System. (Courtesy of Cardio Command Inc., Tampa, FL.)

(**Figure 137-12**). It was designed to insert easily into the wound tract and is inflated up to 400 mL. The device measures 8×2 inches when inflated. These devices have not yet been fully evaluated for use in the prehospital setting or the Emergency Department.

SUTURE LIGATION

Thoroughly inspect briskly bleeding wounds. Place a blood pressure cuff proximally and inflate it until a dry bloodless field is obtained. Large-vessel bleeding will first become apparent as the cuff pressure is slowly dropped. **Large and intermediate-sized vessels will need to be ligated or oversewn for effective control.** Familiarity with the vascular supply to the extremities will help the Emergency Physician anticipate major arterial injuries and look for likely bleeding sources. **Search for the ends of a blood vessel whenever a transected vessel is seen.** A retracted artery in spasm will likely bleed later. Actively search for blood vessel ends and ligate them.

Ligate bleeding vessels that can be visualized (**Figure 137-13**). Grasp the cut end of the bleeding vessel with a hemostat (**Figure 137-13*A***). Pass an appropriate-sized suture around the vessel (**Figure 137-13*B***). Use absorbable sutures that do not lose their tensile strength too soon (e.g., Vicryl, Monocryl, and PDS). Tie and secure the suture around the base of the bleeding vessel (**Figure 137-13*C***). Gently release the hemostat from the blood vessel.

Cut blood vessels, especially arteries and arterioles, often retract into the tissue and are difficult to visualize. A suture can be used to control the bleeding (**Figure 137-14**). Place a figure-of-eight stitch (**Figure 137-14*A***) or a purse-string stitch (**Figure 137-14*B***) to encompass the blood vessel. These sutures are easy to place, quick, and simple.

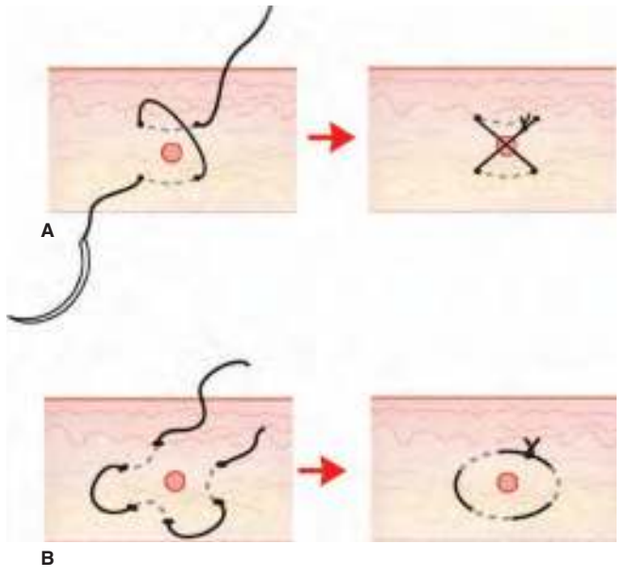

FIGURE 137-14. Control of a bleeding vessel deep or embedded in tissue. ***A.*** The figure-of-eight stitch. ***B.*** The purse-string stitch. Note that both of these stitches are not tied tightly for the sake of clarity. In real use, both of these stitches will be tied tightly to seal the bleeding vessel.

CAUTERY

Place a blood pressure cuff proximally and inflate it until a dry bloodless field is obtained. Slowly deflate the blood pressure cuff. Identify small bleeding vessels as the pressure is gradually reduced. **The most likely source of significant bleeding is from dermal arterioles and veins.** Venous bleeding usually stops with direct pressure. Dermal arterioles tend to resist direct pressure and cause persistent oozing from the wound edges. Blood vessels are best identified by picking up the wound edge and inspecting the dermis. Bleeding vessels can be effectively treated with electrocautery or chemical cauterization.

Electrocautery is surprisingly easy and effective against aggressive bleeding from small vessels less than 2 mm in diameter.[10] Handheld battery-driven electrocautery units use a heated electrode to deliver a thermal burn to the tissue and char the ends of vessels (**Figure 137-15**). They are simple to use, inexpensive, and stocked in most Emergency Departments. More versatile electrosurgical units (e.g., the Bovie and Hyfrecator) are extremely effective coagulators.[42] These devices are in the Operating Rooms but are not routinely available in most Emergency Departments.

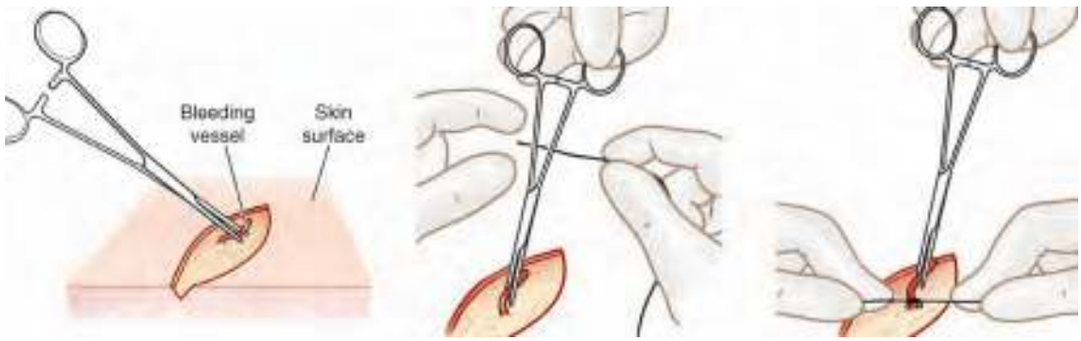

FIGURE 137-13. Control of the bleeding vessel that is visualized. ***A.*** Clamp the cut end of the vessel with a hemostat. ***B.*** Wrap a suture ligature about the base of the vessel. ***C.*** Tie and secure the suture around the base of the bleeding vessel.

FIGURE 137-15. The battery-powered electrocautery device.

Chemical cauterization with silver nitrate ($AgNO_3$) is an effective alternative. The silver nitrate is a dark material that is provided on the end of a wooden applicator stick and resembles a large matchstick (**Figure 137-16**). Rub the silver nitrate over the cut end of the vessel. It forms an insoluble precipitate with tissue protein to form an artificial clot or an eschar that occludes the vessel lumen. **Silver nitrate cannot be used on briskly bleeding vessels as it will coagulate the blood and not the vessel.** The cut vessel must not be bleeding or be just oozing for silver nitrate to coagulate the tissue.

The reduced silver nitrate salts stain the tissue they contact black. Most of the black silver salts are resorbed by the body over several weeks. There is the possibility of permanent staining or tattooing of the skin. **Do not use silver nitrate on light-skinned individuals or close to the skin surface in cosmetically sensitive areas.** Minimize tissue contact with silver nitrate to prevent damage to the underlying tissue.

Two topical solutions can be used for chemical cauterization. Monsel's solution is a 20% ferric subsulfate solution that is thick and dark brown to black in color. **It can permanently stain or tattoo the skin.** Drysol solution is a 30% aluminum chloride solution that is colorless. It will not stain or tattoo the skin. Drysol solution may not be as effective as Monsol's solution for cauterization. Both solutions are applied to a relatively dry or slightly moist area with a cotton-tipped applicator.

Briefly apply the electrocautery or chemical cautery directly to the bleeding source. Neither technique will work well unless the field is dry. This can be achieved with the use of suction, or the wound can be dabbed dry with gauze or cotton-tipped applicators or external pressure. More liberal use to the surrounding tissue will leave unnecessary damage and impair wound healing. Overzealous use of electrocautery and chemical cautery can cause unnecessary tissue necrosis and increase the risk of infection.

VASOCONSTRICTORS

Smaller bleeding vessels will usually constrict and eventually stop on their own once major vessels have been treated. The use of local vasoconstrictors and topical hemostatic agents is effective.[43]

FIGURE 137-16. The silver nitrate ($AgNO_3$) applicator.

Epinephrine is a convenient and effective vasoconstrictor.[44] It can be injected into the wound edges with a local anesthetic or placed directly into the wound. Do not use epinephrine and other vasoconstrictors in a finger, toe, ear, nose, or penis where ischemia may cause tissue loss. **Use topical vasoconstrictors with diligent attention to the total dose administered to avoid systemic side effects (e.g., hypertension, tachycardia, and seizures).**

Commonly available local anesthetic solutions containing epinephrine that are available in the Emergency Department include lidocaine and bupivacaine. Inject 1 to 2 mL of local anesthetic solution containing epinephrine into the tissue surrounding the bleeding vessel, cover the wound with saline-moistened gauze, and apply external pressure for 2 to 4 minutes. **Use caution and aspirate prior to the injection to ensure that the solution is not being injected intravascularly.** Alternatively, spray 1 to 2 mL of 1:1000 epinephrine or an epinephrine-containing local anesthetic solution over the wound surface with a 25 gauge needle, cover the wound with a sterile saline-moistened gauze, and apply external pressure for 2 to 4 minutes.[45]

A more dilute epinephrine solution can be used in larger wounds to minimize potential side effects. Solutions as dilute as 1:100,000 to 1:1,000,000 are used to control brisk bleeding that accompanies tangential burn wound excision and graft donor sites.[46] Topical cocaine (1% to 4%) is a potent vasoconstrictor commonly used on mucous membranes. Combinations of 0.5% tetracaine, 1:2000 epinephrine (adrenalin), and 11.8% cocaine (TEC or TAC) are used for topical anesthesia and hemostasis in pediatric wounds.[47] Apply 1 to 2 mL of these solutions directly into the wound followed by an occlusive dressing.

TOPICAL HEMOSTATIC AGENTS

This group of agents has seen most of the recent innovation in hemorrhage control (**Table 137-3**). Most of these agents were originally developed for operative hemostasis. Their use has expanded into the Emergency Department. More and more hemostatic agents are being developed for hemorrhage control outside the Operating Room or for life-threatening hemorrhage when standard agents have failed. Topical hemostatic agents used in the Emergency Department include cellulose, cyanoacrylate tissue adhesive, dry gelatin, inorganic agents, microfibrillar collagen, polysaccharide-based agents, and thrombin. **Use of hemostatic agents does not replace a methodical approach to wound care and a meticulous search for bleeding vessels.**

Oxidized cellulose (i.e., Surgicel) or dry gelatin (e.g., Gelfoam or Surgifoam) based agents can be used to provide hemostasis when there is diffuse oozing, especially where a small amount of blood impedes wound closure and jeopardizes cosmetic outcome. These agents provide a matrix for platelet deposition and aid hemostasis.[8,43] Troublesome wounds can be treated with these agents and covered with a pressure dressing. The dry field can be approximated with sutures after a few minutes. Another option is to leave the hemostatic agent in the wound, close the wound, and apply a pressure dressing. These techniques will be effective for many wounds. These agents are not free of complications. Absorbable gelatin in a wound can produce excessive granulation tissue and fibrosis. Cellulose can cause a foreign body reaction.

Topical thrombin and microfibrillar collagen (e.g., Avitene, Instat, and Helistat) may be useful hemostatic agents in problematic cases with persistent bleeding.[43,48] Apply topical thrombin in powder form or spray diluted thrombin on the wound. Concentrations of 100 units/mL of thrombin are usually effective. Concentrations of 1000 to 2000 units/mL can be used if the bleeding is severe. Microfibrillar collagen can be used to encourage platelet aggregation. Thrombin and microfibrillar collagen are expensive and are not

TABLE 137-3 Characteristics of Topical Hemostatic Agents

Agent	Active ingredient	How supplied	Advantages	Disadvantages
Celox	Chitosan derivative	Granular powder	Antimicrobial Inexpensive	Difficult application
Combat Gauze	Kaolin	Impregnated gauze	No exothermic production	Gauze roll
Dry Fibrin Sealant Dressing (DFSD)	Calcium chloride Human fibrinogen Human thrombin Vicryl mesh	Block on a compression bandage	Self-polymerizes	Costly Dry field works best Requires careful handling
Hemcon	Chitosan	Pads	Antibacterial Antifungal Well-tested	Can stick to gloves Must apply quickly
Quikclot	Zeolite	Gauze impregnated Powder	Gauze easy to handle	Messy powder form Produces heat
SuperQR	Potassium iron oxyacid salt Hydrophilic polymers	Powder	Effective for bleeding	Difficult application Produces heat
Trauma Stat	Chitosan derivative Polyethylene fibers Silica	Gauze	Can be packed into wounds	Gauze roll
Woundstat	Absorbent polymer Smectite	Granules	Effective for bleeding	Difficult application Small wounds

usually supplied outside the Operating Room. Limit their use to the patient with a coagulopathy or severe bleeding unresponsive to other measures.

Cyanoacrylate tissue adhesive (e.g., Dermabond and Orobase) is commonly used in the Emergency Department for wound closure. It is a helpful adjuvant for hemostasis. It forms an occlusive dressing that provides hemostasis. There are few human data demonstrating the hemostatic activity of cyanoacrylate. It has been shown to promote clot formation, decrease bleeding time, and decrease rebleeding in porcine models for epistaxis and femoral arterial injury.[49,50] Cyanoacrylate works best in a dry, bloodless field that can be obtained by a combination of direct pressure, irrigation, and temporary tourniquet use. Hemostasis was achieved in a porcine arterial hemorrhage model in 90% of the animals after temporary tourniquet placement, irrigation, and cyanoacrylate application via spray.[50] The wound can remain uncovered after cyanoacrylate application, allowing for easier recognition of rebleeding.

A relatively new area in topical hemostasis is the management of life-threatening bleeding outside of the Operating Room. This has focused on hemostatic dressings and agents leading to rapid hemorrhage control after application. These products are placed in the wound, covered with gauze, and pressure is applied. More definitive management can be planned after hemostasis is achieved. These agents fit into the broad categories of inorganic and polysaccharide-based hemostatic agents.

The inorganic hemostatic agents include Quikclot Combat Gauze (Z-Medica, Wallingford, CT). The original Quikclot is a zeolite powder that adsorbs water and produces an exothermic reaction.[51] The newer Quikclot products are kaolin based. Kaolin is an inorganic compound that activates the intrinsic clotting cascade and promotes clot formation. Quikclot Combat Gauze is a gauze roll impregnated with kaolin nanoparticulate minerals. The gauze is packed into the wound and pressure is applied until hemostasis is achieved. Human studies are limited. There are case series showing high rates of hemorrhage control in junctional and nonjunctional extremity applications.[52]

Self-expanding hemostatic polymer is a compound that contains a highly absorbent polymer capable of absorbing 30 gm of water for each gram of polymer and a wicking binder. This is contained within a 4 inch microporous nylon bag. It swells rapidly when exposed to liquids and produces a tamponading effect in the wound bed.

The polysaccharide-based agents are classified as *N*-acetylglucosamines containing glycosaminoglycans and microporous polysaccharide hemispheres. The data supporting the use of these agents are limited and mostly derived from animal studies with multiple differing models, making direct comparison between the products difficult. *N*-acetylglucosamine glycosaminoglycans are complex polysaccharides derived from marine microalgae or crustacean shells in the form of chitin or chitosan.[53] These agents are thought to provide hemostasis through multiple mechanisms including attraction of circulating red blood cells, tissue adhesion, and vasospasm.[54] *N*-acetylglucosamine-based agents include Hemcon ChitoGauze (Tricol, Portland OR), Celox (SAM Medical Products, Portland OR), and Modified Rapid Deployment Hemostat (MRDH) (Marine Polymer Technologies, Danvers MA). Hemcon ChitoGauze is available as a coated bandage. Celox is available as granules, granules in a disposable bag, coated flexible gauze, and a plunger for deep application. MRDH is available as a coated gauze. Hemcon ChitoGauze, one of the best-studied agents, has demonstrated mixed results in animal studies but has favorable reports from its limited use in the United States military.[55] There have been prehospital studies of ChitoGauze showing effectiveness in anticoagulated patients with an 89% rate of cessation or slowing of bleeding.[56] A limited study of 10 trauma patients with intraabdominal injuries achieved hemostasis in 90% of the patients with MRDH.[57]

The last type of hemostatic agent and dressings are the procoagulant supplements. They include Dry Fibrin Sealant Dressing (DFSD) and SeraSeal (Wortham Laboratories, Chattanooga, TN). DFSD is composed of clotting proteins purified from donated blood and plasma. The DFSD is a multilayered dressing composed of fibrin, calcium chloride, thrombin, and an absorbable mesh. The dressing combines with blood to become activated and adherent to the tissues. DFSDs require special handling and are very expensive, both of which will limit its use in the Emergency Department. SeraSeal is an FDA-approved clotting factor product derived from cows. This compound contains activated factors II, V, VIII, and XIII in an agar matrix. SeraSeal is applied as drops to the site of active bleeding and promotes the formation of a clot. The studies of effectiveness are scant and its role in the Emergency Department is limited.

The limited human evidence, case reports, and small animal studies make comparison of the various hemostatic agents difficult. The application of these agents is simple. Determining which of these

agents to use is difficult and is guided by local availability and by personal experience with these products. These agents are useful adjuncts in topical hemostasis in the early phase of trauma management in the Emergency Department.

TRANEXAMIC ACID

Tranexamic acid (TXA) is an antifibrinolytic agent that inhibits plasminogen activation. It stops the conversion of plasminogen to plasmin. The main indications for TXA are dental postextraction bleeding, epistaxis, and severe trauma with bleeding. TXA is contraindicated if the patient is allergic to it or has a thrombosis (e.g., arterial and venous). TXA can cause allergic reactions, thrombosis, and toxic epidermal necrolysis.[58,59]

TXA can be used topically for the control of hemorrhage.[59] TXA is available in 100 mg/mL intravenous solution and 650 mg tablets. Soak gauze in liquid TXA, put the gauze in the wound, and cover the wound with a compression dressing. The tablets are much less expensive than the liquid. The tablets can be crushed and placed directly in the wound followed by application of a compression dressing. A paste can be made from the tablets. Crush three tablets of TXA. Add 0.5 mL aliquots of sterile water and mix well. Continue to add sterile water in aliquots up to 3 mL and mix after each water addition until a thick paste is formed. Apply the paste to the wound followed by a compression dressing. Allow the TXA 20 to 30 minutes to work and stop the bleeding. Remove the compression bandage and reevaluate the wound. Remove the TXA if the bleeding has stopped. The powder and paste may be difficult to remove.

ENDOVASCULAR DEVICES

Severe hemorrhage uncontrollable by traditional techniques of direct pressure, tourniquets, and hemostatic agents can be controlled with minimally invasive nonoperative hemostatic tools. Endovascular tools may be used to tamponade bleeding by intrinsically clamping the aorta. Similar tools were first used during resuscitation of ruptured abdominal aortic aneurysms. The resuscitative endovascular balloon occlusion of the aorta (REBOA) device has been successfully implemented in cases of severe life-threatening hemorrhage. This device can be used as an alternative to thoracotomy with aortic cross clamping by providing proximal aortic pressure. REBOA has less physiologic disturbance and higher success rates than aortic cross clamping.

The indication for placement of a REBOA catheter is life-threatening intraabdominal or pelvic hemorrhage where immediate operative control cannot be obtained. It is indicated for bleeding unable to be controlled by less invasive measures (e.g., direct pressure). This is a temporizing measure to stabilize a hemorrhaging patient while definitive methods of hemorrhage control are pursued. See Chapter 74 for the complete details of using REBOA. A brief description is included below.

REBOA can be introduced through the femoral artery using a Seldinger technique under ultrasound guidance. A guidewire is then placed, followed by a 12 French catheter and a 9 French balloon catheter. This procedure is commonly performed under fluoroscopic guidance. There are new kits available that do not require fluoroscopy and allow the operator to insert the device in one pass via an 8 French catheter in the femoral artery. Proper placement in the thoracic aorta was estimated by measuring the distance between the inguinal crease and the midsternum. A study showed a successful placement rate of 87.5% with the fluoroscopy-free technique.[60] Another study examined the use of ultrasound to determine the proper location of the contrast-filled balloon catheter.[61] Ultrasound-guided placement is faster and safer than the traditional technique and has the distinct benefit of not requiring fluoroscopy to determine placement.[61]

There are three different zones of the aorta for the balloon. Zone 1 is the area distal to the left subclavian artery and proximal to the celiac trunk. Zone 2 represents a 3 cm area of the aorta distal to the celiac trunk and proximal to the last renal artery. Zone 3 represents the aorta distal to the renal arteries and extends to the bifurcation into the common iliac arteries. Any of these three zones can be targeted with this technique, but zones 1 and 3 are the most commonly targeted.[62]

There is no definitive time to safely apply REBOA. **There is a risk of ischemia and aortic injury any time REBOA is used.** The least amount of time used the better. Times of 60 minutes or less are thought to be safe.[63] The rates of organ dysfunction and failure, especially kidney failure, increased significantly with times of greater than 90 minutes.

The REBOA device may be used as a bridge to surgery or to interventional vascular procedures for embolization of severe pelvic or splenic bleeding. REBOA may be implemented in the prehospital setting.[64] These devices are primarily used in trauma care. REBOA can be used in nontraumatic cases (e.g., pelvic hemorrhage). Pelvic hemorrhage is hard to access surgically and is generally managed endovascularly. REBOA would likely be the best temporizing measure for hemorrhage.[65]

The main risks are the same with any procedure where a vessel is accessed. They include bleeding, damage to surrounding structures, and infection. One complication unique to the REBOA is rupture of the aorta due to overinflation of the balloon.

REBOA is a newer technique for hard-to-control intraabdominal and pelvic bleeding. More providers are being trained in the use of REBOA. It may become more commonly used in the future to prolong the "golden hour" in patients with significant intraabdominal and pelvic hemorrhage.

TISSUE CLAMPS

Another novel tool for hemorrhage control is a proprietary tissue-clamping tool called the iTClamp (Innovative Trauma Care Inc., San Antonio, TX). The device contains eight small needles that are inserted into the skin on opposite sides of the wound and tightened together by pushing together the top of the device (**Figure 137-17**). The iTClamp can be used to tightly approximate the edges of wounds unable to be controlled by direct pressure. Pressure from the device will tamponade the bleeding. Recent case reports performed in trauma patients showed that the iTClamp was successfully able to tamponade penetrating neck wounds with minimal discomfort.[66] The application of the iTClamp allowed patients to be safely transferred to the computed tomography scanner and subsequently to the Operating Room. Another study used cadaveric hemorrhage models to evaluate the efficacy of the iTClamp for hemorrhage control.[67] Use of the iTClamp reduced fluid loss in any wound, resulted in no alteration in fluid losses with or without movement of the patient, and did not occlude distal arterial blood flow.[67]

FIGURE 137-17. The iTClamp. (Courtesy of Innovative Trauma Care Inc., San Antonio, TX.)

FIGURE 137-18. The Xstat. (Courtesy of RevMedX Inc., Wilsonville, OR.)

WOUND CLOSURE VERSUS PACKING

The wound can be approximated, a pressure dressing applied, and the limb elevated when oozing cannot be controlled by any other method. Alternatively, the wound can be packed with saline-moistened gauze until better hemostasis is achieved. **Apply as much packing into the wound as possible and then pack some more.** Obtain a coagulation profile in anyone with persistent diffuse bleeding. The wound can be approximated after the coagulopathy is corrected if a correctable coagulopathy is identified.

Xstat can be used for all wounds (RevMedX Inc., Wilsonville, OR).[68] It is meant to be used on life-threatening junctional wounds not amenable to standard tourniquets. It should not be used in the abdomen, pelvis, or thorax. It looks like a big syringe filled with pellets (**Figure 137-18**). It contains 92 compressed cellulose sponges coated with a shellfish extract. The Xstat is for single use and may be used for up to 4 hours for bleeding control. Place the applicator in the wound and depress the plunger to fill the wound with the pellets. The number of pellets to inject will vary with the size of the wound. Up to three applicators may be used on large wounds. The sponges will rapidly expand to tamponade the bleeding. Cover the wound with a compression dressing. Remove the sponges after the bleeding has stopped. Each sponge contains a radiopaque marker for easy radiograph detection.

ALTERNATIVE TECHNIQUES

The general techniques discussed above apply to bleeding from most sites. There are techniques of hemorrhage control applicable to specific anatomic sites.

THE HAND

Hand injuries pose special problems. Strict hemostasis is necessary to examine the wound and identify any associated damage to joint capsules, nerves, and tendons. Place a tourniquet to exsanguinate the extremity and facilitate wound inspection. Elevate the limb and wrap it with an elastic bandage to "milk" the venous return toward the heart. Apply a blood pressure cuff to the forearm or arm and inflate it above the systolic blood pressure. This prevents arterial inflow while minimizing the backflow from venous engorgement to reliably provide a bloodless field.

A digital tourniquet may expedite the examination if the injury is confined to a single digit.[69,70] A Penrose drain can be wrapped about the base of the finger and secured with a hemostat (**Figures 129-3 and 137-19*A***). Mark a 0.25 inch Penrose drain with two lines placed 26 mm apart. Stretch the Penrose drain about the base of an average adult finger until the lines meet. Clamp the Penrose drain with a hemostat to generate a sufficient but safe pressure.[69] An alternative

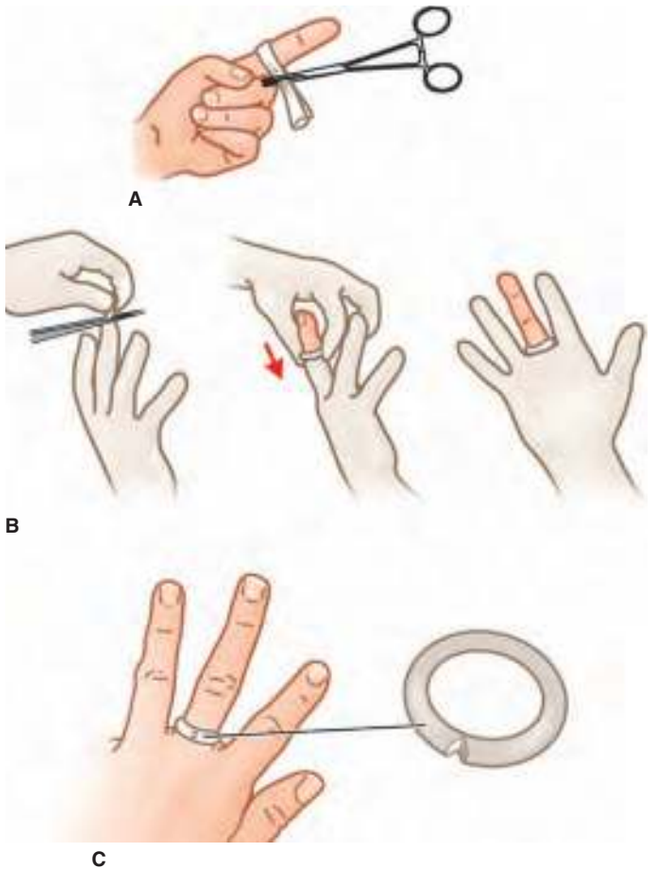

FIGURE 137-19. Finger tourniquets. ***A.*** A Penrose drain wrapped about the base of the finger provides effective hemostasis. ***B.*** A finger of a surgical glove has been cut and rolled down the finger. ***C.*** A commercial finger tourniquet.

is to use a surgical glove with the fingertip cut off and rolled down to leave a tight band at the base of the digit (**Figures 129-4 and 137-19*B***). Use a glove size larger than what would typically fit the patient for general use to avoid generating excessive pressure.[69] Disposable, preformed, rubber digital tourniquets are commercially available (**Figures 129-5, 129-6, and 137-19*C***).[71] Refer to Chapter 129 for a more complete discussion of digital tourniquets. These tourniquets exsanguinate the digit and prevent arterial inflow. **Do not apply digital tourniquets for more than 20 to 30 minutes to avoid injury to the digital nerves.**

Hemostasis is important but should not be pursued without regard to the surrounding tissues. **Do not blindly probe hand wounds deep to surface structures. Blind exploration or clamping is never advised.** Probing and clamping can damage small nerves and other structures. **Do not use vasoconstrictors (e.g., epinephrine) on the digits.** Consult a Hand Surgeon if wounds require deep exploration, a digital artery is injured, or hemostasis is difficult to achieve.

THE SCALP

Scalp wounds frequently occur in association with other major injuries. Control of scalp bleeding is frequently not the priority in the multiple trauma patient. Continued brisk bleeding from the scalp can contribute to hemorrhagic shock.[72] Techniques for vascular control of the damaged scalp should be simple, fast, and not interfere with the ongoing assessment and treatment of other injuries.

A few techniques can help gain rapid control of scalp bleeding with a minimal investment of time or personnel (**Figure 137-20**).

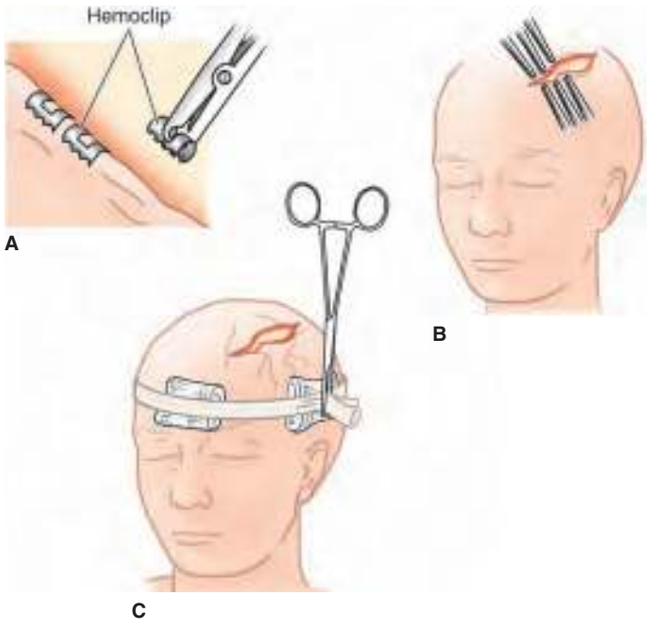

FIGURE 137-20. Hemorrhage from scalp wounds can easily be controlled. ***A.*** Hemostatic Raney scalp clips seal the wound edge. ***B.*** Hemostats applied about the edge of the wound. ***C.*** A Penrose drain wrapped about the head.

The fastest and most effective method is the application of Raney scalp clips (**Figure 137-20*A***). These have been used for years by Neurosurgeons performing craniotomies.[73] **Use scalp clips on the thick skin of the scalp. Use elsewhere can crush and devitalize thin skin or damage subcutaneous structures.** Apply hemostats at the wound edges if scalp clips are not available and where the bleeding is brisk (**Figure 137-20*B***). Inject local anesthetic solution with epinephrine into the wound edges to constrict smaller vessels. A Penrose drain can be wrapped about the head as a temporary tourniquet (**Figure 137-20*C***).[13] Figure-of-eight sutures, simple running sutures, mattress sutures, or surgical staples temporarily close a scalp wound and achieve hemostasis. A more definitive closure can be performed after the patient has been stabilized. Use one or more iTClamps to stop scalp bleeding (**Figure 137-17**).

The use of Raney clips can be cumbersome. This is especially true if the Emergency Physician has little or no experience with the system. It requires a special applier, individually loading the clips on the applier, and manipulating the clips. A much simpler system is a Raney clip gun (**Figure 137-21**). The clips are preloaded in a magazine that snaps into the clip gun. A clip is applied by touching the tip of the gun to the scalp edge and squeezing the handle. The clip gun then ejects and applies a clip, loads the next clip, and is set to apply the next clip. The process and technique of applying a clip is like using a skin stapler. The clip gun is much easier, quicker, and simpler to use than the traditional method.

MAJOR EXTREMITY INJURY

Amputations, major crush wounds, soft tissue avulsions, and fractures of the extremity may present with active bleeding. Diffuse bleeding from muscle and soft tissue may be difficult to localize and treat. Immobilize the extremity and apply direct pressure if discrete bleeding sites cannot be identified. Reduction of long bone fractures and immobilization of soft tissue injuries can stabilize the damaged tissue and minimize blood loss. The application of a MAST suit (Chapter 230) or air splint may stabilize bony fragments and tamponade active bleeding.[74] Consider the application of a topical hemostatic agent such as Hemcon ChitoGauze or Quikclot Combat Gauze. These conservative and simple measures can dramatically reduce ongoing blood loss.

FIGURE 137-21. The Medtronic Clip Gun Kit (Medtronic Neurosurgery, Goleta, CA). It contains the clip gun, three magazines preloaded with Raney-type clips, a clip remover tool, and an instruction manual.

EXPOSED BONE

Exposed bone tends to ooze. This can be especially troublesome in amputations and crush wounds. Bone wax can tamponade these sites and temporarily halt the bleeding until more definitive action can be taken. Open a sterile package of bone wax and hold it in a sterile-gloved hand to warm it up and make it more pliable. Remove a piece of the bone wax and mold it over the end of the broken bone. Firmly push the bone wax into the bone to seal the edges. **Use care to prevent lacerating the glove and finger, resulting in a potentially significant bloodborne pathogen exposure.** Possible complications associated with the use of bone wax include granulomatous reactions, infection, and interference with osteogenesis. An alternative to bone wax is Ostene (Baxter Healthcare Corp., Los Angeles, CA), a water-soluble alkylene oxide copolymer that dissolves within 24 hours.

ARTERIAL INJURIES

Puncture wounds, open fractures, amputations, and deep lacerations may be complicated by arterial injuries. These may be obvious if they present with dramatic pulsatile bleeding. The elastic recoil of arteries frequently causes the damaged vessel to retract deep within the wound and rebleed later after wound closure. Recurrent pulsatile bleeding and deep hematoma formation are characteristics of unrecognized arterial injuries. This is particularly true of puncture wounds where the damage may be deep and not visible to the examiner's eye. These wounds may require angiography, embolization, or wound exploration to identify the source if they rebleed despite local measures.

ASSESSMENT

The ideal goal in wound care is to achieve a dry bloodless field without compromising the vitality of the tissue. Simply controlling the hemorrhage and preserving life is the goal in major trauma victims. Expediting wound closure and preventing hematoma formation is a more modest goal for minor injuries.

AFTERCARE

A healthy wound is proof of adequate hemostasis. Routine wound care should verify a healthy incision line and the absence of a hematoma or an infection. Many of the novel hemostatic agents may present unique challenges in wound care after their application, as many are hard to remove from the wound bed. Refer to Chapters 114 through 119 regarding the details of wound care and repair. Update the patient's tetanus immune status as required.

COMPLICATIONS

The techniques in this chapter are all safe and effective when used as described. Complications occur when the described techniques are used in excess or in the wrong setting. The specific complications of each technique are discussed under each specific section in this chapter.

FUTURE CONSIDERATIONS

The area of hemostasis is very active in terms of research and development. Injectable foam can be used in the abdomen.[75-77] It is injected into the abdomen and expands to fill all the areas and tamponade the bleeding. It may be developed to work on extremity wounds. Polystat is a synthetic polymer that is injected into a vein. It seeks out clots and binds to them, making them stronger and longer lasting. Remedium Technologies is developing a foam to clump blood cells and reduce external hemorrhage. Gel-e is a biopolymer that is biocompatible and form fits to external hemorrhage. It creates rapid coagulation, is durable, and has antibacterial properties. AC5 is a synthetic peptide made of amino acids that is sprayed onto a wound and intercalates with connective tissue to form a lattice-like gel. Hemospray is a powder spray being tested for lower and upper gastrointestinal bleeding.[78] Hydrogels are being developed.[79] These agents are not ready as yet for use in the Emergency Department but have promise.

SUMMARY

There are numerous techniques available for the control of hemorrhage. A methodical approach to the bleeding wound will optimize the outcome. Use simple measures first and take progressive systematic steps until hemostasis is achieved. All bleeding eventually stops! The goal is to halt the bleeding before irreparable harm occurs.

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138 Trigger Point Injections

Danielle Campagne

INTRODUCTION

Musculoskeletal pain is a significant health problem for the North American population.[1-9] Such pain affects between 10% and 20% of the population and is a major cause of morbidity.[1] It is estimated that approximately half of chronic pain complaints result from a musculoskeletal origin.[2] Myofascial trigger point (MTrP) injections may alleviate much of this pain.[10-13] It is imperative that the Emergency Physician perform a thorough history and physical examination with an emphasis on the neurologic and orthopedic examination to exclude other causes of musculoskeletal pain.[3] The performance of MTrP injections by Emergency Physicians is underused.[14,15]

ANATOMY AND PATHOPHYSIOLOGY

The etiology and pathogenesis of MTrPs have yet to be elucidated. The precise mechanism by which MTrP injections inactivate the trigger point is unknown. Researchers agree that acute trauma or repetitive microtrauma appears to lead to the development of a MTrP.[6] The risk for a MTrP is increased when other factors are present (e.g., poor physical conditioning, poor posture, and prolonged bending).[7] MTrPs mostly affect the muscle groups used to maintain posture (i.e., muscles of the neck, shoulders, and back). The patient may present with a tension headache or temporomandibular joint pain when the head and neck regions are affected.[6]

MTrPs are hyperirritable points located within a taut band of skeletal muscle or fascia.[2] When compressed, these points may

cause autonomic pain and local tenderness.[2] Pain may be diffuse if not localized to the MTrP. The pain can be described as burning, dull, sharp, or some combination of these. Autonomic changes associated with a MTrP include dizziness, edema at the site, lacrimation, piloerection, salivation, and tinnitus. The compression of a MTrP can further lead to muscle spasm, stiffness, shortening, and fatigue.[1] This may progress to impaired muscle coordination, reduced muscle strength, and decreased range of motion.[1]

DIAGNOSIS OF MTrPs

The diagnosis of a MTrP relies on the criteria of a tender spot with an underlying taught band, pain on palpation of the tender spot, and a local twitch response (i.e., a transient local contraction of skeletal muscle fibers in response to palpation or needling).[3,10] The data on clinical outcomes provide no definitive answer. The best outcomes appear to occur in patients who exhibit a local twitch response with palpation.[4] The current literature provides no pathophysiologic explanation for this result. There are no laboratory, pathology, or radiology studies to identify or verify a MTrP.

Identifying the palpable, taut band is critical in locating the MTrP (Figure 138-1). The MTrP can be identified by flat palpation, snapping palpation, pincer palpation, and/or deep palpation. Flat palpation uses a fingertip to slide across the skin over the affected muscle to find the MTrP (**Figure 138-2**). The taut band may be felt under the sliding fingertip. Snapping palpation uses the tip of the index finger to pluck the skin to feel the underlying taut band. This motion is similar to plucking a guitar string. Pincer palpation uses the dominant thumb and index finger to firmly grasp the skin and muscle as a unit and roll it to identify the taut band (**Figure 138-3**). Deep palpation can be used to identify a deep MTrP or a superficial MTrP in an obese patient. Use the tip of the index finger to press slowly and deeply to reproduce the patient's symptoms and identify the MTrP.

Studies have used a variety of injectant fluids and solutions during injection of MTrPs (e.g., botulinum toxin, corticosteroid suspensions, ketorolac, local anesthetic solutions, sterile saline, and sterile water).[1,16-18] **No specific fluid or solution has demonstrated a clearly superior clinical outcome.** The optimal injectant fluid or solution varies by Physician preference. The duration of pain relief has been found to last longer than the duration of action of the injectant. A meta-analysis of eight randomized controlled clinical trials examined the type of injectant used and the resulting effect on symptom relief.[1] The authors concluded that the injection of either lidocaine or botulinum toxin provided greater symptom relief than placebo (i.e., dry needling alone). A Cochrane analysis looked at four studies totaling 233 patients that evaluated botulinum toxin A as the injectant versus placebo.[17] It concluded there was inconclusive evidence to support the use of botulinum toxin A in MTrP. **No injectant was more efficacious than any other injectant.** Another study looked at pain relief with an injection of lidocaine versus

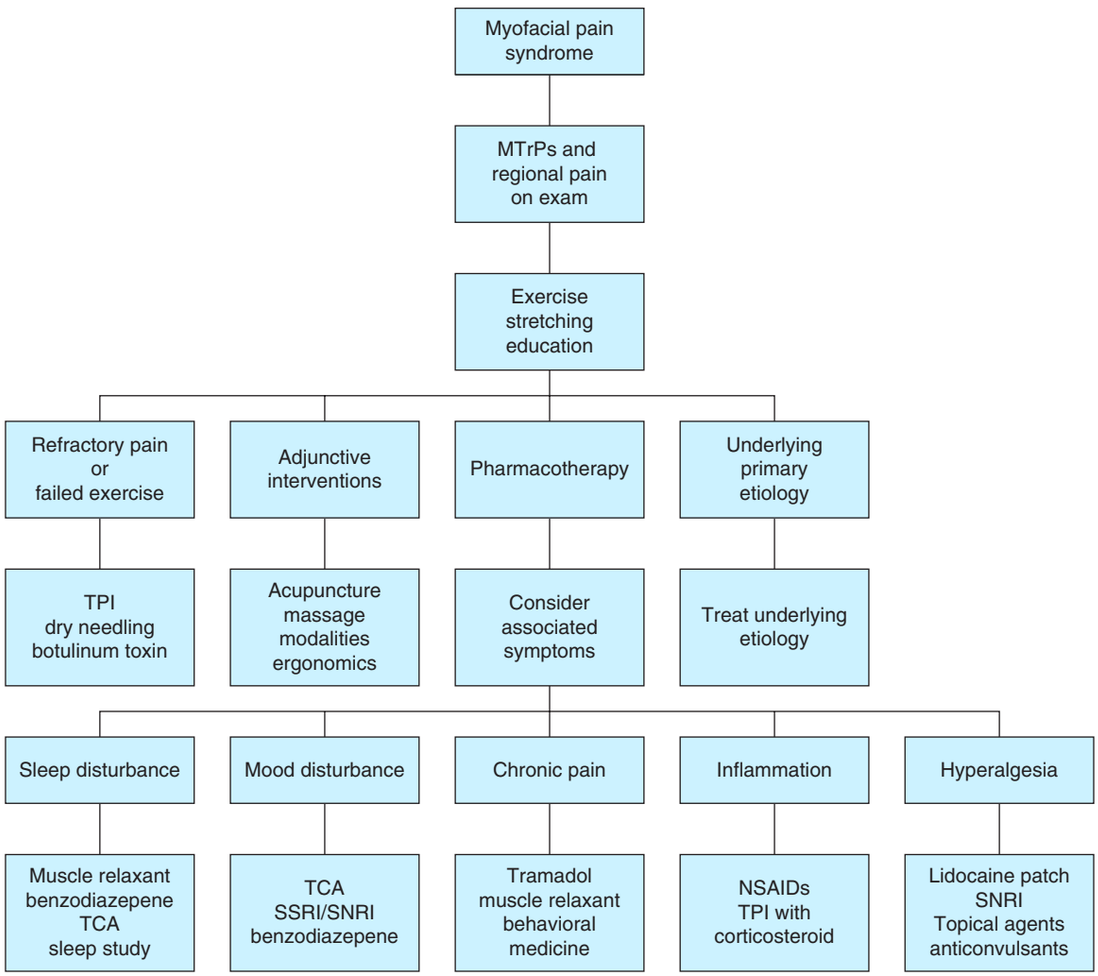

FIGURE 138-1. MTrP treatments. NSAID, nonsteroidal anti-inflammatory drug; SNRI, serotonin-norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor; TCA, tricyclic antidepressant; TPI, trigger point injection. (Used with permission from reference 10.)

FIGURE 138-2. Flat palpation to identify a MTrP or taut band. ***A.*** The index finger pushes down over the MTrP or taut band. ***B.*** The index finger rolls off the tender spot and pushes the skin to one side. ***C.*** The index finger pushes the skin back to the tender spot to feel the taut band. ***D.*** The index finger rolls off the tender spot and pushes the skin to the opposite side.

physical therapy versus both modalities (i.e., MTrP lidocaine injection and physical therapy) combined.[18] The study showed no difference between the three groups. Pain was relieved in all groups. This finding is consistent with the general medical literature on the topic which is still in its infancy and provides no clear indication of effectiveness. More research is required in this area.

NONINVASIVE MTrP MANAGEMENT

Numerous noninvasive techniques have been used to treat MTrPs (**Figure 138-1**).[2,5,8,10,11,19-21] One of the more common techniques uses spray vapocoolant (e.g., ethyl chloride) in combination with passive muscle stretch to relax the taut band. Ischemic compression therapy is the application of pressure to the MTrP to produce ischemia and ablate the MTrP. Digital pressure is applied and increased until the taut band relaxes. A deep pressure or stroking massage can be used to stretch the affected muscle and relax the taut band. Physical therapy can stretch and relax the affected muscles. Transcutaneous electrical stimulation units with the electrodes placed over the MTrP can be used to stimulate and relax the underlying muscle. Ultrasound can transmit heat and vibration to a superficial MTrP with the goal of muscle relaxation.

Some apply massage followed by an injection. The application of pressure to the MTrP is considered critical in the treatment. Warn the patient what to expect when you apply pressure to the MTrP. Apply pressure over the point of maximal tenderness with both thumbs. An elbow or forearm can apply pressure to larger areas. Hold the pressure until the underlying MTrP relaxes and goes away. It usually takes 2 to 4 minutes of pressure to relieve the pain. Inject the MTrP and reapply pressure.

INDICATIONS

MTrP injections have been advocated for points of muscle pain that are not assisted by noninvasive therapy (e.g., ischemic compression therapy, massage, physical therapy, spray vapocoolant, transcutaneous electrical stimulation, and ultrasound) (**Figure 138-1**).[8,10] There is no emergent indication for a MTrP injection in the Emergency Department.

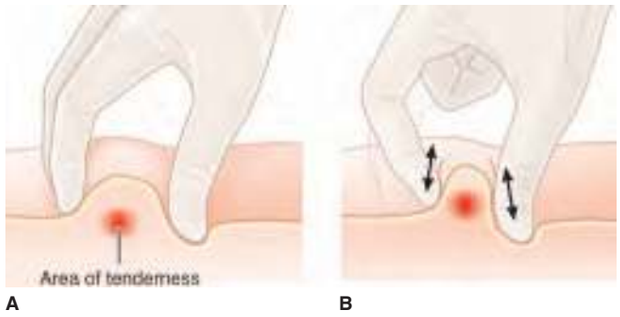

FIGURE 138-3. Pincer palpation to identify a MTrP or taut band. ***A.*** The skin, subcutaneous tissue, and muscle are grasped between the thumb and index finger. ***B.*** The fingers are moved back and forth (*arrows*) to feel the taut band as it is rolled between the fingertips.

CONTRAINDICATIONS

Contraindications to MTrP injections are similar to other injection procedures (e.g., anticoagulation therapy, a bleeding disorder, a dermatologic condition, a local or systemic infection, or malignancy over the injection site).[5] The traditional teaching of anticoagulation therapy being a hard contraindication to any invasive pain procedure (including a MTrP) is being challenged in the literature. A study looked at invasive pain procedures in 4766 patients and found no complications associated with it. Patients who stopped anticoagulation to receive the procedure had more serious complications (e.g., stroke and death).[22] Relative contraindications include allergies to local anesthetic agents, patients with needle phobias, and uncooperative patients.

It is important to differentiate between a patient with one or more isolated MTrPs and fibromyalgia (**Table 138-1**). **A patient with fibromyalgia can have multiple MTrPs. Do not perform MTrP injections in a patient with fibromyalgia.** The injection may worsen their pain.

EQUIPMENT

- Sterile gloves
- Sterile skin marking pen
- Povidone iodine or chlorhexidine solution
- Alcohol swabs
- Sterile gloves
- Gauze 4×4 squares
- 25 or 27 gauge, 1.5 inch needle for superficial trigger points
- 25 or 27 gauge, 2 inch needle for deeper trigger points
- 3 or 5 mL syringe
- Injection solutions

TABLE 138-1 Determining Isolated MTrPs Versus MTrPs Associated with Fibromyalgia

Characteristic	Isolated MTrPs	Fibromyalgia
Immediate response to injection therapy	Resolution of symptoms	Poor or none
MTrPs	Few, discrete, and localized	Many and widespread
Muscle tissue	Taut bands palpable	Soft, no taut bands palpable
Muscle range of motion	Stiff and decreased	Normal
Pain and tenderness	Local or regional	Generalized and widespread
Sexual predilection	None	Female

Source: Modified from reference 2.

- Lidocaine without epinephrine
- Bupivacaine without epinephrine
- Sterile water
- Sterile normal saline
- Botulinum toxin A, 20 units or 0.4 mL (50 U/mL) (Botox; Allergan, Irvine, CA) diluted to 1 mL with normal saline

- Ultrasound machine with a 5 to 7.5 MHz probe
- Ultrasound gel

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Place the patient in a comfortable position on a gurney with the MTrP(s) exposed. The muscle with the MTrP(s) is ideally positioned so that it is relaxed. Identify the MTrP site(s). Clean the skin of any dirt and debris. Use ultrasound to ensure there are no neurologic, tendinous, or vascular structures in the area that can be injured by the MTrP injection. Mark the MTrP skin site with a pen. Apply povidone iodine or chlorhexidine solution over the injection site(s) and surrounding skin and allow it to dry. Follow aseptic technique for the injection procedure.

Prepare multiple 3 or 5 mL syringes armed with a 25 or 27 gauge needle and containing the injection solution. The editor recommends local anesthetic solution as the injectant in the Emergency Department. The number of syringes to prepare depends upon the number of MTrP sites to be injected and the volume injected at each site.

TECHNIQUES

INJECTION TECHNIQUE

Reidentify the MTrP site. Warn the patient what to expect (i.e., exacerbation of their symptoms which is usually pain) upon the injection. Use the nondominant index and middle fingers to locate and isolate the MTrP or taught band (**Figures 138-4*A* and 138-4*B***). Insert the needle into the skin approximately 1 cm away from the MTrP or taught band, at a 30° angle to the skin, and aimed at the MTrP or taut band. Advance the needle into the MTrP or taut band. **Insert the needle briskly and in a controlled manner.** Use a "fast in, fast out" approach to elicit a local twitch response when the tip of the needle hits the MTrP or taut band.[8] **Aspirate to ensure that the tip of the needle is not within a blood vessel.** Inject the solution within the syringe.

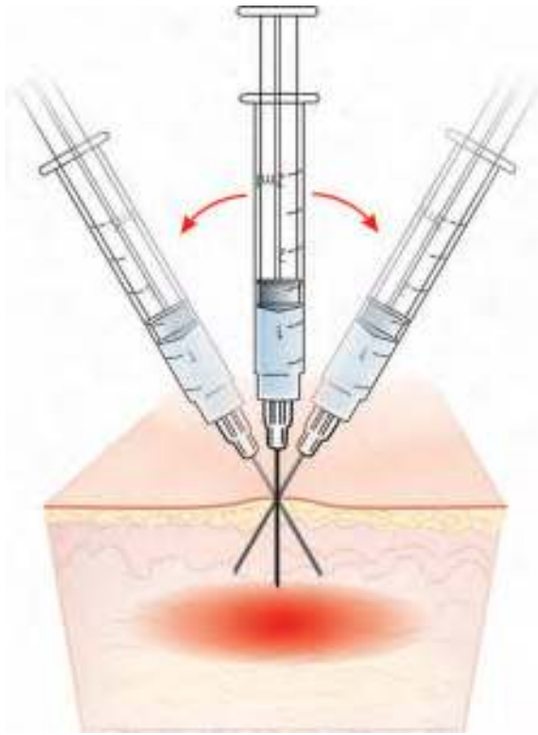

FIGURE 138-5. Injection of the MTrP or taut band. The needle is inserted through a single skin puncture and into multiple places within the MTrP or taut band in a fanlike pattern.

Common practice is to inject between 0.5 and 2.0 mL total per MTrP.[5] This volume can be injected into the one location of maximal tenderness or numerous sites in a fanlike pattern within the MTrP (**Figure 138-5**).[9] Withdraw the needle to the skin surface once the injection is completed but do not completely remove it from the skin. Allow the injection solution to work for up to a minute. Reinsert the needle into the MTrP in a fanlike pattern until the local twitch response is no longer elicited or resisting muscle tautness is no longer palpable.[7] Completely withdraw the needle. Apply pressure over the injection site to prevent bleeding and hematoma formation. Instruct the patient to slowly and fully stretch the affected muscle group.[8]

DRY NEEDLING

The technique of dry needling involves inserting a needle into multiple sites within the MTrP or taut band without injecting any fluid or solution.[23-27] This is similar to acupuncture. Insert the needle as described above. Withdraw the needle until the tip is just below the

FIGURE 138-4. Identifying and injecting the MTrP or taut band. ***A.*** Push with the index finger and middle finger, alternating between the fingers, to identify and isolate the MTrP or taut band. ***B.*** Compress the skin with both fingers to secure the MTrP or taut band and inject it.

skin surface. Redirect and reinsert the needle in a fanlike pattern into a different location within the MTrP or taut band. Continue this process several times until the local twitch response is no longer elicited or resisting muscle tautness is no longer palpable.[7] Completely withdraw the needle. Apply pressure over the injection site to prevent bleeding and hematoma formation. Instruct the patient to slowly and fully stretch the affected muscle group.[8]

ASSESSMENT

The patient should experience relief of their symptoms after the injection, especially if a local anesthetic was injected. The injection of a MTrP breaks the pain cycle. Passively and actively stretch the affected muscle in a slow manner to stretch it out. Apply digital compression to the injection site, the MTrP, and the taut band. Repeat the procedure with local anesthetic solution or by dry needling if symptoms are still present. Observe the injection site and apply pressure to control any bleeding.

AFTERCARE

Apply a simple adhesive bandage over the injection site. Postinjection soreness is expected and can be managed with acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs). Warn the patient that the pain often comes back but is less intense. Encourage the patient to use the affected muscle through its full range of motion but avoid strenuous activity for 1 to 3 days after the injection. Instruct the patient to return to the Emergency Department immediately if they develop fever, chills, swelling at the injection site, redness at the injection site, or any drainage from the injection site.

COMPLICATIONS

The complications are the same as any injection procedure (e.g., infections and needle breakage). An allergic reaction to the injection fluid or solution is possible. Treat this as any other allergic reaction. A thorough history may prevent an allergic reaction to the injectant. Never aim the needle at an intercostal space to prevent an iatrogenic pneumothorax.[7] Hematoma formation following injection can be minimized with proper technique and the application of pressure over the soft tissue after the needle is withdrawn.[2] Never inject the patient when they are standing or sitting in a chair. Ensure that the patient is always on a gurney and the side rails are upright to prevent injury if the patient becomes vasovagal or experiences syncope. Injury and inadvertent injection to adjacent structures can be avoided by knowing the local anatomy and using ultrasonography before the injection to identify adjacent structures.

SUMMARY

MTrP injections and nonpharmacologic management can be performed in the Emergency Department (**Figure 138-1**). They are simple, quick, and effective to manage a patient's pain. The efficacy of MTrP injections have not yet been established. MTrP injections serve as part of a treatment plan to offer quick relief of myofascial pain when other noninvasive therapies have been unsuccessful or when opioids are contraindicated. The current opioid epidemic that is occurring worldwide may result in more Emergency Physicians performing injection of MTrPs in patients.

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139 Escharotomy

Michael A. Schindlbeck and Carlos E. Brown, Jr.

INTRODUCTION

Few injuries have the same capacity for physical destruction and emotional devastation as do thermal burns. They are relatively common presentations that often require resource-intensive management. The preceding decade saw nearly 500,000 burn injuries per year receiving medical care in the United States.[1-3] Over 40,000 of these patients required inpatient treatment for their injuries and up to 73% of these injuries occur at home. The associated expenses are staggering. The mean hospital stay was over 1 week and at an average cost over $50,000 per admission. An average of over 4500 individuals per year died from burn-related injuries. Fires and burns represent the fifth, third, sixth, and eighth leading causes of unintentional injury deaths in the United States for the age groups of 1 to 4 years, 5 to 9 years, 10 to 14 years, and ≥ 65 years, respectively.[1] Less than 6% of the above patients who were admitted to a recognized burn center subsequently died from their injuries.[2-4] **These data underscore the need for rapid and effective emergency care focused on facilitating the successful transfer of these patients to specialized burn centers.** The initial management of these patients invariably falls upon the Emergency Department. The Emergency Physician needs to be well versed in the recognition of acute thermal injuries, their associated complications, and their appropriate treatment.

Thermal injuries have the potential to affect any body surface, both internally and externally, with which a heated medium comes into contact. The overall depth and degree of injury is multifactorial. It is typically proportional to the temperature of the source medium, its unique specific heat, the actual rate of energy transfer, and the overall duration of tissue exposure. This chapter focuses on the skin and its response to burn injuries.

ANATOMY AND PATHOPHYSIOLOGY

Skin exposure to any significant heat source results in a spectrum of pathophysiologic responses. An initial coagulation necrosis occurs as thermal energy is transmitted directly into living tissue. These cells subsequently die and lyse, spilling their intracellular contents and increasing the surrounding interstitial oncotic pressure. These processes serve to trigger a secondary edematous reaction in the surrounding tissues. Cellular breakdown releases a host of generalized inflammatory markers (e.g., histamines, prostaglandins, cytokines, and interleukins). These agents further exacerbate the localized edematous reaction via vasodilation and increased capillary permeability.[5]

Burns involving over 20% of a patient's total body surface area (TBSA) can result in secondary injury extending beyond the locally involved tissues. The inflammatory outburst can become significantly large enough to produce a systemic pathophysiologic response of internal fluid shifts and external fluid losses. Inadequate fluid resuscitation can result in tissue hypoperfusion and multisystem organ dysfunction. A secondary systemic inflammatory response syndrome (SIRS) can further complicate the clinical course of patients whose burns involve greater than 30% TBSA. This frequently results in widespread intravascular hemolysis, acute renal failure, and acute lung injury.[6,7] A global hypermetabolic state tends to accompany these injures. Secondary catecholamine release can raise the resting metabolic rate approximately two to three times above baseline when greater than 30% to 40% of a patient's TBSA is involved. The consequent catabolic tissue breakdown further exacerbates the emerging multisystem organ dysfunction.[5,8] These burns are associated with significant morbidity and mortality. Survivability rates are indirectly proportional to the TBSA involved. Of special note is the loss of a protective functioning epidermis as severe and overwhelming infection becomes a significant potential complication in those who survive beyond the initial resuscitative period.[9] Systemic organ failure from the "burn sepsis" continues to be a leading cause of death in these individuals.

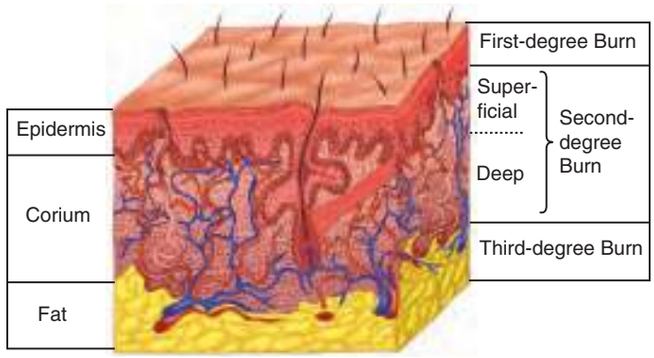

FIGURE 139-1. The depth of burn wounds. (Used with permission from Schafermeyer R, et al: *Strange and Schafermeyer's Pediatric Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2015.)

Burns are classified based on the overall depth of injury sustained. First-degree burns are limited solely to the epidermal layer (Figure 139-1). They typically present as a tender erythematous region of skin. This redness arises as the underlying dermal capillaries dilate in response to the overlying injury. Deeper tissues are not directly involved and the capillary walls remain intact. This explains the blanchable nature of these burns on physical examination. Blistering of the skin indicates involvement of the underlying dermis and is not seen with first-degree burns. Classic examples of first-degree burns include flash burns and sunburns. These burns do not generally illicit the systemic pathophysiologic response described above despite extensive surface area involvement. **Do not include first-degree burns when calculating the overall TBSA affected.**

Second-degree burns are the partial thickness injuries. These burns extend into but not through the dermis (Figure 139-1). They are subdivided into superficial second-degree and deep second-degree burns. Superficial second-degree burns are typically moist to the touch, pink to reddish in color, blanchable with gentle pressure, and covered with tensely distended blisters. Cutaneous nerves, located in the uninvolved underlying deeper dermal layer, remain viable, bestowing an exquisitely painful character to these burns. Deep second-degree burns involve the deeper reticular dermis and differ noticeably in appearance. They tend to be drier in texture and have a more blanched whitish complexion. These injuries tend to be less painful than their superficial counterparts due to destruction of the cutaneous nerves.

Full-thickness burns involve the destruction of the entire epidermal and dermal tissues and are classified as third-degree burns (Figure 139-1). Coagulation necrosis imparts a dry leathery appearance to these burns. The overlying tissue is insensate secondary to the obliteration of the cutaneous nerves. The dermal proteins in third-degree burns are heated and tend to contract, facilitating the formation of a constrictive eschar of scar tissue. Rare scenarios where the tissue destruction extends though the dermis and into the underlying tissues (e.g., muscle, bone, and fascia) are categorized as fourth-degree burns. These injuries are catastrophic in appearance and tend to result in extensive tissue loss.[10]

Burn injuries can generate an extensive redistribution of fluids into the interstitial spaces. Aggressive fluid resuscitation will help

to limit the consequent invariable hypoperfusion but does nothing to limit further leakage of fluids into the interstitium. This third spacing can lead to significant elevations in the localized hydrostatic pressures in tissues already constricted by an extensive overlying eschar. Impairment in regional lymphatic and venous drainage further exacerbates this adulteration of the local pressure gradient. The normal arteriolar perfusion pressure will be eventually overwhelmed and result in distal tissue hypoperfusion. Distal tissue necrosis can progress to limb loss if the burn occurs on an extremity. Swelling and scarring can result in the loss of a functional airway if the burn occurs over the neck. Burns occurring across the torso can produce an abdominal compartment syndrome, progressing to intraabdominal organ ischemia, impaired diaphragmatic excursion, and reduced cardiac output secondary to diminished venous return.[11-13] Significant thoracic burns can impede the dynamic chest wall motions of respiration producing a further restrictive ventilation that can progress to respiratory failure.[14]

INDICATIONS

The general indication to perform an escharotomy is to limit the circulatory or respiratory insult caused by an overlying circumferential burn. These indications can be present with a significant noncircumferential burn. The development of a significant restrictive physiology often requires several hours after the initial burn.[15] Most patients could be successfully transported to a specialized burn center within this time span. Invasive means to assess compartmental pressures (Chapter 93) should not be undertaken at the expense of proper fluid resuscitation and preparation for transport. The Emergency Physician must maintain a high index of suspicion to limit any further injury to viable tissues. **An escharotomy should be performed without delay once the decision is made to perform an escharotomy.** Perform the escharotomy in consultation with the accepting burn center and/or Burn Surgeon if possible and time permits.

Any signs and symptoms of limb hypoperfusion should be taken seriously. **A restrictive etiology should be considered only if hypoperfusion persists despite adequate volume resuscitation as hypovolemia is the most likely etiology of impaired tissue perfusion immediately after the burn injury.** Perform and document frequent and repeat physical examinations including an assessment of overall skin appearance, distal capillary refill, peripheral pulse checks, any motor deficits, and any sensory deficits. **Depending on the presence of palpable pulses as the sole means to approximate compartmental pressures will grossly underestimate the need for decompression.**[16]

Several methods are available to aid the Emergency Physician in frequently assessing for tissue hypoperfusion. Bedside arterial Doppler provides an easy means to gauge peripheral perfusion. It is concerning if any signs of a progressive reduction in arterial flow occur. The absence of arterial flow on a bedside Doppler is an indication for emergent escharotomy. **Keep in mind that the presence of Doppler pulses does not necessarily indicate adequate perfusion.** Pulse oximetry provides another useful adjunct. A distal oxygen saturation of less than 95% in a circumferentially burned extremity has been shown to be an indicator for an emergent escharotomy.[17] Compartmental pressures can usually be measured quickly at the bedside (Chapter 93). An intracompartmental pressure of ≥ 40 mmHg is an indication for an escharotomy. Consider an escharotomy for intracompartmental pressures between 25 and 40 mmHg.

Significant burns to both the chest and abdomen can result in restricting ventilation. Respiratory distress is generally multifactorial in burn patients. A concurrent inhalation injury or secondary acute respiratory distress syndrome should be entertained in the differential diagnosis. **Early intubation and mechanical ventilation is essential in any burn patient exhibiting respiratory distress.** Mechanically ventilated patients with severe truncal burns, persistent arterial hypercapnia, and elevated peak inspiratory pressures (although often confounded by concurrent airway edema and secondary bronchospasm) are objective signs suggesting a significant restrictive respiratory physiology. An emergent escharotomy could be a lifesaving procedure.

Facial burns can result in eschar formation around the eyes and mouth. Measure intraocular pressure if an eschar surrounds the eye. Elevated intraocular pressure (Chapter 188) should be decompressed with a lateral canthotomy (Chapter 194).

CONTRAINDICATIONS

No specific contraindications exist for performing an escharotomy provided the above indications are satisfied. The concern for possible medical futility in patients with no chance of salvageability is very difficult to determine in the Emergency Department.

EQUIPMENT

- Sterile gloves and gown
- Face mask with an eye shield or eye protection
- Hat
- Povidone iodine or chlorhexidine solution
- #10 scalpel blade on a handle
- Electrocautery unit, optional but highly recommended
- Gauze 4×4 squares
- Local anesthetic solution
- Needles and syringes
- Suture to tie bleeding vessels
- Procedural sedation medications and supplies (Chapter 159)

PATIENT PREPARATION

This procedure is considered life and/or limb sparing. Inform the patient and/or their representative for the need to perform an escharotomy, its risks and benefits, and the outcome if not performed. Document the discussion in the medical record. The patient's medical condition often precludes them from signing a consent form. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Escharotomies are performed on tissues previously destroyed by full-thickness burns. Some intermittent nerve function can persist. This may necessitate the use of local anesthesia and/or procedural sedation (Chapter 159). Administer some form of procedural sedation (Chapter 159) in the conscious patient for pain control and to limit the profound anxiety elicited by this procedure. **Strict aseptic technique should be followed.** Clean the skin of any dirt or debris. Apply dilute povidone iodine or chlorhexidine solution to the skin and allow it to dry. **This procedure has the potential to introduce a devastating infection.**

TECHNIQUES

Make the skin incisions along the proper plane with a #10 scalpel blade or an electrocautery unit. Electrocautery has the added ability to coagulate the potential bleeding encountered from inadvertently incised superficial subcutaneous vessels (**Figure 139-2*A***). **Limit the**

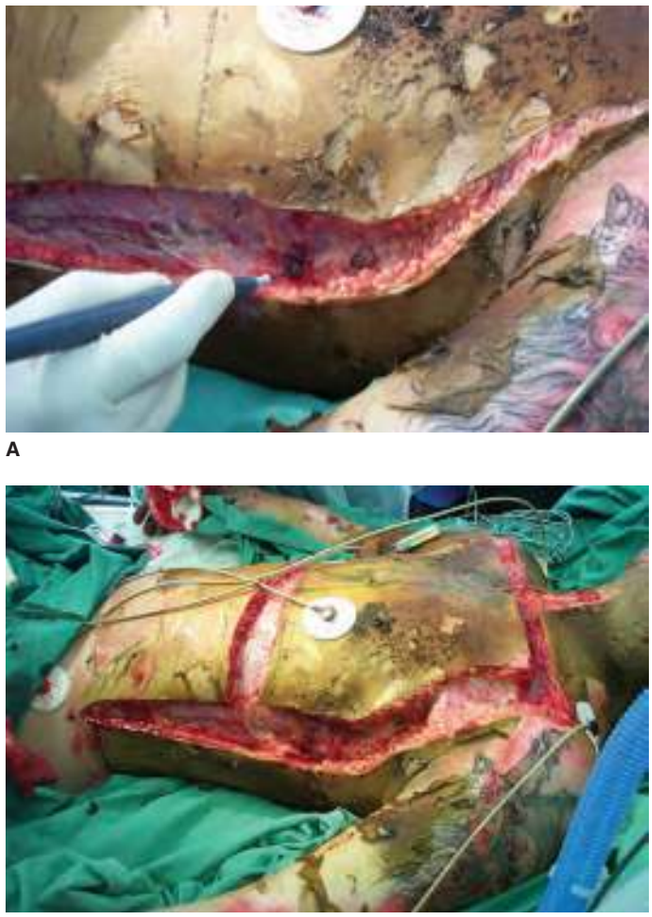

FIGURE 139-2. The escharotomy in a burn patient. *A.* Using electrocautery. *B.* An escharotomy has been made in the abdomen, chest, and neck. (Photos used with permission of www.lifeinthefastlane.com.)

depth of the incision to the dermis. Use extreme caution to avoid overaggressively extending these incisions too deeply and injuring the underlying deep investing fascia, muscles, and/or tendons. There is a pressure buildup underlying the constricting tissue. **A properly placed incision should elicit a rapid separation of the eschar exposing the underlying subcutaneous fat.** Carefully run a gloved fingertip along the incision lines to detect any residual connecting bands of tissue requiring further incision. To ensure an adequate release, continue the incision across the entire eschar and extending 1 to 2 cm into the unscarred tissue on either end. Cosmetic concerns are not a concern as the incised tissue will often require eventual skin grafting if the patient survives.

FACE ESCHAROTOMY

The face is commonly involved in burn injuries. Eschars can form on the face just as they can form on other areas of the body. A patient complaint of any visual disturbance or burns around the eye requires a thorough investigation. This includes measuring visual acuity, a topical fluorescein examination, a funduscopic examination, and measurement of intraocular pressure (Chapters 185 and 188). Elevated intraocular pressure may require a decompressive lateral canthotomy and cantholysis (Chapter 194).

Perioral burns can form eschars. These eschars may limit or prevent mouth opening. An escharotomy may be required to aid in oropharyngeal suctioning and orotracheal intubation. Make the

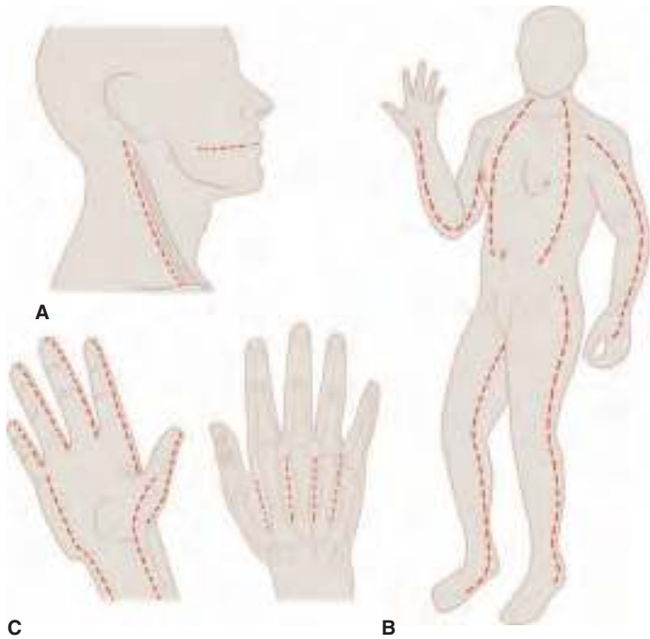

FIGURE 139-3. The location of incisions for escharotomies. *A.* The head and neck. *B.* The extremities and torso. *C.* The hand and fingers.

escharotomy incisions at the bilateral corners of the mouth and extending directly posterior (in the supine patient) and approximately 4 to 5 cm long (**Figure 139-3*A***). **Make the incisions very superficial to prevent injury to the underlying facial artery, cranial nerves, superficial facial structures, and parotid gland.**

NECK ESCHAROTOMY

Significant burns to the neck can result in the formation of a constrictive eschar that can compromise the airway. Make paired vertical incisions on the posterolateral surfaces of the neck from the mastoid process to the clavicle (**Figure 139-3*A***). **Meticulous care and attention should be given to always remain posterior to the clavicular border of the sternocleidomastoid muscle.** This will avoid injury to the internal jugular vein, carotid artery, thyroid gland, trachea, and vagus nerve.

TORSO ESCHAROTOMY

Perform a thoracic escharotomy by extending bilateral vertical incisions from the lateral clavicle to the costal margins along the anterior axillary lines (**Figures 139-2*B* and 139-3*B***). Use caution in female patients to avoid incising directly through the breast tissue. Instruct an assistant to grasp the breast and move it medially to avoid the escharotomy incision line. Extend the thoracic incision inferiorly beyond the costal margins and across the entire eschar to ensure adequate decompression in patients whose burns involve a significant portion of the abdominal wall (**Figures 139-2*B* and 139-3*B***). Make a horizontal bridging incision along the inferior costal margin in patients whose burns extend from the thorax and onto the abdomen to support proper respiratory mechanics (**Figure 139-2*B***).

Circumferential burns of the penis require an escharotomy to decompress the area and prevent ischemia. Distal ischemia can rapidly progress and produce significant tissue loss. Make bilateral incisions along the lateral margins of the body of the penis. **Meticulous care and attention should be given to avoid injuring the dorsally located neurovascular structures that lie between the 10 o'clock and the 2 o'clock position.**

UPPER EXTREMITY ESCHAROTOMY

Perform an upper extremity escharotomy on the medial and lateral aspects of the involved limb, extended along the line dividing the flexor and extensor surfaces (**Figure 139-3*B***). Place the upper extremity supine to ensure adequate landmark identification. The areas surrounding joints are sites of severe potential restriction due to relatively tight tissue adherence. Use caution when extending the incisions across these locations. **Meticulous care and attention should be given to avoid the ulnar nerve along the medial surface of the elbow as it courses posterior to the median epicondyle and the superficial branch of the radial nerve along the lateral surface of the wrist as it courses superficially above the radius.**

Extend the forearm incisions to include the thenar and hypothenar eminences if the hand is involved (**Figure 139-3*C***). Further hand decompression can be accomplished by making vertical incisions down the four intermetacarpal grooves on the dorsum of the hand (**Figures 139-3*C* and 139-4**). The fingers can be decompressed with linear incisions along the middle of the radial and ulnar surfaces, between the flexor and extensor surfaces (**Figures 139-3*C* and 139-4**). **Limit the potential damage to a patient's grasping surfaces.** Incise the ulnar finger surfaces or the radial surface on the thumb. Assess whether this unilateral incision is adequate to restore perfusion prior to automatically incising the corresponding opposing surface.[18]

LOWER EXTREMITY ESCHAROTOMY

The lower extremity should be approached with the incisions extending along the groove between the flexor and extensor surfaces (**Figure 139-3*B***). **Meticulous care and attention should be given to avoid damaging the common peroneal nerve along the lateral knee as it courses superficial to the fibular head and the posterior tibial artery along the medial ankle as it courses posterior to the medial malleolus.** Extend the incisions along the medial and lateral borders of the foot to the great toe and the fifth toe, respectively, if the foot is involved (**Figure 139-3*B***). Decompress the toes in a manner like the fingers.

TECHNIQUE FOR PEDIATRIC PATIENTS

Pediatric patients have a higher surface area–to–volume ratio than do adults. Burns pose a greater risk for significant external fluid losses and the rate of fluid resuscitation needs to be adjusted. More aggressive fluid resuscitation increases the likelihood for pathologically significant elevations in the perfusion pressures of affected tissues. **Infants and younger children depend on diaphragmatic excursion and abdominal wall mobility for normal respiratory function.** They are more susceptible to significant respiratory compromise from extensive truncal burns. This necessitates that the treating Emergency Physician possesses a high index of suspicion and the technical ability to intervene surgically. The patient preparation and escharotomy techniques are the same as in an adult.

FIGURE 139-4. Escharotomies of the hand and fingers. (Used with permission from reference 15.)

ASSESSMENT

The response to an escharotomy should be almost immediate with the signs of improving distal perfusion of the extremities or improved ventilation. The distal extremity should demonstrate decreased pallor and return of a natural skin color, return of sensation, appropriate Doppler arterial flow, and appropriate pulse oximetry. Distal pulse oximetry readings should rapidly climb into a normal range provided adequate respiratory mechanics.[19] **Lack of improvement in distal perfusion should alert the Emergency Physician to either an inadequate surgical decompression or an insufficient fluid resuscitation.** No improvement in perfusion despite appropriately addressing these two concerns requires consideration of an underlying intrafascial compartment syndrome that would require an emergent fasciotomy (Chapters 93 and 94).

AFTERCARE

Continued routine burn care and fluid resuscitation as necessary. Manage any continued bleeding from the escharotomy incisions with the application of pressure or electrocautery. Cover the escharotomy incisions with sterile saline-soaked gauze and an outer dressing. Frequently reassess the patient to rule out the development of additional tissue ischemia or respiratory compromise that would indicate the need for further extension of the initial escharotomy incisions. Transfer the patient to a specialized burn center or an intensive care unit to continuously monitor and manage the patient.

COMPLICATIONS

Bleeding can be a significant complication. This is rare when the escharotomy incisions are properly limited to the dermis. Bleeding can be extensive when an underlying subcutaneous vessel is incised. This is often complicated by the consumptive coagulopathy that frequently accompanies these burns. Electrocautery is an attractive option to limit such bleeding. Direct pressure should otherwise generally suffice. Some blood vessels may require ligature for definitive hemostasis.

The burn-damaged epidermis can no longer function to protect deeper tissues from the external environment. The overlying eschar of necrotic tissue provides an ideal environment for uncontrolled bacterial colonization. Escharotomy incisions provide a direct route for bacterial penetration into susceptible subcutaneous tissues if sterile precautions are not properly maintained. This can result in catastrophic infection and burn sepsis.

Inadvertent injury of deeper structures (e.g., blood vessels, nerves, and tendons) can occur. This is preventable by controlling the depth of the incisions and not extending into and through the subcutaneous tissues. This is especially true when incising over high-risk areas.

Making an escharotomy incision on the fingers is controversial. There is little to no muscle at risk of ischemia in the fingers. The fingers primarily consist of bone, ligaments, subcutaneous fat, and tendons. These structures are quite resistant to ischemia. A finger

escharotomy can expose the metacarpophalangeal and interphalangeal joints, make them prone to infection, and require a subsequent fusion or amputation.

A final complication is the inadequate release of the overlying restrictive eschar. Ongoing tissue ischemia can lead to permanent disability from disfiguring muscle contractures, irreparable neurologic damage, or limb loss. Rhabdomyolysis can lead to life-threatening electrolyte abnormalities and acute renal failure. Several authors advocate extending the escharotomy incisions 1 to 2 cm beyond the eschar border into viable tissue. A secondary reperfusion injury can follow the primary decompression as blood once again streams into previously hypoperfused tissue. The consequent swelling can cause the formation of a secondary underlying intrafascial compartment syndrome.

SUMMARY

Thermal burns are frequently encountered injuries prompting patients to seek medical care. Their associated morbidity, mortality, and costs of treatment can be astronomical. Emergency Physicians often provide an essential role in the acute resuscitation and stabilization of these patients prior to their ideal transfer to a specialized burn center. Severe burns can destroy the natural elastic properties of skin and result in the formation of constrictive overlying eschars. These eschars can result in catastrophic limb loss, airway compromise, or respiratory failure. An escharotomy can be a limb-saving and life-saving procedure that grants the patient a significant chance to undergo more specialized and definitive burn care.

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140 Subcutaneous Hydration (Hypodermoclysis)

Mary J. O

INTRODUCTION

Subcutaneous rehydration therapy (SCRT), also known as hypodermoclysis, is a method of fluid replacement first described in the 1880s as a proposed treatment for cholera in India.[1] It involves the infusion of fluids into the subcutaneous space where it slowly diffuses into the circulation.[2] This method was widely used for rehydration until the 1950s until it was superseded by the intravenous (IV) route.[3] This was due to reports of serious adverse events that were most likely caused by improper choice of fluids for SCRT (e.g., shock secondary to osmotic shift caused by infusion of hypertonic or electrolyte-free fluids).[4] Subsequent studies have confirmed the safety and efficacy of hypodermoclysis.

SCRT offers many advantages over IV hydration. It is often much easier to obtain and maintain access in patients who may be dehydrated with collapsible veins, agitated, at risk of pulling out IV lines. Nursing time to obtain subcutaneous access is less than with IV access.[5] SCRT does not cause thrombophlebitis and there is less risk of fluid overload than with IV fluid administration.[6] The popularity of SCRT is once again increasing in geriatrics, palliative care, and limited-resource environments. Its use remains unfamiliar to and overlooked by many Emergency Physicians.

ANATOMY AND PATHOPHYSIOLOGY

Hyaluronidases are enzymes that break down hyaluronic acid, a compound found in the intercellular matrix responsible for stabilizing the structure of the connective tissue.[1] They are commonly used for subcutaneous hydration, extravasation of hyperosmolar radiologic contrast, and eye surgery.

Hyaluronidases are divided into three groups or classes (**Figure 140-1**).[7] These include the β-endoglucuronidases (found in hookworms and leeches), endo-β-*N*-acetyl-D-hexosaminidases (found in ants, bees, hornets, mammalian testicles, wasps, and yellow jackets), and the hyaluronic acid lyases (found in bacteria). They all are responsible for the hydrolysis of bonds in hyaluronic acid. These enzymes are responsible for insect venom spreading and causing allergic reactions. There are numerous forms that are available for human use (**Table 140-1**). Hyaluronidase is inactivated by blood.

Hyaluronidase temporarily breaks down the extracellular matrix and allows infused fluid to be absorbed up to five times more rapidly, decreasing the amount of localized edema, and increasing efficiency of SCRT.[8] The matrix will rebuild itself within 24 to 48 hours after the injection of hyaluronidase. Its use in hypodermoclysis is optional but helpful.[9-11]

Preferred injection sites are the upper back in between scapulae, anterolateral thighs, anterior abdomen sparing a 2.5 cm diameter

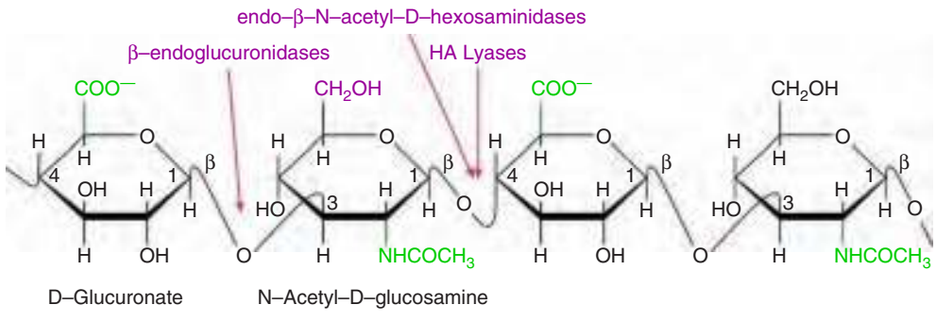


FIGURE 140-1. The action of various classes of hyaluronidases. (Used from reference 7.)

around the navel, and the subclavicular chest wall sparing breast tissue (**Figure 140-2**). The outer sides of the upper arms may be used if other sites are unavailable. Subcutaneous tissue tends to diminish in peripheral areas with age and more centrally located sites are preferred for infusion.[12]

The advantages of hypodermoclysis are numerous. The insertion of the catheter is easier than an IV catheter and less painful. It has fewer complications than IV access. It avoids the first-pass elimination by flowing through the liver. It needs less nursing supervision, needs less equipment, and costs are lower than with IV access. The IV is avoided in those with a needle phobia. Hypodermoclysis can be used for several days. It does not cause thrombophlebitis, venous clotting, or IV catheter clotting, and has few local effects. It does not cause fluid overload because of the limited fluid administered. Patients are unlikely to experience systemic symptoms from fluid administration.

There are numerous disadvantages to hypodermoclysis. The volume administered is limited. Hypodermoclysis causes local edema that resolves after the infusion is stopped. Only administer isotonic fluids. A limited amount of medications can be given subcutaneously (**Table 140-2**). Fluid is limited to that which is isotonic. Fluid is given by a drip to gravity and not a pump. Different rates of absorption occur in different people. Fluid boluses and rapid administration cannot be given. Each site is limited to 1500 mL in 24 hours, with two sites simultaneously used. It rarely happens that two sites are used simultaneously. Hypodermoclysis does not work well if edema is present. Hypodermoclysis can result in hemorrhage in patients who are anticoagulated or have bleeding disorders. Emergency Physicians lack the information and experience with this technique.

INDICATIONS

Hypodermoclysis may benefit patients who are mildly to moderately dehydrated, in whom IV access is difficult or impractical, and oral hydration is not possible.[13-19] It may also serve as a bridge to IV therapy in stable patients whose veins are initially collapsed due to dehydration. It may result in an increased chance of successful venous cannulation after receiving SCRT. Hypodermoclysis is often used for hydration in end-of-life care in the hospice patient. It can be used to administer medications for nausea and vomiting, seizures, pain management, and gamma-globulin (**Table 140-2**).

TABLE 140-1 The Characteristics of Various Hyaluronidase Preparations

	Available for use	Source	U/mL
Hylenex	Yes	Human recombinant	150
Vitrase	Yes	Sheep testis	200
Wydase	Not since 1999	Cow testis	150
Hydase	Not since 2008	Cow testis	150
Amphadase	Not since 2010	Cow testis	150

CONTRAINDICATIONS

SCRT is contraindicated in emergent or life-threatening situations when large and rapid fluid boluses are needed. It should not be used in patients who will require more than 3 L of fluid replacement in a 24-hour period. It is contraindicated in patients with significant peripheral edema as this interferes with the absorption of fluids deposited into the subcutaneous space. Hypodermoclysis should also be avoided in severely cachectic patients who have minimal subcutaneous tissue. Avoid skin sites that are broken, inflamed, infected, burned, scarred, or irradiated. Do not use sites near joints, bones, skin folds, tumors, or large blood vessels. The patient or their caregiver may refuse hypodermoclysis. Avoid infected or inflamed areas as hypodermoclysis can spread a localized infection. Avoid hypodermoclysis in the patient with anticoagulation, blood dyscrasias, circulatory failure, fluid overload states (e.g., congestive heart failure and renal failure), poor skin integrity, severe dehydration, severe electrolyte abnormalities, and shock. Do not use fluids that are hypertonic or hypotonic. Many medications are either not compatible for subcutaneous administration or are not tested.

Allergic reactions, anaphylaxis, and hypersensitivity to hyaluronidase can occur with animal-derived hyaluronidase. This is less likely

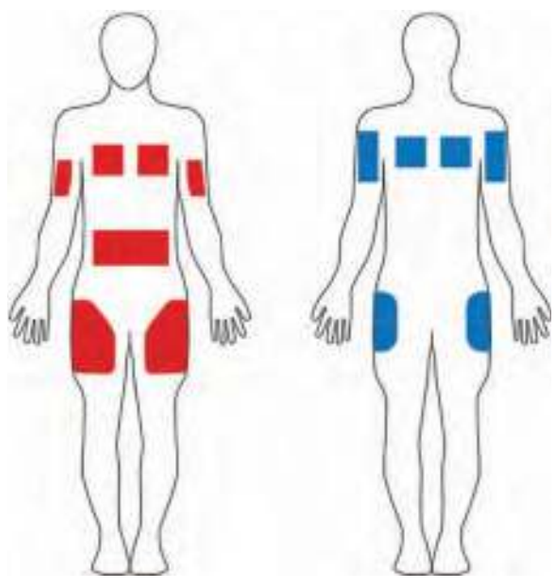


FIGURE 140-2. The potential sites for hypodermoclysis. (Used with permission from reference 23.)

TABLE 140-2 Medications and Their Relation to Subcutaneous Use

Can be used		
Ampicillin	Fentanyl	Metoclopramide
Atropine	Furosemide	Midazolam
Cefepime	Glycopyrrolate	Morphine
Cefotaxime	Granisetron	Naproxen
Ceftazidime	Haloperidol	Octreotide
Ceftriaxone	Hydromorphone	Ondansetron
Chlorpromazine	Hydroxyzine	Phenobarbital
Clonazepam	Ketamine	Potassium
Cyclizine	Ketorolac	Promethazine
Dexamethasone	Levomepromazine	Ranitidine
Diclofenac	Local anesthetics	Scopolamine
Dipyrone	Lorazepam	Tobramycin
Diphenhydramine	Methadone	Tramadol
Famotidine		
Cannot be used		
Alpha-agonists	Diazepam	Phenytoin
Compazine	Dopamine	Thorazine

Source: Modified from reference 23.

for the recombinant hyaluronidase. A skin test can be used to check for sensitivity. Inject 0.02 mL (3 units) intradermally of a 150 U/mL solution. A positive reaction is a skin wheal within 5 minutes that persists for 20 to 30 minutes.

EQUIPMENT

- Alcohol swab
- Chlorhexidine or povidone iodine
- 21 or 23 gauge butterfly needle or angiocatheter
- Commercial subcutaneous infusion device, optional
- Parenteral fluid
- Tubing with drip chamber, primed with the chosen solution
- Transparent dressing
- Tape
- Hyaluronidase (**Table 140-1**)

There are commercially available subcutaneous infusion devices that may be used in place of the standard butterfly needle or angiocatheter (**Figures 140-3 and 140-4**). They include the Saf-T-Intima (Becton Dickinson, Franklin Lakes, NJ), the Aqua-C (Norfolk Medical, Skokie, IL), and the ClearView Sub-Q (Norfolk Medical, Skokie, IL). These devices are discussed below. Other devices less used in the Emergency Department are made by MarCal Medical (Millersville, MD) and Churchill Medical Systems (Lansdale, PA). The advantages of these devices are the increased ease of use and the use of catheters that are designed to remain in place longer. These devices are costlier than using an angiocatheter or butterfly needle.

Electrolyte-free fluids (e.g., dextrose 5% in water) have been reported to draw fluid into the interstitial space causing third-spacing and cardiovascular collapse.[4] **These solutions should be avoided.**

PATIENT PREPARATION

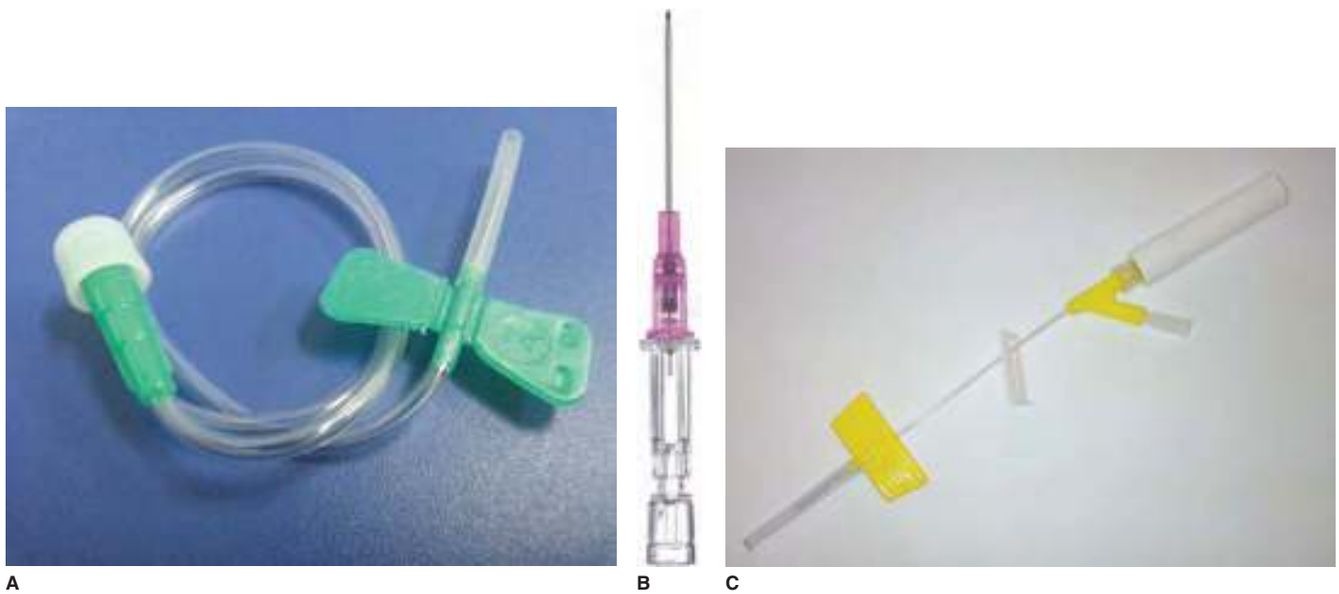
Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Assemble all necessary equipment. Wash hands thoroughly or use an alcohol-based hand cleaner. Don a clean pair of gloves. Prime the infusion tubing with the chosen fluid. Select an appropriate site for subcutaneous hydration. Clean and prepare the skin site. Clean any dirt and debris from the skin. Apply chlorhexidine or povidone iodine to the skin and allow it to dry. Cleaning off the chlorhexidine or povidone iodine with an alcohol swab is optional.

TECHNIQUES

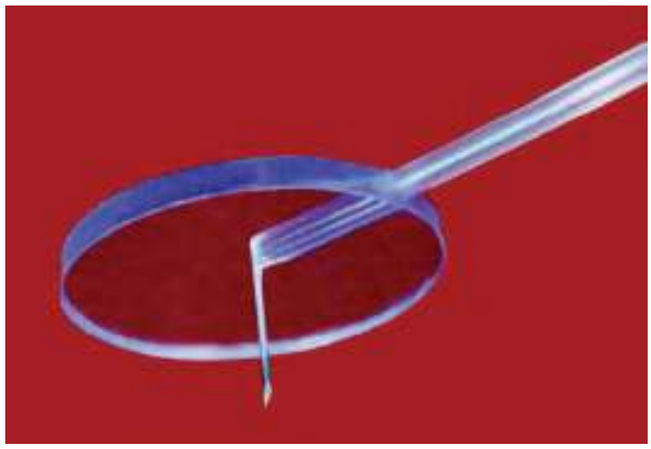
SAF-T-INTIMA

The Saf-T-Intima was designed for hypodermoclysis (Becton Dickinson, Franklin Lakes, NJ). It is a butterfly-type catheter (**Figure 140-3*C***). Grasp the catheter (**Figure 140-5*A***). Grasp the clean and prepared skin with a thumb and index finger (**Figure 140-5*B***). Grasp an area of approximately 2.5 cm



A B C

FIGURE 140-3. The catheters used for hypodermoclysis. ***A.*** The butterfly. ***B.*** The angiocatheter. ***C.*** The Becton Dickinson Saf-T-Intima.



A

B

FIGURE 140-4. Some of the devices used for hypodermoclysis. ***A.*** The ClearView Sub-Q. ***B.*** The Aqua-C.

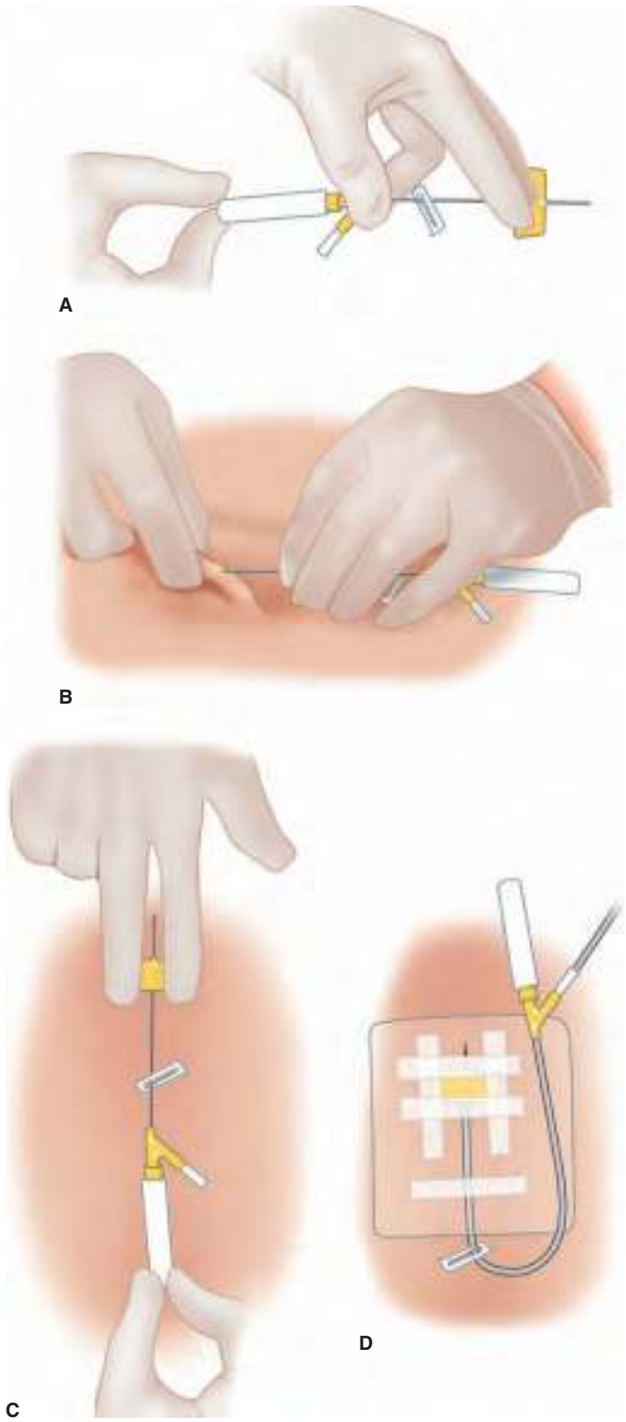
surrounding the insertion site to separate the subcutaneous space from the underlying muscle (**Figure 140-5*B***). Lift the skin upward. Insert the needle with the bevel up and at a 30° to 45° angle into the subcutaneous space located at the base of the area pinched. Advance the needle until the catheter hub is against the skin. Immediately withdraw the needle and select a new insertion site at least 2.5 cm away if blood appears in the tubing. Hold the catheter hub and remove the needle. Lay the catheter and tubing flat against the skin and hold the wings of the butterfly (**Figure 140-5*C***).

If using hyaluronidase, inject the desired amount through the catheter.[11] This is usually 1 mL of 150 U/mL. Attach the primed IV tubing to the catheter. Secure the catheter with tape and a transparent dressing (**Figure 140-5*D***). Start the IV fluid flowing.

The recommended infusion rate is 1 to 2 mL/min or approximately 1.5 to 3 L/day with a maximum of 1.5 L at each site.[2] Small studies have reported patients tolerating boluses of up to 500 mL/hr with the use of hyaluronidase. This requires the use of up to 750 units of hyaluronidase.[10]

ANGIOCATHETER

Use an angiocatheter or butterfly needle if a Saf-T-Intima is unavailable. Grasp the angiocatheter. Grasp the clean and prepared skin with a thumb and index finger. Grasp an area of approximately



A

B

C

D

FIGURE 140-5. Hypodermoclysis using the Saf-T-Intima catheter. ***A.*** Holding the device. ***B.*** Inserting the device. ***C.*** Stabilization of the device. ***D.*** Securing the device.

2.5 cm surrounding the insertion site to separate the subcutaneous space from the underlying muscle. Lift the skin upward. Insert the needle with the bevel up and at a 30° to 45° angle into the subcutaneous space located at the base of the area pinched. Advance the angiocatheter until the catheter hub is against the skin. Immediately withdraw the needle and select a new insertion site at least 2.5 cm away if blood appears in the tubing. Hold the catheter hub and remove the needle. Lay the catheter and tubing flat against the skin.

If using hyaluronidase, inject the desired amount through the catheter.[11] This is usually 1 mL of 150 U/mL. Attach the primed IV tubing to the catheter. Secure the catheter with tape and a transparent dressing. Start the IV fluid flowing.

The recommended infusion rate is 1 to 2 mL/min or approximately 1.5 to 3 L/day with a maximum of 1.5 L at each site.[2] Small studies have reported patients tolerating boluses of up to 500 mL/hr with the use of hyaluronidase. This requires the use of up to 750 units of hyaluronidase.[10]

COMMERCIAL DEVICES

The insertion of a commercial device is simple. Clean and prepare the skin as described previously. Insert the Aqua-C or ClearView Sub-Q perpendicular to the skin. Push the unit until it is flat against the patient's skin. The needle only penetrates a certain depth and into the subcutaneous tissue. Inject hyaluronidase, attach IV tubing, start the fluid infusion, and secure the catheter as described above.

PEDIATRIC CONSIDERATIONS

The use of hypodermoclysis in pediatric patients is similar to that in adults. There have been a few studies showing that SCRT may be a reasonable alternative to IV therapy in children but it is not yet included in current guidelines for the management of dehydration.[8,20,21] Only inject 1 mL of the 150 U/mL hyaluronidase and begin the infusion. Continued research will be needed to clarify the safety and role of SCRT in the pediatric population.

AFTERCARE

Initial swelling is expected at the insertion site when fluids start infusing. This will resolve after completion of the infusion. Check the infusion site for signs of redness, tenderness, or leakage at least once every 4 hours. Switch the site if any of the above are seen, if the patient has discomfort, or the patient has significant swelling. Raise the IV fluids if the drip is running slow. Switch the site if it continues to flow slowly. Observe the painful site for any signs of infection. Change the site if blood backflows at any time into the tubing. Pooling of fluid at the site can reduce the flow rate. Change the site. Bruising results from a blood vessel leaking. Change the site.

COMPLICATIONS

SCRT generally has fewer complications compared to IV hydration and most complications are not significant. The most common complications are local reactions (e.g., inflammation, swelling, redness), extravasation, bleeding, and bruising.[1,22] Swelling is mostly attributed to the rapid rate of infusion and can be lessened by local massage, slowing the flow rate, or diuretics. Do not use hypotonic or hypertonic solutions. Accidental venipuncture can occur. There have been rare reports of pulmonary edema due to fluid overload, although this rate is much lower than with IV fluid administration.[5] The use of hyaluronidase may produce a local allergic reaction, although the switch to a human recombinant version from an animal-derived version has lessened this risk. Rotate the site if complications are noted, if a dose of over 2000 mL is administered, if used over 24 hours, or as needed for patient positioning.

SUMMARY

Subcutaneous hydration or hypodermoclysis is an alternative method for rehydration that offers many advantages over the IV route. It is a safe, simple, and effective method of fluid administration to help correct mild to moderate dehydration with few side effects. The rate of fluid absorption is much slower than with IV access. Hypodermoclysis should not be used as a substitute for the IV route in life-threatening situations when large and rapid fluid boluses are required. The role of SCRT in the pediatric population continues to be investigated.

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141 Subcutaneous Extravasation and Infiltration Management

Henry Swoboda

INTRODUCTION

Extravasation and infiltration events are common occurrences with intravenous (IV) therapy and diagnostics (**Figure 141-1**). Some sources cite an incidence of 10% to 30%.[1] The incidence of extravasation of specific agents (e.g., intravenous contrast or vasopressors) is low.[2,3] Carefully placed IV catheters for chemotherapy have an incidence of extravasation as low as 0.01%.[4] Acute chemotherapeutic medication extravasations are infrequently encountered in the Emergency Department. **Understanding extravasations and their treatment is important due to the high degree of morbidity, the limited application of antidotes, and the increasing presence of standalone Emergency Departments and infusion centers.**[5] An understanding of the early recognition and treatment of chemotherapeutic extravasations is increasingly important.[6]

Most extravasations cause some degree of discomfort for the patient but display limited associated toxicity.[3,7,8] There is swelling distal or proximal to the IV site with the extravasate. The dependent portion of the limb swells. There is skin tightness at the IV site. This can be increased over an area depending on the amount of extravasation. The skin can be blanched and cool to the touch. Extravasation can stop the infusion and set off alarms on the IV pump. Extravasation may seep onto the skin and wet dressings. The Infusion Nurses Society has developed an infiltration and extravasation scale (**Table 141-1**).[9]

FIGURE 141-1. Subcutaneous infiltration of radiologic contrast. (Courtesy of Eric Heffernan, MD, and www.svohradiology.ie.)

TABLE 141-1 The Infusion Nurses Society Infiltration and Extravasation Scale

Grade	Criteria
0	No symptoms
1	Cool skin to the touch Edema in any direction of < 2.5 cm Pain may or may not be present Skin blanched
2	Cool skin to the touch Edema in any direction of 2.5–15.0 cm Pain may or may not be present Skin blanched
3	Cool skin to the touch Edema in any direction of > 15.0 cm Numbness may or may not be present Pain is mild to moderate Skin blanched or translucent
4	Circulation impairment Cool skin to the touch Edema in any direction of > 15.0 cm Infiltration of blood products, irritants, or vesicants Pain is moderate to severe Pitting edema Skin blanched or translucent Skin bruised, discolored, swollen Skin tight and leaking

Prevention of extravasation of medications with known toxicity is a key focus to prevent morbidity. Anything administered in an IV may infiltrate and extravasate into the extremity. Certain medications cause tissue damage when extravasated (**Table 141-2**). **It is recommended that medications with known extravasation toxicity be administered via a central venous catheter in nonemergent situations.**[10] This can limit but does not eliminate extravasation.[10] Sometimes central venous access is impractical for the administration of potentially toxic medications.[11] **Morbidity from extravasation may be minimized by frequent checks of the IV site during potentially toxic drug administration and using recently placed forearm IVs with progression from distal to proximal in placement attempts.** Extravasations in the antecubital fossa carry a high degree of morbidity.[12] Extravasation frequency increases in IVs placed near joints.[12]

Some agents have specific "antidotes" or therapies that are recommended in the event of extravasation (**Table 141-3**). There is poor-quality evidence supporting their use. The recommendations are based on known risks and theoretical benefits.[3,13] Warm compresses are recommended for agents in which increased blood flow and dispersal of the offending agent is desired. Cold compresses are ideal when limitation of inflammation and cytotoxicity is desired. The use of topical, intralesional, or systemic corticosteroids has been advocated. They are not recommended due to conflicting and negative data, lack of an inflammatory cause of tissue damage in most cases, and known deleterious effects on wound healing.[7,14] The use of a washout technique has mixed recommendations.[13] Routine surgical or washout procedures are not recommended due the poor supporting evidence and potential morbidity related to the procedures. Consider a saline washout with or without hyaluronidase in severe extravasations (e.g., those with extreme pain; significant swelling due to large-volume extravasation; clear erythema, blistering, or vascular compromise to the overlying tissue; or involving known vesicant medications without an available antidote).[6,15,16]

Many other specific therapies have been proposed. There are scant data to support their use and evidence of possible harm. Use

TABLE 141-2 Fluids and Medications That Can Cause Tissue Damage after Extravasation

Fluids	
Blood products	Furosemide
Calcium-containing fluids	Haldol
Dextrose solutions ≥ 10%	Interferon
Electrolytes	Interleukin
Hyperosmolar solutions	Ketamine
Hypertonic glucose	Mitomycin
Hypertonic saline	Mitoxantrone
Mannitol ≥ 20%	Nafcillin
Potassium-containing fluids	Norepinephrine
Potential for any fluid ≥ 60 mEq/L	Quinine
Sodium bicarbonate	Paclitaxel
Sodium chloride ≥ 30%	Pancuronium
Total parenteral nutrition	Penicillins
Medications	Pentamidine
Acyclovir	Pentobarbital
Adenosine	Phenobarbital
Alkylating agents	Phenylephrine
Allopurinol	Phenytoin
Amiodarone	Potential for any medication
Aminophylline	Promethazine
Antibiotics	Propofol
Arginine	Ranitidine
Atracurium	Remifentanil
Buprenorphine	Sedatives
Chemotherapy agents	Tetracyclines
Chlordiazepoxide	Thiamine
Contrast media	Thiopental
Dactinomycin	Valproate
Daunorubicin	Vancomycin
Diazepam	Vasopressin
Digoxin	Vasopressors
Dobutamine	Verapamil
Dopamine	Vinblastine
Doxorubicin	Vincristine
Epinephrine	Vindesine
Fentanyl	Vinorelbine

alternative therapies based on expert preference and institutional protocol. Therapies recommended in this chapter have a relatively low risk-to-benefit ratio for the management of severe extravasations. It is best to initiate a specific therapy without delay as many of the effects are considered time-dependent. Obtain a surgical consultation based on institutional policy, need for follow-up, and severity of the extravasation. Onset of toxicity for vesicant medications may be delayed.[6] These patients may benefit from surgical consultation and a washout up to 72 hours after the extravasation.[16]

Extravasation injuries typically occur after IV catheter displacement with ensuing localized swelling, burning, pain, and blanching or erythema. Recognition can be delayed in patients with abnormal mental status. Skin ulcerations and damage are frequent and not initially apparent. Injuries can progress to full-thickness skin sloughing in a delayed fashion. Subcutaneous extravasations can be managed via the general approach outlined in this chapter, in conjunction with the treatments listed in **Table 141-3**, and described in their respective sections.

Extravasations from chest and neck central venous access devices carry extremely high morbidity. These injuries typically present with chest pain and benefit from emergent aspiration of the extravasate, consideration of computed tomography scanning, and emergent surgical consultation.[7] Further discussion of extravasation from central venous catheters is outside of the scope of this chapter.

TABLE 141-3 Treatment of Common Extravasations

Nonchemotherapeutic medications	Treatment(s)
Concentrated electrolyte solutions (e.g., $NaHCO_3$, KCL, CaCl, NaCl, $MgSO_4$)	Warm compresses Consider hyaluronidase
Hypertonic solutions (e.g., D25, D50, total parenteral nutrition, radiographic contrast)	
Hyperosmolar, caustic, or vesicant medications (e.g., arginine, aminophylline, chloramphenicol, mannitol, ampicillin, oxacillin, nafcillin, propofol)	
Vasopressors	Warm compresses plus phentolamine or Terbutaline or 2% Nitroglycerin ointment
Phenytoin	Warm compresses
Promethazine	Frequent neurovascular checks Consider vascular imaging Consider Surgeon consultation
Chemotherapeutic medications	**Treatment(s)**
Etoposide	Warm compresses
Taxanes (paclitaxel, docetaxel)	Hyaluronidase
Vinca alkaloids (vinblastine, vincristine)	
Anthracyclines (doxorubicin, daunorubicin, epirubicin)	Cold compresses Dexrazoxane or DMSO
Bendamustine	Cold compresses
Cisplatin	Sodium thiosulfate
Mechlorethamine	
Mitomycin	Cold compresses DMSO

ANATOMY AND PATHOPHYSIOLOGY

Most extravasation toxicity is caused by one of four mechanisms. Tissue damage can be caused by (1) vasospasm and perfusion abnormalities related to vasoactive medications, (2) cellular dehydration and direct toxic effects of hyperosmolar medications, (3) pH-related tissue damage, or (4) direct cytotoxicity from vesicant chemotherapeutic medications.[2,3,17] Chemotherapeutics can be divided into vesicants and irritants with some overlap. Vesicant chemotherapeutics are typically associated with severe tissue damage while irritant chemotherapeutics are not. These compounds can be further classified as DNA-binding versus non-DNA-binding. The latter's concentration-dependent toxicity is more amenable to drug dispersion techniques.[7] Mechanical complications can occur from large-volume extravasation from IV or intraosseous catheters. These can cause direct compression of soft tissues or a compartment syndrome (Chapters 93 and 94). Treatment of a compartment syndrome is based on clinical evaluation, compartment pressures, and surgical consultation. Management of volume-related complications should focus on prevention and surveillance.[18,19] Dispersion of the extravasate is the aim of many treatments.[18,19] Examples of extravasated medications associated with tissue toxicity and their accompanying suggested treatments are listed in **Table 141-3**.

VASOPRESSOR EXTRAVASATION

There is support for the limited administration of vasopressors through peripheral IV catheters.[11] Administer ongoing vasopressors through a central venous catheter due to the potential morbidity associated with extravasations.[20] The management of vasopressor extravasation can include antidotes (e.g., nitroglycerine paste, phentolamine, or terbutaline) (**Figure 141-2**). Phentolamine is considered first-line therapy and is approved by the U.S. Food and Drug Administration (FDA) for norepinephrine extravasations. Its

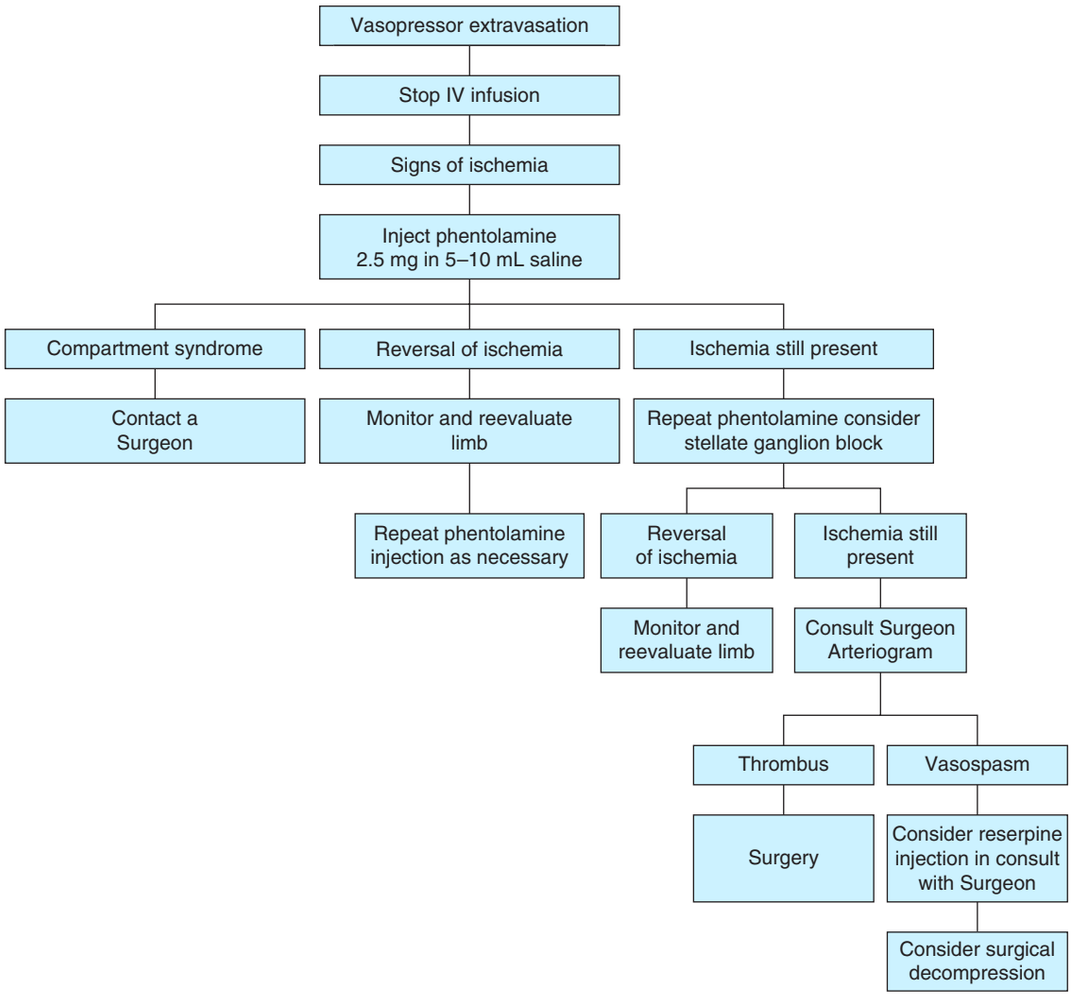

FIGURE 141-2. Treatment of vasopressor extravasation.

alpha-adrenergic antagonism is a focused therapy for all vasopressors with alpha-adrenergic agonism.[3] Phentolamine can be used as a nonspecific therapy for other vasopressor extravasations.[3]

NONNEOPLASTIC MEDICATION EXTRAVASATION

Medications and electrolyte solutions that have an osmolarity greater than 600 mOsm/L have the potential to cause tissue damage via cellular dehydration and osmotic stress (**Table 141-2**).[3] Calcium salts have been associated with significant morbidity due to protein precipitation and vasospasm. Hyaluronidase is a medication that increases connective tissue permeability via the temporary hydrolysis of hyaluronic acid. There is a significant amount of low-quality evidence supporting the safe use of hyaluronidase as a dispersion agent in extravasations. Administration of hyaluronidase can be combined with a washout procedure or it can be used independently. A new, ready-to-use recombinant product has replaced the old powdered formulation. The dosing recommendations have changed.

Local toxicity may occur from medications containing propylene glycol as a preservative (e.g., diazepam, digoxin, etomidate, lorazepam, nitroglycerin, phenobarbital, and phenytoin). Some medication solutions have pHs within the caustic range (e.g., acyclovir, amiodarone, conivaptan, doxycycline, pentamidine, phenytoin, promethazine, thiopental, and vancomycin). Treatment for these extravasations includes warm compresses and routine wound care. Hyaluronidase has been suggested for refractory cases. **Do not attempt neutralization.**[3]

Phenytoin administration has been associated with vascular occlusion and local toxicity, referred to as the purple glove syndrome (PGS).[21] No extravasation is detected in most cases. The PGS may be due to micro-extravasation and other mechanisms. Patients with suspicion for PGS require close neurovascular monitoring, aggressive vascular imaging, and early Surgeon consultation. PGS is rare, consistently reported, and all specific treatments (e.g., heparin and hyaluronidase) are unproven. PGS has been associated with extravasation, intraarterial administration, undiluted medication administration, use of small IV catheters, distal IV location, and concomitant administration of other vesicants.[22] Over 95% of reported cases were associated with one of these factors.[22] Their routine avoidance in phenytoin administration would be prudent.[22] Use of fosphenytoin may decrease the incidence of PGS.

Promethazine has been associated with a similar syndrome in the setting of extravasation or intraarterial injection. There are similarly no proven therapies even though some advocate for sympathetic block and heparinization. The focus has been on prevention by using a dilute solution and employing intramuscular or slow IV administration.[3]

CONTRAST EXTRAVASATION

Extravasation of IV contrast is an infrequent event (**Figures 141-1 and 141-3**). Studies indicate an incidence of 0.34% to 0.7% of contrast administrations. It remains an important issue given the common use of IV contrast for imaging (e.g., > 10,000 administrations

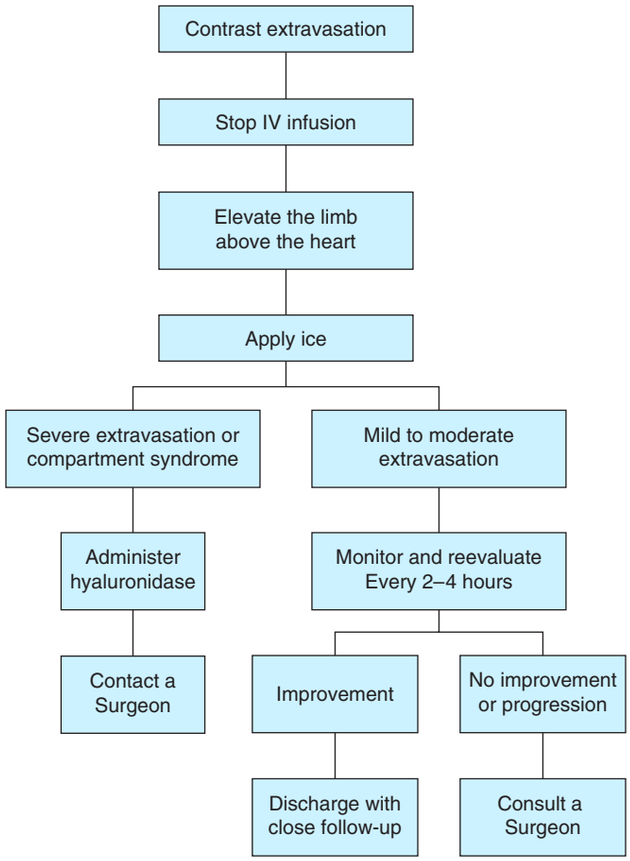

FIGURE 141-3. Treatment of contrast extravasation.

per year at high-volume institutions).[23,24] Severe injuries from contrast extravasations have decreased greatly with the advent of low osmolarity IV contrast. **Most contrast extravasations have minimal associated toxicity. Patients do well with observation and conservative management.** High-risk features include extremes of age, preexisting subcutaneous atrophy, distal IV placement, compromised vascular or lymphatic flow, use of a power injector, high osmolar contrast extravasation, and high-volume (> 50 mL) extravasation.

CHEMOTHERAPEUTIC EXTRAVASATION

Chemotherapeutics are divided into vesicants and irritants based on their potential tissue toxicity. The vesicants are associated with potentially severe toxicity and skin sloughing. Both classes of medications have been associated with local skin reactions during infusion. This can confound the early diagnosis of extravasation. Local reactions frequently resolve quickly once the infusion is stopped. Treat reactions observed by experienced personnel concerning for extravasation. Chemotherapeutic agents have a wide range of toxicities depending upon their mechanism of action. Antidotes are most frequently employed for vesicant chemotherapeutics due to the severity of their toxicity.

Special attention has been paid to increasing dispersion for extravasations of vinca alkaloids (e.g., vincristine and vinblastine) and epipodophyllotoxins (e.g., etoposide) with hyaluronidase and the use of warm compresses. Cold compresses may cause harm.

Dimethylsulfoxide (DMSO) is a free radical scavenger that has been used for a variety of extravasation injuries. It is currently indicated for extravasations of dactinomycin and mitomycin. Apply 99% DMSO (or the highest concentration as available) topically at the site of extravasation. This can be accomplished in the outpatient setting.[25]

Extravasation of nonliposomal anthracyclines (e.g., doxorubicin, daunorubicin, and epirubicin) has been associated with full-thickness skin sloughing and severe toxicity in up to 50% of cases.[26] These cases are subject to aggressive treatment. Dexrazoxane is an iron chelator and antidote used for systemic anthracycline toxicity. It is FDA approved for use in local extravasation injury.

The extravasation of mechlorethamine or a high-risk extravasation of cisplatin or bendamustine (e.g., overlying a joint, a high concentration, or a large volume) can be deactivated with 4% sodium thiosulfate. Manage other chemotherapy extravasations with cold packs and general management as described below.[2,7]

INDICATIONS

Phentolamine is indicated for significant vasopressor extravasation. Terbutaline and/or nitroglycerin ointment can be administered for vasopressor extravasation if phentolamine is not available. Hyaluronidase is specifically indicated for vinca alkaloid extravasation, potentially for high osmolar substances, and large radiographic contrast extravasations. Sodium thiosulfate is indicated for mechlorethamine, bendamustine, and cisplatin extravasations. Administer DMSO for mitomycin extravasations and for anthracycline extravasations if dexrazoxane is not available. Administer dexrazoxane for confirmed anthracycline extravasations that occurred within 6 hours. Use the saline washout technique for severe extravasations of medications without an available antidote or as directed by a surgical consultant.

CONTRAINDICATIONS

Do not give DMSO with dexrazoxane. Do not use the saline washout technique with mild extravasations or extravasations with effective antidotes available. Do not use hyaluronidase near infected or cancerous tissue for fear of spread. Recombinant hyaluronidase has < 1% incidence of allergy and should not be given to patients who are known to be allergic.

EQUIPMENT

- Syringes, various sizes
- Needles, various sizes
- Antidotes (**Table 141-3**)
- Cold packs
- Warm packs
- Povidone iodine or chlorhexidine solution
- Lidocaine without epinephrine
- Hyaluronidase, 150 units/mL
- Blunt needles
- Scalpel with #11 blade
- Sterile 0.9% saline
- Irrigation basin

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Place the patient on the cardiac monitor, noninvasive blood pressure cuff, pulse oximetry, and supplemental oxygen to monitor them as an added level of precaution. Mark the area of extravasation with a pen. Take digital photographs if possible.

TECHNIQUES

GENERAL APPROACH TO EXTRAVASATIONS

The general approach to extravasations is noted in this section (**Figure 141-4**).[3,7,18] Halt the infusion at the first sign of extravasation. Leave the IV catheter in place and do not flush it. Aspirate as much of the extravasate as possible through the IV catheter. Administer analgesics if required. Inject the antidote through the IV catheter and at additional sites as specified for each antidote. Consider a saline washout if a severe extravasation occurs without an available antidote. Remove the IV catheter. Apply dry warm or cold compresses. Warm compresses cause vasodilation which can result in increased systemic absorption, the spread of injury, and increased surface area of the injury. Cold compresses cause vasoconstriction which can concentrate the extravasate, decrease the spread, and worsen the local injury. Apply topical antidotes (e.g., DMSO or nitroglycerin ointment). Elevate the extremity to prevent dependent edema and improve lymphatic outflow. Follow routine wound care with antiseptics (e.g., silver sulfadiazine or topical antibiotic ointment), neurovascular checks, and wound checks. Obtain a consult by a Surgeon per institutional policy and for the development of a compartment syndrome, skin necrosis, and skin ulceration.

SALINE WASHOUT

The saline washout technique can be used for extravasations.[6,15,16,27] Halt the infusion at the first sign of extravasation. Leave the IV catheter in place and do not flush it. Aspirate as much of the extravasate as possible through the IV catheter. Inject the antidote through the IV catheter and at additional sites as specified for each antidote.

Cleanse the skin with chlorhexidine or povidone iodine solution. Allow the antiseptic to dry. Anesthetize the area of extravasation with subcutaneous lidocaine. Place 1 mL of hyaluronidase (i.e., 150 units/mL) into a tuberculin syringe. The remainder of the procedure can be completed without hyaluronidase if it is unavailable. Inject 0.2 mL of hyaluronidase subcutaneously through the infiltrated IV catheter. **Do not inject hyaluronidase if blood draws from the IV catheter.** Inject an additional 150 units of hyaluronidase through the IV catheter for large extravasations. Inject the remaining hyaluronidase (i.e., 0.8 mL) through a 25 gauge or smaller needle at four points along leading edges of the extravasation. Make four stab incisions with a #11 blade scalpel around the periphery of the extravasation. Insert a blunt needle subcutaneously and manipulate it gently to make subcutaneous tracts. Flush the area of extravasation with 20 to 50 mL aliquots of 0.9% saline solution through each stab incision. Ensure drainage of the saline solution through the remaining incisions. Irrigate a total volume of 500 mL of saline. Continue with routine wound care.

VASOPRESSOR EXTRAVASATION

Stop the infusion when extravasation is detected. Examine the site for signs of toxicity, swelling, or blanching. Leave the IV catheter in place and aspirate to remove as much extravasate as possible. Inject 1 to 2 mg of phentolamine subcutaneously through the IV catheter for significant extravasations. Inject an additional 1 to 2 mg (5 to 10 mg total dose diluted in 10 mL normal saline in adults and 0.1 to 0.2 mg/kg up to 10 mg in children) in four places at the leading edge of extravasation. Terbutaline (1 mg diluted in 10 mL of saline for adults and 0.005 to 0.01 mg/kg in children) can be administered in a similar manner if phentolamine is unavailable. Use 2% nitroglycerin ointment (0.5 to 1 inch in adults and 4 mm/kg up to 1 inch

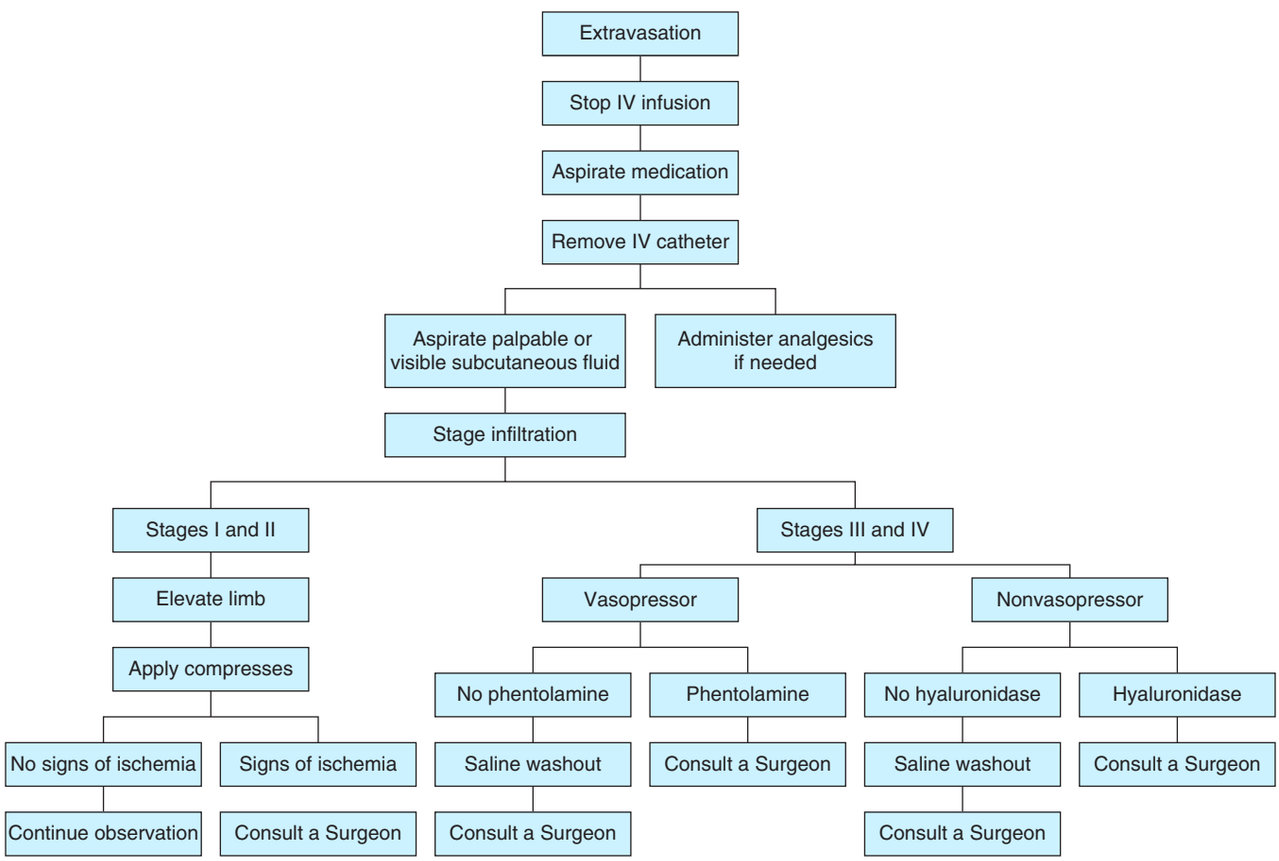

FIGURE 141-4. The general treatment of an extravasation.

in children) applied to the area of extravasation. Similar treatment may be employed for refractory effects of accidental digital epinephrine administration, although patients typically do well regardless of the care given.[28] The side effects of phentolamine and nitroglycerin administration include mild hypotension.[17] Continue resuscitation and reinstitute vasopressor therapy at an alternate IV site.

NONNEOPLASTIC MEDICATION EXTRAVASATION

Administration of hyaluronidase can be combined with a washout procedure or it can be used independently. Cleanse the skin with chlorhexidine or povidone iodine solution. Allow the antiseptic to dry. Anesthetize the area of extravasation with subcutaneous lidocaine. Place 1 mL of hyaluronidase (i.e., 150 units/mL) into a tuberculin syringe. Inject 0.2 mL of hyaluronidase subcutaneously through the infiltrated IV catheter. **Do not inject hyaluronidase if blood draws from the IV catheter.** Inject the remaining hyaluronidase (i.e., 0.8 mL) through a 25 gauge or smaller needle at four points along leading edges of the extravasation.[3] Some sources recommend dilution of the hyaluronidase solution with saline to 15 units/mL for use in neonates and infants. The package insert does not require this but it may facilitate administering the hyaluronidase over a larger area.

CONTRAST EXTRAVASATION

Treat patients with mild contrast extravasation with extremity elevation, cold or hot packs, and observation for 2 to 4 hours (**Figure 141-3**). They can be managed as outpatients in the absence of a worsening clinical exam. Hyaluronidase as previously described has been used safely for large subcutaneous extravasations but should not be used routinely.[23] Consult a Surgeon for signs of a compartment syndrome. Severe extravasations, severe symptoms, or signs of neurovascular compromise may benefit from hyaluronidase administration.[18,29] Consult a Surgeon for severe cases and apply silver sulfadiazine for skin blistering and skin ulceration.[18,29]

CHEMOTHERAPEUTIC EXTRAVASATION

Special attention has been paid to increasing dispersion of extravasations of vinca alkaloids (e.g., vincristine and vinblastine) and epipodophyllotoxins (e.g., etoposide). Use hyaluronidase as previously described and warm compresses. Cold compresses may cause harm. Apply 99% DMSO (or the highest concentration available) topically at the site of extravasation at 4 drops per 10 cm^2 to an area twice the size of the extravasation every 8 hours for 7 days. Allow the DMSO to air dry. The patient should apply cool compresses for 60 minutes three times a day for 3 days between DMSO applications. This can be accomplished as an outpatient.[25]

Dexrazoxane is an iron chelator and antidote used for systemic anthracycline toxicity. It has been FDA approved for use in local extravasation injury. Administer IV dexrazoxane within 6 hours at a renally adjusted dose of 1000 mg/m^2 daily for 2 days and 500 mg/m^2 on day 3. Discontinue the cool compresses 15 minutes before the administration of dexrazoxane to allow circulation of the antidote to the site. Patients will require monitoring of complete blood counts and liver transaminases following dexrazoxane therapy due to the risk of bone marrow suppression and elevated liver enzymes.[26,30] Dexrazoxane is costly, specialized, and may not be available. Apply topical DMSO as described previously if dexrazoxane is not available.[25]

Extravasation of mechlorethamine or high-risk extravasation of cisplatin or bendamustine (e.g., overlying a joint or high concentration or volume) may occasionally occur. This can be deactivated with 4% sodium thiosulfate. Inject 2 mL of thiosulfate per milligram of extravasated agent divided between the extravasated IV catheter and multiple injections around the area of extravasation followed by intermittent cool compresses for 48 to 72 hours. Sodium thiosulfate at a 4% concentration may be ordered from pharmacy. It may be compounded by mixing 1.6 mL of 25% sodium thiosulfate with 8.4 mL of sterile water or 4 mL of 10% sodium thiosulfate with 6 mL of sterile water. Manage other chemotherapy extravasations with cold packs and general management.[2,7]

ASSESSMENT

Document any changes due to the extravasation. Note the initial site changes and any further changes. Note the substance extravasated, its concentration, the estimated volume that extravasated, and the circumstances of the extravasation. What were the symptoms of the extravasation and did this change? What size IV catheter was used and what is its length? How was the medication and/or fluid administered (e.g., IV to gravity, IV piggyback, intramuscular pump, or IV push)? Document any problems with the pump if used (e.g., did not alarm) and its serial number. Label the pump for a check by biomedical engineering or the company and set it aside.

AFTERCARE

Document at the time the patient leaves the Emergency Department. Note the extent of injury, the patient symptoms upon leaving, the site of the injury, and any patient comments. Note any patient education. Document all techniques and treatments used on the extravasation. Note the responses, if any, to these techniques and treatments. Document all personnel involved in the patient's care. Most patients will be admitted to the hospital for moderate or severe extravasations. Place patients with mild extravasations in observation. Patients with mild extravasations may be discharged if the patient is reliable and close follow-up can be arranged.

COMPLICATIONS

Most complications are from the extravasation. There are complications associated with treatments. The patient may have an allergic reaction to any treatment antidote. The use of a scalpel to make incisions and blunt needle manipulation can injure arteries, muscle, nerves, tendons, and veins. The use of certain treatments (e.g., nitroglycerine or phentolamine) can result in hypotension. The use of nitroglycerine with the patient using erectile dysfunction medications can result in life-threatening hypotension.

The best way to prevent complications is to prevent the extravasation.[12,31,32] Ensure that all staff are trained on IV cannula insertion. Start low and move proximally when attempting an IV cannulation. Avoid veins that can cause the IV to dislodge or kink (e.g., antecubital fossa, deep brachial, dorsal hands, and over joints). Do not use small veins or veins used recently. Inject the worst offending medications first. Monitor patients receiving IV infusions. Use caution and extra monitoring in patients with altered mental states, circulatory impairment, confusion, or limited veins.

Other devices may be added for patient safety. The ivWatch (ivWatch, Williamsburg, VA) monitors the IV site to detect leaks, extravasation, and infiltration (Figure 59-25). It is comprised of the patient monitor, a reusable sensor cable, single-use disposable sensor applied next to the IV site, and an AC adapter. The monitor gives an audible and visual alert. Refer to Chapter 59 for a discussion of other devices.

SUMMARY

Infiltration of IV catheters is a common occurrence in the Emergency Department. Prevention, conservative management, and good wound care are the cornerstones of management. Washout procedures and antidotal therapy may ameliorate the effects in

severe extravasations or those involving toxic solutions. The level of evidence supporting these therapies is low and the Emergency Physician must determine when they should be employed.

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SECTION 8

Neurologic and Neurosurgical Procedures

142 Lumbar Puncture

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INTRODUCTION

Meningitis and subarachnoid hemorrhage (SAH) are serious, life-threatening conditions. They require prompt and accurate diagnosis in the Emergency Department (ED) due to their significant morbidity and mortality.[1] There are many diagnostic modalities available to the Emergency Physician (EP) to assist in the diagnosis. However, the lumbar puncture (LP) is still considered the gold standard. The LP is a procedure that is often performed in the ED to obtain information about the cerebrospinal fluid (CSF) to aid in the diagnosis of a variety of medical conditions. Knowledge about the proper indications, contraindications, various techniques, equipment, and recognition and treatment of potential complications is vital to any EP who performs this procedure. **It is important to perform a thorough neurologic examination prior to the LP, as significant neurologic deficits may suggest increased intracranial pressure (ICP). Significant morbidity and mortality can result if the procedure is performed on the wrong patient.**

ANATOMY AND PATHOPHYSIOLOGY

The entire cavity of the brain and spinal cord has a volume of approximately 1650 mL. CSF occupies only approximately 150 mL of this volume. The brain literally floats in the CSF because the specific gravity of the CSF and brain are approximately the same. Approximately 500 mL of CSF is produced every day. This is approximately 0.35 to 0.5 mL/min. It takes less than 20 minutes for the patient to replace the CSF removed from the LP procedure. Most of the CSF is produced by the choroid plexus within the lateral ventricles. Small amounts of choroid plexus can also be found in the third and fourth ventricles. Lesser amounts of CSF are secreted by the ependymal surfaces of the ventricles. A minimal volume of CSF is produced by the brain through the small perivascular spaces that surround the blood vessels entering the brain substance.

The flow of CSF through the ventricular system is rather simple (**Figure 142-1**). CSF produced in the lateral ventricles flows through the foramina of Monro into the midline third ventricle. It then passes through the aqueduct of Sylvius into the fourth ventricle. From the fourth ventricle, the CSF flows into the cisterna magna via two lateral openings (foramina of Luschka) and one midline opening (foramen of Magendie). The cisterna magna is located beneath the medulla and cerebellum. It is continuous with the subarachnoid space that surrounds the brain and spinal cord. The CSF then flows through the subarachnoid space to bathe the brain and spinal cord. The CSF is absorbed back into the venous system by way of the arachnoid villi.

The average CSF pressure is 130 mmH_2O when measured in the lateral decubitus position. It can range from 70 to 200 mmH_2O in a normal person after 8 years of age and up to 250 mmH_2O in the obese patient.[2] It ranges from 10 to 100 mmH_2O in children up to 8 years of age. The pressure is regulated by the rate of CSF absorption by the arachnoid villi as the CSF production rate is constant. These structures act as one-way valves for CSF to flow into the venous blood of the dural sinuses. Disease states that impede reabsorption can lead to increased ICP.[3]

Familiarity with the anatomy of the spinal column is important when performing an LP. The anatomy will be briefly reviewed from superficial to deep as the spinal needle traverses the midline structures. The skin and subcutaneous tissue are the first layers encountered. These are followed by the supraspinous and intraspinous ligaments, located between the spinous processes of adjacent vertebrae. Deep to these ligaments is the thick ligamentum flavum that accounts for the characteristic "pop" that is described when performing an LP. The next layers encountered are the epidural fat, in the epidural space, followed by the dura mater and finally the subarachnoid space.

There are subtle anatomic differences when considering the lateral approach. The layers include skin and subcutaneous tissue followed by the paraspinal ligaments. The intraspinous ligament is less likely to be encountered with an extreme lateral approach, as this is a midline structure. The ligamentum flavum and deeper structures should be encountered in the same fashion regardless of the approach.

INDICATIONS

There are many indications for performing an LP. The primary indications to perform an LP are suspicion of either a central nervous system infection (e.g., meningitis, encephalitis) or an SAH. LP may also be performed for the evaluation of new-onset seizures, to obtain CSF biomarkers, to diagnose and treat idiopathic intracranial hypertension (IIH; formerly known as pseudotumor cerebri), to evaluate for central nervous system diseases (e.g., Guillain-Barré syndrome, multiple sclerosis, systemic lupus erythematosus), to confirm demyelinating or inflammatory diseases, to administer antibiotics or chemotherapeutic agents, to aid in radiologic imaging procedures (e.g., cisternography or myelography), or to diagnose meningeal carcinomatosis.

SUSPICION OF MENINGITIS IN ADULTS

An LP should be performed in adults when there is a clinical suspicion of a central nervous system (CNS) infection.[4] The absence of fever does not exclude a CNS infection. Meningeal signs include nuchal rigidity, Kernig's sign, and Brudzinski's sign. Other signs of a possible CNS infection include a severe headache, photophobia, or a petechial rash. Many of these signs may not be present. This is especially true in elderly, young, or immunocompromised patients. **An LP should be strongly considered in febrile adults with an altered mental status and no alternate source of fever**.

Two commonly cited meningeal findings include the Brudzinski's sign and the Kernig's sign (**Figure 142-2**). Both tests are performed in the supine patient. For the Brudzinski's test, passively flex the

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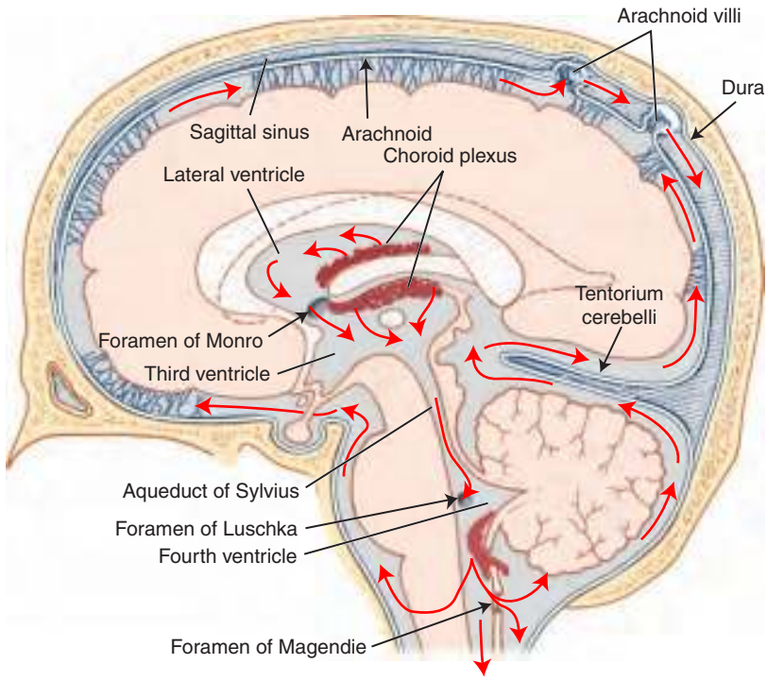


FIGURE 142-1. CSF circulation around the brain and upper spinal cord.

patient's head until their chin touches the sternum. Flexion of the patient's hips and knees in response to the head flexion is considered a positive Brudzinski's sign (**Figure 142-2*A***). The patient may also experience neck pain and resistance to flexion if meningitis is present. For the Kernig's test, passively flex one of the patient's legs to 90° at the hip and to 90° at the knee (**Figure 142-2*B***). Passively extend the knee. Pain in the lower back or resistance to knee extension is known as a positive Kernig's sign.[5] Neither test is very sensitive or specific for meningitis.[6]

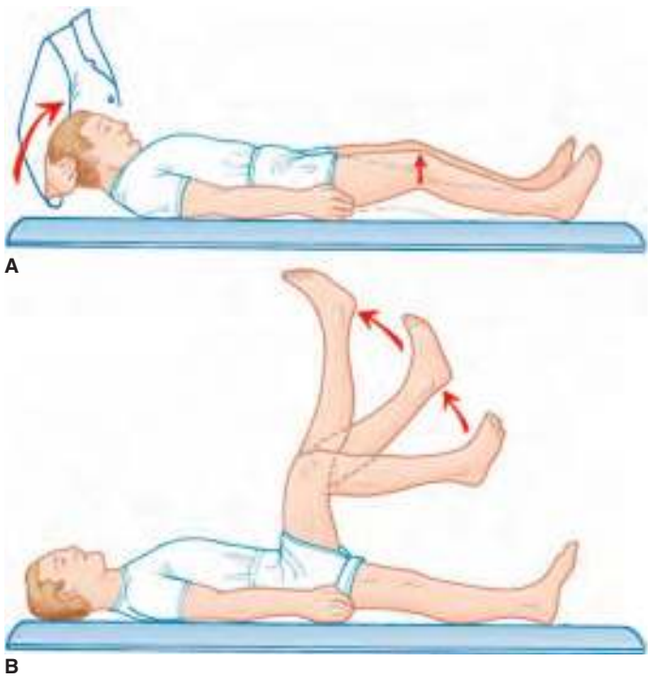


FIGURE 142-2. Physical examination of meningeal signs. ***A.*** Brudzinski's sign. Upon passive elevation of the head, the patient complains of neck and low back pain and may have involuntary flexion of the knees and hips suggesting meningeal irritation. ***B.*** Kernig's sign. Begin with the patient starting in a supine position with their hips and knees flexed 90°. Gradually extending the knee causes the patient to complain of neck or lower back pain.

SUSPICION OF MENINGITIS IN CHILDREN

The decision of whether to perform an LP on a febrile infant is based on the clinical suspicion of meningitis, the age and appearance of the child, and whether an identifiable source of fever is present.[4] The EP will often be faced with a well-appearing febrile infant with no obvious source of fever. In the past, many institutions managed all febrile infants less than 3 months old with a full sepsis evaluation, to include an LP and admission to the hospital. Newer recommendations suggest that the full septic evaluation is now indicated for infants less than 1 month old and strongly considered in infants less than 60 days old.

Guidelines are available to identify patients at low risk for serious bacterial infection, where LP and hospitalization may or may not be indicated.[7] Examples of serious bacterial infections include meningitis, sepsis, osteomyelitis, septic arthritis, urinary tract infections, pneumonia, and enteritis. The clinical evaluation alone is inadequate to exclude serious bacterial infections in infants. It must be combined with laboratory studies that help to define low-risk criteria. These criteria only apply to non–toxic-appearing infants with reliable parents and follow-up. A more detailed discussion regarding the evaluation of the child with a fever is beyond the scope of this chapter.

It should be noted that any febrile child, regardless of age, who appears "toxic" should have an LP as an integral part of the sepsis evaluation. Certain bacterial infections have a high propensity for dissemination and systemic bacteremia. Examples include epiglottitis, buccal cellulitis, periorbital cellulitis, and septic arthritis. These children should be considered for an LP as part of their evaluation depending on the clinical scenario.

IS AN LP INDICATED FOR THE FIRST FEBRILE SEIZURE?

The EP will be faced with the decision of whether to perform an LP when evaluating infants and children with a first febrile seizure. The American Academy of Pediatrics (AAP) recommends that an LP should be performed for any child if there is a clinical suggestion of meningitis.[8] Clinical signs and symptoms that have been shown to correlate with the presence of meningitis include petechiae, nuchal rigidity, coma, persistent drowsiness, Kernig's or Brudzinski's sign, status epilepticus, and paralysis.[9] The AAP also recommends performing an LP if the child has not received their *Haemophilus influenzae* type b (Hib) immunization or *Streptococcus pneumoniae* immunization, or if the immunization status cannot be determined.[8]

Benign febrile seizures are those that occur in children 6 through 60 months of age, are associated with a fever at the onset of an illness, and involve a single generalized seizure lasting less than 15 minutes in a child that did not recur within 24 hours.[8] **These children generally appear well except for the fever and usually do not require an LP.**

Complex febrile seizures are those that do not meet the criteria of a benign febrile seizure. These children require a complete septic evaluation including an LP. Criteria include seizures that begin focally, seizures lasting over 15 minutes, children with a prolonged postictal period, suspicious findings on physical examination, children less than 6 months of age, children already receiving antibiotics, or children who have had multiple seizures during a single period of illness. Suspicious findings on physical examination that suggest a complex seizure include rashes, petechiae, cyanosis, hypotension, abnormal respirations, increased or floppy tone, stiff neck, difficult to console, deviated eyes, doll's eyes, nystagmus, ataxia, photophobia, bulging or tense fontanelles, eyes that are unable to fix and follow, and no response to voice or painful stimuli.

SUBARACHNOID HEMORRHAGE

A suspected SAH is the other common indication for an LP.[10-17] The classic description of an SAH is the sudden onset of an excruciating headache (often described as a "thunderclap" headache) during exertion that may or may not be associated with syncope, nausea, vomiting, diaphoresis, or meningeal signs. Physical examination findings may include nuchal rigidity, an altered level of consciousness, papilledema, retinal hemorrhage, third-nerve palsy, sixth-nerve palsy, bilateral lower leg weakness, nystagmus, ataxia, aphasia, or hemiparesis. Additional risk factors for a SAH include cigarette smoking, hypertension, alcohol abuse, a family history of SAH, polycystic kidney disease, connective tissue disorders, or sickle cell anemia.

It is estimated that 20% to 50% of patients with an SAH may have sentinel bleeds that precede the major bleeding event. **It is important to diagnose a sentinel bleed because early management and intervention can improve the overall outcome for the patient. A sentinel bleed may precede a major SAH by hours, days, weeks, or months.[18,19]**

A computed tomography (CT) scan of the head is often the first study used to investigate a patient complaining of a sudden-onset headache concerning for SAH.[20] The small volume from a sentinel bleed into the subarachnoid space diffuses and is diluted away from the source and hemolyzes. The sensitivity of CT scan for SAH can range from 92% to 98% when performed within 24 hours of the onset of symptoms.[21-23] The sensitivity decreases markedly when performed 48 to 72 hours after onset of symptoms.[24,25] The head CT scan can be negative in the patient with a sentinel bleed. The head CT scan can be negative in the patient with anemia.

Should an LP be performed if the CT is negative? This issue is currently debated and controversial. The sensitivity of CT scanners is increasing. Some have suggested that the LP can be omitted if the head CT is negative within 6 hours of the headache onset due to the low incidence of positive LPs.[26-32] There is not enough evidence now to justify not doing an LP. Performing an LP in this group of patients may become unnecessary in the future.[33]

An LP must be performed if the CT scan is negative and an SAH is still suspected.[14-17,20,30] The presence of either xanthochromia or red blood cells in the CSF will confirm the presence of bleeding. While the CSF will usually confirm an SAH, it is possible that the LP may be negative despite a recent SAH if there has not been sufficient time for the red blood cells to migrate to the lumbar spine area. In this case, despite a negative head CT scan and a negative LP, a cerebral angiogram or follow-up LP in 12 to 18 hours should be performed.

CONTRAINDICATIONS

Knowledge of the contraindications to performing an LP is important.[34] The EP will often have to weigh the potential risks of performing the procedure with the benefits of obtaining the CSF. The decision must be made whether the procedure should be performed immediately or can be delayed until further studies are completed. Absolute contraindications to performing an LP include a cellulitis or abscess at the skin puncture site or signs and symptoms of increased ICP (except for IIH). The LP should be delayed in patients with an unstable airway, hypotension, shock, or status epilepticus until the patient has been stabilized. Relative contraindications to performing an LP include the presence of a brain abscess, epidural or subdural fluid collection, brain tumors, and spinal cord tumors.

Note that antibiotics should not be delayed if meningitis is suspected and the LP must be delayed.[35,36] If meningitis is highly suspected and the patient is unstable, treatment should be initiated with parenteral antibiotics and steroids. The LP can be delayed until the patient's condition is stabilized.

Do not perform an LP if the patient has an intrathecal pump. An LP can damage the catheter, even if going inferior to the scar. The catheter can loop below the scar and be damaged by the spinal needle. Consult an Interventional Radiologist to perform the LP under fluoroscopy.

INCREASED ICP

An LP is relatively contraindicated in the presence of increased ICP.[34,37-39] This includes patients with space-occupying lesions (e.g., tumor or abscess), lateralizing signs (e.g., hemiparesis), or when signs of uncal herniation are present (e.g., unilateral third-nerve palsy). Brain herniation or coning has been reported in patients with meningitis and increased ICP due to the sudden drop in spinal pressure induced by the removal of CSF from the LP.

IIH has been treated with a combination of medicines and repeated LPs to lower ICP. Removal of CSF for IIH is a common diagnostic and therapeutic procedure in the ED. The performance of an LP for IIH in the ED is controversial. Many of these patients are obese, which can make an LP more difficult to perform, require multiple attempts, and be painful for the patient. The CSF removed reforms within a few hours. The LP provides only temporary relief of the patient's symptoms. It is recommended to consult a Neurologist before performing an LP in the patient with a known diagnosis of IIH. An LP may be required to make the initial diagnosis of IIH but may be unnecessary in the patient when the diagnosis has been made previously.

BRAIN ABSCESS

Patients with a brain abscess are at high risk for herniation.[34,40,41] Brain abscesses may present with a progressively worsening headache, low-grade fever, or the development of focal neurologic signs (e.g., hemiparesis, papilledema, visual field deficits, and mild obtundation). Suspect a brain abscess in patients with a history of otic or paranasal sinus infection, orbital cellulitis, chronic pulmonary or intraabdominal infection, endocarditis, congenital heart disease, recent dental procedures or abscesses, recent neurosurgery, craniofacial trauma, open skull fractures, or recent meningitis.

WHEN IS A CT SCAN INDICATED BEFORE AN LP?

Increased ICP by itself is not necessarily a contraindication to an LP. ICP is usually mildly elevated in patients with IIH or meningitis.[34,42-44] A CT scan does not need to be routinely performed in straightforward cases of suspected meningitis when the patient has a normal neurologic examination. The inability to visualize the optic discs does not constitute a focal finding and, by itself, is not an indication for a head CT scan prior to an LP.[45] Despite this, there are occasional cases of herniation in patients with a normal CT scan.[37,38,43]

Most literature suggests that CT scans be performed prior to LP when patients are comatose or altered, have focal neurologic signs, are human immunodeficiency virus (HIV) positive, have a progressively worsening headache, or have papilledema.[34,45-47] **The lack of papilledema is not always a reliable sign of normal ICP, as it often takes greater than 48 hours to develop papilledema**.[48] Papilledema may be absent in up to 15% of adults and up to 50% of children with early increased ICP.

The CT scan should be evaluated for mass lesions, shift of midline structures, or hydrocephalus due to obstructing masses, cisternal obstruction, and cerebral edema.[34,47] There are three findings that may predispose a patient to herniation if an LP is performed.[49] The first finding is midline shift. This suggests unequal pressures across the midline. The second finding is loss of the suprachiasmatic and basilar cisterns. This suggests unequal pressures between the supratentorial and infratentorial compartments. The third finding is any evidence of a posterior fossa mass, obliteration of the superior cerebellar cistern, or obliteration of the quadrigeminal plate cistern which lies caudal to the midbrain. These findings all suggest the presence of increased infratentorial pressure.

Do not delay the initiation of antibiotics if meningitis is suspected and a CT scan is indicated before performing an LP.[35,36] Administer the antibiotics and steroids before the patient undergoes CT scanning. Several studies have shown that delays in the initiation of antibiotics are common in the ED. These delays are often EP generated and result from the need for a CT scan prior to LP, waiting for LP results before administering antibiotics and steroids, and not giving antibiotics and steroids before a patient is transferred to the ward.[50-53]

COAGULATION ABNORMALITIES

An LP is relatively contraindicated in patients with a coagulopathy.[34] This includes hemophiliacs, those on anticoagulants, and patients with thrombocytopenia. An LP can result in a spinal epidural or subdural hematoma with subsequent spinal cord compression. Appropriate replacement of platelets, replacement of clotting factors, and/or administration of reversal agents should be undertaken prior to attempting an LP if the procedure can be delayed.[54-56] **The most experienced EP should perform the procedure with a small-gauge needle when an immediate LP is indicated in these patients**.

BACTEREMIA

Some sources list bacteremia as a contraindication to an LP, especially in children. While there is some suggestion that there may be an association between performing an LP in a bacteremic patient and the later development of meningitis, the risk of this is low. **An LP should not be withheld for fear of inducing meningitis in these patients.** The risk of delaying the diagnosis of meningitis clearly outweighs the small chance of causing meningitis with an LP.[57]

EQUIPMENT

- Sterile gloves and gown
- Face mask and cap
- 1% lidocaine solution
- Povidone iodine or chlorhexidine solution
- LP needles, various gauges and lengths
- Topical anesthetic agent (e.g., eutectic mixture of local anesthetics [EMLA]), optional
- LP kit

Most of the equipment necessary for performing an LP is available in prepackaged, sterile, disposable, and single patient use commercial kits (**Figure 142-3**). These kits usually contain a 20 gauge Quincke spinal needle, syringes, needles (e.g., 25 gauge and 22 gauge) for local anesthesia, a manometer with a stopcock, sterile drapes, specimen tubes, 1% lidocaine, gauze, brushes for prepping the skin, and a bandage. Some kits provide povidone iodine or chlorhexidine swab sticks, whereas other kits have a small basin that needs to be filled. The EP should become familiar with the kit used at their institution.

Additional supplies may be needed to perform the procedure without interruption. For example, some EPs prefer a smaller gauge Quincke needle or a nontraumatic needle such as a Sprotte or Whitacre which are not often provided in the commercial kits (**Figure 142-4**). The use of a 25 gauge spinal needle is recommended as it causes a smaller puncture hole and a lower risk of postprocedural headache.[58-62] It has been suggested that atraumatic needles should be the standard when performing an LP.[63,64] It is a good idea to have extra spinal needles, lidocaine, gauze, and skin antiseptic (e.g., povidone iodine or chlorhexidine solution) when performing an LP. There are numerous formulae to determine the LP depth and the length of the spinal needle required.[65,66] A reliable formula for

FIGURE 142-3. A commercially available LP kit.

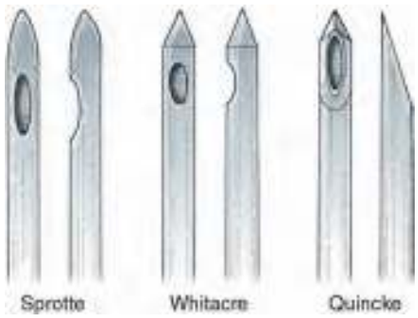


FIGURE 142-4. The three types of spinal needle tips. The standard Quincke needle has a sharp, beveled end. The Whitacre and Sprotte needles are designed to spread atraumatically, rather than cut dural fibers.

estimating the required LP needle length is LP depth (cm) = 1 + [17 × (weight [kg] ÷ height [cm])].[66]

There are subtle differences among the spinal needles commonly available (**Figure 142-4**). The standard Quincke needle has a sharp tip with a broad bevel at the end. The Whitaker and Sprotte needles have smaller tips with smaller diameter bevels. The bevel of the Sprotte needle is broader with a rounded tip to separate the fibers of the dura as opposed to cutting through them. **Always remember to keep the bevel oriented parallel to the fibers of the dura when performing an LP regardless of needle style.**

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Include the post-LP headache, its occurrence, and its treatment.[67] Obtain an informed consent for the procedure and place this in the medical record.[68] When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.[69] Ask the parents or caregivers if they would like to be present for the LP.

There are a variety of different patient positions that can be used to perform the LP (**Figure 142-5**).[58,70,71] Knowledge and proficiency in more than one approach will be useful for the EP when encountering a difficult LP. The position of the patient will be chosen based upon the patient's body habitus, their ability to assume a position, their level of cooperativeness, and EP preference.

The sitting position is more commonly used in adults than the lateral decubitus position. It is easier to identify the midline and

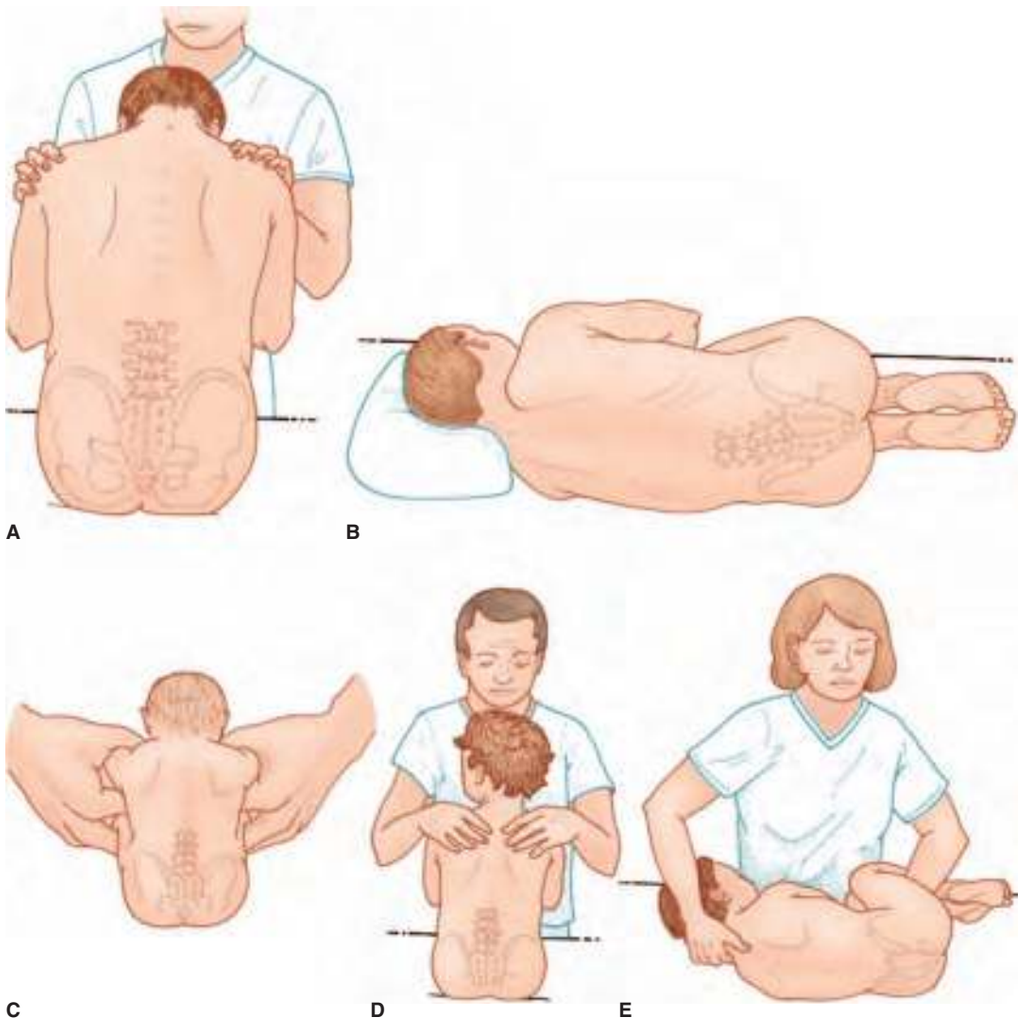


FIGURE 142-5. Patient positioning for an LP. ***A.*** An adult in the sitting position. ***B.*** An adult in the lateral decubitus position. ***C.*** An infant restrained in the sitting position. ***D.*** A child held in the sitting position. ***E.*** A child held in the lateral decubitus position.

palpate the spinous processes with the patient sitting (**Figure 142-5*A***). The sitting position is particularly useful when patients are obese. While the LP can still be performed in the lateral decubitus position for obese patients, palpating the spinous processes and identifying the midline can be difficult. **The measurement of hydrostatic pressure in the CSF is not accurate with the patient sitting, as it can give a falsely elevated pressure reading.**

The lateral decubitus position is often the most comfortable position for the patient (**Figure 142-5*B***). Place the patient with their knees flexed and the upper back curled forward to widen the interlaminar spaces. Ensure that the patient's shoulders, back, and hips are exactly perpendicular to the stretcher and floor. This will increase the chances of keeping the needle in the midline as it is introduced parallel to the surface of the stretcher. This is particularly important in infants and children where an assistant may be called upon to hold the patient in position. **Severe neck flexion is not necessary and can lead to airway obstruction or lack of CSF flow.**

Children can be placed in the sitting or lateral decubitus position.[72,73] An assistant can easily maintain neonates and infants in the sitting position (**Figure 142-5*C***). Toddlers and school-aged children can often be maintained in the sitting position (**Figure 142-5*D***). The lateral decubitus position can be used for any child (**Figure 142-5*E***).[74] **It is important to assess the child visually and with pulse oximetry during the procedure.[74] These positions may cause improper neck flexion that can result in respiratory compromise, oxygen desaturation, hypoxemia, and anoxic encephalopathy.[75,76] The largest interspinous space is achieved with the patient sitting upright with their hips flexed, leaning forward, and with slight neck flexion based on ultrasonographic studies.[76-79]**

The subarachnoid space must be entered below the termination of the spinal cord that is situated at the lower level of L1 or the body of L2 (Figure 142-6). Identify by palpation the vertebral spinous processes in the midline and the posterior superior iliac spines. The patient can help to determine if the needle is in the midline by providing real-time feedback of where they feel the needle.[80,81] Just ask them if the needle is in the midline. Another option is to draw a line between the spinous process of C7 and the gluteal cleft.[82] An imaginary line connecting the posterior superior iliac spines (i.e., Tuffier's line) should intersect the midline at approximately the L4 spinous process or the L3-L4 interspace. **One can select any of the spaces between L2-L3 and L5-S1 to perform an LP.** Palpate the intended interspace before prepping the area. Some EPs mark the site lightly with a pen or make a small indentation with the hub of a needle. Adjust the bed height so that you can sit in a comfortable position while performing the procedure.

Prepare the LP kit. Open the kit using sterile technique and place it on a bedside table. Place povidone iodine or chlorhexidine solution into the basin provided with the kit. Place any additional needles or supplies onto the sterile field. **This procedure requires strict aseptic technique. The EP should don full sterile and personal protective equipment at this point.[83-85]** This should include sterile gloves, a sterile gown, a face mask, and a cap. Prepare the stopcock and manometer. The manometer is usually in two pieces that slide together. Insert the manometer into the vertical port of the stopcock. Turn the handle toward the outflow side of the stopcock. The stopcock handle will occlude the port that it points to. Pull the stylette out of the spinal needle to ensure there is no damage.[86] Replace the stylette into the spinal needle.

Prepare the patient's back. Clean the skin of any dirt and debris. Apply povidone iodine or chlorhexidine solution using a circular motion from the intended site of entry outward. Allow the solution to completely dry prior to inserting the spinal needle.[87] Chlorhexidine solution introduced into the spinal canal can result in arachnoiditis and neurotoxicity.[88] Avoid chlorhexidine solutions above 0.5%.[87] Prepare an area of at least 10 cm in diameter. Most kits include a solid drape and a fenestrated drape. Place the solid drape between the patient's hips and the bed. Place the fenestrated drape with the adhesive side toward the patient's back and the opening centered at the desired level for the procedure.

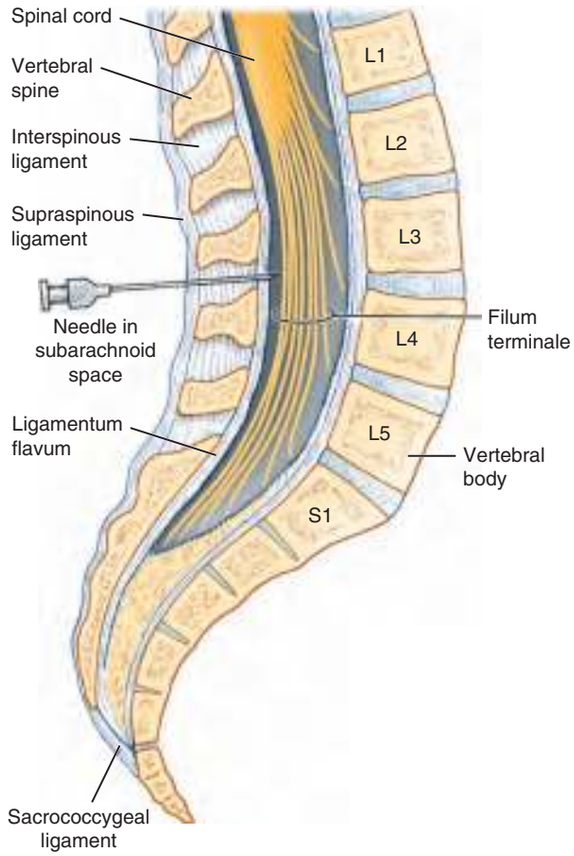


FIGURE 142-6. Midsagittal section of the lumbosacral region with a spinal needle in the L3-L4 interspace. The needle has penetrated the supraspinal ligament, the interspinous ligament, the ligamentum flavum, the dura mater, and the arachnoid mater.

Reidentify the anatomic landmarks. Place a finger over the desired interspace to use for the procedure. Place a skin wheal of local anesthetic solution subcutaneously over the desired interspace using a 25 gauge needle. Infiltrate and anesthetize the deeper tissue of the interspace along the projected needle track using the 22 gauge needle. **The infiltration of local anesthetic solution, unless contraindicated, should be used in all patients, including neonates and young children.** Local anesthetic makes it more likely the LP will be successful.[77,78,89]

Alternatively, a field block can easily be performed to produce anesthesia of the skin, interspinous ligaments and muscles, and the periosteum. The interspinous ligament and the periosteum are supplied by the recurrent spinal nerves branching off the nerve roots exiting the spinal canal at the same level. Inject local anesthetic solution into the interspinous ligaments, between the spinous processes superior and inferior to the intended puncture site, and on either side of the interspinous space (**Figure 142-7**).[58] A topical anesthetic (e.g., EMLA cream) may be applied over the interspace for 30 to 60 minutes prior to performing the LP if the patient is awaiting a CT scan of the head and antibiotics have been administered. The topical anesthetic only anesthetizes the skin and superficial subcutaneous structures. The patient will still require an anesthetic injection. A jet injector (Chapter 154) can be used to deliver the local anesthetic.[90,91]

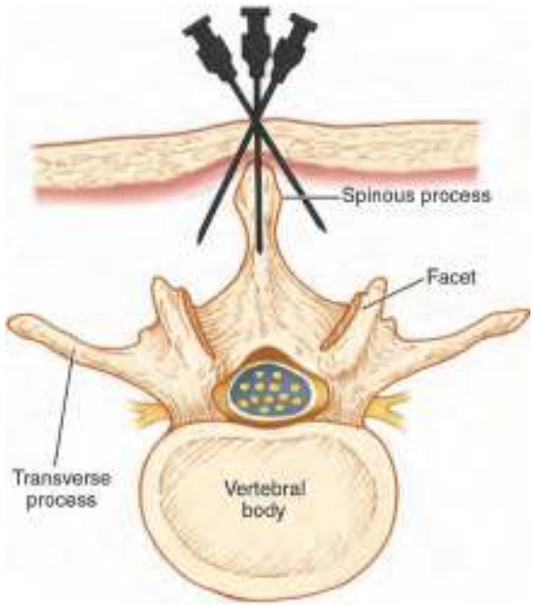


FIGURE 142-7. Field block for LP anesthesia.

It may occasionally be difficult for some patients to remain still and cooperative with the procedure. This can include anxious patients, those with an altered mental status, and small children. These patients may require an intravenous anxiolytic, nitrous oxide administration (Chapter 158), or procedural sedation (Chapter 159).

TECHNIQUES

LATERAL DECUBITUS POSITION AND MIDLINE APPROACH

Palpate the intended interspace. Introduce the needle in the middle of the interspace and parallel to the bed. **Orient the bevel of the spinal needle parallel to the longitudinal dural fibers to increase the chances that the fibers will be separated rather than cut by the tip of the needle.**[92] This has been shown to decrease the incidence of postdural puncture headache.[93-95] The bevel should point up or down with the patient in the lateral decubitus position. Angle the needle 10° cephalad, or toward the umbilicus, and advance it slowly. The needle can be held between both index fingers and advanced with the thumbs (**Figure 142-8*A***). Alternatively, it can be guided with a thumb and forefinger near the puncture site while the other hand holds the hub of the needle and advances it (**Figure 142-8*B***).

Resistance will usually be felt as the needle penetrates the interspinous ligaments. Stop advancing the needle and remove the stylet frequently to check for the presence of CSF.[96] Many describe a characteristic "pop" that is felt when the needle enters the subarachnoid space. The commonly used Quincke needles often decrease or eliminate this sensation.[96] If bone is encountered, withdraw the needle to the subcutaneous tissue, confirm your landmarks, and readvance the needle in the midline. If bone is still encountered, redirect the needle slightly more cephalad and readvance it. Perform the procedure at a different level if still unsuccessful.

CSF should flow freely once the subarachnoid space is entered. The rate of CSF flow from the hub of the spinal needle may be slow. The flow rate can be increased by one of the following: ask the patient to Valsalva by gently coughing or bearing down, rotate the spinal needle 90°, or repeat the procedure with a larger bore

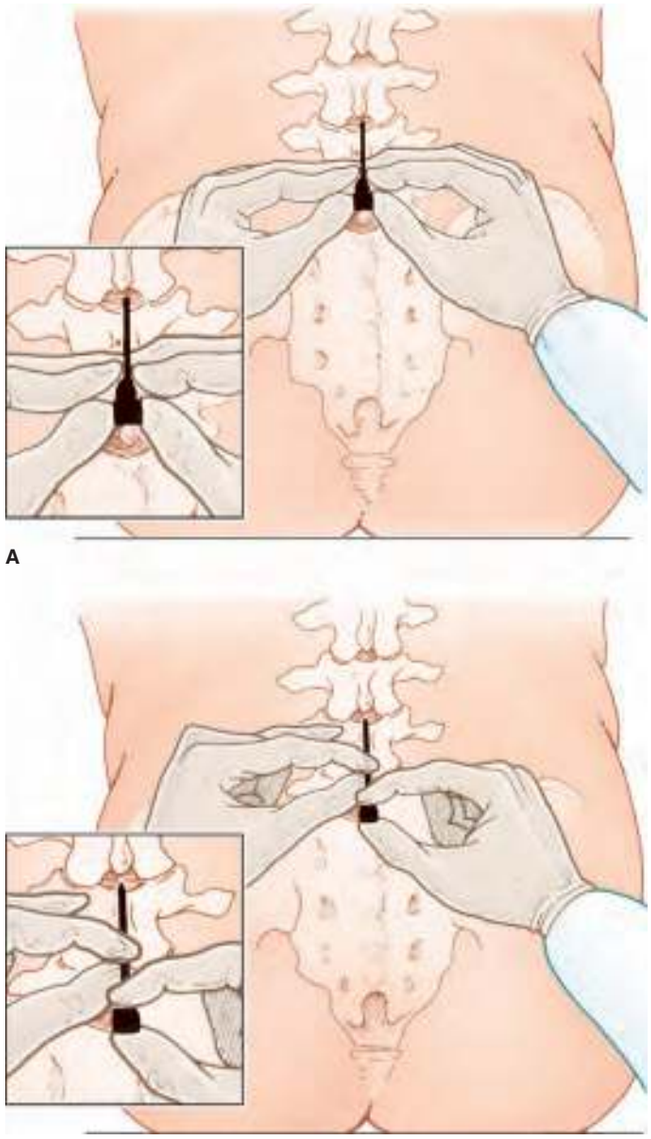


FIGURE 142-8. Two-handed techniques for spinal needle insertion. ***A.*** The index fingers guide the tip while the thumbs advance the needle. ***B.*** An alternative technique. One hand is placed at the needle tip and the other is at the base of the needle.

spinal needle. Attach the stopcock and manometer directly to the needle or use the short extension tubing provided in most kits as a spacer. **Hold the hub of the needle firmly between the thumb and index finger and brace your hand against the patient's back when attaching or removing anything from the spinal needle**. This will prevent the needle from advancing or withdrawing. The stopcock handle should point posteriorly, allowing CSF to fill the manometer.

Ensure that the manometer hub remains at the level of the needle in order to get an accurate reading if using the extension tubing. Phasic changes with respirations should be noted as the manometer fills. Instruct the patient, or an assistant, to gently extend the patient's legs to decrease intraabdominal pressure and lower the reading. Normal opening pressure ranges from 70 to 180 mmH_2O in most patients. Obtain the pressure reading once the CSF flow stops. Turn the stopcock handle toward the needle hub (i.e., the patient) to empty the contents of the manometer into the

first tube for collection. To continue collection, remove the stopcock or simply remove the manometer and continue collection through the stopcock by pointing the handle toward the manometer port.

In general, 1.0 mL of CSF in each of the four tubes should be adequate to perform the CSF analysis. Collect 2 mL in each tube if cytology or antigen testing is necessary. The rate of CSF collection can be increased. Ask the patient to cough or bear down (e.g., as in doing a Valsalva), instruct an assistant to push on the patient's abdomen, turn the spinal needle 90°, or use a larger diameter spinal needle to increase the flow of CSF. **Carefully replace the stylet and remove the needle when the samples have been collected.**

There is disagreement regarding the use of the spinal needle stylet.[97] The literature notes an increased incidence of intraspinal epidermoid tumors after an LP if a stylet is not used. This led to the habit of leaving the stylet in place while inserting the spinal needle to prevent transferring epidermal cells into the spinal canal. Some experts now recommend removing the stylet after the tip of the spinal needle has been inserted past the epidermis.[77,98] This method has been shown to result in a greater success rate when performing an LP.

Reinserting the stylet into the spinal needle is potentially problematic. Many will hold and stabilize the spinal needle with one hand and reinsert the stylet with the other hand. This risks a potential needlestick injury. The book editor inserts the tip of the stylet with one hand without using the other hand to stabilize the spinal needle. Once the stylette is partially inserted, grasp the spinal needle with the other hand and fully insert the stylette. This prevents punctures by the stylette. Reinserting the stylet prior to removal of the spinal needle has been shown to decrease the incidence of the post-LP symptoms of dizziness, headache, nausea, and tinnitus.[99] The method of using the stylet is left to EP preference.

SITTING POSITION AND MIDLINE APPROACH

Place the patient sitting on the edge of the bed. Ask the patient to flex their lower back and lean forward onto some support (e.g., an assistant or bedside stand) to open the interlaminar spaces in the lumbar area. Orient the bevel of the needle laterally (i.e., to the left or right). The remainder of the procedure is the same as previously described.

LATERAL APPROACH

The lateral approach may be useful in avoiding the calcified supraspinous and interspinous ligaments often encountered in elderly patients. This approach may be performed with the patient in the lateral decubitus position or the sitting position. Although it is less commonly used than the midline approach, it is a good idea for the EP to become familiar with this technique as an alternate approach if the midline approach has failed. This may prove easier in the patient who has had multiple previous midline LPs.

Position the patient and select an appropriate interspace. Cleanse, drape, and anesthetize the area as previously described. Insert the spinal needle 1.5 to 2.0 cm lateral to the midline. The needle can approach from either side (i.e., left or right) if the procedure is being performed in the sitting position. Approach from the inferior side if performing the LP in the lateral decubitus position (**Figure 142-9*A***). Direct the needle 10° cephalad and approximately 20° to the midline. This angle will direct the needle through the erector spinae muscles and lateral to the supraspinous and interspinous ligaments. The needle will penetrate the ligamentum flavum, the dura, and then the subarachnoid space (**Figure 142-9*B***). If bone is encountered, partially withdraw the needle and redirect it at the same angle toward the midline but slightly more cephalad. The remainder of the procedure is as described previously.

LP IN INFANTS AND CHILDREN

Performing an LP in an infant or child is similar to that of an adult. Place the patient in the lateral decubitus position or the sitting position.[72,73,100] Place the neck in mid-flexion in the lateral decubitus position. **Severe flexion of the neck does not facilitate the procedure and can result in lack of CSF flow or airway obstruction.**

Hypoxemia has been reported during LP in infants. The increased intraabdominal pressure caused by flexing the knees

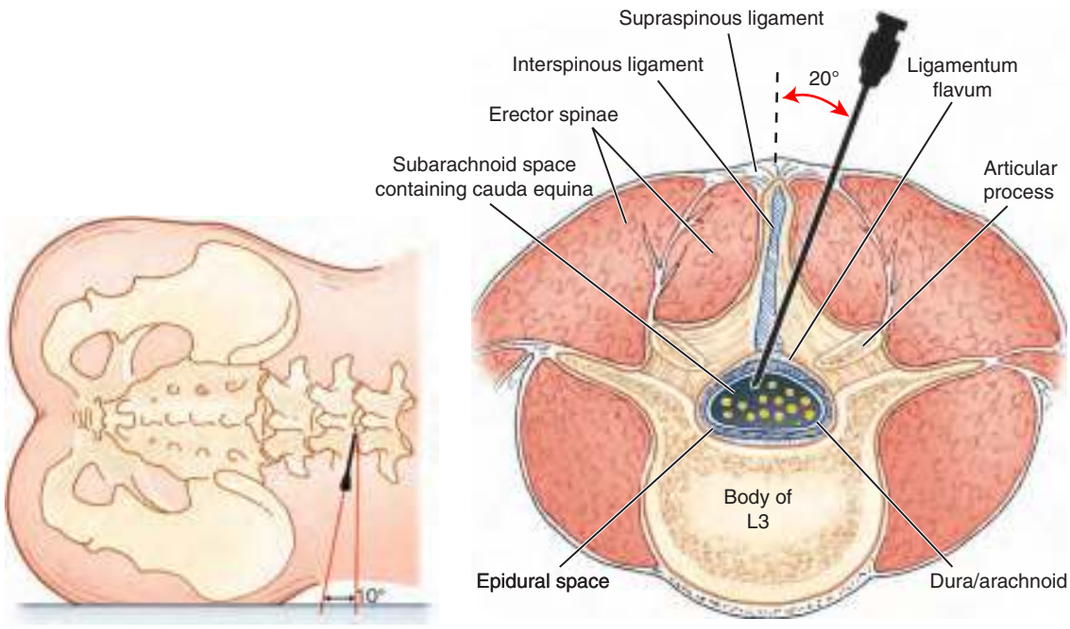


FIGURE 142-9. The lateral approach. ***A.*** The patient is in the lateral decubitus position. Insert the spinal needle approximately 2 cm below the midline, directed approximately 20° toward the midline and 10° cephalad. ***B.*** Cross-section of the spinal column showing the path of the spinal needle during the lateral approach. Notice that it avoids the calcified supraspinal and interspinal ligaments.

into the abdomen may lead to compression of the diaphragm, ventilation-perfusion mismatch, and hypoxemia.[101] The sitting position or modified lateral decubitus position (e.g., hips only flexed to 90°) is preferred.[102] Preoxygenation with 100% oxygen via face mask for 2 to 5 minutes may prevent hypoxemia.[103] **Consider the use of continuous pulse oximetry in infants and young children undergoing an LP.**

Some authors advocate the use of a butterfly needle and using the tubing as a manometer to get a general idea of the opening pressure. The use of nonstyletted needles, however, may occasionally result in the implantation of cells and a subsequent epidermoid tumor. In some low ICP syndromes, CSF may fail to flow during the procedure and gentle suction with a 1 mL syringe can be used.

Local anesthesia should be used in all patients, including neonates. There is evidence that pain perception is present even in premature neonates.[104] The use of local anesthetic is often omitted in the neonate and young infant, possibly in fear of obscuring anatomic landmarks. The success rate of LP in neonates given local anesthetic, the amount of struggling during lidocaine injection, and the amount of struggling during spinal needle insertion were studied.[105] The study found that local anesthesia did not alter the success rate of the procedure and led to a decreased amount of struggling during spinal needle insertion.

The use of EMLA cream or similar topical anesthetic is common and effective for venipuncture in children. It also can be used successfully before LP in children and adults (Chapter 154).[106,107] In some studies on adults, EMLA has been shown to be more effective than lidocaine infiltration.[108,109] The major disadvantage of EMLA cream is that it requires application for a minimum of 30 minutes before the procedure is performed. It is more effective if it stays on longer. It does not anesthetize the deeper tissues and local infiltration is still required.

Procedural sedation is usually reserved for children getting routine LPs for intrathecal chemotherapy. Procedural sedation can be used while performing a diagnostic LP. Procedural sedation should not be used unless the child has a normal mental status and is hemodynamically stable. The decision regarding procedural sedation should be considered on a case-by-case basis.

The angle of the spinal needle insertion was found to be different in different age groups.[110] The angle was more acute in infants compared with younger children. It remains to be seen whether this clinical difference affects the success rate of an LP.

ULTRASOUND-GUIDED LP

Anesthesiologists have been using ultrasound (US) to assist in various spinal procedures. EPs have reported on the use of US to assist in performing an LP or to evaluate spinal anatomy.[111-123] US-guided LP was performed easily and was successful in patients in whom multiple attempts at landmark-guided LP were unsuccessful.[113] The authors concluded that the technique has great potential as a time-saving tool. A randomized, double-blind study compared LP using landmarks identified by palpation versus those identified by US.[115] The use of US significantly reduced the number of failures in all patients and improved the perceived ease of the procedure in obese patients. A group of Radiologists studied US guidance for LPs in children.[124] They found US superior to fluoroscopy as it allowed three-dimensional guidance in real time and provided visualization of soft tissue structures.

US is more readily available in EDs to assist the EP in performing many procedures including an LP.[111,112] US can visualize superficial and deep tissue structures. EPs who consider using US in assisting with an LP should be properly trained in the use and theory behind US before attempting to use it in the clinical setting.

There are three possible approaches to establishing the indications for the use of US to guide an LP. US guidance could be used for an LP when the traditional landmark-guided approach has failed. This approach may result in more patients having multiple attempts at an LP. US guidance could be used as the initial method in patients predicted to have a higher failure rate with landmark-guided LP with spinal landmarks that are difficult or impossible to palpate.[112,125,126] These include patients in whom the landmarks are increasingly difficult to palpate as a result of being in the body mass index (BMI) categories of overweight (BMI 25 to 29.9) and obese (BMI ≥ 30).[111] Patients in whom an LP is not predicted to be difficult would have it attempted initially with the landmark palpation method, reserving US guidance for failures. The final approach uses US guidance initially for all patients, regardless of their BMI or the predicted ease of an LP.

US can be used to visualize the anatomic structures and mark the point of needle insertion. It can also be used to visualize the deep soft tissue structures, allowing the EP to estimate the depth that the needle needs to be inserted to enter the subarachnoid space. Using US to identify landmarks improves success rates, decreases the number of attempts, and decreases the number of traumatic LPs.[120,127-131] The use of US may decrease the hospitalization rate for traumatic or unsuccessful LPs.[132]

A high-frequency linear US transducer in the range of 5 to 10 MHz is the transducer of choice. The linear transducer may not allow visualization to the depth required in patients with a higher BMI. Use a lower frequency US transducer (e.g., a curvilinear array abdominal transducer in the range of 2 to 4 MHz). A pen or surgical skin marker is needed to mark the skin at the point of needle insertion. Surgical skin markers have the advantage of using scrub-resistant, nonsmearing ink.

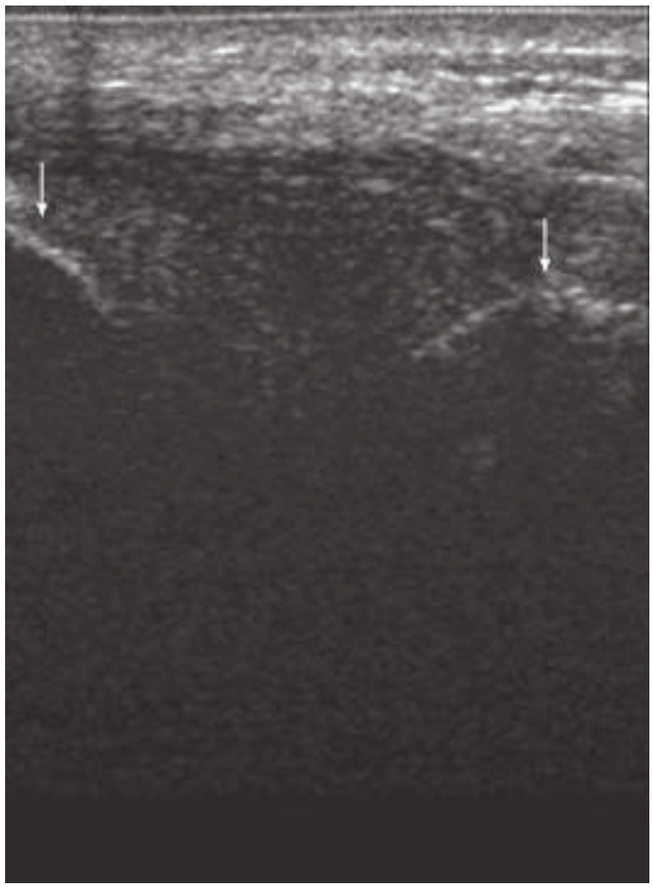
Transverse and longitudinal midline images centered over the lumbar spine are used.[111,112] There is a preference for the paramedian longitudinal view.[112] This view was felt to allow better visualization of deep soft tissue structures such as the ligamentum flavum and dura mater. The spinous process causes a shadow that extends from near the top to the bottom on the monitor screen in the transverse view (**Figure 142-10**).[113,125] The distal tips of the spinous processes are seen as a series of echogenic convexities in the longitudinal view (**Figure 142-11**).[117]

Place the patient into the position they will be in during the LP, whether the lateral decubitus position or sitting upright. It is important that the patient be in the exact position (e.g., with spinal flexion and their knees drawn up) that will be used during the LP. **Any change in patient position between the time of the US and the LP could change the location of the landmarks and decrease the likelihood of success.**

Use US to locate the entry site of the spinal needle. Use the transverse view at the level of the iliac crests and move the US transducer until the shadow caused by the spinous process is centered on the monitor screen (**Figure 142-10**). Use the skin marker to place a mark on each side of the transducer at its midpoint (**Figure 142-12**). The two lines are connected and will form a single line that marks the midline of the spine. Rotate the US transducer 90° to a midline longitudinal view. Move the US transducer until the tops of two adjacent spinous processes are seen with the gap between them located in the center of the screen (**Figure 142-11*A***). Again, make two marks on the patient's skin, one on each side of the US transducer at its midpoint (**Figure 142-13**). These two marks are connected to form a single transverse line that indicates the center of the gap between adjacent spinous processes. Color flow Doppler can be used to visualize the vascular structures and help decide the location of the LP.[118]

US can be used to measure the depth to the CSF and the depth of spinal needle insertion. This can guide the EP more than

FIGURE 142-10. US image of the lumbar spine in the transverse view. The spinous process (*arrow*) causes a shadow that extends from the top to the bottom of the monitor screen.



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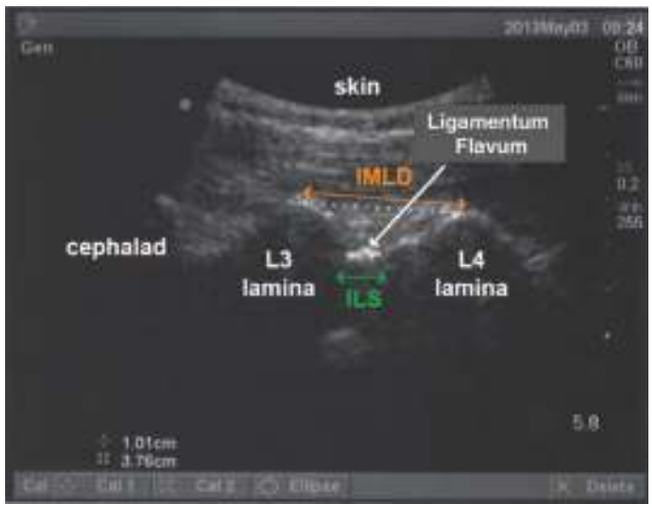

B

FIGURE 142-11. US image of the L3-L4 lumbar spine in the longitudinal view. ***A.*** Midline view. The distal tips of the spinous processes are seen as a series of echogenic convexities (arrows). ***B.*** Lateral view. IMLD, inter-middle laminar distance, ILS, interlaminar space. (Used with permission from reference 117.)

the equations. US answers the question of how deep to insert the spinal needle.

Set aside the US transducer. Connect the two pairs of skin marks to form two lines that intersect at a right angle (**Figure 142-14**). Their intersection marks the site of needle entry (**Figure 142-14**). Clean and prep the patient's skin and perform the LP as described previously.

RISK FACTORS FOR TRAUMATIC OR UNSUCCESSFUL LP

There are numerous risk factors for obtaining a traumatic or unsuccessful LP.[98,133] It is important to recognize these, as traumatic or unsuccessful attempts may cause diagnostic ambiguity and lead to unnecessary antibiotic use, hospitalization, and patient discomfort. Steps may be taken to minimize patient discomfort and maximize the chance of a successful LP in the setting of a high likelihood of a difficult LP. Have the most experienced EP perform the procedure. Consider the use of US or fluoroscopic guidance. **The EP should never delay administering antibiotics, or other potentially life-saving treatment, because an LP may appear difficult.**

The inability to either visualize or palpate the spinous processes is predictive of a difficult LP in the adult patient. The inability to visualize the spinous processes is associated with an increased risk of a traumatic LP.[133] The use of bedside US can assist in the identification of the spinous processes.

There are several risk factors in children associated with an unsuccessful or traumatic LP.[98] These include patient-related factors (e.g., young age and inability to visualize the spinous processes), physician factors (e.g., less experience performing an LP), and procedural factors (e.g., no local anesthetic use, patient movement, and advancement of the spinal needle with the stylet in place).[98]

FIGURE 142-12. The US transducer is oriented transversely across the lumbar spine. A mark is placed on each side of the US transducer's midpoint.

EPs have traditionally been taught to keep the stylet in place as the spinal needle is advanced into the subarachnoid space. This will avoid the introduction of epidermal cells into the subarachnoid space and the subsequent development of an epidermoid spinal canal tumor. The stylet-out technique has not been linked to this complication as long as the stylet is used as the needle penetrates the skin.[98] The greatest benefit of stylet removal appears to occur in young infants when the EP is less likely to feel the spinal needle penetrate the dura. The stylet-out technique allows for continuous monitoring of penetration into the subarachnoid space by direct visualization for CSF return.

AFTERCARE

Clean the excess povidone iodine or chlorhexidine from the patient's back and apply a dressing or bandage to the puncture site. Recumbent positioning will decrease the postural headache that sometimes follows LP, but it has not been shown to decrease the incidence of postdural puncture headache (PDPH).[134]

FIGURE 142-13. The US transducer is oriented longitudinally along lumbar spine. A mark is placed on each side of the US transducer's midpoint.

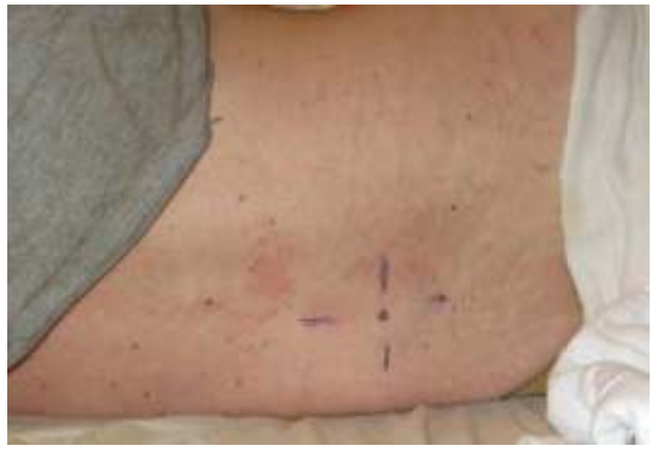

FIGURE 142-14. Two pairs of marks are obtained. Their intersection (purple dot) marks the spot for the spinal needle entry.

COMPLICATIONS

The use of proper technique is essential when performing an LP. It is important for the EP to be aware of potential complications, know how to recognize them, and know how to manage them. Refer to the article by Evans for a complete review of LP complications.[135] The most common complication is PDPH whereas the most life-threatening is cerebral herniation. Localized cellulitis, dural abscesses, discitis, and localized bleeding are also potential complications.

CEREBRAL HERNIATION

The most serious complication that may result from an LP is brain herniation or coning.[38] Theoretically, if a large pressure gradient exists between the cranial and lumbar compartments, herniation across the tentorial incisura or foramen magnum may occur after removal of CSF from the lumbar area. Patients with increased ICP secondary to intracranial mass lesions, cerebral edema, and acute hydrocephalus are at greater risk for cerebral herniation or coning. Herniation has also been known to occur in patients with meningitis. The role of LP in precipitating brain herniation has been the subject of debate. Several studies suggest that an LP is relatively safe in the patient with increased ICP.[136-138] The elevated ICP in IIH is uniform throughout the CNS, so there is no pressure gradient to result in brain herniation. Each individual patient's risks and benefits must be considered before proceeding.

There are case reports of herniation occurring after an LP in patients with meningitis and SAH.[139-141] The actual role that an LP has in precipitating or facilitating the process of herniation is not known.[139] **Patients with decorticate or decerebrate posture, focal neurologic signs, or no response to pain should receive antibiotics but not an LP.[139,141] This is true even when the CT appears normal in cases of suspected meningitis.[139,141] Obtain a CT scan before performing an LP if there is a suspicion for an SAH.[142,143]**

POSTDURAL PUNCTURE HEADACHE

The PDPH is the most common complication of an LP.[144,145] It is thought to be the result of continued CSF leakage at the puncture site. The reason why this causes a headache remains unclear.[146] It is hypothesized that the lower CSF pressure induced by the leakage causes the brain to "sag." This leads to traction on pain-sensitive structures in the brain such as the dura, nerves, and bridging veins.

Intracranial venous dilation and increased brain volume may lead to a neurohumoral response identified as pain.

The headache begins within 24 hours of the procedure in 65% of cases and within 48 hours in 90% of cases.[147] Delayed development of a PDPH 5 to 14 days after the procedure has been reported. The headache typically resolves within 7 days. It has been reported to last several months in rare individuals. The headache is usually located in the frontal or occipital area. It may vary in intensity. The PDPH is usually described as bilateral pressure that is throbbing or achy and improves with supine positioning. Associated symptoms may include nausea, vomiting, neck stiffness, auditory symptoms, and vestibular symptoms.[147]

The incidence of PDPH has been reported to be anywhere from 1% to 70%. The wide range is most likely due to the fact there are several identifiable risk factors that influence its development. Age and gender play a significant role.[147-149] The highest incidence occurs in 18 to 30 year olds. There seems to be a decreased incidence after the age of 60, the reason for which is unknown.

The type of needle and its diameter also influence the development of a PDPH.[59-63] This is based upon the amount of trauma and the size of the hole it makes in the dura. Smaller diameter needles and atraumatic needles lower the incidence of PDPH. However, a 22 or larger gauge needle must be used to determine the opening pressure and to collect samples in a timely fashion when performing a diagnostic LP.[150,151] The bevel orientation should be parallel to the longitudinal dural fibers when using a Quincke needle. This significantly reduces the incidence of PDPH.[95,152-154] The use of an atraumatic needle may also decrease the incidence of PDPH.[155,156] These needles are designed to separate rather than shear the dural fibers. Replacement of the stylet before removing the spinal needle has been shown to decrease the incidence of PDPH.[157] Repeated dural punctures have been associated with an increase in the incidence of PDPH.[158]

Other nonproven risk factors for a PDPH include psychogenic factors, the rapidity of CSF withdrawal, race, patient positioning, and hydration status. Bed rest for 24 hours was often widely recommended. It has not been shown to decrease the incidence of PDPH.[159-162] Other studies have shown bed rest to increase the risk of PDPH.[163,164] Early ambulation does not increase the incidence of PDPH.[165] Dehydration was once felt to impair the patient's ability to produce CSF to compensate for the leaking CSF. The incidence of PDPH was independent of daily fluid intake.[166] They postulated that it is the closure of the dural defect and not the CSF loss that is the critical factor in the termination of the PDPH.

Many treatments have been tried to relieve a PDPH. Supine positioning can provide some symptomatic relief for initial or mild PDPHs. A single 300 mg dose of oral caffeine may provide transient relief.[167,168] Administer 500 mg of intravenous caffeine for more severe headaches.[169] Intravenous caffeine administration resulted in an approximately 70% success rate in treating PDPH.[170] Intravenous caffeine administered prophylactically may minimize the incidence of PDPH.[171] There is some promise in the use of triptans.[172,173] The use of cosyntropin, an ACTH analog, has shown promise in the treatment of PDPH.[174,175] An occipital nerve block may resolve a PDPH.[176,177] The pain of a PDPH can be ceased with the administration of gabapentin, hydrocortisone, and theophylline.[168] A greater occipital nerve block can decrease the pain of a PDPH and prevent the more invasive blood patch or transnasal sphenopalatine block.[178] The epidural pumping of normal saline has shown some promise.[145] Transnasal sphenopalatine ganglion block can alleviate the pain.[179,180]

A more definitive but invasive treatment for the PDPH is the epidural blood patch (Chapter 143).[181-183] This procedure is typically performed by an Anesthesiologist. It involves injecting 10 to 20 mL of autologous blood into the epidural space at the level of the previous LP. The blood acts to tamponade any further CSF leakage and allows healing of the dural opening. Epidural blood patching is successful in 85% of patients after one injection and about 98% of patients if a second blood patch is required.[120-122,184-186] Epidural blood patching should be performed no sooner than 24 hours after the LP. Complications of the blood patch include back pain, paresthesias, radiculopathies, and weakness, all of which are transient. A rare complication is the spinal subdural hematoma.[187] Other modalities for PDPH relief that have been used but not widely studied are epidural saline injections, dextrose injections, gelatin injections, and epidural morphine.

INFECTIONS

Local infections including cellulitis, abscesses (e.g., lumbar epidural or spinal cord), and discitis can result from an LP. Performing an LP through an area with a local infection (i.e., a cellulitis or an abscess) can introduce bacteria into the CSF and lead to meningitis. Contamination of the needle by airborne pathogens can also occur. **Always wear a sterile gown and gloves, a mask, and a cap while performing an LP.**[83,188,189] It is possible for the EP's oral flora to contaminate the field and equipment, resulting in an iatrogenic meningitis.[83-85]

It was once thought that an LP could induce meningitis in a bacteremic patient. Further studies have shown this idea to be unfounded.[190-192] **Bacteremia is not a contraindication to performing an LP**. Proper cleaning and disinfecting of the skin, avoiding infected areas with the spinal needle, and using sterile technique will minimize but not eliminate any risk of infection.

HEMORRHAGE

A traumatic LP is a common occurrence and can occur in up to 30% of LPs.[193] Up to 72% of LPs have anywhere from 1 to over 50 red blood cells.[194] This is a common and usually uncomplicated occurrence in patients with a normal coagulation profile. Traumatic LPs can result in a spinal epidural or spinal subdural hematoma in patients regardless of coagulation abnormalities.[195-200] Epidural hematomas most likely result from needle trauma to the internal vertebral plexus or radicular vessels (**Figure 142-15**).[194]

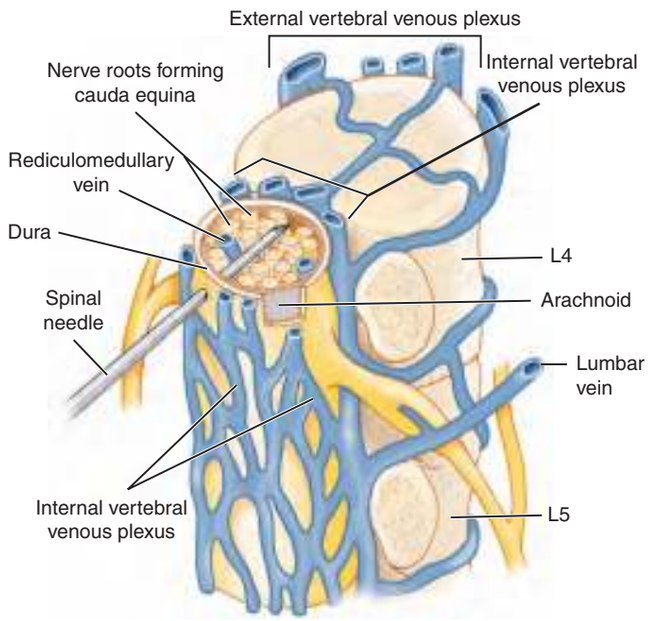

FIGURE 142-15. Illustration of the spinal cord and the potential sources of spinal needle-induced bleeding. The correct path of the spinal needle usually avoids the internal vertebral venous plexus.

The radicular vessels course down the length of each nerve root. It has been suggested that the bevel of the spinal needle can induce trauma and bleeding to these vessels much like they produce paresthesias when touching the nerve roots. Spinal epidural hematomas, if large enough, can result in a cauda equina syndrome.[201]

Subdural hematoma or SAH is a rare but catastrophic complication in patients with or without a coagulopathy.[199] One author recommended that the procedure be performed only if necessary in patients with a thrombocytopenia.[202] Platelets should be transfused prior to an LP in patients with platelet counts less than 20,000 or if platelet counts are dropping rapidly. The most skilled EP should perform the LP using a 22 gauge or smaller needle.[202] The patient should be observed after the LP for the development of neurologic signs suggesting a hematoma (e.g., lower extremity weakness, sensory deficits, or incontinence).

A rare bleeding complication is an intracranial subdural hematoma.[203,204] This may result from the same mechanism causing a PDPH, namely the downward displacement of the brain from decreased CSF volume and persistent leakage after an LP. This may occasionally cause tearing of the bridging veins and lead to a unilateral or bilateral subdural hematoma. Suspect this diagnosis when a headache sounding like a PDPH lasts for more than a week, is no longer postural in nature, or returns after initially improving.[205]

MISCELLANEOUS COMPLICATIONS

Neuropathies involving cranial nerves III, IV, V, VI, VII, and VIII have been reported. They most likely result from traction on the nerves caused by low ICP after the LP. Typical complaints may include visual and auditory symptoms. Epidural fluid collections of CSF after an LP can be significant.[206] They usually resolve spontaneously with time but can compromise the thecal sac if large enough.

Mild low back pain is a common complaint that results from the local trauma of the needle tract. Transient dysesthesias are common and result from spinal needle contact with the nerve roots. A spinal needle that passes beyond the subarachnoid space into the annulus fibrosis can cause disc herniation. This can result in a discitis and vertebral collapse.[207]

Intraspinal epidermoid tumors are composed of well-differentiated stratified squamous epithelium surrounding a mass of caseous substance formed by the desquamation of epidermal tissue. They are often congenital but may result from the introduction of epidermal fragments into the spinal canal. This may occur if the stylet of the needle is not used. Spinal epidermoid tumors may present months to years after an LP.[208-211]

A dry tap is often the result of lateral displacement of the spinal needle. Maintain the spinal needle in the midline while it is being advanced. Not penetrating deep enough with the spinal needle can result in a dry tap. This is especially true in obese patients who may require long (i.e., 7 to 10 inch) spinal needles to gain access to the subarachnoid space. It has been suggested that the standard 3.5 inch spinal needle is adequate for 97% of patients.[212]

Mechanical complications with the equipment do occur. The spinal needle can break.[213] A bent stylet cannot be pulled out of the spinal needle.[86] Check the equipment before using it.

Prevent secondary injury by always inserting the stylet into the spinal needle before removing it.[99] Withdrawing the spinal needle without the stylet can result in the aspiration of a lumbar nerve root or arachnoid tissue into the epidural space. The patient may require a laminectomy if this occurs to replace the nerve root or arachnoid tissue. The reinsertion of the stylet will also decrease the incidence of post-LP symptoms (i.e., dizziness, headache, nausea, and tinnitus).[99]

The ICP can be falsely elevated. **Positioning the patient is important when measuring the opening pressure.** Place the patient in the lateral decubitus position and ensure that they relax during the procedure. Do not flex or extend the patient's neck, hip, or knee. Try not to have the patient Valsalva during the procedure. Carefully instruct the patient from the fetal position for needle placement to the lateral decubitus position to measure opening pressure.

It is now common for adolescents and adults to have tattoos on their lower back. It is recommended to perform an LP in an area void of tattoo ink, at a higher or lower interspace, or nicking the skin with a needle or scalpel prior to inserting the spinal needle.[214,215] All these techniques avoid the spinal needle penetrating through the tattoo ink. The tattoo ink may contain substances that can be irritating or toxic if introduced into the spinal canal.

CSF INTERPRETATION

Proper interpretation of the CSF is an important skill for the EP who performs the LP. **Tables 142-1 and 142-2** list the normal CSF values and the CSF values in a variety of different medical conditions.[216,217]

CSF PRESSURE

Normal CSF pressure ranges from 70 to 180 mmH_2O in adults and from 50 to 80 mmH_2O in infants and children. Note that many manometer kits use cmH_2O on the demarcations, whereas results are commonly interpreted in mmH_2O. Elevated CSF pressure may be seen in bacterial meningitis, viral meningitis, brain abscesses, tuberculous meningitis, fungal meningitis, encephalitis, meningeal carcinomatosis, SAH, pseudotumor cerebri, and Guillain-Barré syndrome. It may be falsely elevated when the patient is tense or creating a lot of intraabdominal pressure by flexing their knees into the abdomen. The pressure will also be falsely elevated if the patient is in a seated position. Although CSF pressure is not routinely recorded in infants and children (most likely because they are often crying, struggling, or difficult to hold), it should be recorded whenever possible. Low CSF pressure may be the result of a spinal root obstructing the flow of CSF into the needle or obstruction of flow from a spinal mass.

TABLE 142-1 Normal CSF Values

	Preterm infant	Term infant	Child	Adult
White blood cell count	9 (range 0–32)	8 (range 0–22)	0–7	0–5
(WBC/mm^3)	57% PMNs	61% PMNs	0% PMNs	0% PMNs
Glucose (mg/dL)	24–63 (mean 50)	34–119 (mean 52)	40–80	50–80
CSF/blood glucose ratio				
Normal ratio	55–105%	44–128%	50%	60–70%
Abnormal ratio	< 0.5–0.6	< 0.5–0.6	< 0.4–0.5	< 0.4–0.5
Protein (mg/dL)	65–150 (mean 115)	20–170 (mean 90)	5–40	15–45

TABLE 142-2 CSF Values in Various Neurologic Conditions

Condition	Appearance	Pressure	Cell count (mm^3)	Glucose (mg/dL)	Protein (mg/dL)
Bacterial meningitis	Clear, cloudy, or purulent	Elevated	500–10,000 + cells with 90–95% PMNs	0–40	> 50
Partially treated bacterial meningitis	Possibly cloudy	Normal or elevated	1–500 cells, lymphs or monos may predominate	Low or normal	> 50 and < 500
Brain abscess	Clear, cloudy, or purulent	Elevated	Possibly > 100,000 cells if abscess ruptures. PMNs predominate.	Normal	< 200
Tuberculous meningitis	Clear, opalescent, or ground glass	Elevated	25–500 WBCs, PMNs early but usually lymphs predominate	10–40	50–500
Fungal meningitis	Clear or cloudy	Elevated	10–500 WBCs, lymphs predominate. PMNs early.	< 40	< 600
Viral meningitis or encephalitis	Clear, may have faint opalescence	Normal or elevated	6–1000 cells, predominance of lymphs. PMNs early.	Normal but may be low with herpes or mumps	< 200
Acute syphilitic meningitis	Clear or turbid	Elevated	100–500 WBCs, usually lymphs	Normal or decreased	< 200
Meningeal carcinomatosis	Clear or mucinous	Elevated	10–500 WBCs, lymphs predominate	< 40	< 500
Subarachnoid hemorrhage	Bloody, xanthochromia, clear	Elevated	1000–3.5 × 10^6 RBCs	Normal, but can be decreased in 10–15% of cases	Increased
Multiple sclerosis	Clear	Normal	0–20 lymphocytes, > 50 rare	Normal	45–75
Progressive multifocal leukoencephalopathy	Clear	Normal	< 10 monos	Normal	Normal
Guillain-Barré syndrome	Clear or xanthochromic	Normal or elevated	Normal, but 10–200 WBCs, predominantly lymphs	Normal	May be as high as 1000
Pseudotumor cerebri	Clear	Elevated	Normal	Normal	Normal
Subacute sclerosing panencephalitis	Clear	Normal	Usually normal	Normal	Increased; check CSF measles titers and CSF gamma-globulin
Neuro-Behçet's syndrome	Clear	Normal or elevated	Up to 3000 WBCs, PMNs predominate	Normal	Increased

A novel device to measure pressure is the Compass LP (Centurion Medical Products, Williamston, MI). This device is a single patient use and disposable unit that attaches to the spinal needle (**Figure 142-16**). It provides a digital readout of the CSF pressure. Further research is required before this device replaces the standard manometer.

FIGURE 142-16. The Compass LP Pressure Monitor. (Photo courtesy of Centurion Medical Products.)

CELL COUNTS AND DIFFERENTIAL

A variable amount of white blood cells (WBCs) may be normally present in the CSF depending upon the age of the patient.[218] Neonates may have up to 32 WBCs/mm^3 with 60% polymorphonuclear leukocytes (PMNs). Infants 4 to 8 weeks of age may have up to 22 WBCs/mm^3. Most sources consider anything greater than 8 to 10 WBCs/mm^3 to be abnormal.[219,220] Normal adult CSF should contain no more than 5 WBCs/mm^3 with a differential consisting predominately of mononuclear cells or lymphocytes. The presence of more than one PMN should be considered abnormal.

Bacterial meningitis cell counts are usually greater than 500 WBCs/mm^3 with a predominance of PMNs; though lymphocytosis can uncommonly occur.[221,222] The CSF will usually contain less than 1000 WBCs/mm^3 in patients with viral meningitis and have a differential of 100% lymphocytes. An early viral meningitis (i.e., 48 hours) will have a predominance of PMNs in the CSF, making it difficult to distinguish it from bacterial meningitis.[1,223,224] Approximately 90% of patients will show a switch to a mononuclear pleocytosis within 8 to 12 hours on repeat LP.[225]

Normal cell counts and differentials do not always exclude meningitis. Approximately 95% of the population does not normally have any PMNs in their CSF. The presence of one PMN could represent an abnormality. One PMN may be seen in approximately 5% of normal children. Bonadio reviewed 424 LPs of which 106 had PMNs but no pleocytosis.[226] All 106 patients had negative Gram's stains and cultures. The authors concluded that the older child without pleocytosis or abnormal CSF chemistries can be considered at very low risk for meningitis. Neither the presence of bands nor the quantity of bands was a predictor of bacterial meningitis.[227] Close clinical observation and hospitalization for treatment and repeat LP should be considered until CSF culture results are negative if meningitis is suspected and the CSF is normal or has PMNs.

A traumatic LP can often make the interpretation of the CSF difficult as peripheral WBCs can be introduced into the CSF. Clearing of the red hue of the CSF from the first to last tube suggests that the tap was traumatic. This is not always a reliable sign. The ratio of the RBCs to the WBCs in the blood is compared to the ratio in the CSF. This is based on the assumption that blood introduced into the CSF keeps the ratio of RBCs to WBCs the same. Some use a set RBC to WBC ratio of 1000:1, 750:1, or 500:1.[228] This is not always accurate as a peripheral leukocytosis may often be present. By comparing the ratios, a predicted WBC count for the CSF can be obtained (predicted CSF WBC = CSF RBC × blood WBC ÷ blood RBC). The actual WBC count will then be the predicted WBC subtracted from the observed or measured WBC (actual CSF WBC = observed CSF WBC − predicted CSF WBC).

Most studies on patients with traumatic taps that did not have meningitis have shown that these formulae are often inaccurate.[229] The observed CSF WBC count is often less than the predicted CSF WBC count.[230] This raises concerns that the diagnosis can be missed in the presence of meningitis. The use of the formula in patients who had meningitis found many false-positive and false-negative results.[231] They investigated the value of an O:P ratio (observed CSF WBC/predicted CSF WBC) in predicting the presence of meningitis. They found that a ratio greater than 10 had a sensitivity of 88% and a specificity of 90% in predicting culture-positive meningitis. **They concluded, along with others, that pleocytosis in bacterial meningitis is rarely masked by a traumatic tap.**[231,232] The O:P ratio of ≤ 0.01 can be used to identify patients with traumatic LPs who do not have meningitis.[233]

GLUCOSE

Normal values for CSF glucose are listed in **Table 142-1**. Compare the ratio of CSF glucose to a simultaneously determined blood glucose level to determine if low CSF glucose (hypoglycorrhachia) exists.[224] The ratio is abnormal in preterm infants if it is lower than 0.5 to 0.6.[235] A ratio of less than 0.4 to 0.5 in children and adults is abnormal. Approximately 58% of patients with bacterial meningitis will have a glucose of < 40 mg/dL. The sensitivity for detecting bacterial meningitis increases to about 70% if a CSF-to-serum glucose ratio of < 0.31 is used.[236] The normal steady state of 0.6 tends to decrease as serum glucose increases. Ratios of less than 0.3 should be considered abnormal in cases of severe hyperglycemia. The CSF-to-serum glucose ratio is less accurate when there are rapid changes in the serum glucose. **A low CSF-to-serum glucose ratio should always raise the concern of bacterial or fungal meningitis.** Other conditions such as tuberculous or syphilitic meningitis, meningeal carcinomatosis, or SAH can be the etiology. Approximately 15% to 20% of patients with an SAH will have hypoglycorrhachia.[237,238] Normal CSF-to-serum glucose ratios are usually seen with aseptic meningitis, encephalitis, brain abscesses, and subdural empyemas.

PROTEIN

The normal CSF protein levels are listed in **Table 142-1**. Elevated CSF protein levels, often greater than 150 mg/dL, are seen in acute bacterial meningitis. Other causes of increased CSF protein include any type of meningitis, encephalitis, CNS tumors, SAH, demyelinating syndromes, and a traumatic LP.[239] Correct the CSF protein by subtracting 1.1 mg/dL of protein for each 1000 RBCs in traumatic LPs.[240]

GRAM'S STAIN

The Gram's stain is a very reliable test when performed by properly trained individuals. It is positive in identifying approximately 80% of bacterial CNS infections.[241] The probability of detecting bacteria on a Gram's stain depends upon the number of bacteria present in the CSF.[242] Approximately 25% of smears are positive with ≤ 10^3 colony-forming units (CFU)/mL, 60% with 10^3 to 10^5 CFU/mL, and 97% with > 10^5 CFU/mL. False negatives can result from partially treated meningitis where the sensitivity decreases to about 60%.[243] False positives can result from the use of contaminated LP trays, contaminated reagents, or the use of an unoccluded spinal needle.[244]

CSF CULTURES

Obtain CSF cultures in all patients suspected of having meningitis.[4] Positive cultures are assumed to be 100% specific but may only occur in 80% of patients thought to have bacterial meningitis.[218] Transport the CSF specimens to the laboratory promptly, as *H. influenzae* and meningococcus will not survive storage or variations in temperature. A small number of patients treated for meningitis with antibiotics have a final diagnosis of bacterial meningitis.[245,246] The decreased prevalence of bacterial meningitis is due to vaccination.[245,246] Antibiotics administered prior to LP can sterilize the CSF. There is probably a window of about 2 to 3 hours where antibiotics do not affect the culture results.[247] This depends upon the amount of bacteria in the CSF and the elapsed time from initiation of antibiotics. The percentage of positive CSF cultures decreases from 33% to 4% and Gram's stains from 41% to 7% when antibiotics are given prior to LP.[248] The effect of full intravenous antibiotic treatment on CSF cultures was studied by performing an initial LP and then a repeat LP in 44 to 66 hours.[249] All but one of the cultures became negative, whereas the cytology and biochemistry were not affected. They concluded that partial treatment with antibiotics may alter the culture results but does not distort the other characteristics of a "bacterial" CSF.

SUBARACHNOID HEMORRHAGE

It is imperative to interpret the CSF results correctly when an LP is performed after a negative head CT to rule out the possibility of an SAH.[250] Most sources agree that the presence of xanthochromia, which results from lysis of red blood cells, confirms the presence of intracranial bleeding.[251-253] Xanthochromia by spectrophotometry was found to be only slightly better than visual inspections.[254] Xanthochromia, when measured by spectrophotometry, has a sensitivity that approaches 100% when performed between 12 hours and 2 weeks from the initial SAH. Do not accept gross xanthochromia as a positive finding.[255-257] Gross xanthochromia can be a false-positive result. Always rely on a spectrophotometric determination of xanthochromia.[258,259] Xanthochromia can develop in specimens within a few hours if the tap was traumatic.[260] One cannot rely on the finding of xanthochromia if the WBC count is > 10,000 or if the analysis of CSF samples is delayed.[260]

So how should patients who present within 12 hours of their symptom onset be managed? Delaying an LP for 12 hours would require holding patients in the ED or admitting everyone who required an LP for evaluation. This presents a legitimate logistical problem. Edlow and Caplan suggest that patients that have a negative CT should undergo immediate LP.[261] If the CSF is persistently bloody without xanthochromia and clinical suspicion is high, vascular imaging should be the next step.[20]

FUTURE CONSIDERATIONS

The Injeq System (Injeq Oy, Tampere, Finland) may make LPs easier. The system consists of the specialized spinal needle, the cable, and the monitor (**Figure 142-17**).[262] The needles come in a variety of sizes. The needle has an electrode at the tip of the stylette that senses the

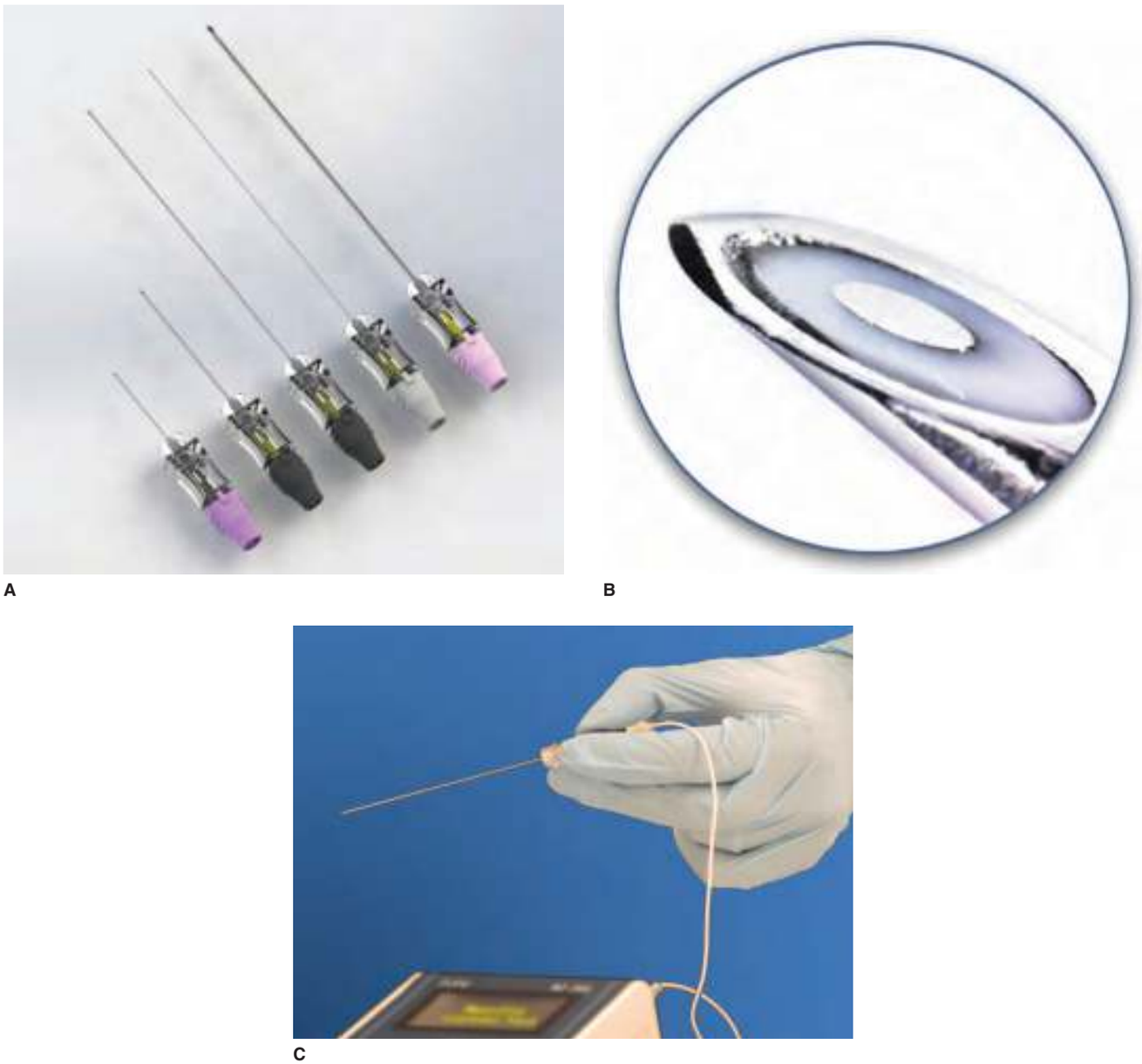

FIGURE 142-17. The Injeq System. ***A.*** The IQ spinal needles. ***B.*** The sensor on the stylette tip inside the spinal needle. ***C.*** The spinal needle attached to its cable and monitoring unit. (Photos courtesy of Injeq Oy.)

environment of the needle tip. The sensor uses bioimpedance analysis to differentiate tissue types. The sensor measurement is different for CSF versus other tissues. The company is hoping to get U.S. FDA approval in 2018.

SUMMARY

Most LPs will be performed in suspected cases of meningitis or SAH after a negative head CT. The risks of performing an LP need to be weighed against the potential benefits of diagnosing these two potentially life-threatening illnesses promptly. Knowledge of the proper indications, contraindications, technique, and interpretation of the CSF findings will undoubtedly help the EP to minimize the complications that can be associated with the procedure. Although most complications are rare, awareness of their existence, presentation, and proper treatment is imperative.

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143 Blood Patching for Postdural Puncture (Lumbar Puncture) Headache

Gunar Subieta, Maria Torres, and Ned Nasr

INTRODUCTION

The epidural blood patch (EBP) has been used for the effective relief of postdural puncture headache (PDPH) for well over 50 years.[1] It continues to be the gold standard for therapy of this debilitating condition.[2-4] The advent of spinal and epidural anesthesia during the early twentieth century required techniques for treating PDPH. The risk of PDPH was recognized from a dural puncture. Gormley was the first to describe EBP as a therapy for PDPH in 1960.[5] EBP was not widely used until nearly a decade later.[6]

TABLE 143-1 The International Headache Society Criteria for PDPH[7]

Dural puncture was performed
Headache associated with at least one of the following:
Hyperacusis
Nausea
Neck stiffness
Photophobia
Tinnitus
Headache develops within 5 days of dural puncture
Headache improves within 15 minutes after lying
Headache resolves either:
Spontaneously within 1 week
Within 48 hours of EBP
Headache worsens within 15 minutes after sitting or standing

The International Headache Society classifies the PDPH as an instance in which the criteria for low cerebrospinal fluid (CSF) pressure is met without any other possible explanation (**Table 143-1**).[7] It can and does occur after a lumber puncture.[8] The patient presents with a bilateral positional headache aggravated by sitting or standing. The headache is better or relieved by the recumbent position. It is associated with at least one typical symptom (e.g., nausea) and an atypical symptom (e.g., visual and/or auditory disturbances). The onset can occur at any time within 5 days of any procedure involving the puncture of the dural membrane (i.e., PDPH).

A spontaneous intrathecal CSF leak is uncommon. It occurs in 0.05% of the population. A spontaneous leak is usually associated with congenital structural abnormalities or connective tissue diseases (e.g., Marfan's syndrome or Ehlers-Danlos syndrome).

A breach of the dural membrane may be intentional or accidental depending on the circumstances. The highest frequency of accidental punctures occurs in young females during the perioperative delivery period in conjunction with an epidural placement. Ruptures associated with diagnostic procedures may present with headache and are usually seen in pediatric or geriatric patients.[9]

ANATOMY AND PATHOPHYSIOLOGY

The dural sac creates a fluid-filled space that surrounds the brain, is continuous throughout the spine, and ends at the level of the midsacrum. This space contains approximately 150 mL of CSF. The CSF is constantly secreted and recycled by reabsorption. More detailed information regarding the CSF can be found in Chapter 142.

PDPH development is still a subject of discussion in terms of its etiology.[10] There are many theories for the PDPH. One theory is the loss of CSF at a higher rate than it is produced.[11] This results in a reflex vasodilation and an increased cerebral blood flow to fill the gap left by this loss and venous congestion.[11] The body responds by trying to maintain a constant intracerebral volume. This theory may explain why vasoconstrictors such as caffeine and theophylline have a role in the relief of PDPH. The EBP is thought to work by forming a clot over the dural tear that is causing the CSF leakage and by increasing intracranial pressure via a mass effect. The EBP relieves tension on cerebral bridging veins and subsequently reduces the cerebral vasodilation. Another theory suggests that the loss of the CSF cushion of the brain results in traction of the meninges, cranial nerves, and cervical structures.[12] It is hypothesized that traction on the nerves is responsible for the symptoms of a PDPH. Traction on the trigeminal nerve causes a frontal headache. Traction on the abducens nerve causes visual symptoms. Traction on the vagus nerve causes nausea. Traction on the cervical nerves may

cause referred pain in the occipital nerve distribution, the neck, and the shoulders. The patient will present with symptoms based on the nerve or nerves under traction.

The type of needle affects the outcome (Chapter 142). Pencil needles are more traumatic to the dura and may result in a higher inflammatory response, decreasing the amount of leakage. The larger the caliber of the needle, the higher is the likelihood of a CSF leak. Many attribute worse symptoms with larger punctures.[13]

The incidence of PDPH has been associated with age between 18 and 30 years, female sex, low body mass index, prior history of headaches, and prior motion sickness.[4,14] Patients with a prior history of migraines are not at a higher risk than other populations.[15] Obesity is protective and prevents a PDPH.[16,17]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for a PDPH is broad. A PDPH may present with a frontal to occipital headache occurring within 12 hours of a procedure and up to 5 days after (**Table 143-1**). It is usually accompanied by symptoms of nausea, vomiting, and neck stiffness. Unusual symptoms may include visual and auditory disturbances. These include diplopia, hyperacusis, photophobia, and tinnitus. **Always consider the timing of symptoms.**

The history and physical examination are paramount. They must be comprehensive since the differential diagnosis includes the possibility of an abscess, hematoma, hypertensive emergency, pneumocephalus, postpartum cerebral angiopathy, preeclampsia, progression of an intracranial lesion, subarachnoid hemorrhage, or a thrombosis.[18,19] Low back pain makes the differential diagnosis include arachnoiditis, aseptic meningitis, myofascial pain, neurologic symptoms from local anesthetics, or septic meningitis.[20] The presenting patient without associated symptoms raises the possibility of primary headaches (e.g., classic migraine, common migraine, or tension headache) or secondary headaches (e.g., cervicogenic headache, giant cell arteritis, or medication overuse headache).[21] **Always be cognizant of red flags such as altered mental status, scalp tenderness, fever, history of cancer, neurologic deficits, and recent travel.** Obtain a complete lab work and imaging studies as warranted.[22]

Approximately 70% of PDPHs spontaneously resolve within 2 weeks. Some PDPHs may take up to months or years to resolve.[23,24] Surgical intervention may be required to relieve the PDPH in a minority of patients.[25]

INDICATIONS

Anesthesiologists and Pain Management Physicians recommend aggressive hydration, caffeine supplementation, analgesia with nonsteroidal anti-inflammatory drugs (NSAIDs), and opiates as first-line treatments.[2,3,8,26-28] These have been proven to be partly efficacious in multiple randomized controlled studies.[29] Additional interventional therapies include peripheral nerve blocks at the level of the sphenopalatine ganglion and the occipital nerve.[28,30,31] Their mechanisms are not well understood. Promising medical management strategies include cosyntropin (i.e., synthetic adrenocorticotropic hormone) with the belief that this hormone will increase CSF production.[28,32,33] Receptor agonists (e.g., sumatriptan) have been used with mixed results.[28,34,35]

The autologous EBP has become the standard of care for the management of persistent symptoms.[3,36,37] It was first described in 1960 to improve PDPH symptoms.[5] Multiple studies have demonstrated that the volume ideally is 15 to 20 mL.[38] The mechanism of action is thought to be due to immediate compression of the space causing cephalic displacement of the fluid, delayed clot formation at the level of the puncture site, and fibroblastic activity at the level of the puncture site.[39] It has been shown to be effective if performed sooner for the treatment of a PDPH. The EBP may reduce the patient's length of stay and improve satisfaction. A Cochrane review performed in 2010 does not advocate for a prophylactic EBP on every dural puncture.[40]

CONTRAINDICATIONS

The presence of an active infection at the site of epidural needle placement, bacteremia, presumed meningitis, and sepsis are absolute contraindications to performing an EBP.[41] The headache pain in these cases may have an etiology other than the PDPH. Discuss treatment with an EBP with patients with cancer or immunodeficiency syndromes and their Oncologist or Primary Care Provider before proceeding.

EQUIPMENT

- Angiocatheter, 20 gauge or larger
- Chlorhexidine or povidone iodine swabs
- Tuohy needle, usually 18 gauge
- Needles, various sizes
- 1% lidocaine
- 0.9% saline
- Syringes, 5 and 10 mL
- 5 mL glass or plastic syringe for loss of resistance (LOR)
- 10 mL ampule of 0.9% sterile saline to be used in syringe for LOR
- Sterile adhesive drape
- Sterile gauze 4×4s
- Surgical cap for the patient and providers
- Surgical masks and sterile gloves
- Butterfly needle(s) for blood draw and tourniquet
- Fluoroscopic unit, optional

The Touhy needle was designed to enter through the dura with minimal trauma. The Touhy needle comes in epidural kits and separately (**Figure 143-1*A***). The tip is curved (**Figure 143-1*B***). The shaft has markings every 1 cm (**Figure 143-1*A***). The stylet fits within the Touhy needle and functions to close off the inside (**Figure 143-1*C***).

The sterile epidural catheter kit contains all the required equipment (**Figure 143-2**). This includes the local anesthetic, skin antiseptic, and drapes. It is wise to have additional 5 and 10 mL sterile syringes.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Positioning is a crucial step in successfully accessing the epidural space. Place the patient in a sitting position on the level gurney with their arms and shoulders relaxed (**Figure 143-3**). The legs may be crossed or extended with both legs in the same position; ensure the patient is comfortable. Ensure the patient's spine is perpendicular to the floor. Minimize the distance from the edge of the

A

B

C

FIGURE 143-1. The Touhy needle. ***A.*** The needle. ***B.*** Two views of the needle tip. ***C.*** The stylet in the needle.

FIGURE 143-2. An example of a prepackaged commercial epidural kit.

bed to the patient's back. Ask the patient to push their lower back toward the edge of the bed while tucking their head into their chest to assist in proper positioning. The EBP may be attempted with the patient lying in the lateral position, but identification of the appropriate structures may be more challenging.

Carefully inspect the prior puncture site and surrounding tissues. The prior puncture site can be determined in most cases. **The appropriate location for needle insertion should be at the same level.** Confirm the insertion site of the needle by gentle palpation of the posterior iliac crests and the corresponding interspinous space at the same level. This is usually L3-L4. Bruising and tenderness to palpation at the previous puncture level are common. **Any erythema, purulence, and signs of infection are absolute contraindications to proceeding with an EBP.**

FIGURE 143-3. Positioning of the patient.

Don a cap, face mask, sterile gown, and sterile gloves. Have the patient apply a cap and a mask. Move the patient's hair under the cap and out of the way. Have an assistant wipe, clean the skin of the patient's back to remove any debris, dirt, and dried blood. Apply the chlorhexidine or povidone iodine to the area and allow it to dry.

TECHNIQUE

Apply sterile drapes to demark a sterile field. Anesthetize the skin at the puncture site with 1% lidocaine. Insert the Tuohy needle with its introducing stylet. Advance the Touhy needle slowly until the supraspinous ligament is encountered. Firmly hold the position of the Touhy needle so it does not move. Remove the stylet. Attach a sterile syringe with sterile saline to the Tuohy needle. Advance the Tuohy needle with constant or intermittent pressure on the syringe plunger. The Touhy needle will pierce and pass through the interspinous ligament and then the ligamentum flavum. A loss of resistance will be felt upon passing through the ligamentum flavum from pressure on the syringe plunger. Saline should be able to be injected freely.

A second provider should be obtaining the blood to inject through the Tuohy needle for the EBP while the first provider is obtaining access to the epidural space. The hand, forearm, and antecubital fossa are the easiest locations for access. **Strict attention to sterile technique is essential. The second provider should use universal precautions (i.e., a cap, a mask, a sterile gown, sterile gloves) and drapes.** Clean the patient's arm of any dirt and debris. Apply chlorhexidine or povidone iodine and allow it to dry. Apply sterile drapes to demarcate a sterile field. Use a large-bore butterfly needle or a standard angiocatheter. Obtain at least 20 mL of blood divided into 10 mL aliquots in syringes. **Ensure the syringes used to collect the blood remain sterile throughout the process.** The sterile syringes with the patient's blood will be given to the provider obtaining epidural access when ready. **Do not obtain the blood too soon or it may clot in the syringes before it is injected into the patient's back.**

Hand the sterile syringes with the patient's blood to the practitioner who accessed the epidural space. Attach the sterile syringe with the collected blood to the Tuohy needle. **Inject the blood into the epidural space slowly and in 5 mL increments (Figure 143-4).** The patient commonly feels increased pressure and tightness at the injection site. There is no set agreed upon amount of blood that needs to be given for relief of a PDPH. The range of 10 to 20 mL is usually sufficient.[41,42]

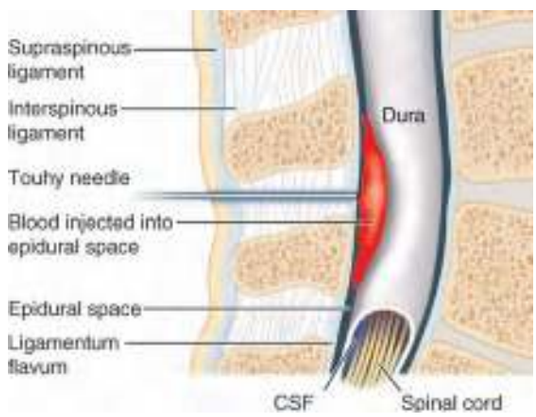

FIGURE 143-4. The epidural blood patch.

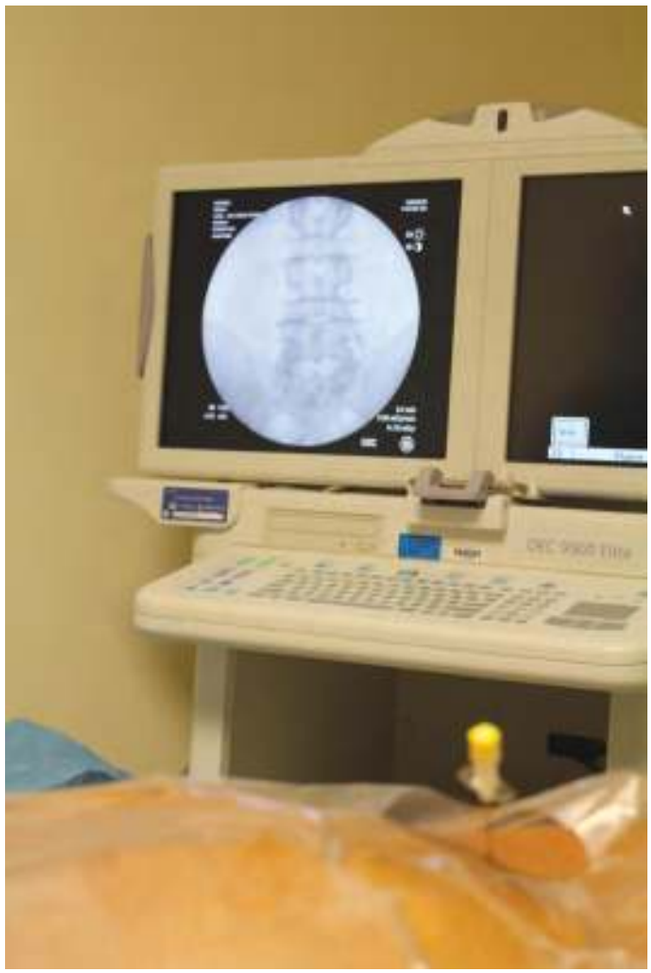

FIGURE 143-5. Place the fluoroscopy unit so the screen is visible while inserting the Touhy needle.

Remove the Touhy needle after the blood has been injected. Hold pressure to the injection site. Apply a bandage or a small sterile dressing.

FLUOROSCOPY GUIDANCE

The use of fluoroscopy with the EBP procedure can increase the effectiveness when compared to the blind insertion technique.[43] Position the fluoroscopy unit so that the monitor can be easily seen (**Figure 143-5**). Position the foot pedal in a convenient location to be stepped upon to activate the fluoroscopy when required. Position the patient, clean and prep the skin, and anesthetize the puncture area as described above. Insert the needle under fluoroscopy until the tip is in the epidural space (**Figure 143-6**). Some physicians inject 9 to 10 mL of autologous blood mixed with 1 mL of contrast material (**Figure 143-6**). The allows visualization of the blood entering the epidural space and ensures that it is being injected into the proper location.[43]

AFTERCARE

It is recommended for patients to lay flat for at least 2 hours after the EBP with intravenous fluid running at a maintenance rate. Instruct the patient upon discharge not to do any heavy lifting, straining, or strenuous activity. Increase oral fluid intake for at least 24 hours. Arrange appropriate follow-up. The patient should

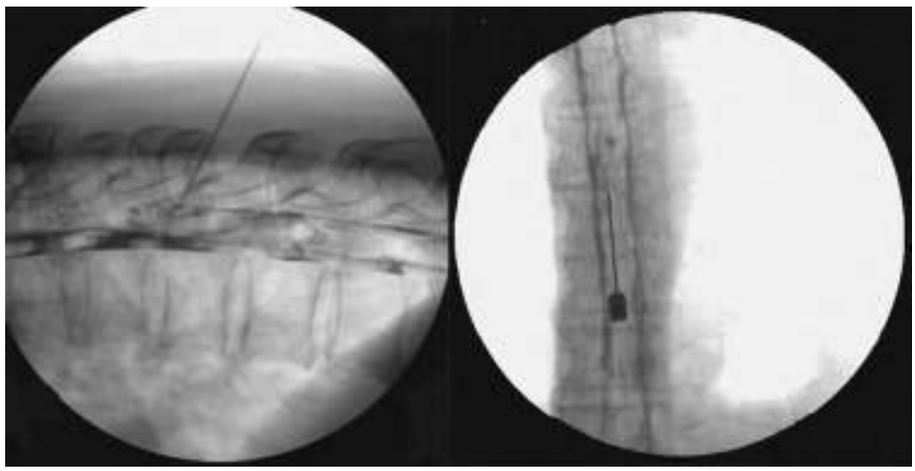

FIGURE 143-6. Fluoroscopy images. Positioning of the Touhy needle (*right*). Injection of autologous blood and contrast (*left*).

return to the Emergency Department immediately if they experience bowel or bladder dysfunction, erythema or tenderness at the injection site, fever, headache with worsening intensity, neck stiffness, numbness or weakness in the lower extremities, progressively worsening photophobia, or worsening nausea and vomiting.

Severe headaches may take up to 24 hours to resolve after an EBP. A second EBP may be necessary in some cases. Over 75% of patients with a PDPH attain complete resolution of symptoms after a single EBP. Over 95% of the remaining patients had complete resolution of symptoms after a second EBP.

COMPLICATIONS

The complications associated with an EBP are similar to those of lumbar puncture (Chapter 142) and epidural catheter placement. There is the additional complication of possibly contaminated blood injected into the epidural or intrathecal space. EBP is commonly avoided in febrile or septic patients as the risks of developing meningitis are quite real. A low-grade fever of a known cause being appropriately treated is not an absolute contraindication for an EBP in the presence of a PDPH. Neurologic complications can be related to compression (e.g., subdural hematoma, cauda equina syndrome, and lumbovertebral syndrome).[44] These can usually be seen with larger blood volumes injected. Back pain is the most common complication and usually resolves after 48 hours. The back pain can last up to a month in some cases. Other symptoms may include neck pain, lower extremity radicular pain, and temperature elevation. Inflammatory acute arachnoiditis is a rare complication that may be seen within the week following an EBP. The symptoms consist of severe radicular back pain and magnetic resonance imaging diagnosis of adhesions between the lumbar nerve roots.

SUMMARY

An EBP is a consistently reliable means of alleviating a PDPH in patients who have failed conservative management of their symptoms. An EBP is a relatively low-risk procedure when done properly, using sterile technique, and appropriate patient selection. It should be offered to patients presenting with a PDPH. Appropriate diagnosis of a PDPH is essential. An EBP in the setting of meningitis or other neurologic syndromes may be deleterious to the patient.

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144 Burr Holes

Caleb P. Canders, Noah T. Sugerman, and Amir Rouhani

INTRODUCTION

Skulls with burr holes have been found dating back 10,000 years.[1] One of the oldest written records of a burr hole placement was between 2000 and 1700 BC.[2] The ancient history of burr holes is well described.[1-4] A surgical algorithm included the placement of burr holes in the Continental Army.[4]

Patients with a closed head injury and expanding intracranial hematomas require urgent decompression and control of hemorrhage. Burr hole drainage of epidural and subdural hematomas is uncommonly performed in the Emergency Department but can be lifesaving when timely neurosurgical intervention is not possible. **Burr hole drainage is indicated in patients with suspected increased intracranial pressure and evidence of tentorial herniation or upper brainstem dysfunction.**[5] There has been less need to make exploratory burr holes in head-injured patients since computed tomography (CT) scanning is widely available.

Burr holes can be lifesaving on rare occasions when the patient is worsening neurologically or has blown a pupil and CT scan is unavailable. Suspect a space-occupying lesion when there is clinical evidence of tentorial herniation or upper brainstem dysfunction. This includes progressive deterioration in the patient's level of consciousness, a fixed and dilated pupil, hemiparesis, posturing (i.e., decerebrate or decorticate), or flaccidity.

The placement of a temporal burr hole on the side of the fixed and dilated pupil to decompress an epidural or subdural hematoma can be lifesaving.[6] Up to 70% of patients with evidence of brainstem dysfunction soon after head trauma have significant intracranial mass lesions, most of which are extra-axial blood collections.[7] With adequate training, burr hole drainage of acute intracranial hematomas can be successfully performed in the Emergency Department by Emergency Physicians.[8-12]

ANATOMY AND PATHOPHYSIOLOGY

A significant proportion of patients with fatal head injuries die before reaching the hospital. Death is usually secondary to an expanding intracranial hemorrhage, basilar skull fractures with associated injury to the venous sinuses, intracranial carotid artery lacerations, or major cortical blood vessel lacerations. Skull fractures are present in up to 90% of adults who develop a traumatic intracranial hematoma. Children are less likely to suffer a skull fracture after head trauma than adults due to the relative plasticity and malleability of their skulls.

Injury to the middle meningeal artery, or its branches, is a frequent cause of severe intracranial hemorrhage following head trauma. This artery is a branch of the maxillary artery and enters the cranium via the foramen spinosum. It is usually located between the periosteal and meningeal layers of the dura mater. It divides into anterior and posterior branches after entering the skull. The larger branches of the middle meningeal artery lie within the dura and are accompanied by veins. Their superficial location in the dura produces grooves on the interior of the cranium and makes them vulnerable to injury, especially from temporal bone fractures. The bony vault of the skull is fairly thick, approximately 5 mm in thickness, and shows considerable individual and regional variation. The temporal bone, in particular the squamous temporal bone, is much

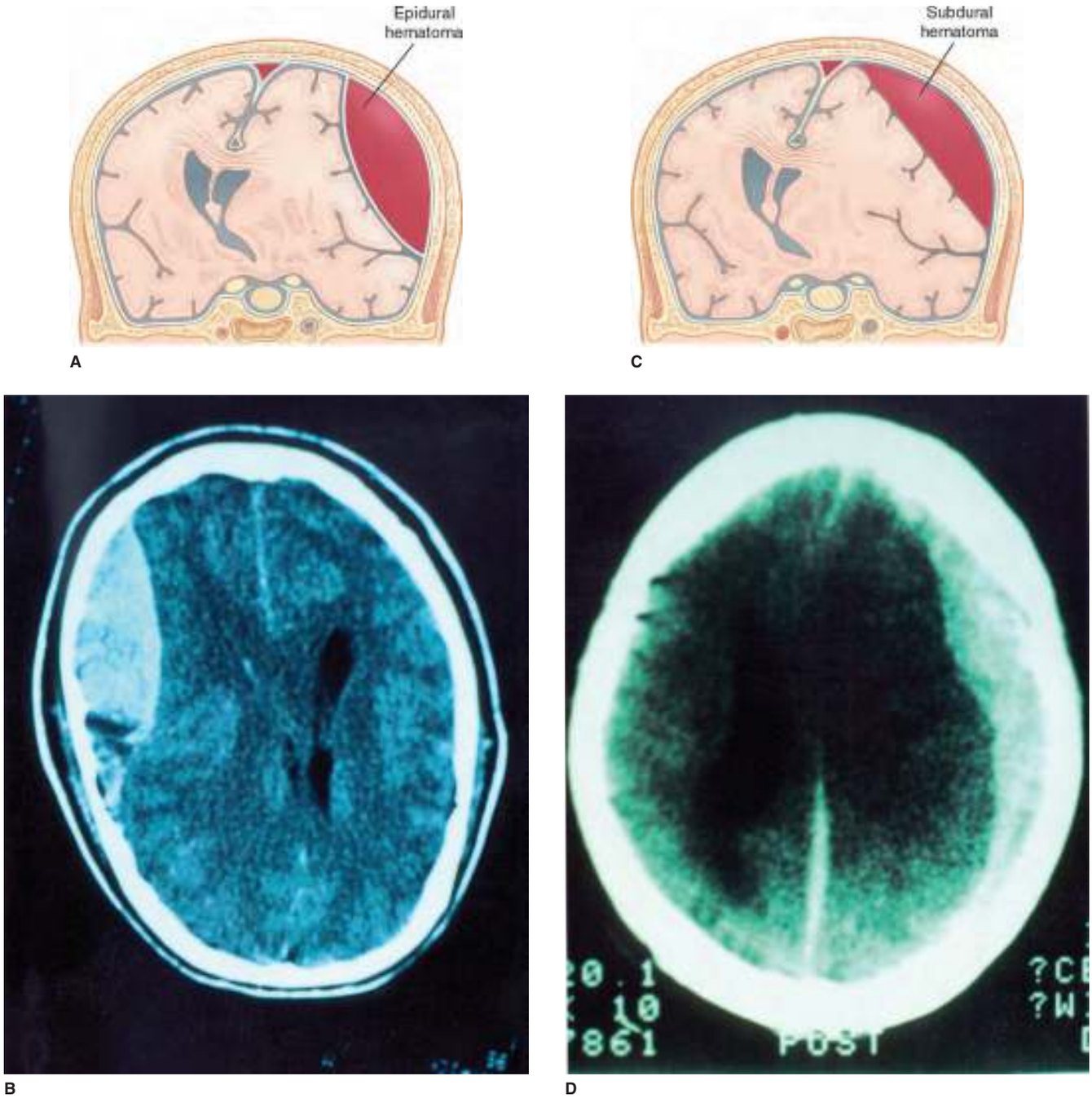

FIGURE 144-1. Hematomas requiring drainage through a burr hole. ***A.*** Illustration of an epidural hematoma. ***B.*** CT scan of an epidural hematoma. ***C.*** Illustration of a subdural hematoma. ***D.*** CT scan of a subdural hematoma.

thinner than other areas of the skull. This makes it more vulnerable to a fracture with an associated injury to the underlying middle meningeal vessels.

Posttraumatic epidural hematomas usually develop in the temporal or temporoparietal location as a result of an injury to the middle meningeal vessels (**Figures 144-1*A* and 144-1*B***). More than half of all epidural hematomas result from an injury to the middle meningeal artery. Epidural hematomas occur laterally over the cerebral hemispheres with the epicenter at the pterion in approximately 70% of patients (**Figure 144-2**). The remaining epidural hematomas are distributed in the frontal area, occipitoparietal area, and the posterior fossa. Other sources of epidural hematomas include a torn venous sinus or an injury to the carotid artery before it enters the intracranial dural mater.

Subdural hematomas are collections of blood between the dura mater and the brain (**Figures 144-1*C* and 144-1*D***). They usually result from tearing of a bridging vein as the brain moves within the skull during blunt head trauma.[7] The patient will present with an abnormal neurologic examination minutes to hours after the acute injury.

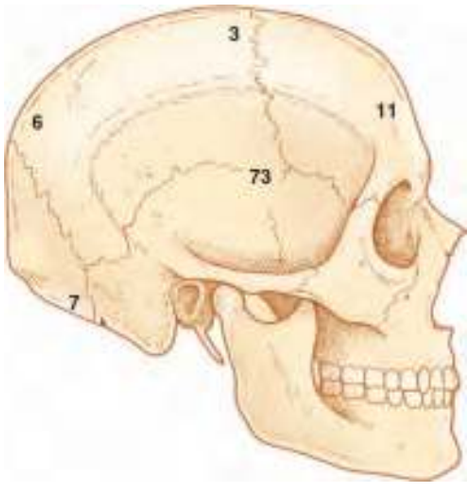

FIGURE 144-2. Percentages of epidural hematomas by anatomic location.

Pupillary changes are not an early sign of an intracranial hematoma but rather a sign of transtentorial (i.e., uncal) herniation causing compression of the occulomotor nerve. Other causes of acute pupillary changes need to be ruled out. Hematomas are found ipsilateral to the pupillary change in 85% of cases.

INDICATIONS

There are a few indications to emergently place a burr hole in the Emergency Department. These include the emergent drainage of an intracranial hematoma in a patient with a reduced Glasgow Coma Score (GCS) of < 8 with a fixed and dilated pupil, monitoring of intracranial pressure, and the emergent cannulation of the ventricular system (Chapter 147). This procedure may be performed by trained Emergency Physicians if a Neurosurgeon has been consulted and is not immediately available. Consider a burr hole if a Neurosurgeon is not available and the signs of brain herniation are present before transferring the patient.[13,14]

CONTRAINDICATIONS

The only absolute contraindication to burr hole placement is a patient who is coagulopathic. Otherwise, the available Emergency Physician with the most skill and experience in performing this technique should be the one to place the burr hole. Relative contraindications include localized infections of the scalp and patients who are thrombocytopenic. This procedure should not be performed by those unfamiliar with the technique and its complications. Performing it is controversial if it delays transfer of the patient to a center with neurosurgical services if the patient has signs of brain herniation.[13,14]

A coagulopathy or thrombocytopenia makes a burr hole dangerous to perform. The use of anticoagulants and antiplatelet agents by the patient increases their risk of hemorrhagic complications. Consider reversing these conditions by administering prothrombin complex concentrate (PCC), fresh frozen plasma, or platelets prior to the procedure. A mild elevation in the international normalized ratio (INR) up to 1.6 may be acceptable to place a burr hole.[15]

EQUIPMENT

- Sterile prep kit
- Sterile gloves and gown
- Face mask with an eye shield or goggles
- Cap
- Povidone iodine or chlorhexidine solution
- Sterile drapes
- 1% or 2% lidocaine containing epinephrine
- 22 gauge needles
- 5 mL syringe
- #10 scalpel blades
- #11 scalpel blades
- #3 scalpel handle
- Bipolar cautery, optional
- Self-retaining mastoid retractors
- Handheld drill, Hudson brace or air-powered
- Skull perforator bits
- Conical burr bits
- Small hook
- Bone wax
- Thrombin-soaked Gelfoam
- Periosteal elevator
- Suction catheter kit
- Head covers, masks, and sterile gowns
- Ventriculostomy catheter (optional)
- Bone rongeur
- Mayo scissors
- 4–0 nylon suture
- Potts scissors

The required equipment is stored and contained within a prepared sterile tray that can be obtained from the Operating Room or hospital central supply (**Figure 144-3**). A completely disposable, sterile, and single patient use instrument set is also available (Spectrum Surgical Instruments Corp., Stow, OH). The Hudson brace drill is a handheld device consisting of a stabilizer handle in series with a rotating handle (**Figure 144-4**). The distal end has a snap lock chuck that slides to allow easy insertion and removal of the bits. The perforator bits have a sharp point and come in a variety of shapes and sizes (**Figure 144-5**). The tip is designed to penetrate the inner table of the skull and lock without allowing it to puncture the dura or the brain (**Figure 144-6**). **Exercise extreme caution when using**

FIGURE 144-3. The contents of the sterile hospital-prepared burr hole tray.

FIGURE 144-4. The Hudson brace drill.

the perforator bit, as the bit may not lock when it penetrates the inner table of the skull. The burr bits are rounded and used to enlarge the hole made by the perforator bit (**Figure 144-6**).

There have been several modifications of a twist drill for making a burr hole.[16,17] These modifications are meant for the Operating Room or for use by Neurosurgeons. They are not for general Emergency Department use. These devices are more complicated than using the Hudson brace. Very little information exists on their use in the literature.

FIGURE 144-5. Examples of perforator bits (*left*) and burr bits (*right*).

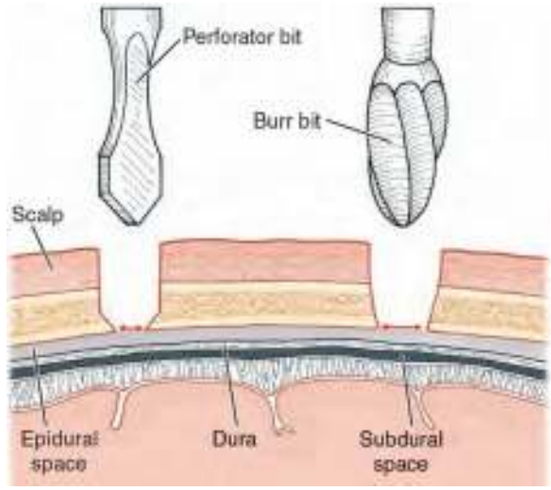

FIGURE 144-6. The perforator bit is used to make a hole through the skull and just penetrate the inner table of bone (red *arrow on the left*). The burr bit is used to enlarge the hole (*red arrow on the right*).

PATIENT PREPARATION

The patient should be fully monitored with a noninvasive blood pressure cuff, pulse oximetry, cardiac monitor, and end-tidal carbon dioxide monitor (if available). Obtain a CT scan of the head to determine the presence of an acute subdural or epidural hematoma to determine the location and extent of the hematoma, to rule out the presence of a mass, and to determine if there is herniation. Obtain a complete blood count and a coagulation profile to ensure that the patient is not thrombocytopenic or coagulopathic. These may require reversal with the administration of PCC, fresh frozen plasma, and/or platelets.

Explain the risks, benefits, and complications of the procedure to the patient and/or their representative. Obtain an informed consent if the patient is conscious. However, the patient who is deteriorating neurologically with tentorial herniation usually loses consciousness and time is of the essence. This may limit obtaining an informed consent. Document the circumstances of not getting consent in the medical record.

Determine the site for the skin incision and the burr hole (**Figure 144-7**). The burr hole should be placed over the center of the intracranial hematoma. CT images can assist in determining the location. A frontal or anterior burr hole is typically made just anterior to the coronal suture and 3 cm lateral to the midline, approximately along the midpupillary line (**Figure 144-7*A***). The coronal suture is often palpable. If not, draw a perpendicular line midway between the lateral canthus of the orbit and the external auditory meatus. The frontal burr hole can be used to drain an intracranial hematoma or to perform a ventriculostomy. The temporal burr hole is made two finger breadths above the zygomatic arch and two finger breadths anterior to the external auditory meatus (**Figure 144-7*B***). The parietal or posterior burr hole is made two finger breadths behind the external auditory meatus and three finger breadths above the mastoid process (**Figure 144-7*B***).

Prepare the patient. Orotracheally intubate the patient to protect and secure the airway. Insert a nasogastric tube to decompress the stomach. Shave the scalp at least 5 cm in all directions from the proposed skin incision. **This procedure requires strict aseptic technique.** Clean the skin of any hair remnants from shaving, dirt, or debris. Cleanse the skin first using 70% alcohol followed by povidone iodine or chlorhexidine solution. Allow the povidone

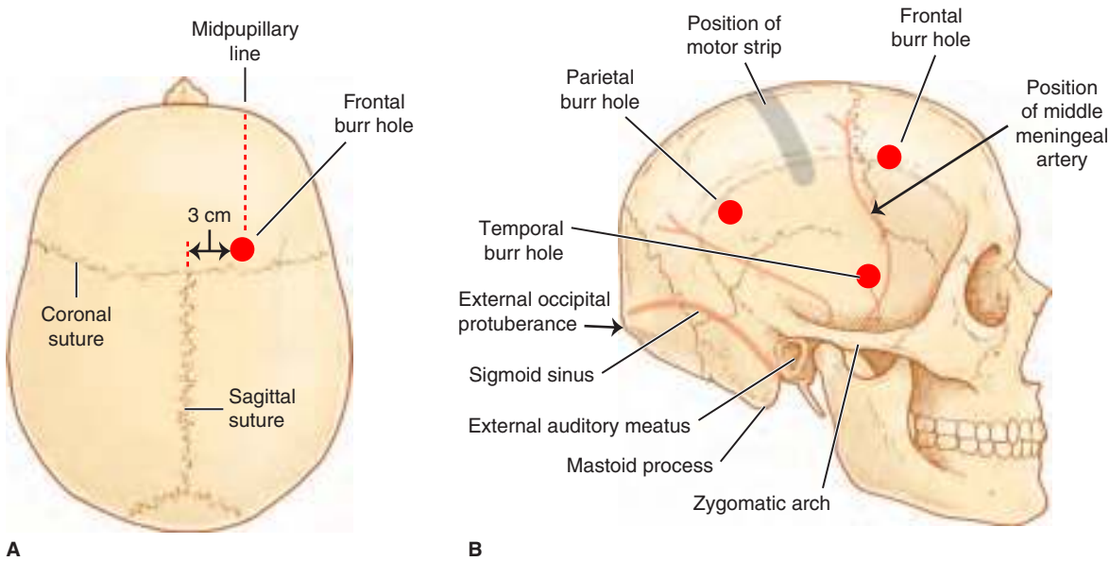

FIGURE 144-7. Typical locations for burr holes. ***A.*** Superior view of the skull. ***B.*** Lateral view of the skull.

iodine or chlorhexidine solution to dry. The Emergency Physician should don full sterile and personal protective equipment at this point. This should include sterile gloves, a sterile gown, a face mask with an eye shield or goggles, and a cap. Isolate the surgical field by using sterile drapes. Prophylactic intravenous antibiotic coverage is recommended if time permits. Administer a broad-spectrum antibiotic that covers gram-positive skin flora. Position the patient so that the proposed incision site is visible and easily accessible. Place the patient supine with a folded blanket or towel under the ipsilateral shoulder. Turn the head to the contralateral side if the cervical spine has been cleared. Instruct an assistant to hold and steady the patient's head.

TECHNIQUES

BURR HOLES

Identify the site to make the burr hole. Infiltrate 5 mL of lidocaine containing epinephrine along the proposed incision site and down to the level of the periosteum on the skull. This will result in analgesia and vasoconstriction that may aid in hemostasis. Make a 2 cm skin incision centered about the site to make the burr hole and straight down to bone. The incision must traverse all layers of the scalp including the skin, subcutaneous tissue, temporalis muscle (if present), and the periosteum. Remove the periosteum overlying the skull by scraping it away with a periosteal elevator. **The periosteum will otherwise get caught in the perforator bit and make it difficult to turn.** Insert a small self-retaining retractor into the incision (**Figure 144-8*A***). Hemostasis can often be obtained with the use of the retractor or cautery. Small bleeding vessels may be tied off with absorbable suture (**Figure 144-8A**).

Fit the Hudson brace drill with a perforator bit. Grasp the stabilizing handle of the Hudson brace with the nondominant hand. Grasp the rotating handle with the dominant hand. Place the tip of the perforator bit against the skull (**Figure 144-8*B***). Turn the rotating handle clockwise with the dominant hand using a smooth and slow motion. **Always maintain the drill perpendicular to the skull with firm, controlled pressure on the Hudson brace drill.** Frequently remove the perforator bit to examine the hole. Irrigate the area with normal saline. Use suction to remove the bone fragments and the irrigation fluid. Gently probe the hole to determine if the inner table has been penetrated. Continue to drill until the inner table has been penetrated or the perforator bit locks (**Figure 144-8*C***). **Do not apply too much downward pressure on the brace to prevent it from plunging into the brain. Exercise extreme caution as the bit does not always lock when the inner table is perforated.**

Remove the perforator bit from the Hudson brace drill. Place the burr bit on the Hudson brace drill. Place the burr bit into the hole in the skull. Hold the Hudson brace drill as described above. Rotate the handle clockwise to enlarge the hole in the skull (**Figure 144-8*D***). Frequently remove the burr bit to examine the hole. Irrigate the area with normal saline. Use suction to remove the bone fragments and the irrigation fluid. Continue to drill until the hole in the inner table is enlarged enough to accept the tip of the bone rongeur. **Do not apply too much downward pressure on the Hudson brace drill to prevent it from plunging into the brain.**

The clot of an epidural hematoma will be obvious as it separates the inner table of the skull from the dura. This clot will be gelatinous in consistency. Drainage through a single burr hole can be difficult. Free the underlying dura from the bone edge with a Penfield elevator. **Gently insert the elevator between the inner table of the skull and the dura. Gently separate the dura from the skull.** Enlarge the burr hole in order to facilitate aspiration of the blood clot. Insert a bone rongeur into the hole. Take small bites of the skull to enlarge the hole (**Figure 144-8*E***). **Do not be concerned with making the hole smooth or symmetric.** The Neurosurgeon will later trim and repair the bony defect if and when the patient goes to the Operating Room. Bleeding from the bone can be controlled with bone wax or a local hemostatic (e.g., Gelfoam).

HEMATOMA DRAINAGE

An epidural hematoma is aspirated by gentle suction and irrigation with normal saline through an adequate bone opening (**Figure 144-8*E***). **Pay close attention to the temperature of the irrigation solution. It should ideally be body temperature.** Use wall suction with a #9 or #11 French aspirator.

Epidural and subdural hemorrhages are usually clotted in the acute stages. In the event that an epidural hematoma is not identified

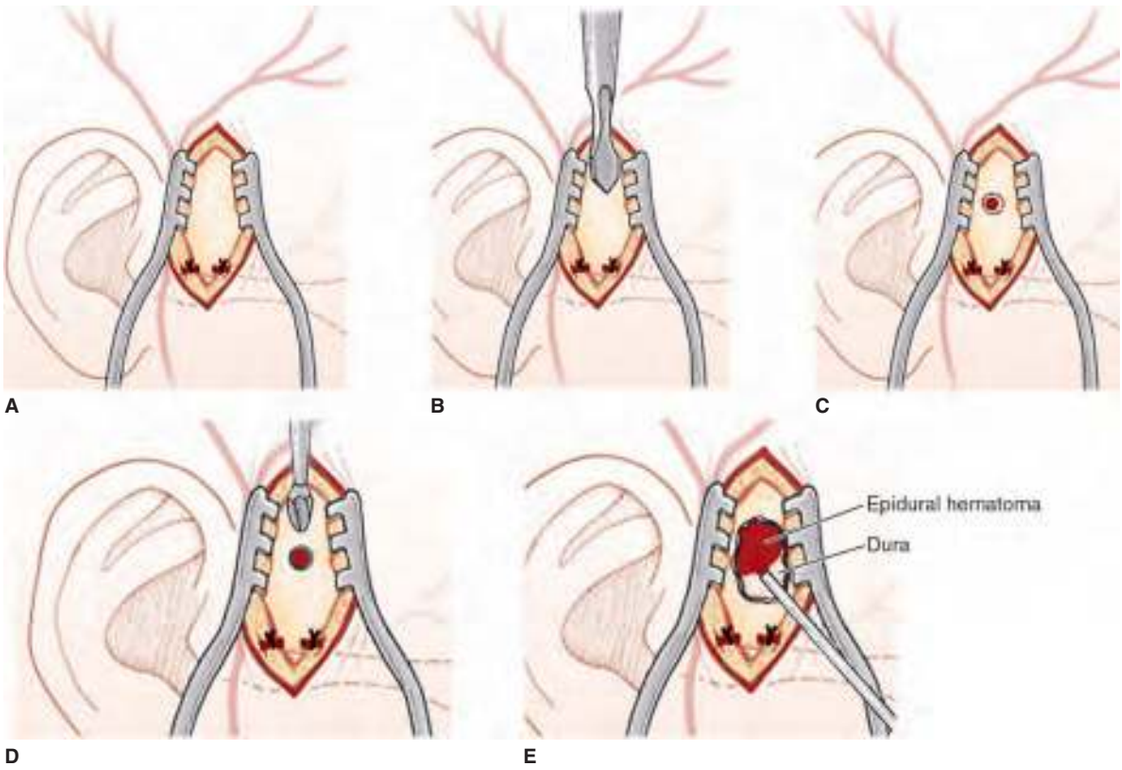

FIGURE 144-8. Drainage of an epidural hematoma. ***A.*** An incision is made through the skin, subcutaneous tissue, temporalis muscle, and galea aponeurotica. The incision is held open with self-retaining retractor. ***B.*** A Hudson brace drill fitted with a perforator bit is used to penetrate the skull to the inner table. ***C.*** A hole has been made with the perforator bit. ***D.*** The hole is enlarged with a burr bit on the Hudson brace drill. ***E.*** The bone edges have been removed with a rongeur to expose the epidural hematoma. The hematoma is gently removed by suction.

after placement of the burr hole, inspect the underlying dura for a possible subdural hematoma. The presence of a subdural hematoma causes the dura to have a bluish hue or tinge (**Figure 144-9*A***). Carefully place a traction suture in the middle of the exposed dura using 4–0 nylon (**Figure 144-9*B***). Apply traction on the suture to elevate the dura. Incise the dura with a fine Mayo scissors or a #11 scalpel blade (**Figure 144-9*B***). **Exercise extreme caution during the maneuver to prevent lacerating the brain.** Open the dura in a cruciate fashion. Drain the subdural clot using suction and gentle irrigation with normal saline. **At no time should any pressure be placed on the brain. Care should be taken not to irrigate with any force directly against the brain surface.**

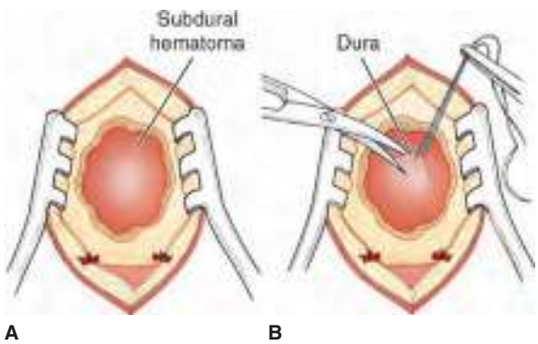

FIGURE 144-9. Drainage of a subdural hematoma. ***A.*** The dura is exposed and a hematoma is visible below it. ***B.*** Traction is placed on a suture that has been placed through the center of the exposed dura. The tented dura is carefully opened with a scissors or scalpel to expose the underlying hematoma.

VENTRICULOSTOMY

The burr hole can be made in order to place a ventriculostomy catheter. Make a nick in the dura with an 18 gauge needle or a #11 scalpel blade. Perform a ventriculostomy using an appropriate ventricular catheter. Insert the catheter perpendicular to the skull and direct it toward the ipsilateral inner canthus to enter the ventricular system.[18] Advance the catheter to a depth of approximately 5 to 6 cm. If unsuccessful after three attempts, place a parenchymal monitor or a subarachnoid bolt. Refer to Chapter 147 for the complete details of performing a ventriculostomy.

ASSESSMENT

Assessment and stabilization of the head injury victim prior to placement of the burr hole, during the procedure, and post-procedurally requires attention to securing the patient's airway, adequate and aggressive treatment of hemodynamic instability and shock, stabilization of the cervical and thoracolumbar spine, and concomitant treatment of any extracranial injuries. **Aggressive management of hypoxia and hypotension cannot be overemphasized. Hyperventilation in the first 24 hours after severe head injury should be avoided as it can reduce cerebral blood flow.** The assessment should include hemodynamic parameters, GCS, and frequent neurologic examinations. The neurologic examination should include pupillary size and reaction, extraocular muscle function, and motor movements of the extremities. Intubation using the rapid sequence technique often precedes burr hole placement in the patient with severe head injury. This precludes a detailed neurologic examination as most patients will have received neuromuscular blockade.

Reduction in pupillary size can be appreciated after evacuation of the hematoma in patients who have had pupillary changes preceding the burr hole placement. Repeat and document the hemodynamic and neurologic assessments every 5 minutes. Obtain a postprocedural CT scan of the head as early as possible to check the status of the hematoma. CT imaging also verifies catheter location and reduction in ventricular size in patients who have undergone ventricular catheter placement.

AFTERCARE

Patients who have had burr hole drainage require definitive management by a Neurosurgeon. A craniotomy is indicated for a more thorough evaluation, a thorough evacuation of the hematoma, irrigation of the epidural or subdural space, and hemostasis.[19,20] Postprocedural CT imaging is not required if definitive management by a Neurosurgeon is available and the patient can proceed directly to the Operating Room. Cranioplasty is often postponed following burr hole drainage to minimize infectious risk. Postprocedural management often requires airway protection with continued endotracheal intubation, fluid resuscitation, management of hypoxia, management of hypotension, management of seizure prevention, and management of coagulopathy. Secondary injuries can evolve, even after adequate hematoma evacuation. They need to be anticipated, recognized, and treated aggressively.

A two-layer closure is recommended in the event a craniotomy is not to follow or will be delayed. The dura is usually not closed. Cover the dura with a small piece of thrombin-soaked Gelfoam. Close the galea with 3–0 absorbable suture. Close the scalp skin with 3–0 nylon suture. Apply a dry dressing to the scalp wound. Alternatively, apply sterile saline-soaked gauze over the wound and cover this with a dry dressing.

COMPLICATIONS

Wound infections, abscesses, hemorrhage, and postoperative hematomas are major complications.[18] These can be avoided by using sterile precautions, antibiotic prophylaxis, and fine surgical technique. Other complications include plunging with the perforator bit or the burr bit and causing a penetrating injury to the brain, cortical laceration, or cortical contusion. Blunt or penetrating brain injuries can result in delayed stroke, hemorrhage, and seizures. Posttraumatic aneurysms and arteriovenous malformations have also been reported. A multitude of coagulopathic abnormalities can occur including disseminated intravascular coagulation, fibrinolysis, and hypercoagulability.

Significant bleeding complications can occur from the procedure.[18] Penetration of the sagittal sinus can result in significant hemorrhage and possible exsanguination. Prevent this by staying at least 2 cm from the midline and properly identifying the landmarks before drilling into the skull. Avoid lacerating the middle meningeal artery or its branches. Prevent injuries to these arteries by not drilling beyond the inner table and carefully separating the dura from the skull before using the bone rongeur. Another option is to obtain a lateral plain radiograph of the skull. Note the position of the grooves in relation to the external auditory meatus. Avoid these grooves, and thus the branches of the middle meningeal artery, when determining the exact site to place the burr hole.

SUMMARY

The prognosis for the severely head-injured patient with clinical evidence of tentorial herniation and brainstem compression is poor. Rapid evacuation of an intracranial hematoma may help to improve neurologic outcomes. Ideally, patients are resuscitated and a noncontrast CT scan of the head is performed in order to confirm the presence of an intracranial hematoma. Patients may at times undergo rapid neurologic deterioration prior to CT scanning or CT scanning may not be readily available. Diagnostic burr hole exploration and evacuation of an extra-axial hematoma can be a lifesaving measure. The authors do not wish to suggest that exploratory surgery should replace CT scanning in the management of patients with a severe head injury. The CT scan is invaluable in assessing and identifying the location of an intracranial hematoma. Burr hole evacuation in a trauma setting should be considered only in the presence of rapid neurologic deterioration, evidence of brain herniation, evidence of brainstem compression, and the unavailability of a Neurosurgeon to perform the procedure.

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145 Lateral Cervical Puncture

Eric F. Reichman

INTRODUCTION

The safest procedure to obtain cerebrospinal fluid (CSF) is lumbar puncture. However, there are situations where lumbar puncture is either contraindicated or technically not feasible. This includes infections in the lumbar area, obesity, previous spinal surgery, previous spinal fusion, a history of arachnoiditis, and the previous injection of chemotherapeutics. The usual and safe alternative method is a lateral cervical puncture under such circumstances.

Cisternal puncture describes the suboccipital access to cisterna magna, a CSF-containing space. It is a less frequently used procedure due to the high incidence of complications. As a result, cisternal puncture should be performed by a Neurological Surgeon for patients whose CSF cannot be accessed by lumbar puncture or lateral cervical puncture.[1] It will be described in this chapter for the sake of completeness.

Dr. Mullan introduced a method for performing a percutaneous cordotomy using a lateral cervical puncture in the early 1960s.[2] He introduced a strontium-90 needle through the C1-C2 interspace and into the subarachnoid space under fluoroscopic guidance. He then directed the needle anteriorly toward the anterior dura mater to interrupt the spinal thalamic fibers in an attempt to control intractable pain. The lateral cervical puncture is a direct derivative of this technique.

ANATOMY AND PATHOPHYSIOLOGY

Lateral cervical puncture involves the placement of a spinal needle into the C1-C2 interspace, posterior and inferior to the vertebral artery. The vertebral artery ascends through the foramina in the transverse processes of the cervical vertebrae beginning at the sixth cervical vertebra. It winds behind the lateral mass of the atlas (C1) to enter the skull through the foramen magnum (**Figure 145-1**). Inserting the needle 1 cm inferior to the tip of the mastoid process and 1 cm posterior from that point will avoid puncturing the vertebral artery (**Figure 145-2**).

The spinal canal is formed by sequential vertebral foramina and is triangular in shape. Its lateral width is greater than the anteroposterior width. The spinal canal is more spacious in the upper cervical spine, allowing for safe placement of a needle into the C1-C2 interspace. The sagittal diameter of the spinal canal is approximately 23 mm at C1 and 20 mm at C2. The cross-sectional area of the cervical spinal canal is greatest at C2 and progressively decreases. It is smallest at the level of C7. The vertebral canal is narrower in women than in men. The spinal canal at the level of C1-C2 can be divided into three parts. The anterior third is occupied by the odontoid process. The middle third is occupied by the spinal cord itself. The posterior third is occupied by the subarachnoid space. The spinal cord is suspended and cushioned within the subarachnoid space by CSF. The anterior boundary of the spinal canal is formed by the posterior aspect of the vertebral bodies and the intervertebral disks. The lateral wall of the spinal canal is formed by the pedicles and the intervertebral foramen. The posterior wall of the spinal canal is formed by the lamina, the ligamentum flavum, and the lateral masses or articular processes.

The morphology of the spinal cord demonstrates considerable individual variation in size and shape. The spinal cord changes in morphology throughout the entire spinal canal. It is cylindrical in shape and larger in transverse diameter than anteroposterior diameter. The spinal cord is largest from C3 to C6, obtaining approximately 13 to 14 mm in maximal transverse diameter. The average sagittal diameter of the cervical spinal cord is approximately 11 mm at C1 and 10 mm at C2.

The cervical nerve roots usually occupy the inferior one-third of the neural foramen. The first cervical spinal nerve root exits between the occiput and C1. The C2 through C7 spinal nerves exit above their corresponding numbered vertebra. Each nerve root innervates a specific dermatome and myotome, with considerable anatomic variation and overlap. The C1-C2 interspace is guarded laterally by the ligamentum flavum. The ligamentum flavum is composed of a yellow elastic tissue, the fibers of which are almost perpendicular in direction. It is attached to the lower part of the anterior surface of the lamina above and the posterior surface of the upper margin of the lamina below.

INDICATIONS

The lateral cervical puncture is an alternative method for obtaining CSF when lumbar puncture is not feasible or successful. Conditions that make lumbar puncture difficult are considered contraindications such as lumbar arachnoiditis, marked obesity, infections in the lumbar area, prior lumbar spine surgery, prior administration of lumbar intrathecal chemotherapeutics, and known congenital anomalies of the lumbar area (e.g., meningocele and myelomeningocele). The lateral cervical puncture is performed, like the lumbar puncture, in order to obtain CSF for analysis. CSF analysis

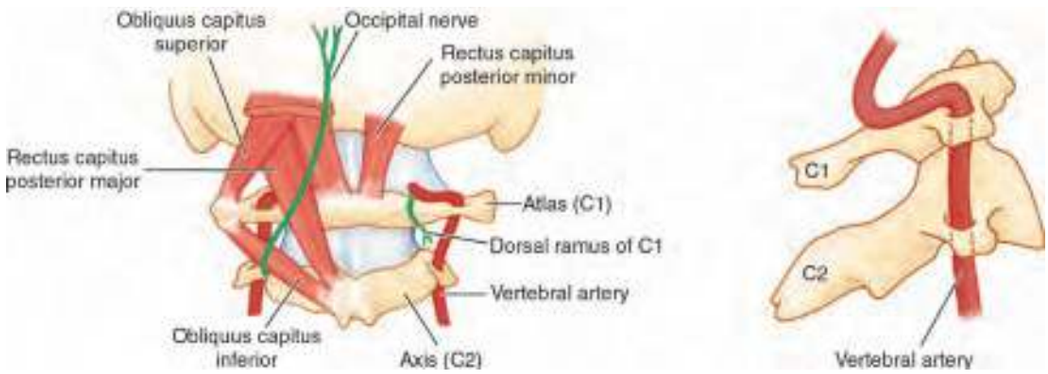

FIGURE 145-1. The course of the vertebral artery at the level of C1-C2. ***A.*** Posterior view. ***B.*** Lateral view.

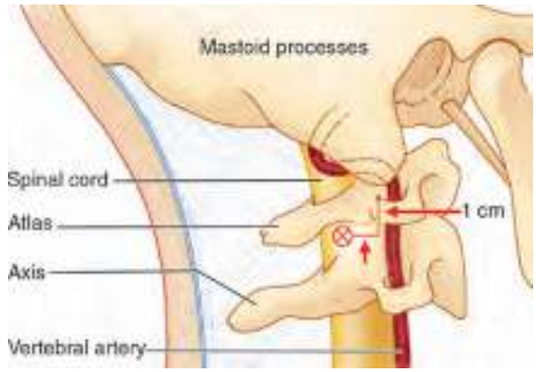

FIGURE 145-2. Anatomic landmarks for the lateral cervical puncture. The site for insertion of the needle is represented by an ⊗.

is indicated in patients suspected of having a central nervous system infection or a subarachnoid hemorrhage. Other indications for lateral cervical puncture include the installation of antineoplastic or antimicrobial agents. Lateral cervical puncture may also be necessary for the introduction of dye for radiographic studies.[3,4]

CONTRAINDICATIONS

Contraindications for a lateral cervical puncture include brain abscesses, brain tumors, cervical spine anomalies and deformities, coagulopathy, increased intracranial pressure, inflammatory adhesions, local infections, posterior fossa abscesses, posterior fossa tumors, thrombocytopenia, and vertebral artery anomalies (course or location). Arnold-Chiari malformations and other congenital abnormalities in the region of the foramen magnum are also a relative contraindication. These include achondroplasia, basilar impression, Dandy-Walker malformation, Klippel-Feil syndrome, and syringomyelia.

EQUIPMENT

- Sterile gloves and gown
- Face mask with an eye shield or goggles
- Cap
- 20 gauge spinal needle with a stylette
- 23 gauge spinal needle with a stylette
- 3 mL syringe
- 20 gauge needles
- 22 gauge needles
- Three-way stopcock
- Manometer
- Extension tubing, optional
- Four specimen vials with caps
- Gauze pads
- Sterile towels
- Fenestrated sterile drape
- Lidocaine hydrochloride, 1%
- Sterile gloves
- Povidone iodine or chlorhexidine solution
- Fluoroscopy unit, optional

All of the basic equipment can be found in commercially available lumbar puncture kits. The kit needs to be supplemented with personal protective equipment and skin antiseptic.

PATIENT PREPARATION

The patient should be fully monitored with a noninvasive blood pressure cuff, pulse oximetry, cardiac monitor, and end-tidal carbon dioxide monitor (if available). Obtain a computed tomography (CT) scan of the head if a complete neurologic examination cannot be performed or if it is abnormal in any way, the patient has a history of malignancy, of the patient may have a potential mass-occupying lesion (e.g., the patient with human immunodeficiency virus [HIV]). Obtain a complete blood count (hemoglobin, hematocrit, and platelet count) and a coagulation profile (prothrombin time, partial thromboplastin time, and international normalized ratio) to ensure that the patient is not thrombocytopenic or coagulopathic. Do not delay the administration of intravenous antibiotics pending these studies if meningitis is in the differential diagnosis and the reason for the lateral cervical puncture.

Explain the procedure, its risks, and benefits to the patient and/or their representative. Explain the postprocedural care. Obtain an informed consent for the procedure. Place the patient supine on the gurney, without a pillow, and the neck as straight as possible. Limit any head rotation from the true supine position. The landmark for needle insertion is 1 cm caudal and 1 cm posterior to the tip of the mastoid process (**Figure 145-2**).

This procedure requires strict aseptic technique. Clean the skin of any dirt and debris. Swab the area with alcohol pads. Shave the area so that the mastoid tip is contained within the sterile field. The Emergency Physician should don full sterile and personal protective equipment at this point. This should include sterile gloves, a sterile gown, a face mask with an eye shield or goggles, and a cap. Apply povidone iodine or chlorhexidine solution to the skin and allow it to dry. Apply sterile towels and drapes to delineate a sterile field. Inject local anesthetic solution subcutaneously at the above identified landmark. Consider the administration of intravenous diazepam or midazolam, if not contraindicated, to relax the patient. Some patients may require the administration of procedural sedation. The aid of an assistant to hold the patient's head straight and upright is recommended.

TECHNIQUE

Maintain the patient in the supine position with absolutely no head movement. An assistant should stabilize the patient's head. Introduce a 21 or 22 gauge spinal needle perfectly horizontal, parallel to the plane of the bed, and perpendicular to the neck (**Figure 145-3**). The needle will cross a number of tissue planes including the skin, subcutaneous tissue, trapezius muscle, suboccipital muscles, and the meninges. Advance the needle slowly and in 2 to 3 mm increments. Remove the stylette frequently to check for CSF. The subarachnoid space is approximately 6 cm from the skin surface in most adults. Puncture of the dura is often felt as a "pop" or loss of resistance. Frequent checks for CSF prevent excessive penetration of the needle through the subarachnoid space, overshooting the spinal canal, or inadvertent puncture of the spinal cord or vertebral artery. **Immediately remove the spinal needle if the patient develops any neurologic symptoms.**

CSF flow through the needle signifies that the tip is within the subarachnoid space. If CSF is not draining after puncture of the dura and removal of the stylet, rotate the needle 30°. The use of a portable fluoroscopic unit can confirm the needle's trajectory and exact position. Carefully apply the three-way stopcock and manometer

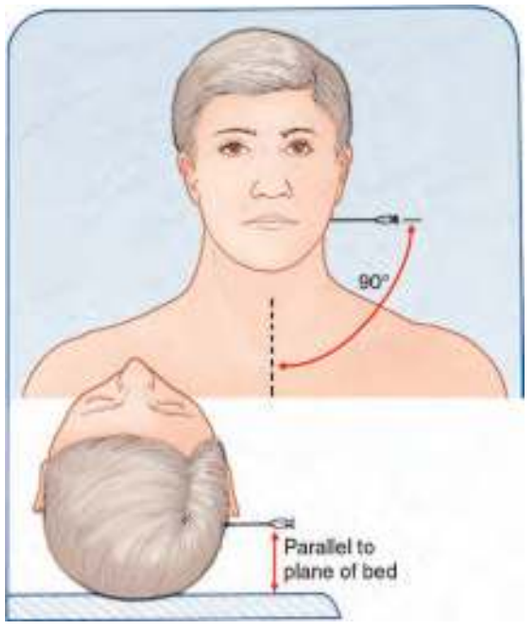

FIGURE 145-3. Proper needle trajectory for a lateral cervical puncture.

to the spinal needle to measure the pressure of the CSF. Carefully remove the stopcock and the manometer from the spinal needle. **Do not allow the spinal needle to move while applying and removing the stopcock and manometer. Keep in mind that the needle is not very well supported by the soft tissue as in the lumbar puncture. Hence, the needle must be supported more carefully.** Collect 1 to 2 mL of CSF in each specimen tube. Insert the stylet into the spinal needle. Remove the spinal needle and stylet as a unit. Apply a bandage to the skin puncture site.

Encountering arterial blood from the spinal needle usually indicates that it was pointing too far anteriorly and that the vertebral artery was penetrated. The venous plexus surrounding the vertebral artery may also be penetrated. Remove the spinal needle and apply manual pressure if the vertebral artery is inadvertently entered. Reattempt the procedure with the tip of the spinal needle directed slightly more posteriorly. Directing the spinal needle too far posteriorly causes it to enter the spinal musculature and miss the spinal canal. In the event that bone is encountered, meaning that either the lateral arch of C1 or C2 is encountered, redirect the spinal needle slightly rostrally or caudally and advance it into the subarachnoid space.

ALTERNATIVE TECHNIQUE

The cisternal puncture is an alternative to the lateral cervical puncture. Place the patient in the lateral decubitus position on the gurney. Place a pillow under the patient's head to keep the cervical and thoracic spine as straight as possible. Limit any head rotation from the true position. **This procedure also requires strict aseptic technique.** Clean, prep, and anesthetize the skin from the external occipital protuberance to the mastoid process.

Maintain the patient in the lateral decubitus position with absolutely no head movement. An assistant should stabilize the patient's head. Flex the patient's head toward the chest while not moving the cervical spine. Introduce a 21 or 22 gauge spinal needle between the C2 spinous process and the occiput, aiming cephalad. The needle will cross a number of tissue planes. Advance the needle slowly until the occiput is encountered. Slightly withdraw the needle and redirect it horizontally. Advance the needle in 2 to 3 mm increments until the cisterna magna is entered. The remainder of the procedure is as described for the lateral cervical puncture. The complications are similar to the lateral cervical puncture with the additional complication of laceration of the posterior inferior cerebellar artery.[5]

AFTERCARE

Maintain the patient in a supine position after the procedure. A small bandage is usually sufficient to control any soft tissue bleeding or continued CSF leakage. Postdural puncture headaches can be minimized by using a small gauged spinal needle inserted with the bevel parallel to the fibers of the dura. Resting in the supine position for 24 hours also reduces the incidence of postdural puncture headaches. Monitor the patient's neurologic status. Any change in patient's baseline neurologic examination requires a CT scan of the brain, posterior fossa, and occipital-cervical junction in order to rule out a herniation, epidural hematoma, or intradural hemorrhage.

Send the aspirated fluid for the appropriate laboratory analysis if an etiology other than an acute traumatic hemorrhage is suspected.[6] This can include a biochemistry analysis (glucose and protein level), cell count and differential, culture (e.g., bacterial, fungal, viral), cytology, and Gram's stain.

COMPLICATIONS

The complications associated with lumbar puncture are also possible during the lateral cervical puncture. Specific complications from lateral cervical puncture include penetration of the vertebral artery with subsequent hematoma formation and puncture of the spinal cord with resultant neurologic deficits.[6-11] Minimize complications by using a small gauge spinal needle. These complications should result in no serious consequences if unilateral and recognized.[6] Approximately 0.4% of the population has an anomalously positioned vertebral artery. There has been a single case report of a death from a subdural hematoma due to puncture of this vessel.[7] A nerve root may be irritated with passage of the needle, resulting in local pain or possibly a headache. Other complications include infection, herniation syndromes, neck pain, and headache. The incidence of postdural puncture headache is less with the lateral cervical puncture than with a lumbar puncture. Refer to Chapter 142 for the complete details regarding the complications associated with a lumbar puncture. An ultrasound-guided technique has been described in animals.[12] This technique has not been described, as of the publication of this book, in humans.

SUMMARY

A lumbar puncture is still the preferred technique to obtain CSF for analysis as well as for injection of contrast material. The lateral cervical puncture is a safe alternative method. One can avoid the complications of puncturing the vertebral artery or the spinal cord by observing maximum attention to detail. The lateral cervical puncture can be performed at the bedside. However, the availability of a portable fluoroscopy or ultrasound unit can facilitate the procedure.

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146 Intracranial Pressure Monitoring

Hannah Kirsch, Shahed Toossi, and Debbie Yi Madhok

INTRODUCTION

Increased intracranial pressure (ICP) can be a life-threatening result of brain injury (e.g., traumatic), edema (e.g., encephalopathy, postoperative, stroke, or trauma), expanding intracranial masses (e.g., abscesses, epidural hemorrhage, intraparenchymal hemorrhage, subdural hemorrhage, and tumors), hydrocephalus (e.g., aqueductal stenosis, Chiari malformation, or lesions obstructing cerebrospinal fluid [CSF] flow), infection (e.g., abscesses and meningitis), ischemic stroke, hemorrhagic stroke, metabolic abnormalities (i.e., encephalopathy, hypo-osmolality, and uremia), neurologic disorders, and pseudotumor cerebri. Elevated ICP can lead to disability, death, and permanent neurologic damage. It is often seen in the Emergency Department in relation to head trauma. High ICP may be seen just before death. Control of elevated ICP and fluctuations may improve recovery.

Most of this chapter is devoted to the current gold standard for ICP monitoring (i.e., ventriculostomy).[1-3] There are two basic types of ICP monitoring devices (**Figure 146-1**). One provides ICP data and the other provides ICP data and allows drainage of CSF. ICP monitors that use fluid are most accurate when closed to drainage. The methods of epidural, intraparenchymal, subdural, and ultrasonic ICP monitoring will be discussed briefly (**Figure 146-2**).

A

B

C

FIGURE 146-1. Examples of ICP monitoring devices. ***A.*** ICP monitor device. ***B.*** ICP monitoring device with CSF drainage. ***C.*** ICP "bolt."

ANATOMY AND PATHOPHYSIOLOGY

The Monro-Kellie doctrine was conceived in the eighteenth century by Scottish Physician Alexander Monro and his pupil George Kellie. **The cranial vault contains a fixed volume. An increase in the volume of one or more cranial constituents (i.e., blood, brain, and CSF) must be compensated for by a decrease in another constituent(s).** There is a balance between these components, making a dynamic equilibrium. The brain parenchyma is virtually incompressible. Backflow of CSF through the cerebral aqueduct and the relative vasoconstriction of cerebral vessels occurs after an acute brain injury to compensate.[4] Examples are the presence of

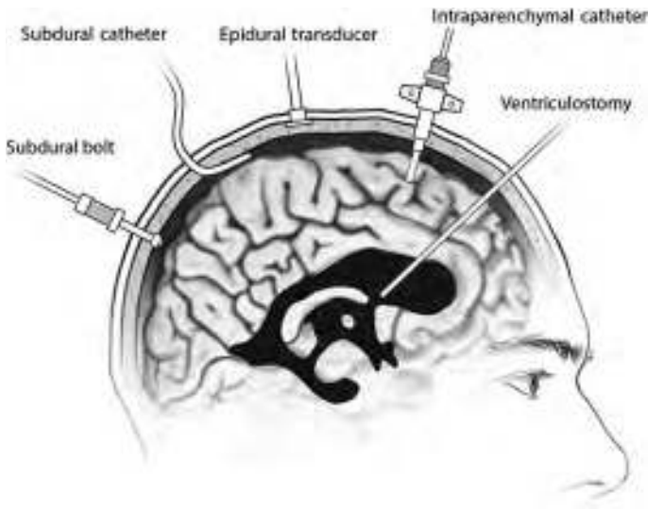

FIGURE 146-2. Locations of ICP monitoring devices. (Used with permission from reference 3.)

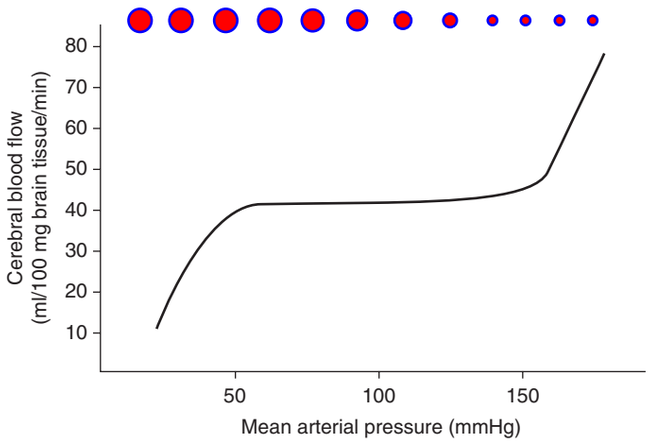

FIGURE 146-4. CBF is approximately constant between MAPs of 50 and 150 mmHg in a healthy individual without long-standing hypertension. Cerebral arterioles (represented along the top) can no longer change size to regulate flow over a MAP of 150 mmHg.

extravascular blood or edematous brain parenchyma.[4] There can be an abrupt increase in ICP once the initial compensatory shifts in blood volume and CSF are maximized (**Figure 146-3**).[5]

The elevation in ICP can result in herniation or shifting of brain tissue from an area of high pressure to an area of low pressure. Infratentorial mass effect can eventually lead to tonsillar herniation without appropriate pressure relief. Supratentorial mass effect can eventually lead to transtentorial herniation without appropriate relief of pressure. Herniation of the temporal lobe uncus can cause altered mental status and ipsilateral pupillary dilation. The less common central herniation causes bilateral pinpoint pupils and weakness. Tonsillar herniation may occur suddenly with examination signs of cardiac instability, coma, pinpoint pupils, and respiratory instability.[4]

One of the primary goals of ICP monitoring is to maintain an optimal cerebral perfusion pressure (CPP) that delivers the appropriate cerebral blood flow (CBF). Normal CBF averages 55 to 60 mL/100 gm brain tissue/min. The CBF is higher in gray matter (i.e., 75 mL/100 gm brain tissue/min) and lowest in white matter (i.e., 45 mL/100 gm brain tissue/min). CPP is vital to supporting cerebral perfusion. A CPP of 60 to 70 mmHg is adequate for maintaining brain functions. CPP is calculated as mean arterial pressure (MAP) – ICP. MAP is calculated as the diastolic blood pressure plus one-third of the pulse pressure or two-thirds of the diastolic blood pressure plus one-third the systolic blood pressure. CBF is calculated as CPP divided by cerebrovascular resistance. CBF can be maintained at a near constant level over a MAP range of about 50 to 150 mmHg by manipulating arteriolar diameter, which dictates cerebrovascular resistance (**Figure 146-4**). **This is the principle of cerebral autoregulation.**[6] CBF is insufficient to adequately perfuse tissue and can cause ischemic injury when below this MAP range. The CBF increases directly with MAP and can cause injury from hyperperfusion and elevated ICP by increasing the amount of blood volume in the brain when above this MAP range. Vasoconstriction decreases the CBF and cerebral blood volume.

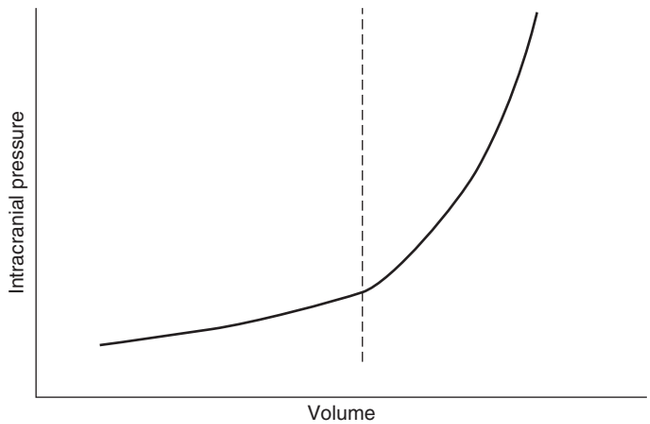

FIGURE 146-3. Intracranial pressure is relatively stable over a range of increasing volume of intracerebral contents. ICP begins to increase exponentially above this range.

The normal ICP ranges up to 20 mmHg. The range of 15 to 20 mmHg is abnormal and > 20 mmHg is pathologic. ICP elevation for more than 5 minutes is referred to as sustained intracranial hypertension. The monitor catheters can be used for several days before requiring replacement. The normal ICP waves have a steep upward slope during systole and a dicrotic notch during the downstroke in diastole (**Figure 146-5**). This indicates normal ICP.

External ventricular drains (EVDs) and other monitors provide the absolute measure of ICP and the ICP waveforms. Interpret the waveforms to provide additional context to the ICP measurement (**Figure 146-5**).[5] A normal ICP waveform has three upstrokes, each with a lower amplitude than the preceding one. The percussion wave (i.e., P1) corresponds to systole. It represents choroid plexus pulsations. It is normally the tallest of the three types of waves. The tidal wave (i.e., P2) corresponds to intracranial compliance. It represents brain volume and can be variable in shape. It is elevated in response to a rapidly increasing mass in the cranial cavity. A P2 wave higher than P1 represents decreased intracranial compliance. The dicrotic wave (i.e., P3) corresponds to the aortic valve closing. This is the lowest of the three waves normally.

ICP waveforms can be recorded with trend recording (**Figure 146-6**). A-waves or plateau waves have an amplitude of 50 to 100 mmHg above baseline and last up to 20 minutes. They are associated with decreased consciousness, increased extremity tone,

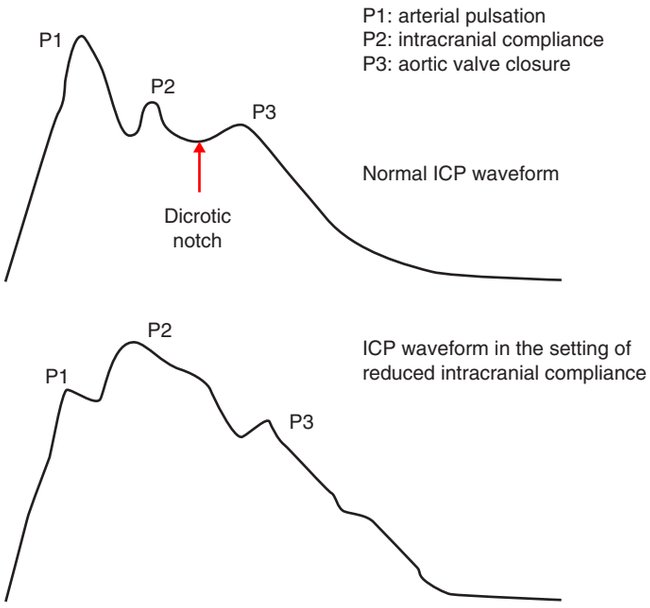

FIGURE 146-5. The normal ICP waveform is contrasted with an ICP waveform in the setting of decreased intracranial compliance.

restlessness, and tonic-clonic movements. They may represent surges in ICP due to increased cerebral blood volume. B-waves have an amplitude of 10 to 20 mmHg above baseline and are short lasting (i.e., < 2 minutes). They often precede A-waves, correlate with respiratory changes, and increasingly occur in decompensation. C-waves or Traube-Meyer-Hering waves are low amplitude, rapid, and may be superimposed on other waves. They can be seen with normal or increased ICP and fluctuate with respirations and changes in blood pressure.

Intracranial compliance is the ability of the brain to tolerate stimulation in other body regions and the accommodative potential of the intracranial space. It can be assessed by several methods. Injection of 1 mL aliquots of preservative-free saline into the intracranial vault and the ICP is measured. An increase of 2 mmHg or more for each 1 mL injected indicates compromised intracranial compliance. ICP can be measured before and after an external stimulus (e.g., endotracheal intubation). The ICP should return within 5 minutes to baseline, which indicates appropriate intracranial compliance. The ICP waveforms can be examined. An elevated P2 can be seen with impaired intracranial compliance.

One strategy to decrease ICP and deliver an optimal CPP is to augment the MAP to trigger the brain to vasoconstrict arterioles and decrease the total blood volume in the closed cranial vault. This only works with a brain with an intact cerebral autoregulation

INDICATIONS

The neurologic physical examination is the cornerstone of assessing whether a patient requires ICP monitoring. The Brain Trauma Foundation and European Brain Injury Consortium guidelines state that ICP monitoring is indicated in traumatic brain injury patients with an abnormal head computed tomography (CT) scan and Glasgow Coma Scale score less than 9 after resuscitation.[7] An abnormal CT scan is defined as intracranial contusions, diffuse edema, and hematomas. ICP monitoring is indicated in patients with severe traumatic brain injury and a normal CT scan with asystolic blood pressure < 90 mmHg, age over 40, or motor posturing (i.e., unilateral or bilateral).[7] The latter recommendation is based on a 1982 study regarding the risk of intracranial hypertension in patients with normal versus abnormal initial CT scans.[8]

Nontraumatic brain injury (e.g., hemorrhagic or ischemic stroke) requires ICP monitoring when the patient has a decline in alertness and signs of impending herniation. Anticipatory monitoring is useful in patients when sedation and/or paralysis impedes the physical examination assessment. Consider an anticipatory ventriculostomy in patients with injuries that are at high risk of progression to elevated ICP (e.g., extensive intraventricular hemorrhage or subarachnoid hemorrhage).

Interventricular catheters, commonly called EVDs, are considered the gold standard for ICP measurement. They are the only monitoring method that allows simultaneous measurement of ICP and treatment of elevated ICP via CSF drainage.[9] A less invasive monitoring option is an intraparenchymal device placed through a so-called "bolt" in the skull that can provide simultaneous access for other types of monitoring (e.g., cerebral blood flow). Recalibration of the zero point is not possible with these devices. A drift in the zero point is calculated as the difference between the ICP recorded after a device is removed and zero ICP measured while the device is in place and is a concern. Several observational studies have suggested that zero drift is not as significant a problem as first anticipated.[10,11] Most recent studies show accuracy ranges from 2 to 7 mmH_2O when compared to intraventricular pressures.[9,11-14] Hemorrhage and infection rates are comparable to those of intraventricular catheters, and numerous studies suggest lower complication rates.[9,15] One of the biggest disadvantages of this type of monitor (i.e., a "bolt") compared to an EVD is the inability to divert CSF. Subdural and epidural monitoring devices have been developed as less invasive options.

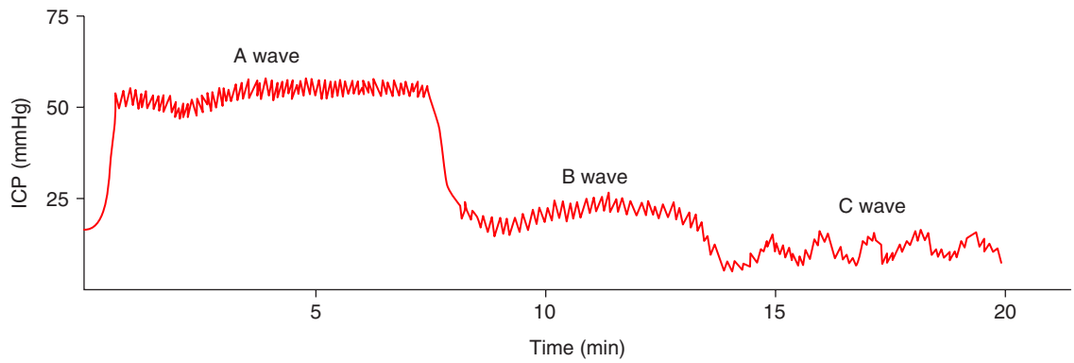

FIGURE 146-6. Examples of ICP waveforms using trend recording. (Used with permission from www.clinicalgate.com.)

Their accuracy is more limited than that of intraparenchymal devices. Their use is plagued by zero drift and frequent dislodging after placement. These devices are not frequently used.

CONTRAINDICATIONS

Contraindications for ventriculostomy specifically include the presence of increased infratentorial pressure without an occipital craniectomy, which may induce upward herniation if intraventricular pressure is relieved. Other general procedural contraindications include thrombocytopenia and coagulopathy. There are no validated guidelines. Typical acceptable parameters include an international normalized ratio (INR) of up to 1.6 and platelets of less than 100,000/μL.[16,17] Emergent platelet transfusion or administration of fresh frozen plasma may be warranted under these circumstances. Perform a noncontrast head CT scan prior to ICP monitor insertion to ensure that there is no hemorrhage, mass, or other lesion along the anticipated tract.

EQUIPMENT

- ICP monitor (**Table 146-1**)
- Skull penetration equipment (Chapters 144 and 147)
- External monitoring device (**Figure 146-7**)
- Sterile gloves
- Sterile gown
- Cap
- Face mask with eye shield or goggles

ULTRASOUND (US) EQUIPMENT

- US machine
- US gel
- High-frequency US transducer
- Clear adhesive dressing (e.g., Tegaderm)

The Codman Microsensor can read intraparenchymal, intraventricular, and subdural pressure depending on its location. It drains CSF if placed in the ventricle. It uses a strain gauge located at the tip and transmits pressure changes over a diaphragm.

The Gaeltec ICP monitor measures ICP subdurally. This system is reusable. It measures pressure with a balloon-covered sensor.

The Integra Neuroscience products are most numerous. Some systems allow CSF drainage and ICP data. The drain must be leveled with the foramen of Munro, zero-balanced daily, and its level adjusted to regulate flow. Other systems measure ICP data without CSF drainage. These systems use a diaphragm to measure pressure or a transducer.

TABLE 146-1 Some Available ICP Devices

Codman
Microsensor Monitor
Gaeltec
Subdural ICP Monitor
Integra
Camino ICP Monitor with Licox Bolt
Camino Micro Ventricular Bolt Pressure Monitor
Camino OLM ICP Monitor
Camino Post Craniotomy Subdural Pressure Monitor
Ventrix Ventricular Tunneling Pressure Monitor
Raumedic
Neurovent
Neurovent-P

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the ICP catheter insertion on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Maintain sterility of the ICP catheter and skull entry site. This includes accessing CSF through the catheter. Don full personal protective equipment before placing the ICP catheter. Use a cap, face mask, and sterile gloves if accessing the CSF through the ICP catheter or maintaining the skull puncture site.

Note and document the physical examination with an emphasis on the nervous system. Early signs and symptoms of elevated ICP include altered mental status, headache, nausea, and vomiting. **Note the vital signs frequently.** This can progress to coma and motor posturing in response to noxious stimuli. Cushing's response (i.e., the triad of bradycardia, irregular respirations, and systolic hypertension) may be present. Document the patient's alertness and Glasgow Coma Scale score (**Tables 146-2 and 146-3**). Note language articulation, comprehension, fluency, and repetition. Assess the corneal reflexes, extraocular movements, pupillary response, and visual fields. Document the blinking, presence of doll's eyes, and visual pursuit if the patient does not follow commands. Examine the fundus for changes to the optic nerve suggestive of papilledema and increased ICP (e.g., changes of the blood vessels, changes to the cup-to-disk ratio, and changes to disk margins). Perform and document the vital signs frequently.

INVASIVE TECHNIQUES

A full technical description of EVD placement in adult and pediatric patients is detailed in Chapter 147 titled "Ventriculostomy." The directions below will vary depending on the exact model of the ICP monitor. The advantages and disadvantages of these invasive techniques are listed in **Table 146-4**. The invasive techniques are more accurate than noninvasive techniques.[18-21] The general instructions are below.

Insert the ICP device using sterile conditions. Connect all cables. Connect the cable to the ICP cable in the patient's skull. Connect the other end of the cable to the external monitor. Turn the external monitor on. Zero the external monitor. Calibrate the patient monitor. Begin measuring the quantitative ICP and waveforms.

NONINVASIVE TECHNIQUES

These techniques can be used to estimate the ICP.[18-21] They are not as accurate as invasive techniques.

OPTIC NERVE SHEATH DIAMETER

The diameter of the optic nerve sheath can be used to estimate ICP.[22-26] This can be performed using CT scans or US.[19,18-30] The bony eye socket and the fluid-filled globe are suited for US examination. Papilledema is visualized as a hyperechoic protrusion from the optic disk.[23] Increased ICP is transmitted through the subarachnoid layer to the optic nerve sheath. This results in an enlarged optic nerve sheath.[22] The advantages of using US are as follows: it is convenient, easily learned, and easy to perform at bedside; it lacks ionizing radiation; and it can be performed without moving the patient to the CT scanner. Papilledema is uncommon in acute ICP elevations

A

B

C

D

FIGURE 146-7. Examples of external ICP monitoring devices. ***A.*** Duet External Drainage and Monitoring System (Medtronic, Minneapolis, MN). ***B.*** The handheld Pressure Monitor NPS3 (Raumedic AG, Helmbrechts, Germany). ***C.*** ICP-Monitor HDM (Spiegelberg, Hamburg, Germany). ***D.*** ICP Express Monitoring System (Codman Neuro, Raynham, MA).

TABLE 146-2 The Glasgow Coma Scale Score*

Score	6	5	4	3	2	1
Eye opening	N/A	N/A	Spontaneously	In response to speech	In response to painful stimuli	Do not open in response to stimuli
Motor response	Follows commands	Localized movement to painful stimuli	Nonpurposeful movement to noxious stimuli	Flexes upper extremities and extends lower extremities to painful stimuli	Extends all extremities to painful stimuli	No response to stimuli
Verbal response	N/A	Orientated to person, place, and time	Converses but confused	Replies inappropriately	Incomprehensible	No response

*The total score is the sum of the eye opening, motor response, and verbal response components. N/A, not applicable.

TABLE 146-3 The Pediatric Glasgow Coma Scale Score*

Score	6	5	4	3	2	1
Eye opening > 1 year old	N/A	N/A	Spontaneously	In response to speech	In response to painful stimuli	Do not open in response to stimuli
Eye opening < 1 year old	N/A	N/A	Spontaneously	In response to shouting	In response to painful stimuli	Do not open in response to stimuli
Motor response > 1 year old	Follows commands	Localized movement to painful stimuli	Flexion; withdraws to painful stimuli	Flexes extremities to painful stimuli	Extends extremities to painful stimuli	No response to stimuli
Motor response < 1 year old	Follows commands	Localized movement to painful stimuli	Flexion; withdraws to noxious stimuli	Flexes extremities to painful stimuli	Extends extremities to painful stimuli	No response to stimuli
Verbal response > 5 years old	N/A	Orientated to person, place, and time	Converses but confused or disoriented	Replies inappropriately	Incomprehensible	No response
Verbal response 2–5 years old	N/A	Appropriate words or phrases	Inappropriate words	Persistent cries and screams	Grunts	No response
Verbal response < 2 years old	N/A	Smiles or coos appropriately	Cries inconsolably	Persistent inappropriate crying and/or screaming	Grunts, agitated, and restless	No response

*The total score is the sum of the eye opening, motor response, and verbal response components. N/A, not applicable.

TABLE 146-4 Advantages and Disadvantages of ICP Methods

ICP monitor	Advantages	Disadvantages
Epidural	Decreased risk of infection	Cannot determine pressure-volume relationship
	Easily placed	Decreased accuracy of ICP
	Least invasive method	Decreased waveform quality
	Quickly placed	Increased baseline drift with time
		No CSF drainage or sampling
Intraparenchymal	Baseline drift of 1 mmHg per day	Baseline drift if used for multiple days
	Easily placed	Cannot be recalibrated after placement
	Easy to move patient location	Cannot determine pressure-volume relationship
	Good-quality waveforms	Easily damaged
	Less artefact than other devices	Fragile cable
	No need for adjustment with patient position changes	Manipulation can damage fiberoptics
	Only requires zeroing during insertion	No CSF drainage or sampling
	Uses fiberoptic transducer at tip	Requires periodic replacement
Intraventricular	Accurate ICP measurements	Bleeding with quick CSF drainage
	Can determine pressure-volume relationship	Catheter occlusion
	Contrast administration	CSF leakage
	CSF drainage to decrease ICP	Difficult insertion with compressed ventricles
	CSF sampling	Difficult insertion with displaced ventricles
	Medication administration	Difficult insertion with edema
		Difficult insertion with small ventricles
		Frequent transducer recalibration
		Infection to center of brain
		Ventricle collapse with quick CSF drainage
		Very invasive
Subarachnoid	Easily placed	Dampened waveforms
	Can be used with collapsed or small ventricles	Less accurate than other methods
	Lower infection rates than ventricular catheters	Frequent recalibration
	No brain penetration	No CSF drainage or sampling
	No locating ventricles	Recalibration after patient position changes
	Quickly placed	
Subdural	Same as epidural	Same as epidural

and is often a late sign of elevated ICP. A normal optic nerve sheath does not exclude elevated ICP. The normal optic nerve sheath diameter will vary among patients and the normal values are difficult to define.

The patient can be in any position. The pediatric patient can be restrained (Chapter 232) or held by a parent. Have the patient close the eye. Apply a clear dressing over the eye to keep the eyelid closed. This also decreases the anxiety of the transducer approaching and pushing on the eye. It also prevents the US gel and transducer from irritating the eye or causing a corneal abrasion. Apply US gel liberally to obtain a good acoustic window and prevent any pressure on the eye. Scan the eye in the longitudinal and transverse planes. Scan the eye with the patient looking in four directions to find the best optic nerve sheath dimensions. Looking inward often moves the optic nerve the most perpendicular to the US waves.[28]

TRANSCRANIAL DOPPLER

Transcranial Doppler (TCD) can be used in children, sedated patients, trauma patients, and when invasive ICP monitoring is contraindicated.[27] TCD can be performed at the bedside, is noninvasive, and is portable. It measures cerebral blood flow velocities. TCD relies on operator-dependent factors (e.g., locating the acoustic window, the angle of the transducer, and obtaining a strong pulse signal). It may be difficult to obtain a sufficient Doppler signal limiting the use of TCD.

OCULAR VASCULATURE

Third Eye Diagnostics Inc. of Bethlehem, Pennsylvania, has developed the Cerepress. It uses the blood vessels of the eye to estimate ICP. It noninvasively measures blood pressure in the central retinal vein as a correlate to ICP. The Cerepress measures intraocular pressure and ICP. This hand-held device can be used at bedside and requires little training.

ASSESSMENT

Perform a noncontrast head CT to confirm accurate placement of the ICP monitor. Hemorrhage along the insertion tract may or may not become clinically significant. Repeat the CT scan if hemorrhage is present to ensure the stability of hemorrhage size. The EVD and other monitors provide the absolute measure of ICP and the ICP waveforms. Interpret the waveforms to provide additional context to the ICP measurement (**Figures 146-5 and 146-6**).[5]

AFTERCARE

Monitor all patients requiring invasive ICP monitoring in an Intensive Care Unit. Closely monitor hemodynamic and respiratory status. Perform frequent examinations of the patient's vital signs and neurologic examinations as noted in the patient preparation section earlier (**Tables 146-1 and 146-2**).

COMPLICATIONS

Major complications are hemorrhage and infection. Tract hemorrhage upon catheter insertion and removal occurs between 5% and 41% of patients.[31-34] The rate of clinically significant hemorrhage is low and ranges from 0.5% to 5% of patients. Studies on hemorrhage rates are limited and most are retrospective.

There is a wide range of reported incidences of ICP device–associated infection (e.g., abscesses and meningitis).[14,35-37] Infection rates are associated with a longer duration of monitoring. Sterile technique reduces the risk of infection. **Use sterile technique when placing a ventricular catheter.** Some retrospective studies showed an increased risk of infection in devices inserted outside the Operating Room. These differences were not statistically significant. Staphylococci are the most common bacteria isolated. There is mixed evidence for the use of single-dose antibiotic prophylaxis at the time of ICP catheter insertion.[38,39] Consult institutional guidelines prior to performing the procedure regarding antibiotic administration.

Technical issues can lead to inaccurate ICP measurements. Zero-balance the catheters daily and whenever the catheter is moved. Catheters can become occluded with arachnoid villi, brain tissue, or blood clots. Waveforms can be dampened by clots, by air in the catheter, or if the catheter tip is against the brain. Catheters can bend or kink. Waveforms can be misinterpreted.

SUMMARY

Patients with traumatic brain injury or other pathology often require monitoring of ICP in response to signs of intracranial hypertension (e.g., clinical or radiologic) or in anticipation of impending elevations of ICP. EVDs are the gold standard method of ICP monitoring and a way to relieve elevated pressures via CSF drainage. Less invasive methods of monitoring are currently less accurate and largely confined to more specialized centers but may become more reliable and accessible in the future.

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147 Ventriculostomy

John Burke, Shahed Toossi, and Debbie Yi Madhok

INTRODUCTION

An external ventricular drain (EVD) is the gold standard for measuring intracranial pressure (ICP) because it is both diagnostic and therapeutic.[1] Elevated ICP can be quickly ascertained and subsequently relieved by draining cerebrospinal fluid (CSF) through the EVD catheter. Occasionally, placement of an EVD represents an emergent lifesaving procedure that needs to be done within minutes in a patient who is deteriorating rapidly from a neurologic perspective.[2] This chapter will discuss some of the situations when this procedure may be considered, other therapeutic options, and an explanation of how to perform an emergent ventriculostomy.

ANATOMY AND PATHOPHYSIOLOGY

The cranium is a fixed space after infancy that has little capacity for added volume or mass. This concept is the fundamental principal behind the Monro-Kellie doctrine which states that the fixed space of the skull must accommodate brain tissue, blood, and CSF. **An increase in volume of any one of these substances without a compensatory decrease in the amount of the other will lead to an increase in ICP.** All states of increased ICP can be understood within this framework. Examples include tumors that may increase ICP either by adding volume to the cranium (i.e., the mass itself), blocking CSF outflow (i.e., increasing the amount of CSF), causing vasogenic edema (i.e., expanding the amount of brain tissue), hemorrhaging into the tumor cavity (i.e., increasing the amount of blood in the cranium), or any combination of these mechanisms. Other pathologic conditions including edema, infection, intracranial hemorrhage, and massive cerebral infarctions can increase ICP.

The patient with increased ICP may display the classic clinical signs of headache, vomiting, and papilledema.[2] Vomiting is particularly associated with acute increases in ICP. Other signs include a cranial nerve VI (i.e., the abducens nerve) palsy that causes diplopia, decreased consciousness, and an elevated blood pressure with bradycardia (i.e., Cushing's phenomenon). **An increase in ICP may eventually progress to herniation which occurs when there is a force applied to a part of the brain great enough to push other parts of the brain into different cranial compartments.** The cranial contents are divided into compartments by invaginations of the dura mater (**Figure 147-1**).[3] The supratentorial space is separated from the infratentorial space by the tentorium cerebelli. The right and left hemispheres are separated by the falx cerebri.

When a unilateral supratentorial mass exerts enough force, the ipsilateral cerebral hemisphere is pushed medially toward the opposite hemisphere (**Figure 147-2*A***). A tentorial herniation is when the medial aspect of the temporal lobe is pushed down toward the brainstem and over the edge of the tentorium cerebelli (**Figure 147-2*B***). The symptoms of a tentorial herniation include a worsening of any headache with vomiting, progressively decreasing consciousness, anisocoria, hemiparesis, and Parinaud's syndrome (i.e., an upward gaze paresis) from compression on the dorsal midbrain. Compression of the oculomotor nerve results in a sluggish and dilated pupil, usually on the same side as the mass lesion. **A progression to a fixed and dilated pupil with decerebrate rigidity (i.e., extensor posturing) is an ominous sign of increased ICP.**

Mass effect in the infratentorial compartment of the skull may produce downward pressure of the cerebellum into the foramen

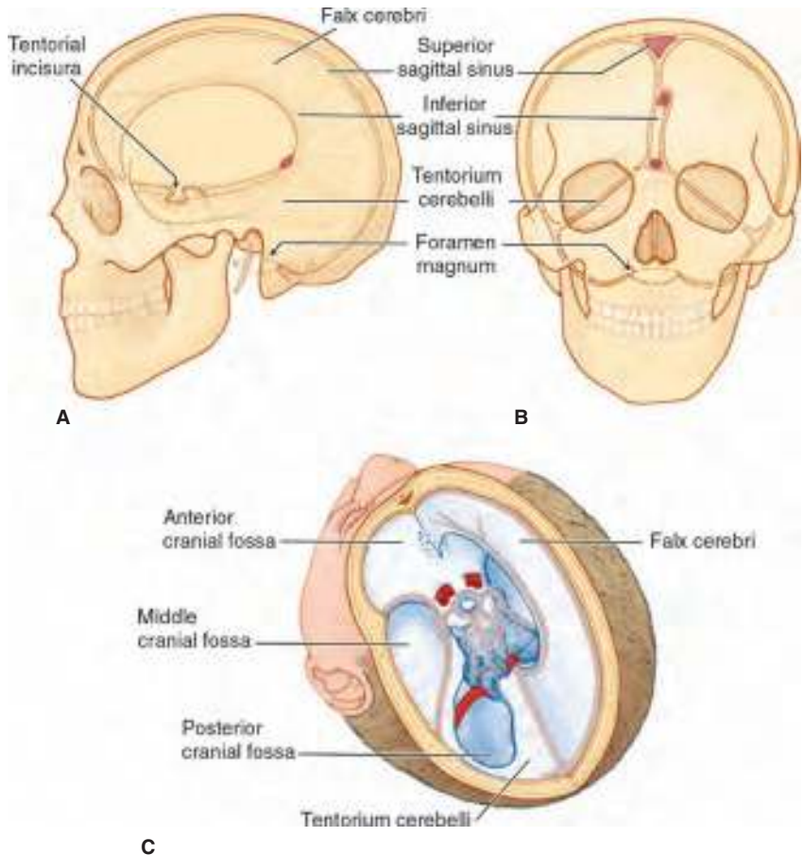

FIGURE 147-1. The falx cerebri and tentorium cerebelli divide the skull into compartments. ***A.*** Sagittal view. ***B.*** Coronal view. ***C.*** Top of the skull removed with a section of tentorium cerebelli also removed.

magnum (**Figure 147-3*A***) or upward pressure of the midbrain into the supratentorial compartment (**Figure 147-3*B***), the former being more common. The downward pressure is known as tonsillar or cerebellar herniation (**Figure 147-3*A***). The terminal events surrounding this condition may occur more urgently and fatally than a supratentorial herniation. The urgency of cerebellar herniation stems from both the tight space in the posterior fossa compared to the supratentorial compartments as well as the vital structures found in this area. For example, an acute respiratory arrest may occur from compression of the medulla. Other important signs of tonsillar herniation include profuse vomiting, irregular respirations (e.g., ataxic breathing), neck pain, and neck stiffness. **The patient may not necessarily lose consciousness or have pupillary changes prior to the terminal events of tonsillar herniation.** Upward herniation forces the cerebellum and upper brainstem through the tentorial notch (**Figure 147-3*B***). The patient is usually obtunded or comatose with small or anisocoric pupils that may at first be reactive. There may be an associated paresis of the extremities which progresses to decorticate posturing.

Any patient with these potentially life-threatening neurologic findings should be considered unstable and worked up accordingly. An emergent head computed tomography (CT) scan is the diagnostic test of choice after the patient's airway, breathing, and circulation have been stabilized. The signs of subfalcine herniation on the head

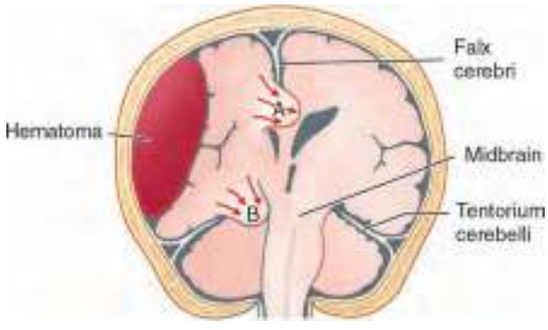

FIGURE 147-2. Herniation of the brain. ***A.*** Subfalcial herniation. ***B.*** Tentorial herniation.

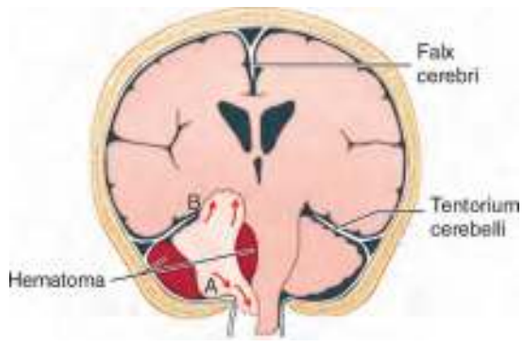

FIGURE 147-3. Herniation of the brain. ***A.*** Downward cerebellar tonsillar herniation through the foramen magnum. ***B.*** Upward cerebellar herniation into the supratentorial compartment.

CT include an anterior midline shift with brain tissue from one side pushed under the falx and into the opposite side of the brain (**Figure 147-2*A***). Other signs include generalized cerebral edema with effacement of the sulci, the ventricles, the basilar cisterns, and the fourth ventricle.

The term herniation is often confused with mass effect. These are two terms for independent variables. Herniation is an emergent clinical syndrome that portends acute neurologic decompensation and implies a rapid onset of symptoms. Mass effect and increased midline shift are radiologic phenomena that may or may not correlate with the clinical syndrome of herniation. For example, a well-compensated chronic increased ICP may not directly lead to herniation despite the radiologic presence of a mass effect. Clinical status is the best predictor of herniation and is the most important indicator for the emergent placement of an EVD. A patient with a Glasgow Coma Score of 15 will rarely require an EVD no matter what the head CT shows.

There are several clinical interventions that should be implemented immediately once increased ICP, hydrocephalus, or herniation is suspected and the patient's condition is declining.[4] The airway must be stabilized and rapid sequence intubation is the procedure of choice. Pretreatment with lidocaine may be useful in diminishing the elevation in ICP that occurs during direct laryngoscopy. Propofol and etomidate are the induction agents of choice as they may decrease the ICP. Thiopental is another choice but is rarely used in the United States due to it causing hypotension. There is a theoretical risk that ketamine can increase ICP secondary to increasing cerebral blood flow and increasing cerebral metabolic rate of oxygen. There is a dearth of evidence supporting these possibilities.[5] An advantage to ketamine is that it is more hemodynamically stable than both etomidate and propofol. Neuromuscular blockade is accomplished with succinylcholine after administering a defasciculating dose of a nondepolarizing neuromuscular blocking agent. Paralytics may only be necessary for a brief period during intubation. **It is preferable to avoid paralysis for any length of time when the patient's neurologic status needs to be followed closely and any changes readily identified.** However, the need to follow the patient's neurologic course should be balanced by the need to reduce elevated ICP that may be accomplished, in part, with sedatives and paralytics. The potential for succinylcholine increasing ICP is most prevalent in patients with neoplasms of the central nervous system and may not be significant for acute trauma or bleeds.[6] Maintain the patient's oxygen saturation at 100%. The importance of avoiding unnecessary sedatives and paralysis cannot be overstated, as the most important determinant of whether a patient needs an EVD is the clinical exam. Basic maneuvers to control increased ICP should always be implemented including elevating the head of the bed to 30° or placing the patient in the reverse Trendelenburg position if a cervical spine injury is suspected.

It is desirable for the initial cerebral perfusion pressure (CPP) to be 60 mmHg or higher to diminish the risk of cerebral ischemia. The lower limit of an acceptable mean arterial blood pressure (MAP) is usually 70 to 80 mmHg.[7] Higher MAP goals may be indicated if the patient has elevated ICP with an intact cerebral autoregulation. Hyperosmolar therapy with an osmotic diuresis and a induced hypernatremia are commonly used with the effectiveness of the former dependent on adequate renal function. Hyperventilation to reduce the pCO_2 in the range of 30 to 35 mmHg can reduce ICP in the acute resuscitation phase by causing arterial vasoconstriction with lower pCO_2 goals increasing the risk of cerebral ischemia. **Hyperventilation to lower ICP should only be used for a short time as a bridge to another ICP management strategy given the risk of causing rebound intracranial hypertension once the brain pH equilibrates.** The use of osmotic diuretics, such as mannitol, can reduce ICP quickly. Unfortunately, any extended duration of use of osmotic diuretics places the patient at an increased risk for cerebral ischemia. Other methods to consider include the use of dexamethasone in patients with edema surrounding a neoplastic mass or abscess. Barbiturates such as pentobarbital and phenobarbital can be used to induce a coma and reduce ICP by decreasing the cerebral metabolic rate of oxygen but cause hypotension. Paralysis decreases intrathoracic pressure and facilitates venous drainage from the brain resulting in lower ICP. **It is imperative that an ICP monitoring device is put into place since the ability to do sequential neurologic checks is lost if barbiturates or paralysis are employed as a means of lowering ICP.** Consider obtaining an electroencephalogram. The commonly used modality of osmotic diuresis is still used in the acutely deteriorating patient but is limited because of the ischemic and metabolic complications related to its use.[8]

INDICATIONS

The primary indication for a ventriculostomy is to decrease elevated ICP unresponsive to less invasive management methods.[9] The presence of an EVD and the management of ICP have been shown to decrease mortality.[10] This may change in the future if other studies show no benefit.[11] Care of the traumatic brain injury did not show keeping ICP by monitoring at 20 mmHg and or less to be superior to care based on imaging and examination.[11] A ventriculostomy can also be performed to measure ICP, drain CSF to treat hydrocephalus, introduce contrast for radiographic studies, or collect CSF from the ventricular system.[12]

It is preferable to treat an identifiable pathologic process as soon as possible. This usually requires intervention by a Neurosurgeon. The Neurosurgeon may be able to take the patient to the Operating Room to place an indwelling ventricular shunt system in an expedited fashion.[13] The ICP can be monitored and reduced by draining CSF via a ventriculostomy in the Emergency Department if the patient continues to deteriorate and a definitive procedure cannot be immediately arranged.[14]

CONTRAINDICATIONS

Performing a ventriculostomy could precipitate or worsen brain herniation and hasten the patient's mortality if an infratentorial mass has been identified and it appears to be inducing upward pressure into the supratentorial compartment. A coagulopathy or thrombocytopenia makes a ventriculostomy dangerous to perform. The use of anticoagulants and antiplatelet agents by the patient increases their risk of hemorrhagic complications. Consider reversing these conditions by administering fresh frozen plasma and/or platelets prior to performing a ventriculostomy. A mild elevation in the international normalized ratio (INR) up to 1.6 may be acceptable to place a ventriculostomy.[15]

EQUIPMENT

- Skin razor
- Skin prep kit with povidone iodine or chlorhexidine solution
- Sterile gloves and gown
- Face mask with an eye shield or goggles
- Cap
- Skin marker
- Ventricular catheter with stylet
- Tunneling device for catheter
- Drainage tubing and collection system

- Measuring implement (e.g., ruler)
- Scalpel handle
- #15 and #11 scalpel blade
- 10 mL syringe
- 25 gauge needle
- Lidocaine with epinephrine, 1%
- Self-retaining retractor
- Twist drill with a ¼ inch bit
- Sterile towels or drapes
- Bipolar cautery
- Needle driver
- 3–0 nylon suture
- Suction source
- Suction tubing
- Suction catheters
- Pressure transducer
- Blunt tip catheter to probe palpate the dura, optional
- Spinal epidural needle to puncture the dura, optional
- Headlight, optional
- Curette, optional
- Three-way stopcock, optional

It is most useful to have either a commercially available ventriculostomy kit or assemble one to have available in the resuscitation area. Complete, disposable, and single patient use kits are convenient and cost-effective (**Figure 147-4**).

PATIENT PREPARATION

This procedure is often performed emergently. Explain the procedure and its risks and benefits to the patient and/or their representative if possible. Explain the general surgical risks and the risk of placement-related hemorrhage. If it will not unduly delay the treatment, obtain an informed consent to perform the procedure.

The patient should be fully monitored with a noninvasive blood pressure cuff, pulse oximetry, cardiac monitor, and end-tidal carbon dioxide monitor if available. Before the procedure, it is important to implement four steps summarized by the 4-C mnemonic of consent, coagulation labs, coagulation medications, and CT scanning. Coagulation medications should be reviewed, as the presence of anticoagulation medications (e.g., warfarin) or antiplatelet medications (e.g., aspirin) significantly increases the chance of a symptomatic placement-related hemorrhage and may alter the risk-benefit profile of the procedure. Obtain a complete blood count (i.e., hemoglobin, hematocrit, and platelet count) and a coagulation profile (i.e., prothrombin time, partial thromboplastin time, and INR) to ensure that the patient is not thrombocytopenic or coagulopathic. These may require reversal with the administration of fresh frozen plasma, prothrombin complex concentrate, and/or platelets. The head CT should be reviewed. It can determine the presence of an acute subdural or epidural hematoma and the location and extent of the hematoma, rule out the presence of a mass, and determine if there is any herniation. The head CT can be used to obtain measurements that aid in the placement of the EVD. This includes the presence of a midline shift, the distance from the skull to the ventricles, and the distance to the foramen of Monro.

FIGURE 147-4. An example of the contents of a commercially available ventriculostomy kit.

Set up the room and collect the necessary equipment. **Position the patient so their head is at the edge of the bed, the head of the bed is elevated to facilitate drilling and catheter placement, and there is adequate space for the Emergency Physician to work.** Perform a "time out" to confirm the side of the procedure, that consent was received, and that the CT and laboratory studies were reviewed. Shave the patient's head for 4 to 5 cm surrounding the burr hole entry point. The hair from the entire frontal area back to the ear may be shaved, but it is probably unnecessary.

Identify Kocher's point.[16-20] Kocher's point is found by identifying the glabella and measuring 10.5 cm posteriorly in the midline. Mark the point 2.5 laterally cm from the end of the line 10.5 cm posterior in the midline to identify Kocher's point (**Figure 147-5**). The final entry point should be centered in the midpupillary line. This is approximately 1 cm anterior to the coronal suture in the midpupillary line (**Figure 147-5**).

Clean the skin of any dirt and debris. Cleanse the area once with a chlorohexidine swab. Inject local anesthetic solution with epinephrine at the site of the entry. Inject local anesthetic solution with epinephrine over the forehead to block the innervation from the V1 branches providing sensation to the scalp in this region. Repeat the prep of the scalp with povidone iodine and allow it to dry. Dress in full sterile attire (i.e., hat, mask, eye shield or goggles, sterile gown, and sterile gloves). Drape the field using sterile towels or prefabricated drapes. **It is imperative that strict sterility is maintained throughout the procedure. When creating the sterile field, it is important that proper aim of the catheter is obtained before insertion. It is necessary to have the ipsilateral medial canthus and the tragus fully visualized in the skin preparation or for marks to these structures to be made before the skin preparation with a sterile marker to allow the proper aim of the catheter to be maintained.** Organize the equipment in the order that it is to be used during the procedure. This step minimizes fumbling for equipment when the catheter is in the parenchyma. Note the premade markings on the ventricular catheter or mark 4, 5, and 7 cm on the catheter prior to its insertion.

It is strongly encouraged to prepare the drill bit before the skin incision so that no time is wasted preparing the drill with the skull exposed. Choose the appropriate drill bit and insert it into the drill. Adjust the safety stop on the drill bit to the estimated skull thickness. Secure the safety stop on the drill bit firmly with an Allen wrench.

Consider the administration of prophylactic intravenous broad-spectrum antibiotics immediately prior to the procedure. The usual infecting organisms are *Staphylococcus epidermidis* and *Staphylococcus aureus*. Appropriate antibiotic coverage includes third-generation cephalosporins and penicillinase-resistant penicillins.

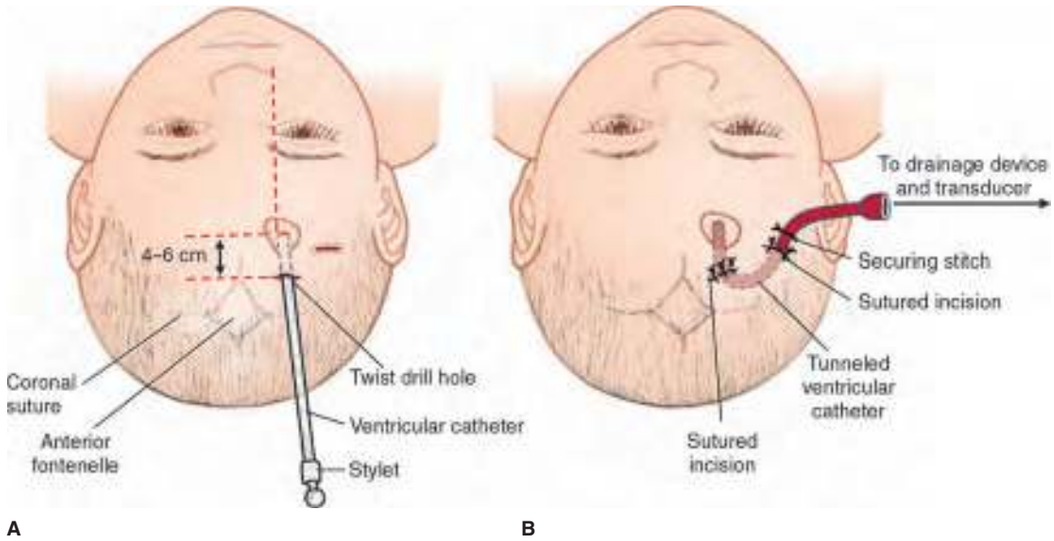

FIGURE 147-5. Placement of a ventriculostomy catheter. ***A.*** Insertion of the ventricular catheter. ***B.*** The catheter is tunneled and the skin closed.

TECHNIQUE

Infiltrate Kocher's point with 3 to 5 mL of lidocaine with epinephrine. Make the incision over Kocher's point marked on the skull. The size of the incision varies by institution. Little more than a stab incision is necessary to complete the procedure. A larger incision of 1 to 2 cm may be useful to visualize the trajectory of the catheter perpendicular to the skull, especially if a large amount of local anesthetic was used. Carry the incision from the skin surface down to and through the periosteum of the skull. Tie off or cauterize any bleeding vessels. Insert the self-retaining retractor into the incision to provide adequate exposure. It is a poor use of time to achieve absolute hemostasis while a patient is decompensating and awaiting the placement of the ventricular catheter. It may be useful to scrape the periosteum off the exterior surface of the skull to avoid skiving of the drill on the skull during drilling.

Aside from placement of the catheter, drilling the skull is one of the highest risk steps in the procedure. Carefully and slowly drill through the outer and inner tables of the skull. **Take care not to penetrate the dura. Do not apply too much force against the drill to prevent plunging the drill bit into the brain.** It is important to develop a tactile feedback as the drill passes through different layers of skull. If there is an anatomy lab or a cadaver dissection available, the best practice is to make multiple passes through the skull on a cadaver model. Drilling has three stages with its own tactile feedback. The bone is dense and tough as the drill passes through outer table. The drill may catch on the skull. The cancellous bone is more easily passed once through the outer table. The drill should not catch until it approaches the inner table. Rotate the drill using the hand crank while slightly pulling back on the drill away from the brain. The threads of the drill will pull it through the inner table. **Stop drilling when a loss of resistance is felt.** Flush the hole with warm sterile saline to remove any debris. Carefully make an opening in the dura (i.e., a controlled durotomy) with an 18 gauge needle or a #11 scalpel blade.

Place the catheter. Placement of the catheter can be associated with trauma to the brain, especially if an incorrect trajectory is used. An incorrectly placed catheter may easily penetrate vital structures such as the thalamus or the prepontine cistern and lead to hematomas. A few basic guidelines should be followed before insertion to avoid such complications. Aim the catheter in two planes. In the medial-to-lateral plane, aim the catheter to a line through the medial canthus of the ipsilateral eye. In the anterior-to-posterior plane, aim the catheter to a line through the ipsilateral tragus (**Figure 147-5*A***). These trajectory lines may be drawn before creation of the sterile field. The final trajectory of the catheter should be perpendicular to the skull.

A simple ventricular catheter or a fiberoptic catheter with a screw-in mechanism should be available. Apply gentle pressure while inserting the catheter into the brain matter and aiming for the medial canthus of the ipsilateral eye (**Figure 147-5*A***). A loss of resistance or a "give" will often be felt as the catheter enters the ventricle. There should be a return of CSF at a depth of 4 to 5 cm of catheter penetration. Withdraw the stylet and advance the catheter 1 cm farther. **Do not insert the catheter more than 7 cm deep.** If no CSF is obtained, withdraw the catheter and reinsert it while aiming slightly more medially and posteriorly, as the position of the ventricles may be distorted by the underlying pathologic process. Attach the pressure transducer to measure ICP.

If CSF is not obtained after the third pass, it is important to stop. Leave the catheter in place and obtain a head CT because multiple continuous passes may increase the risk of hematoma formation.[21] Reposition the catheter to obtain ventricular placement if the head CT shows an obvious correction that could be made to access the ventricle.[22] If CSF is still not obtained, take the catheter out and possibly place a pressure monitor or other monitoring device that does not require precise ventricular placement.

ALTERNATIVE TECHNIQUES

The gathering of equipment, making the burr hole, and the insertion of the ventriculostomy catheter can take significant time and experience. The required equipment may not be readily available. An easier, quicker, and simpler technique is to perform a transorbital decompression.[23,24] This technique is adapted from the frontal lobotomy procedure which was developed in the 1930s. Clean and prep the skin of the eyelids and periorbital area. Moisten sterile gauze with sterile saline. Instruct an assistant to grasp the upper eyelid with a piece of saline-moistened gauze. Instruct the assistant to retract the upper eyelid outward then upward and maintain this position (**Figure 147-6*A***). Grasp a piece of saline-moistened gauze in the nondominant hand. Apply this to the sclera at the 12 o'clock

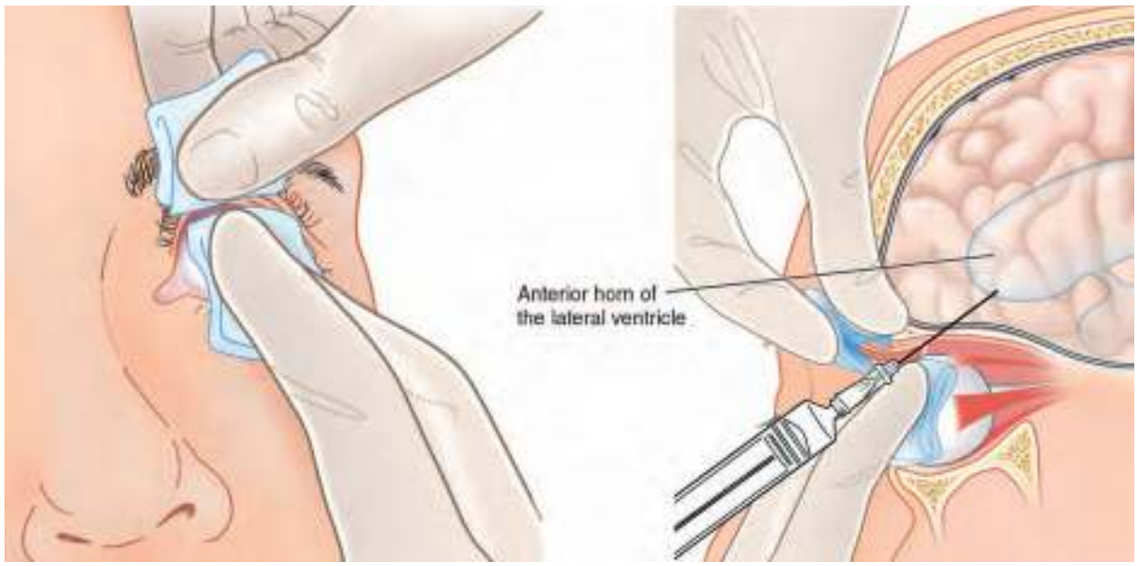

FIGURE 147-6. Transorbital ventricular decompression. ***A.*** The upper eyelid is retracted and the globe is displaced downward. ***B.*** Insertion of the needle.

position and displace the globe downward (**Figures 147-6*A* and 147-6*B***). **Do not place the gauze on the cornea as this can cause a significant corneal abrasion.** Apply an 18 gauge, 1.5 to 2.5 inch angiocatheter or spinal needle on a syringe. Insert the needle through the orbital roof, 1 cm posterior to the superciliary arch (**Figure 147-6*B***). Aim the needle toward the coronal suture at the midline or vertex of the skull. Advance the needle approximately 3 cm so that its tip enters the anterior horn of the lateral ventricle. Gently aspirate after the needle is inserted to a depth of 3 cm and CSF is aspirated. If using an angiocatheter, the soft plastic catheter can be secured to the orbital roof with a suture and the ICP monitored through the pressure transducer.

A relatively new three-dimensional ultrasound phased array transducer has been developed for burr holes.[25] It is 4 to 10 mHz in power. The transducer visualizes the ventricles to increase the EVD placement on the first pass.

AFTERCARE

It is desirable to tunnel the drainage tubing posteromedially subcutaneously for several centimeters to reduce the risk of infection (**Figure 147-5*B***).[2,26] Suture the ventricular catheter and tubing to the skin so that it remains in place (**Figure 147-5*B***). Close the skin around the ventricular catheter with 3–0 suture using a figure-of-eight technique (or a running suture if a larger incision was made). Secure the catheter to the skin using a simple drain stitch. If a pressure transducer is being used, obtain a measurement prior to allowing any quantity of CSF to drain. Attach the sterile fluid collection system. If a simple drainage bag is being used, ensure that it is kept at the level of the ventricle to avoid overdrainage. **Frequently recheck and document the patient's neurologic status.** It is useful to send a baseline sample of CSF for culture, cell count, chemistry, and cytology (if applicable) after ventriculostomy placement.

PEDIATRIC CONSIDERATIONS

Young children have an open anterior fontanelle. This large area devoid of bone is ideal for the introduction of a needle to drain CSF from the ventricular system. This procedure may be required in cases of acute patient decompensation due to an acute nonobstructive hydrocephalus (e.g., meningitis), an acute obstructive hydrocephalus (e.g., brain abscess, brain tumor, epidural hematoma, intracranial hemorrhage, or subdural hematoma), or the acute decompensation of a chronic hydrocephalus (e.g., aqueductal stenosis, posterior fossa tumors, or other congenital malformations).[12]

The ventriculostomy procedure in a young child is similar to that in an older child, adolescent, and adult with a few exceptions. A burr hole is not required as the anterior fontanelle allows an easy access point. Use an 18 or 20 gauge, 1.5 inch long angiocatheter instead of the spinal needle. The procedure is similar to inserting an intravenous catheter. Insert the angiocatheter perpendicular to the skin and at the most lateral aspect of the anterior fontanelle or the coronal suture to avoid the midline sagittal sinus. Stop advancing the angiocatheter when CSF flows from the hub of the needle. Slightly advance the plastic catheter while withdrawing the needle. Apply the pressure transducer to the hub of the catheter to measure ICP. Remove enough CSF to decrease the ICP to an appropriate level or until the signs and symptoms of herniation are reversed.[26,27] Remove the catheter or secure it to the scalp with suture. The secured catheter can be capped or left attached to the transducer to continue to monitor ICP.

COMPLICATIONS

The most common complication of placement of an emergent ventriculostomy is a CSF infection.[28,29] The risk of infection increases the longer the drain is left in place. Infections are rare for catheters in place for fewer than 4 days. Tunneling the drainage tubing, adhering to strict aseptic technique, and administering prophylactic antibiotics can reduce the incidence of infection.[28,30] Antibiotic-coated catheters tend to decrease the rates of infection.[31]

Ventricular puncture can result in an acute bleed in the subdural, intraparenchymal, or ventricular spaces. This can occur from direct trauma to the vascular system or excessive CSF drainage that shrinks the brain and tears the bridging vessels to the subdural space. It is estimated that the risk of hemorrhage associated with a ventriculostomy is 5.7%, with 1% of ventriculostomy patients having a clinically significant hemorrhage.[32] **An emergent head CT is indicated if the patient's condition deteriorates further after ventriculostomy**

placement. Patients should always be tested for normal blood clotting function prior to performing a ventriculostomy.

The most feared complication of this procedure is plunging with the drill bit uncontrollably into the brain substance. This procedure should not be performed by those unfamiliar with and untrained in the technique. Apply the minimal amount of pressure to the drill so that the bit penetrates the bone. **Never use a hand twist drill without the safety stop.**

SUMMARY

Patients with traumatic brain injury, acute nontraumatic intracranial hemorrhage, or acute or chronic hydrocephalus from infectious and noninfectious etiologies who present with a rapidly deteriorating neurologic exam may have elevated ICP and/or be in the process of brain herniation. There are several tools in addition to emergent ventriculostomy placement that can temporarily stabilize the patient's changing neurologic status. These include intubation, sedation, hyperventilation, osmotic diuresis, maintenance of cerebral perfusion pressure, dexamethasone administration, and barbiturate coma. Although the ultimate goal is to treat the primary pathologic condition, this chapter was designed to describe the use of CSF drainage via a ventriculostomy and its use in the context of the other methods commonly used to reverse the life-threatening complications of an acutely increasing ICP.

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148 Ventricular Shunt Evaluation and Aspiration

Daniel W. Weingrow and Jacob F. Lentz

INTRODUCTION

Pediatric and adult patients with ventricular shunts frequently seek medical attention in the Emergency Department (ED) with complaints that may or may not be caused by a malfunction and/or infection of these indwelling devices.[1-3] Patients with shunts may present with clinical entities as benign as a viral upper respiratory infection or with a life-threatening condition such as hydrocephalus. The wide range of possibilities is a challenge to the Emergency Physician's (EP's) diligence and clinical acumen. The challenge for the EP is to determine if the shunt system is functioning properly and if it is a direct cause of the patient's acute problem. This chapter will discuss the complications of ventricular shunt complications and includes tapping the shunt reservoir in the appropriate circumstance.

Ventricular shunts are lifesaving devices that are particularly prone to complications.[4] Complications of shunts typically occur soon after placement or revision.[1,5,6] Approximately 30% of patients who undergo a shunt revision present to the ED shortly after discharge. Children who had shunts placed as infants will require two shunt revisions secondary to obstruction within their first 10 years.[7] The overall shunt infection rate is 10% to 20%.[7-9] Approximately 90% of these infections will present within 3 months of the shunt placement.[7] Routine injection of the reservoir when placing it may decrease the infection rates.[10] The mortality of shunt-related complications has been estimated to be 4.6%.[11] This rate is highly dependent on the type of complication as well as the patient's initial neurologic examination. There is an enormous financial cost associated with ventricular shunts and their complications.[11] The use of best practice guidelines may decrease infection rates.[12] All of the above statistics apply to the population with hydrocephalus treated using indwelling ventricular shunt devices. Many of the complications discussed in this chapter may be greatly reduced in the future with the development of neuroendoscopic techniques.[13] Patients will continue to present frequently to the ED with a variety of problems related to their ventricular shunts. **Unless stated otherwise, the reader should assume that reference is being made to the more common ventriculoperitoneal (V-P) shunt device.**

Undershunting is the most common complication and can result from obstruction, malfunction, or breakage of the device.[14] The next most common category of shunt complication is infection despite being impregnated with an antimicrobial coating. Overshunting is the next most common complication. It may be associated with overshunting syndrome which may be associated with a subdural hematoma, hygroma, and slit ventricle syndrome.

Seizures tend to occur at a high rate after shunt placement. There is controversy as to whether shunt placement is the cause of the seizure or whether seizure activity is actually related to other associated conditions.[15] Patients may have problems related to the distal catheter (e.g., peritoneal hernia for abdominal shunts or endocarditis for atrial shunts).[16] There are other unique complications related to ventriculoatrial (V-A) and ventriculopleural shunt systems that will be discussed separately. There are also complications unique to infancy and childhood (e.g., cranial deformities) that are not managed in the ED.

The patient's significant other or caretaker will usually be familiar with the patient's baseline behavior and the signs and symptoms associated with previous shunt malfunctions.[17] **They represent important sources of collateral information when taking a history.** Neurosurgeons are adept at the evaluation of the ventricular shunts and are a critical resource when a shunt requires revision. **The EP bears the responsibility to recognize a shunt malfunction and other acute diseases that may masquerade as a shunt malfunction. It is in the best interest of patient care that the EP work with patients, families, and Neurosurgeons to achieve the optimal management.**

ANATOMY AND PATHOPHYSIOLOGY

Hydrocephalus is defined by an excessive quantity of cerebrospinal fluid (CSF) and is the condition most frequently associated with the initial need for a ventricular shunt or a shunt revision due to the malfunction of a preexisting shunt. An increase in the volume of CSF can overwhelm the nervous system's compensatory mechanisms and lead to an increased intracranial pressure (ICP). This in turn can lead to problems with cerebral perfusion and central nervous system (CNS) herniation. Obstructive hydrocephalus can cause dilation of the temporal tips of the lateral ventricles, produce compression of the uncus of the temporal lobe against the brainstem, and lead to herniation. Nonobstructive hydrocephalus can cause pressure on the cerebellar tonsils leading to brain herniation.[18] Increased ICP due to hydrocephalus can be temporized with acetazolamide therapy and ultimately managed surgically by shunt placement.[19]

Hydrocephalus is usually due to decreased CSF absorption. It may rarely be caused by increased production (e.g., a choroid plexus papilloma) or because of cerebral atrophy (e.g., hydrocephalus ex vacuo). Hydrocephalus ex vacuo is a radiographic diagnosis that does not require surgical intervention. **There are two main subdivisions among patients with decreased CSF reabsorption, noncommunicating hydrocephalus, and communicating hydrocephalus. Patients have an obstruction proximal to the arachnoid granulations preventing the normal flow of CSF in noncommunicating hydrocephalus. Patients have unobstructed flow of CSF between the choroid plexus of the ventricular system and the spinal cord but suffer from diminished reabsorption at the arachnoid granulations in communicating or nonobstructive hydrocephalus.** This most frequently occurs in patients who have had a hemorrhage or infectious process that resulted in particulate matter interfering with the normal reabsorption of CSF.

Normal pressure hydrocephalus features the radiographic appearance of hydrocephalus with dilated ventricles along with the clinical triad of ataxia, urinary incontinence, and dementia. Normal pressure hydrocephalus is most common in the elderly population. It is, as the name suggests, associated with a normal ICP. The patient usually accumulates CSF due to decreased absorption.

Once the diagnosis of hydrocephalus is made, the Neurosurgeon will be responsible for treating the primary causes (e.g., tumor, bleed, congenital abnormality) as well as placing an indwelling ventricular shunt. The components of a standard shunt system include a ventricular catheter, a one-way valve, and distal tubing that enters the cavity into which the fluid is being shunted (**Figure 148-1**). It is a standard practice to enter the right lateral ventricle through a burr hole in the skull. The proximal catheter is then situated in the right lateral ventricle. It has numerous openings to prevent the choroid plexus from blocking the catheter outflow from the ventricles (**Figure 148-2**). The valve system is placed subcutaneously along the right temporoparietal region. The distal catheter is tunneled subcutaneously along the skull, lateral neck, anterolateral chest wall, and finally into the peritoneal space just superior or lateral to the umbilicus (**Figure 148-3**). The shunt system may occasionally be found on the left side in some patients.

There are two main types and many kinds of valve systems (**Figure 148-4**). The differential pressure valve allows the Neurosurgeon to select a low-, medium-, or high-pressure system. The CSF will flow out of the ventricle through the one-way system when the CSF pressure or CSF flow rate builds to a specified level. They are

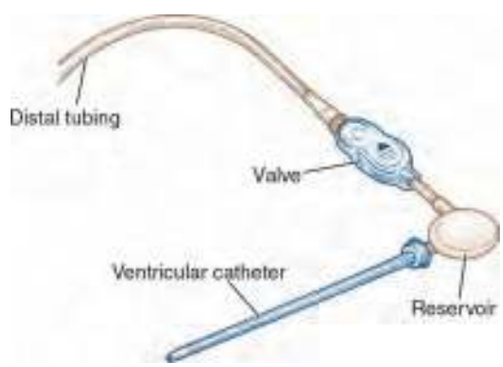


FIGURE 148-1. The components of a ventricular shunt.

FIGURE 148-2. The intraventricular catheter with holes to prevent the choroid plexus from blocking it.

adjusted with magnetic tools. A variable resistance constant flow valve keeps CSF rates constant and provides a more physiologic system by varying the resistance in the valve as the individual changes position. Some of these valve systems are palpable on physical examination but do not allow access to the CSF for aspiration. A single or double bubble reservoir (i.e., two bubbles in tandem) for the draining of CSF is a common addition to the standard shunt system (**Figure 148-5**). The reservoir can be inserted anywhere in the system. It is most frequently adjacent to the valve mechanism on the lateral aspect of the skull (**Figure 148-3**).

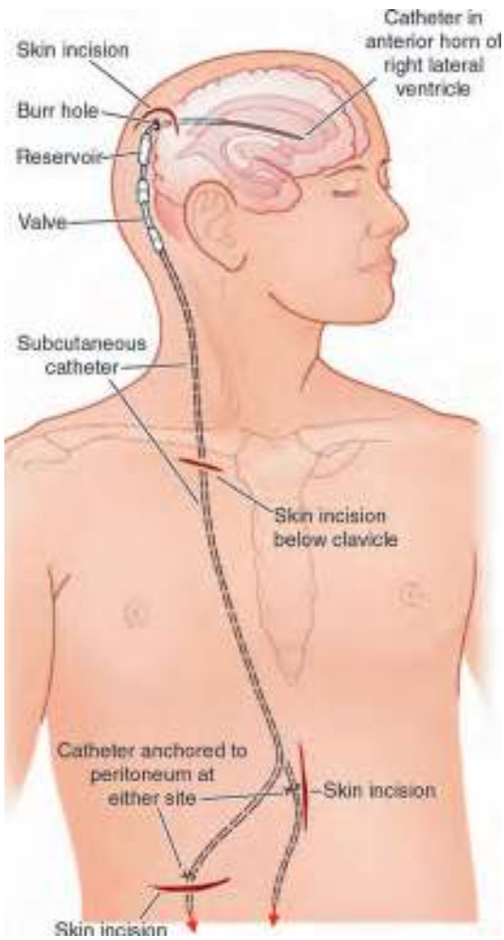


FIGURE 148-3. The anatomic pathway of a ventriculoperitoneal shunt.

FIGURE 148-4. Examples of different shunt valves. Flow is in the direction of left to right.

The distal catheter has a wire spring embedded in the length of its silicone tubing in order to decrease kinking. The distal tip has numerous openings to prevent blockage. A small incision into the peritoneal cavity allows the distal catheter tubing to exit the subcutaneous tissue and enter the peritoneum. A generous portion of tubing is left free floating inside the peritoneal cavity. This extra tubing allows for the distal end to lengthen as the patient grows and lends further stability to its place within the abdominal cavity. The proximal and distal catheters are impregnated with radiopaque markings to allow verification of the catheter position on radiographs.[20]

There are many types of configurations of the distal catheter placement. The distal tubing in ventriculoatrial and ventriculopleural shunt systems will terminate in the right atria and right

FIGURE 148-5. Example of a double bubble system.

pleural cavity, respectively. These configurations are not used frequently but it is important to be aware of them. Ventriculoperitoneal shunts have been the preferred modality since the 1960s for most types of hydrocephalus due to their lower complication rate.[21,22] The EP may come across a patient with a lumboperitoneal shunt that avoids any direct surgical invasion of the brain.[23] The proximal catheter is placed in the lumbar subarachnoid space, the valve and reservoir system are above the iliac crest, and the distal tubing is tunneled subcutaneously in the abdominal wall ending into the abdominal cavity.

A patient with a ventricular shunt is subject to a host of complications from mechanical malfunctions that may occur anywhere in the path of the shunt system.[23] **As with any indwelling medical device there is a risk of infection.** The remainder of this chapter will guide the EP through the process of identifying the various types of shunt problems.

SIGNS AND SYMPTOMS

The clinical presentation of hydrocephalus varies with its severity, rate of progression, and age of the patient. Preverbal pediatric patients with open fontanelles typically have a nonspecific examination. Further collateral information from caretakers is usually necessary to make the diagnosis. Experienced caretakers are accurate in diagnosing shunt malfunction.[24] **It is crucial that the EP rely upon collateral information to distinguish shunt malfunction from other acute or nonacute disease entities.** Isolated bradycardia without hypertension is common in children with a shunt malfunction.[25]

Pediatric patients with a recent ventricular shunt insertion who have nausea and vomiting, irritability, decreased level of consciousness, and a bulging fontanelle are predictive of a shunt malfunction.[26] Only decreased level of consciousness and loss of developmental milestones were found to be significant predictive factors in patients with presentations late after shunt insertion.[26] Lethargy and shunt site swelling were signs of shunt malfunction in the pediatric population.[27]

Cranial nerve palsies (e.g., supranuclear upward gaze palsy or Parinaud's syndrome [i.e., sunset eyes or the setting sun sign]) can result from compression of the mesencephalic tectum by a dilated suprapineal recess in patients with more severe hydrocephalus.[28,29] This can give the patient a "scared" or surprised appearance and cause the patient to extend their neck to compensate. The sixth cranial nerve's long intracranial course leaves it susceptible to compression which can manifest as unilateral or bilateral palsies.[30]

Elevated ICP can manifest as an engorgement of the scalp veins due to reversal of flow from the intracerebral sinuses.[31] This may result in a "puffy eyes" appearance, the subtlety of which may escape the EP who is unfamiliar with the patient's normal appearance.[32] Papilledema is seen in only 2% of patients with shunt malfunction.[33]

Symptoms of shunt malfunction are similar to increased ICP in other disease entities in patients who are verbal with closed sutures.[34] They may describe headaches that are classically worse in the morning after awakening from sleep due to relative hypoventilation during sleep which dilates the cerebral veins and the head being at the level of the heart. They may exhibit ataxia and other movement disorders (e.g., hyperreflexia and spasticity that involves the legs greater than the arms).[35] Nausea and vomiting may be present due to increasing pressure on the vagal nucleus of the area postrema (i.e., the "vomiting center").[36] Patients will display diminishing levels of responsiveness, coma, and an associated Cushing reflex (i.e., bradycardia and hypertension) as ICP continues to rise and prior to the terminal event of herniation.[30]

Examine the shunt from the site of the burr hole to its insertion cavity. Much of the shunt is palpable. Begin at the burr hole on the scalp. Examine the surrounding scalp carefully for any bogginess (i.e., indicating that CSF may have extravasated from the system), erythema, tenderness, or warmth. Examine the suture lines. A shunt reservoir may be palpated under the scalp. It should typically feel like a fluid-filled bubble. Gently compress the bubble to assess if there is brisk refilling of the CSF. Quick filling of the reservoir is an indication that the proximal portion of the system may be patent.[37,38] **This is by no means a perfect test.[37,38] Resistance to compression indicates a distal shunt malfunction. Avoid pumping the reservoir repeatedly.**

A double bubble reservoir system may be palpated. Compress the proximal reservoir to empty it and fill the distal reservoir. Compress the distal reservoir while still compressing the proximal reservoir. **Any resistance to compression of the distal reservoir indicates a distal malfunction with the shunt system.** Release the proximal reservoir while still compressing the distal reservoir. It should refill very quickly. **A delay in filling of the proximal reservoir indicates a proximal malfunction.**

Continue to palpate the temporal region of the scalp and the neck for any gaps in the system that could indicate a disconnection. The individual components of the shunt system are commonly joined by small connectors that can dislodge from one another. Continue to palpate the tubing along the neck and chest until reaching the abdomen and note any gaps. It is common to fracture the tubing in the area over the clavicle and lower ribs. Perform a thorough abdominal examination.

The distal end of the shunt may not function for several reasons. The tip may have withdrawn out of the peritoneal cavity and into the preperitoneal fat or subcutaneous tissue. This may have occurred because the patient had grown and used up the extra length of catheter that was originally left in the peritoneal cavity. The distal tip can migrate and cause a bowel perforation with or without resultant peritonitis, bowel obstruction, hernias, pleural effusions, and a hydrocele.[20,39]

Intraperitoneal infections may cause loculations around the catheter tip and form a pseudocyst.[40,41] It is important to note that these patients may not have any signs of a systemic infection. Additional causes of distal catheter obstruction include kinking of the intraabdominal tubing, debris collection around the tube openings, compression secondary to pregnancy, or other processes that increase the intraabdominal pressure.

Unexplained fever should prompt an evaluation of the distal catheter as a possible nidus of infection in patients with ventriculoatrial shunts.[42] A new murmur, skin findings consistent with endocarditis, or unexplained end-organ dysfunction should raise the possibility of bacterial seeding from the distal port.[43]

INDICATIONS

The EP is obliged to assess the patency, placement, and integrity of the shunt system when a patient presents with any symptoms even remotely related to hydrocephalus. The presence of systemic signs (e.g., fever, tachycardia, or hypotension) or local signs of infection (e.g., warmth, tenderness, bogginess, or redness along the shunt tract) is a reason to consider aspirating CSF from the system. This is not to say that every child with an upper respiratory infection requires that the shunt be tapped.[3] It is not uncommon to have overlapping findings in that an infection may obstruct the system and therefore require both mechanical and infectious complications to be addressed. **The withdrawal of CSF from the shunt can be lifesaving and sustain the patient who is herniating, in extremis, or deteriorating neurologically until a Neurosurgeon can repair the shunt.**

CONTRAINDICATIONS

There are no contraindications to performing a thorough physical examination of the shunt system, obtaining a head computed tomography (CT), and obtaining plain radiographs to assess its integrity. There is some controversy that surrounds the tapping of a shunt by personnel other than a Neurosurgeon.[44-46] **It is possible to disrupt the pressure and valve mechanism by the manipulation of the needle that is required to perform the tap. The reservoir may develop a leak if the correct technique is not employed or if the shunt is tapped too frequently.** There is the risk that bacteria will be introduced into the system. Many patients have had multiple complications related to both a primary disease and the shunt, with some already having undergone a number of shunt revisions. **It is prudent to assume a conservative approach with each of these patients and consult a Neurosurgeon prior to tapping the shunt.**

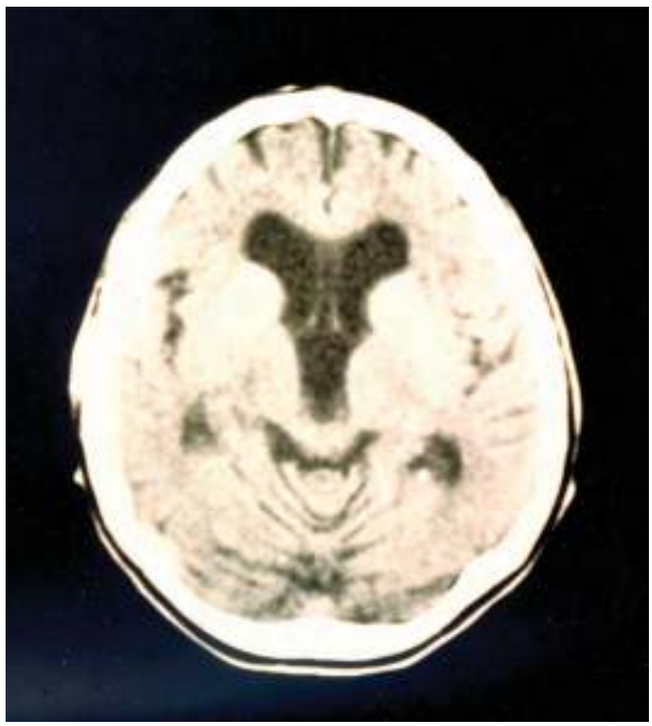
SHUNT ASSESSMENT THROUGH IMAGING

The history and physical are critical in determining if a patient's symptoms are due to shunt malfunction or an alternate diagnosis. No single collection of signs or symptoms is sensitive in ruling in or out ventricular shunt malfunction.[26,27,38] An EP's judgment along with the patient's and caretakers' assessment of symptoms can obviate the need for advanced imaging if the presenting complaint can confidently be attributed to a cause other than a malfunctioning shunt.[17] Imaging of the shunt system and the brain may be required in cases where there is concern for shunt malfunction but clinical uncertainty remains. The EP must use clinical judgment to balance the risk of a shunt malfunction with the cost and radiation associated with advanced imaging.

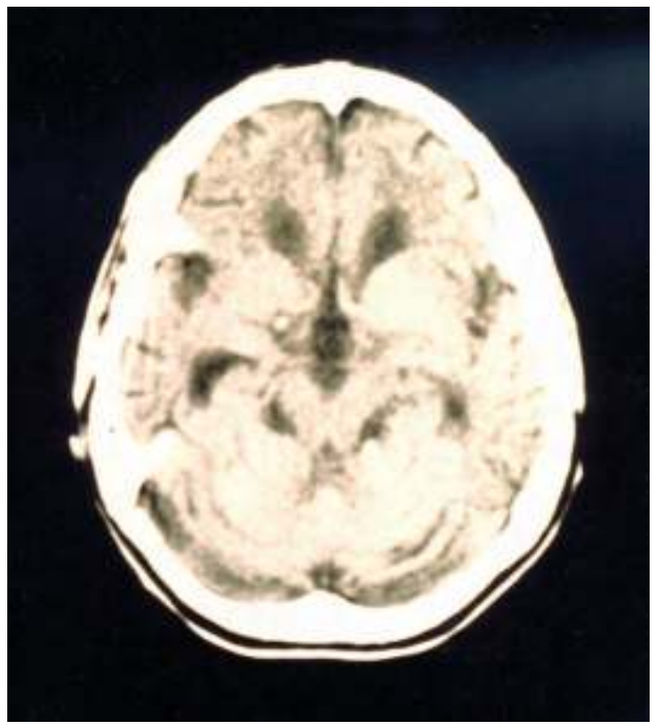
Both CT and magnetic resonance imaging (MRI) can aid in visualizing the ventricular shunt, characterize and grade the severity of hydrocephalus, and demonstrate secondary signs of increased ICP.[47] **It can be difficult to determine if there is a change in the ventricular system because preexisting congenital or chronic abnormalities can give the ventricles an abnormal appearance at baseline. The best approach is a comparison to a previous scan.**[48]

The EP should not feel completely reassured by a scan demonstrating ventricles that are "normal" or "unchanged." Patients with a shunt malfunction may not have radiographic findings supporting the diagnosis.[48,49] There are multiple explanations for this finding. Patients with small ventricles may have noncompliant pressure volume curves requiring extremely high ICPs for significant changes in ventricular size. Small ventricles may be seen in overshunting where the CSF is draining too vigorously. This results in slit ventricle syndrome or intracranial hypotension in which the symptoms are similar to a spinal headache.[50] **It is important to carefully examine the CT scan for any subdural hematomas or hygromas that can result from the brain shrinking away from the dura and tearing bridging veins from a siphoning effect of overdrainage of CSF.**[51]

Scans of patients with both obstructive and nonobstructive hydrocephalus can have a similar appearance to those patients with hydrocephalus ex vacuo due to loss of brain tissue. This can complicate distinguishing normal pressure hydrocephalus from cerebral atrophy in patients with dementia. Two main measurements are used to distinguish these entities. The first is a size greater than 2 mm of the temporal horns of the lateral ventricles when the sylvian and interhemispheric fissures are not visible on the CT or MRI image.[52] A bilateral frontal horn width greater than half the width of the internal diameter of the skull at the same level is suggestive of hydrocephalus (**Figure 148-6**).[52] In pediatric patients, a ratio of



A



B

FIGURE 148-6. Head CT of a patient with hydrocephalus. ***A.*** The ventricles are enlarged with effacement. ***B.*** Pronounced temporal horns.

the frontal horns of the lateral ventricles to the maximal biparietal diameter as measured in the same slice greater than 0.3 suggests hydrocephalus.[53]

MRI and CT scans can show signs of increased ICP beyond simple hydrocephalus. These include impending herniation wherein the brain appears to be under increased pressure with a loss of sulcal markings, a loss of differentiation between the gray and white matter, and/or an inability to visualize the fourth ventricle. **Herniation is characterized by compression of the basilar cisterns due to compression at the tentorial notch.[18]**

A MRI scan does not yet have the same ubiquitous availability to most EPs. It can give similar information to a CT scan when evaluating hydrocephalus.[54] An MRI has the added benefit of not exposing the patient to ionizing radiation.[55,56] The exposure to ionizing radiation has been implicated as a disproportionate risk factor for malignancy in pediatric patients receiving advanced imaging.[57] Pediatric patients have incompletely ossified calvaria which leads to increased radiation penetration of the brain. Most patients with ventricular shunts undergo many CT scans in their lifetimes due to malfunction, revision, periodic observation, and maintenance.[58-60] Limited CT scans of four slices instead of whole brain CT can be used if MRI is unavailable.[61] **The EP should coordinate management with the consulting Neurosurgeon to avoid unnecessary radiation from repetitive scans.**

A protocol was developed for the MRI scanning of pediatric patients with a suspected shunt malfunction.[58,62] The single-shot fast-spin echo MRI has a short scan time.[47,55,63-65] There were concerns of movement artifacts in obtaining MRI sequences in children. This is eliminated with a single axial T2 diffusion-weighted sequence in as little as 8 seconds which obviates the need for sedation.[66] The MRI does have the limitations of identifying bleeds and venous sinus thrombosis.[65] **Programmable ventricular shunts must be evaluated by a Neurosurgeon after an MRI. Programmable shunt valves tolerate up to a 3 Tesla (T) MRI without permanent damage, but the pressure setting may be altered and should be rechecked after having an MRI for any reason.[30]** There are now MRI-resistant programmable valves (e.g., Codman, Raynham, MA, and Medtronic, Minneapolis, MN).

A shunt series of plain radiographs can reveal any mechanical kinks, breaks, fluid collections, or disconnections along the shunt tubing. Shunt materials are impregnated with radiopaque substances and markings which allow the entire device to be visualized on plain radiographs. The threshold to obtain a plain film shunt series should be very low if an EP has the slightest suspicion of a shunt malfunction or infection.[67] **A shunt series consists of anteroposterior and lateral plain radiographs of the skull, neck, chest, and abdomen.[64]** These films are useful for assessing shunt positioning and the integrity of the connections within the system.[68] **Always review the lateral abdominal films as it is impossible to determine if the shunt tubing lies within the peritoneal cavity without it.** Inspect the entire path of the shunt system and note placement of the catheter inside the ventricle, the integrity of the connections around the valve and/or the reservoir, the identification of the distal catheter within the peritoneal cavity, and any kinks or breaks in the system. Examine the chest radiographs closely for any pleural effusion or pneumothorax from migration of the distal end of the tubing.[39]

Radionucleotide scanning with technetium along with manometry is highly sensitive for shunt malfunctions when a high suspicion of shunt malfunction exists but CT or MRI imaging is unremarkable. This scanning technique is invasive and involves radiation exposure.[69] The EP should be aware of these highly sensitive tests. It should be left to the discretion of the consulting Neurosurgeon to make the determination for radionucleotide scans.

SHUNT ASPIRATION

The indications for tapping a shunt reservoir in the ED include the following: to obtain a CSF specimen if there is a clinical concern of a shunt infection; to evaluate shunt function if there is suspected obstruction; and as a temporizing measure in a potentially distally occluded shunt when the patient is in extremis, deteriorating neurologically, or has signs of herniation on CT scan.[70] This can temporarily restore cerebral perfusion and sustain the patient until a Neurosurgeon can repair the shunt. **It is recommended that a Neurosurgeon be consulted and given the first option to perform the procedure.** The Neurosurgeon may have already manipulated the hardware multiple times and will ultimately be responsible for any shunt revisions and follow-up.

Patients with ventriculoatrial shunts are a particular concern when there are any signs of an infection. The distal catheter sits at the intersection of the right atrium and the superior vena cava. This puts the patient at risk for life threats such as endocarditis and septic emboli. It may be possible to evaluate the majority of shunt malfunctions without tapping the device.[44]

Tapping a ventricular shunt is a quick and simple procedure. Palpate the lateral aspect of the skull for the reservoir. Plain radiographs or bedside ultrasound may be helpful if it is difficult to identify upon palpation.[71] Prepare for the procedure by having the following equipment available: four sterile fluid specimen tubes with tight-fitting tops (those supplied in the standard lumbar puncture kit are ideal), a 23 to 25 gauge butterfly needle, a 5 to 10 mL syringe, a shave-prep set, an antibacterial cleansing preparation (e.g., povidone iodine or chlorhexidine solution), a fenestrated drape, and sterile personal protective equipment. A lumbar puncture tray supplemented with a butterfly needle and attached tubing will provide all the supplies. Consider the application of cerebral regional tissue oxygen (rSO_2) before, during, and after the tap.[72] It may help in the detection the malfunction.

Local anesthetic is not required for this procedure. Place the patient in whatever position is mutually comfortable. Shave a small patch of hair overlying the reservoir. Clean the skin of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. **This procedure requires strict aseptic technique.** The EP should don full sterile and personal protective equipment at this point. This includes sterile gloves, a sterile gown, a face mask with an eye shield or goggles, and a cap. Place the drape over the patient's head so that the fenestration overlies the reservoir and prepped skin. Set up a bedside table with a sterile drape and all the supplies.

Insert the needle at approximately 45° from the vertex of the reservoir bubble with the tip pointed toward the center of the reservoir (**Figure 148-7**). This is where the greatest amount of CSF is located. **Avoid inserting the needle into any of the connections or internal pressure mechanisms that may be at either end of the reservoir.** Allow the CSF to passively drain from the butterfly tubing into sterile tubes.

Spontaneous CSF flow indicates that the proximal portion of the ventricular shunt is not completely occluded. Attach a manometer to the end of the butterfly tubing to measure the opening pressure. Alternatively, hold the length of the butterfly needle tubing upward as the length of a typical tube is approximately 30 cm. Manometry readings indicating that the intraventricular pressure is greater than 20 cmH_2O is a concern for distal obstruction.[73] The normal pressure is 10 to 20 cmH_2O in a relaxed lateral recumbent patient with the reservoir facing upward. Attempt to gently aspirate CSF with a 5 mL sterile syringe if there is no spontaneous CSF flow. The proximal (i.e., ventricular) end is likely obstructed if CSF is difficult to aspirate.[46] Withdraw the needle from the shunt reservoir and apply pressure to tamponade any bleeding.

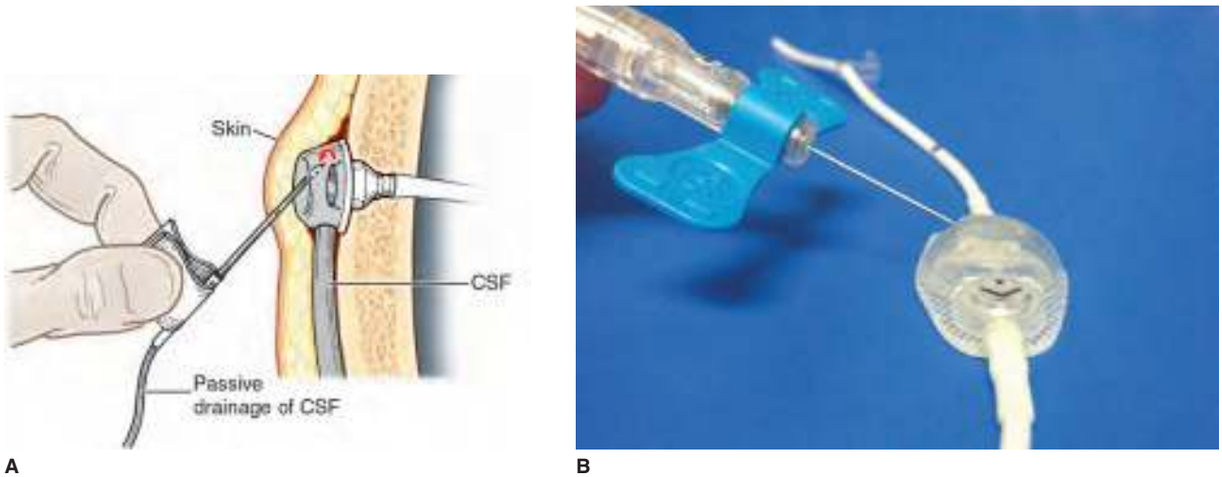


FIGURE 148-7. Tapping the shunt reservoir. ***A.*** Artist Illustration. ***B.*** A shunt and butterfly needle. (Used with permission from reference 6.)

Label the tubes of CSF and have them transported to the laboratory for a cell count and differential, Gram stain, culture (e.g., bacterial, viral, and fungal), and chemistry (e.g., protein and glucose). Obtain a sample of the patient's serum glucose at the same time so as to more accurately determine what level of glucose in the CSF to call abnormal. **A normal level of CSF glucose should not be any lower than 50% to 66% of the serum level.**

Obtain a nuclear medicine scan (i.e., a shunt-o-gram) if CSF cannot be aspirated to determine the proximal patency of the system. A radioactive isotope (e.g., technetium) is injected into the reservoir after manually occluding the distal shunt tubing. The flow, or lack thereof, of the radioactive isotope is then imaged.[74] This form of imaging can increase the sensitivity for the detection of a shunt malfunction.[75]

A lumbar puncture should never be performed in lieu of tapping the shunt. Obstruction of the shunt system can result in obstructive hydrocephalus. Lumbar puncture in a patient with obstructive hydrocephalus can cause a significant pressure differential and precipitate brain herniation.

CSF ASSESSMENT

The CSF obtained from the shunt is interpreted in the same way as CSF is interpreted from a lumbar puncture. A high white cell count with a large number of polymorphonuclear leukocytes, low glucose, and a high protein level indicates a high likelihood of bacterial infection. The positive Gram stain and culture make the definitive diagnosis.[75] There is no standard for the number of white cells that definitively indicates an infection. Some patients with indwelling shunts may have a chronically elevated CSF white blood cell count with more lymphocytes or eosinophils than polymorphonuclear leukocytes.[76] Refer to Chapter 142 for a more detailed discussion regarding the evaluation of CSF. **Antibiotic coverage should be initiated if a shunt is being tapped.** *Staphylococcus epidermidis* causes the vast majority of infections, with *Staphylococcus aureus* and gram-negative bacilli following in frequency.[77] The current management of shunt-related infections has been reviewed.[78] Empiric therapy includes vancomycin with either cefotaxime or ceftriaxone for children and vancomycin with rifampin for adults.[79] Consider additional coverage for methicillin-resistant *S. aureus* if indicated.

COMPLICATIONS

There are a few complications associated with the aspiration of a ventricular shunt. The introduction of bacteria into the reservoir can result in meningitis, peritonitis, brain abscesses, shunt occlusion, or infection anywhere along the course of the shunt tubing.[80,81] **It is of the utmost importance to maintain strict sterile technique.** The needle can damage the reservoir and result in a permanent hole necessitating its removal and replacement.[82] A misplaced needle can damage the connections or valves of the shunt system.

FUTURE CONSIDERATIONS

The use of bedside ultrasound can be very helpful. It can identify a fluid collection along the subcutaneous tubing as an abscess.[83] It can identify an extracranial tubing fracture by visualizing the abrupt ending of the tubing and an associated fluid collection.[68,84] The benefits of bedside ultrasound are that it can be readily repeated, lacks ionizing radiation, and is quickly available for an unstable patient. Transcranial sonography has some utility in patients with open fontanelles. The majority of studies on the use of transcranial ultrasound have been performed in the Neonatal Intensive Care Unit where germinal matrix intraventricular hemorrhage and subsequent posthemorrhagic hydrocephalus in premature neonates often necessitates surgical intervention.[85] Serial measurements of the lateral ventricles aid in the therapeutic evaluation of hydrocephalus. The precise reference values are critical, as intervention may be based on ventricular size changes of only a few millimeters.[86]

This research has expanded to include the sonographic estimation of ICP using optic nerve sheath diameter.[87] There have been reports of bedside ultrasound being used for the evaluation of problems with shunt tubing using spectral Doppler to evaluate presence or absence of flow in a ventricular shunt model.[88,89] Ultrasound can be used to locate a difficult to palpate shunt reservoir.[71]

The ShuntCheckT (NeuroDx Development, Princeton, NJ) is a device that allows the noninvasive detection of CSF flow through a shunt.[90] The procedure requires placing an ice cube over the shunt. A single patient use and disposable sensor is placed on the skin of the neck overlying the shunt tubing. The sensor is attached to the ShuntCheckT device with a cable. The ShuntCheckT analyzes the temperature readings from the sensor and determines if CSF flow is present or not.

SUMMARY

Patients with indwelling ventricular shunt devices are subject to many complications throughout their lives. These most commonly include obstruction, infection, and malfunction. The EP will see shunt patients of all ages who present with headache, fever, nausea, vomiting, seizures, irritability, or a change in mental status. The challenge is to determine whether or not the patient's complaint is related to the shunt. This chapter discussed some of the common shunt complications and an approach to their diagnosis. A careful history and a skilled physical examination will set the EP on a path toward a good patient outcome. The brain imaging, the plain film shunt series, and the shunt tap will prove most useful. A Neurosurgeon should always be consulted. A conservative approach to the diagnosis, management, and patient disposition is strongly recommended due to the life-threatening nature of shunt complications.

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149 Subdural Hematoma Aspiration in the Infant

Sarah Christian-Kopp

INTRODUCTION

Extra-axial fluid collections are described as fluid collection that are external to the brain parenchyma. These include epidural hematomas, subdural hematomas, and subarachnoid hemorrhages. Intra-axial fluid collections develop within the brain parenchyma. Extra-axial fluid collections in children are classified as symptomatic and asymptomatic. Symptomatic extra-axial fluid collections have been classified as effusions, hematomas, or hygromas. Their appearance on computed tomography (CT) scans and the treatment for each are identical (**Figure 149-1**).[1] CT scan images of symptomatic extra-axial fluid collections usually demonstrate ventricular compression and flattening or obliteration of the cerebral sulci on CT scans (**Figure 149-1**). Asymptomatic or benign subdural fluid collections usually appear as a hypodensity over the frontal lobes with dilation of the cortical sulci, interhemispheric fissure, and Sylvian fissure. The ventricles are usually normal in size or slightly enlarged with no evidence of transependymal flow.

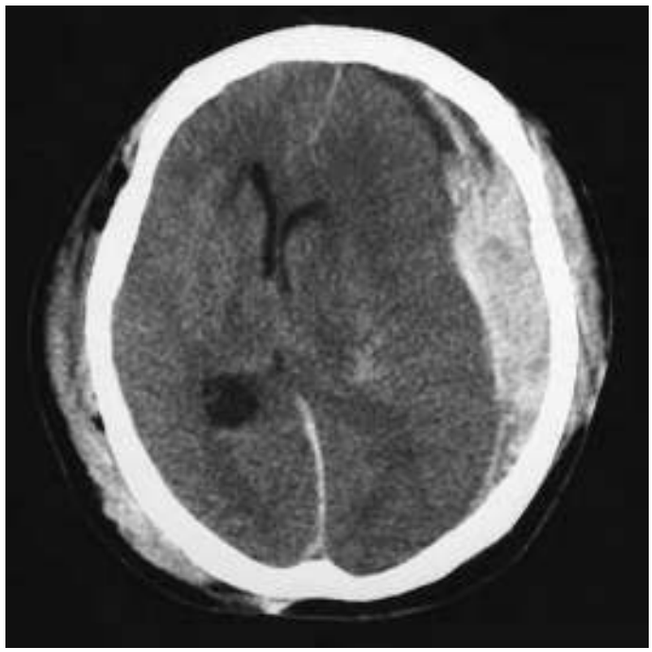

FIGURE 149-1. CT scan image of a subdural hemorrhage.

Common presenting symptoms of a symptomatic extra-axial fluid collection include a depressed level of consciousness, lethargy, irritability, a large head, seizures, or vomiting. The physical examination may reveal fever, a full fontanel, gaze paresis, hemiparesis, increased tone, lethargy, macrocephaly, and retinal hemorrhages. Markwalder has done an excellent review of the pathophysiology and experimental studies of chronic subdural hematomas.[2]

Most extra-axial fluid collections result from head trauma, whether due to accidental trauma or nonaccidental trauma (NAT) formerly known as abuse.[3] Other causes include the placement of a ventriculoperitoneal shunt and postinfectious bacterial meningitis. The etiology of intracranial hemorrhage and extra-axial fluid collections is quite varied (**Table 149-1**). Acute and chronic subdural fluid collections are not rare problems during infancy. Males are affected more commonly than females.

TABLE 149-1 The Etiologies for Intracranial and Extra-Axial Fluid Collections

Brain atrophy
Child abuse
Coagulopathy
Hydrocephalus
Infection
Masqueraders
Craniocerebral disproportion
External hydrocephalus in a child with ventriculomegaly and extracerebral fluid
Extracerebral space is enlarged and filled with CSF-like fluid from the head being too large for the enclosed brain
Neoplastic
Metastatic
Primary
Prior surgery
Spontaneous
Trauma
Vascular
Aneurysm
Vascular malformation
Ventriculoperitoneal shunt malfunction

Look for a clear history of injury or trauma in the presence of an acute or chronic subdural hematoma.[3] Consider the possible etiology of NAT if a history of an injury is not forthcoming or if the history does not make sense. It is incumbent upon the medical team to rule out abuse. This may require a period of inpatient observation, social services consultation, forensics consultation, a radiographic skeletal survey, a bone scan, and possibly an ophthalmological consultation. **The presence of retinal hemorrhages in association with a subdural fluid collection is highly suspicious for NAT.[4] Admission to the hospital for further observation is warranted if NAT cannot be ruled out.** Err on the side of precaution. The presence of congenital anomalies may predispose the child to subdural hematoma formation.[5] **An extra-axial fluid collection alone is not pathognomonic for NAT.**

Percutaneous removal of the subdural fluid is useful in diagnosing an active infection and rapidly lowering the intracranial pressure in the symptomatic patient.[6] Repeated removal of the fluid by percutaneous aspirations has been advocated by some Neurosurgeons for definitive treatment of chronic extra-axial fluid collections.[7,8] Subdural fluid collections in infants do increase in size, are often bilateral, can be difficult to diagnose, and are most often seen in children under the age of two years. The combination of CT and magnetic resonance imaging (MRI) is usually diagnostic.

ANATOMY AND PATHOPHYSIOLOGY

A subdural aspiration or tap is usually performed by puncturing the anterior fontanel. It can be approached through the coronal suture or the soft cranium. The diamond-shaped anterior fontanel is formed by the junction of the sagittal, coronal, and frontal sutures (**Figure 149-2**). It measures approximately 4 cm in the anteroposterior plane and 2.5 cm in the transverse plane. The bones of the infant skull are separated by connective tissue bands referred to as sutures. The fontanels are readily palpable at the junctions of the sutures. Six of the fontanels are located at the corners of the two parietal bones. Two of these, the anterior and posterior fontanels, are in the midline.

The anterior fontanel is the largest of the neonatal fontanels and is utilized for percutaneous subdural aspiration. The diamond-shaped anterior fontanel is formed by the junction of the coronal suture and sagittal suture (**Figure 149-2**). It measures approximately 4 cm in the anteroposterior plane and 2.5 cm in the transverse plane. The posterior fontanel is at the junction of the lamboidal and sagittal sutures. The anterior fontanel has usually closed by 18 months of age but can be patent up to the age of 2 years. The posterior fontanel usually closes by 6 to 8 weeks of age.

The anterior fontanel is readily palpable and bulging in cases of symptomatic extra-axial fluid collections. The lateral extent

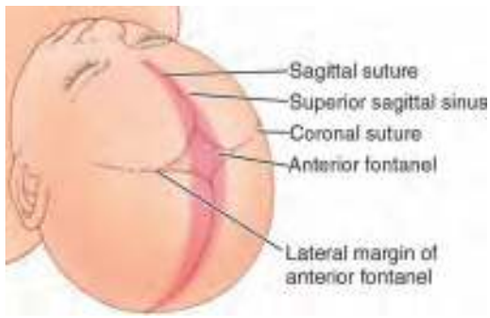

FIGURE 149-2. Surface anatomy of the infant skull. The shaded area represents the underlying superior sagittal sinus.

of the anterior fontanel is continuous with the coronal suture (**Figure 149-2**). The anterior fontanel is composed of connective tissue and easily perforated with a spinal needle.[6] Continued advancement of the needle would then perforate the underlying dura, allowing the tip of the needle to rest within the subdural space. Traversing the dura is usually associated with a definite change in resistance as the subdural space is entered. Successful entry of the tip of the needle into the subdural fluid collection can be confirmed by removal of the stylet and observing spontaneous drainage from the needle hub.

INDICATIONS

A subdural aspiration is performed for diagnostic purposes and for rapid decompression of subdural fluid collections (i.e., acute or chronic).[6] Perform a subdural aspiration in the young child with a subdural fluid collection and radiographic signs of elevated intracranial pressure, a depressed level of consciousness, and/or a changing level of consciousness if a Neurosurgeon is not available.[9] A subdural aspiration may be considered in the presence of other clinical signs and symptoms associated with elevated intracranial pressure (i.e., bulging fontanelles, coma, cranial nerve palsies, hemiparesis, hypotonia, irritability, lethargy, posturing, seizures, somnolence, and vomiting that is repeated or intractable). **Aspiration of extra-axial fluid collections can reduce intracranial pressure dramatically.** Subdural aspiration of fluid allows for culture and sensitivity, identification of microorganisms, and aids in the selection of bacterial specific antimicrobial agents if an infectious etiology is the cause of the extra-axial fluid collection.

CONTRAINDICATIONS

The main contraindications to performing a subdural aspiration include absence of the anterior fontanel from premature closure, age of 2 or older, coagulopathy, congenital anomalies of the skull or brain, scalp infections, solid clots that are not liquefied, and thrombocytopenia. **Do not perform this procedure if unfamiliar with the technique and its complications. Consult a Neurosurgeon, if available, prior to performing this procedure.** Repeated aspirations are rarely indicated as blood or fluid reaccumulation often requires the placement of a drain or a surgical procedure.

EQUIPMENT

- Sterile skin prep kit
- Povidone iodine or chlorhexidine solution
- Sterile gloves and gown
- Face mask with an eye shield or goggles
- Sterile drapes
- 1% or 2% lidocaine, with or without epinephrine
- 18 to 22 gauge, 1.5 inch spinal needle or angiocatheter
- Tincture of benzoin or Mastisol
- Cotton swab
- Bandage
- Bundling blanket
- Commercially available immobilization device
- Syringe for aspirating fluid
- Sterile specimen containers
- Culture bottles or swabs
- IV extension tubing, optional

FIGURE 149-3. An example of a lumbar puncture kit.

The basic equipment can be found in commercially available pediatric lumbar puncture kits (**Figure 149-3**). The kit may need to be supplemented with personal protective equipment and skin antiseptic.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. It is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Apply full monitoring with a cardiac monitor, end-tidal carbon dioxide monitor, noninvasive blood pressure cuff, and pulse oximetry. Obtain a CT scan of the head to determine the presence of an extra-axial fluid collection, the location and extent of the fluid collection, and the type of fluid (e.g., blood, fluid, or pus), and to rule out the presence of a mass (**Figures 149-1 and 149-4**). A sonographer with bedside ultrasound training for the presence of a subdural hematoma can substitute (**Figures 149-4 and 149-5**).[10] Consider an ultrasound when the patient's condition prevents moving them for a CT or MRI. Obtain a complete blood count (i.e., hematocrit, hemoglobin, and platelet count) and a coagulation profile (i.e., prothrombin time, partial thromboplastin time, and international normalized ratio) to ensure that the patient is not coagulopathic or thrombocytopenic.

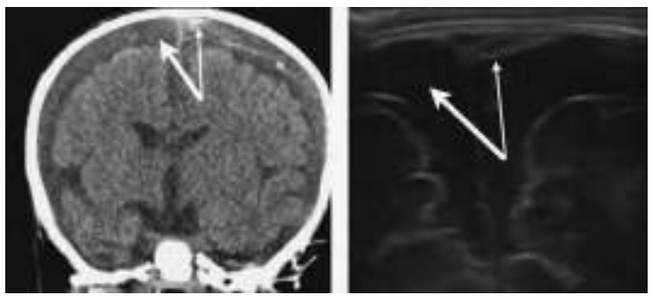

FIGURE 149-4. Coronal CT image (*left*) and corresponding ultrasound image (*right*) of an acute (*thin arrow*) on chronic (*thick arrow*) subdural hemorrhage in a 2-month-old. (Used with permission from reference 13.)

FIGURE 149-5. Sagittal ultrasound image of a subdural hemorrhage (*arrow*) in a 10-month-old. (Used with permission from reference 13.)

Place the patient supine on a stretcher. The child may need to be restrained to prevent movement during the procedure (Chapter 232). Bundling an infant in a blanket and placing their head close to the edge of the bed facilitates aspiration. An alternative is to use a commercially available immobilization device (e.g., Papoose board). The aid of an assistant to hold the child's body with the head straight and upright is recommended if the child is restless. Some children may require the administration of parenteral sedatives or procedural sedation (Chapter 159).

This procedure requires strict aseptic technique. Shave the frontal and parietal regions of the child's head. Clean any dirt and debris from the scalp. Identify by palpation the anterior fontanel, the coronal suture, and the lateral margin of the coronal suture. Don full sterile and personal protective equipment (i.e., cap, face mask with an eye shield or goggles, sterile gloves, and sterile gown). Apply povidone iodine or chlorhexidine solution to the skin over the entire scalp and allow it to dry. Ensure that the solution is not allowed to drain onto the baby's face or eyes. Apply sterile drapes to leave the frontal and parietal regions of the skull exposed. A local anesthetic agent is usually not required but may be used at the Emergency Physician's discretion. Place a skin wheal using 0.25 to 0.50 mL of 1% or 2% lidocaine or apply a topical lidocaine cream at the site the needle will enter the scalp.

TECHNIQUE

Form a Z-tract by gently sliding the scalp skin laterally (**Figure 149-6**). Insert a 22 gauge spinal needle perpendicular to the skull into the lateral margin of the anterior fontanel, about 2.5 to 3 cm off the midline (**Figure 149-6**). Advance the spinal needle beneath the frontal bone until the subdural space is penetrated (**Figure 149-7**). This is usually within 5 to 8 mm from the skin surface. A loss of resistance is usually appreciated as the dura is penetrated and the subdural

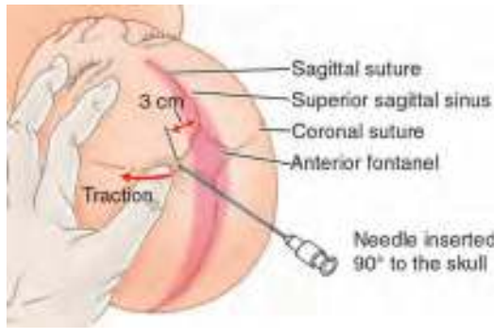

FIGURE 149-6. Subdural fluid aspiration. The needle is inserted at the lateral border of the coronal suture and at 90° to the skull.

space is entered. **Do not advance the needle any further. Securely hold the spinal needle so that it does not move (Figure 149-7).** An alternative is to insert the needle at a 45° angle to the skin surface and tunnel it under the scalp into the subdural space (**Figure 149-8**). Remove the stylet. Spontaneous drainage is often appreciated. The fluid may appear hemorrhagic. It is not uncommon to drain xanthochromic fluid.

The initial subdural aspiration usually results in spontaneous drainage through the spinal needle. **The spontaneous cessation of flow through the needle suggests that the extracranial and intracranial pressures are equalized, not that the fluid has been completely evacuated.** If the fluid does not spontaneously drain, carefully and gently apply a syringe to the spinal needle and gently aspirate the fluid. **Use minimal negative pressure to just aspirate the fluid collection and prevent pulling the brain or any bridging veins into the needle.** Alternatively, apply digital pressure to the anterior fontanelle to increase the flow through the spinal needle. Intravenous extension tubing may be connected to the spinal needle to allow the subdural fluid to drain away from the patient and directly into the specimen tubes. This is left to the Emergency Physician's preference.

Observe the gradual flattening of the anterior fontanel to determine the endpoint for the procedure.[11] Aspirate no more than 25 to 30 mL of subdural fluid during the procedure. Larger volumes up to 80 mL have been safely aspirated.[12] Remove the spinal needle and apply a bandage to the skin puncture site.

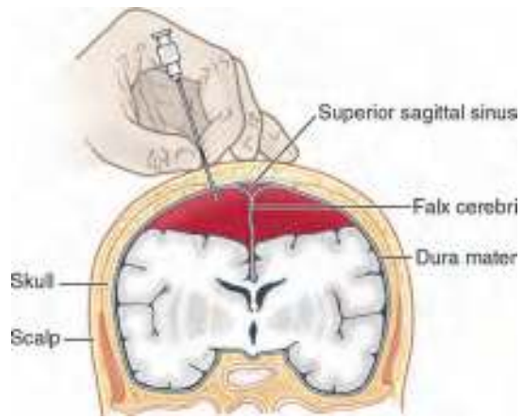

FIGURE 149-7. Coronal section through the skull at the level of the coronal suture. The needle is visible along its trajectory.

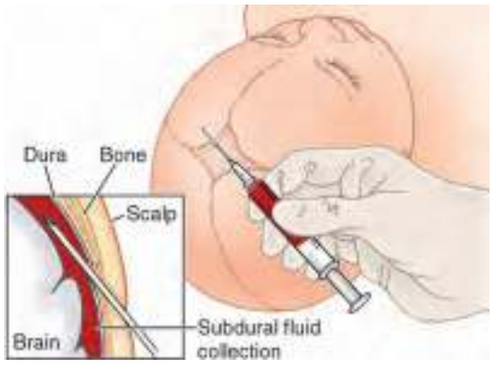

FIGURE 149-8. An alternative method for subdural fluid aspiration. The inset shows the oblique trajectory of the needle into the subdural space.

ALTERNATIVE TECHNIQUES

There are numerous alternatives to the aspiration of the fluid collection. These include burr hole drainage, craniotomy, drainage through an external collecting system, observation, and placement of a subdural-to-peritoneal shunt. Do not perform the aspiration with symptomatic nonliquefied clot or extensive membrane development. Consult a Neurosurgeon to evaluate and manage the patient.

Some Physicians prefer to use an intravenous catheter rather than the spinal needle. Removal of the metal needle leaves a soft catheter in place that minimizes the risk of injury to the brain or intracranial vascular structures. The use of an intravenous catheter set rather than a spinal needle is left to the discretion of the Emergency Physician.

ASSESSMENT

Monitor the child for a change in their level of functioning after subdural aspiration. Comparison to the child's baseline neurologic examination is essential. It is mandatory to document timed serial neurologic examinations in the medical record. The clinical examination is age-related. It is often prudent to observe the infant in the arms of a parent or health care worker while monitoring their level of alertness, extraocular movement, facial expression, limb movement, and pupillary response. Obtain daily head circumference measurements and plot them on a chart. Note the size and feel of the anterior fontanel. Note any signs of infection. **Infection can be prevented with careful attention to aseptic technique.** A simple bandage placed over the puncture site is usually sufficient and can be removed 48 to 72 hours after the procedure. Monitor continued spontaneous drainage through the puncture site as it poses an infectious risk.

AFTERCARE

Perform an ultrasound or CT scan of the head to determine if the fluid collection has been adequately drained.[10,13] Bedside ultrasonography is an effective tool in neonates and young children. Any change in the child's neurologic examination requires an emergent head CT scan or ultrasonography to determine if the subdural fluid collection has reaccumulated.[10,13]

Send the aspirated fluid for the appropriate laboratory analysis if an etiology other than an acute traumatic hemorrhage is suspected. This can include a biochemistry analysis (e.g., glucose and protein level), cell count and differential, culture (e.g., bacterial, fungal, or viral), cytology, and Gram's stain. Admit the child to the Intensive Care Unit where they can be appropriately monitored and observed.

COMPLICATIONS

The primary complication of aspiration of an extra-axial fluid collection in the infant is injury to the superior sagittal sinus or draining veins. This can result in further bleeding, brain compression, and brain herniation. This is best avoided by inserting the spinal needle 2.5 to 3 cm lateral to the midline. **At no time should the trajectory of the needle be in the midline or parasagittal in location to avoid this complication.** Overpenetration of the spinal needle can result in brain or intracerebral hemorrhage. Have an assistant immobilize the child to prevent them from moving and the needle lacerating the brain or a blood vessel.

Persistent leakage of subdural fluid from the puncture site can be the result of insufficient fluid aspiration or failure to use a Z-tract. This complication can be prevented by tunneling the needle under the scalp prior to penetrating the lateral margin of the anterior fontanel (**Figure 149-6**). Leakage can usually be controlled with local pressure, head elevation, and rarely by using cotton soaked with tincture of benzoin or Mastisol. Placement of a single suture at the puncture site will usually control continued leakage if less invasive methods fail.

Introducing infection into the subdural space has been reported following a subdural aspiration.[14] Avoid this by maintaining strict sterile technique. Nervous system disability is the result of the subdural hemorrhage and not the procedure.[15,16] An infection can make this disability worse.

SUMMARY

Extra-axial fluid collections are common problems during infancy. They are regarded as posttraumatic lesions in the great majority of cases. Birth trauma is sometimes implicated but is not common. It appears that minor injuries during infancy or more violent injuries (e.g., cranial impact and shaking) are the inciting events. It is incumbent upon the Emergency Physician to rule out any possibility of NAT! Report actual or suspected abuse to the appropriate state agency as required by law. Emergent aspiration of extra-axial fluid collections in the symptomatic child can be completed safely with very little risk to the child. Admission and continued observation are mandatory.

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150 Skeletal Traction (Gardner-Wells Tongs) for Cervical Spine Dislocations and Fractures

Thomas Engel and Rebecca Roberts

INTRODUCTION

Traumatic injuries to the cervical spine result from forces acting on the head and neck. The incidence of spinal cord injury in the United States is approximately 5 per 100,000.[1] Approximately 60% to 80% of spinal cord injuries involve the cervical spine. Cervical spine injuries can occur at all ages with predilection for teens and young adults. Emergency Physicians must be aware of cervical spine injuries in the elderly with seemingly minor mechanisms of injury.[2] Motor vehicle collisions are the most common cause and account for almost half of cervical spine injuries.[3] The remaining cervical spine injuries result from falls, sports injuries, violence, penetrating wounds, and miscellaneous causes. Few other injuries have the potential to cause such high levels of morbidity.

The primary aims of therapy with an acute spinal cord injury are to safely extract and transport patients from the scene, to identify injury early, to minimize secondary injury to the spinal cord by preventing hypoxia and hypotension, to realign the spine, to improve neurologic recovery, to maintain spinal stability, and to obtain an early functional recovery. This is achieved by decompression of the spinal cord through restoration of the normal sagittal diameter of the spinal canal using cervical traction and/or removing a compressive lesion surgically. This is particularly important in patients who have sustained an incomplete spinal cord lesion and are found to have a progressing neurologic deficit. Restoring the normal anatomic position provides pain relief.

Consider all cervical spine injuries unstable until proven otherwise. Neurosurgical guidelines recommend the use of a rigid cervical collar with supportive blocks and taping of the patient's forehead to the backboard to provide for the greatest degree of cervical stabilization.[4] Operative intervention has become more common in the management of cervical trauma with improved imaging and surgical tools. The use of skeletal traction in the acute spinal cord injury patient remains a safe and straightforward method of reducing fractures and maintaining the spinal canal in anatomical alignment during initial management, as a surgical adjunct, or as definitive therapy.

Fabricius Hildanus utilized forceps in treating fractures or dislocations of the cervical spine as early as 1646. Crutchfield is credited for introducing skeletal tongs in the management of cervical spinal injuries.[5] Crutchfield developed a pair of self-tightening tongs in 1933 that allowed him to apply traction to the cranium in a patient with a cervical spine fracture.[5] These tongs were subsequently modified and have essentially been replaced by the Gardner-Wells tongs.[6] Gardner-Wells tongs were introduced in the early 1970s and utilize the principle of a spring-loaded point for cervical traction.[6]

ANATOMY AND PATHOPHYSIOLOGY

Cervical spinal cord injuries can be divided into upper (i.e., occiput to C3) and lower (i.e., C3 to C7) injuries. Numerous classification systems exist. These are based upon the morphology and the mechanism of injury.[7] No classification is ideal. **Critical to all cervical spine injury classifications is the determination of the stability of a fracture or dislocation.** Stability of the vertebral column is dependent upon the integrity of the vertebra, the intervertebral disk, the facet joints, and the ligamentous structures.

Clinical stability of the cervical spine is determined by the ability of the spine under physiological loads to maintain its normal anatomic relationship so that there is no damage to the spinal cord or nerve roots. It has been proposed that spinal instability be separated into mechanical, neurologic, and combined types. Mechanical instability implies that the injured spine could collapse or distort under normal physiological stresses. Neurologic instability implies a risk of neural injury (i.e., spinal cord and/or nerve root) subsequent to the initial injury.

It is imperative that a thorough clinical and radiologic evaluation be completed to determine if the patient has suffered an unstable cervical spine injury. Begin with a thorough history and physical examination. Perform a complete neurologic examination with particular attention to spinal cord function. Cervical spine radiography is indicated to evaluate fracture patterns, disk disruption, vertebral subluxation, vertebral dislocation, vertebral angulation, and ligamentous injury. A computed tomography (CT) scan is recommended with possible addition of magnetic resonance imaging (MRI) and myelography if available in a timely fashion and if indicated. The radiograph for ligamentous injury evaluation should look for disruption of each of the major spinal ligaments (i.e., the anterior longitudinal ligament, the apophyseal ligamentous complex, the posterior ligamentous complex, and the posterior longitudinal ligament). Radiologic findings that indicate instability include diastasis of the apophyseal joints, dislocations, disruption of the posterior vertebral bodyline, interspinous process widening, vertebral displacement, and widening of the spinal canal.

White and Punjabi proposed a checklist point value system (**Table 150-1**).[8] A point value is assigned to each of the injuries appreciated on the cervical spine radiographs. The total point values are then summated. A score of five or more is suggestive of clinical instability. The indications for the application of the Gardner-Wells tongs includes evidence of cervical spine instability.

The spring-loaded pin design of the Gardner-Wells tongs and tilting of the pins in the direction of pull ensures a firm and prolonged grasp of the skull. Gardner-Wells tongs have the capacity to tolerate at least 65 pounds or more of traction. The amount of weight necessary to accomplish reduction varies considerably. As a rule, 5 pounds of weight is applied for each vertebral level above the level of the fracture or the dislocation. Many authors have recommended the use of up to 50 to 60 pounds of weight.[9] Others have applied weights in excess of 100 pounds to reduce a cervical subluxation.[10]

TABLE 150-1 Checklist for the Diagnosis of Clinical Instability in the Lower Cervical Spine[8]

Element	Point value*
Anterior element destroyed or unable to function	2
Posterior element destroyed or unable to function	2
Relative sagittal plane translation > 3.5 mm	2
Relative sagittal plane rotation > 11°	2
Positive stretch test	2
Spinal cord damage	2
Root damage	1
Abnormal disk narrowing	1
Dangerous loading anticipated	1
Developmentally narrow spinal canal	1

*Summate the points. A total of 5 or more points is considered clinically unstable.

The concern for using an excessive amount of traction weight is overdistraction that can result in a traction type of spinal cord injury. Begin by applying 5 to 10 pounds of weight.[3] Add additional weight in 3 to 5 pound increments if the initial application of weights fails to achieve spinal alignment. Obtain lateral cervical spine radiographs 10 to 15 minutes after each addition of weight until the cervical spine is realigned. This time delay is needed after the application of weight to allow the soft tissues to accommodate. **It is of paramount importance to monitor the patient's neurologic status during the entire procedure to prevent iatrogenic injury from overdistraction of an unstable motion segment.**

INDICATIONS

Neurosurgical or Orthopedic Spine consultation is advised if available. Immediate surgical intervention or placement of a halo device often provides more definitive management, a greater degree of immobilization, and improved outcomes. The definitive management of the cervical spine injury can include skeletal traction with Gardner-Wells tongs if clinical instability is demonstrated or suspected, or if reduction of a displacement (i.e., a dislocation or fracture) is needed and consultant availability is limited or delays in intervention are present.[11-13] Other indications for the application of Gardner-Wells tongs are certain cervical infections or neoplasms, anterior cervical discectomy with graft fusion in which distraction of the disk space is required, cervical spine facet fractures, cervical spine spondyloptosis, and cervical spondylosis.[14,15] Only apply Gardner-Wells tongs if the patient requires temporary longitudinal traction.[16] Their use requires the patient to remain bedridden as compared to halo vest traction.

CONTRAINDICATIONS

Cervical traction is contraindicated in someone who has sustained posttraumatic disk herniation with spinal cord compression. Perform a good neurologic evaluation, a CT scan, or MRI scan prior to applying cervical traction. Some have advocated for an MRI evaluation prior to all cervical traction due to fears of iatrogenic spinal cord compression from disk herniation. The literature has not shown that time delays due to MRI imaging have proven detrimental to functional recovery.[17] Fractures of the skull and infections overlying the potential pin sites are contraindications to the use of skeletal traction. Other contraindications include diseased bone and children less than 3 years of age. Atlantooccipital dislocations and C1-C2 dislocations are contraindications as applying Gardner-Wells tongs to someone who survives these injuries could worsen the patient's condition and/or neurologic deficit.

FIGURE 150-1. The Gardner-Wells tongs. (Photo courtesy of Eric F. Reichman, PhD, MD.)

EQUIPMENT

- Skin prep kit
- Hair clippers
- Povidone iodine solution, chlorhexidine may damage nerve tissue
- 1% or 2% lidocaine with epinephrine
- 5 mL syringe
- 18 gauge needles
- 22 gauge needles
- Gardner-Wells traction tongs
- Pulley traction system
- Traction weights, 3 to 5 pound increments
- Topical antibiotic ointment

The Gardner-Wells tongs are a simple device (**Figure 150-1**). It consists of a contoured and rigid stainless steel rod that follows the coronal contour of the calvarium. Gardner-Wells tongs are available in versions composed of graphite or titanium alloy. These versions are compatible with and facilitate subsequent CT and MRI scans without producing artefacts. The Gardner-Wells tongs have a threaded hole on each end that accommodates the pins. The spring-loaded pins are threaded screws with sharp cone-shaped points. One of the pins is the calibration pin (**Figure 150-2**). It contains an indicator pin that extends and retracts as the tip of the calibration pin penetrates the skull. The pins screw into the rigid rod so that the points are tilted in the direction of the pull. This results in the pins pressing into the skull when traction is applied and ensuring that they do not pull out. A squeezing pressure of 30 pounds is applied to the skull when the pins are properly positioned.[5] An S-shaped hook is permanently attached to the top of the Gardner-Wells tongs. The rope and traction weights are attached to this hook. Permanently attached to the S-hook is a metal plate that is engraved with the instructions (**Figure 150-3**). **The instruction plate faces**

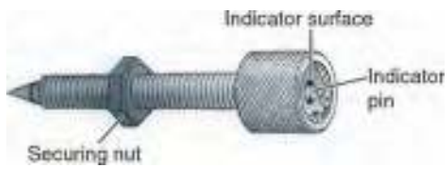

FIGURE 150-2. The calibration pin of the Gardner-Wells tongs.

FIGURE 150-3. Instructions for the use of the Gardner-Wells tongs. (Photo courtesy of Eric F. Reichman, PhD, MD.)

FIGURE 150-4. Application of the Gardner-Wells tongs to a human skull. (Photo courtesy of Eric F. Reichman, PhD, MD.)

upward and is readable when the Gardner-Wells tongs are properly applied (Figures 150-4 and 150-5).

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain an informed consent to apply the Gardner-Wells tongs. Signed consent may be omitted in cases where the patient is immobilized or has an altered mental status. Document the reason for the lack of a signed consent in the medical record. Intravenous sedation may be required in certain cases and is at the discretion of the treating Emergency Physician.

Consider the use of some form of deep venous thrombosis prophylaxis since the application of Gardner-Wells tongs requires the patient to remain bedridden. The use of sequential compressive devices is simple, easily applied in the Emergency Department, and will not cause complications. Consult a Spine or Neurological Surgeon before prescribing intravenous, oral, or subcutaneous anticoagulants that could have grave bleeding complications in a cervical trauma patient.

Identify the anatomic landmarks required to place the Gardner-Wells tongs. The pins are introduced in the temporal region, two to three fingerbreadths (i.e., 3 to 4 cm) cephalad to the pinna of the ear (**Figure 150-5**). Place them directly above the external auditory meatus for neutral distraction, 2 to 3 cm posterior to the external auditory meatus for flexion distraction, and 2 to 3 cm anterior to the external auditory meatus for extension distraction. A helpful landmark is the squamosal line where the temporalis muscle inserts into the skull. **Place the Gardner-Wells tongs below the squamosal line to allow adequate traction.** Another useful landmark is to observe for the widest biparietal diameter of the patient's skull. This usually corresponds to the landmark just inferior to the squamosal line.

The use of Gardner-Wells tongs does not require shaving the patient's hair at the proposed pin sites. Clipping the hair can improve patient comfort and postapplication care. Clean the skin and hair of any blood, dirt, and debris. Apply povidone iodine solution to the skin and hair at the proposed pin sites. Allow the povidone iodine to dry. Use sterile technique and infiltrate 1 mL of lidocaine with epinephrine into each of the two proposed pin sites. Infiltrate subcutaneously and down to the level of the periosteum of the skull.

TECHNIQUE

The Gardner-Wells tongs are fast and easy to apply by one person. An assistant standing at the patient's feet can confirm that tongs are placed symmetrically. Another assistant may be needed to maintain

FIGURE 150-5. Application of the Gardner-Wells tongs. ***A.*** It can be positioned and applied without shaving the patient's entire head. ***B.*** Schematic demonstrating the pin position into the skull.

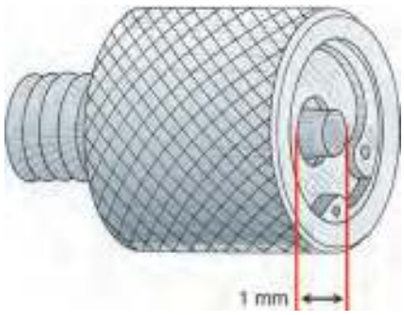

FIGURE 150-6. The calibration pin indicator is at 1 mm when the pins are properly seated in the skull.

the patient's head position during the procedure. The patient should already be supine on the gurney. Stand above the patient's head. Assemble the Gardner-Wells tongs by inserting the pins into the threaded holes.

Place the pins of the Gardner-Wells tongs over the proposed pin insertion sites of the patient's head. **The instructions on the S-hook must be facing upward and readable. The Gardner-Wells tongs are upside down if the instructions are not facing upward.** Apply the Gardner-Wells tongs so the pins are symmetrically located on each side of the head. The assistant at the foot of the bed can ensure symmetry of pin placement. Screw in the pins equally and simultaneously on both sides of the tongs (**Figure 150-5*B***). Note that the small spring-loaded indicator pin is observed on one side of the tongs when adequate tension is applied (**Figures 150-2 and 150-6**). Recommendations by the manufacturer suggest that the pins should be tightened simultaneously until there is a 1 mm protrusion of the indicator pin beyond the flat surface (**Figure 150-6**). Tighten the securing nuts to prevent the tongs from loosening (**Figure 150-2**).

The points of the pins will not pull out when properly applied. The depth of penetration of the pins is self-limited by a gradual lessening of the spring tension and an increase in the surface area of contact between the tips of the pins and the skull. The pressure exerted by each pin is exactly the same, regardless of whether one pin has been advanced farther than the other. The curve of the rigid rod allows the traction loop to find its proper position.

CONSIDERATIONS FOR PEDIATRIC PATIENTS

There are significant anatomic differences in children below the age of 8 years. The large occiput of children produces a cervical kyphosis. It is recommended to elevate the torso with soft padding to prevent excessive kyphosis and allow for a more anatomic alignment of the cervical spine when immobilized on a spine board or a stretcher.

ALTERNATIVE TECHNIQUES

There are halo tongs that can be used. They usually include four pins instead of two with the Gardner-Wells tongs. They can be attached to a pulley system with weights to reduce and stabilize the cervical spine. They can alternatively be attached to a halo vest for stability (**Figure 150-7**). This device is reserved for the consultant.

APPLICATION OF TRACTION WEIGHTS AND ASSESSMENT

Gently rock the Gardner-Wells tongs to assure that the pins are properly seated within the outer table of the skull (**Figure 150-8**). Place the patient in the reverse Trendelenburg position (**Figure 150-9**).

A

B

FIGURE 150-7. The halo tongs attached to a vest. ***A.*** Overview. ***B.*** Close-up of the tongs. (Photos courtesy of Eric F. Reichman, PhD, MD.)

Tie the provided rope to the S-shaped hook. Feed the other end of the rope through a pulley at the head of the bed and apply weights. Proper attention to the head position and the axis of distraction are important elements in achieving closed reduction. Initially apply a 10 pound weight.[8] Obtain a lateral cervical spine radiograph 10 to

FIGURE 150-8. The Gardner-Wells tongs have been applied. The arms are grasped and gently twisted to confirm proper seating of the pins.

15 minutes after the application of the weight. Add 3 to 5 pound weights, one at a time, for each vertebral segment above the level of the injury. Obtain a lateral cervical spine radiograph 10 to 15 minutes after each 3 to 5 pound weight is added and reassess the neurologic exam. Continue to increase the traction weight in 3 to 5 pound increments. Continue to repeat lateral cervical spine radiographs and neurologic exams 10 to 15 minutes after each additional weight is added. **Stop adding weights when the radiographs demonstrate appropriate alignment of the cervical spine. Careful assessment and documentation of the patient's neurologic function are mandatory throughout the application of weights and skeletal traction to prevent overdistraction and iatrogenic injury.**

AFTERCARE

Clean the pin sites every shift with povidone iodine or hydrogen peroxide solution followed by iodine ointment. Obtain daily cervical spine radiographs to follow the spinal alignment and pin placement. Very slowly reduce the weight by 50% to maintain the alignment if spinal realignment is obtained with traction. Monitor the patient's airway status, anxiety level, and for the development of pressure ulcers given their immobilization. Confirm that sequential compression devices are applied to the legs and functional to prevent thromboembolic complications. Ensure proper patient padding as pressure ulcer development is common and preventable.

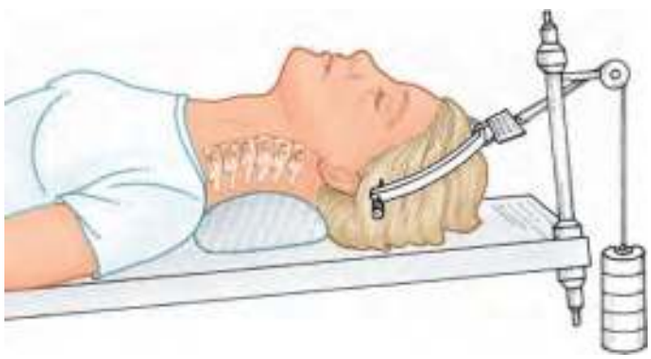

FIGURE 150-9. The patient is placed in reverse Trendelenburg and traction is applied.

The points of the pins tend to penetrate the outer table of the skull due to the continuous pressure exerted by the springs on a very small area. **Readjust the pins in 24 hours, again setting the indicator pin so that it protrudes approximately 1 mm from the flat surface. Further adjustment of the Gardner-Wells tongs is not necessary and is not recommended as it can result in erosion of the pinpoint through the skull.**

Interfaculty transport may become necessary to provide higher level of patient care after the application of Gardner-Wells tongs. Expert advice is to place the patient back into a ridged cervical collar, use side-by-side immobilizers, and tape the patient's head to a long spine board. Secure the weights attached to the pulley system to prevent their movement. **Take extreme caution when transferring the patient to prevent excessive distraction or loss of traction.**

COMPLICATIONS

Skull penetration from placing the pins too low in the temporal region where the skull is thinnest can lead to dural tears, epidural hematomas, and possibly intracranial injury.[18-20] Pins located in the temporal fossa pierce the temporalis muscle and can cause painful mastication. Overdistraction can lead to iatrogenic injury. This is best prevented by the initial use of a minimal amount of weight necessary to distract or reduce the cervical spine injury. Pin site infections are prevented by close attention to surgical technique and daily hygiene with the addition of topical antibiotic ointment. Other complications reported include intracranial aneurysms, cerebrospinal fluid leaks, and osteomyelitis of the skull.

SUMMARY

The application of skeletal traction in the form of Gardner-Wells tongs is a safe, simple, and quick procedure when there is evidence of clinical or radiologic cervical spine instability and no immediate plans for definitive therapy. It requires only local anesthesia and antiseptic preparation of the skin. Careful attention to the application technique using the suggested anatomic landmarks will reduce the chances of complications. Monitoring realignment and/or reduction procedures with frequent cervical spine radiographs and neurologic exams after each traction weight applied is essential. Gardner-Wells tongs are not recommended as a long-term immobilization technique. Definitive management of cervical spine instability requires surgical stabilization and/or halo bracing.

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151 Reflex Eye Movements (Caloric Testing and Doll's Eyes)

Atilla B. Üner

INTRODUCTION

A central nervous system-induced coma is either the result of bilateral diffuse impairment of the hemispheres or impairment of the paramedian reticular formation in the high pons of the brainstem. Examination of the pupils and reflex horizontal eye movements (i.e., vestibulo-ocular reflex and oculocephalic reflex) in the Emergency Department will aid in determining the location of the lesion responsible for the comatose state to either the brainstem, the hemispheres, or both.[1,2] When more information is available, a neurologic exam including reflex eye movements can aid in determining the prognosis of patients in a nontraumatic coma including patients successfully resuscitated from cardiac arrest.[3] **This testing is considered to be part of a thorough and complete neurologic examination in the comatose patient.**

ANATOMY AND PATHOPHYSIOLOGY

EXAMINATION OF THE PUPILS

It is imperative to first assess the pupillary reflex to light and the presence of spontaneous eye movements in the comatose supine patient. The pupillary light reflex involves the pretectal nuclei in the upper midbrain of the brainstem.[4] Damage to areas of the brainstem can result in characteristic abnormalities of the pupils.[1] Pontine lesions can produce pinpoint pupils with preserved reaction to light upon close examination. Midbrain tegmental lesions can result in midrange pupils that may be irregular, unequal, and unreactive to light. Midbrain pretectum lesions may cause midrange fixed pupils that do respond to accommodation.

Impaired brainstem function may be seen in patients who are in a toxic or metabolic coma. This can present with impaired eye movements or a complete ophthalmoplegia. Pupillary function is preserved in these cases. **Preserved pupillary function in a comatose patient suggests decreased brainstem function likely caused by a toxic or metabolic disorder and not structural damage to the brainstem.[1]**

Spontaneous roving eye movements in the comatose patient are typically slow and horizontal, indicating bilateral hemispheric disease with a relatively intact brainstem (e.g., the pons and the midbrain).[1,2] Ocular dipping describes the slow downward movement of the eyes with rapid return to a neutral position with preserved spontaneous roving horizontal eye movements and suggests global hypoxic encephalopathy.[1] Ocular bobbing describes an intermittent rapid downward movement of the eyes with delayed return to a neutral position in the absence of horizontal eye movements and suggests severe damage to the pons (e.g., the "locked-in syndrome").[1]

Spontaneous full roving eye movements or impaired eye movements with preserved pupillary reaction to light permit adequate localization of the cause of coma. Further testing of reflex eye movements is only indicated if spontaneous lateral eye movements are limited or absent.[1,2]

OCULOCEPHALIC REFLEX (DOLL'S EYES)

The brainstem is comprised of the medulla, the pons, and the midbrain. It contains all three nuclei involved in the oculocephalic reflex. **Hence an intact oculocephalic reflex response suggests intact brainstem function.[2]** The lateral semicircular canals, the vestibulocochlear nerve, the abducens nerve, the oculomotor nerve, and the medial and lateral ocular rectus muscles must also be intact for a physiologic response to occur. **The optic nerve is not involved and vision or light perception is not required for this reflex to function.**

A simplified model of the physiologic oculocephalic reflex is described in this paragraph. Neural stimulation of the lateral semicircular canal is mediated by the inertia of the endolymph fluid resulting in a deflection of the cupula that is directly proportional to instantaneous head velocity.[5] Fairly rapid movements are necessary to elicit a response. Neural excitation from the lateral semicircular canal travels via the ipsilateral vestibulocochlear nerve to the ipsilateral medial vestibular nucleus in the medulla. It continues from there to the contralateral abducens nucleus in the pons and results in abduction of the contralateral eye via the abducens nerve and the lateral rectus muscle. The contralateral medial longitudinal fasciculus then excites the ipsilateral oculomotor nucleus in the ipsilateral midbrain, resulting in ipsilateral eye adduction via the oculomotor nerve and the medial rectus muscle (**Figure 151-1**).[1]

Turning the head to one side will cause an increase in the resting neuronal discharge rate of the lateral semicircular canal on that side. **Turning the head to the right will result in abduction of the left eye and adduction of the right eye in a comatose patient whose brainstem function is intact (Figures 151-1*A* and 151-1*B*). The eyes then spontaneously return to the midline (Figure 151-1*C*). Turning the head to the left will result in abduction of the right eye and adduction of the left eye in a comatose patient whose brainstem function is intact (Figure 151-1*D*). The eyes then spontaneously return to the midline (Figure 151-1*E*). The result is conjugate (i.e., parallel) eye movement contralateral to the direction of head deviation.** It will appear as though the patient is compensating for the passive head motion by moving both eyes to the other side and maintaining visual fixation of a stationary target

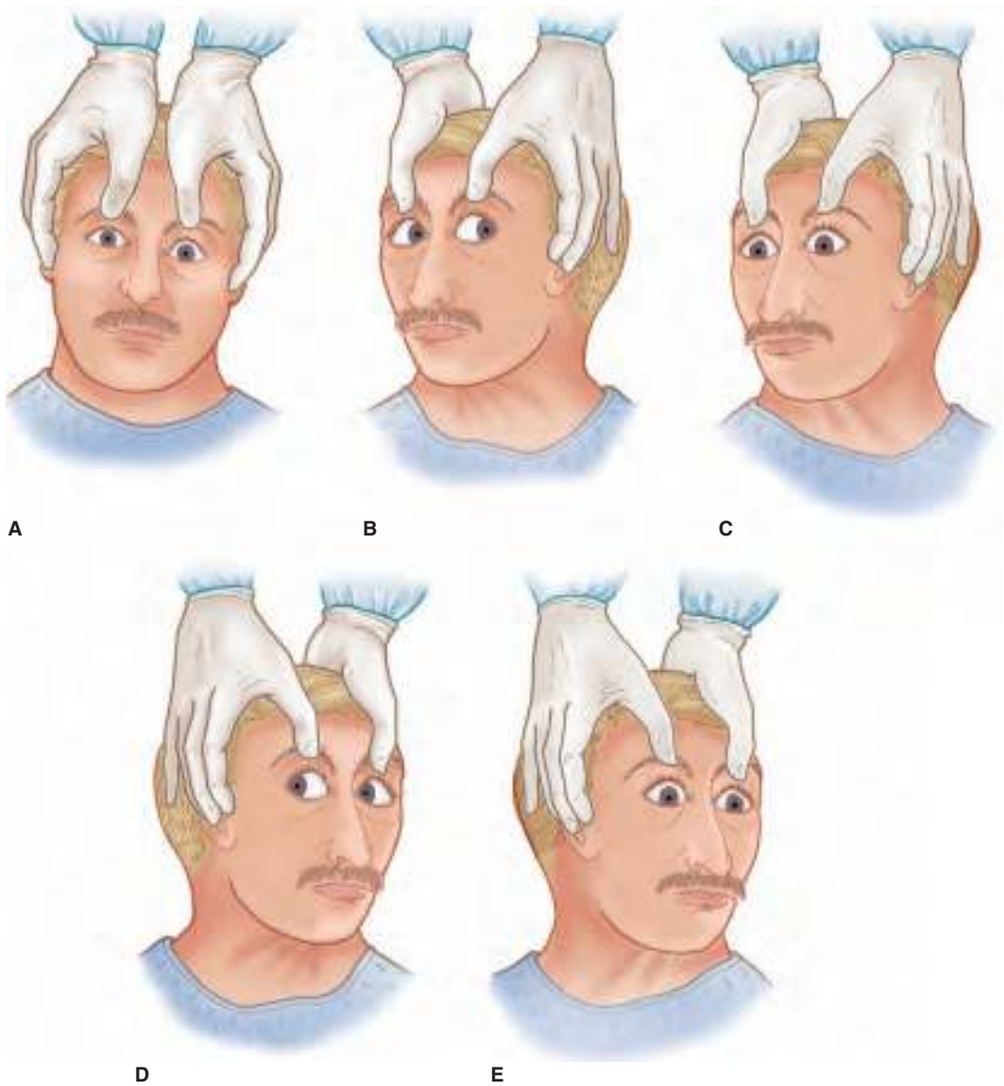

FIGURE 151-1. The oculocephalic or doll's eyes reflex in a patient with an intact brainstem. ***A.*** The head is facing upright and grasped. ***B.*** The head is rotated 90° to the right and the eyes deviate to the left (i.e., the opposite side). ***C.*** The eyes spontaneously return to the midline. ***D.*** The head is rotated 180° to the left and the eyes deviate to the right (i.e., the opposite side). ***E.*** The eyes spontaneously return to the midline.

when the head is turned. This indicates an intact brainstem function and is termed a positive oculocephalic reflex (i.e., positive doll's eyes). A negative oculocephalic reflex ensues if the patient's eyes remain fixed in the orbits in a neutral position and do not move in response to passive turning of the head, implying impaired brainstem function. A partial response may be caused by impaired brainstem function, oculomotor nerve palsy, or abducens nerve palsy.[2]

A vertical oculocephalic response can similarly be tested by moving the head up and down.[6] This should result in compensatory vertical eye movements. This test will only be useful if the horizontal oculocephalic reflex is negative. A positive (i.e., intact) vertical oculocephalic reflex with a negative horizontal oculocephalic reflex suggests a pontine lesion. However, the vertical oculocephalic reflex is often negative in normal elderly patients and is only helpful if positive.[1]

VESTIBULO-OCULAR REFLEX

Ice and warm water caloric testing of the vestibulo-ocular reflex involves the same structures as the oculocephalic reflex (**Figure 151-2**). This test needs only to be performed when the oculocephalic reflex is negative or cannot be performed.[2] Instilling ice water into the external auditory canal generates a temperature gradient that causes movement of the endolymph of the lateral semicircular canal.[7,8] This results in an ampullifugal deflection of the cupula in the supine patient, leading to a decrease in the lateral semicircular canal resting neuron discharge rate on the ipsilateral side and an active increase of the resting neuron discharge rate of the medial vestibular nucleus on the contralateral side. This leads to an activation of the contralateral medial vestibular nucleus of the medulla resulting in conjugate eye deviation to the ipsilateral side (i.e., toward the ice water irrigated side) if all involved brainstem structures are intact.[1,2]

Irrigation of the external auditory canal with warm water will cause an increase in the resting neuron discharge rate of the ipsilateral lateral semicircular canal and conjugate eye deviation to the contralateral side (i.e., away from the warm water irrigated side). The results are reverse if the patient is prone.[1,2,9]

Density changes caused by temperature changes of the endolymph demonstrate the greatest effect when the lateral semicircular

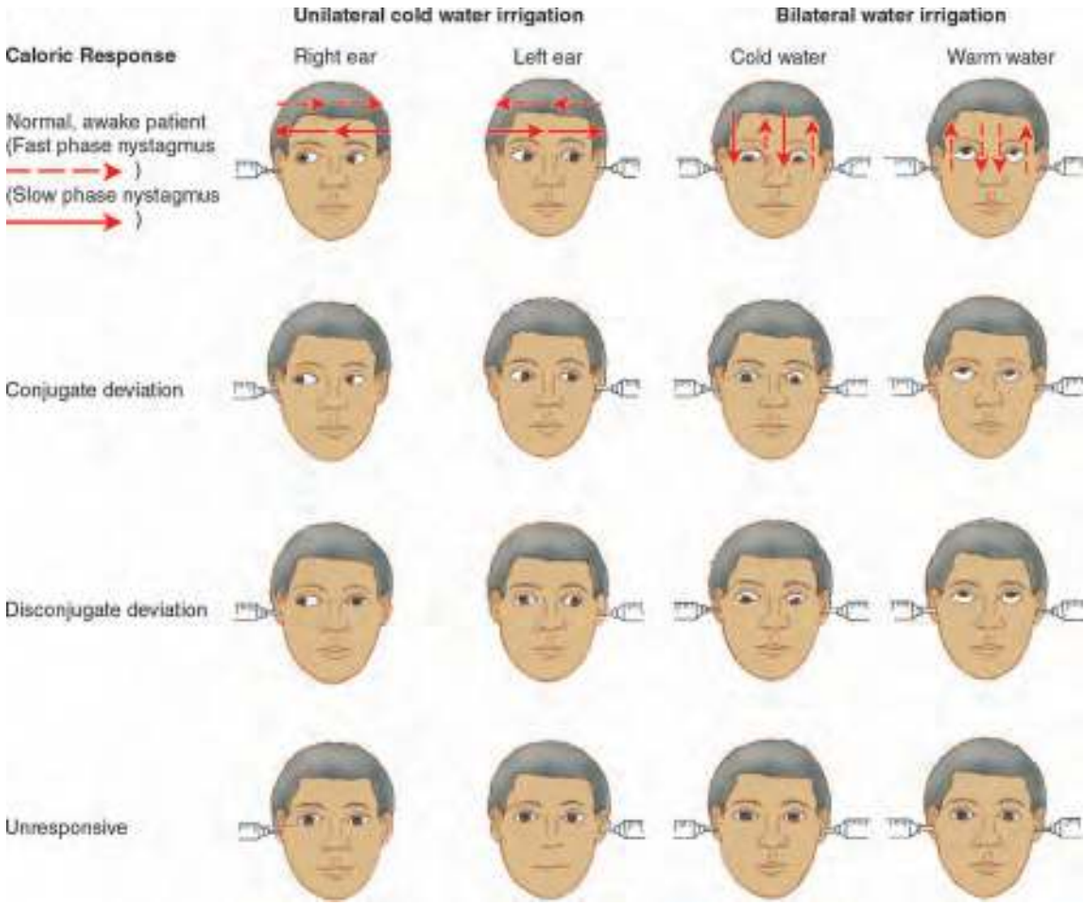

FIGURE 151-2. The vestibulo-ocular reflexes associated with unilateral cold water irrigation, bilateral cold water irrigation, and bilateral warm water irrigation.

canals are in the vertical planes for maximum gravity effect. This is achieved by elevation of the supine patient's upper body to 30° above the horizontal.[7-9] The observable oculomotor response is terminated after about 100 seconds by neural adaptation, although the residual thermal stimulation of the lateral semicircular canal can last up to 10 minutes.[7]

Unilateral vestibulo-ocular testing in an awake patient with ice water results in slow eye movements (i.e., slow phase nystagmus) ipsilateral to (i.e., toward) the irrigated side and rapid eye movements (i.e., fast phase nystagmus) contralateral to (i.e., away from) the irrigated side (**Figure 151-2**). Warm water testing in an awake patient results in slow eye movements (i.e., slow phase nystagmus) contralateral to (i.e., away from) the irrigated side and rapid eye movements (i.e., fast phase nystagmus) ipsilateral to (i.e., toward) the irrigated side. This quick phase, if present, indicates alertness of the pontine or midbrain reticular formation.[1] **The direction of nystagmus is named after the direction of the quick phase, giving rise to confusion.**

Bilateral vestibulo-ocular testing in an awake patient can be used to test vertical eye movements (**Figure 151-2**). Bilateral cold water irrigation results in slow eye movements (i.e., slow phase nystagmus) downward and rapid eye movements (i.e., fast phase nystagmus) upward. Bilateral warm water irrigation results in slow eye movements (i.e., slow phase nystagmus) upward and rapid eye movements (i.e., fast phase nystagmus) downward.

The presence of nystagmus indicates a noncomatose state. Typical examples of noncomatose states include catatonia, conversion reactions, malingering, psychiatric illness, or schizophrenia. Hypoactive responses can be due to neurologic disorders, vestibular disorders, or medications that depress labyrinth function.[8] Hyperactive responses in unresponsive patients can be due to mastoid disease or a perforated tympanic membrane.

Rapid phase nystagmus is absent in the patient with an acute coma. The quick phase may return with persistent vegetative states. It is important to note the slow and full eye deviation in response to caloric stimulation when assessing the comatose patient and not nystagmus.[1,2]

INDICATIONS

Reflex eye movement testing is indicated when information is needed regarding the brainstem function of a comatose patient. Oculocephalic reflex (i.e., doll's eyes) testing is indicated in any comatose patient if both spontaneous roving horizontal eye movements and pupillary reactions to light are limited or absent. Vestibulo-ocular reflex testing is indicated in any comatose patient if the oculocephalic reflex assessment is abnormal or cannot be performed.[1,3]

CONTRAINDICATIONS

Any suspicion of an occult cervical spine injury or basilar skull fracture is an absolute contraindication to oculocephalic reflex (i.e., doll's eyes) testing. It cannot be performed if the patient's cervical

spine is immobilized. A history of rheumatoid arthritis increases the risk of atlantoaxial subluxation with resulting spinal cord compression. Osteoporosis and cervical spine ankylosis increase the risk of injury to the cervical spine with manipulation. Other than an occult cervical spine injury or basilar skull fracture, these are relative contraindications to oculocephalic reflex testing.

Contraindications to vestibulo-ocular reflex testing include perforation of the tympanic membrane, the presence of tympanostomy tubes, any suspicion of cerebrospinal fluid otorrhea, and basilar skull fractures. There is a significant risk of introducing bacteria into the central nervous system through a tear in the dura mater. Reflex testing with cold air by a subspecialty service may be requested for patients with perforated tympanic membranes.[8]

EQUIPMENT

- Otoscope
- Warm water at 44°C
- Ice water
- 16 to 18 gauge angiocatheter
- 60 mL syringe
- Emesis basin
- Towels or chux

PATIENT PREPARATION

Explain the procedure, its utility, and potential outcomes to the patient's representatives. Place the patient supine for testing. **The cervical spine must be cleared radiographically and clinically to rule out any fractures, dislocations, or ligamentous instability before oculocephalic reflex (i.e., doll's eyes) testing.**

The external auditory canals and tympanic membranes must be visualized by otoscopy before performing vestibulo-ocular reflex testing. The absence of blood, cerebrospinal fluid, tympanostomy tubes, and tympanic membrane perforation must be ascertained before performing this test. **Remove any cerumen from the external auditory canal so that the irrigation fluid can reach the tympanic membrane.** Place towels and an emesis basin under the ear to be irrigated to catch the draining fluid.

TECHNIQUES

OCULOCEPHALIC REFLEX TESTING

Stand above the head of the bed and grasp the patient's head with both hands (**Figure 151-1*A***). Use the thumbs of both hands to open the patient's eyelids. Rapidly move the patient's head to one side while simultaneously observing for the presence or absence of horizontal eye movements (**Figures 151-1*B* and 151-1*C***). Observe the eyes for several seconds. Repeat the test by rotating the patient's head toward the other side (**Figures 151-1*D* and 151-1*E***). Document the presence or absence of horizontal eye movements.

VESTIBULO-OCULAR REFLEX TESTING

Elevate the head of the bed to 30°. Draw ice water into a 60 mL syringe. Remove the plastic angiocatheter from the needle. Discard the needle. Place the catheter on the syringe. Place the catheter into the external auditory canal. Irrigate the external auditory canal over 30 to 40 seconds with the ice water. Simultaneously observe the patient's eyes for the presence, or absence, and direction of horizontal eye movements.[1,8] The water should freely enter and exit the external auditory canal.[9] The stimulus is dependent upon the water temperature and not water pressure. Therefore, slow irrigation will suffice. Reflex horizontal eye movements may be delayed for up to 1 minute after irrigation of the external auditory canal.[1] It may be easier for the physician to observe the patient's eye movements while an assistant irrigates the external auditory canals.

At least 5 to 10 minutes should elapse before testing the contralateral side.[1] The same external auditory canal can also be irrigated with warm water at 44°C (111.2°F) if, for any reason, the contralateral side cannot be tested. Warm water testing may be performed if there is no response to cold testing. Some physicians also perform simultaneous bilateral external auditory canal irrigation and observe for vertical eye movements. This requires the simultaneous irrigation of both external auditory canals with equal volumes of water.

ASSESSMENT

OCULOCEPHALIC REFLEX TESTING

Head movement to the right should result in conjugate eye movement to the left and return to midline, whereas head movement to the left should result in conjugate eye movement to the right and return to midline (**Figure 151-1**).[10] **This is a positive oculocephalic reflex and indicates a functionally intact brainstem in the comatose patient. Incomplete or absent horizontal eye movements in response to head movement (i.e., the eyes remain in the midline) is a negative oculocephalic reflex. This indicates impairment of the brainstem and vestibulo-ocular reflex testing should be performed if not contraindicated.**

VESTIBULO-OCULAR REFLEX TESTING

Full horizontal eye movements indicate that the etiology of the coma is located in the cerebral hemispheres and not in the brainstem. This is true whether the eye movements are noted spontaneously, upon oculocephalic reflex testing, or upon vestibulo-ocular reflex testing. Unilateral ice water irrigation of the external auditory canal resulting in ipsilateral conjugated full eye deviation (i.e., toward the irrigated ear) followed slowly by a returning to midline is a positive response. Unilateral warm water irrigation of the external auditory canal resulting in contralateral full eye deviation (i.e., away from the irrigated ear) followed slowly by a return to midline is a positive response. **A positive response indicates intact brainstem function. Impaired or absent reflex horizontal eye movements indicates impaired brainstem function at or below the level of the oculomotor nucleus.** This may or may not be the sole cause of the patient's coma.[1,2]

Full vertical eye movements indicate that the etiology of the patient's coma is located in the cerebral hemispheres and not in the brainstem. Bilateral ice water irrigation of the external auditory canals resulting in downward movement of the pupils followed slowly by a return to midline is a positive response. Bilateral warm water irrigation of the external auditory canals resulting in upward movement of the pupils followed slowly by a return to midline is a positive response. **A positive response indicates an intact brainstem function. Impaired or absent reflex vertical eye movements indicate impaired brainstem function at or below the level of the oculomotor nucleus.**

Absent bilateral vestibulo-ocular reflexes in a comatose patient may be due to a variety of reasons. Inadequate irrigation can be due

to cerumen impaction, low irrigation volumes, or poor technique. These causes are obvious and easily corrected. The patient may have other causes for the coma including toxin-induced and metabolic. The patient may also have a preexisting dysfunction of their vestibular apparatus.

AFTERCARE

Dry the patient off to prevent skin maceration from moisture if vestibulo-ocular reflex testing was performed. Examine the tympanic membranes and external auditory canals to assess for any injury related to the testing.

COMPLICATIONS

No significant complications are to be expected if patients with contraindications are excluded. Injury to the tympanic membrane and external auditory canal can occur from the angiocatheter or forceful irrigation. Never use a sharp or metal object to irrigate the external auditory canals. Other potential complications from vestibulo-ocular reflex testing include meningitis, otitis media, and vomiting.

SUMMARY

Central nervous system-induced coma can occur only in the presence of bilateral or diffuse hemispheric lesions, brainstem lesions, or a combination of both. Spontaneous roving eye movements are seen in bilateral hemispheric disease with a relatively intact brainstem. Perform oculocephalic reflex testing if spontaneous roving eye movements in the comatose patient are impaired or absent. The brainstem function is intact if passive turning of the patient's head results in conjugate contralateral eye deviation.

Vestibulo-ocular reflex testing needs only to be performed when oculocephalic reflex testing is abnormal or cannot be performed. Irrigation of the ear canal with ice water will result in eye deviation toward the irrigated ear in the comatose patient with intact brainstem function. Intact full reflex horizontal eye movement indicates that the lesion or lesions causing the coma lie in the cerebral hemispheres and not in the infratentorial brainstem. Incomplete or absent horizontal eye movements suggest a lesion in the brainstem, which may or may not be the sole cause of coma.

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152 Myasthenia Gravis Testing

Eric F. Reichman

INTRODUCTION

Myasthenia gravis is an autoimmune disorder that occurs when polyclonal antibodies bind to a significant number of postsynaptic acetylcholine receptors at the neuromuscular junction leading to inadequate neuromuscular transmission.[1-4] It most commonly affects 10 to 30 year old females and 70 to 90 year old males. Multiple tests are available to diagnose myasthenia gravis.[3,5-12] These include the use of muscle biopsies, curare, edrophonium chloride (Tensilon), electromyography, ice packs, neostigmine, nerve stimulation, and serologic testing. The edrophonium test is the most commonly used diagnostic test for myasthenia gravis.[13-16] Many of these techniques are seldom used and not feasible to perform in the Emergency Department. For these reasons, only the edrophonium test and the ice pack test will be described in this chapter.

ANATOMY AND PATHOPHYSIOLOGY

Acetylcholine is a neurotransmitter of the neuromuscular junction that is released by the presynaptic nerve terminals when stimulated.[5,17] An electrical potential is produced at the myoneural end plate when sufficient numbers of the postsynaptic receptors at the neuromuscular junction are bound by the released acetylcholine. This electric potential then propagates and ultimately leads to muscle contraction. Simultaneously, acetylcholinesterase rapidly terminates the neurotransmission by metabolizing the acetylcholine in the synaptic cleft of the neuromuscular junction.

Myasthenia gravis is an autoimmune disorder that occurs when polyclonal antibodies bind to postsynaptic acetylcholine receptors at the neuromuscular junction.[2-5,17] This leads to inactivation of the receptors and inadequate neuromuscular transmission. Myasthenia gravis is characterized clinically by muscle weakness that develops after repetitive muscle contraction.

Patients are divided into two clinical groups. The first are patients who present with ocular complaints.[10] Patients most commonly present with some degree of ocular muscle involvement. Diplopia is the most common patient complaint. Ptosis is the most visible sign noted on the physical examination. Diplopia with disconjugate gaze can be elicited by having the patient maintain a vertical gaze for approximately 3 minutes. Ptosis can also be made to worsen by having the patient maintain an upward gaze for the same duration of time. These patients may or may not have associated weakness of the pharyngeal and facial muscles that present with the complaints of dysarthria and dysphagia. These symptoms can often be elicited by having the patient count backward from 100.

The second group are patients with proximal muscle weakness. Weakness of the limbs usually involves the proximal muscles and may be asymmetric. Weakness of the muscles can be elicited by having the patient perform repetitive exercises involving the muscle groups in question. **Involvement of respiratory and pharyngeal muscles should be taken very seriously as it may lead to respiratory failure or aspiration.**[2,3,18-21]

Edrophonium chloride is a short-acting acetylcholinesterase inhibitor (anticholinesterase) used in the diagnosis of myasthenia gravis.[3,17] Its onset of action is rapid (within 1 to 2 minutes) and the duration of action is brief (2 to 5 minutes). These characteristics make it ideal to use in the Emergency Department. Edrophonium

given intravenously to patients with myasthenia gravis briefly inhibits the actions of the acetylcholinesterase, thus prolonging the interaction between the acetylcholine and the postsynaptic receptors. This results in a temporary but noticeable improvement in muscle contraction.[1,20] Edrophonium can rapidly, and temporarily, reverse the signs and symptoms of myasthenia gravis.

INDICATIONS

Testing is indicated when the patient's condition is suggestive of myasthenia gravis.[5,6] Edrophonium chloride can be used for patients presenting with diplopia, facial muscle weakness, appendicular muscle weakness, or ptosis suggestive of myasthenia gravis. The ice pack test is reserved to evaluate patients presenting with ptosis.[22-24]

CONTRAINDICATIONS

There is the possibility of worsening the patient's symptoms, causing respiratory distress or arrest, and cardiac dysrhythmias. Do not administer edrophonium if resuscitative equipment, Advanced Cardiac Life Support medications, and additional support personnel are not immediately available. Edrophonium should not be administered if the Physician is not properly trained in airway management and rescue techniques. Use caution when administering edrophonium if the patient's medications can cause atrioventricular node blocking or slowing of transmission (e.g., beta-blockers, calcium channel blockers, and digoxin). These can make the edrophonium associated dysrhythmias more pronounced.

Edrophonium testing is relatively contraindicated in patients with known myasthenia gravis who are taking oral pyridostigmine (Mestinon) and present with increasing weakness. The weakness may be due to insufficient drug treatment (myasthenic crisis) or too much drug treatment (cholinergic crisis).[3] The terminology regarding these types of crises is controversial and beyond the scope of this chapter.[25,26] The patient will improve with edrophonium testing if the etiology of the weakness is a myasthenic crisis. The symptoms will worsen with edrophonium testing if the etiology of the weakness is a cholinergic crisis. Consult a Neurologist, for these reasons, prior to performing an edrophonium test on a patient with known myasthenia gravis who is already taking oral anticholinergic medications.

A patient with known myasthenia gravis complaining of respiratory distress should be assessed and managed similar to any other patient with respiratory compromise. An edrophonium test is contraindicated if it is being used to improve a patient's respiratory distress. A cholinergic crisis treated with edrophonium can further compromise the patient's respiratory status, possibly leading to a respiratory arrest.

The use of edrophonium is contraindicated in pregnancy. It may induce the patient into preterm labor. A neostigmine test is safer if testing is required. Testing should be performed only after consultation with an Obstetrician and a Neurologist.

Edrophonium testing is relatively contraindicated in patients with asthma, bronchospastic disease, cardiac dysrhythmias, or if a group of muscles that are weak are not easily observable. Edrophonium testing should be deferred in favor of neurologic consultation and consideration of other testing methods.

EQUIPMENT

- 10 mg edrophonium chloride (Tensilon)
- Tuberculin syringe
- 10 mL syringe containing 9 mL of sterile normal saline
- Intravenous access with normal saline solution attached to the IV catheter
- Cardiac monitor
- Pulse oximeter
- Noninvasive blood pressure monitor
- Supplemental nasal oxygen
- Resuscitative equipment and medications
- Digital camera or Polaroid camera with instant film
- Ice pack, ice cubes, or instant cold packs

PATIENT PREPARATION

Explain the risks, benefits, and potential complications to the patient and/or their representative. Obtain a signed consent for the procedure. Place the patient sitting upright or supine in a bed. Obtain intravenous access. Apply supplemental oxygen, pulse oximetry, cardiac monitoring, and noninvasive blood pressure monitoring. **Resuscitative equipment and medication must be immediately available in the room if required**.

Identify a group of muscles that can easily be observed and monitored for improvement of function. If possible, the muscle group to be tested should be fatigued. For example, have the patient look upward for 3 minutes to accentuate ptosis. Muscle groups of the extremities can be exercised for several minutes until the patient experiences fatigue. Take a picture, if a camera is available, of the muscle group to be observed after it has been fatigued. This is the "before" photograph.

Prepare the edrophonium chloride. **It is essential that the edrophonium concentration be accurate regardless of who (i.e., Emergency Physician, Nurse, or Pharmacist) prepares the solution.** It is supplied in a concentration of 10 mg/mL. Using sterile technique, transfer 10 mg (1 mL of 10 mg/mL) of edrophonium into a syringe containing 9 mL of sterile normal saline. The resulting solution will contain 1 mg of edrophonium chloride per mL of fluid. **Verify the concentration of the edrophonium solution prior to use, especially if it was made by someone else.**

TECHNIQUE

Administer the edrophonium soon after the weakened muscle is identified, carefully noted, and fatigued. Ideally, one person should administer the medication while another person observes the patient for the effects of the edrophonium chloride. **It may be administered in increasing doses up to a total of 10 mg.** Inject 1 mg (1 mL) of edrophonium chloride intravenously followed by a saline flush. Physical improvement in the observed muscle group should be seen within 30 seconds to 2 minutes if the edrophonium is effective. Muscle improvements will revert to their original state after 2 to 3 minutes. **The test is concluded if there is a positive response to the edrophonium in the observed muscle group.** Inject 3 mg (3 mL) of edrophonium chloride intravenously followed by a saline flush if there is no improvement after the first dose. **The test is concluded if improvement is seen in the muscle group.** Inject the remaining 6 mg (6 mL) of the edrophonium chloride intravenously followed by a saline flush if there is no response within 2 to 3 minutes after the second dose. **The test is considered negative and concluded if there is no response to the third dose of edrophonium (total of 10 mg). A negative test argues against myasthenia gravis but does not completely exclude the diagnosis.**[19,21]

Some Neurologists prefer to give the entire 10 mg (10 mL) dose of edrophonium chloride as a single intravenous bolus and observe the muscle group for improvement. **This has the potential to cause significant bradycardia and is not recommended.**

The total dose of edrophonium to administer to children is 0.15 mg/kg, not to exceed 10 mg of edrophonium.[6] An initial dose of 1 mg is appropriate for children. Administer subsequent doses of 1 to 2 mg to a maximum of 0.15 mg/kg or 10 mg of edrophonium.

ALTERNATIVE TECHNIQUE

ICE PACK TEST FOR OCULAR MYASTHENIA GRAVIS

The ice pack test can be used to diagnose patients with ocular signs of myasthenia gravis.[22-24,27] This test is reserved for the patient presenting with ptosis and/or diplopia suggestive of myasthenia gravis. The basis of this test is the finding that patients with myasthenia gravis have symptoms that worsen in warm weather and that improve in cold weather. Based on this clinical observation, studies have shown that placement of a bag of ice directly over the eyes of myasthenia patients with ptosis actually relieved the symptoms and signs of ocular myasthenia gravis.[22,23] This test is quick, simple, inexpensive, and easy to perform in the Emergency Department. It has none of the potential complications associated with the intravenous administration of edrophonium chloride. There are no contraindications to performing this test.

Place a bag of ice directly over the eye with ptosis and/or diplopia for 2 minutes or until the patient is no longer able to tolerate the cold. An alternative to an ice pack is ice cubes placed in a glove or instant cold packs that are activated by compression. Remove the ice pack from the eye and observe the patient for any improvement in the ptosis or diplopia. **A clear improvement of the ptosis indicates a positive test**. This test may be limited by the patient's intolerance to the cold of the ice pack.

ASSESSMENT

These tests can be positive and confirm the diagnosis of myasthenia gravis. **Objective findings of improved muscle function must be observed to identify the test as positive**. It is very common to observe fasciculations of the facial muscles or tongue after the intravenous administration of edrophonium chloride. **Muscle fasciculations are not considered a positive test**. The patient having the subjective feeling of "feeling better" without objective evidence of improved muscle function is also not considered a positive test. Document improvement by taking a photograph. This is the "after" photograph. Place both the "before" and "after" photographs in the patient's medical record.

AFTERCARE

Many medications used in the Emergency Department and prescribed to a patient at discharge can worsen myasthenia gravis and cause weakness (**Table 152-1**). Look up all current medications as they may be the cause of worsening symptoms in a known myasthenia gravis patient. Look up all medications before administering them in the Emergency Department or writing a prescription.

The patient may be safely discharged if they are ambulatory and without respiratory distress. All patients with suspected or proven myasthenia gravis should be referred to their Primary Care Physician and a Neurologist for further evaluation and management.

TABLE 152-1 Some of the Medications That Worsen Myasthenia Gravis[28-30]

Aminoglycosides	General anesthetics	Penicillins
Antidysrhythmics	Interferon	Phenothiazines
Antiepileptics	Iodinated contrast media	Procainamide
Beta-blockers	Local anesthetics	Quinidine
Calcium channel blockers	Magnesium	Quinine
Chloroquine	Narcotic analgesics	Statins
Clindamycin	Neuromuscular blockers	Steroids
Estrogens	Nitrofurantoin	Sulfonamides
Fluoroquinolones	Ophthalmic beta-blockers	Telithromycin

COMPLICATIONS

Muscarinic side effects can be seen due to the prolonged cholinergic stimulation in patients hypersensitive to edrophonium chloride. These effects include increased salivation, increased lacrimation, and miosis. Some patients may experience bradycardia, junctional rhythms, and ventricular dysrhythmias due to the increased vagal effects of the prolonged acetylcholine stimulation of the heart.[17] Older patients may experience a resultant postural syncope. **These effects are usually transient and self-limited. Intravenous atropine in low doses (0.5 mg) is effective to counteract any of the above symptomatology.**

There are no complications associated with the proper use of the ice pack test. Leaving the ice pack on the eyelids too long can result in a frostbite injury.

SUMMARY

Myasthenia gravis is an autoimmune disease that results in muscle weakness. The edrophonium test allows for a rapid, simple, and safe way to diagnose myasthenia gravis in the Emergency Department. The ice pack test for ocular myasthenia gravis offers a simple alternative to diagnose myasthenia gravis. Obtain prompt Neurologic consultation for all patients presenting with signs and symptoms suggestive of myasthenia gravis.

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SECTION 9

Anesthesia and Analgesia

153 Local Anesthesia

Mark Supino and Daniel Yousef

INTRODUCTION

Modern medicine can trace the use of local anesthetics back to the year 1884 when the Austrian Physician Karl Koller first used topical cocaine to assist with an ophthalmologic operation.[1] The premier Surgeon William Halstead first used injected cocaine to generate the intentional blockade of nerve transmission the following year.[2] Halsted's experiments with cocaine soon led to a concurrent dependency.[3] The emerging illicit market for this compound soon prompted the search for a less toxic agent.[3] Procaine, more commonly known by its trade name Novocain, was the first synthetic local anesthetic. It is a benzoic acid ester derivative developed by the German chemist Alfred Einhorn in 1904. Procaine had fewer drawbacks than its cocaine predecessor but was far from the ideal agent. Lidocaine was the first amide local anesthetic agent and was initially produced in 1945. The market for more effective agents continued to blossom. No less than 20 additional agents were developed for use as local anesthetics, each possessing unique pharmacokinetic properties to tailor its utility to specific clinical applications. They are all synthetic derivatives of cocaine.

The clinical utilization of these agents has become widespread throughout all medical specialties parallel to the proliferation in pharmacologic development. The daily practice of Emergency Medicine presents multiple scenarios that necessitate their use. **The Emergency Physician must maintain a familiarity with the local anesthetic agents available and their individual characteristics, have an expertise in their delivery, be well-informed of the potential side effects, and know how to avoid and treat adverse reactions to ensure the safest and most optimal pain relief.**

PHARMACOLOGY AND PATHOPHYSIOLOGY

Neuronal function and signal conduction are dependent upon a negative resting intracellular electrical potential (approximately –70 mV) as compared to the surrounding extracellular environment at a cellular level. This polarity results from the abundance of sodium (Na^+) cations found within the extracellular space. Membrane-based sodium-potassium (Na^+/K^+) pumps establish this sodium gradient. Adjacent voltage-gated sodium channels maintain the gradient by inhibiting the concentration of mediated and electrically driven sodium influx that would invariably result. A small intracellular sodium influx ensues upon the stimulation of an idle neuron. This sodium influx results in a slight depolarization of the resting membrane potential. The voltage-gated sodium channels reflexively open when a critical threshold of sodium influx is met. This results in a massive sodium ion influx and widespread membrane depolarization.[4-6] Impulse transmission proceeds as this membrane depolarization is propagated down the entire length of the nerve fiber.[7]

Local anesthetic agents function by reversibly binding to membrane-based sodium channels and inhibiting the initial sodium influx that results upon the stimulation of an idle neuron.[4-9] If a significant number of these channels are blocked, the critical threshold of sodium influx required for the voltage-gated sodium channel opening cannot be met, widespread membrane depolarization cannot occur, and nerve impulses will not be transmitted. Careful examination of neuronal cells reveals that the binding sites for local anesthetic agents are located on the internal surfaces of their cellular membranes. The local anesthetic agent must first diffuse across the bilipid cellular membrane as an uncharged lipophilic compound. The local anesthetic agent is converted intracellularly to its active cationic state which terminates signal transduction and produces tissue anesthesia.[10]

All local anesthetic agents generally share the same molecular framework. They contain a lipophilic aromatic group linked to an ionizable group, usually a tertiary amine, via an intermediate linkage.[4,7] This intermediate linkage is either an amino-amide or an amino-ester bond. This property is used to classify the local anesthetics as either "amides" or "esters." Further variations on this generalized structure generate the altered pharmacokinetics that impart the unique qualities to the individual local anesthetic agents.[7]

The potency of a given local anesthetic agent is directly proportional to its lipid solubility.[9,11] More lipophilic agents tend to be more potent. The more soluble agent will readily diffuse across the cellular membrane despite a lower concentration of the extraneuronal anesthetic. A lower quantity of local anesthetic is needed to elicit the same endpoint response.[12] Variable potencies are rarely of significance to the Emergency Physician. The available preparations utilize concentrations of the local anesthetic specifically formulated to correct for this variation.

The primary determinant of a local anesthetic's onset of action is its pKa.[13] **The pKa is the pH at which a given drug exists in equal proportions as ionized and unionized molecules.** The unionized molecules more readily cross the nerve cell membrane. A portion of the molecule becomes charged once inside the neuron. The ionized portion binds most completely to receptor proteins within the sodium channels.[4,11] Commercially available local anesthetic agents are weak bases with pKas of 7.6 to 8.9.[4] Agents with a lower pKa at physiologic pH (7.4) will have relatively more uncharged molecules free to cross the nerve cell membrane (i.e., faster onset) than agents with a higher pKa.[6,11] Low tissue pH results in so little local anesthetic in the uncharged form that the agent is ineffective.[6] This phenomenon explains why local anesthetics function poorly in acidic environments (e.g., abscess cavities) as a greater percentage of the agent is secondarily converted to its ionized state within the extracellular environment. It also explains why the addition of a buffering agent such as sodium bicarbonate increases the pH of the injected local anesthetic solution, increasing the ratio of nonionized local anesthetic agent present, and enhancing the rapidity of anesthesia onset. Increasing the total quantity of local anesthetic injected, whether by manipulating its concentration or increasing the overall volume, will hasten the onset of activity by augmenting the diffusion gradient and more aggressively driving the local anesthetic agent intracellularly.[7,8] Local anesthetic agents with a higher intrinsic pKa generate more effective sodium channel blockade intracellularly via their tendency to persist in an ionized and functionally active

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state.[8,10] The rate at which a local anesthetic agent diffuses through surrounding nonneuronal tissues also appears to be an important variable.[5,8]

A local anesthetic agent's duration of activity is typically proportional to the overall degree of protein binding.[6-8,11] Those that possess a higher affinity for the target receptor are less likely to be dislodged and exhibit a longer duration of action.[7,11] **All the available local anesthetic agents produce a degree of localized vasodilation.** This serves to increase regional blood flow, promote systemic absorption of extracellular fluid, and decrease the total duration of activity.[5,7,11] Counteracting this phenomenon by compounding a vasoconstrictor such as epinephrine with the local anesthetic agent represents another practical way to increase the duration of action.[4,14]

The two classes of local anesthetic agents undergo metabolism via different mechanisms.[5,7,15-17] Esters are metabolized rapidly by plasma pseudocholinesterase to the major metabolite para-aminobenzoic acid (PABA).[5,7,15] PABA is allergenic and likely responsible for the infrequent allergic reactions to ester agents.[5,7,16] **Patients with atypical pseudocholinesterase activity are at increased risk for systemic toxicity from ester anesthetic agents.[16,17]** Amide anesthetic agents are metabolized more slowly by the liver to a variety of metabolites that are unrelated to PABA. **Patients with impaired liver function are at increased risk for systemic toxicity from amide anesthetic agents.[16,17]**

INDICATIONS

Local anesthetic agents are used in the Emergency Department for local infiltration, regional nerve blockade (Chapter 156), peripheral nerve blockade, and topical application (Chapter 154). General principles concerning their use and local infiltration will be discussed in this chapter. A complete review of specific techniques and procedures is available elsewhere in this text.

Localized injected anesthesia, or infiltrative anesthesia, represents the most widespread use of local anesthetic agents within the Emergency Department. It is generally a safe procedure that is technically easy to perform and well tolerated by the patient. It can be readily used for surgical procedures that are routinely performed by the Emergency Physician. This includes wound repair, foreign body removal, cutaneous abscess drainage, intravascular catheter placement, and hematoma blocks.

CONTRAINDICATIONS

The primary and most important contraindication to the use of a local anesthetic agent is a history of an allergic reaction. Less than 1% of all adverse reactions from local anesthetic agents are due to true IgE-mediated allergic phenomena.[18,19] Ester agents are responsible for most of such cases, whereas allergic reactions to amide anesthetics are exceptionally rare.[7,15,20-22] The physiology behind this finding is rooted in the divergent metabolism of the two classes of local anesthetic agents.

The ester-type local anesthetic agents are rapidly metabolized within the bloodstream by serum pseudocholinesterase which explains their very short plasma half-lives. **The secondary metabolites are derivatives of the organic compound PABA. They exhibit allergenic properties and are the agents responsible for the allergic response.** This property makes ester-type local anesthetic agents (e.g., cocaine, procaine, and tetracaine) a less commonly used option in daily practice.

Amide-type local anesthetic agents are metabolized in the liver into nonallergenic byproducts via cytochrome P450-mediated pathways. Allergic reactions to the amide-type local anesthetic agents are very rare but do occur. Methylparaben is a close analog of PABA and a preservative commonly used in multidose preparations of amide agents. It may account for the rare occurrence of an apparent allergic reaction to the amide class of local anesthetic agents.[7,15,16,22-25] **A simple way to remember if an agent is an ester or an amide is that all amides will have two "i's" in their generic name (e.g., lidocaine and bupivacaine) whereas esters only have one (e.g., procaine and tetracaine).**

An agent from the opposite class may be chosen if a history of a prior allergic reaction to an agent is obtained from a patient requiring local anesthesia. There is no true cross reactivity between the esters and the amides.[7] Choose a preparation free of preservatives to avoid possible reactions.[23] Solutions intended for single and/or intravenous uses tend to be free of preservatives. Intravenous formulations of lidocaine can be found in any standard Emergency Department "crash cart."

Infiltration of a wound with 1% diphenhydramine is clinically effective as an alternative agent in the rare circumstance that a patient has a true IgE-mediated allergy to the local anesthetic agents.[26] **Diphenhydramine is more painful to inject than the local anesthetic agents and is less effective at attaining adequate anesthesia when compared to local anesthetic agents. Reserve the use of diphenhydramine for the rare instance of a patient with an actual local anesthetic allergy.[26]** Dilute the 5% parenteral formulation of diphenhydramine to 1% (i.e., add 1 mL of diphenhydramine to 4 mL of normal saline) to make a solution for local infiltration.

The size of the anesthetic field required for a given procedure can further dictate the utility of infiltrative anesthesia. Larger fields will require the injection of larger quantities of local anesthetic solution. **Do not exceed the maximum safe dose of a local anesthetic agent (Table 153-1). Consider an alternative anesthetic technique (e.g., regional nerve blockade or procedural sedation) if infiltrative anesthesia requires potentially toxic local anesthetic doses.** An alternative is to dilute the local anesthetic solution in half using sterile normal saline. This diluted local anesthetic solution may not provide adequate anesthesia.

TABLE 153-1 Properties and Dosages for Injectable Local Anesthetic Agents

Local anesthetic class	Anesthetic agent	Relative potency	Relative onset of action	Duration of action (minutes)*	Maximum dose without epinephrine		Maximum dose with epinephrine	
					(mg/kg)	(mg)	(mg/kg)	(mg)
Ester	Procaine (Novocain)	1	Slow	45–90	8.0	500	10.0	600
Ester	Chloroprocaine (Nesacaine)	2	Rapid	30–90	11.0	800	14.0	1000
Ester	Tetracaine (Pontocaine)	8	Slow	180–600	1.5	100	2.5	200
Amide	Lidocaine (Xylocaine)	2	Rapid	120–240	4.5	350	7.0	500
Amide	Mepivacaine (Carbocaine)	2	Rapid	180–360	5.0	300	7.0	500
Amide	Bupivacaine (Marcaine)	8	Slow	240–480	2.5	175	3.0	225
Amide	Etidocaine (Duranest)	8	Rapid	240–480	2.5	175	4.0	300

*Longer times represent the addition of epinephrine to the local anesthetic solution.

Certain procedures necessitate meticulous tissue reapproximation (e.g., facial lacerations). The injection of local anesthetic solution can distort the surrounding tissue and diminish the likelihood of an optimal outcome. Consider an alternative anesthetic technique in these situations.

Topical anesthetic agents are contraindicated on mucous membranes, the eye, denuded skin, or burned skin as they are rapidly absorbed through these tissues. Absorption can produce severe systemic toxicity and death. Eye contact can produce corneal injury. Topical agents containing cocaine and epinephrine are contraindicated in regions of end artery flow because they can result in intense vasoconstriction.[27] The U.S. Food and Drug Administration (FDA) has required a boxed warning against the use of prescription viscous lidocaine for infant and children teething.[28] Studies have shown that topical pain relievers are largely ineffective, wash out of the child's mouth rapidly, and if ingested in large enough quantities may result in complications (e.g., seizures, central nervous system [CNS] injury, and cardiac dysrhythmias).[29-31] Caregivers are encouraged to alleviate dental pain by using chilled teething rings or by gently massaging the affected gums with fingers.[32]

EQUIPMENT

- Syringes, 1 mL to 20 mL sizes
- Needles, various sizes and lengths
- Local anesthetic solution, with or without epinephrine
- Alcohol swabs, povidone iodine, or chlorhexidine solution
- Gauze squares
- 8.4% sodium bicarbonate solution (i.e., 1 meq/mL)
- Gloves
- Face mask with an eye shield or goggles
- 5% parenteral diphenhydramine solution

Universal precautions are of utmost importance in performing any procedure using a local anesthetic agent. It is vital to wear gloves when administering topical anesthetic agents to protect the fingers, to prevent absorption of the local anesthetic agent through the fingers of the healthcare worker, and to prevent introduction of bacteria into the wound. The use of sterile as compared to nonsterile clean gloves has not been shown to alter the rate of infection for the repair of simple traumatic lacerations.[33] Nonsterile nonlatex gloves are generally sufficient for most repairs.[33] Wear a mask with a face shield or goggles to prevent accidental mucous membrane exposure if the injectable local anesthetic solution shoots out of the wound margins.

One of the most important determinants of pain response during administration of a local anesthetic agent is needle size. Use a 25 or 27 gauge needle for infiltration to minimize pain. A long needle allows for the infiltration of a larger region with a single needle pass and decreases the number of times tissues must be punctured. A 2.0 inch, 27 gauge needle is typically an optimal choice. Do not insert the needle more than two-thirds of its length to prevent inadvertent breakage within the tissues.[13] **Another important factor is the rate of infiltration.** A slow steady method minimizes the pain response.

Many Emergency Departments have a small local anesthesia tray or basket containing the necessary equipment for providing local anesthesia. Such a kit may include the following items: needles in sizes from 18 to 30 gauge, 1 to 2 cm long, and 4 cm long; syringes ranging from 1 mL (tuberculin) to 10 mL; cotton-tipped applicators for the application of topical agents; local anesthetic agents such as 1% and 2% lidocaine, 0.25% and 0.5% mepivacaine or bupivacaine, and the same agents combined with 1:200,000 epinephrine. Sodium bicarbonate may be used for buffering the local anesthetic agent. Alcohol swabs, povidone iodine swabs or solution, or chlorhexidine swabs or solution are required for cleansing the skin. Nonsterile and sterile examination nonlatex examination gloves are required for the infiltration of the local anesthetic agent and performing the procedure.

PATIENT PREPARATION

Discuss the procedure with the patient and/or their representative. The most common adverse reaction to a local anesthetic agent is a vasovagal reaction.[13] This complication is more likely when patients are anesthetized while sitting in an upright position (e.g., dental procedures). The Emergency Physician must take precautions to alleviate secondary injury to the patient. Place the patient supine in a bed whenever possible with the side rails up to prevent injury no matter how minor the procedure. Friends and family members have been reported to experience syncope upon witnessing injections. Ask them to leave the room prior to starting or keep them in a sitting position throughout the duration of the procedure. Sedation (Chapter 159) can minimize the response to treatment while maintaining stable vital signs and spontaneous respirations when the patient exhibits considerable anxiety.

Prepare the area prior to injecting local anesthetic solution. Clean the skin of any dirt and debris. Apply an alcohol swab, povidone iodine, or chlorhexidine to the skin over the injection site and the surrounding area. Allow the solution to dry. Apply sterile drapes, if applicable, to delineate a sterile field.

LOCAL INFILTRATION AND PERIPHERAL NERVE BLOCKADE

LOCAL ANESTHETIC DOSING

Infiltration anesthesia refers to the injection of a local anesthetic solution directly into the subcutaneous tissues about to be manipulated. The first step to be considered is selection of the proper anesthetic. Barring a history of an allergic reaction to a given class of local anesthetic agents, the amide anesthetics lidocaine (i.e., Xylocaine) and bupivacaine (i.e., Marcaine) are the most common agents employed within the Emergency Department. Tailor the choice between these agents to the individual patient and situation. Lidocaine exhibits a quicker onset of activity whereas bupivacaine exhibits a longer duration of action (**Table 153-1**). Lidocaine possesses a wider margin of safety, with larger doses required to illicit a toxic response. The ester agent procaine (i.e., Novocain) is a reasonable alternative in amide-allergic patients.

It is of utmost importance to remember the maximal recommended doses for the chosen local anesthetic agent to avoid systemic toxicity (Table 153-1). The disadvantage of local infiltration is that a large volume of local anesthetic may be required for a small area. Extensive wounds may require toxic doses of local anesthetic agents to achieve adequate anesthesia. A lower concentration or the addition of epinephrine will allow a larger volume of local anesthetic to be used. Local infiltration may distort the wound edges and complicate the repair.

The maximal recommended dose of a local anesthetic agent is the same for local infiltration or regional nerve blockade. It is important to properly calculate the amount of local anesthetic agent administered to a patient. Local anesthetic solutions are supplied with the concentration denoted as a percentage (e.g., 1% lidocaine, 0.25% bupivacaine, 4% cocaine). This percentage must be converted to mg/mL. A 1% local anesthetic solution is prepared by dissolving 1 gm (i.e., 1000 mg) of the local anesthetic agent in 100 mL

of diluent. This results in a concentration of 10 mg/mL (i.e., 1 gm/100 mL = 1000 mg/100 mL = 10 mg/mL). **A simple method to calculate the strength of a local anesthetic solution is to move the decimal point one place to the right to convert from a percentage to a concentration in mg/mL (e.g., 0.25% = 2.5 mg/mL, 2% = 20 mg/mL, 4% = 40 mg/mL). This value must be multiplied by the volume to be administered to determine the total amount in mg of local anesthetic agent. This value must be compared to the maximal allowable dose (Table 153-1) to ensure it is a safe dose.**[34]

EPINEPHRINE-CONTAINING AGENTS

Epinephrine can be added to a local anesthetic solution to prolong its duration of action, to assist with hemostasis by local vasoconstriction, and to slow the absorption of the local anesthetic agent. This allows for larger quantities of local anesthetic solution to be injected and diminishes the concern for systemic toxicity. Local anesthetic agents generally compounded with epinephrine in a 1:100,000 or 1:200,000 solution are available from the manufacturer.

One can be readily formulated if a compounded solution is not available. Begin by obtaining 1:1000 epinephrine, which is usually administered to patients for severe allergic reactions or bronchospasm. This solution of 1:1000 contains 1 gm of epinephrine per 1000 mL or 1 mg/mL of epinephrine. Diluting this solution by a factor of 100 will result in the desired 1:100,000 concentration. Place 0.1 mL of 1:1000 epinephrine into 10 mL of local anesthetic solution to make a dilution of 1:100,000 or 0.010 mg/mL. Place 0.1 mL of 1:1000 epinephrine into 20 mL (i.e., 0.05 mL in 10 mL) of local anesthetic solution to make a dilution of 1:200,000 or 0.005 mg/mL.

REDUCING THE PAIN OF INFILTRATION

Local anesthetic agents are weak bases. They are packaged as hydrochloride salts with a pH of 4 to 6 to increase their solubility and shelf life. This acidic pH causes much of the pain associated with the injection of local anesthetic agents.[4,6] Epinephrine is unstable at a physiologic pH. Commercial solutions containing epinephrine are generally formulated with similarly acidic pH values. **Buffering lidocaine, mepivacaine, or bupivacaine with sodium bicarbonate has been shown to reduce the pain of injection significantly.**[27,35,36] Raising the pH of the local anesthetic agent increases the percentage of local anesthetic molecules in the nonionized diffusible state.[35] This may allow nearly instantaneous penetration of the nerve cell membrane by the anesthetic molecules and block or reduce the pain of infiltration.[35,37,38] **Buffering local anesthetic agents does not affect the duration of anesthesia, degree of anesthesia, the serum levels of the local anesthetic agent, or the toxicity of buffered anesthetic agents.**

Caution must be exercised when buffering highly lipophilic and less soluble anesthetic agents (e.g., bupivacaine) because precipitation can occur.[4] Use the prepackaged 50 mL ampules of 8.4% (i.e., 1 meq/mL) sodium bicarbonate found in any "crash cart." Add 1 mL of 8.4% (i.e., 1 meq/mL) sodium bicarbonate to 10 mL of 1% lidocaine or 1% mepivacaine, with or without epinephrine, to achieve buffering.[35] Add 0.05 to 0.10 mL of 8.4% (1 meq/mL) sodium bicarbonate to each 10 mL of 0.5% bupivacaine.[36]

The use of warm lidocaine [37°C (98.6°F) to 42°C (107.6°F)] decreases the pain of infiltration.[39-41] The exact etiology of this is unknown. It is hypothesized that warm lidocaine does not stimulate cold receptors and diffuses into tissues faster. Warm the local anesthetic agent by placing it in a blanket warmer or a water bath. The combination of warming and buffering a compound results in an even less painful procedure.[40,42]

Other simple measures can help reduce the pain of the local anesthetic injection. Limit the infiltration of a local anesthetic solution to the subdermal space. This will help decrease the overall pain of injection, minimize the degree of tissue distortion, and protect the patient from the inadvertent injection into an intravascular space. Make every attempt to infiltrate through the exposed wound edges rather than puncturing through the adjacent intact skin when anesthetizing an open wound (**Figure 153-1**).[43] This will provide a much less painful experience for the patient. Infiltrate grossly contaminated wounds percutaneously through clean and intact skin to decrease the risk of spreading the contamination and to decrease the risk of an infection related to the infiltration (**Figure 153-2**). Limit the number of needle punctures through uninjured skin in contaminated wounds. The application of gentle pressure to the injection site prior to the injection (e.g., pressing a sterile cotton tipped applicator against the skin) limits the pain associated with childhood vaccinations.[44] Dentists have been using similar distraction techniques for many years to perform dental blocks. The use of this technique in the Emergency Department is limited by the fact that infiltrations into previously inflamed tissue and secondary stimulation may expose the patient to unwarranted additional pain. A slower rate of local anesthetic solution injection is associated with less pain.

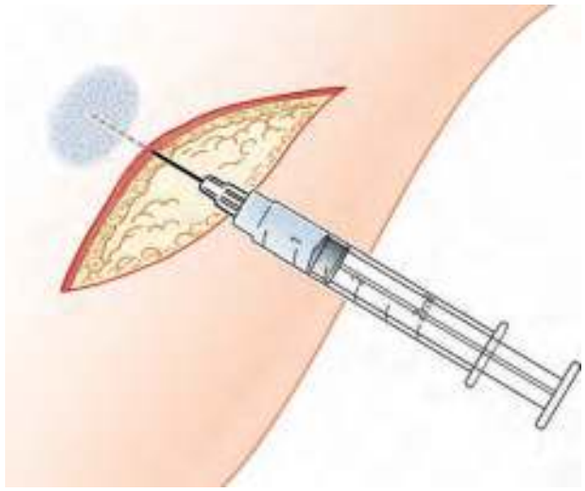

FIGURE 153-1. The needle is inserted through the wound edge to inject local anesthetic solution.

Insert the needle into an open wound at its apex (**Figure 153-2**). Tunnel the needle its entire length down the wound margin. Inject

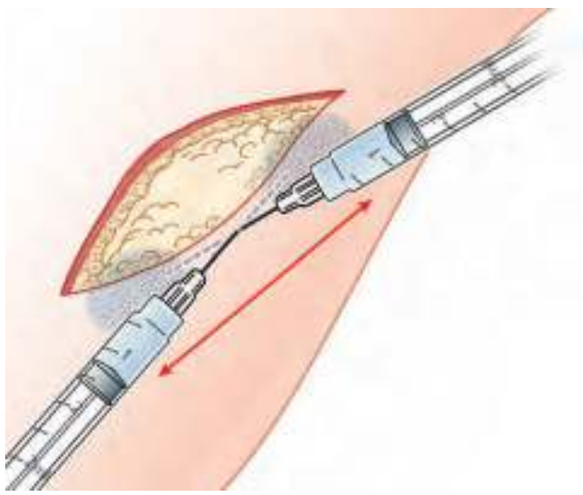

FIGURE 153-2. The needle is inserted through the intact skin to inject local anesthetic solution.

the local anesthetic solution while slowly withdrawing the needle. Do not completely withdraw the needle from the skin. Redirect it along the opposing wound margin and repeat the technique. This can be performed down the entire length of the wound as necessary. Minimizing the number of skin punctures will help to minimize pain.

In summary, the pain upon injection of local anesthetic agents can be reduced by following a few simple suggestions.[45,46] Warm and/or buffer the local anesthetic agent. Inject the local anesthetic agent slowly. Inject open wounds through the wound edges and not through intact skin except when the wound is grossly contaminated. Infiltrate subdermally to minimize pain and tissue distention. Insert and advance the needle to create a tract and inject as the needle is withdrawn to minimize tissue distention. Do not totally withdraw the needle after infiltration when possible. Leave the tip of the needle within the skin and redirect the needle to prevent excessive skin punctures.

COMBINING LOCAL ANESTHETIC AGENTS

Physicians have combined various local anesthetic agents to exploit the unique properties of each individual local anesthetic agent, to achieve a rapid onset, and to achieve a prolonged duration of action.[47,48] The rapid-acting lidocaine can be safely mixed in a 1:1 ratio with longer acting but slower onset bupivacaine. Employing this mixture may be no more dangerous than sequentially administering equal doses of either parent compound. **The value of such an approach is questionable.** Enough concern persists regarding the potential toxicity of this mixture to often preclude its use within the Emergency Department. The combined benefits might not be as relevant within the Emergency Department as most studies referenced come from the anesthesia and surgical literature.

It is important to recognize that the toxic effects in an overdose situation are additive, even if an amide and ester are combined.[49,50] Mixing two local anesthetic agents can complicate the overall dosage calculations and enhance the potential for a toxic injection. **It is difficult to determine the maximum dose if two local anesthetic agents are mixed together.** It will be impossible to determine which of the two anesthetics is the causative agent if the patient develops an allergic reaction. **The combination of local anesthetic agents cannot currently be recommended as general practice. Use lidocaine containing epinephrine, bupivacaine, or bupivacaine containing epinephrine to prolong the anesthetic effect rather than combining two local anesthetic agents.**

ALTERNATIVE TECHNIQUES

It has been known since the 1940s that injected antihistamines exhibit anesthetic properties. A 1956 study comparing the infiltration of diphenhydramine to procaine demonstrated equal anesthetic properties. Subsequent Emergency Medicine-based studies have shown equal anesthetic results when comparing the injection of 1% solutions of either lidocaine or diphenhydramine, although the latter was associated with a more painful infiltration. A further study comparing 0.5% diphenhydramine to 1% lidocaine demonstrated resolution in the disparity regarding the pain of injection, albeit at the expense of creating a disparity in the anesthetic effect.[51] **The overall duration of anesthesia produced by infiltrated diphenhydramine is shorter than that of lidocaine.** A concern has arisen regarding the possible destruction of local tissue and subsequent skin necrosis associated with diphenhydramine infiltration. Multiple early experiences had reported this complication. An appropriate dilution prior to injection eliminates this potential complication. Dilute 1 mL of standard parenteral 5% diphenhydramine solution with 4 mL of sterile normal saline to achieve a 1% solution that is suitable for injection.[52,53] **Consider diphenhydramine a viable alternative to the more commonly used local anesthetic agents when they are contraindicated.**

LIPOSOMAL BUPIVACAINE

Liposomal bupivacaine (Exparel) is a long-acting form of bupivacaine. This formulation slowly releases bupivacaine from liposomes, thereby delaying the peak plasma concentrations and increasing the duration of action with a half-life of greater than 24 hours. It is approved by the U.S. FDA for pain relief by local infiltration in postsurgical patients. The preponderance of data is specifically for bunionectomy and hemorrhoidectomy surgeries. Studies have shown it to be effective for postoperative pain relief with opioid-sparing effects. It has been found to have an acceptable adverse effect profile.

Liposomal bupivacaine can be administered undiluted or diluted to increase volume up to a concentration of 0.89 mg/mL (i.e., 1:14 dilution by volume) with normal saline or lactated Ringer's solution. The dosing in the trial for a bunionectomy was a total of 8 mL or 106 mg administered as 7 mL infiltrated into the tissues surrounding the osteotomy and 1 mL infiltrated into the subcutaneous tissue.[54] The dosing in the trial of hemorrhoidectomy surgery was a total of 20 mL or 266 mg with 10 mL of saline divided into six 5 mL aliquots injected by visualizing the anal sphincter as a clock face and slowly infiltrating one aliquot to each of the even numbers to produce a field block.[55]

Different formulations of bupivacaine are not bioequivalent. It is not possible to convert dosing from any other formulations of bupivacaine to liposomal bupivacaine. Non–bupivacaine-based local anesthetics may cause an immediate release of bupivacaine from Exparel if administered together locally. The administration of liposomal bupivacaine may follow the administration of lidocaine after a delay of 20 minutes or more.[56]

Dosing recommendations and the preparations of the product for local infiltration in nonsurgical procedural cases have not been established. The use of liposomal bupivacaine has not been evaluated in pediatric patients or in pregnancy. More studies are needed to establish the safety and efficacy for liposomal bupivacaine in epidural or intrathecal use, regional nerve blocks, and intravascular or intraarticular use. The application of liposomal bupivacaine in Emergency Department practice cannot be recommended, although it could prove to be a useful adjunct in the very near future.[53]

ASSESSMENT

It is important to wait long enough after the local anesthetic agent is administered to allow the onset of adequate anesthesia before the planned procedure is started. A period of 5 to 10 minutes is usually adequate for subcutaneous local infiltration. A period of 15 to 30 minutes may be required for peripheral nerve blocks. A simple examination of the area being anesthetized with fine touch and pinprick is important to ensure adequate anesthesia prior to the procedure. It may be necessary to inject additional local anesthetic solution in areas of continued sensitivity while keeping in mind the total dose injected to avoid toxicity.

AFTERCARE

Observe the patient for a minimum of 15 minutes following the use of local anesthesia to assure no evidence of an adverse reaction. The length of time analgesia is maintained depends upon the agent used for the procedure (**Table 153-1**). The patient need not wait in the Emergency Department for normal sensation to return prior to discharge. They should be instructed to return to the Emergency Department if normal sensation has not returned within 12 to 24 hours.

COMPLICATIONS

Toxic reactions to local anesthetics agents are far more common than allergic sequelae.[13,17,57] The propensity for toxicity is directly proportional to the potency of the drug.[7,9,11] **Table 153-1** lists the recommended maximal doses for commonly used local anesthetic agents. **These are only estimates, and in certain circumstances, the toxic dose may be considerably less.[7,21] Infiltration into highly vascular areas, inadvertent intravascular injection, or application to mucous membranes may cause toxicity at accepted standard doses.[7]**

It is unlikely that toxic serum concentrations of local anesthetic agents will be reached in most clinical situations in the Emergency Department. For example, the maximum dose of 1% lidocaine without epinephrine in a 70 kg patient would be approximately 31.5 mL. This is a volume that is more than adequate for most wounds. **It is important to be vigilant of the total dose administered, especially in patients with large or multiple lacerations in whom higher doses of local anesthetics may be required.** A less concentrated form of the local anesthetic agent (e.g., 0.5% lidocaine as opposed to 1%) may be used when the maximum dose could be exceeded; however, this may decrease the degree of anesthesia. Procedural sedation (Chapter 159) at the bedside or general anesthesia in the Operating Room may be required to repair larger wounds.

The major manifestations of local anesthetic toxicity occur in the CNS and the cardiovascular system.[6,9,13,15] Initial signs and symptoms are of CNS excitation. This occurs from the suppression of inhibitory cortical neurons and leads to unopposed functioning of facilitatory pathways. Signs and symptoms include ataxia, delirium, disorientation, dizziness, lightheadedness, nystagmus, psychosis, restlessness, sensory disturbances (e.g., visual difficulties, tinnitus, perioral tingling, metallic taste in the mouth), unconsciousness, and an unsteady gate.[58-60] Muscle twitching, slurred speech, and/or tremors may immediately precede seizures. There may also be augmentation of medullary and sympathetic activity with resultant hyperpnea, hypertension, tachycardia, and tachypnea. Generalized depression of the entire CNS can occur and is manifested as coma, drowsiness, and ultimately respiratory arrest.[9,15]

Management of CNS toxicity should begin with an assessment of the patient's airway, breathing, and circulation. Treatment is primarily supportive. Hypoxia and acidosis enhance CNS and myocardial absorption of local anesthetic agents and must be addressed aggressively. Hypocapnia raises the seizure threshold to prevent convulsions by inducing cerebral vasoconstriction and by decreasing the delivery of the local anesthetic agent to the CNS.[7,61] An alert and cooperative patient may be instructed to hyperventilate if early signs of toxicity are present.[6,21,62] Manage seizures with intravenous benzodiazepines to raise the CNS threshold to local anesthesia-induced convulsions. The metabolism of local anesthetics is short enough that loading patients with long-acting antiepileptic drugs (e.g., phenytoin) is generally not required. The effect of phenytoin upon sodium channel conduction can potentiate the arrhythmogenic property of local anesthetics. Administer short-acting neuromuscular blocking agents (e.g., succinylcholine or vecuronium) until serum levels of the local anesthetic agent decline if the seizures fail to respond to benzodiazepines.[7] Succinylcholine is metabolized by pseudocholinesterase. This is the same plasma enzyme that metabolizes ester anesthetic agents. Avoid succinylcholine in ester-induced seizures.[7,6,21]

The cardiovascular system is relatively resistant to local anesthetic toxicity in comparison with the CNS.[17,25] The cardiovascular system does not exhibit toxicity until much higher blood levels are reached.[17,25] Cardiovascular toxicity results from the direct effects upon cardiac and vascular smooth muscle and the indirect effects upon autonomic tone. Complications are the result of negative inotropism, peripheral vasodilatation, and slowing of the myocardial conduction system.[9,15] Arterial dilation combined with decreased cardiac contractility leads to progressive hypotension and eventual cardiovascular collapse. The sodium and calcium channel blockade exhibited at more toxic doses predisposes the patient to fatal arrhythmias. The end results are hypotension, bradycardia, prolonged electrocardiographic intervals, and eventually cardiac arrest.[9,11,15] Treatment is supportive with inotropes, intravenous fluids, and vasopressors. Avoid class IB antidysrhythmics (e.g., lidocaine, mexiletine, and tocainide) due to their potential to accentuate cardiac sodium channel blockade. Vasopressors with positive inotropic effects (e.g., dopamine) will treat profound cardiovascular depression.[7]

Bupivacaine is more cardiotoxic than other local anesthetic agents. Treatment of bupivacaine cardiotoxicity has shown a potential benefit with the use of high-dose intravenous insulin (e.g., 2 IU/kg) in concert with supplemental intravenous glucose and intravenous potassium boluses.[63] The infusion of a lipid emulsion (i.e., Intralipid) has also been proposed for use in bupivacaine, levobupivacaine, lidocaine, and ropivacaine.[64-76] The actual mechanism of action is unclear. Hypotheses include the extraction of lipophilic anesthetics from their target tissues via the fatty emulsion and/or the direct antagonization of anesthetic-mediated suppression of cardiac fatty acid metabolism.[77,78] Always attempt the rescue interventions with ongoing Advanced Cardiac Life Support protocols.

Treatment of symptomatic local anesthetic toxicity involves a large initial IV bolus of 20% lipid emulsion at 1.5 mL/kg over 1 minute followed by an IV drip of 0.25 mL/kg/min.[64,79] Use ideal body weight to calculate the dose. The bolus can be repeated two times at 5 minute intervals and the drip rate doubled. Continue the drip at 0.025 mg/kg/min as needed for up to 6.5 hours. The maximum total dose of Intralipid administered should not exceed 12 mL/kg.[78] Consultation with a Toxicologist is recommended. Although the lipid-based drug propofol is often more readily available in the Emergency Department than Intralipid, the former is compounded in a 10% lipid solution. **The volume of propofol solution needed to reverse local anesthetic toxicity requires the administration of a toxic dose of propofol and is therefore contraindicated.[80-82] Intralipid administration is strictly contraindicated in any patient with soy or egg allergies.**

Intralipid also comes in a 30% emulsion.[74,83] The dosing is different from the above 20% emulsion. Administer the 30% emulsion in a bolus of 1 mg/kg over 1 minute followed by an infusion of 10 mL/kg/h over 15 minutes. The bolus can be repeated two times at 5 minute intervals and the drip rate doubled. The maximum total dose of Intralipid administered should not exceed 6 mL/kg.[74,83]

Adding epinephrine to a local anesthetic agent increases both the amount of drug that can be administered and the duration of action.[4,14] It decreases bleeding into the surgical field. There are significant drawbacks to the use of epinephrine. These include increased pain of infiltration, increased wound inflammation, increased wound infection rates, increased uncomfortable side effects in susceptible patients (e.g., palpitations, tremors, syncope), and the potential for severe tissue ischemia if used in regions of end arterial circulation.[13] Emergency medicine dogma cautions against the use of an epinephrine-containing local anesthetic agents in regions of end arterial circulation (e.g., digits, the tip of the nose, the pinna, or the penis) to avoid potential distal tissue infarction and necrosis.[84] The clinical evidence supporting this concern is scant and the little that does exist is dated. Reviews of digital nerve blocks performed with epinephrine found no cases of digital necrosis or gangrene.[85-87] Most of these studies were carried out in healthy patients without peripheral vascular disease. The few studies carried out in patients with poor circulation did not demonstrate any adverse effects from using epinephrine.[85,87] Current practice guidelines suggest that the danger of using epinephrine in extremities is likely overstated and

any appreciable vasoconstriction is transient.[88] Literature focused on the inadvertent infiltration of epinephrine into fingers via improperly used autoinjectors has similarly failed to demonstrate a single significant case of tissue necrosis.[89] Most of these cases demonstrated a spontaneous return of perfusion with only conservative treatments (i.e., warm soaks and digital massage). Prolonged vasospasm and ischemia in these areas can be reversed with the subcutaneous infiltration of 1.0 mL of 1:1000 phentolamine (i.e., 1 mg) diluted with saline in a 1:1 mixture at the local anesthetic injection site.[90-94] A less invasive alternative is to apply topical nitroglycerin paste to the affected area. Caution must be used when administering epinephrine to patients who are elderly, are taking beta-blockers, or have a history of coronary artery disease, hypertension, hyperthyroidism, or pheochromocytoma.[6,13] Inadvertent intravascular injection of epinephrine can have fatal consequences.[95,96]

The potential systemic complications unique to epinephrine are not often an issue. One must infiltrate 30 mL of local anesthetic solution to attain a cumulative dose of 0.3 mg using 1:100,000 preparations, a quantity commonly used subcutaneously for anaphylactic allergic reactions. Such a large volume is rarely encountered with infiltrative anesthesia. Consider an alternative anesthetic technique if a large volume of local anesthetic solution containing epinephrine is required. Rare reports of patient mortality secondary to an inadvertent intravenous injection have been reported. Use extreme care when using these preparations in any patient with suspected cardiovascular disease.

True allergic reactions are rare adverse events.[19] Effective management of an allergic reaction depends upon its severity and may include the use of epinephrine, antihistamines, steroids, and vasopressors. A complete review of the management of allergic reactions and anaphylaxis can be found in standard Emergency Medicine textbooks.

A rare but acute nonallergic type of reaction to lidocaine is called Hoigne syndrome. It is characterized as a "doom anxiety" and is a non–dose-dependent primarily psychiatric reaction to lidocaine.[97] Patients may complain of anxiety, lightheadedness, palpitations, and feeling like they are about to die. There are various theories as to the pathophysiology of Hoigne syndrome. The exact mechanism is still unknown. Treatment is supportive and standard benzodiazepine administration is usually sufficient. The condition is self-limited and patients may be discharged home once symptoms resolve. Warn the patient that they may experience subsequent anxiety, bad dreams, or muscle tension for up to 7 days after the initial inciting reaction.[97]

Methemoglobinemia has been reported to occur following the use of both classes of local anesthetic agents.[98-102] It is most commonly seen after the use of the topical agent benzocaine.[98-102] The use of this medication is discussed elsewhere in this book. The Emergency Physician must be aware of this potential complication and intervene appropriately. **Methemoglobin is an altered state of hemoglobin where the ferrous (i.e., Fe^{2+}) irons of heme are oxidized to the ferric (i.e., Fe^{3+}) state.** The ferric irons are unable to bind oxygen, resulting in impaired oxygen delivery to tissues.

Initial methemoglobinemia is characterized by a cyanotic-looking appearance and low oxygen saturation that does not respond to supplemental oxygen. Patients may complain of dizziness, dyspnea, exercise intolerance, fatigue, headache, and weakness when methemoglobin levels reach approximately 20% or more. Serious complications (e.g., chest pain, CNS depression, dysrhythmias, seizures, and tachypnea) occur at methemoglobin levels above 50%. Death is associated with methemoglobin levels exceeding 70%. Patients with preexisting anemia may become symptomatic at lower methemoglobin levels due to an already reduced oxyhemoglobin concentration.[103]

Other topical anesthetics besides benzocaine (e.g., Hurricane spray) that may induce methemoglobinemia include benzocaine/tetracaine/butamben (e.g., Cetacaine) and lidocaine plus prilocaine (e.g., EMLA cream).[104-106] Formation of methemoglobin from these products is dose-dependent. Emergency Physicians are advised to strictly follow the age-based dosing recommendations and the maximum dose recommendations.

First-line treatment of methemoglobinemia is intravenous administration of methylene blue at a dose of 1 to 2 mg/kg over 5 minutes. Administer treatment to symptomatic patients with methemoglobin levels greater than 10% to 15%. Treat all patients with methemoglobin levels greater than 25% regardless of symptoms.[107]

Injection of local anesthetic agents can cause complications. These agents have not been shown to increase the incidence of wound infections. Do not inject local anesthetic agents into a joint prior to obtaining synovial fluid. They can result in false-negative culture results, false-negative crystal analysis due to dissolving crystals, and false-positive crystal analysis due to anesthetic crystal formation. Needle punctures of arteries, nerves, and veins are usually a temporary inconvenience with no long-lasting consequences. Intraneural injection can result in temporary or permanent nerve injury. **Never inject local anesthetic agents if the patient experiences paresthesias indicating intraneural needle placement.** Withdraw the needle 1 to 2 mm and allow the paresthesias to resolve before infiltrating with the local anesthetic solution. Never redirect the needle when more than the tip is subcutaneous to prevent needle breakage.

SUMMARY

Local anesthetic agents have become an indispensable tool in the practice of Emergency Medicine. Emergency Physicians often rely on local anesthetic agents to relieve patient discomfort and provide wound care. Infiltrative anesthesia represents one of the most prevalent uses for these agents. It is relatively quick, easy to perform, well tolerated, and has a large margin of safety. Complications can and do arise. The Emergency Physician must be ready to recognize the warning signs of local anesthetic toxicity and intervene appropriately. In-depth knowledge of the appropriate administration of these common agents plays an important role in the practice of medicine. Emergency Physicians have sought to relive the pain and suffering of our patients. These agents allow us to come closer to attaining that goal.

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154 Topical and Noninvasive Anesthesia

Sonali Gandhi and Michael Schindlbeck

INTRODUCTION

Invasive procedures can cause significant anxiety in patients both young and old, much of which is related to fear of the associated pain. Noninvasive anesthesia has been shown to decrease pain and anxiety surrounding procedures (e.g., lumbar puncture, intravenous access, and laceration repair).[1,2] This chapter will discuss topical anesthetic agents, noninvasive anesthetic agents, and the range of techniques that are available for delivery of these agents. Not discussed are many of the nonpharmacologic techniques to decrease pain and anxiety except oral sucrose.[3-10]

ANATOMY AND PATHOPHYSIOLOGY

Mechanical, thermal, and chemical stimuli are detected by nerve endings called nociceptors. These pain receptors are in the dermis and the epidermis of the skin (**Figure 154-1**). Nociceptors are free nerve endings that have their cell bodies outside the spinal column in the dorsal root ganglia. The intact stratum corneum, the outer layer of cornified epithelial cells of the skin, is an effective barrier to the outside environment. Local anesthetics must traverse the stratum corneum to be delivered to the terminals of cutaneous sensory nerve fibers. The local anesthesia can block or reduce the action potential of the nerve fibers and prevent depolarization of the nerve ending. The three methods by which the stratum corneum can be bypassed to deliver a local anesthetic are direct injection, passive diffusion, and needle-free drug delivery strategies. This chapter will focus on the techniques for enhancing passive diffusion and needle-free administration of local anesthetic agents, collectively known as topical anesthesia.

INDICATIONS

Topical anesthesia is commonly utilized in the Emergency Department. The patient experiencing pain from an injury (e.g., a laceration, abrasion, or contusion) can benefit from the application of topical anesthesia. The use of topical anesthesia is beneficial in the patient who will undergo a painful procedure (e.g., venipuncture, lumbar puncture, abscess incision and drainage, or laceration repair).[11,12]

Topical anesthetic agents offer several potential advantages over local infiltration anesthesia. They are less painful to apply, overcome aversions toward needles, do not distort the wound margins, decrease the infection rate, are easy to use, and decrease the need for sedation.[2] The major limitations of topical anesthesia have been the extended time required to achieve anesthesia (**Table 154-1**) and the lack of sufficient analgesia that often requires supplemental infiltration anesthesia. These constraints have limited the use of topical

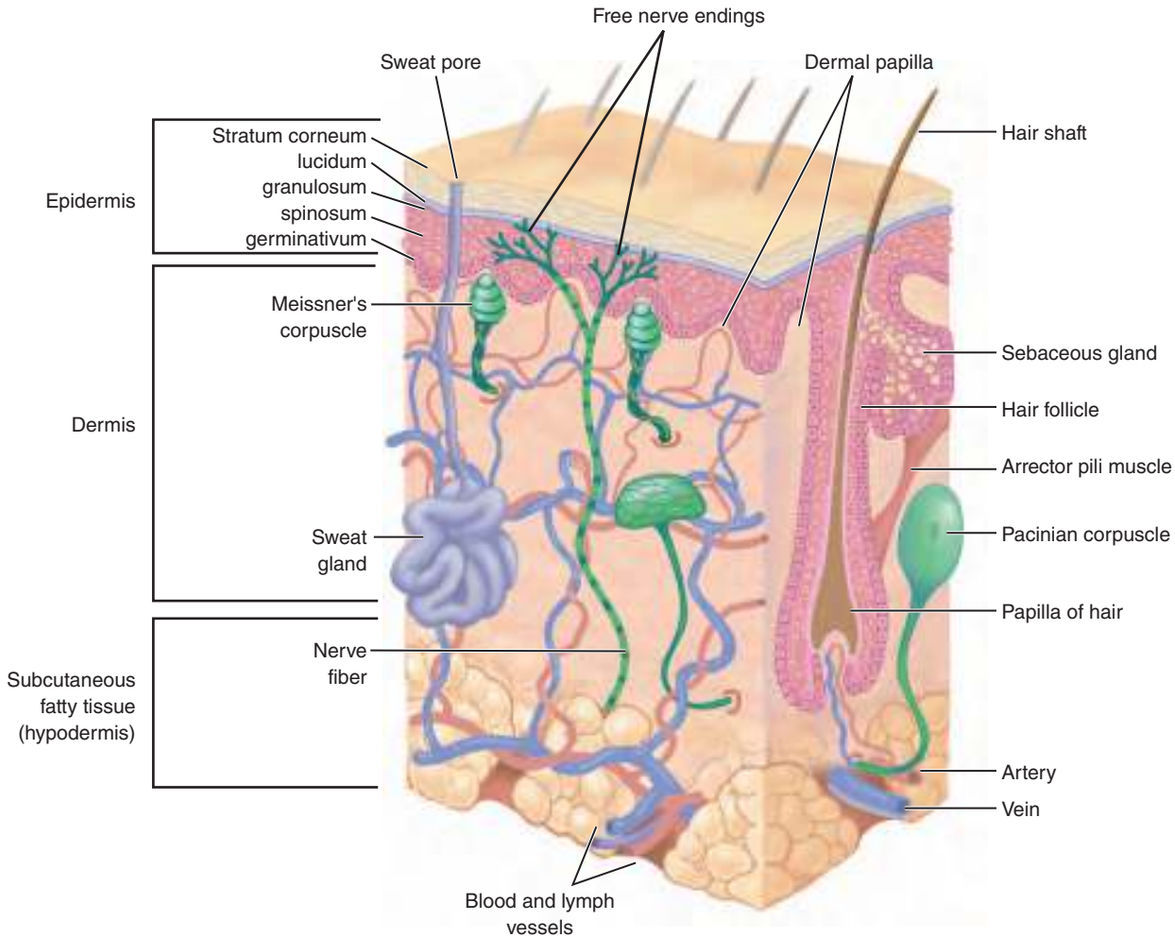


FIGURE 154-1. Cross-section of skin showing cornified stratum corneum surface with underlying sensory nerve endings.

anesthesia in the Emergency Department. Several new agents and delivery techniques have addressed these limitations with some success, offering the Emergency Physician more options for providing anesthesia.

CONTRAINDICATIONS

There are very few contraindications to the use of topical anesthetics. The topical anesthetic agent is typically not systemically absorbed to any significant degree. They can cause local adverse reactions and should not be used in patients who are allergic to the medication or its components (e.g., preservatives). **Use care when applying topical anesthetics to mucous membranes. Absorption from mucous membranes is typically more rapid and efficient than through skin, and more substantial systemic absorption can occur.** A relative contraindication to using topical anesthetic agents are patients taking class I antiarrhythmics (e.g., mexiletine and tocainide) as they can produce additive and possibly toxic effects. Many of the topical anesthetics are contraindicated or require special ophthalmic formulations for use in the eye. **Special care and attention to dosing is imperative in neonatal patients to avoid toxicity.** Specific contraindications and concerns for each topical agent and application technique are addressed later in this chapter and Chapter 153. Use caution if the patient is taking medications that may cause methemoglobinemia (**Table 154-2**).[13]

Do not use lidocaine products on the oral mucosa of patients less than 3.5 years of age for relief of teething and stomatitis pain.[14-16] Young children can swallow these preparations instead of swishing and spitting them out. Swallowing can cause serious adverse reactions, seizures, and death.

EQUIPMENT

GENERAL SUPPLIES

- Alcohol swabs
- Povidone iodine or chlorhexidine solution
- Gauze squares, 2×2 and 4×4
- Gloves

TOPICAL CREAM, GEL, OR LIQUID

- TAC (i.e., 0.5% tetracaine, 1:2000 epinephrine, and 11.8% cocaine)
- LET (i.e., 0.5% tetracaine, 1:2000 epinephrine, and 4% lidocaine)
- EMLA (i.e., 2.5% lidocaine and 2.5% prilocaine)
- LMX-4 or LMX-5 (i.e., 4% or 5% liposomal lidocaine)
- Topicaine (i.e., 4% lidocaine gel)

IONTOPHORESIS

- 2% lidocaine hydrochloride containing 1:100,000 epinephrine
- Device to administer iontophoresis (**Figure 154-2**)
- Iontophoresis system

TABLE 154-1 Characteristics of Topical Anesthetic Agents

Medication or method	Time to anesthesia (minutes)	Duration of anesthesia (minutes)	Dose	Side effects
Amethocaine	30–45	240–360 after removal	1 tube covers up to 30 cm^2 < 1 month: do not use 1 month–5 years: 1 tube 5 years of age: 2 tubes Adults: 7 tubes	Local skin reaction
EMLA (eutectic mixture of lidocaine and prilocaine)	60 superficial dermal, 120 deep dermal	60 after removal (shorter in more vascular areas)	0–3 months or < 5 kg: 1 gm/10 cm^2 body surface area (BSA) for 1 h 3–12 months and > 5 kg: 2 gm/20 cm^2 BSA for 4 h 1–6 years and > 10 kg: 10 gm/100 cm^2 BSA for 4 h 7–12 years and > 20 kg: 20 gm/200 cm^2 BSA for 4 h	Local skin reactions
Heat-enhanced diffusion patches (Synera or Rapydan)	10–30	100 after removal	1 patch for > 3 years of age	Local skin reactions
Iontophoresis with lidocaine with epinephrine	10–20	30–45	Calculate dosage	Burns, local skin reactions
Jet lidocaine injection	1–3	45	Inject area, calculate dosage	Contusions, minor bleeding, cellulitis, administration pain
Laser-assisted transdermal	5	30–45	Calculate dosage	Local skin reactions, administration pain
LET (lidocaine, epinephrine, and tetracaine)	15–30	45–60	Apply 1–3 mL to open wound	None
Lidocaine gel 4% or 5% (Topicaine)	20–60, mean 30	20–60	Apply 1/8 inch thick layer in 2 year olds and older	Local skin reactions
Liposomal lidocaine (LMX-4 and LMX-5)	30	60 after removal	1 gm/10 cm^2 BSA. Do not apply > 1 gm in < 1 year of age. Do not leave on > 1 h in < 3 months of age, > 4 h in 3–12 months of age, > 5 h in 12 months of age and older. Do not use in those < 1 month of age	Skin erythema
Peels (S-Caine and Pliaglis)	20–60	600+	Only use in adults. Apply a thin layer	Local skin reactions
Powdered lidocaine injector	1–3	10–20	Inject once to intact skin	Contusions, minor bleeding, cellulitis
TAC (tetracaine, adrenaline, and cocaine)	20–30	45–60	Apply 1–3 mL to open wound. Avoid mucous membranes	Seizures, cardiac arrest (rare)
Ultrasound-assisted delivery	5	30–45	Calculate dosage	Local skin reactions
Vapocoolant sprays	0.1–0.2	1	Use in 3 years or older	Frostbite, skin erythema

ULTRASOUND-ASSISTED LOCAL ANESTHETIC DELIVERY

- Ultrasound device (**Figure 154-3**)
- Topical local anesthetic agent

POWDERED ANESTHETICS

- Needle-free powder lidocaine delivery system (**Figure 154-4**)

LIDOCAINE NEEDLE-FREE INJECTION

- Needle-free injection system, prefilled or fillable (**Figure 154-5**)
- 1% or 2% lidocaine

TABLE 154-2 Some of the Medications that Can React with Topical Anesthetics and Cause Methemoglobinemia

Acetaminophen	Nitrites
Aniline dyes	Nitrofurantoin
Benzocaine	Nitroglycerine
Chloroquine	Phenobarbital
Cyclophosphamide	Phenytion
Dapsone	Prilocaine
Flutamide	Primaquine
Lidocaine	Procaine
Mesalamine	Sodium nitroprusside
Naphthalene	Sulfonamide
Nitrates	Trimethoprim-sulfamethoxazole

HEAT-ENHANCED DIFFUSION

- Lidocaine-tetracaine thermal patch (**Figure 154-6**)

LASER-ASSISTED TRANSDERMAL PASSAGE

- Cutaneous resurfacing laser (**Figure 154-7**)
- Topical anesthetic agent

TOPICAL VAPOCOOLANT SPRAYS

- Topical vapocoolant spray (**Figure 154-8**)

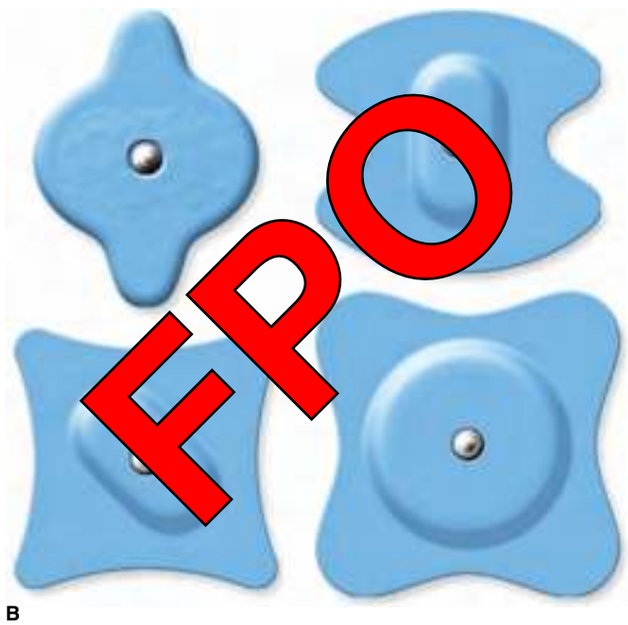
PATIENT PREPARATION

Discuss the procedure and its risks, benefits, complications, and alternatives with the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Use precautions to prevent further injury or pain to the patient. Cleanse the skin of any dirt and debris. This will aid in providing appropriate anesthesia to the affected area.[17] Apply povidone iodine or chlorhexidine solution to the skin and allow it to dry. Consider administering a supplemental anxiolytic or procedural sedation (Chapter 159).

Young children do not talk and are afraid of any procedures performed. Their actions demonstrate anxiety and pain (**Table 154-3**). There are several methods described to characterize the child's pain.[18]

A



B

FIGURE 154-2. An example of an iontophoresis unit. ***A.*** The Dupel iontophoresis unit. ***B.*** The patches. (Photo courtesy of EMPI, Clear Lake, SD.)

PASSIVE DIFFUSION OF LOCAL ANESTHETIC AGENTS

Topical local anesthetic agents are often applied in the form of a gel or cream.[19,20] There are numerous combinations of anesthetic agents to use topically on wounds. TAC (i.e., tetracaine, adrenaline, and cocaine), LET (i.e., lidocaine, epinephrine, and tetracaine), and EMLA (i.e., eutectic mixture of lidocaine and prilocaine) are three commonly applied combinations (**Table 154-1**). Other topical local anesthetic agents include lidocaine gel and liposomal lidocaine.

FIGURE 154-3. The Sontra Sonoprep ultrasound-assisted anesthetic delivery unit. (Photo courtesy of Sontra Medical Corp., Franklin, MN.)

TAC

Many Emergency Departments use TAC for topical anesthesia. It is a combination of 0.5% tetracaine, 1:2000 or 0.05% adrenalin (i.e., epinephrine), and 11.8% cocaine. TAC provides topical anesthesia and vasoconstriction. The vasoconstrictor action reduces blood flow in the affected area and systemic absorption of the drug. The degree of anesthesia achieved with TAC is comparable to that of local lidocaine infiltration for wounds on the face and scalp. TAC is superior to lidocaine infiltration in pediatric patients, which research attributed to patient compliance.[21,22] The effects are less profound for wounds on the trunk and extremities.

The cocaine component in TAC is a powerful and effective local anesthetic agent and a vasoconstrictive agent. **TAC is a controlled substance.** This makes its storage, utilization, and monitoring subject to stringent regulation and documentation requirements. This may limit the availability and use of TAC in some institutions.

Dose recommendations generally call for 5 mL of TAC for lacerations smaller than 3 cm in length and 10 mL for lacerations greater than 3 cm in length.[23] Apply the chosen volume of TAC liquid or gel onto a 2×2 gauze square or a cotton ball. Invert the gauze square or cotton ball to apply TAC within the wound margins. Allow TAC to remain for up to 15 minutes or until visible skin blanching occurs. **Blanching indicates the presence of vasoconstriction and adequate analgesia.** The gauze pad can be held in place by the patient's gloved hand, the patient representative's gloved hand, or with tape.

TAC is not free of complications. **Always wear gloves when handling TAC to prevent percutaneous absorption.** Do not apply TAC to tissues with an end-arteriole supply due to the vasoconstrictive effects of cocaine. This includes the fingers, toes, nose, ear, and penis. There is the possibility of vasoconstrictive ischemic injury to these tissues. The systemic absorption of cocaine from TAC

FIGURE 154-4. Zingo needle-free powdered lidocaine delivery system. (Photo courtesy of 7T Pharma LLC.)

FIGURE 154-5. Needle-free injectors. ***A.*** The INJEX. (Photo courtesy of INJEX Ltd. UK, Herefordshire, United Kingdom.) ***B.*** The J-Tip. (Photo courtesy of National Medical Products, Orange, CA.) ***C.*** The PharmaJet. (Photo courtesy of PharmaJet, Golden, CO.) ***D.*** The Bioject ZetaJet. (Photo courtesy of ZetaJet, Malvern, Australia.)

administration has been implicated in rare episodes of respiratory arrest, seizures, and death.[24,25]

LET

LET is an alternative to TAC.[26] It is a combination of 4% lidocaine, 0.1% (1:1000) adrenaline (i.e., epinephrine), and 0.5% tetracaine.[27] It is considered safer, similarly effective, more practical, and more

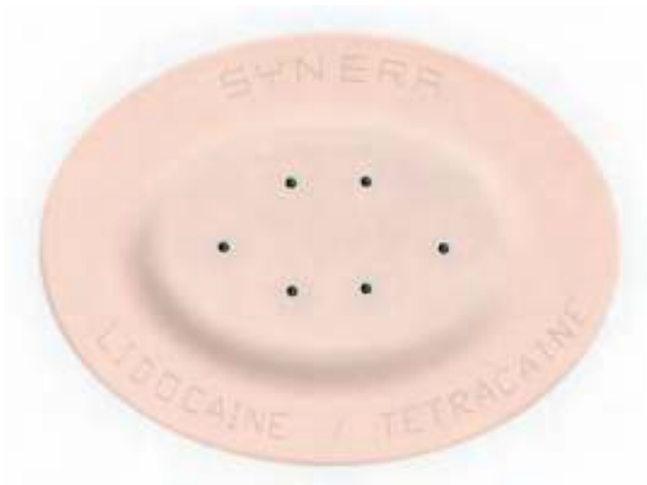


FIGURE 154-6. The Synera lidocaine-tetracaine patch. (Photo courtesy of Galen US Inc., Souderton, PA.)

cost effective to use than TAC. No statistical significance was found between anesthesia for pediatric and adult patients using LET compared to TAC on the face and scalp for uncomplicated lacerations.[27,28] **LET does not contain a controlled substance and has no potential for abuse.**[26,27] The resultant security and documentation requirements are much less complicated than those required for TAC.

Apply liquid LET by dripping it into the wound or taping a LET-soaked cotton ball or 2×2 gauze square over the wound. Allow it to dwell 20 to 30 minutes or until the wound edges blanch. The addition of methylcellulose to liquid LET makes a gel that is more adherent and can be painted on wounds. The gel form will not drip or run. Common guidelines suggest it should not be placed on the digits, ear, nose, penis, or other areas that are supplied by end arteries

FIGURE 154-7. The Norwood Abbey laser-assisted resurfacing laser. (Photo courtesy of Norwood Abbey LTD., Frankston, Australia.)

A B

FIGURE 154-8. Gebauer vapocoolant spray. ***A.*** Spray bottle. ***B.*** Aerosolized can.

due to the strong vasoconstrictive effect and risk of ischemia. These guidelines are currently being challenged.[29] It can be used for lacerations on all other locations except those grossly contaminated.[20]

EMLA CREAM

The use of EMLA (i.e., eutectic mixture of local anesthetics) cream has gained significant popularity, particularly in pediatric patients. It is an emulsion of 2.5% lidocaine and 2.5% prilocaine in a 1:1 ratio by weight. Each gram of EMLA contains 25 mg of lidocaine and 25 mg of prilocaine. It is nonsterile and preservative free. It is also commercially available in prepackaged transdermal disks. **Apply EMLA only onto intact skin and not into open wounds.** It is not licensed for use in injured or violated skin.

TABLE 154-3 Pain Signs and Symptoms in Preverbal Children

Back arching	Increase in respiratory rate
Changes in facial expression	Limb thrashing
Finger clenching	Oxygen desaturation
Flushing	Pallor
Head banging	Poor feeding
Increase in blood pressure	Sleep disturbances
Increase in heart rate	Sweating
Increase in muscle tone	Writhing

EMLA is indicated prior to venipuncture, arterial punctures, access of indwelling ports and reservoirs, lumbar puncture, minor skin procedures, tympanic membrane and external auditory canal anesthesia, or regional nerve blockade. Apply EMLA cream to intact skin and cover it with an occlusive dressing (e.g., Tegaderm) or apply a transdermal disk. Allow at least 1 hour for the EMLA to take effect. Analgesia is usually satisfactory after 1 hour, peaks at 2 hours, and persists for about 1 hour after it is removed from the skin. The prolonged time required for anesthesia to take effect limits its practical use in the Emergency Department. EMLA should not be applied to infants less than 3 months of age due to the risk of methemoglobinemia.[30] The contraindications to using EMLA include allergies to lidocaine or tetracaine and a history of methemoglobinemia.

AMETHOCAINE CREAM

Ametop is another name for the commercial form of amethocaine gel (i.e., 4% amethocaine). It is an alternative to EMLA and is indicated for intact skin. Apply the cream to intact skin and cover with an occlusive dressing. Amethocaine can be used prior to performing lumbar puncture, venous cannulation, minor skin procedures, external auditory canal anesthesia, or regional nerve blockade. It is faster in onset and has greater efficacy than EMLA. Ametop provides adequate anesthesia at approximately 30 to 45 minutes.[31,32] It works for the pain of intravenous cannulation when compared to

EMLA.[33] Amethocaine decreases the pain of intravenous cannulation and is comparable to 4% liposomal lidocaine.[34]

LIPOSOMAL AGENTS

Liposomal lidocaine (e.g., LMX-4 or LMX-5) is a 4% or 5% lidocaine cream that is encapsulated in a liposomal matrix. The encapsulation of lidocaine keeps it from being rapidly metabolized. The lipid content of the liposomes allows for better drug penetration of the stratum corneum. The LMX-4 cream has several advantages over other topical anesthetic agents. These include a more rapid onset of action and no requirement for an occlusive dressing. The most frequent adverse reaction is local erythema. No serious side effects have been reported with the use of LMX-4. It is recommended that it be applied for 30 to 60 minutes to an area less than 100 cm^2 in patients who weigh less than 20 kg.[35] Apply the LMX thick and covered with an occlusive dressing. Do not flatten the LMX with the occlusive dressing as it works best when applied thick. Intravenous cannulation after the application of LMX-4 showed improved success rates on the first attempt among children when compared to a placebo.[36] A dwell time of 15 minutes was not effective and may be too short.[37] The recommended time is 30 to 60 minutes. LMX-4 is as effective as EMLA.[38] Buffered lidocaine injection decreased the pain associated with intravenous catheter insertion greater than lidocaine cream.[2]

TOPICAINE

Topicaine is a 4% lidocaine gel that is used for procedures in the Emergency Department including placement of Foley catheters and nasogastric tubes. It is readily available and easy to use. Patients can develop a contact dermatitis, particularly if they have an allergy to amide-type local anesthetic agents. Apply Topicaine for 30 minutes prior to a procedure for maximum efficacy.

ACTIVE NEEDLE-FREE LOCAL ANESTHETIC DELIVERY

An alternative to either the injection of local anesthetic solution or the use of topical creams and gels are needle-free drug delivery systems. These techniques utilize various strategies designed to expedite the delivery of the local anesthetic agent across the stratum corneum. This can reduce wait times to achieve anesthesia from 30 to 60 minutes to within 10 minutes with the use of additional devices (e.g., ultrasound or iontophoresis).[39] The clinical utility of these devices is offset by disadvantages including cost of the devices, the bulky equipment, and the limited surface area that can be anesthetized.[40]

ULTRASOUND-ASSISTED LOCAL ANESTHETIC DELIVERY

Ultrasound has been used to accelerate the delivery of topical anesthetic agents (**Figure 154-3**). Ultrasound disrupts the layers of the stratum corneum, forming temporary pores that facilitate the distribution of topical anesthetic agents below the epidermis.[41] Focal ultrasound followed by a 5 minute application of 4% liposomal lidocaine provided more effective anesthesia when compared to a placebo or standard care.[42,43] Ultrasound has been shown to reduce the time to achieve anesthesia with EMLA cream from 60 minutes to 5 minutes.[44] The use of ultrasound prior to a 5 minute application of liposomal lidocaine produced the same amount of pain relief as the typical 30 minute application of liposomal lidocaine.[45] Ultrasound is effective for intravenous catheter placement and phlebotomy.[46] It is not associated with the adverse effects of iontophoresis.[46]

IONTOPHORESIS

Iontophoresis is a process by which direct electrical current facilitates dermal penetration of positively charged lidocaine molecules when placed under a positive electrode (**Figure 154-2**). This allows for enhanced absorption of local anesthesia.[47] The dose is calculated by multiplying the duration of delivery with the current used to deliver the local anesthetic agent. Studies evaluating this method typically use 20 to 30 mA and 2% lidocaine hydrochloride with 1:100,000 epinephrine.[48,49] It takes approximately 10 minutes to obtain adequate analgesia for intravenous catheter placement.[50] The side effects of iontophoresis include skin blanching, erythema, tingling, itching, and burning sensations.[40] Burns have been reported to rarely occur during iontophoresis with an incidence of 1 per 15,000 to 20,000 treatments.[51] Iontophoresis has been successfully used prior to intravenous catheter placement and lumbar puncture.[52]

POWDERED ANESTHETICS

Another alternative to the injection of lidocaine solution is the transepithelial injection of powdered lidocaine using a specialized delivery system.[53] The delivery system is a prefilled, disposable, single-use device that is designed to produce rapid analgesia for venipuncture and intravenous catheter placement. The system is applied 3 minutes before venipuncture or intravenous cannulation. The device works by releasing pressurized helium, which in turn ruptures a cassette of lidocaine powder. The particles of lidocaine powder are accelerated to a velocity that is sufficient to penetrate the stratum corneum and become deposited in the subepithelial layer. The onset of anesthesia is approximately 1 to 3 minutes after the injection.[54] This delivery system results in a significant reduction in pain.[55] The potential adverse effects of powdered lidocaine administration include local skin contusion, application site bleeding, and a subsequent cellulitis.

JET LIDOCAINE

Another form of needle-free lidocaine injection is commonly referred to as jet lidocaine. The device uses pressurized CO_2 to inject up to 0.5 mL of aqueous lidocaine into subcutaneous tissue (**Figures 154-5 and 154-9**). The lidocaine is drawn up into the reservoir and the device is then held firmly against the skin where the proposed procedure is to occur. The CO_2 cartridge is activated, forcing the micro-aerosolized lidocaine through the stratum corneum and depositing it into the subcutaneous tissues. The local anesthetic travels approximately 5 mm through the skin. The onset of anesthesia is approximately 1 to 3 minutes after the injection. Approximately 20% of patients in a randomized trial experienced pain from administration of the system.[56] Other complications include device failure 10% of the time and minor local bleeding. It can be used for arterial blood gases, dental anesthesia, facial lacerations, intravenous cannulation, and lumbar punctures.[57-59] Jet lidocaine was found to provide more effective anesthesia in studies comparing jet

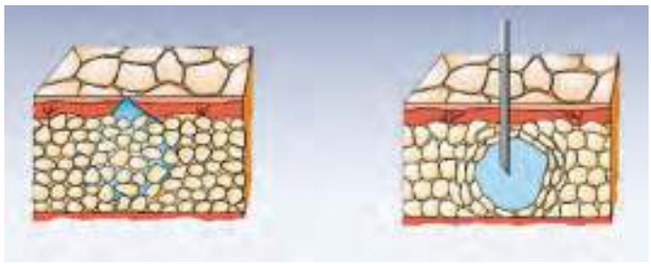


FIGURE 154-9. Comparison of anesthetic delivery with a needle-free device (*left*) versus a needle (*right*). (Photo courtesy of National Medical Products, Orange, CA.)

lidocaine to EMLA or liposomal lidocaine.[61,62] Jet lidocaine has been found to be more effective than placebo.[63,64] Jet lidocaine reduced venipuncture pain in pediatric patients when compared to vapocoolant sprays and placebo.[59] It reduced pain in infants requiring a lumbar puncture when compared to placebo.[60] One study found jet lidocaine to be no more effective at providing local anesthesia than jet saline.[65] This suggests that the jet injection itself may play a role in providing anesthesia. The J-tip can cause local anesthetic toxicity.[66-68]

HEAT-ENHANCED DIFFUSION

The application of heat improves the dermal absorption of topical anesthetics.[69] The lidocaine-tetracaine topical patch consists of 70 mg of each local anesthesia agent, combined with an air-activated heating system incorporated into an adhesive patch (**Figure 154-6**). It is specifically designed to warm the skin and accelerate the absorption of the local anesthetic agents. The heating component of the patch is automatically activated when the patch is removed from its packaging. It can be used safely in children over the age of 5 months. The only contraindications to using this patch are neonates, allergies to lidocaine, allergies to tetracaine, and a history of methemoglobinemia. The lidocaine-tetracaine patch can be used for minor invasive procedures on intact skin (e.g., lumbar puncture, intravenous catheter placement, and arterial puncture).[69] The lidocaine-tetracaine patch can provide effective anesthesia after 10 minutes. It was more effective than EMLA or a placebo at all application times shorter than 60 minutes.[70,71] The patches are just as effective as injection anesthesia for abscess incision and drainage.[72] Common side effects include skin erythema, blanching, and edema.

LASER-ASSISTED TRANSDERMAL PASSAGE

Another technique to break down the stratum corneum skin barrier involves the use of laser.[73] A cutaneous resurfacing laser is used to ablate the outer layer of skin in an area about 6 mm in diameter (**Figure 154-7**). Topical anesthetic is then applied to this area and rapidly penetrates through the ablated skin to the subcutaneous pain receptors. The ablation procedure is typically painless. Anesthesia occurs approximately 5 minutes after the local anesthetic application. The amount of energy used for the ablation varies but typically is in the range of 2.0 to 3.5 J/cm^2. Side effects may include mild pain, erythema, and itching at the ablation site. The patient may experience temporary hyperpigmentation or hypopigmentation at the treatment site. The biggest drawback to this technique is the cost of the handheld laser.

VAPOCOOLANT SPRAYS

Vapocoolant sprays, also known as vapocoolants and refrigerant sprays, are an alternative to topical local anesthetic agents. Vapocoolant sprays are a good choice for patients requiring brief procedures or with allergies to the medications used in topical anesthetics. Vapocoolant sprays work immediately, are easy to use, are cost effective, and may be repeatedly applied without the risk of methemoglobinemia or systemic toxicity. Vapocoolant sprays are sterile liquids that vaporize upon contact with the skin (**Figure 154-8**). They cool the skin surface as the liquid vaporizes and provide brief topical anesthesia lasting approximately 30 seconds. Commonly used vapocoolants include ethyl chloride, fluorohydrocarbons, and alkane mixtures. Do not use vapocoolants in patients with a known hypersensitivity or allergy to the vapocoolant or its propellant. The topical spray may have antibacterial activity.[74]

Vapocoolants are commercially available in containers with a tip that directs a precise stream of liquid to the desired area (**Figure 154-8**). The vaporization of the liquid lowers the skin temperature in the area of contact to approximately –20°C (–4°F) as it vaporizes. The skin becomes temporarily cold then frozen. Vapocoolant sprays are convenient, effective, "needle-less," and provide immediate anesthesia.[75] Their use is limited by a very brief duration of action of approximately 30 seconds. **These sprays only provide superficial anesthesia and do not traverse the stratum corneum.** Vapocoolant sprays can be used prior to accessing indwelling ports and reservoirs, arterial puncture, venipuncture, intravenous catheter placement, intramuscular injections, local anesthetic injection, lumbar puncture, and minor skin procedures.[76-80]

Using the vapocoolant spray is very simple. Assemble and gather all the equipment required for the procedure to be performed. Clean and prep the skin. Apply sterile drapes if applicable. Hold the inverted container of refrigerant spray 10 to 15 cm above the skin surface. Spray the liquid onto the skin until a white frost appears and the skin turns white. This usually takes 7 to 12 seconds. Immediately perform the procedure to ensure an adequate anesthetic effect. The area can be surrounded with lubricant to limit skin exposure under the covered areas.

Avoid spraying the liquid onto the skin for prolonged periods as it may result in frostbite, skin hypopigmentation, and atrophic scarring.[81,82] Do not use vapocoolant sprays on mucous membranes. The vapocoolants are significantly absorbed through mucous membranes and can result in adverse reactions and toxicity. These agents are highly volatile and must be used in well-ventilated areas.[81] Neurologic symptoms from inhalation include headache, nausea, vomiting, dizziness, incoordination, disorientation, central nervous system depression, respiratory depression, cardiac arrest, and dysrhythmias from myocardial sensitization to catecholamines.[81] Never spray around open flames or an electrocautery unit as some vapocoolant sprays are highly flammable. Do not use vapocoolant spray on open wounds as it may delay wound healing. Patients were happier with the use of vapocoolant than ice.[83] The application of ice packs work, but takes longer to achieve anesthesia.

MUCOUS MEMBRANE ANESTHESIA

Mucous membranes warrant a special note as the systemic absorption from intact mucous membranes is much more rapid and effective than absorption through the skin and results in higher blood levels of the anesthetic agent. Mucous membranes include the nose, mouth, throat, and genitourinary region. Numerous agents may be used to provide anesthesia to mucous membranes (e.g., benzocaine, cocaine, lidocaine, and tetracaine). These local anesthetic agents produce only superficial anesthesia like vapocoolant sprays. They do not provide any pain relief that originates submucosally or deeper. **The total dose applied should be reduced to one-third to one-half the dose used for infiltration to reduce toxicity. Use these agents cautiously on the oral mucosa as they can suppress the gag reflex and increase the risk for aspiration.**

Benzocaine is a commonly used mucous membrane anesthetic for the nose, mouth, and throat. It is available in spray, liquid, and gel form in a concentration of 14% to 20% (e.g., Cetacaine, Hurricaine, Americaine). It is nontoxic when applied to intact mucous membranes due to its poor water solubility. It provides brief analgesia and is often used for tube placement (e.g., nasogastric and orotracheal). **Overuse of benzocaine has the potential to produce methemoglobinemia.[84-86]**

Cocaine is supplied in solution at concentrations of 4% and 10%. It is an extremely effective mucous membrane anesthetic with significant vasoconstrictive properties which can be helpful in instances such as epistaxis. Systemic absorption is enhanced when it is applied to inflamed mucous membranes. The maximum

dose is 3 mg/kg. This dose still has the potential to result in toxicity and serious complications. **Do not use cocaine in patients with hypertension, cardiomyopathy, or known or suspected coronary artery disease, or patients sensitive to exogenous catecholamines. Cocaine is further limited by its risk of potential abuse.**

Lidocaine is available in jelly and liquid form with a concentration of 2% to 10%. The 2% and 4% viscous solutions are often used in the Emergency Department. Instruct the patient to swish the solution in their mouth for 30 to 60 seconds and then spit it out. It can result in significant systemic absorption if swallowed. The maximal dose is 300 mg in adults (15 mL of 2% or 7.5 mL of 4%) and 3 mg/kg in children. **Use extreme caution when sending patients home with viscous lidocaine for intraoral use.** Frequent use and swallowing can result in elevated blood levels of the parent drug and its metabolites, both of which can result in potential adverse and toxic effects.[87,88]

Tetracaine is available in liquid and aerosol form with a concentration of 0.25% to 1.0%. It has significant cardiotoxic effects. Therefore, it is not often used for mucous membrane anesthesia. Tetracaine should not be used for mucous membrane anesthesia in the Emergency Department.

MISCELLANEOUS AGENTS

Several alternative topical anesthetic techniques have been used to provide analgesia. These techniques all have the potential to decrease the pain due to procedures. More information and trials are required before these can be used in the Emergency Department.

S-Caine Peel (Zars Pharma) has been developed as a local anesthetic peel. It is composed of 7% lidocaine and 7% tetracaine in an inert base. It is applied to an area for 20 to 30 minutes. It dries into a flexible membrane that can be peeled off after the application period. This agent is easily applied to nonflat body surfaces. Its primary use has been for dermatologic filler procedures, facial laser resurfacing, laser vein treatments, laser tattoo removal, and pulse dye laser treatment.[89-91] It may be used on intact skin for nonemergent arterial blood gases, intravenous cannulation, and lumbar puncture in the future.

Acupressure has been used for hundreds of years to relieve pain. It has been recently used in acute painful conditions.[92] It has been successful in reducing or eliminating pain. Most Emergency Physicians are not trained in this modality.

The ShotBlocker (Bionix, Toledo, OH) is a plastic device that is pressed against the skin just before and during an injection.[93] It has small bumps on the back that go against the patient's skin. It may be used to decrease the pain of local anesthetic injections. It follows the gait control theory of pain management. The noxious impulses of the ShotBlocker applied to the skin distract from the needle pain.

An anesthetic putty has been developed to relieve pain before laceration repair.[94] It contains 4.94% lidocaine, which is equivalent to a 4% base solution or 1% injectable lidocaine. The putty is put in the laceration and left to dwell to provide anesthesia. The putty dries as it provides anesthesia. It is removed completely in one mass and does not distort the anatomy. The application and removal are pain-free. This putty is not yet available commercially.

Oral sweet solutions, usually sucrose, have been used for several years for nonemergent procedures.[8,95-99] These include arterial blood gases, bladder catheterization, heel sticks, intravenous access, and lumbar puncture. The use of these solutions is simple, effective, and easy up to the age of 6 months. It is believed they work through distraction and the release of endogenous opioids. Apply 1 mL in each cheek or allow sucking from a pacifier just before the procedure. The only contraindications are suspected or actual necrotizing enterocolitis and postoperative abdominal surgery. One study in children 1 to 3 months of age showed sweet solutions did not decrease pain scores or heart rates but did decrease the crying time.[100] There is no real downside for trying sweet solutions to reduce pain.

ASSESSMENT

Wait long enough after the topical agent is administered to allow the onset of adequate anesthesia before the planned procedure is started. The time necessary for the onset of anesthesia is highly variable and dependent on the topical anesthetic used and the delivery technique utilized. A simple examination of the area being anesthetized with fine touch and pinprick ensures adequate anesthesia prior to the procedure. It may be necessary to apply additional anesthetic or use an alternative anesthetic technique in areas of continued sensitivity, keeping in mind the total dose applied to avoid toxicity.

AFTERCARE

Observe the patient for a minimum of 15 minutes following the use of topical anesthesia to ensure no evidence of an adverse reaction or toxicity. The patient need not wait in the Emergency Department for normal sensation to return prior to discharge.

COMPLICATIONS

The use of topical and noninvasive anesthesia occasionally causes adverse effects. There are case reports of patients developing central nervous system toxicity after the application of topical anesthetics.[101] There have been cases of methemoglobinemia reported in patients, particularly with the use of benzocaine.[84] Topical anesthetics in patients taking class I antiarrhythmics (e.g., mexiletine and tocainide) can produce additive and possibly toxic effects. EMLA has been reported to cause ulceration of the gingival mucosa and should not be used for mucosal anesthesia.[102] Many of the topical anesthetics are contraindicated or require special ophthalmic formulations for use in the eye. Special care and attention to dosing are imperative to avoid toxicity. This is especially true when using topical anesthetics in neonatal patients

SUMMARY

There are effective techniques available to decrease the pain and anxiety associated with invasive procedures. Topical and noninvasive anesthetic agents, particularly those with fast-acting properties, are excellent adjuncts for use in the Emergency Department. Topical creams, active medication delivery, or alternative techniques should be considered prior to performing a painful invasive procedure.

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155 Hematoma Blocks

Thomas P. Graham

INTRODUCTION

Distal extremity fractures are commonly seen in the Emergency Department. These fractures often require closed reduction by manipulation which can be a painful and frightening experience for the patient. Achieving adequate analgesia is important to

facilitate reduction and to minimize patient discomfort. Physicians frequently provide inadequate analgesia to patients, and particularly children, with extremity fractures.[1,2]

ANATOMY AND PATHOPHYSIOLOGY

The hematoma block is a technique to inject a local anesthetic solution into the hematoma between the fractured bone fragments. Fracture manipulation can often be undertaken painlessly or with significantly reduced pain after performing a hematoma block. Hematoma blocks of the distal forearm are considered safe in children and adults.[3-8] A hematoma block may be superior to intravenous sedation in alleviating discomfort during fracture reduction.[9] Other advantages are the relative ease of the procedure, no worries concerning aspiration, and ability to potentially manage the airway. The hematoma block avoids the side effects of sedating drugs, does not require intravenous access and cardiac monitoring, and is not associated with a prolonged recovery phase. The Emergency Physician can safely perform a hematoma block alone whereas two health care providers are needed for procedural sedation. The disadvantages of the hematoma block include the discomfort and anxiety resulting from injecting into the fracture site. There is the potential for a rare complication.

Extremity fractures that are displaced or angulated result in the formation of a hematoma between the fracture fragments. The hematoma is easily accessible with a needle. The injection of local anesthetic solution can significantly alleviate pain. Most important neurovascular structures in the upper extremity are contained in the volar soft tissue, making the dorsal or lateral approach preferred. Most important neurovascular structures in the lower extremity are contained in the proximal anterior thigh or posteriorly in the leg, making the lateral approach preferred in the proximal thigh and the anterior or lateral approach from the mid-thigh distally.

Other techniques are available to provide analgesia and anesthesia. A Bier block (Chapter 157) may lead to more effective anesthesia and require fewer repeat manipulations for the reduction of forearm fractures when compared to the hematoma block.[10-12] The Bier block is a technique with which most Emergency Physicians are not familiar and have not developed proficiency. It often requires equipment not commonly available in the Emergency Department and can be associated with adverse outcomes. Intraarticular injection of local anesthetic solution for the reduction of intraarticular fracture-dislocations has been advocated as safe and effective.[13,14] The joints of the extremities are easily entered by performing an arthrocentesis (Chapter 97). Local anesthetic solution injected intraarticularly diffuses throughout the joint cavity and exits through the fracture site to alleviate pain.

INDICATIONS

A hematoma block is indicated in adult and pediatric patients with closed fractures of the extremity that require manipulation or closed reduction.[15-18] Consider performing a hematoma block when medical resources are limited or scarce. It is an alternative when procedural sedation is impossible or impractical.[15,17,19] A hematoma block may be performed purely for analgesia when fracture manipulation is unnecessary and/or other methods of analgesia are ineffective or contraindicated.

CONTRAINDICATIONS

Hematoma blocks are contraindicated when there is a history of allergic reactions to local anesthetic agents. The procedure is contraindicated in the setting of an open fracture, cellulitis overlying the fracture site, the presence of a neurovascular deficit, or the presence of a vascular deficit. Do not perform a hematoma block if a sterile field cannot be maintained or the safety of the medical staff cannot be assured due to an uncooperative patient. Relative contraindications include patients with bleeding disorders and those taking anticoagulants. The potential additional hemorrhage from the injection of local anesthetic solution into a closed space may result in a compartment syndrome (Chapters 93 and 94).

EQUIPMENT

- Sterile gloves and gown
- Face mask and cap
- Povidone iodine or chlorhexidine solution
- Sterile drapes
- Lidocaine (i.e., 1% or 2%) or bupivacaine (i.e., 0.25% or 0.5%), without epinephrine
- Syringes, various sizes
- 22 or 23 gauge, 2 inch long needles
- Spinal needles for obese patients

OPTIONAL EQUIPMENT

- Ultrasound machine
- 7.5 to 10 MHz ultrasound transducer
- Sterile ultrasound gel
- Sterile ultrasound transducer cover

PATIENT PREPARATION

Explain the risks, benefits, complications, and alternatives to the patient and/or their representative. The technique for adult and pediatric patients is identical. An adult usually tolerates the hematoma block injection without any supplemental analgesia or sedation. Some adults and children may require supplemental nitrous oxide (Chapter 158), intravenous sedation (Chapter 159), or an intravenous anxiolytic agent to facilitate the hematoma block.

Prepare for the procedure. **The hematoma block must be performed using aseptic technique.** Cleanse the skin of any dirt and debris over the fracture site and surrounding skin. Apply povidone iodine or chlorhexidine solution onto the skin and allow it to dry. Apply sterile drapes to delineate a sterile field. Don sterile gloves, a sterile gown, a face mask, and a cap during the procedure. Use sterile technique and draw up the local anesthetic solution into a syringe armed with a 22 or 23 gauge, 2 inch long needle. Bupivacaine can be used by itself or mixed with lidocaine in a 50-50 ratio if postprocedure analgesia is desired. A longer spinal needle may be required to reach the fracture in the obese patient. Fill the syringe to a maximum of 10 mL of local anesthetic agent to be injected. Larger volumes maybe required in adults and with larger fractures. Always be aware of the maximum safe dose of lidocaine or bupivacaine (Chapter 153).

TECHNIQUES

LANDMARK (BLIND) HEMATOMA BLOCK

Follow strict sterile technique throughout the procedure. Inform the patient of the early signs of local anesthetic toxicity. These include circumoral and tongue numbness, dizziness, lightheadedness, mental status decline, tinnitus, and visual disturbances. Instruct the patient to inform you immediately if they experience any of these symptoms.

FIGURE 155-1. Schematic illustration of the hematoma block.

Place a wheal of 1% lidocaine subcutaneously over the fracture site. Allow 1 to 2 minutes for the anesthetic to take effect. Slowly insert and advance the 23 gauge needle attached to the local anesthetic solution in the syringe. Insert the needle through the skin wheal and aimed at the fracture site. Continue to slowly advance the needle toward and into the expected location of the gap between the fracture fragments (**Figures 155-1 and 155-2**). **Aspirate with the syringe to ensure there is no free flow of blood. This indicates the tip of the needle is within a blood vessel. Do not inject the local anesthetic solution.**[20,21] A flash of blood indicates entry of the tip of the needle into the hematoma. Withdraw the needle and redirect it to enter the hematoma if the needle strikes bone or if no flash of blood is returned. Slowly inject the local anesthetic solution into the hematoma. Withdraw the needle. Apply a bandage to the skin puncture site.

Some will reposition the needle to different areas within the hematoma and inject small amounts of the local anesthetic solution into each area (**Figure 155-2*B***). This technique distributes the local anesthetic solution to increase the efficacy of the hematoma block. Injection into multiple areas also minimizes the risk of intravascular injection of the entire dose of local anesthetic solution.

HEMATOMA BLOCK FOR INTRAARTICULAR FRACTURE-DISLOCATIONS

The procedure is identical to that described above except for the anatomic landmarks and the volume of local anesthetic solution injected. Refer to Chapter 97 for the complete details of an arthrocentesis.

ULTRASOUND-GUIDED HEMATOMA BLOCK

The use of ultrasound (US) can facilitate the injection into the correct site.[12-27] Identify the fracture site and hematoma using US. Place the US transducer on the patient's skin above the fracture site. Turn the US transducer so its long axis is aligned with the long axis of the bone. Identify the fracture site and hematoma. Rotate the US transducer 90° to visualize the short axis or cross-sectional view of the fracture site and hematoma.

Clean, prep, and sterilely drape the patient as described previously. Inject local anesthetic solution subcutaneously over the fracture site. Prepare the US transducer. Apply US gel to the footprint of the US transducer. Apply a sterile transducer cover or a sterile glove over the US transducer. Squeeze any air out of the space between the US transducer and the cover. Apply sterile US gel on the transducer cover.

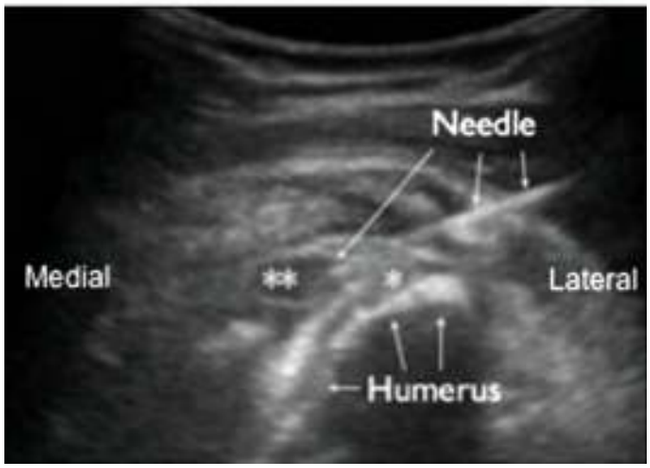
Grasp the sterile US transducer with the nondominant hand. Align the long axis of the US transducer along the long axis of the bone and approximately 8 to 10 mm proximal to the fracture site. Aim the syringe containing the local anesthetic solution downward and in the direction of the fracture site. Slowly insert and advance the needle through the skin under US guidance. Advance the needle

A

B

FIGURE 155-2. An ankle fracture. ***A.*** The radiograph. ***B.*** The hematoma block. (Photos used with permission from reference 19.)

A



B

FIGURE 155-3. A hematoma block using US. ***A.*** Positioning of the transducer and needle. ***B.*** US view of the hematoma block. The hematoma (*) and the infiltration of local anesthetic (**) are seen. (Photo used with permission from reference 25.)

into the hematoma at the fracture site (**Figure 155-3**). Inject the local anesthetic solution. Remove the needle and apply a bandage to the skin puncture site.

ALTERNATIVE TECHNIQUES

Several adjuncts to the hematoma block may aid in increasing its efficacy. The addition of hyaluronidase to the local anesthetic solution has been advocated at some centers as a means of increasing the speed and efficiency of the hematoma block. The hyaluronidase breaks down the connective tissue and allows better penetration of the local anesthetic solution into the area. The evidence for the efficacy of hyaluronidase is currently lacking.[28] **It is not currently recommended to add hyaluronidase to the local anesthetic solution.** Some authors recommend the use of other agents (e.g., anxiolytics) in combination with the hematoma block for fracture manipulation. Combining the hematoma block with nitrous oxide has shown positive results.[29]

ASSESSMENT

Reassess and document a thorough neurologic and vascular examination distal to the fracture site immediately after the injection. Assess the patient's level of pain with gentle range of motion in 10 minutes. A second injection of local anesthetic solution may be performed if required for analgesia if the patient is not experiencing any side effects or toxicity and the total combined doses do not exceed the maximum allowable limit.

AFTERCARE

The extremity can be manipulated to reduce the fracture once adequate analgesia has been obtained. Splint the extremity. **Perform and document another neurologic and vascular examination of the extremity after any manipulation and splinting.** Instruct the patient to immediately return to the Emergency Department for severe pain, significant swelling, numbness, paresthesias, or pallor of the extremity. Arrange appropriate follow-up with an Orthopedic Surgeon. Prescribe analgesics as appropriate.

COMPLICATIONS

The complications associated with a hematoma block are rare.[15] These include a compartment syndrome, local anesthetic toxicity, and osteomyelitis.[30-36] The early signs of local anesthetic toxicity include circumoral and tongue numbness, dizziness, lightheadedness, mental status decline, tinnitus, and visual disturbances. The cardiovascular toxic effects include asystole, atrioventricular blocks, bradycardia, cardiac depression, dysrhythmias, and hypotension. The neurologic toxic effects include agitation, coma, confusion, headaches, seizures, and possibly death.[21,37] Seizures associated with local anesthetic toxicity are usually short lasting and respond to barbiturates, benzodiazepines, and propofol. Refer to Chapters 153, 154, and 157 for the complete details regarding local anesthetic complications and toxicity. Introducing bacteria into a previously closed fracture and injury to vascular structures are potential complications. These can be minimized or eliminated by using strict sterile technique and carefully identifying the anatomic landmarks.

SUMMARY

A hematoma block is an effective technique to facilitate manipulation of extremity fractures and intraarticular fracture-dislocations in adults and children. It may be performed purely for analgesia when no manipulation is required. Complications are rare if proper techniques are used. The procedure requires fewer Emergency Department resources than procedural sedation or a Bier block. Consider performing a hematoma block when procedural sedation is impractical or contraindicated. Consider patient preference. Most patients would prefer a shorter Emergency Department stay with a hematoma block rather than a much longer one involving procedural sedation and the associated risks.

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156 Regional Nerve Blocks (Regional Anesthesia)

Eric F. Reichman and Jehangir Meer

INTRODUCTION

Regional anesthesia or regional nerve blocks are defined as infiltration of a peripheral nerve with local anesthetic agents to attenuate motor output and sensory input. It provides anesthesia to allow problems to be treated efficiently and with minimal discomfort. Patients typically tolerate nerve blocks better than direct wound infiltration. Nerve blocks often require less local anesthetic solution than does infiltration of large wounds.

Regional anesthesia provides sensory blockade of a region without altering the normal anatomic features of the area to be repaired.[1] It may be considered for use in the repair of extensive wounds, incision and drainage of abscesses, foreign body removal, wound exploration, burn care, fracture reduction, or pain control. Once familiar with the body's sensory innervation, the Emergency Physician can easily employ regional anesthesia techniques within the Emergency Department.

Locating and anesthetizing a peripheral nerve is accomplished in one of four ways. First is to identify the general location of the nerve using anatomy and landmarks. Infiltrate local anesthetic solution at that site and allow it to diffuse over the area. The second is to locate a nerve by using the injecting needle to elicit paresthesias. Once paresthesias are elicited, withdraw the needle 1 to 2 mm and allow the paresthesias to resolve before injecting the local anesthetic solution. Third, a nerve stimulator can be used to accurately locate peripheral nerves with motor fiber components. Use of a nerve stimulator does not require cooperation on the part of the patient. However, due to its complexity, a physician skilled in its use is required. Nerve stimulators are rarely available in the Emergency Department. Finally, ultrasound can be used to identify the target nerve and to inject the local anesthetic solution.

The traditional method used by Anesthesiologists to perform regional anesthesia involves a combination of surface landmarks and nerve stimulation. Over the past 10 to 15 years, ultrasound (US) has gained a prominent role in guiding nerve blocks. It offers the advantages of visualizing the nerve and the needle, as well as directly visualizing the deposition of local anesthetic solution around the nerve. Several small initial studies have shown that Emergency Physicians can safely perform US-guided nerve blocks.[2-5]

It is common to encounter children complaining of pain in the Emergency Department. Regional anesthesia is frequently overlooked in children. Its use is increasing and serves as an excellent opportunity to minimize pain in the pediatric population.[6] It can be administered safely and effectively in these patients. A child may require intravenous or intramuscular sedation in conjunction with nerve blockade in more complicated cases. The use of nitrous oxide with pediatric patients in the Emergency Department has been found to be successful when used for forearm fracture manipulation.[7] It can also be used for other procedures. Refer to Chapter 158 regarding the use of nitrous oxide as a supplement to performing the regional nerve block. The disadvantages of performing regional nerve blocks in children include the extra time required to perform the block, mandatory technical dexterity, and assistant support because the child may not remain still for the procedure.

This chapter covers the commonly performed Emergency Department regional anesthetic blocks of the head, neck, upper extremity, lower extremity, and two of the many torso blocks (**Table 156-1**). Dental blocks are discussed in Chapter 209. Refer to Chapter 153 for a more complete discussion on the properties of local anesthetic agents.

TABLE 156-1 Nerves and Anatomic Areas That Can Be Anesthetized in the Emergency Department Using a Regional Block

Head and neck	Lower extremity	Upper extremity	Torso
Supraorbital nerve block	Femoral nerve block	Brachial plexus block	Intercostal block
Supratrochlear nerve block	Saphenous nerve block	Median nerve block	Penile nerve block
Infraorbital nerve block	Lateral femoral cutaneous nerve block	Ulnar nerve block	
Mental nerve block	Obturator nerve block	Radial nerve block	
Greater occipital nerve block	Sciatic nerve block	Wrist block	
Lesser occipital nerve block	Popliteal fossa block	Digital nerve block	
Greater auricular nerve block	Common peroneal nerve block		
Scalp block	Superficial peroneal nerve block		
External ear block	Deep peroneal nerve block		
External auditory canal block	Sural nerve block		
Cervical plexus block	Posterior tibial nerve block		
	Ankle block		
	Digital nerve block		

ANATOMY AND PATHOPHYSIOLOGY

There is a topographic arrangement of axons within peripheral nerves (**Figure 156-1*A***).[8,9] Axons located in the outer or mantle layer innervate proximal structures. Axons in the center of the nerve or core layer innervate distal structures. Local anesthetic solution injected near a nerve diffuses from the mantle layer to the core layers. **This explains why anesthesia slowly spreads along the nerve distribution in a proximal to distal direction.**

Avoid intraneural injection when performing peripheral nerve blocks. The nerve has a tough, fibrous outer sheath that acts as a physical barrier to trap intraneural fluid (**Figure 156-1*B***). Injection of local anesthetic agents into the nerve bundle will compress the fragile axons and their capillary blood supply.[10,11] This can result in axonal necrosis and permanent nerve damage. **Paresthesias elicited upon needle insertion indicate that the tip of the needle is within the nerve bundle. Withdraw the needle 1 to 2 mm and allow the paresthesias to resolve, usually within 15 to 30 seconds. The anesthetic agent can be safely injected when the paresthesias resolve.**

Cutaneous innervation is referenced to a segment known as a dermatome.[8] This is defined as an area of skin supplied by a single spinal or segmental nerve. This type of innervation is best represented in worms where each body segment has its own nervous supply. The pattern of segmental innervation still holds true with some minor modifications as one moves up the phylogenetic tree. The truncal dermatomes in humans are represented as simple bands while the extremity dermatomes are serpiginous and follow the embryonic rotation of the limb buds. The most commonly used dermatomal chart is that developed by Keegan and Garrett (**Figure 156-2**).[9] Their model of the extremity dermatomes is in strips of innervation, all originating from the limb base and extending distally. This system is used in clinical medicine today.

INDICATIONS

Regional anesthesia produces profound analgesia with minimal physiologic or anatomic alteration. These techniques are especially useful in large or extensive lacerations that would otherwise require the infiltration of a large and potentially toxic volume of

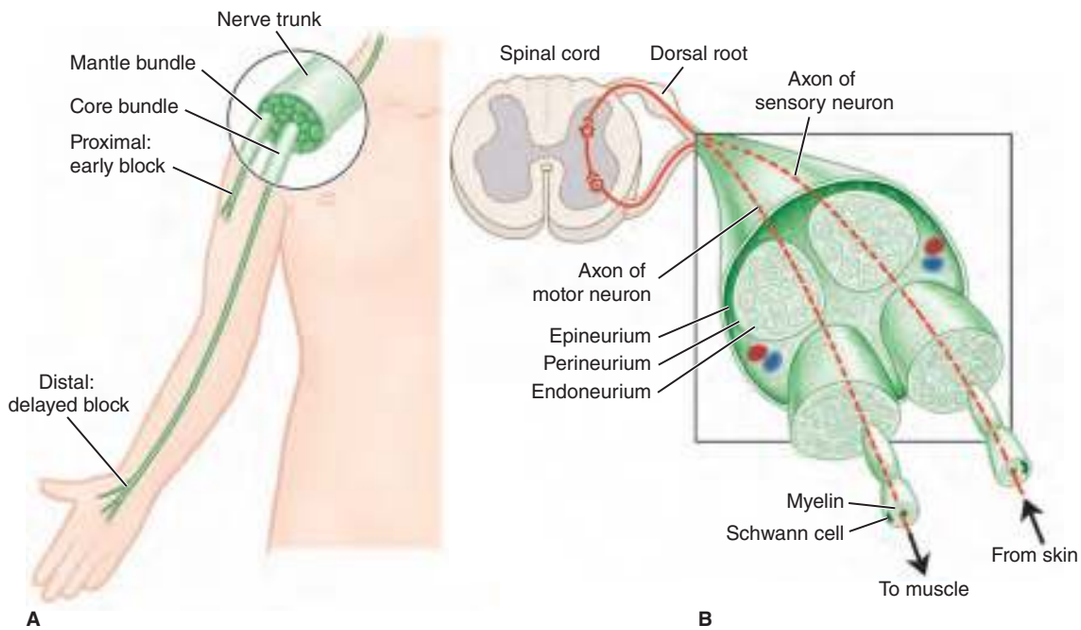


FIGURE 156-1. The anatomy and topographic arrangement of axons in a peripheral nerve. ***A.*** The gross anatomy of a peripheral nerve. ***B.*** The microscopic anatomy of a peripheral nerve.

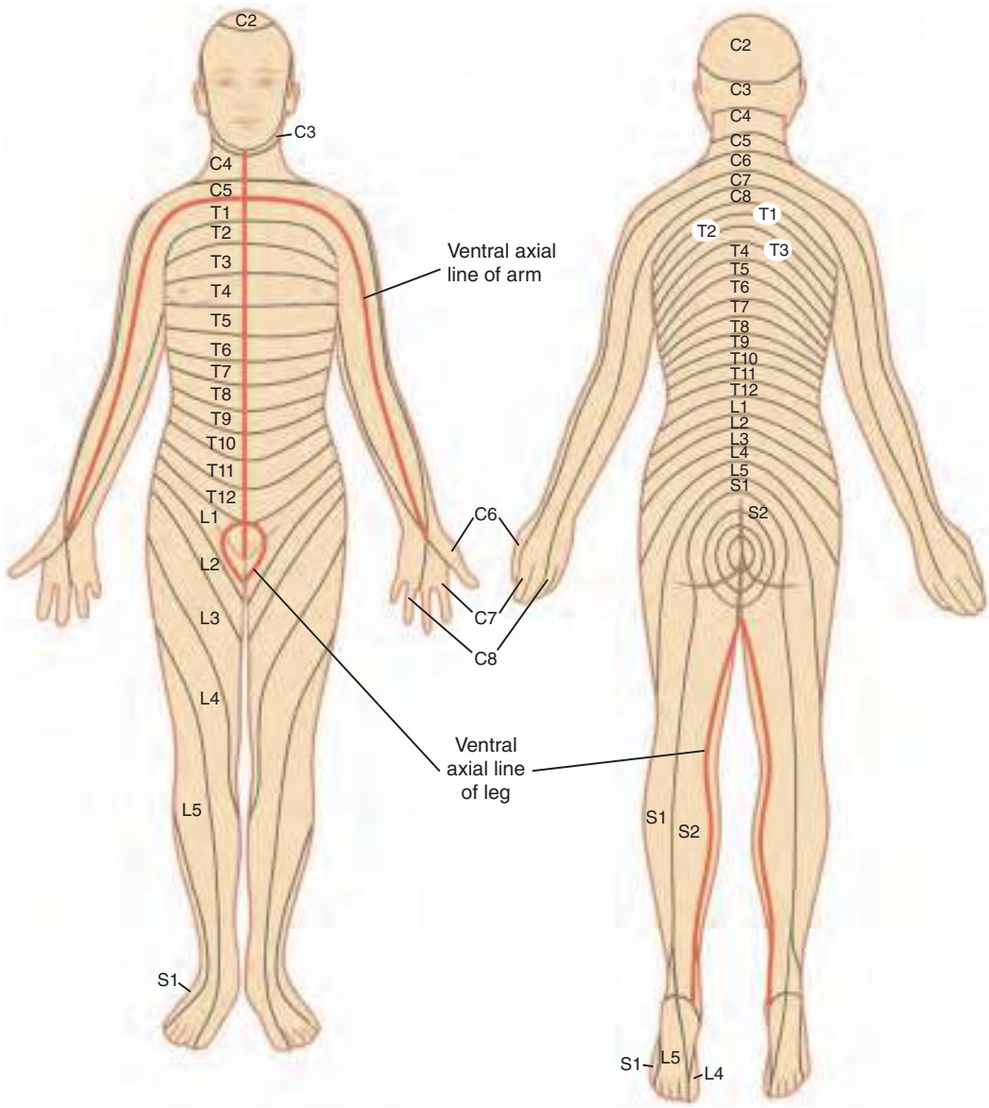


FIGURE 156-2. The dermatomal chart of the human body.

local anesthetic solution. Nerve blocks can avoid a patient being taken to the Operating Room because the volume of local anesthetic required for extensive wound repair may require toxic doses. These techniques are also useful in cosmetic repairs where local infiltration may cause distortion of tissues or loss of anatomic landmarks making approximation and repair difficult. The necessity to palpate deep tissue for excision is also an indication for regional anesthesia.[12,13] Regional nerve blocks can be performed prior to burn care, dislocation or fracture reduction, foreign body removal, incision and drainage of abscesses, pain control, wound exploration, and wound care.

US-guided regional anesthesia offers a number of advantages when compared with the anatomic landmark. It is safer and results in fewer complications (e.g., vascular puncture, pneumothorax, intravascular injection of local anesthetic).[14] US achieves higher rates, over 95%, of successful blocks and delivers a more rapid onset of anesthesia. In addition, a smaller volume of local anesthetic solution is required for the block.[14] US can eliminate the need for procedural sedation and analgesia (PSA) and its potential complications (e.g., apnea, hypoxia, and hypotension). Preprocedural fasting is not required as it is a potential requirement in PSA. US-guided nerve blocks reduce Emergency Department lengths of stay compared with patients receiving PSA.[2] It facilitates procedures in patients with higher American Society of Anesthesiology (ASA) classification scores who would otherwise be put at higher risk for complications by the administration of PSA.

CONTRAINDICATIONS

There are few contraindications to regional anesthesia.[8,15,16] Absolute contraindications include injection through infected tissue, history of a bleeding disorder or a coagulopathy, or an allergy to the anesthetic agent. **Relative contraindications include preexisting neurologic damage prior to the procedure. This should carefully be documented before any anesthetic injection.** Patients with altered mental status or the inability to cooperate (e.g., developmental delay, intoxication, or young children) should have an alternative treatment plan instead of a regional nerve block.[6,17,18] They would not be able to provide the feedback of paresthesias in case of intraneural needle placement.

There are no patient contraindications to the use of US to guide nerve blocks. Emergency Physician contraindications include the lack of familiarity and training with the procedure. Psychomotor

TABLE 156-2 Maximum Doses of Local Anesthetic Agents

Anesthetic agent	Maximum dose (mg/kg)	Duration of action (min)
Procaine (2%)	6	15–30
Procaine (2%) with epinephrine	8	30–90
Tetracaine (0.25%)	1	120–140
Tetracaine (0.25%) with epinephrine	2	240–280
Chloroprocaine (2%)	8	15–30
Chloroprocaine (2%) with epinephrine	10	30–90
Lidocaine (1%)	3	30–120
Lidocaine (1%) with epinephrine	5	60–400
Etidocaine (0.5%)	3	30–120
Etidocaine (0.5%) with epinephrine	4	60–200
Mepivacaine (1%)	3	30–120
Mepivacaine (1%) with epinephrine	5	60–400
Bupivacaine (0.25%)	1.75	120–240
Bupivacaine (0.25%) with epinephrine	2.25	240–480
Prilocaine (4%)	5	30–60
Prilocaine (4%) with epinephrine	6	120–300

coordination and experience with US are required. Caution is advised for the novice ultrasonographer.

EQUIPMENT

ANATOMIC LANDMARK-GUIDED NERVE BLOCKS

- Sterile gloves
- Sterile drapes
- Povidone iodine or chlorhexidine solution
- Alcohol swabs
- Local anesthetic solution (**Table 156-2** and Chapter 153)
- 18 gauge needle to draw up local anesthetic solution
- 20 to 27 gauge noncutting or Quincke needles for injection, 2 inches long
- 22 to 24 gauge noncutting or Quincke spinal needles
- 1, 3, 5, 10, and 60 mL syringes
- Intravenous extension tubing

US-GUIDED NERVE BLOCKS

- Above listed supplies
- US machine
- High-frequency, linear-array, US transducer
- Sterile US transducer cover
- Sterile US gel
- Noncutting needles

All of the required equipment is readily available in any Emergency Department. Some have a preprepared tray or tackle box containing all the required equipment.

Numerous additives are used to supplement the local anesthesia agent injected for regional anesthesia.[19] The authors do not recommend their use in the Emergency Department. The additives can have numerous side effects and have not been studied for their use outside of the anesthesia literature.

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Emphasize that there are few complications with this procedure. However, all possible complications should be discussed beforehand. Inform the patient of the possibility of paresthesias during the procedure and of the expected duration of action of the local anesthetic agent (**Table 156-2**). **Obtain an informed consent for the regional nerve block in addition to the procedure for which it is performed.** Ideally, the consent should be documented in the medical record and signed by the patient. Some Emergency Physicians prefer to note on the patient's chart "indications, risks, and benefits were discussed with the patient" rather than having the patient sign a consent form. This decision is specific to each Emergency Physician, their institution, and state requirements.

Perform and document a neurologic examination of the area to be anesthetized before performing regional anesthesia. Include a description of any neurologic deficit in the document of informed consent for the procedure. Have the patient sign an agreement that the defect was present prior to the administration of the local anesthetic solution.

Position the patient based upon the specific regional block to be performed. Place the patient supine on a gurney or procedure table prior to the procedure in most cases. If the patient must sit upright, place the patient on an adjustable bed. The patient's comfort should be optimized to prevent unexpected complications such as vasovagal syncope.[8,20,21] Expose the area of the injection and identify the anatomic landmarks required for proper needle placement. Clean all dirt and debris from the skin. Scrub the needle insertion site with povidone iodine or chlorhexidine solution and allow it to dry. Apply sterile drapes to delineate a sterile field.

If using US guidance, prepare the US transducer. **Always use a sterile US transducer cover and sterile US gel when performing nerve blocks to prevent deep infections.** Set up a sterile field on a bedside table. Open the US transducer cover set onto the sterile field. Instruct an assistant to hold the US transducer upright and place standard or sterile US gel on the footprint of the US transducer (**Figure 63-6*A***). Apply the sterile transducer cover over the US transducer (**Figure 63-6*B***). Smooth all the air bubbles away from the footprint of the US transducer to prevent imaging artifacts. Secure the cover with rubber bands to prevent it from sliding off the US transducer (**Figure 63-6*C***). Place the US transducer on the sterile field (**Figure 63-6*D***). Apply sterile US gel onto the cover over the US transducer footprint just before scanning.

TECHNIQUE

The general procedure will first be described and specifics will be addressed with each individual nerve block.

ANATOMIC LANDMARK TECHNIQUE

Position the patient. Identify the nerve or nerves to be blocked and their associated anatomic landmarks. Carefully clean and prepare the skin over the injection site in a sterile fashion. Draw up the local anesthetic solution to be injected. The amount will vary based on the specific block. Always keep in mind the maximum allowable dose of local anesthetic (**Table 156-2**). Reidentify the anatomic landmarks. Insert the needle into the site. Withdraw the plunger to ensure that the tip of the needle is not within a blood vessel, thus avoiding intravascular injection. If paresthesias are elicited, withdraw the needle 1 to 2 mm and allow them to resolve. Inject the local anesthetic solution. Apply an appropriate bandage to the site. Allow 5 to 15 minutes for the block to take effect. Confirm that anesthesia has been achieved with pinprick prior to performing the procedure for which regional anesthesia was performed. Document the regional anesthesia procedure, the procedure for which regional anesthesia was performed, and any complications in the medical record. A sample regional anesthesia procedure note is provided in **Table 156-3**.

TABLE 156-3 A Sample Regional Anesthesia Procedure Note

After informed consent and identification of the necessary landmarks, the skin overlying _____ (location) was cleaned and prepped with povidone iodine or chlorhexidine solution. Using sterile technique, a skin wheal of local anesthetic solution was placed. A ____ gauge needle was used to anesthetize the _____ nerve with _____ mL of ____ % _____ (lidocaine, marcaine, procaine, etc.). Anesthesia was confirmed with needle pinprick testing. No complications were noted.

ULTRASOUND-GUIDED TECHNIQUE

Position and prepare the patient as described above for the anatomic landmark technique. Find the general location of the nerve using anatomic landmarks. Use US to identify the targeted nerve. Use the nondominant hand to hold the US transducer. Manipulate the needle with the other hand. Center the target nerve on the monitor screen.

Use a longitudinal or in-plane needle approach for all US-guided nerve blocks. The US transducer should image the nerve bundle in the short axis, allowing the entire length of the needle to be visualized as it approaches the nerve. Use a noncutting needle, such as a Quincke spinal needle, to reduce the risk of intraneural injection or nerve injury. Place the US machine on the patient's opposite side to allow the Emergency Physician to glance easily and quickly from the field to the US monitor. Connect a short IV extension tubing to allow easier manipulation of the needle if an assistant is available. The assistant can hold the syringe containing the local anesthetic and inject it under the direction of the Emergency Physician who is controlling the needle. An extra level of safety for the novice performing this injection is to have the supervising physician control the syringe and injection.

An important concept regarding the injection of local anesthetic solution around the nerve bundle is the "donut sign." This is the circumferential spread of local anesthetic solution around the nerve. This is the desired location of the local anesthetic solution around the nerve and it is usually associated with a successful block.

REGIONAL ANESTHESIA TECHNIQUES FOR THE HEAD AND NECK

SUPRAORBITAL NERVE BLOCK

Anatomy: The supraorbital foramen lies on the supraorbital ridge along a line drawn through the pupil in the midposition (**Figures 156-3 and 156-4**). The supraorbital foramen may be palpable as an indentation. The supraorbital nerve is a branch of the ophthalmic division of the trigeminal nerve. It emerges through the supraorbital foramen, or notch, at the midline of the superior orbital ridge (**Figure 156-4*A***). Its area of innervation includes the forehead beginning at the superior orbital ridge and extending superiorly to the vertex of the scalp. It is blocked simultaneously with the supratrochlear nerve as there is considerable overlap in their areas of innervation.

Patient positioning: Place the patient supine, or sitting, and facing the Emergency Physician.

Landmarks: Identify, under the eyebrow, the superior orbital rim and the supraorbital foramen by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution over the midline of the forehead at the level of the eyebrow. Insert a 25 or 27 gauge needle through the skin wheal and aimed laterally (**Figure 156-4*B***). Advance the needle while infiltrating subcutaneously with 3 to 5 mL of local anesthetic solution. Always maintain the needle just above the supraorbital ridge while advancing it (**Figure 156-4*B***). Stop infiltrating when the needle passes the midline of the bony orbit.

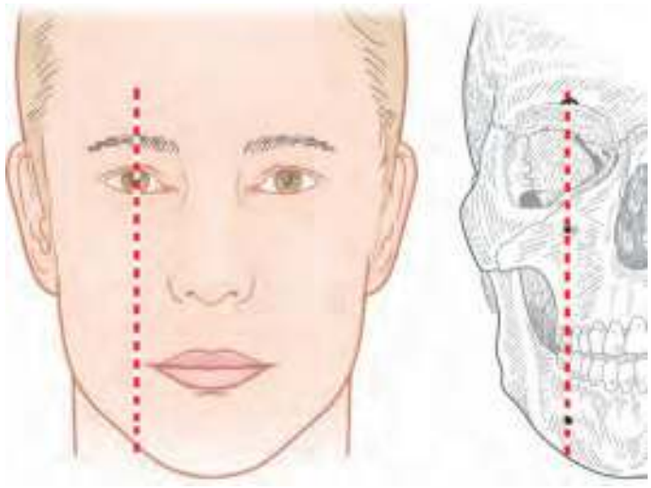


FIGURE 156-3. The supraorbital foramen, the infraorbital foramen, and the mental foramen all lie along a straight line drawn through the pupil in the midposition.

US-guided block: The nerve can also be located exiting the foramen with US. Find the foramen where the nerve exits with US. Inject 1.5 mL of the local anesthetic just outside the foramen. **Do not inject into the foramen as this can put pressure on the nerve and result in necrosis.**

Remarks: This technique will anesthetize both the supraorbital and supratrochlear nerves. Infiltrate 2 mL of local anesthetic solution directly over the supraorbital foramen or notch, if it is palpable, rather than subcutaneously infiltrating above the supraorbital ridge.

SUPRATROCHLEAR NERVE BLOCK

Anatomy: The supratrochlear nerve is a branch of the ophthalmic division of the trigeminal nerve. It emerges through the trochlea at the supermedial aspect of the bony orbit (**Figure 156-4*A***). It provides innervation to the middle of the forehead beginning at the superior orbital ridge and extending superiorly to the vertex of the scalp. It is often blocked simultaneously with the supraorbital nerve as there is considerable overlap in their areas of innervation.

Patient positioning: Place the patient supine, or sitting, and facing the Emergency Physician.

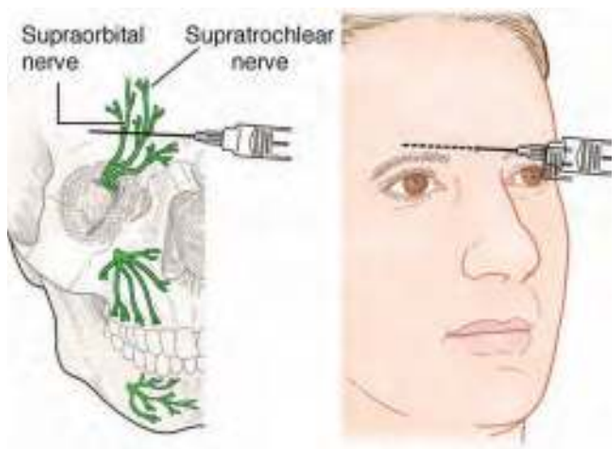


FIGURE 156-4. The supraorbital and supratrochlear nerve block. ***A.*** The location of the nerves. ***B.*** Insertion of the needle.

Landmarks: Identify, under the eyebrow, the superior orbital rim by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution over the midline of the forehead at the level of the eyebrows. Insert a 25 or 27 gauge needle through the skin wheal, aimed laterally (**Figure 156-4*B***). Advance the needle while infiltrating subcutaneously with 2 to 3 mL of local anesthetic solution. Always maintain the needle just above the supraorbital ridge while advancing it (**Figure 156-4*B***). Stop infiltrating when the needle passes the midline of the bony orbit.

Remarks: Stop infiltrating 1.5 cm from the skin wheal so as to block only the supratrochlear nerve.

INFRAORBITAL NERVE BLOCK, EXTRAORAL APPROACH

Anatomy: The infraorbital nerve is a branch of the maxillary division of the trigeminal nerve. It emerges through the infraorbital foramen 1 cm below the middle to medial third of the inferior orbital ridge (**Figure 156-5*A***). It lies in the same plane as the supraorbital foramen and pupil that is in the midposition (**Figures 156-3 and 156-5**). The nerve exits the infraorbital foramen and travels inferiorly and medially. It provides sensory innervation to the medial cheek, nasal ala, upper lip, and skin between the upper lip and nose. The infraorbital nerve terminates as the anterior and middle superior alveolar nerves. They provide sensory innervation to the maxillary incisors, canine, and premolar teeth, as well as their bony support and surrounding soft tissues.

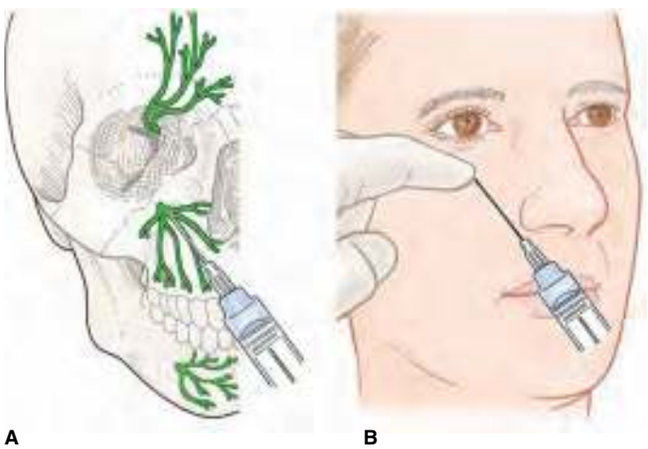
Patient positioning: Place the patient supine, or sitting, and facing the Emergency Physician.

Landmarks: Identify the infraorbital foramen by palpation. Significant tenderness will be elicited when the infraorbital nerve is palpated as it exits the infraorbital foramen.

Needle insertion and direction: Insert a 25 or 27 gauge needle just above the infraorbital foramen (**Figure 156-5*B***). Advance the needle until the maxilla is contacted. Inject 1 to 2 mL of local anesthetic solution.

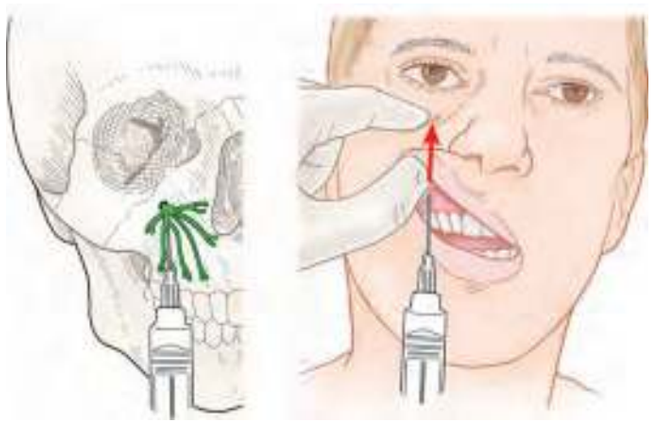
US-guided block: The nerve can also be located exiting the foramen with US. Find the foramen where the nerve exits with US. Inject 1.5 mL of the local anesthetic just outside the foramen. **Do not inject into the foramen as this can put pressure on the nerve and result in necrosis.**

Remarks: The infraorbital nerve can be blocked intraorally. The intraoral route results in the patient experiencing less pain than the extraoral route.



A B

FIGURE 156-5. The extraoral approach to the infraorbital nerve block. ***A.*** Location of the nerve. ***B.*** Insertion of the needle.



A B

FIGURE 156-6. The intraoral approach to the infraorbital nerve block. ***A.*** Location of the nerve. ***B.*** Insertion of the needle.

INFRAORBITAL NERVE BLOCK, INTRAORAL APPROACH

Anatomy: The anatomy and innervation of the infraorbital nerve is described in the previous section.

Patient positioning: Place the patient supine, or sitting, and facing the Emergency Physician.

Landmarks: Identify the infraorbital foramen by palpation. It lies in a plane with the supraorbital foramen and the pupil in the midposition (**Figures 156-3 and 156-6*A***).

Needle insertion and direction: Place the index finger of the nondominant hand over the infraorbital foramen (**Figure 156-6*B***). Use the nondominant thumb to retract the upper lip. Insert a 2.5 to 4.0 cm, 25 or 27 gauge needle through the mucous membranes and directed toward the index finger. Advance the needle until its tip is palpable at the infraorbital foramen by the index finger. The estimated depth of needle penetration is 1.0 to 1.5 cm. Inject 2 mL of local anesthetic solution.

US-guided block: The nerve can also be located exiting the foramen with US. Find the foramen where the nerve exits with US. Aim and advance the needle toward the US transducer. Inject 1.5 mL of the local anesthetic just outside the foramen. **Do not inject into the foramen as this can put pressure on the nerve and result in necrosis.**

Remarks: This is the preferred route to block the infraorbital nerve. It can also be blocked extraorally.

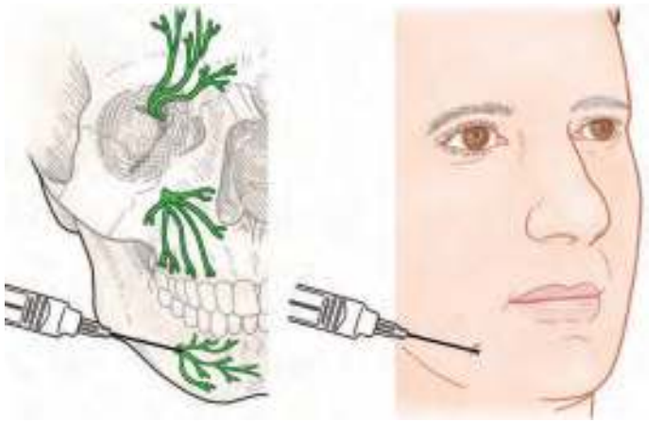
MENTAL NERVE BLOCK, EXTRAORAL APPROACH

Anatomy: The mental nerve is a branch of the mandibular division of the trigeminal nerve. It emerges from the mental foramen. The foramen lies in a vertical plane with the supraorbital foramen, the infraorbital foramen, and the pupil that is midposition (**Figures 156-3 and 156-7**). The nerve travels inferiorly and anteriorly to provide sensory innervation to the skin of the lower lip and chin.

Patient positioning: Place the patient supine, or sitting, and facing the Emergency Physician.

Landmarks: Identify the vertical plane consisting of the supraorbital foramen, the infraorbital foramen, and the midposition pupil (**Figure 156-3**). Identify the point where the vertical plane crosses the middle of the body of the mandible (**Figure 156-7*A***). This is where the mental nerve exits the mental foramen.

Needle insertion and direction: Insert a 25 or 27 gauge needle at the above identified landmark (**Figure 156-7*B***). Place a skin wheal



A B

FIGURE 156-7. The extraoral approach to the mental nerve block. ***A.*** Location of the nerve. ***B.*** Insertion of the needle.

of local anesthetic solution at this intersection. Advance the needle through the skin wheal until the mandible is contacted. Inject 1 to 2 mL of local anesthetic solution.

US-guided block: The nerve can also be located exiting the foramen with US. Find the foramen where the nerve exits with US. Inject 1.5 mL of the local anesthetic just outside the foramen. **Do not inject into the foramen as this can put pressure on the nerve and result in necrosis.**

Remarks: This nerve can also be blocked intraorally. The intraoral route results in the patient experiencing less pain than the extraoral route.

MENTAL NERVE BLOCK, INTRAORAL APPROACH

Anatomy: The anatomy and innervation of the mental nerve are described in the previous section.

Patient positioning: Place the patient supine, or sitting, and facing the Emergency Physician.

Landmarks: Retract the lower lip and identify the junction of the first and second premolars. The patient's mouth may be open or closed.

Needle insertion and direction: Insert a 25 or 27 gauge needle directed inferiorly and posteriorly through the gingival mucosa at the junction of the first and second premolars (**Figure 156-8**). Advance the needle one-third the depth of the mandibular body and contact the mandible. Inject 1 to 2 mL of local anesthetic solution.

US-guided block: The nerve can also be located exiting the foramen with US. Find the foramen where the nerve exits with US. Inject 1.5 mL of the local anesthetic just outside the foramen. **Do not inject into the foramen as this can put pressure on the nerve and result in necrosis.**

Remarks: This is the preferred approach to block the mental nerve. The intraoral route results in the patient experiencing less pain than the extraoral route.

FIGURE 156-8. The intraoral approach to the mental nerve block.

GREATER OCCIPITAL NERVE BLOCK

Anatomy: The greater occipital nerve is a branch of the dorsal ramus of the second cervical nerve. It provides sensory innervation to the posterior neck, extending superiorly to the vertex of the scalp (**Figure 156-9**). It emerges on the posterosuperior neck just below the line connecting the external occipital protuberance and the mastoid process (**Figure 156-10**). The posterior occipital artery accompanies the greater occipital nerve.

Patient positioning: Place the patient prone.

Landmarks: Identify the external occipital protuberance and mastoid process by palpation (**Figure 156-10*A***). Connect these landmarks with a line. Identify the occipital artery by its palpable pulse approximately one-third the distance from the external occipital protuberance.

Needle insertion and direction: Place a skin wheal of local anesthetic solution over the pulse of the occipital artery. Insert a 25 gauge needle 1 to 2 mm to the left of the occipital artery pulse. Inject 1 mL of local anesthetic solution. Redirect the needle 1 to 2 mm to the right of the pulse and inject 1 mL of local anesthetic solution.

US-guided block: Several small studies have shown that these injections can be performed safely.[22,23] Place the patient prone. Place the linear transducer below the external occipital protuberance in a transverse orientation (**Figure 156-10*C***). Turn on the color Doppler box. Slide the transducer laterally until the occipital artery is seen. The greater occipital nerve is medial to the occipital artery (**Figure 156-10*D***). Insert a 25 gauge needle under the lateral border of the transducer. Advance the needle to the nerve and inject 1 to 2 mL of local anesthetic solution.

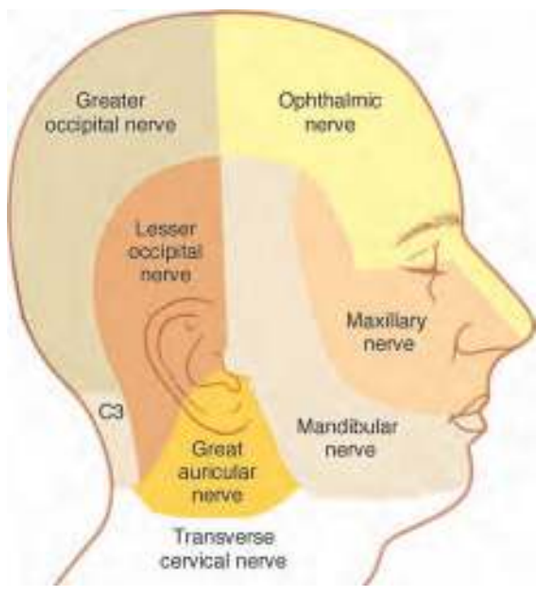


FIGURE 156-9. The cutaneous nerve supply to the face, scalp, and upper neck.

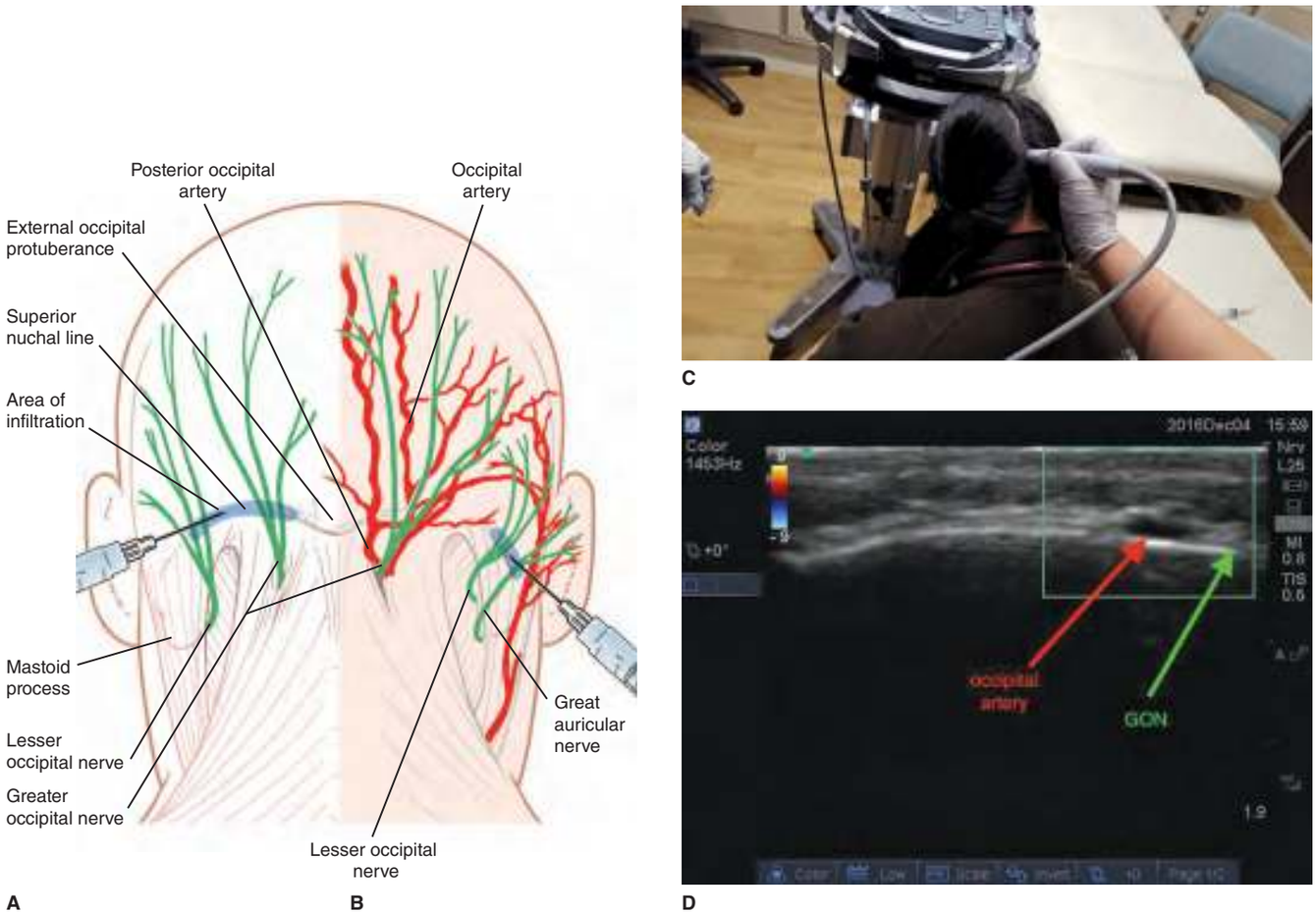


FIGURE 156-10. Regional anesthesia of the posterolateral scalp. ***A.*** The greater occipital nerve block. ***B.*** The lesser occipital and great auricular nerve blocks. ***C.*** Transducer placement for blocking the greater occipital nerve. ***D.*** US image of the greater occipital nerve.

Remarks: This block is useful for laceration repair as well as relief of occipital muscular or tension headaches.[24] If the pulse of the posterior occipital artery is not palpable, divide the line between the external occipital protuberance and the mastoid process into thirds. Infiltrate the middle third subcutaneously with 5 to 8 mL of local anesthetic solution (**Figure 156-10*A***). This technique will also anesthetize the lesser occipital nerve.

LESSER OCCIPITAL NERVE BLOCK

Anatomy: The lesser occipital nerve is a branch of the cervical plexus. It provides sensory innervation to the skin and scalp between the ear and the mastoid process (**Figure 156-9**). It emerges at the middle third of the posterior border of the sternocleidomastoid muscle and travels superiorly toward the mastoid process (**Figure 156-10**).

Patient positioning: Place the patient supine or sitting with their head turned toward the side opposite that being anesthetized.

Landmarks: Identify the mastoid process by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution just posterior to the mastoid process. Insert a 25 gauge needle directed anteriorly through the skin wheal. Advance the needle while infiltrating local anesthetic solution subcutaneously until the posterior ear is contacted (**Figure 156-10*B***). This requires 3 to 7 mL of local anesthetic solution.

Remarks: The lesser occipital nerve can be blocked at the level of the cervical plexus. Refer to the section on cervical plexus blockade below.

GREAT AURICULAR NERVE BLOCK

Anatomy: The great auricular nerve is a branch of the cervical plexus. It emerges at the middle third of the posterior border of the sternocleidomastoid muscle and travels superiorly with the external jugular vein (**Figure 156-10*B***). This nerve provides sensory innervation to the skin and scalp behind the ear, the posterior ear, and the lobule (**Figure 156-9**).

Patient positioning: Place the patient supine with their head turned toward the side opposite that being anesthetized.

Landmarks: Identify the lobule of the ear, the mastoid process, and the sulcus behind the ear.

Needle insertion and direction: Place a skin wheal of local anesthetic solution just posterior to the mastoid process. Insert a 25 gauge needle directed anteriorly through the skin wheal. Advance the needle while infiltrating local anesthetic solution subcutaneously until the posterior ear is contacted (**Figure 156-10*B***). This requires 3 to 7 mL of local anesthetic solution. Complete and successful anesthesia occurs within 10 minutes.

US-guided block: The nerve can also be blocked with US.[25] Find the nerve with US. Aim and advance the needle toward the nerve. Inject the 1.5 mL of the local anesthetic. The anesthetic spreads around the nerve to obtain the "donut sign."

Remarks: The great auricular nerve block is indicated for lacerations of the auricle, debridement, and hematoma evacuations.[25] It is rarely performed without simultaneous blockade of the auriculotemporal nerve. The great auricular nerve can be blocked at the

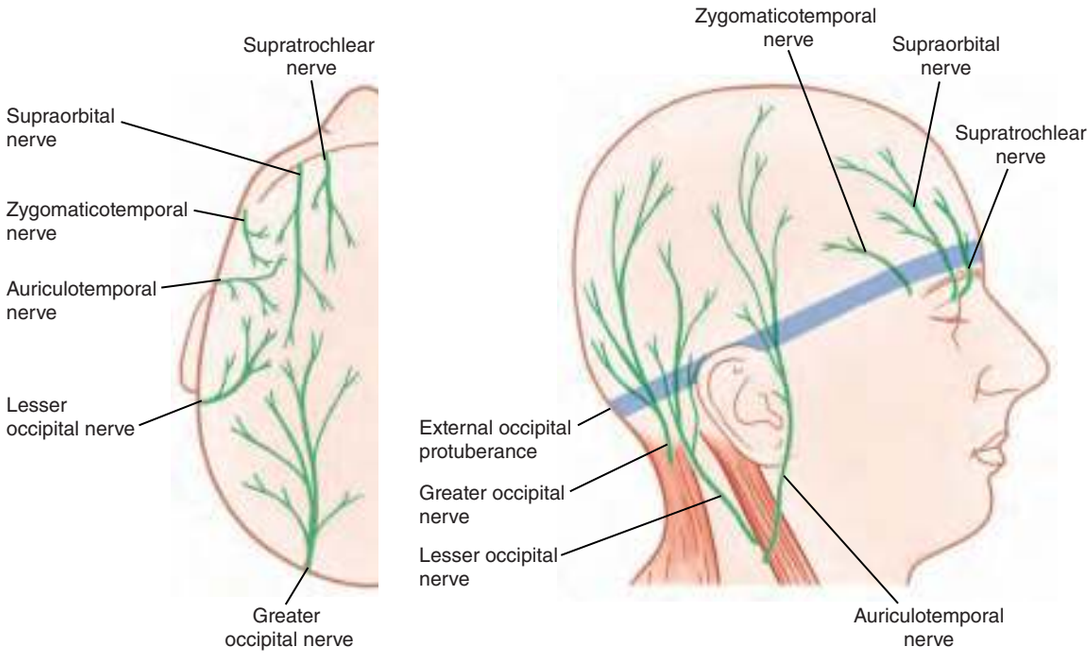


FIGURE 156-11. The scalp block. ***A.*** The sensory innervation of the scalp. ***B.*** Local anesthetic solution is injected subcutaneously along the base of the scalp.

level of the cervical plexus. Refer to the section on cervical plexus blockade below.

SCALP BLOCK

Anatomy: The scalp receives its sensory innervation from branches of the trigeminal nerve anteriorly and the cervical plexus posteriorly (**Figure 156-11**). The scalp may be anesthetized anywhere along the anterior midline to the posterior midline. This involves blocking the supratrochlear, supraorbital, auriculotemporal, lesser occipital, great auricular, and greater occipital nerves.

Patient positioning: Place the patient supine with their head turned toward the side opposite that being anesthetized.

Landmarks: Identify the glabella and the external occipital protuberance by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution over the glabella. Insert a 25 gauge needle through the skin wheal. Infiltrate a continuous line of local anesthetic solution subcutaneously between the glabella and the external occipital protuberance (**Figure 156-11*B***). This requires 15 to 20 mL of local anesthetic solution.

Remarks: Infiltrate the local anesthetic solution subcutaneously along the scalp base inferior to the area in which the procedures will be performed to block only a portion of the scalp. It is useful to add epinephrine (1:200,000) to the local anesthetic solution to cause vasoconstriction and prevent excessive blood loss. Significant systemic absorption of the local anesthetic agent does not occur despite the extensive vascularity of the scalp. Scalp blocks provide anesthesia for laceration repair, drainage of superficial abscesses, and the exploration of scalp wounds. Complications are fairly rare.[26] There is a case report of a temporary facial nerve palsy after a scalp block.[27]

EXTERNAL EAR BLOCK

Anatomy: The ear is a difficult structure to anesthetize. It is innervated by a large number of sensory fibers that originate from the cervical plexus, the trigeminal nerve, and the vagus nerve. The external ear, or pinna, is innervated by the cervical plexus and the auriculotemporal branch of the trigeminal nerve (**Figures 156-9 and 156-11**).

Patient positioning: Place the patient supine or sitting upright with their head turned toward the side opposite that being anesthetized.

Landmarks: Identify the angle of the mandible by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution over the angle of the mandible. Insert a 25 gauge needle through the skin wheal. Infiltrate local anesthetic solution subcutaneously in an anterior and superior direction, from the angle of the mandible to the superior surface of the ear, to block the auriculotemporal nerve (**Figure 156-12*A***). Infiltrate local anesthetic solution subcutaneously in a posterior and superior direction, from the angle of the mandible to the superior surface of the ear, to block the great auricular and lesser occipital nerves (**Figure 156-12*A***). This requires a total of 8 to 10 mL of local anesthetic solution.

Remarks: Some may prefer to anesthetize the auriculotemporal nerve trunk by injecting local anesthetic solution just above the posterior aspect of the zygomatic arch (**Figure 156-12*B***). It requires less local anesthetic solution and hurts less than subcutaneous infiltration. An alternative technique is to circumferentially infiltrate local anesthetic solution subcutaneously around the ear (**Figure 156-12*C***).[28]

EXTERNAL AUDITORY CANAL BLOCK

Anatomy: The external auditory canal (and the tympanic membrane) receives its innervation from the auriculotemporal nerve and the vagus nerve.

Patient positioning: Place the patient sitting upright or supine with their head turned toward the side opposite that being anesthetized.

Landmarks: Identify the helix, the tragus, and the lobule of the ear to be anesthetized (**Figure 156-13**).

Needle insertion and direction: Anesthetize the external auditory canal using a four-quadrant block (**Figure 156-13**). Insert a 25 gauge needle, advance it 0.5 to 0.75 cm, and inject 1 mL of local anesthetic solution at each of the four landmarks identified in **Figure 156-13**.

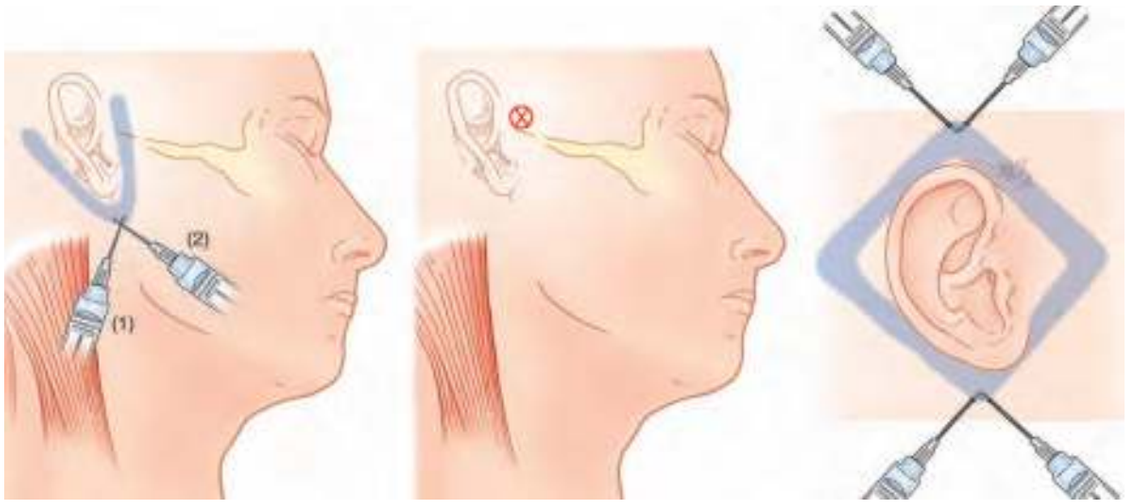


FIGURE 156-12. The external ear block. ***A.*** Infiltration of local anesthetic solution from the angle of the mandible to the anterior (*1*) and posterior (*2*) superior surfaces of the ear. ***B.*** The site for anesthetizing the trunk of the auriculotemporal nerve. ***C.*** An alternative method.

Remarks: This block can be quite painful for the patient. The authors recommend to first anesthetize the external ear before anesthetizing the external auditory canal. This block also anesthetizes the tympanic membrane.

CERVICAL PLEXUS BLOCK

Anatomy: The cervical plexus originates from the anterior rami of cervical nerves two through four. These rami form numerous loops that anastomose to form nerves that provide sensory innervation to the anterolateral neck, the scalp, the ear, and the infraclavicular area. The four nerves of the cervical plexus become superficial at the midportion of the posterior border of the sternocleidomastoid muscle and then distribute to their respective sensory areas (**Figure 156-14*A***). The cervical plexus provides motor innervation to the strap muscles of the neck.

The four superficial nerves of the cervical plexus are the lesser occipital nerve, the great auricular nerve, the anterior (or transverse) cervical nerve, and the supraclavicular nerve (**Figure 156-14*A***). The lesser occipital nerve travels superiorly and posteriorly to provide sensory innervation to part of the posterior surface of the upper ear and the postauricular skin. The great auricular nerve travels superiorly and anteriorly to provide sensory innervation to the skin over the posterior surface of the ear, the anterior lower half of the ear, and over the angle of the mandible. The anterior, or transverse, cervical nerve of the neck travels anteriorly to provide sensory innervation to the skin of the neck from the inferior border of the mandible to the sternum. The supraclavicular nerves travel and innervate the skin of the clavicle down to the second rib.

FIGURE 156-13. External auditory canal block. Inject local anesthetic solution at each of the four landmarks. This block also anesthetizes the tympanic membrane.

Patient positioning: Place the patient supine with their head turned toward the side opposite of that being anesthetized.

Landmarks: Identify the posterior border of the sternocleidomastoid muscle by palpation. Divide this border into thirds.

Needle insertion and direction: Place a skin wheal of local anesthetic solution over the middle third of the posterior border of the sternocleidomastoid muscle. Insert a needle through the skin wheal. Infiltrate 5 to 10 mL of local anesthetic solution subcutaneously over the middle third of the posterior border of the sternocleidomastoid muscle (**Figure 156-14*B***).

Remarks: This block is useful when managing burns or suturing lacerations on the anterolateral neck. The complications of this block include a resultant Horner's syndrome.[29] There is no advantage to using US with this block since the landmark is easy to find and the cervical plexus is consistent in its location.[30]

REGIONAL ANESTHESIA TECHNIQUES FOR THE UPPER EXTREMITY

BRACHIAL PLEXUS BLOCK

The brachial plexus innervates the entire upper extremity. Blockade of the brachial plexus can be performed to repair tendons or extensive lacerations, to reduce fractures and dislocations, or to provide anesthesia for burn care to name a few uses. Protect the arm from injury if this procedure is to be performed by properly supporting the arm, padding the ulnar nerve and pressure points, and not extending or displacing the arm posteriorly.

The brachial plexus arises from the C5 to T1 nerve roots (**Figure 156-15**). The nerve roots fuse to form three trunks. Each trunk divides into an anterior and posterior division that then redistributes to form the lateral, medial, and posterior cords. These cords divide in the region of the axilla to form the musculocutaneous nerve, the median nerve, the ulnar nerve, the axillary nerve,

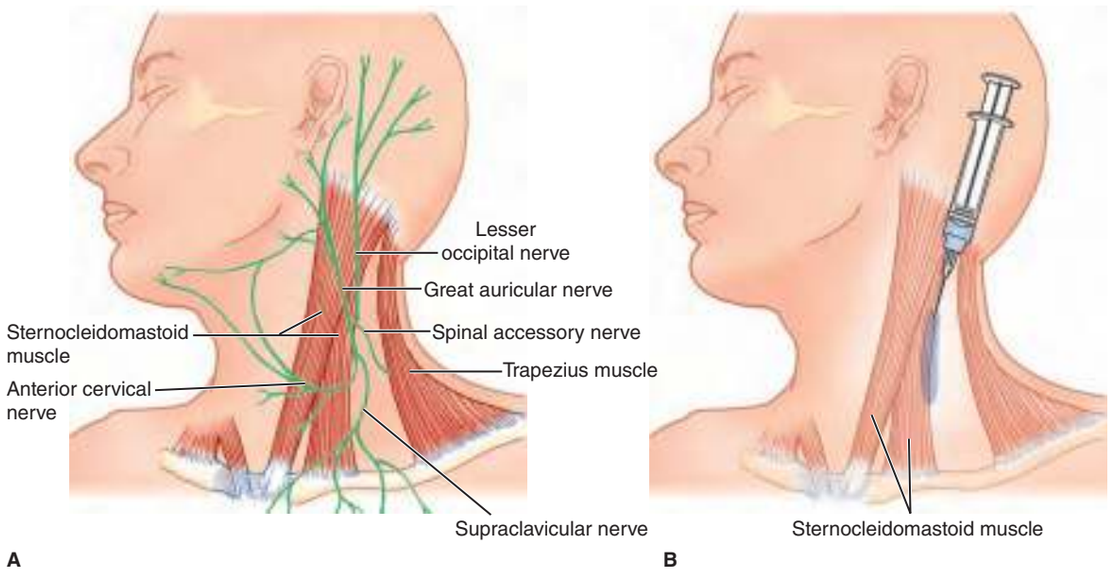


FIGURE 156-14. The cervical plexus block. ***A.*** The cutaneous nerves of the cervical plexus. ***B.*** Injection of local anesthetic solution posterior to the middle third of the sternocleidomastoid muscle.

the radial nerve, and several cutaneous nerves. The brachial plexus crosses the midclavicle to enter the axilla (**Figure 156-16*A***).

The brachial plexus may be blocked from the supraclavicular, interscalene, infraclavicular, or axillary approach. The preferred non-US method for the Emergency Physician is the axillary block which will be described in detail. The other blocks will also be discussed. The lack of US use with these other nerve block techniques requires the use of a nerve stimulator and they have a high risk of associated complications. Blockade of the brachial plexus can result in hemidiaphragm paresis, even if using US for guidance.[31,32]

BRACHIAL PLEXUS BLOCK, SUPRACLAVICULAR APPROACH

Anatomy: The anatomy of the brachial plexus is described above. This approach blocks the brachial plexus at the level of the trunks where it is most compactly arranged.

Patient positioning: Place the patient supine with their head turned 45° from the midline and toward the side opposite that being anesthetized (**Figure 156-16*A***). Place the ipsilateral arm in any position of comfort for the patient.

Landmarks: The subclavian artery is the main landmark. Palpate the subclavian artery immediately lateral to the clavicular head of the sternocleidomastoid muscle in the interscalene groove. Identify the midpoint of the clavicle.

Needle insertion and direction: Place a skin wheal of local anesthetic solution 2 cm above the midclavicle. Insert a 25 gauge needle directed caudally through the skin wheal (**Figures 156-16*B* and 156-16*C***). Advance the needle until the patient experiences paresthesias. Withdraw the needle 1 mm and allow the paresthesias to resolve. Aspirate to ensure that the tip of the needle is not within a blood vessel. Inject 40 mL of local anesthetic solution. Reduce the volume based on the patient's body size and the maximal allowable dose to prevent toxicity.

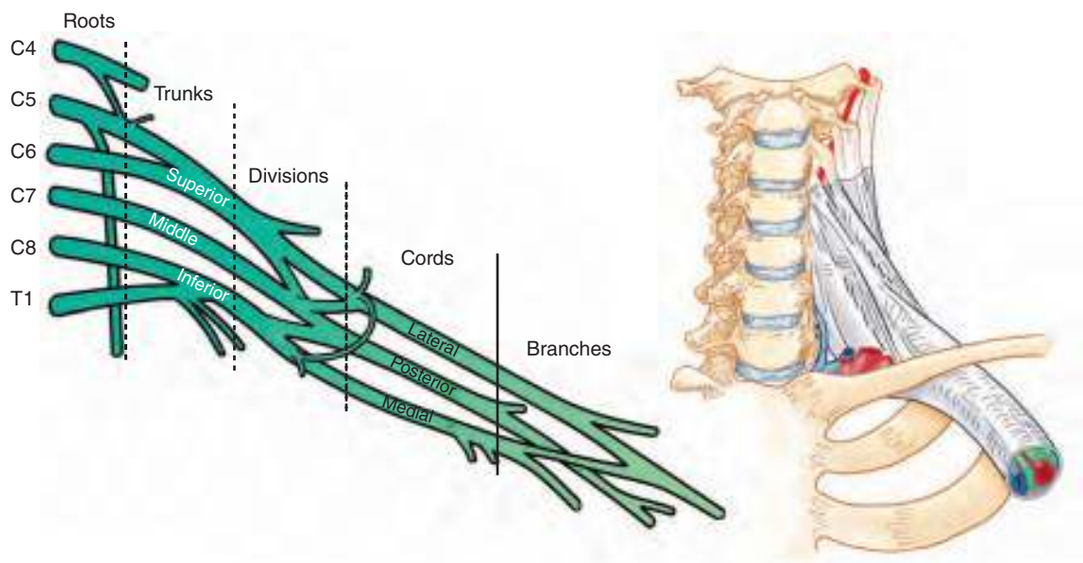


FIGURE 156-15. The brachial plexus. ***A.*** The anatomy of the brachial plexus. ***B.*** The brachial plexus is contained within a sheath. The subclavian artery and vein enter the sheath at the level of the clavicle.

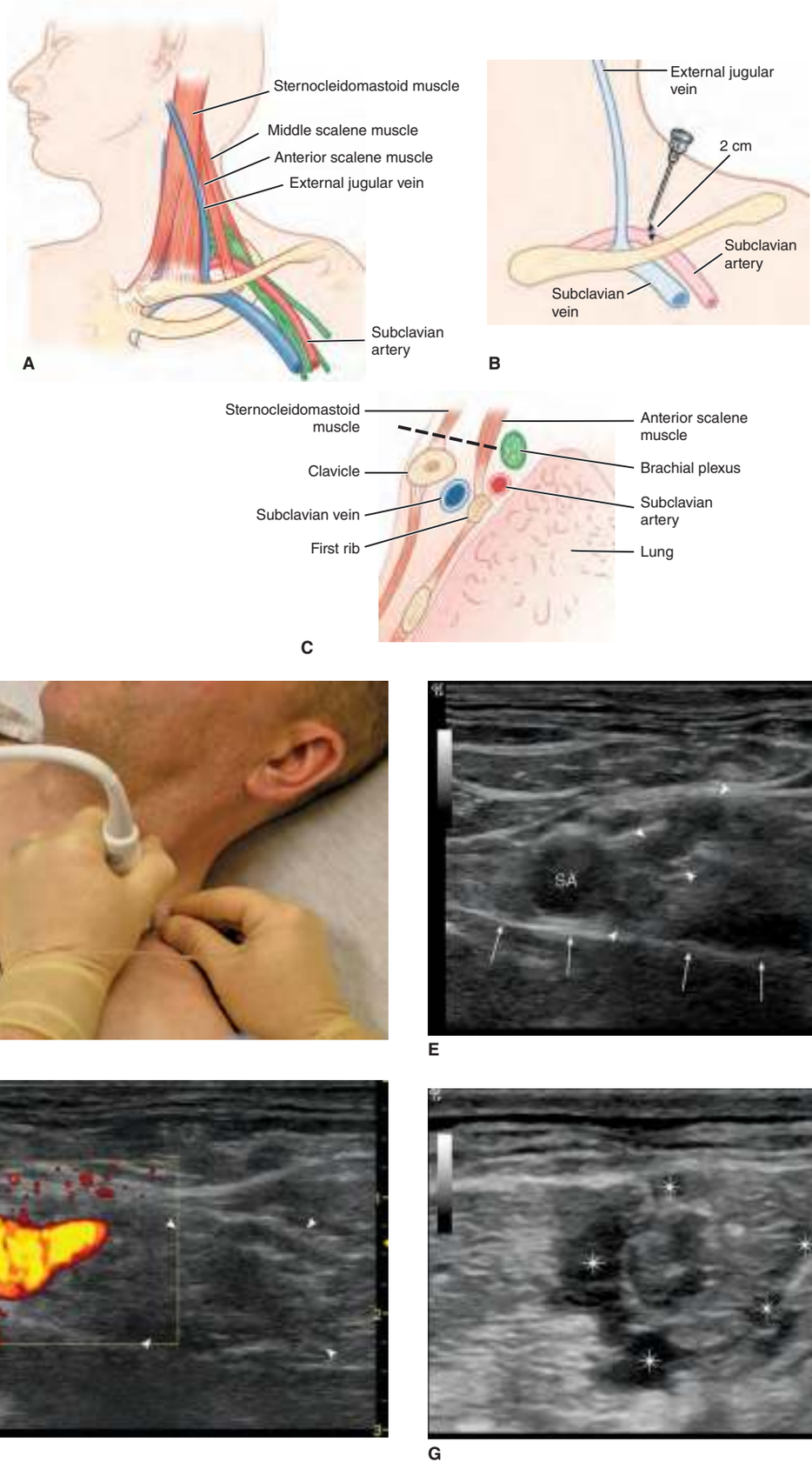


FIGURE 156-16. The supraclavicular approach to the brachial plexus block. ***A.*** The course of the brachial plexus. ***B.*** The needle is inserted perpendicular to the skin and 2 cm superior to the middle of the clavicle. ***C.*** Sagittal section demonstrating the trajectory of the needle (*dotted line*). ***D.*** US transducer and needle placement. ***E.*** The supraclavicular brachial plexus (*arrowheads*) is adjacent to the subclavian artery (SA). The first rib is denoted by the *arrows*. ***F.*** Color Doppler of the supraclavicular brachial plexus (*arrowheads*). The subclavian artery and a branch are visible in color. ***G.*** The "donut sign." Local anesthetic solution (*) surrounds the nerve bundles.

US-guided block: This nerve can be blocked with the aid of US.[33] Place the US transducer in an oblique coronal plane above the clavicle and lateral to the sternocleidomastoid muscle (**Figure 156-16*D***). Locate the subclavian artery. It is a pulsatile, round, and hypoechoic structure in cross-section. The trunks of the brachial plexus are located adjacent to the subclavian artery (**Figure 156-16*E***). Posterior to the subclavian artery lies the first rib and the pleural line can be seen sliding in real time. **Use color/power Doppler to confirm the location of the subclavian artery and any branches or take-offs, all of which must be avoided (Figure 156-16*F*).** Anesthetize the skin. Position the spinal needle lateral to the US transducer and parallel to its long axis. Slowly insert and advance the needle. Visualize the entire length of the needle as it is introduced through the subcutaneous tissue toward the brachial plexus. Advance the needle through the nerve sheath. Aspirate to ensure the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of anesthetic around the nerves. If it is satisfactory, inject the remainder of the local anesthetic solution to achieve the "donut sign" (**Figure 156-16*G***). If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: This block is characterized by a quick onset of anesthesia and a complete block. A high volume of anesthetic is required with a quick onset of anesthesia. There is no chance of missing peripheral or proximal nerve branches because of failure of the local anesthetic solution to spread along the sheath of the brachial plexus. Unfortunately, this technique is difficult to teach and to master without considerable experience. This technique has a high incidence of an iatrogenic pneumothorax, reportedly up to 6%. Other complications include blockade of the phrenic nerve, subclavian artery puncture, and Horner's syndrome. Unintentional intravascular injection can result in high blood levels of the local anesthetic agent.

BRACHIAL PLEXUS BLOCK, INTERSCALENE APPROACH

Anatomy: The anatomy of the brachial plexus is described above. This approach blocks the brachial plexus at the level of the trunks (**Figure 156-17*A***).

Patient positioning: Place the patient supine with their head turned 45° from midline and toward the side opposite that being anesthetized (**Figure 156-17*A***). Place the ipsilateral arm in any position of comfort for the patient.

Landmarks: Identify the posterior border of the clavicular head of the sternocleidomastoid muscle by palpation. Move the palpating finger laterally until it rolls into the interscalene groove between the anterior and middle scalene muscles at the level of the cricoid cartilage (**Figure 156-17*B***).

Needle insertion and direction: Place a skin wheal of local anesthetic solution in the interscalene groove at the level of the cricoid cartilage. Slowly insert a 25 gauge needle through the skin wheal in a dorsal, medial, and caudal direction (**Figure 156-17*B***). Advance the needle until the patient experiences paresthesias. Withdraw the needle 1 mm and allow the paresthesias to resolve. Aspirate to ensure that the tip of the needle is not within a blood vessel. Inject 30 to 40 mL of local anesthetic solution. Reduce the volume based on the patient's body size and the maximal allowable dose to prevent toxicity.

US-guided block: The brachial plexus can be blocked by performing the interscalene block with the aid of US.[34,35] This block is performed at the level of the internal jugular vein and carotid artery in the neck. Place the US transducer on the neck less than a third of the way up from the clavicle (**Figure 156-17*C***). Identify the carotid artery and internal jugular vein. Move the US transducer laterally to find the muscle bellies of the anterior and middle scalene muscles (**Figure 156-17*D***). Between the muscles lie the nerve roots of the brachial plexus in cross-section. It is represented by hypoechoic circles within the hyperechoic rings of the nerve sheaths (**Figure 156-17*D***). **Use color Doppler to identify any blood vessels in the field and note their location so as to avoid them.** Anesthetize the skin. Using a posterior approach, insert the spinal needle connected by extension tubing to a 20 mL syringe filled with local anesthetic solution. Advance the needle and visualize it approaching the brachial plexus. The needle path will usually go through the middle scalene muscle. Once the brachial plexus is close to the needle tip, use a short and controlled jab to penetrate through the nerve sheath. Aspirate to verify that the needle tip is not in a blood vessel. Instruct an assistant to deliver a test dose of 1 to 2 mL of local anesthetic solution. If the anesthetic spreads around the nerves, slowly deliver the remainder of the local anesthetic solution to obtain the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: The advantages and disadvantages are similar to those of the supraclavicular approach with the exception of possibly not achieving anesthesia of the lower trunk. This may require supplementary blockade of the median and ulnar nerves. The interscalene brachial plexus block, although ideal for regional anesthesia of the shoulder, has been associated with recurrent laryngeal nerve paralysis and an almost 100% incidence of phrenic nerve paralysis.[36] This can be significant in patients with respiratory comorbidities (e.g., chronic obstructive pulmonary disease, obesity).[37] The supraclavicular brachial plexus block is associated with lower rates of these complications.[3]

BRACHIAL PLEXUS BLOCK, AXILLARY APPROACH

Anatomy: The anatomy of the axillary brachial plexus nerve block is rather simple. The neurovascular bundle is easily found at the anterior axillary fold by palpating for the pulsations of the axillary artery. The neurovascular bundle is surrounded by the fibrous axillary sheath (**Figure 156-18*A***). The axillary sheath is bound medially by skin and connective tissue, anteriorly by the biceps and coracobrachialis muscles, inferiorly by the triceps muscle, and laterally by the neck of the humerus. The axillary artery is the central reference structure within the neurovascular bundle. Within the axillary sheath the median nerve is anterior, the radial nerve is posterolateral, and the ulnar nerve is posterior to the axillary artery (**Figure 156-18*A***). The axillary vein is medial to the artery. The medial antebrachial cutaneous nerve and the medial brachial cutaneous nerve are medial to the artery (**Figure 156-18*A***). The only sensory nerve outside the neurovascular bundle is the musculocutaneous nerve. This nerve exits the axillary sheath as it crosses the clavicle.

Patient positioning: Place the patient supine with their head turned toward the side opposite that being anesthetized (**Figure 156-18*B***). Abduct the arm 90°. Flex the elbow 90° so that the forearm is parallel to the long axis of the body and the palm is facing upward.

Landmarks: Identify the brachial artery by its palpable pulse. Trace it proximally to the anterior axillary fold formed by the pectoralis major muscle. Use the index and middle fingers of the nondominant hand to secure the neurovascular bundle (identified by the pulse) against the humerus (**Figure 156-18*B***).

Needle insertion and direction: Place the skin wheal of local anesthetic solution overlying the axillary artery pulse just posterior to the anterior axillary fold. Insert a 22 gauge spinal needle just above the fingertip on the axillary pulse, directed toward the apex of the axilla, and in the direction of the neurovascular bundle (**Figure 156-18*B***). Advance the needle. A "pop" will be felt as the axillary sheath is penetrated. The correct needle position is

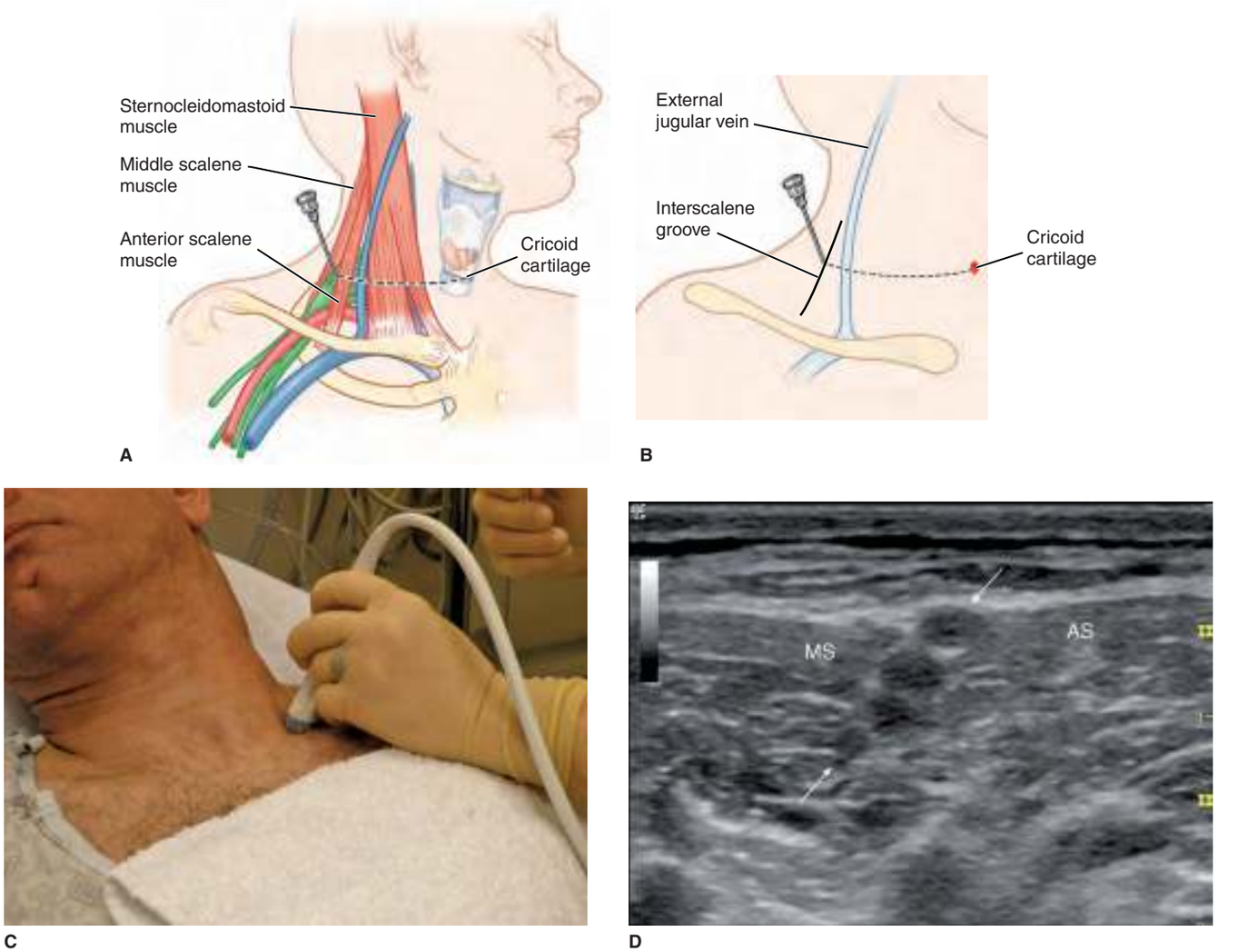


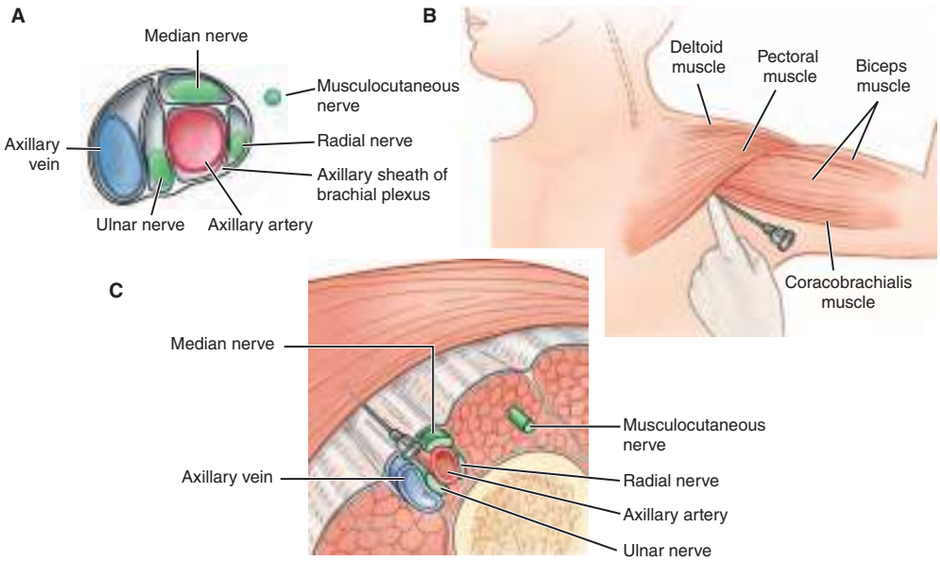
FIGURE 156-17. The interscalene approach to the brachial plexus block. ***A.*** The anatomy of the region. ***B.*** The needle is inserted into the interscalene groove at the level of the cricoid cartilage. ***C.*** US transducer placement. ***D.*** The brachial plexus (between the *arrows*) is located between the anterior scalene (AS) and the middle scalene (MS) muscles.

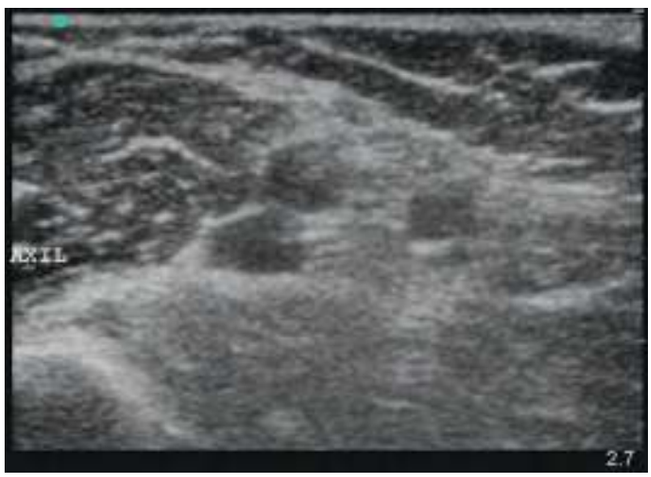
confirmed by eliciting paresthesias, observing blood flow in the needle, or observing pulsations of the needle that match the pulse. Instruct an assistant to attach the distal end of intravenous extension tubing to the hub of the needle and the proximal end to a 60 mL syringe containing local anesthetic solution. The Emergency Physician must always maintain pressure against the neurovascular bundle with the nondominant hand while stabilizing the needle with the dominant hand. Instruct the assistant to aspirate to ensure that the tip of the needle is not within a blood vessel. Withdraw the needle 2 mm if blood flow or paresthesias are elicited.

Apply digital pressure to the neurovascular bundle just distal to the tip of the needle with the nondominant fingers. This prevents the local anesthetic solution from flowing distally. Inject the local anesthetic solution into the axillary sheath after the paresthesias have resolved and a negative aspiration has been achieved. Instruct the assistant to inject a volume of approximately 40 mL in the adult patient. This volume has been shown to consistently block the entire brachial plexus. Reduce the volume based on the patient's body size and the maximal allowable dose to prevent toxicity. **Continue to apply digital pressure to the neurovascular bundle just distal to the needle during and after injection of the local anesthetic solution.**

Withdraw the needle while the assistant simultaneously injects 3 to 5 mL of local anesthetic solution into the subcutaneous tissue. This blocks the medial brachial cutaneous nerve and the intercostobrachial nerve. Abduct the patient's arm 30° to 45° after the needle is withdrawn. Maintain this position while continuing to apply digital pressure to the neurovascular bundle just distal to the needle insertion site, for 2 to 3 minutes.

US-guided block: The brachial plexus can be blocked by performing the axillary block with the aid of US. This block is performed at the level of the terminal branches of the brachial plexus within the axillary sheath. These branches are visualized as hyperechoic nodules around the pulsatile and hypoechoic axillary artery (**Figures 156-18*D* and 156-18*E***). Place the US transducer with the marker pointing cephalad at the superior axillary crease (**Figure 156-18*F***). Locate the axillary artery and vein in cross-section (**Figure 156-18*G***). The musculocutaneous nerve can be seen between the biceps muscle and the coracobrachialis muscle. Use color Doppler to confirm the location of the axillary artery, vein, and any branches or take-offs (**Figure 156-18*G***). Anesthetize the skin. Position the spinal needle inferior or superior to the long axis of the US transducer (**Figure 156-18*F***). Slowly insert and advance the needle. Visualize the entire length of the needle as it is advanced. Aim the needle between the axillary artery and vein.





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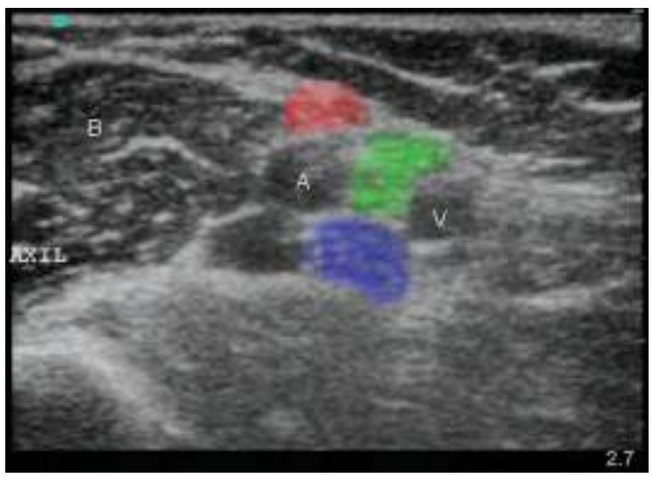

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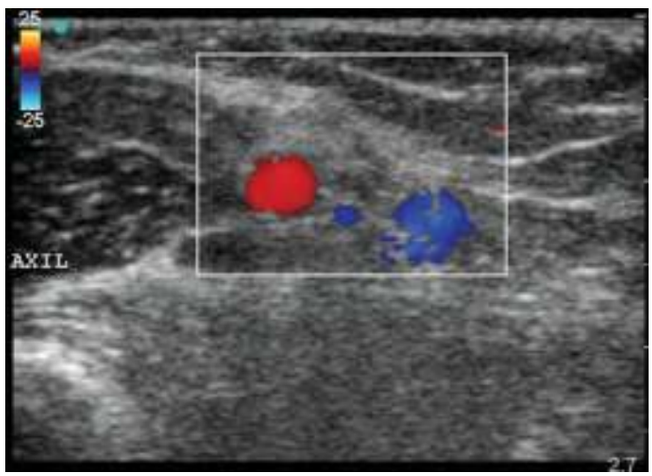

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FIGURE 156-18. The axillary approach to the brachial plexus block. ***A.*** The topographical arrangement of the contents of the axillary sheath at the level of the blockade. ***B.*** The patient is positioned and the axillary artery pulse is palpated with one finger. The needle is inserted above the pulse and along the course of the axillary sheath. ***C.*** The needle may travel above the sheath and enter it at a higher level. ***D.*** US image of the axillary brachial plexus. ***E.*** Same image as in part ***D*** with labels (A, axillary artery; B, biceps muscle; V, axillary vein; red oval, median nerve; green oval, ulnar nerve; blue oval, radial nerve). ***F.*** US transducer and needle placement. ***G.*** Color Doppler of the axillary artery (red) and vein (blue).

Advance through the nerve sheath. Aspirate to ensure the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the subclavian artery. If it is satisfactory, inject the remainder of the local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: The axillary approach to the brachial plexus is the most commonly used and preferred technique. The procedure is easily mastered, has no major complications, and is easily performed in the obese patient. The disadvantages of this technique include insufficient anesthesia of the shoulder and upper arm. The musculocutaneous nerve provides sensory innervation to the radial aspect of the forearm and may be missed by the local anesthetic agent. The subcutaneous infiltration of local anesthetic solution usually blocks the musculocutaneous nerve. **Proximal flow of the local anesthetic solution is required to ensure adequate anesthesia.** Abduction of the arm while maintaining pressure on the neurovascular bundle allows proximal flow of the local anesthetic solution. It also prevents the humeral head from limiting proximal spread due to compression of the brachial plexus.

BRACHIAL PLEXUS BLOCK, INFRACLAVICULAR APPROACH

This approach to the brachial plexus has many advantages and few serious complications. Unfortunately, this technique requires considerable experience on the part of the Emergency Physician and a nerve stimulator to locate the brachial plexus (**Figures 156-19*A* and 156-19*B***). For these reasons, the anatomic landmark approach will not be described. The US-guided technique is described below.

Anatomy: The anatomy of the brachial plexus is described above. This approach blocks the brachial plexus at the level of the cords (**Figure 156-17*A***).

Patient positioning: Place the patient supine with their head turned 45° from midline and toward the side opposite that being anesthetized (**Figure 156-19*A***). Place the ipsilateral arm extended 90°.

Landmarks: Identify the middle third of the clavicle.

Infraclavicular Block. The brachial plexus can be blocked by performing the infraclavicular block with the aid of US.[36] This block is performed below the clavicle. The cords of the brachial plexus lie below the pectoralis major and minor muscles. They appear as distinct hyperechoic nodules positioned laterally, medially, and posteriorly around the subclavian artery (**Figures 156-19*C* and 156-19*D***). Prep and drape the infraclavicular region. Place the US transducer with the marker pointing cephalad below the clavicle, medial to the coracoid process (**Figure 156-19*E***). Locate the subclavian artery (which is pulsatile) and vein in cross-section. Use color Doppler to confirm the location of the subclavian artery, vein, and any branches or take-offs (**Figure 156-19*F***). Anesthetize the skin. Position the spinal needle inferior or superior to the long axis of the US transducer (**Figure 156-19*E***). Insert and advance the needle. Visualize the entire length of the needle as it is advanced. Aim the needle between the subclavian artery and vein. Advance the needle. Advance the needle through the nerve sheath. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the subclavian artery. If satisfactory, inject the remainder of the local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: This block can result in an iatrogenic pneumothorax due to the proximity of the needle to the lung apex.

MEDIAN NERVE BLOCK, AT THE ELBOW

Anatomy: The median nerve innervates all the muscles of the anterior forearm except the flexor digitorum profundus to the ring and little fingers and the flexor carpi ulnaris.[13,37,38] It innervates the thenar muscles and the lumbrical muscles to the index and middle fingers in the hand. It provides sensory innervation to the palmar aspect of the thumb, index finger, middle finger, radial portion of the ring finger, and lateral half of the palm (**Figure 156-20*A***). The median nerve provides a variable amount of sensory innervation to the dorsal distal surfaces of the lateral three and one-half fingers.[13]

Patient positioning: Place the patient supine with their arm abducted 45°, the elbow in full extension, and the hand supinated (**Figure 156-21*A***).

Landmarks: Identify the medial and lateral epicondyle of the humerus by palpation. Connect the epicondyles with a straight line (**Figure 156-21*A***). Identify the brachial artery by palpating for its pulse just medial to the biceps tendon and over the line just drawn. Mark the site of the palpable pulse with a marker.

Needle insertion and direction: Place a skin wheal of local anesthetic solution just medial to the pulse at the level of the

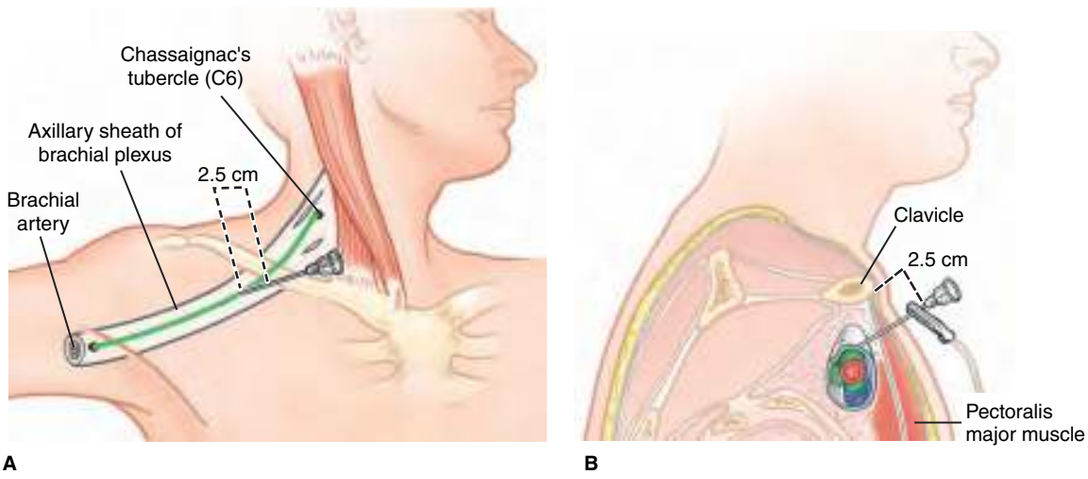

FIGURE 156-19. The infraclavicular approach to the brachial plexus block. ***A.*** Needle insertion and direction. ***B.*** Sagittal section demonstrating needle insertion into the neurovascular bundle. The alligator clip is attached to a nerve stimulator. ***C.*** US image of the infraclavicular brachial plexus. ***D.*** Same image as in part ***C*** with labels (A, subclavian artery; V, subclavian vein; red oval, lateral cord; green oval, medial cord; blue oval, posterior cord). ***E.*** US transducer and needle placement. The clavicle is located between the two red lines. ***F.*** Color Doppler of the subclavian artery (red) and vein (blue).

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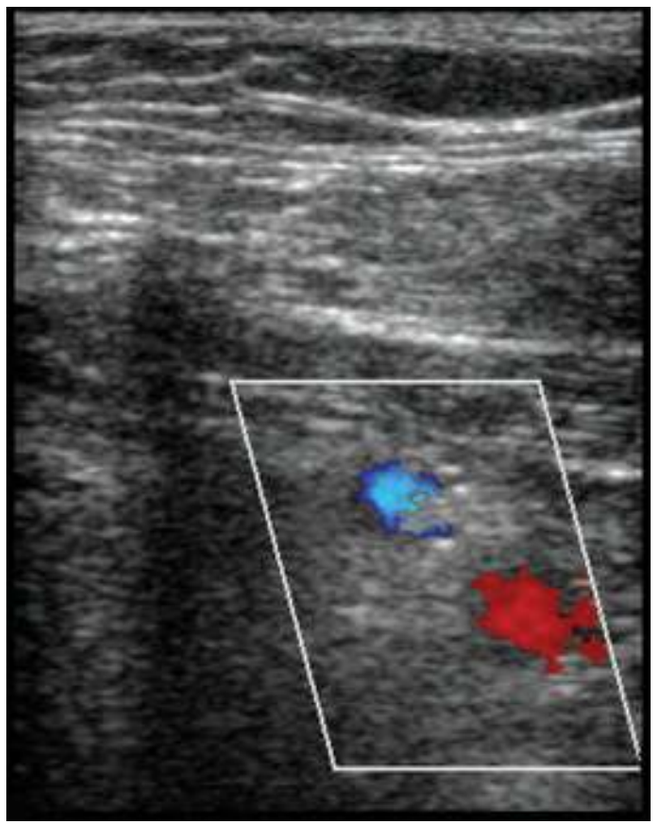

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FIGURE 156-19. (*Continued*)

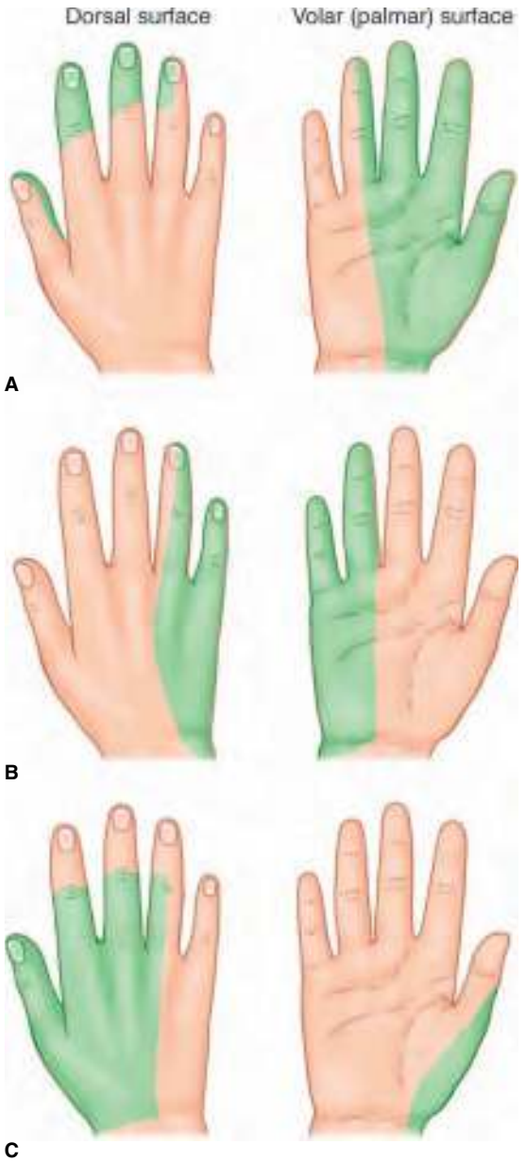


FIGURE 156-20. The sensory innervation of the hand. ***A.*** The median nerve. ***B.*** The ulnar nerve. ***C.*** The radial nerve.

intercondylar line. Insert a 25 gauge needle perpendicular to the skin and slowly advance it. If paresthesias are elicited, withdraw the needle 1 to 2 mm and allow the paresthesias to resolve. Inject 3 to 5 mL of local anesthetic solution. If paresthesias are not elicited, slowly move the needle in a fan-like pattern to elicit paresthesias. Withdraw the needle 1 to 2 mm, allow the paresthesias to resolve, and inject 3 to 5 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US. Place the US transducer in the crease of the antecubital fossa. Locate the pulsatile brachial artery in the middle of the antecubital fossa (**Figure 156-21*B***). Anesthetize the skin medial to the brachial artery. Place the spinal needle inferior to the short axis of the US transducer directly over the median nerve which should be medial to the brachial artery. Only the tip of the needle will be visualized in this view. Insert and advance the needle until its tip is at the median nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of the local anesthetic solution around the median nerve. If satisfactory, inject the remainder of the local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: The median nerve has no sensory branches in the forearm. Therefore, there is no advantage to blocking the median nerve at the elbow. Inserting the needle to elicit paresthesias can be quite painful for the patient. Blockade at the wrist is usually easier to perform, especially in obese patients.[10]

MEDIAN NERVE BLOCK, AT THE WRIST

Anatomy: The median nerve lies in the carpal tunnel on the volar aspect of the wrist. It is located between the tendons of the flexor carpi radialis and palmaris longus muscles (**Figure 156-21*C***). The innervation of the median nerve is described in the previous section.

Patient positioning: Place the patient supine with their arm abducted 45°, the elbow in full extension, and the hand fully supinated (**Figure 156-21*A***).

Landmarks: Identify the palmaris longus tendon by flexing the patient's clenched hand against resistance. Mark the radial border of the palmaris longus tendon. Note the position of the proximal and distal wrist creases.

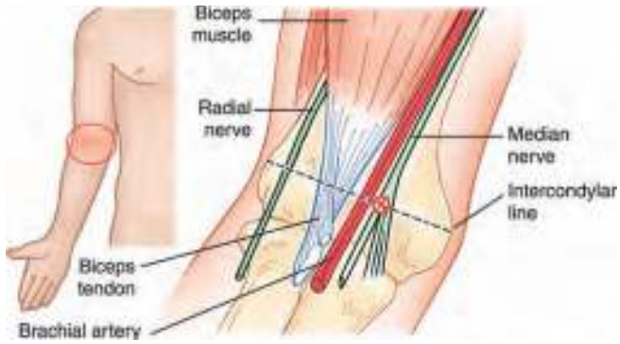
Needle insertion and direction: Place a skin wheal of local anesthetic solution along the radial border of the palmaris longus tendon between the proximal and distal wrist creases. Insert a 25 gauge needle perpendicular to the skin wheal and advance it slowly (**Figure 156-21*C***). If paresthesias are elicited, withdraw the needle 1 to 2 mm and allow them to resolve. Inject 3 to 5 mL of local anesthetic solution. If paresthesias are not elicited, inject 5 to 10 mL of local anesthetic solution. Injection of the local anesthetic solution should not raise a skin wheal and should flow effortlessly if the needle is within the carpal tunnel.[13]

US-guided block: The nerve can be blocked with the aid of US.[39] Place the US transducer over the middle of the wrist with the marker pointing laterally (**Figure 156-21*D***). The palmaris longus tendon should be visible in the middle of the wrist. The median nerve is hyperechoic and directly lateral to this tendon (**Figure 156-21*E***). The radial artery is lateral to the median nerve (**Figure 156-21*C***). Anesthetize the skin lateral to the palmaris longus tendon. Place the needle inferior to the short axis of the US transducer and directly over the median nerve (**Figure 156-21*D***). Insert and advance the needle until its tip is at the median nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of the local anesthetic solution around the median nerve. If satisfactory, inject the remainder of the local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: A small percentage of the population (10% to 15%) does not have a palmaris longus tendon.[40] Identify the flexor carpi radialis tendon by the same method as identifying the palmaris longus tendon. Inject the local anesthetic solution 1 cm medial to the ulnar edge of the flexor carpi radialis tendon between the proximal and distal wrist creases (**Figure 156-21*C***). The technique is otherwise as noted above.

ULNAR NERVE BLOCK, AT THE ELBOW

Anatomy: The ulnar nerve lies in the ulnar groove of the humerus at the elbow between the olecranon process and medial condyle of the humerus (**Figure 156-22*A***). It provides motor innervation to the flexor carpi ulnaris, the ring and little finger portion of the flexor digitorum profundus, the palmaris brevis, the hypothenar muscles, the third and fourth lumbricals, the interossei, and the adductor pollicis muscles. It provides sensory innervation to the medial one-third to one-half of the palm, the palmar aspect of the ulnar half of the ring finger, and the entire little finger (**Figure 156-20*B***). The ulnar nerve



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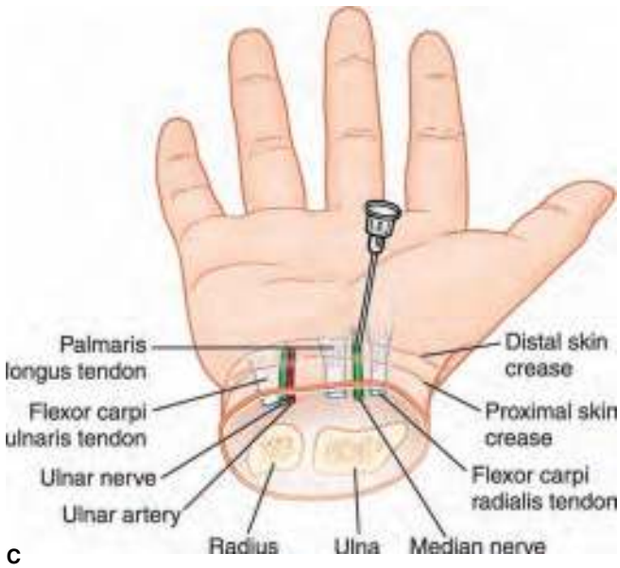

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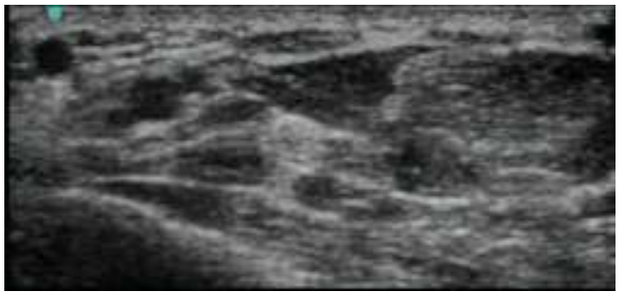
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FIGURE 156-21. The median nerve block. ***A.*** Blockade at the level of the elbow. ***B.*** US image of the median nerve at the antecubital fossa. ***C.*** Blockade at the level of the wrist. ***D.*** US transducer and needle placement at the wrist. ***E.*** US image of the volar wrist. The median nerve is the round, hyperechoic structure in the center of the image.

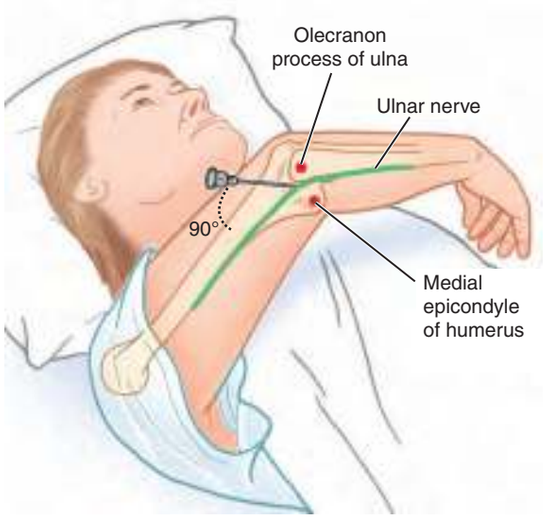
provides sensory innervation to the dorsomedial half of the hand, the little finger, and the ulnar half of the ring finger on the dorsal surface of the hand.[41]

Patient positioning: Place the patient supine with their elbow flexed 90° and the shoulder flexed 45° (**Figure 156-22*A***).

Landmarks: Identify the olecranon process and the medial epicondyle of the humerus by palpation. Palpate the groove between the olecranon process and the medial epicondyle. The ulnar nerve is located within this groove.

Needle insertion and direction: Place a skin wheal of local anesthetic solution 1 to 2 cm proximal to the ulnar groove. Insert a 25 gauge needle through the skin wheal and directed toward the ulnar groove. Aim the needle parallel to the ulnar groove and the course of the nerve (**Figure 156-22*A***). Advance the needle into the ulnar groove and inject 5 to 7 mL of local anesthetic solution. If paresthesias are elicited, withdraw the needle 1 mm and allow them to resolve before injecting the local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US. Place the US transducer in the crease between the olecranon process and the medial epicondyle with the marker pointing laterally. Two hyperlucent structures should be seen, the medial epicondyle medially and the olecranon process closer to the marker. The ulnar nerve is hyperechoic and courses between these two hyperlucent structures. Anesthetize the skin. Place the needle inferior to the short axis of the



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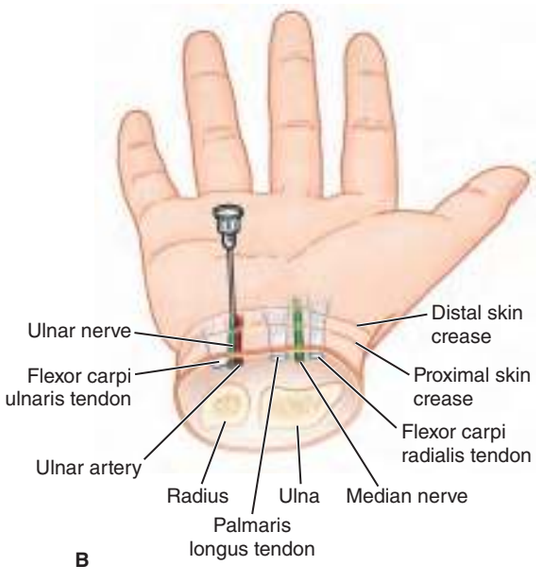

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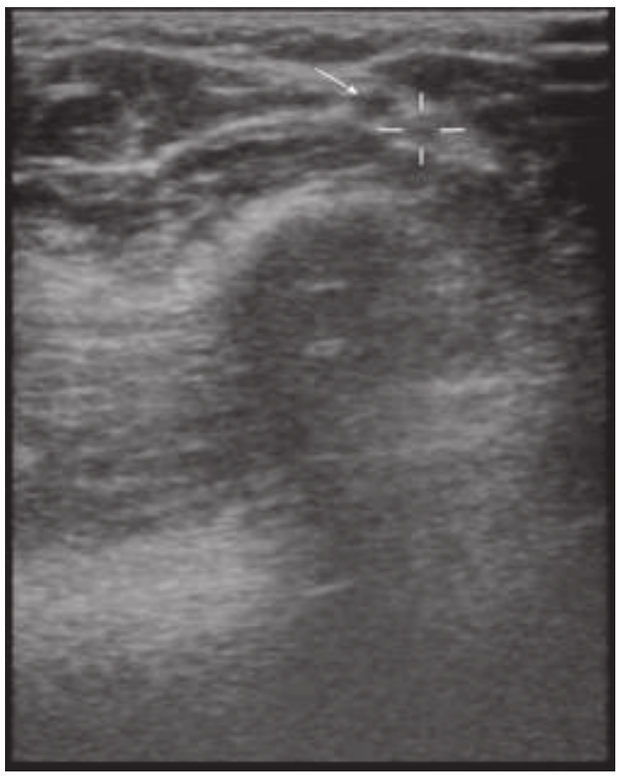

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FIGURE 156-22. The ulnar nerve block. ***A.*** Blockade at the level of the elbow. ***B.*** Blockade at the level of the wrist. ***C.*** US transducer and needle placement at the wrist. ***D.*** US image of the volar wrist. The ulnar artery (*arrow*) is lateral to the hyperechoic ulnar nerve (in the crosshairs).

US transducer and directly over the ulnar nerve. Only the tip of the needle will be visualized in this view. Insert and advance the needle until its tip is at the ulnar nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the ulnar nerve. If satisfactory, inject the remainder of the local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: The ulnar nerve has no sensory branches in the forearm and thus may be blocked at the wrist or at the elbow. Blockade of the ulnar nerve at the elbow is not recommended. A fibrous sheath surrounds the ulnar nerve at the elbow requiring intraneural injection for a successful blockade. This can lead to residual neuritis and nerve dysfunction. Blocking the ulnar nerve several centimeters above the elbow may prevent these complications. Blockade at the wrist is very reliable and does not have the associated complications as at the elbow.

ULNAR NERVE BLOCK, AT THE WRIST

Anatomy: The ulnar nerve lies between the distal and proximal flexor skin creases of the wrist, lateral (i.e., radial) to the flexor carpi ulnaris tendon and medial (i.e., ulnar) to the ulnar artery (**Figure 156-22*B***). The innervation of the ulnar nerve is described in the previous section.

Patient positioning: Place the patient supine with their arm abducted 45° to 90°, the elbow fully extended, and the hand supinated (**Figure 156-22*B***).

Landmarks: Identify the flexor carpi ulnaris tendon by flexing the patient's clenched hand against resistance. Mark the medial aspect of the flexor carpi ulnaris tendon. Identify the ulnar artery by its palpable pulsations between the proximal and distal wrist crease. Note the position of the proximal and distal wrist creases.

Needle insertion and direction: Place a skin wheal of local anesthetic solution in the quadrangle defined by the proximal flexor skin crease, the distal flexor skin crease, the lateral aspect of the flexor carpi ulnaris tendon, and medial to the ulnar artery. Insert a 25 gauge needle perpendicular to the skin wheal and slowly advance it 0.5 cm (**Figure 156-22*B***). If paresthesias are elicited, withdraw the needle 2 mm and allow them to resolve. Inject 2 mL of local anesthetic solution once the paresthesias resolve. If paresthesias are not elicited, inject 3 to 5 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US.[39] Place the US transducer over the middle of the wrist with the marker pointing laterally (**Figure 156-22*C***). The palmaris longus tendon should be visible in the middle of the wrist. Move the US transducer medially until the ulnar artery is visible. The ulnar nerve is hyperechoic and directly medial to the ulnar artery (**Figure 156-22*D***). Anesthetize the skin medial to the ulnar artery. Position the needle inferior to the short axis of the US transducer and directly over the ulnar nerve (**Figure 156-22*C*).** Insert and advance the needle until its tip is at the ulnar nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the ulnar nerve. If satisfactory, inject the remainder of the local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: This is the preferred approach to block the ulnar nerve. Blockade of the ulnar nerve at the wrist is very reliable and does not have the associated complications as at the elbow.[10,39,40]

RADIAL NERVE BLOCK, AT THE ELBOW

Anatomy: The radial nerve and the sensory branch of the musculocutaneous nerve run together in the sulcus between the biceps and the brachioradialis muscle on the anterolateral aspect of the elbow. The radial nerve provides sensory innervation to portions of the dorsal arm and forearm, the dorsolateral half of the hand, and the dorsal proximal aspects of the thumb, index, middle, and radial half of the ring fingers (**Figure 156-20**).[13] It provides motor innervation to the muscles on the posterior aspect of the arm, forearm, and hand.

Patient positioning: Place the patient supine with their elbow flexed 15° to 30°.

Landmarks: Palpate the tendon of the biceps muscle in the antecubital fossa. Identify the flexion skin crease of the elbow. Palpation of the biceps tendon is greatly facilitated by having the patient flex their elbow 90° then contract and relax their biceps muscle. Identify the medial and lateral condyles of the humerus. Draw a line between the humeral condyles (**Figure 156-23*A***). This line should be located at the level of the antecubital crease.

Needle insertion and direction: Place a skin wheal of local anesthetic solution 2 cm lateral to the biceps tendon and 1 cm proximal to the antecubital crease (**Figure 156-23*A***). Insert a 25 gauge needle through the skin wheal and perpendicular to the skin (**Figure 156-23*A***). Advance the needle 1 to 2 cm. Move the transducer in a fan-like pattern until paresthesias are elicited. Withdraw the needle 1 to 2 mm and allow the paresthesias to resolve. Inject 5 to 7 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US. Position the patient with their arm abducted 45°, the elbow in full extension, and the hand supinated. Place the US transducer on the lateral aspect of the crease of the antecubital fossa. The radial nerve is lateral to the biceps tendon and the brachial artery (**Figure 156-23*B***). Anesthetize the skin over the radial nerve. Place the needle inferior to the short axis of the US transducer and directly over the radial nerve. Only the tip of the needle will be visualized in this view. Insert and advance the needle until its tip is at the radial nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the radial nerve. If satisfactory, inject the remainder of the local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

The radial nerve can be blocked above the humeral condyle using US.[42,43] Flex the patient's elbow 90° with the hand pronated. Once the radial nerve is found, the technique is similar to the above, only more proximal on the arm.

Remarks: Blockade of the radial nerve at the elbow is difficult, has limited applications, is painful for the patient, and often results in a large antecubital hematoma. The preferred technique is blockade at the wrist.

RADIAL NERVE BLOCK, AT THE WRIST

Anatomy: The radial nerve at the wrist consists of a trunk and terminal branches that arise in the forearm (**Figure 156-23*C***). The innervation of the radial nerve is described in the previous section.

Patient positioning: Place the patient supine with their arm abducted 45°, the elbow fully extended, and the hand midway between supination and pronation (**Figure 156-23*C***).

Landmarks: Identify the radial artery by its pulsation at the level of the proximal wrist crease.

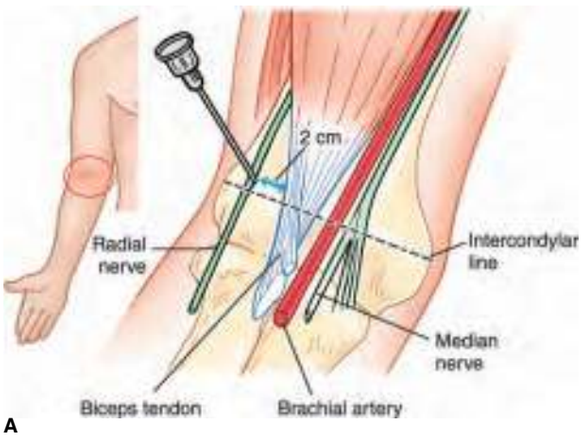
Needle insertion and direction: Place a skin wheal of local anesthetic solution 1 mm lateral to the radial pulse. Insert a 25 gauge needle 1 mm lateral to the radial pulse, through the skin wheal, and perpendicular to the skin (**Figure 156-23*C***). Advance the needle 0.5 cm. If paresthesias are elicited, withdraw the needle 1 to 2 mm and allow them to resolve. Inject 2 mL of local anesthetic solution. If paresthesias are not elicited, inject 3 to 5 mL of local anesthetic solution. This will anesthetize the terminal trunk of the radial nerve. Infiltrate 5 to 7 mL of local anesthetic solution at the level of the extensor wrist crease from the lateral aspect of the radius to the base of the fourth metacarpal (**Figure 156-23*C***). This will anesthetize the terminal branches that arise in the forearm.

US-guided block: The nerve can be blocked with the aid of US.[39] Place the US transducer over the middle of the wrist with the marker pointing laterally (**Figure 156-23*D***). The palmaris longus tendon should be visible in the middle of the wrist. Move the US transducer laterally until the radial artery is visualized. The radial nerve is hyperechoic and directly lateral to the radial artery (**Figures 156-23*E***). Anesthetize the skin lateral to the radial artery and directly over the radial nerve. Place the needle inferior to the short axis of the US transducer and directly over the radial nerve. Insert and advance the needle until its tip is at the radial nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the radial nerve. If satisfactory, inject the remainder of the local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: This is the preferred technique for blockade of the radial nerve.[40]

WRIST BLOCK

Perform the wrist block by blocking the radial, ulnar, and median nerves at the wrist. The technique for each specific nerve block was discussed previously. The wrist block provides complete anesthesia



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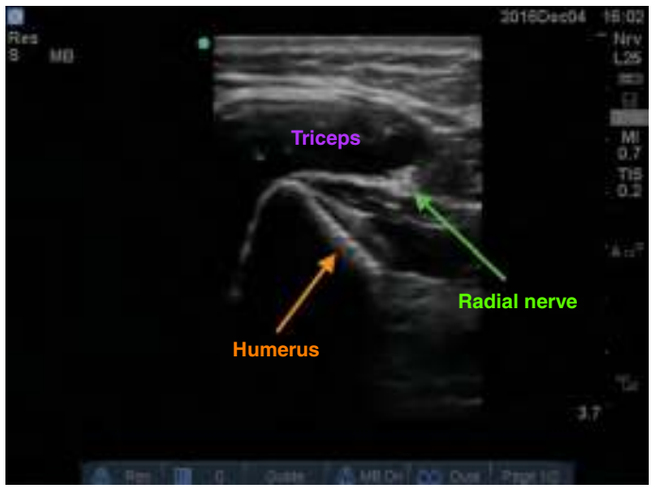

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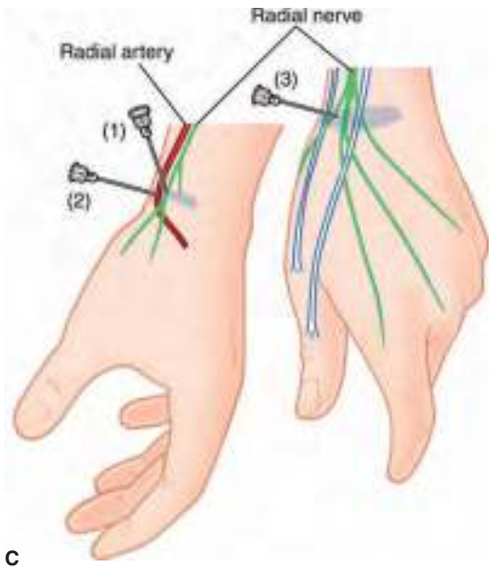

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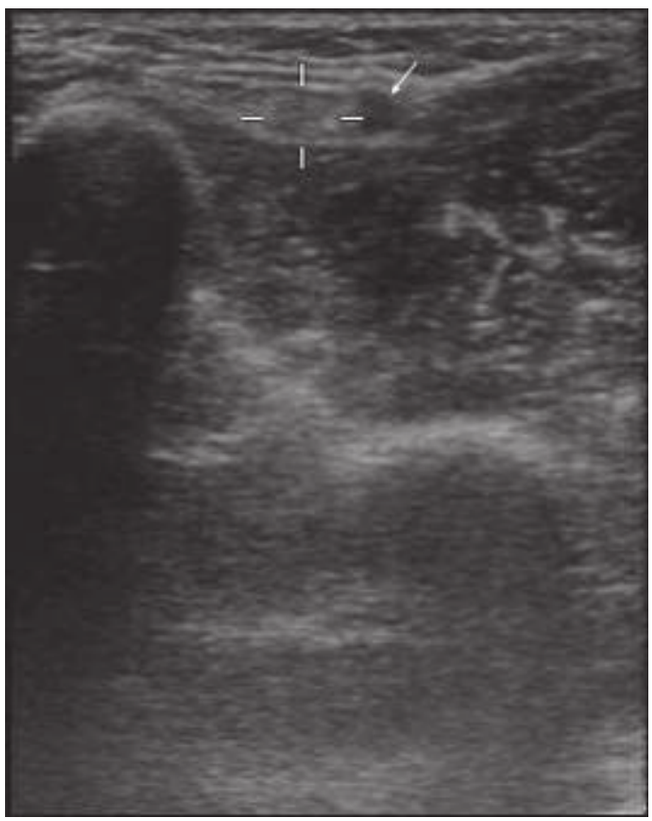

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FIGURE 156-23. The radial nerve block. ***A.*** Blockade at the level of the elbow. ***B.*** US image of the radial nerve at the elbow. ***C.*** Blockade at the level of the wrist. The numbers represent the three injections required to anesthetize the branches of the radial nerve. ***D.*** US transducer and needle placement. ***E.*** US image of the wrist. The radial artery (*arrow*) is adjacent and medial to the radial nerve (in the crosshairs).

to the hand and is commonly used in hand surgery. It can be performed in the Emergency Department to provide anesthesia for burn management, foreign body removal, wound exploration, or extensive laceration repair. Wrist blockade is reliable but slow as it requires extended time to block all three nerves at the wrist.[13,39,40,44]

INTERMETACARPAL NERVE BLOCK

Anatomy: The principal nerves supplying the finger are the palmar digital nerves which originate from the deep volar branches of the ulnar and median nerves in the region of the wrist. These nerves follow the artery along the lateral aspects of the bone and supply sensation to the volar skin, the interphalangeal joints, the distal finger, and the fingertip of all five digits.

Two dorsal and two palmar nerves supply each finger. These nerves run along the phalanxes in the 2, 4, 8, and 10 o'clock positions.[45] These nerves also supply the dorsal, distal aspect of the finger including the fingertip and nail bed. The dorsal digital nerves originate from the radial and ulnar nerves that wrap around the dorsum of the hand. They supply the nail bed of the thumb and small finger and the dorsal aspect of all five digits up to the distal interphalangeal joints. The palmar and dorsal nerves need to be blocked in the case of the thumb and fifth finger.

Patient positioning: Place the patient sitting upright or supine with their hand pronated on a bedside examination table.

Landmarks: Locate the web spaces and the metacarpal heads on each side of the finger to be blocked.

Needle insertion and direction: Insert a 25 gauge needle into the dorsal aspect of the web space on one side of the digit to be anesthetized (**Figure 156-24*A***). Advance the needle approximately 0.5 cm. Inject 1 mL of local anesthetic solution. Repeat the procedure on the other side of the digit to be blocked. When blocking the second and fifth digits, a half-ring block is required on the ulnar aspect of the fifth digit and radial aspect of the second digit. When blocking the thumb, infiltrate the dorsum and sides in a half-ring manner.

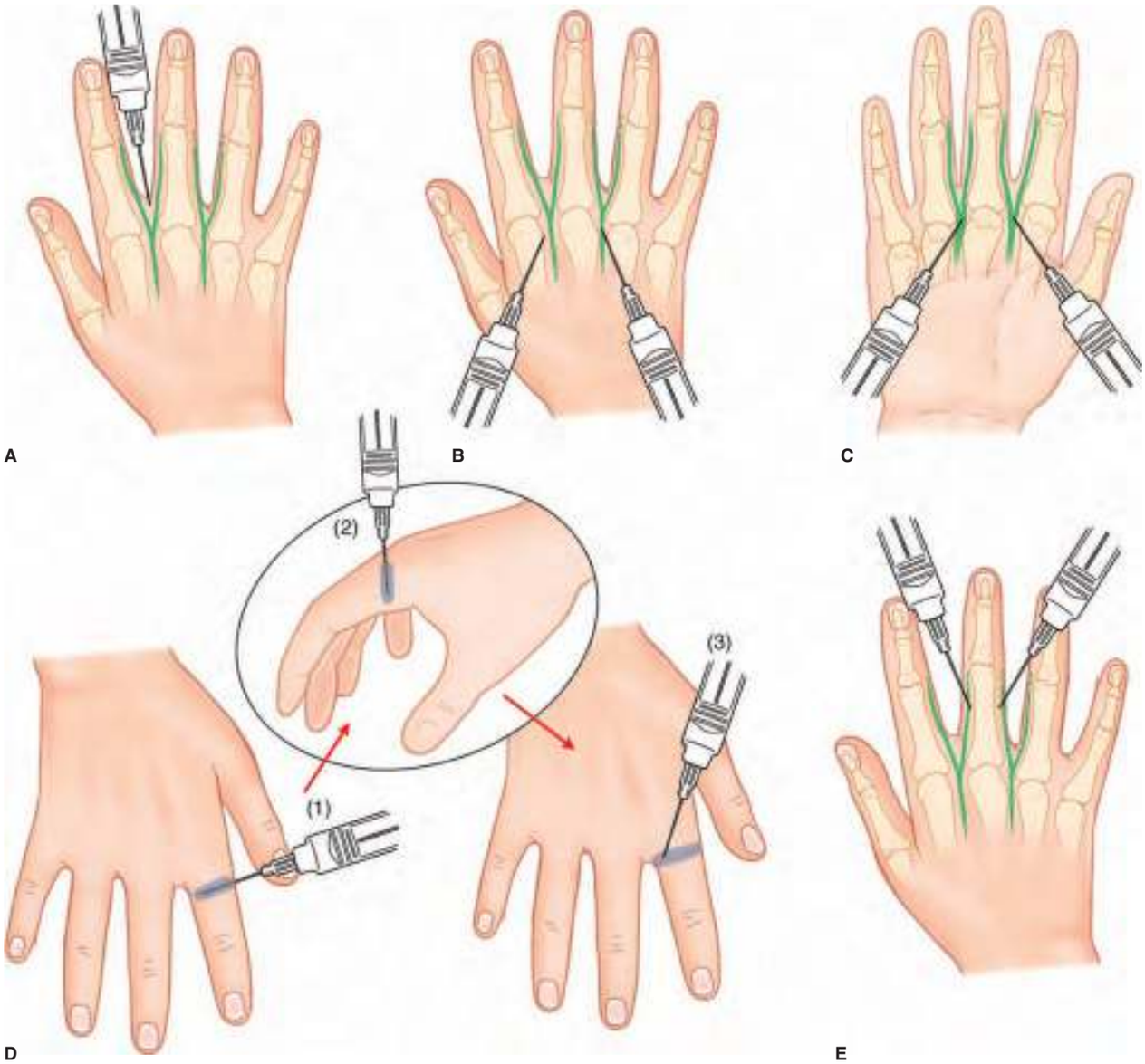


FIGURE 156-24. Techniques to anesthetize the digital nerves of the fingers. ***A.*** Intermetacarpal nerve block through the web space. ***B.*** Intermetacarpal nerve block on the dorsal surface of the hand between the metacarpal heads. ***C.*** Intermetacarpal nerve block on the ventral surface of the hand between the metacarpal heads. ***D.*** The ring block. ***E.*** The half-ring block.

An alternative is the metacarpal head block. This technique can be used to anesthetize any of the fingers. Insert a 25 gauge needle perpendicular to the dorsum of the hand and adjacent to the metacarpal head on one side of the finger to be blocked (**Figure 156-24*B***). Advance the needle 0.5 cm and inject 1 mL of local anesthetic solution. Repeat the procedure on the other side of the finger to be blocked. Some physicians prefer to perform this block on the volar aspect of the hand (**Figure 156-24*C***). This technique is extremely painful and should be avoided.

Remarks: This block produces less swelling than does the ring block. Subsequently, there is less risk of vascular compromise. This is a less painful technique than the ring block. Some use the volar subcutaneous block on top of the tendon sheaths.[46] This block is effective. Some use a block that requires the needle to penetrates the flexor tendon. This cannot be recommended to penetrate the poorly vascular tendon.

DIGITAL NERVE BLOCK (RING BLOCK) OF THE FINGER

Anatomy: The anatomy and innervation of the digital nerves are described in the previous section.

Patient positioning: Place the patient sitting upright or supine with their hand pronated on a bedside examination table.

Landmarks: Locate the dorsum of the proximal phalanx to be anesthetized.

Needle insertion and direction: Insert a 25 gauge needle on the dorsal surface of the base of the proximal phalanx (**Figure 156-24*D-1***). Inject 1 mL of local anesthetic solution along the dorsal surface of the finger. Remove the needle and reinsert it downward, perpendicular to the phalanx and to a depth just past the base of the phalanx (**Figure 156-24*D-2***). Inject 1.0 to 1.5 mL of local anesthetic along the lateral aspect of the finger. Withdraw the needle reinsert it on the other side of the finger to be blocked and inject 1.0 to 1.5 mL of local anesthetic solution (**Figure 156-24*D-3***).

An alternative is the half-ring block (**Figure 156-24*E***). It is a variant of the ring block (**Figure 156-24*D***). Inject 1.0 to 1.5 mL of local anesthetic solution on one side of the base of the proximal phalanx to be anesthetized. Repeat this procedure on the other side of the finger. The injection of local anesthetic on one side of the finger is termed the half-ring block. It takes two half-ring blocks to anesthetize a finger.

Remarks: The indications for a digital block include repair of finger lacerations and amputations, reductions of fractures and dislocations, incision and drainage of infections, removal of fingernails, and relief of pain from burns. Do not inject more than 5 mL of local anesthetic solution into a digit. Using local anesthetic agents that contain epinephrine is controversial because the finger contains end arteries and may experience ischemia from the vasoconstrictive effects of epinephrine. The literature shows that local anesthesia that contains epinephrine is safe to use on the digits.[47-49] The decision to use epinephrine or not use epinephrine in the digits is up to the treating physician. Some use the volar subcutaneous block on top of the tendon sheaths.[46] This block is effective. Some use a block that requires the needle to penetrate the flexor tendon. This cannot be recommended to penetrate the poorly vascular tendon.

REGIONAL ANESTHESIA TECHNIQUES FOR THE TORSO

INTERCOSTAL NERVE BLOCK

Anatomy: The intercostal nerves originate from the thoracic spinal cord and have four major branches (**Figure 156-25**). The first is the gray rami communicans to the sympathetic ganglion. The second branch is the posterior cutaneous nerve that supplies the paravertebral muscles and overlying skin. The third branch is the lateral cutaneous nerve that arises about the midaxillary line. It divides into an anterior and posterior division to supply most of the chest and the abdominal wall. The final branch is the terminal anterior cutaneous nerve. It supplies the anterior chest and abdominal wall adjacent to the midline. Each intercostal nerve travels within a neurovascular bundle behind the inferior border of each rib (**Figure 156-25*B***). The intercostal nerve lies inferior to the intercostal vein and artery. The intercostal nerve may be blocked at several sites along its course. The most common site is at the angle of the rib. The technique described will be the blockade of the intercostal nerves at the angle of the rib.

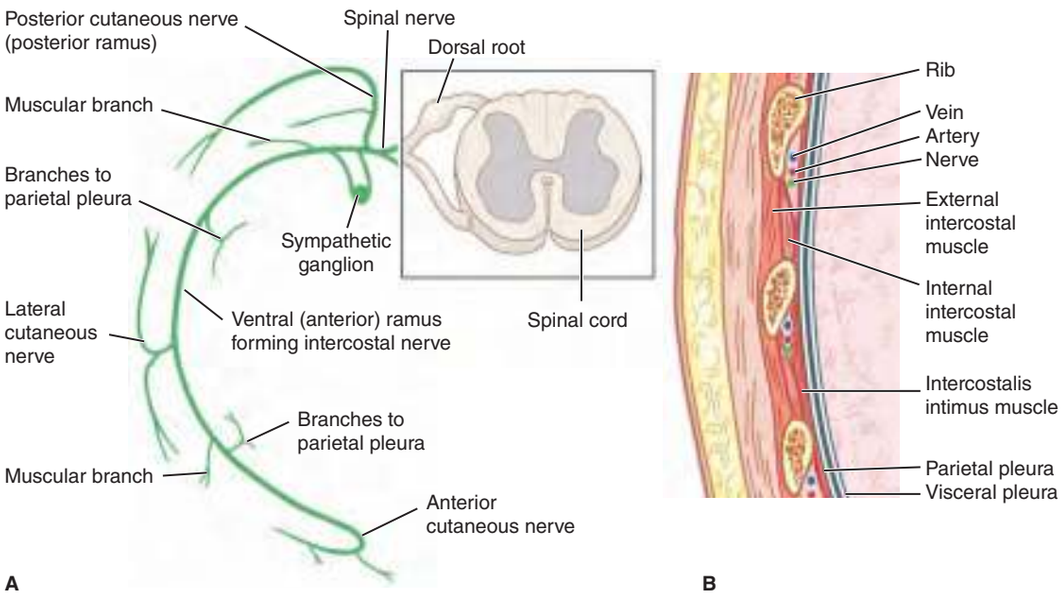


FIGURE 156-25. The intercostal nerve. ***A.*** The anatomy of a typical thoracic spinal nerve. ***B.*** Cross-section through the chest wall. The intercostal nerves are contained within a neurovascular bundle that lies behind the inferior border of each rib.

Patient positioning: Place the patient prone with a pillow under the midabdomen to straighten the lumbar curve and increase the size of the intercostal spaces posteriorly.

Landmarks: **The most important step is to correctly identify the anatomy of the patient.** Draw a line along the vertebral spines corresponding to the levels to be anesthetized. Palpate laterally from the vertebral spines to the edge of the paraspinal muscles. This is the location where the ribs are most superficial. This distance can vary from 6 to 8 cm from the midline in the average adult. Draw vertical lines parallel to the first line and along the edge of the paraspinal muscles (**Figure 156-26*A***). These lines must angle slightly medially over the upper ribs to avoid the scapula. Palpate and mark the inferior edge of each rib along these two vertical lines (**Figure 156-26*A***).

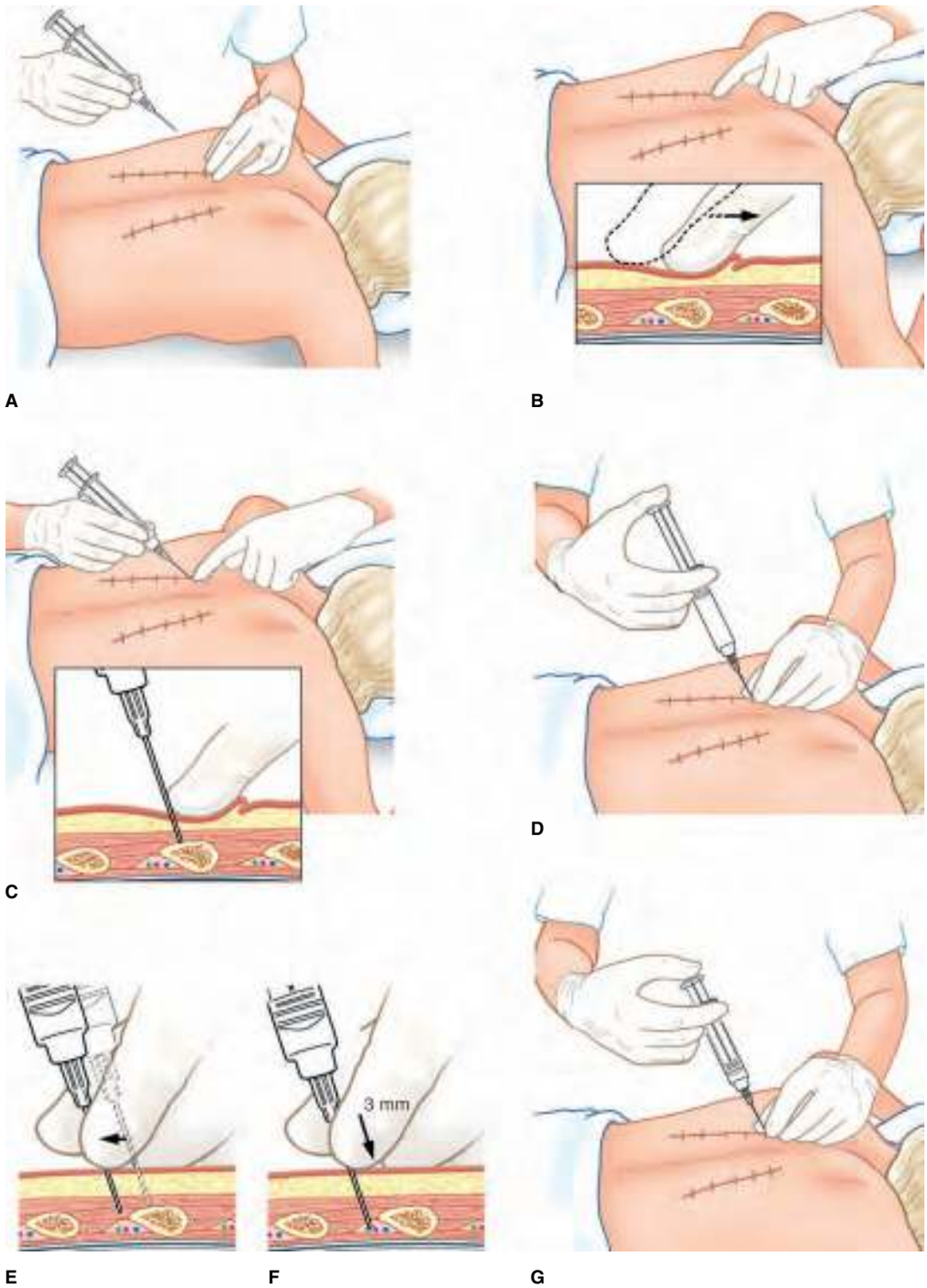


FIGURE 156-26. The intercostal nerve block. ***A.*** The patient is placed prone. A line is drawn along the lateral border of the paraspinal muscles. Note that the upper end is angled medially to avoid the scapula. Cross-marks are drawn to denote the inferior border of the rib and the location to perform the block. ***B.*** The index finger of the nondominant hand pulls the skin overlying the inferior border of the rib upward. ***C.*** The dominant hand is resting against the patient. The needle is inserted at a 60° angle to the skin and advanced until the rib is contacted. ***D.*** The fingers of the nondominant hand grasp and stabilize the needle. ***E.*** The needle is "walked" off the inferior border of the rib. ***F.*** The needle is advanced 3 mm so that the tip is within the neurovascular bundle. ***G.*** Inject 1 to 2 mL of local anesthetic solution while maintaining the needle in a stable position with the dominant hand.

Needle insertion and direction: **This procedure requires the utmost of care to prevent inducing a pneumothorax.** Inject local anesthetic solution to make a skin wheal at the intersection of the horizontal lines with the vertical paraspinal muscle lines. Use the index finger of the nondominant hand to pull the skin at the lower edge of the rib up onto the rib (**Figure 156-26*B***). Grasp the syringe in the dominant hand. Insert the 25 gauge needle at a 60° angle along the tip of the nondominant index finger while the dominant hand is resting on the patient's back (**Figure 156-26*C***). Advance the needle until the rib is contacted (**Figure 156-26*C*, inset**).

Reposition the nondominant hand so that it is resting against the patient's back and holding the needle between the thumb, index, and middle fingers (**Figure 156-26*D***). Use the nondominant hand to slowly and carefully "walk" the needle off the inferior rib margin (**Figure 156-26*E***). Advance the needle 3 mm with the nondominant hand (**Figure 156-26*F***). **It is imperative that the needle not be advanced more than 3 mm after it is "walked" off the inferior border of the rib to prevent an iatrogenic pneumothorax. Aspirate to ensure that the tip of the needle is not within a blood vessel or the lung.** Inject 1 to 2 mL of local anesthetic solution (**Figure 156-26*G***). Remove the needle and repeat the procedure at the other desired interspaces.

Remarks: Local anesthetic solution for intercostal blocks should contain 1:200,000 or less of epinephrine. The nerve can be blocked with the aid of US.[50] The only advantage is visualization of the underlying lung may prevent an iatrogenic pneumothorax. **Obtain a postprocedural upright chest radiograph to ensure that the patient does not have an iatrogenic pneumothorax.**[51]

PENILE BLOCK

The penis may be anesthetized for the purposes of circumcision, laceration repair, foreign body removal, zipper entrapment, or to perform the release of a phimosis or paraphimosis. The dorsal nerves of the penis provide sensory innervation to the penis. They emerge from under the pubis just lateral to the symphysis and course along the dorsal surface of the penis. These nerves are located approximately 0.5 cm from the dorsal penile midline. These nerves are blocked at the base of the penis. Refer to Chapter 177 for the complete details of the penile block.

REGIONAL ANESTHESIA TECHNIQUES FOR THE LOWER EXTREMITY

The sensory innervation of the lower extremity is illustrated in **Figure 156-27**. Note that the nerves provide patches of innervation rather than stripes of innervation beginning at the torso and extending to the foot.

FEMORAL NERVE BLOCK

Anatomy: The femoral nerve is formed from the lumbar plexus. It travels in the pelvis between the iliacus and psoas major muscles. It enters the thigh below the inguinal ligament, midway between the anterior superior iliac spine and the pubic tubercle (**Figure 156-28*A***). The femoral nerve lies anterior to the iliopsoas muscle and lateral to the femoral artery in the proximal thigh (**Figure 156-28*B***). The femoral nerve has both sensory and motor components. It provides motor innervation to the anterior thigh muscles. It provides sensory innervation for the anterior thigh, anteromedial thigh, medial thigh, medial leg, and medial border of the foot (**Figure 156-27**).

Patient positioning: Place the patient supine with their hip and knee extended and the leg slightly externally rotated.

Landmarks: Identify the anterior superior iliac spine and the pubic tubercle by palpation. Connect these landmarks with a straight line to roughly approximate the position of the inguinal ligament. Identify the femoral artery by its palpable pulse 1 to 2 cm below the midpoint of the inguinal ligament.

Needle insertion and direction: Place a skin wheal of local anesthetic solution just lateral to the femoral artery pulse. Insert a 25 gauge needle through the skin wheal and perpendicular to the skin (**Figure 156-28*B***). Slowly advance the needle while remaining perpendicular to the skin. The femoral nerve is identified once paresthesias are elicited. Withdraw the needle 2 mm and allow the paresthesias to resolve. Inject 15 to 20 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US.[52-55] Place the US transducer along the inguinal crease, midway between the anterior superior iliac spine and the pubic tubercle (**Figure 156-28*C***). Identify the femoral artery and femoral vein. The femoral nerve is hyperechoic and located 0.5 to 1 cm lateral and posterior to the common femoral artery (**Figure 156-28*D***). Apply pressure with the US transducer to distinguish the compressible femoral vein from the incompressible femoral artery. Use color Doppler to confirm the location of the femoral artery and any branches or takeoffs. Place the needle lateral to the long axis of the US transducer (**Figure 156-28*C***). Slowly insert and advance the needle. Visualize the entire length of the needle as it is inserted and approaches the femoral nerve. Continue to advance the needle and penetrate the fascia lata and fascia iliaca so that the tip of the needle is adjacent to the femoral nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the femoral nerve. If satisfactory, inject the remainder of the local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: The femoral nerve is contained within a fibrous sheath that is separate from the other contents (i.e., the femoral artery and vein) of the femoral triangle (**Figure 156-28*B***). **Paresthesias must be elicited to confirm the proper position of the tip of the needle before injecting the local anesthetic solution if using the landmark technique.** Deposition of the local anesthetic solution outside of the fibrous sheath will not result in any anesthesia except in the area of the injection.

SAPHENOUS NERVE BLOCK, AT THE KNEE

Anatomy: The saphenous nerve is the terminal branch of the femoral nerve. It travels across the anterior thigh in a medial direction to become superficial at the medial knee after emerging between the tendons of the gracilis and sartorius muscles (**Figure 156-29*A***). It follows the great saphenous vein from above the knee to below the medial malleolus (**Figure 156-29*B***). It provides sensory innervation to the anteromedial leg, medial leg, posteromedial leg, and medial border of the foot to the ball of the great toe (**Figure 156-27**). It has no motor component.

Patient positioning: Place the patient supine with their ankle supported on a pillow or blanket, the knee extended, and the leg externally rotated.

Landmarks: Identify the femoral condyle above the knee or the tibial condyle below the knee by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution over the posteromedial aspect of either condyle (i.e., femoral or tibial). Insert a 25 gauge needle through the skin wheal. Infiltrate 7 to 10 mL of local anesthetic solution subcutaneously in a transverse line from the posteromedial to the anteromedial aspect of either condyle (**Figure 156-29*C***).

US-guided block: The nerve can be blocked with the aid of US. Identify the femoral artery and femoral nerve at the inguinal crease (see femoral nerve block). Move the US transducer inferiorly and

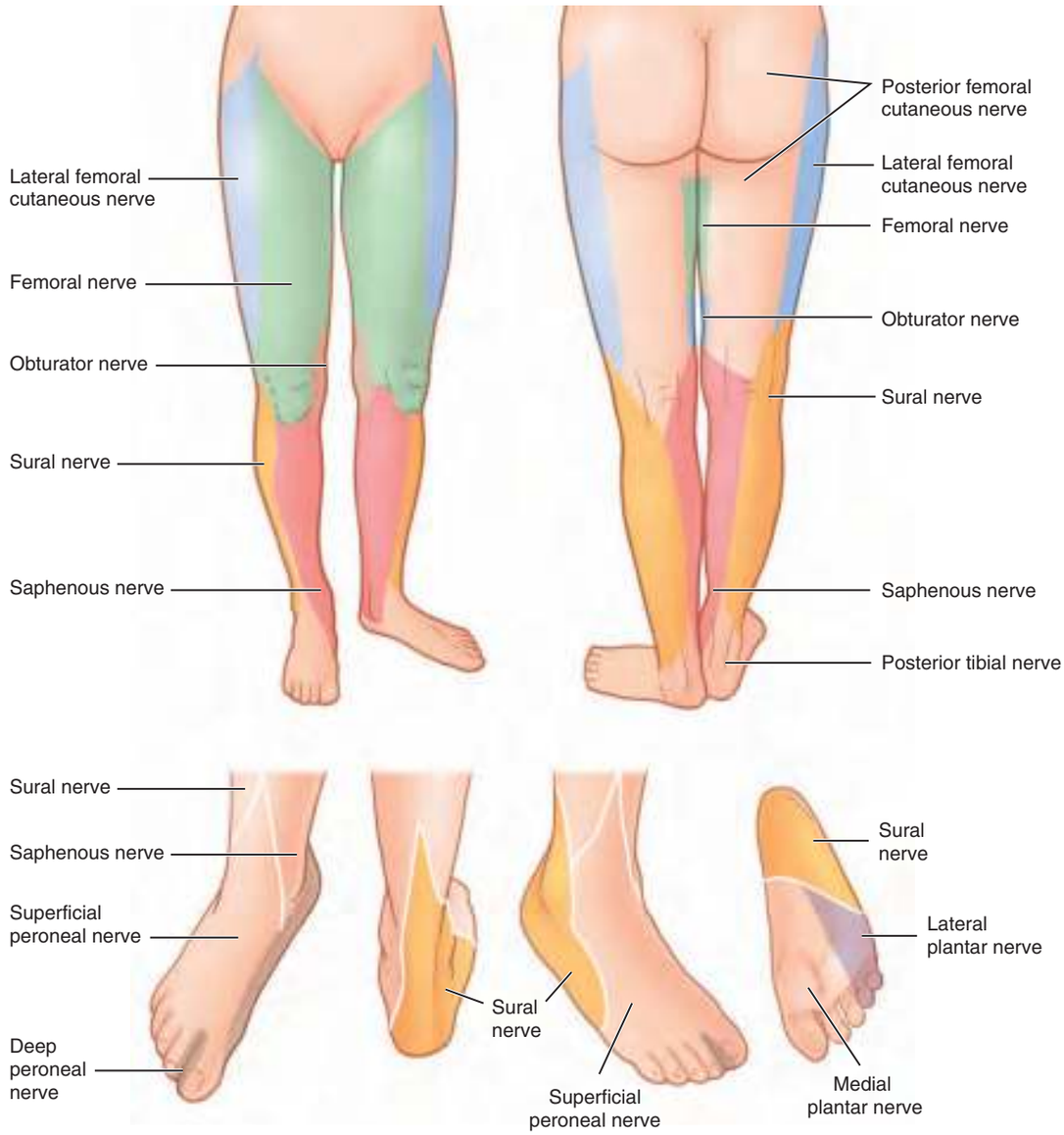


FIGURE 156-27. The sensory distribution of the cutaneous nerves of the lower extremity.

medially keeping the femoral artery in the center of the screen. At the mid-thigh, the saphenous nerve travels with the femoral artery and the nerve to the vastus medialis muscle. These structures lie posterior to the sartorius muscle and medial to the vastus medialis muscle (**Figure 156-29*D***). Continue to move the transducer inferiorly and medially until the transducer is at the distal third of the thigh. Use color Doppler to confirm the location of the femoral artery and any branches or take-offs. The saphenous nerve is medial to the femoral artery and posterior (i.e., deep) to the sartorius muscle (**Figure 156-29*D***). The small saphenous nerve may not be visualized by US at this level. The target is the fascial plane between the sartorius and vastus medialis muscle.

Place the needle lateral to the long axis of the US transducer. Insert and advance the needle. Visualize the entire length of the needle as it is inserted and approaches the fascial plane between the sartorius and vastus medialis muscles. Aspirate to ensure the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the test dose spread the local anesthetic solution around the 12 o'clock to 2 o'clock position of the femoral artery. If it is satisfactory, inject another 5 to 10 mL of the local anesthetic solution. If the test dose is not satisfactory, reposition the needle and inject another test dose.

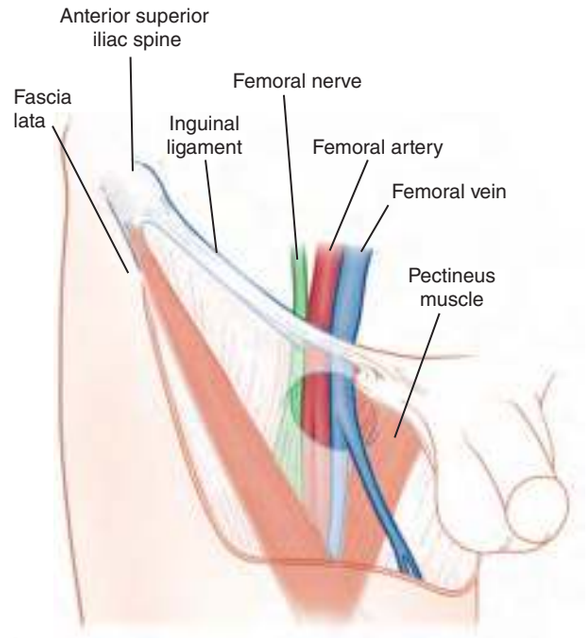
Remarks: The saphenous nerve may be blocked at the ankle if anesthesia of the medial leg is not required.

SAPHENOUS NERVE BLOCK, AT THE ANKLE

Anatomy: The saphenous nerve divides into numerous terminal branches in the distal leg. These terminal branches travel across the anteromedial ankle (**Figure 156-29*B***) to innervate the anteromedial aspect of the ankle and foot (**Figure 156-27**).

Patient positioning: Place the patient supine with their ankle supported on a pillow or blanket, the knee extended, and the leg externally rotated.

Landmarks: Identify the anterior border of the medial malleolus and the great saphenous vein by palpation.



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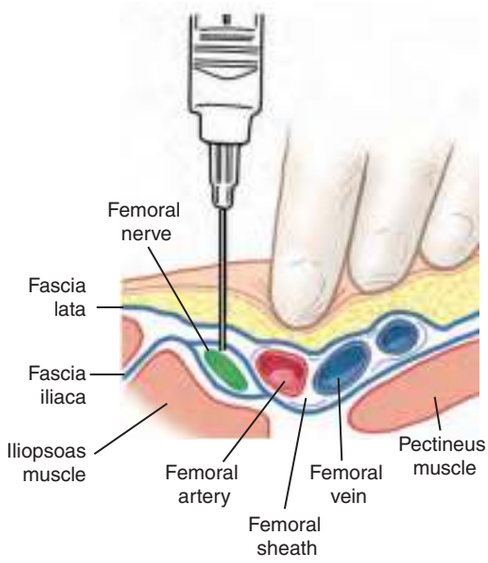

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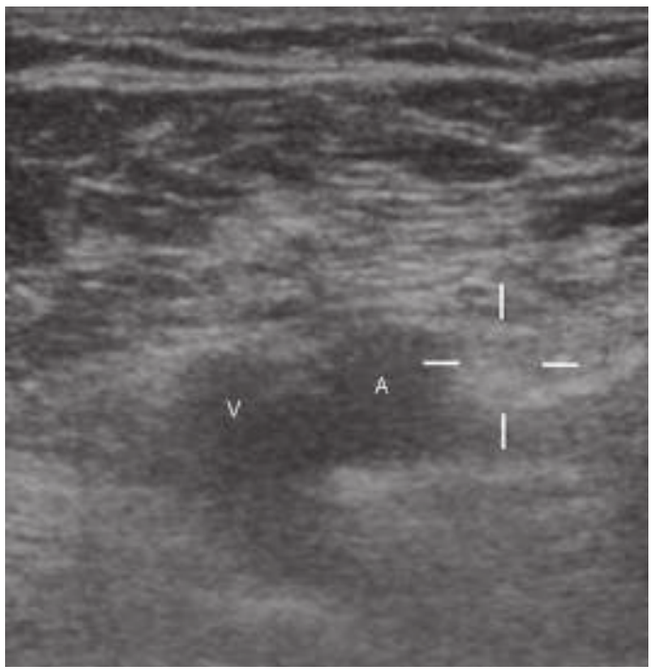


D

FIGURE 156-28. The femoral nerve block. ***A.*** The anatomy of the inguinal region. ***B.*** Blockade of the femoral nerve. ***C.*** US transducer and needle placement. ***D.*** US image of the femoral neurovascular bundle. The femoral nerve is located within the crosshairs (A, femoral artery; V, femoral vein).

Needle insertion and direction: Place a skin wheal of local anesthetic 1.5 cm superior and anterior to the medial malleolus. Insert a 25 gauge needle through the skin wheal. Infiltrate 3 to 5 mL of local anesthetic solution subcutaneously in a fan-like pattern around the great saphenous vein.

US-guided block: The nerve can be blocked with the aid of US. Identify the great saphenous vein anterior to the medial malleolus. Use color Doppler to confirm the location of the great saphenous vein. The saphenous nerve is adjacent to the great saphenous vein. The small saphenous nerve may not be visualized by US at this level. Place the needle lateral to the long axis of the US transducer. Insert the needle until its tip is adjacent to the great saphenous vein. Aspirate to ensure the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the test dose spread the local anesthetic solution around the great saphenous vein. If it is satisfactory, inject another 2 to 3 mL of the local anesthetic solution. If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: Alternatively, infiltrate 5 to 7 mL of local anesthetic solution subcutaneously in a transverse line from the anterior

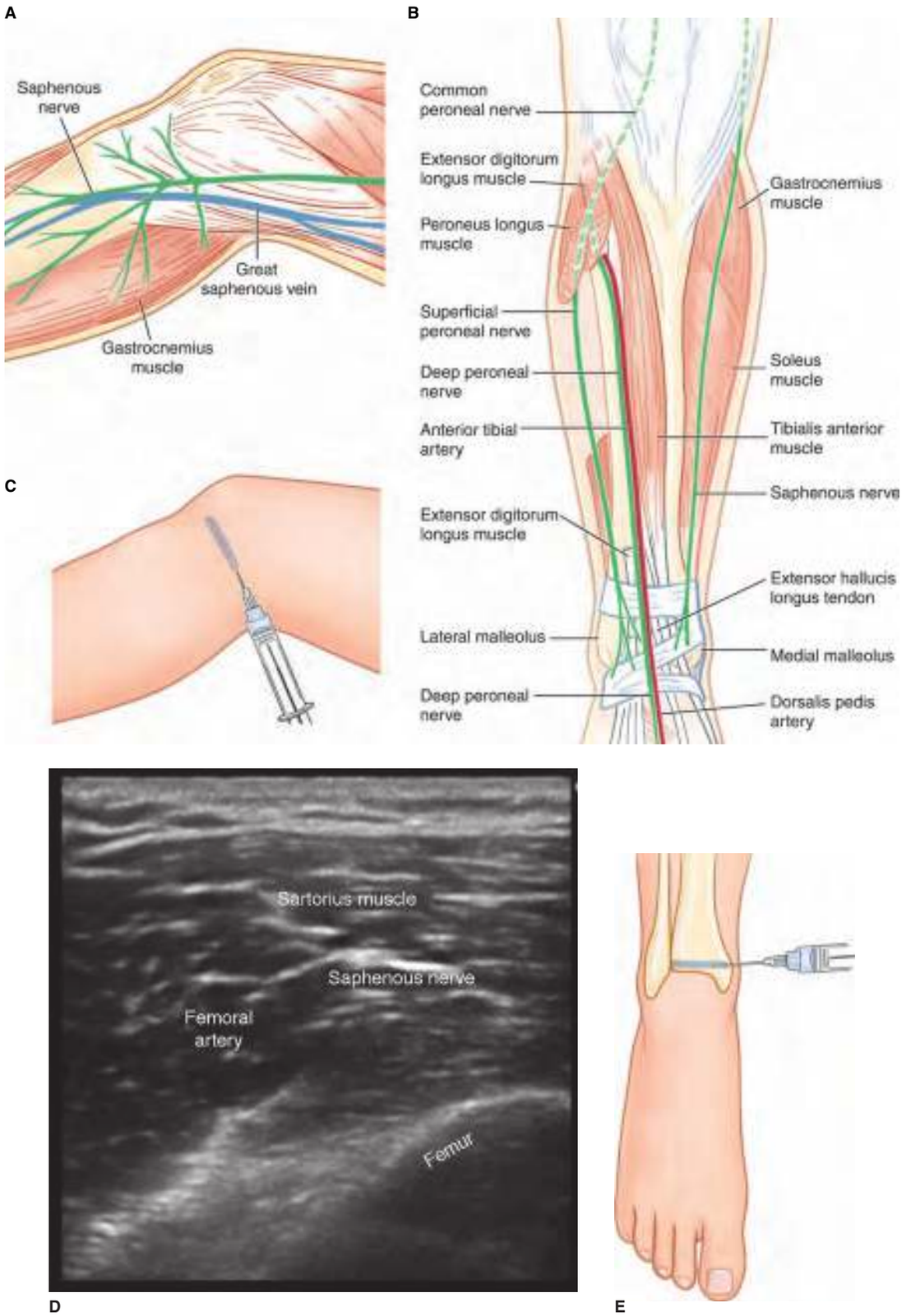


FIGURE 156-29. The saphenous nerve block. ***A.*** The course of the saphenous nerve at the medial knee. ***B.*** The course of the saphenous nerve in the leg. ***C.*** Blockade at the level of the knee. ***D.*** US image of the saphenous nerve in the distal thigh. ***E.*** Blockade at the level of the ankle.

border of the medial malleolus to the anterior border of the anterior tibial ridge (**Figure 156-29*E***).

LATERAL FEMORAL CUTANEOUS NERVE BLOCK

Anatomy: The lateral femoral cutaneous nerve enters the thigh through or below the inguinal ligament, 1 to 2 cm medial to the anterior superior iliac spine (**Figure 156-30*A***). It crosses through or over the sartorius muscle to lie on its anterior surface and deep to the fascia lata. This nerve provides sensory innervation to the anterolateral and lateral thigh (**Figure 156-27**). It has no motor components.

Patient positioning: Place the patient supine with their hip and knee extended.

Landmarks: Identify the anterior superior iliac spine by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution 2 to 3 cm inferior and 2 to 3 cm medial to the anterior superior iliac spine. Insert a 25 gauge needle perpendicular to the skin wheal. Advance the needle through the fascia lata. A "pop" will be felt as the needle traverses through the fascia lata. Infiltrate 10 mL of local anesthetic solution subcutaneously in a superior to inferior fan-like pattern.

A second approach begins with making the same skin wheal. Insert the needle through the skin wheal directed laterally and superiorly. Advance the needle to contact the iliac bone just medial and inferior to the anterior superior iliac spine. Infiltrate 10 mL of local anesthetic solution subcutaneously in a fan-like pattern about the iliac bone.

US-guided block: The nerve can be blocked with the aid of US. Place the US transducer along the inguinal ligament and just medial to the anterior superior iliac spine. Move the US transducer medially and inferiorly. Identify the fascia lata, fascia iliaca, and sartorius muscle (from superficial to deep, respectively). It may be difficult to identify the small lateral femoral cutaneous nerve between the fascia lata and fascia iliaca just above the sartorius muscle.

Place a skin wheal of local anesthetic solution. Place the needle lateral to the long axis of the US transducer. Insert and advance the needle. Visualize the entire length of the needle as it is inserted and approaches the lateral femoral cutaneous nerve. If the nerve cannot be identified, direct the needle immediately medial and inferior to the anterior superior iliac spine near the sartorius muscle insertion site. Advance the needle tip between the fascia lata and fascia iliaca. Aspirate to ensure the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the test dose spread in the fascial plane of fascia lata and fascia iliaca. If it is satisfactory, inject another 5 to 10 mL of the local anesthetic solution. If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: A third, or alternative, approach is often used (**Figure 156-30*B***). Place a skin wheal of local anesthetic solution just medial to the anterior superior iliac spine. Insert the needle through the skin wheal and perpendicular to the skin. Advance the needle until a "pop" is felt as the needle transverses the aponeurosis of the external oblique muscle. Continue to slowly advance the needle until a second "pop" is felt as the needle transverses through the internal oblique muscle and underlying iliac fascia. Inject 5 to 10 mL of local anesthetic solution. This approach blocks the nerve in its canal as it begins to pass under the inguinal ligament.

OBTURATOR NERVE BLOCK

Anatomy: The obturator nerve originates from the lumbar plexus. It passes through the pelvis and obturator canal to exit the obturator foramen into the thigh with its accompanying artery and vein (**Figure 156-31*A***). It divides within the obturator canal into anterior and posterior branches. The anterior branch provides the sensory innervation to the hip and motor innervation to the anterior adductor muscles. There may also be a small and inconsistent area of

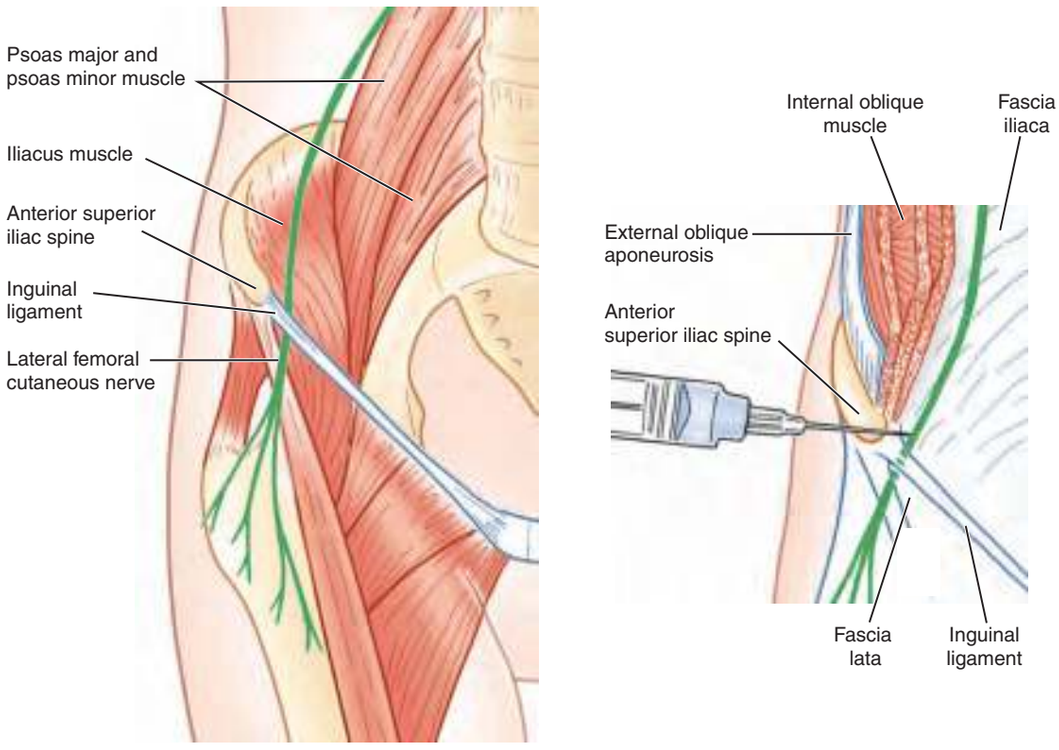


FIGURE 156-30. The lateral femoral cutaneous nerve block. ***A.*** The course of the lateral femoral cutaneous nerve. ***B.*** The third or alternative approach to blockade of the nerve.

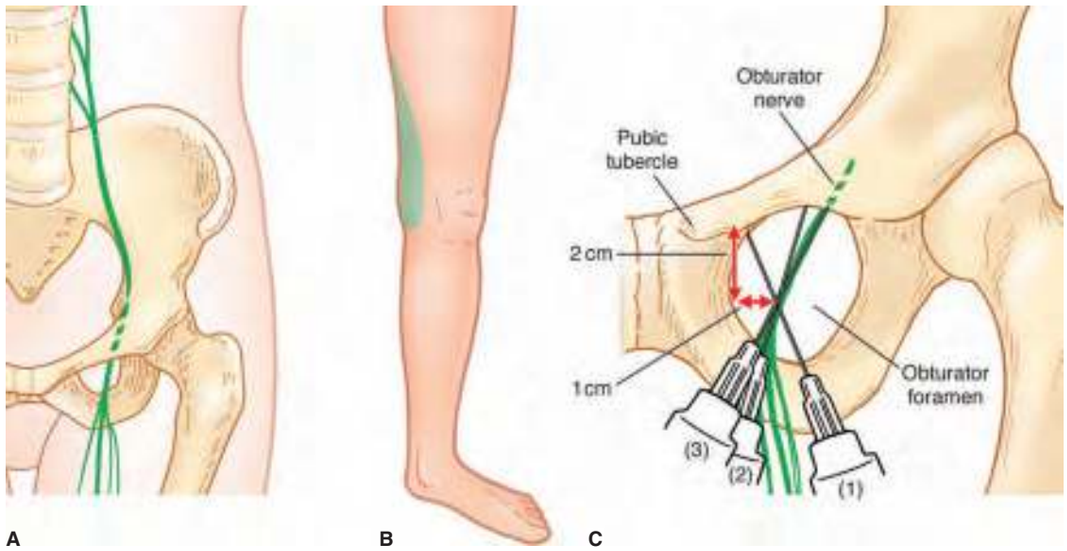


FIGURE 156-31. The obturator nerve block. ***A.*** The course of the obturator nerve. ***B.*** The sensory distribution of the obturator nerve. ***C.*** Blockade of the obturator nerve.

cutaneous innervation over the medial thigh (**Figures 156-27 and 156-31*B***). The posterior branch provides motor innervation to the deep adductor muscles and sensory innervation to the knee joint.

Patient positioning: Place the patient supine with their hip and knee extended. Abduct the hip 10° to 20°.

Landmarks: Identify the pubic tubercle by palpation.

Needle insertion and direction: Place a skin wheal of anesthetic solution 1.5 cm inferior and 1.5 cm lateral to the pubic tubercle. Insert a 7 to 10 cm, 22 to 25 gauge spinal needle directed medially through the skin wheal. Advance the needle until it contacts the horizontal ramus of the pubic bone (**Figure 156-31*C-1***). Withdraw the needle slightly (2 to 3 mm) and redirect it 45° superiorly. Advance the needle to identify the superior bony portion of the obturator canal (**Figure 156-31*C-2***). Withdraw the needle slightly (2 to 3 mm). Redirect the needle slightly laterally and inferiorly towards the obturator canal. Advance the needle 2 to 3 cm (**Figure 156-31*C-3***). Inject 10 to 15 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US. Place the US transducer along the medial third of the inguinal crease. Scan distally from the inguinal crease and identify the adductor longus, adductor brevis, and adductor magnus muscles (from superficial to deep, respectively). The obturator nerve divides into two branches distal to the inguinal crease, the anterior and posterior branches. The anterior branch lies in the fascial plane between the adductor longus and adductor brevis. The posterior branch lies in the fascial plane between the adductor brevis and adductor magnus. Both branches are small nerves and may or may not be identified under US.

Place a skin wheal of local anesthetic solution. Place the needle lateral to the long axis of the US transducer. Insert the needle in the plane of the long axis of the US beam. To block the anterior branch of the obturator nerve, visualize the entire length of the needle as it is inserted and approaches the fascial plane between the adductor longus and brevis. To block the posterior branch, insert the needle into the fascial plane between the adductor brevis and magnus. Once the needle tip is in between the fascial planes, aspirate to ensure the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the test dose spread the two fascial planes. If it is satisfactory, inject another 10 to 15 mL of the local anesthetic solution. If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: The purpose of this block is to provide analgesia for an acute hip fracture when the administration of intravenous analgesics is contraindicated. The technique requires significant experience and is time-consuming. **The obturator nerve must be anesthetized within the bony canal to prevent the needle from perforating the bladder or vagina.** This block is rarely performed in the Emergency Department but its use is increasing as bedside US use and its applications are increasing.

SCIATIC NERVE BLOCK, CLASSIC OR POSTERIOR APPROACH

Anatomy: The sciatic nerve arises from the lumbosacral plexus and leaves the pelvis through the greater sciatic foramen inferior to the piriformis muscle (**Figure 156-32**). It is located midway between the ischial tuberosity and the greater trochanter of the femur. The nerve is superficial and accessible at the inferior border of the gluteus maximus muscle. The sciatic nerve provides the motor innervation to the muscles of the posterior thigh, leg, and foot. It provides sensory innervation to the posterior thigh, anterolateral leg, posterolateral leg, lateral leg, and almost the entire foot. There are four techniques for sciatic nerve blockade. These approaches were developed to allow sciatic nerve blockade from a variety of positions, thus eliminating positioning problems that are encountered in the elderly, the infirm, and the trauma patient.

Patient positioning: Place the patient on the side opposite that to be blocked. Flex the hip and knee of the upper leg until the heel is over the dependent knee (**Figure 156-33*A***).

Landmarks: **Identification of the anatomic landmarks is the key to success (Figure 156-33*B***). Identify the greater trochanter of the femur and the posterior superior iliac spine by palpation. Connect these two landmarks with a straight line. Identify the midpoint of this line and draw a perpendicular bisector downward for 3 cm. This point represents the site of local anesthetic injection. Verify the position for injection with a second line. Reidentify the greater trochanter of the femur and the sacral cornu. Draw a line starting from 1.5 cm below the sacral cornu to the greater trochanter. This line should cross the end of the perpendicular bisector and be directly over the sciatic nerve (**Figure 156-33*B***).

Needle insertion and direction: Place a skin wheal of local anesthetic solution at the identified injection site. Insert a 22 gauge

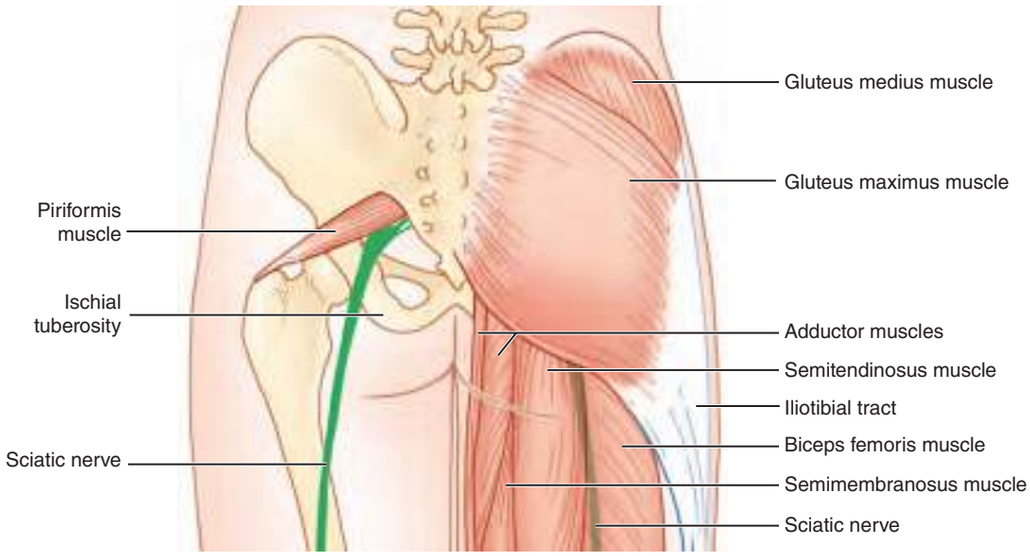
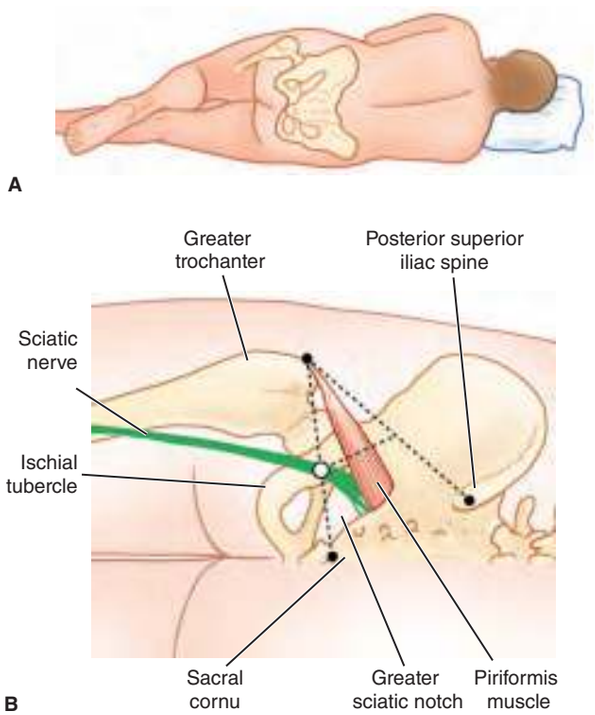


FIGURE 156-32. The course of the sciatic nerve.

needle perpendicular to the skin wheal. Advance the needle until paresthesias are elicited. **If paresthesias are not elicited, redirect the needle to find the sciatic nerve. The nerve must be located prior to infiltration with the local anesthetic solution to achieve proper anesthesia.** Withdraw the needle 2 mm and allow the paresthesias to resolve. Inject 20 to 30 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US.[53] Place the US transducer over the previously identified intersection point (**Figure 156-33*B***). Place the long axis of the US transducer along the line between the greater trochanter and the sacral hiatus. Identify the ischial bone as a hyperechoic line with a bony shadow underneath and medial to the sciatic nerve. Identify the gluteus maximus muscle superficial (i.e., posterior) to the sciatic nerve. The sciatic nerve appears wide and flat between the gluteus maximus muscle and the ischial bone (**Figure 156-33*C***). Scan cephalad and caudad to obtain the best view of the sciatic nerve.

Place a skin wheal of local anesthetic solution. Place the needle lateral to the long axis of the US transducer. Insert the needle in the



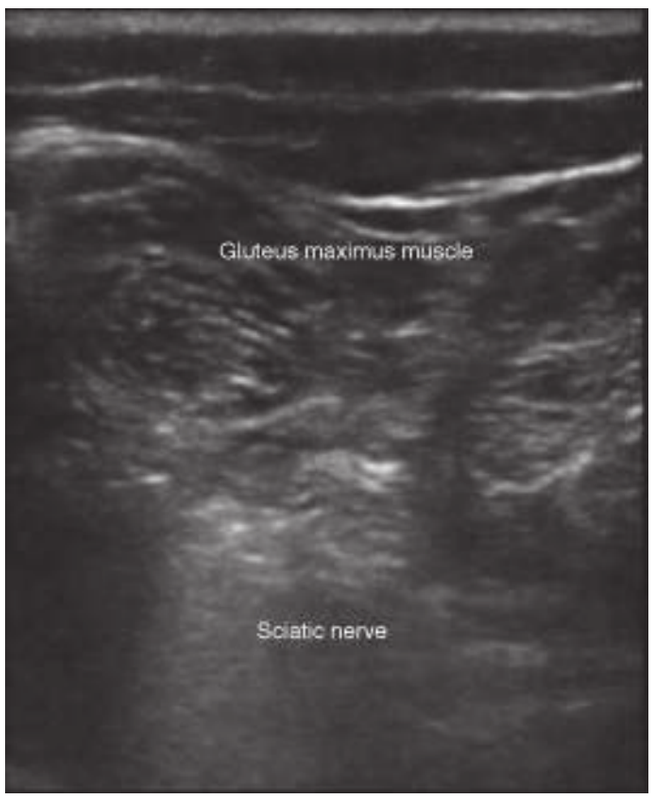


FIGURE 156-33. The classic or posterior approach to the sciatic nerve block. ***A.*** Patient positioning. ***B.*** Identification of the landmarks required to perform the block. ***C.*** US image of the sciatic nerve underneath the gluteus maximus muscle.

plane of the long axis of the US beam. Advance the needle toward the sciatic nerve. Stop advancing the needle when its tip is adjacent to the sciatic nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the sciatic nerve. If satisfactory, inject 15 to 20 mL of local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: Blockade of the sciatic nerve can be used for fracture reduction, extensive laceration repair, incision and drainage of abscesses, wound exploration, or anesthesia from burns or trauma. The posterior approach is the most commonly performed technique.[56]

SCIATIC NERVE BLOCK, ANTERIOR APPROACH

Anatomy: The anatomy and innervation of the sciatic nerve are described in the previous section. This approach can be used for patients who cannot be turned onto their side.

Patient positioning: Place the patient supine with their hip and knee extended.

Landmarks: **Identification of the anatomic landmarks is the key to success (Figure 156-34*A*).** Identify the anterior superior iliac spine and the pubic tubercle by palpation. Connect these points with a straight line (**Figure 156-34*A*, *Line 1***). This line represents the location of the inguinal ligament. Trisect the inguinal ligament line into three equal parts. Draw a line perpendicular from the junction of the medial and middle thirds (**Figure 156-34*A*, *Line 2***). Identify the tuberosity of the greater trochanter by palpation. Draw a line from the tuberosity medially across the anterior thigh and parallel to the line representing the inguinal ligament (**Figure 156-34*A*, *Line 3***). The point of intersection of this line (**Figure 156-34*A*, *Line 3***) with the perpendicular line from the inguinal ligament (**Figure 156-34*A*, *Line 2***) represents the point of injection.

Needle insertion and direction: Place a skin wheal of local anesthetic solution at the identified injection site. Insert a 10 to 15 cm long, 22 gauge needle perpendicular to the skin and aimed slightly lateral. Advance the needle until the femur is contacted (**Figure 156-34*B*-*1***). Withdraw the needle 1 cm and redirect it medially. Advance the needle 5 cm past the location where the bone was found (i.e., 6 cm; **Figure 156-34*B*-*2***). The needle should be within the neurovascular bundle containing the sciatic nerve. The average distance from the surface of the femur to the neurovascular bundle is 4.5 to 6.0 cm in adults. **Inject a small test dose of local anesthetic solution to determine the ease of injection. The tip of the needle is within muscle or a fascial plane if significant resistance is encountered.** Advance the needle until resistance to injection is at a minimum. Eliciting paresthesias is extremely helpful in identifying the correct location of the needle. If paresthesias are elicited, withdraw the needle 2 mm and allow them to resolve. Inject 15 to 30 mL of local anesthetic solution.

Remarks: The posterior femoral cutaneous nerve, which accompanies the sciatic nerve, may sometimes be missed with this approach. This technique is extremely painful and not often performed.

SCIATIC NERVE BLOCK, LITHOTOMY APPROACH

Anatomy: The anatomy and innervation of the sciatic nerve are described in the previous section. The sciatic nerve lies anterior to the gluteus maximus muscle when the patient is in the lithotomy position. The nerve is more superficial in this position than in the other approaches.

Patient positioning: Place the patient supine. Maximally flex the hip and knee of the extremity to be anesthetized. Use a bed or examination table with foot stirrups if available and not contraindicated.

Landmarks: Identify the ischial tuberosity and the greater trochanter of the femur by palpation. Connect these landmarks with a straight line. Identify the midpoint of this line.

Needle insertion and direction: Place a skin wheal of local anesthetic solution at the midpoint of the line from the ischial tuberosity to the greater trochanter. Insert a 10 to 15 cm long, 22 gauge spinal needle perpendicular to the skin wheal. Slowly advance the needle until paresthesias are elicited. Withdraw the needle 2 mm and allow the paresthesias to resolve. Inject 20 to 25 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US. Place the US transducer over the midpoint of the line connecting

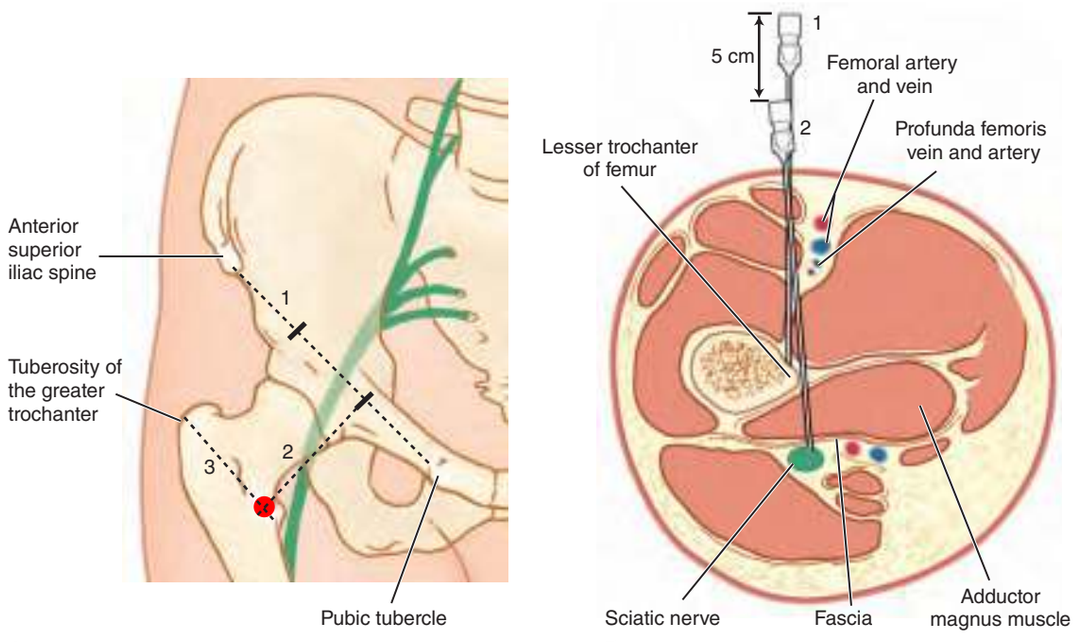


FIGURE 156-34. The anterior approach to the sciatic nerve block. ***A.*** Identification of the landmarks. ***B.*** Blockade of the sciatic nerve.

the greater trochanter and the ischial tuberosity with the long axis of the US transducer along the line between the bony landmarks. Identify the gluteus maximus muscle superior (i.e., posterior) to the sciatic nerve. Identify the sciatic nerve between the greater trochanter and the ischial tuberosity, anterior (i.e., deep) to the gluteus maximus muscle. Scan cephalad and caudad to obtain the best view of the sciatic nerve.

Place a skin wheal of local anesthetic solution. Place the needle lateral to the long axis of the US transducer. Insert the needle in the plane of the long axis of the US beam. Advance the needle toward the sciatic nerve. Stop advancing the needle when its tip is adjacent to the sciatic nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the sciatic nerve. If satisfactory, inject 15 to 20 mL of local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: This approach is more difficult to master than the previous two techniques. It is time-consuming and requires locating the sciatic nerve by eliciting paresthesias prior to injecting the local anesthetic solution.

SCIATIC NERVE BLOCK, LATERAL APPROACH

The lateral approach will not be described. This approach requires considerable experience, a nerve stimulator, and does not provide any advantage over the other approaches.

POPLITEAL FOSSA NERVE BLOCK, ANATOMIC OR POSTERIOR APPROACH

Anatomy: The popliteal fossa is a diamond-shaped area on the posterior aspect of the knee (**Figure 156-35**). Its boundaries are the long head of the biceps femoris muscle superolaterally, the semimembranous and semitendinous muscles superomedially, and the medial and lateral heads of the gastrocnemius muscle inferiorly. This space contains the tibial and common peroneal nerves, the popliteal artery and vein, and loose fatty connective tissue (**Figure 156-35*A***). The nerves are superficial to the arteries and veins, midway between the skin and the posterior surface of the femur. The average distance between the skin and the nerves is 1.5 to 2.0 cm in adults. These nerves are responsible for the motor innervation of all the muscles below the knee. They also provide sensory innervation to the entire leg below the knee except the area innervated by the saphenous nerve.

Patient positioning: Place the patient prone with a pillow or blanket under their ankle to position the knee in slight flexion (10° to 20°).

Landmarks: Identify the borders of the popliteal fossa. Divide the fossa into a superior and inferior triangle with a line drawn medial to lateral across the skin fold or crease (**Figure 156-35*B***). Draw a line from the apex to the base of the superior triangle, making two smaller and equal triangles (**Figure 156-35*B***). Identify the spot 5 cm superior and 1 cm lateral to the midline of the upper triangle. This spot represents the landmark for needle insertion.

Needle insertion and direction: Place a skin wheal of local anesthetic solution 5 cm superior and 1 cm lateral to the midline of the upper triangle (**Figure 156-35*B***). Insert a 25 gauge needle through the skin wheal, directed superiorly, and at a 45° to 60° angle to the skin surface. Advance the needle anteriorly and superiorly until paresthesias are elicited. Withdraw the needle 2 mm and allow the paresthesias to resolve. Infiltrate 35 to 45 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US.[33,57] Place the US transducer 10 cm proximal to the skin crease. The sciatic nerve lies between the biceps femoris muscle laterally and the semimembranosus and semitendinosus muscles medially (**Figure 156-35*C***). Scan cephalad and caudad to obtain the best view of the sciatic nerve and to identify where the sciatic nerve branches into the tibial and the common peroneal nerves.

Place a skin wheal of local anesthetic solution. Place the needle lateral to the long axis of the US transducer. Insert the needle in the plane of the long axis of the US beam. Advance the needle toward the sciatic nerve. Stop advancing the needle when its tip is adjacent to the sciatic nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the sciatic nerve. If satisfactory, inject 10 to 15 mL of local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: This block is difficult to use in patients who cannot lie in the prone position. This includes pregnant patients, the morbidly obese, spinal injury patients, hemodynamically unstable patients, and those on mechanical ventilation.[56]

COMMON PERONEAL NERVE BLOCK

Anatomy: The common peroneal nerve originates in the popliteal fossa as one of the terminal branches of the sciatic nerve (**Figure 156-36*A***). It courses posterolaterally around the neck of the fibula where it can be palpated. It provides motor innervation to the peroneal muscles and the muscles of the anterior leg and foot. It provides sensory innervation to the anterior leg, dorsum of the foot and toes, and the medial great toe (**Figure 156-27**).

Patient positioning: Place the patient lying on the side opposite that being anesthetized. Alternatively, place the patient supine with their leg internally rotated.

Landmarks: Identify the head and neck of the fibula and the common peroneal nerve by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution over the neck of the fibula (**Figure 156-36*B***). Insert a 25 gauge needle through the skin wheal to elicit paresthesias. Withdraw the needle 2 mm and allow the paresthesias to resolve. Inject 3 to 5 mL of local anesthetic solution. If paresthesias are not elicited, subcutaneously infiltrate the area with 5 to 7 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US. Place the patient prone with a pillow or blanket under their ankle to position the knee in slight flexion (10° to 20°). Identify the biceps femoris muscle laterally, the semimembranosus and semitendinosus muscles medially, and the popliteal crease. Place the US transducer 10 cm proximal to the crease. The sciatic nerve is located between the biceps femoris muscle laterally and the semimembranosus and semitendinosus muscles medially (**Figure 156-35*C***). Scan cephalad and caudad to obtain the best view of the sciatic nerve and to identify where the sciatic nerve branches into the tibial and the common peroneal nerves. Move the transducer caudally until the sciatic nerve branches into the tibial nerve medially and the common peroneal nerve laterally.

Place a skin wheal of local anesthetic solution. Place the needle lateral to the long axis of the US transducer. Insert the needle in the plane of the long axis of the US beam. Advance the needle toward the common peroneal nerve. Stop advancing the needle when its tip is adjacent to the common peroneal nerve. Aspirate to ensure that the needle tip is not in a blood vessel. Inject a test dose of 1 to 2 mL of local anesthetic solution. Watch the spread of local anesthetic solution around the common peroneal nerve. If satisfactory, inject 10 to 15 mL of local anesthetic solution to produce the "donut sign." If the test dose is not satisfactory, reposition the needle and inject another test dose.

Remarks: The common peroneal nerve may not always be palpable. Apply digital pressure over the neck of the fibula. Significant

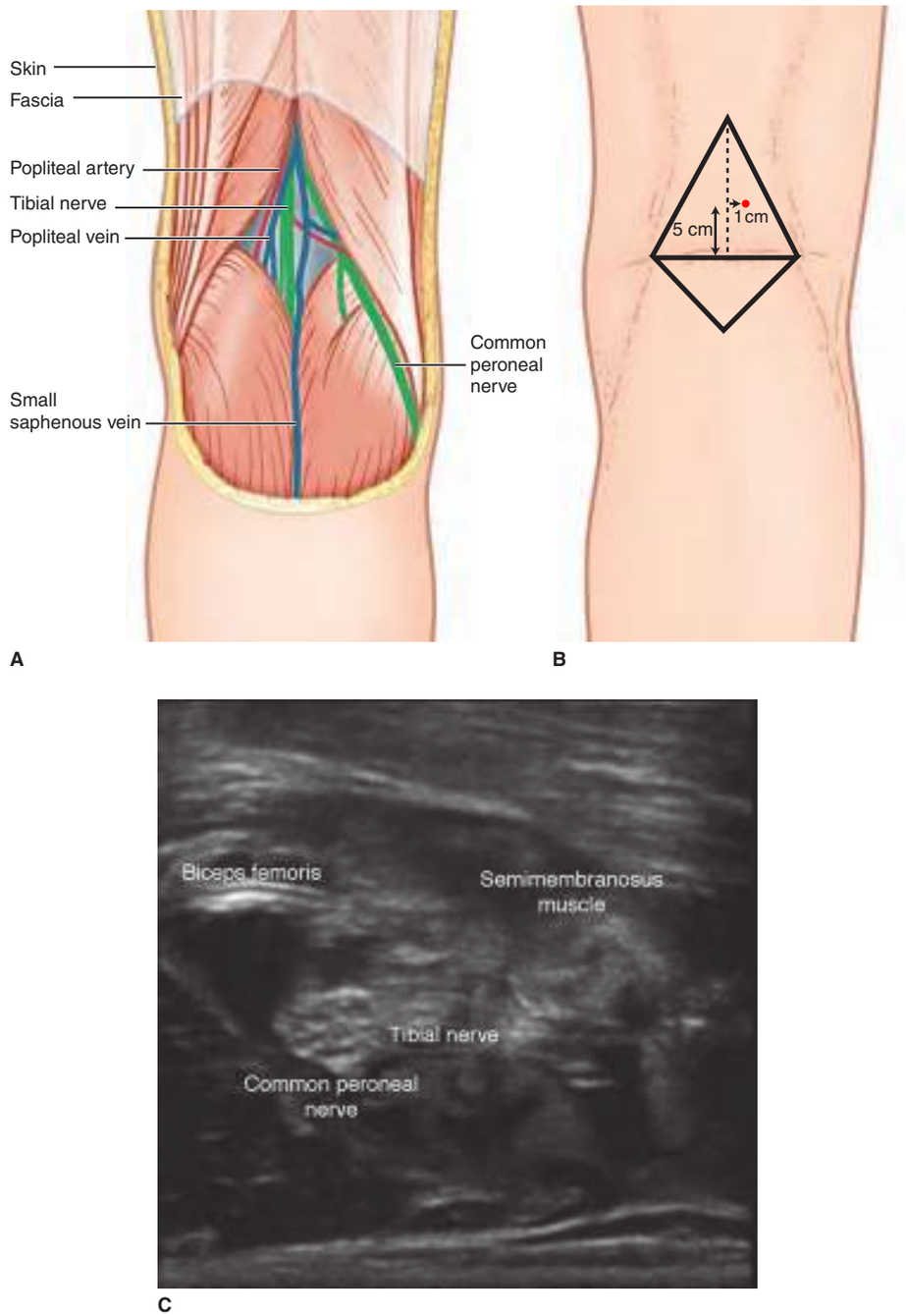


FIGURE 156-35. The anatomic or posterior approach to the popliteal fossa nerve block. ***A.*** The contents of the popliteal fossa. ***B.*** Identification of the landmarks required to perform the block. ***C.*** US image of the popliteal fossa.

discomfort will be elicited when compressing the nerve against the fibula. This site is the location of the common peroneal nerve.

SUPERFICIAL PERONEAL NERVE BLOCK

Anatomy: The superficial peroneal nerve is one of the terminal branches of the common peroneal nerve (**Figure 156-37*A***). It perforates the investing fascia of the anterior leg to become subcutaneous in the lower third of the leg. It provides motor innervation to the peroneus longus and brevis muscles. It provides sensory innervation to the anterolateral leg, medial great toe, and dorsum of the foot and toes except the first web space and the area covered by the saphenous and sural nerves (**Figure 156-27**).

Patient positioning: Place the patient supine with their ankle supported on a pillow or blanket.

Landmarks: Identify the anterior border of the medial and lateral malleolus by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution anterior to the distal aspect of the lateral malleolus. Insert a 25 gauge needle through the skin wheal. Infiltrate 6 to 10 mL of local anesthetic solution subcutaneously in a transverse line to the anterior border of the medial malleolus (**Figure 156-37*B***).

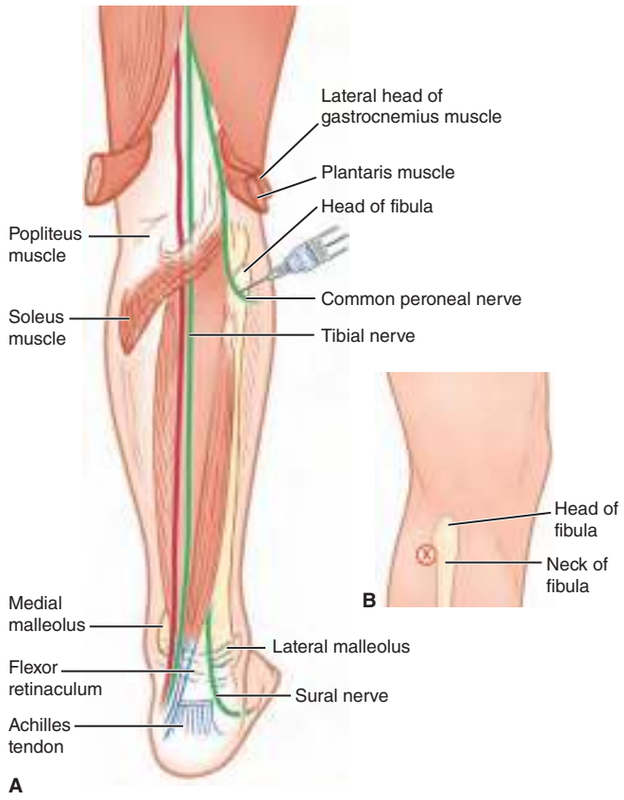


FIGURE 156-36. The common peroneal nerve block. ***A.*** The course of the common peroneal nerve. ***B.*** The landmark for performing the block.

US-guided block: The nerve can be blocked with the aid of US. Place the US transducer along the anterior border of the lateral malleolus. Identify the dorsalis pedis artery. Use color Doppler to confirm the location of the dorsalis pedis artery. Place a skin wheal of local anesthetic solution. Place the needle lateral to the long axis of the US transducer. Insert the needle in the plane of the long axis of the US beam. Visualize the entire length of the needle as it is advanced in the subcutaneous tissues in a transverse line from the lateral malleolus to the medial malleolus, avoiding the dorsalis pedis artery. Aspirate to ensure the needle tip is not in a blood vessel. Inject 3 to 5 mL of local anesthetic solution as the needle is withdrawn from the medial malleolus to the lateral malleolus.

Remarks: The superficial peroneal nerve may also be blocked at the level of the ankle. Infiltrate 4 to 8 mL of local anesthetic solution subcutaneously in a transverse line from the anterior border of the lateral malleolus to the anterior tibial ridge.

DEEP PERONEAL NERVE BLOCK

Anatomy: The deep peroneal nerve is one of the terminal branches of the common peroneal nerve. It descends over the interosseous membrane into the dorsal foot, lateral to the dorsalis pedis artery (**Figure 156-38*A***). It travels between the tendons of the tibialis anterior and extensor hallucis longus muscles at the level of the ankle joint and above. It travels between the tendons of the extensor digitorum longus and the extensor hallucis longus muscles at the level of the malleoli. It may or may not contribute a motor branch to the peroneus longus muscles. It provides sensory innervation to only the first web space (**Figure 156-27**).

Patient positioning: Place the patient supine with their ankle supported on a pillow or blanket.

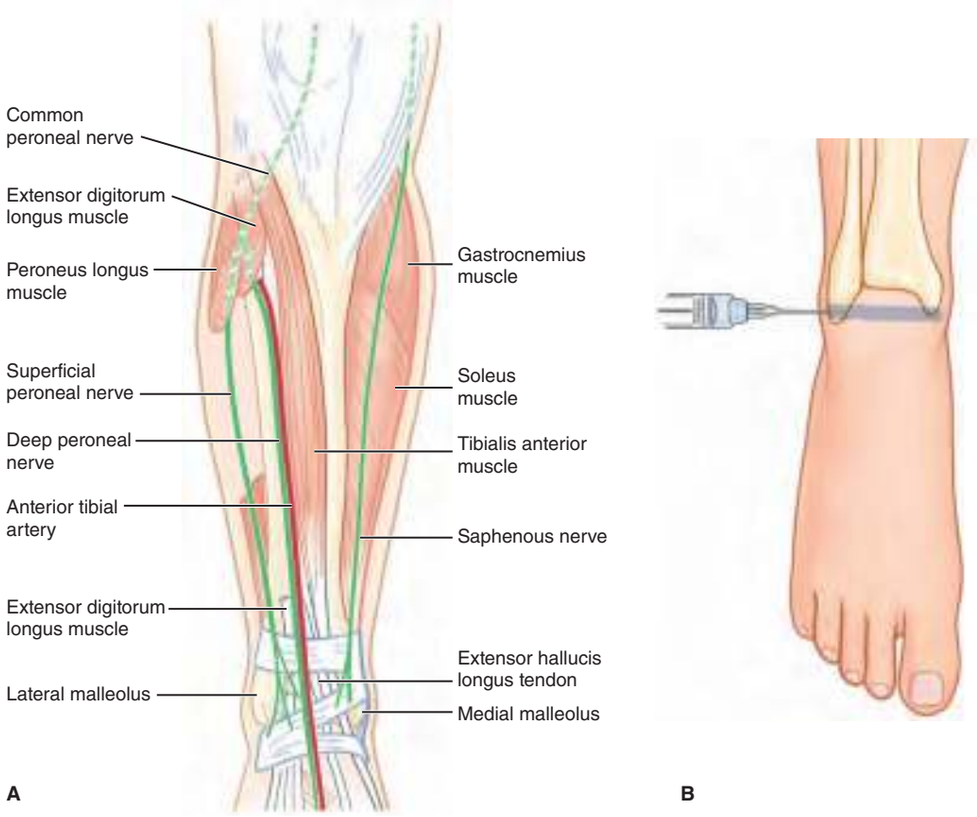


FIGURE 156-37. The superficial peroneal nerve block. ***A.*** The course of the nerve. ***B.*** Blockade of the superficial peroneal nerve.

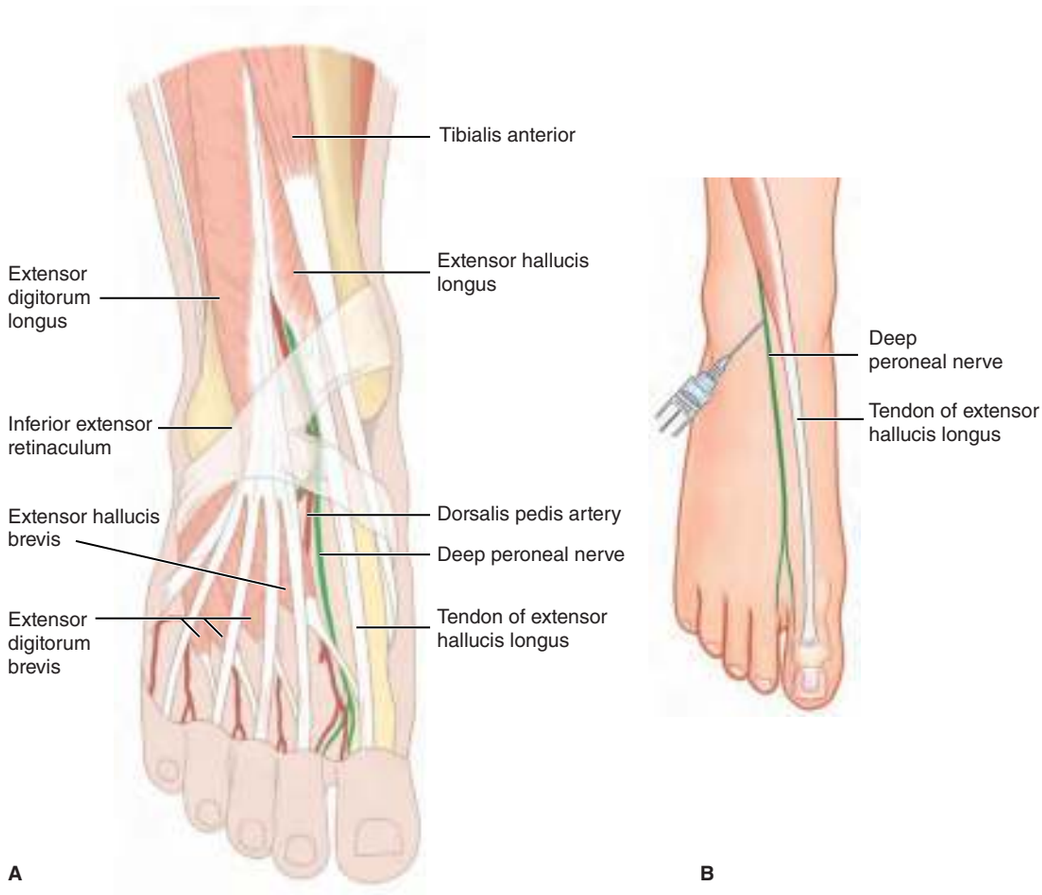


FIGURE 156-38. The deep peroneal nerve block. ***A.*** The course of the deep peroneal nerve. ***B.*** Blockade of the deep peroneal nerve.

Landmarks: Identify the tendons of the tibialis anterior and the extensor hallucis longus muscles by palpation. Identify the anterior tibial artery by its pulse at the level of the ankle joint.

Needle insertion and direction: Place a skin wheal of local anesthetic solution between the two tendons and just lateral to the anterior tibial artery (**Figure 156-38*B***). Insert a 25 gauge needle through the skin wheal and perpendicular to the skin. Advance the needle 3 mm and inject 3 to 5 mL of local anesthetic solution.

US-guided block: The nerve can be blocked with the aid of US. Place the US transducer along the line drawn between the anterior border of the lateral and medial malleolus. Identify the tendons of the extensor digitorum longus and extensor hallucis longus muscles and the dorsalis pedis artery. Use color Doppler to confirm the location of the dorsalis pedis artery. Place a skin wheal of local anesthetic solution lateral to the dorsalis pedis artery. Place the needle lateral to the long axis of the US transducer. Insert the needle in the plane of the long axis of the US beam. Visualize the entire length of the needle as it reaches the lateral aspect of the dorsalis pedis artery. Avoid penetrating the dorsalis pedis artery and any tendons. Aspirate to ensure the needle tip is not in a blood vessel. Inject 3 to 5 mL of local anesthetic solution on the lateral aspect of the dorsalis pedis artery. Watch the local anesthetic solution spread around the dorsalis pedis artery.

Remarks: The deep peroneal nerve may also be blocked at the level of the malleoli. Identify the tendons of the extensor digitorum longus and extensor hallucis longus muscles by palpation and the dorsalis pedis artery by its pulse at the level of the malleoli. Place a skin wheal of local anesthetic solution between the tendons and lateral to the dorsalis pedis artery. Insert the needle perpendicular to the skin. Advance the needle 3 mm and inject 2 to 4 mL of local anesthetic solution.

SURAL NERVE BLOCK

Anatomy: The sural nerve originates from the tibial nerve and the common peroneal nerve in the popliteal fossa. It is superficial after its origin and travels on the lateral leg and foot (**Figure 156-39**). It has already divided at the level of the ankle into numerous superficial branches, all located behind the lateral malleolus. The sural nerve provides sensory innervation to the anterolateral surface of the foot and little toe (**Figure 156-27**).

Patient positioning: Place the patient prone with their ankle supported on a pillow or blanket and the leg externally rotated. Alternatively, place the patient supine with their ankle supported on a pillow or blanket and the leg internally rotated.

Landmarks: Identify the posterior border of the lateral malleolus and the Achilles tendon by palpation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution at the level of the lateral malleolus just lateral to the Achilles tendon. Insert a 25 gauge needle through the skin wheal and angled toward the lateral malleolus (**Figure 156-39**). Infiltrate 5 mL of local anesthetic solution subcutaneously in a transverse line to the lateral malleolus.

US-guided block: The nerve can be blocked with the aid of US. Place the US transducer behind the lateral malleolus. Identify the lateral malleolus. Place a skin wheal of local anesthetic solution. Place the needle lateral to the long axis of the US transducer. Insert the needle in the plane of the long axis of the US beam. Visualize the

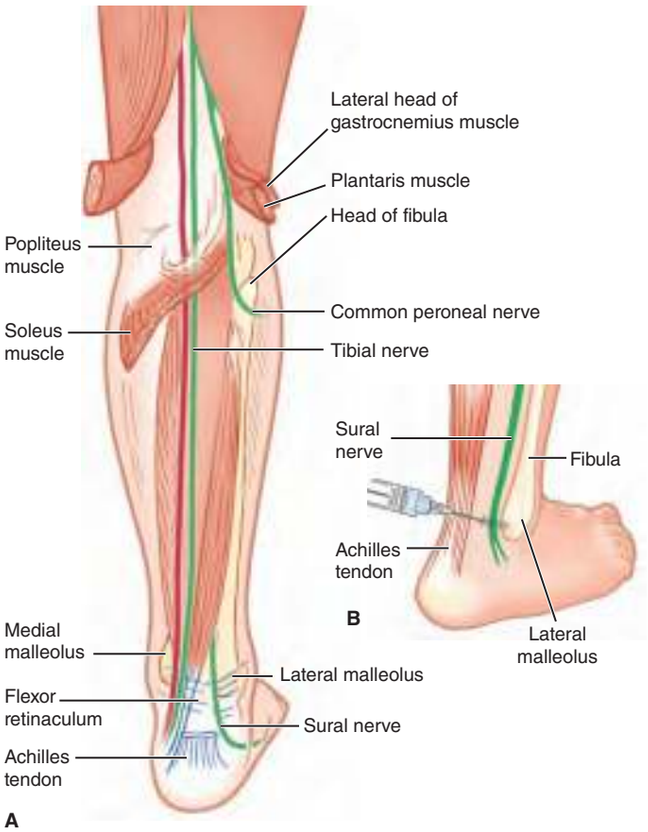


FIGURE 156-39. The sural nerve block. ***A.*** The course of the sural nerve. ***B.*** Blockade of the sural nerve.

entire length of the needle as it is inserted and contacts the posterior aspect of the lateral malleolus. Aspirate to ensure the needle tip is not in a blood vessel. Inject 3 to 5 mL of local anesthetic solution posterior to the lateral malleolus.

Remarks: **Ensure that the needle is not within the Achilles tendon before injecting the local anesthetic solution.**

POSTERIOR TIBIAL NERVE BLOCK

Anatomy: The posterior tibial nerve travels in the posterior leg and exists medial to the Achilles tendon several centimeters above the ankle. It is superficial at the ankle, midway between the medial malleolus and the heel (**Figure 156-40**). It lies between the tendons of the flexor digitorum longus and flexor hallucis longus muscles. It travels with and slightly posterior to the posterior tibial artery. The posterior tibial nerve divides at the inferior border of the calcaneus to form the medial and lateral plantar nerves. It provides motor innervation to the intrinsic foot muscles. The lateral plantar nerve provides sensory innervation to the lateral one-third of the sole and plantar surface of the lateral one and one-half toes (**Figure 156-27**). The medial plantar nerve provides sensory innervation to the medial two-thirds of the sole and the plantar surface of the medial three and one-half toes (**Figure 156-27**).

Patient positioning: Place the patient supine with their ankle supported on a pillow or blanket and the leg externally rotated.

Landmarks: Identify the medial malleolus by palpation and the posterior tibial artery by its pulsation.

Needle insertion and direction: Place a skin wheal of local anesthetic solution, at the level of the upper border of the medial malleolus just posterior to the posterior tibial artery or medial to the Achilles tendon. Insert a 25 gauge needle through the skin wheal and perpendicular to the skin. Advance the needle to the tibia or until paresthesias are elicited. If paresthesias are elicited, withdraw the needle 2 mm and allow them to resolve. Inject 3 to 5 mL of local anesthetic solution. If paresthesias are not elicited, infiltrate 5 to 7 mL of local anesthetic solution starting against the posterior tibia as the needle is withdrawn.

US-guided block: The nerve can be blocked with the aid of US.[58,59] Place the US transducer behind the medial malleolus (**Figure 156-40*B***). Use color Doppler to confirm the location of the posterior tibial artery. Identify the tendon of the flexor hallucis longus lateral to the tibial nerve (**Figure 156-40*C***). Place a skin wheal of local anesthetic solution. Place the needle lateral to the long axis of the US transducer (**Figure 156-40*B***). Insert the needle in the plane of the long axis of the US beam. Visualize the entire length of the needle as its tip reaches the tibial nerve. Aspirate to ensure the needle tip is not in a blood vessel. Inject 3 to 5 mL of local anesthetic solution around the tibial nerve to produce the "donut sign."

Remarks: This block is especially useful before exploring puncture wounds, repairing lacerations, or removing foreign bodies from the sole.

ANKLE BLOCK

The ankle block is performed to achieve complete anesthesia of the foot. It requires anesthesia of the posterior tibial nerve, the sural nerve, the superficial peroneal nerve, the deep peroneal nerve, and the saphenous nerve (**Figure 156-41*A***). The ankle can be thought of as diamond-shaped in cross-section (**Figure 156-41*B***). This diamond requires three subcutaneous and two deep injections (**Figure 156-41*B***). The subcutaneous injections are to anesthetize the sural, superficial peroneal, and saphenous nerves. The deep injections are to anesthetize the posterior tibial and deep peroneal nerves. The techniques for these individual nerve block injections were described previously.

DIGITAL BLOCK OF THE TOE

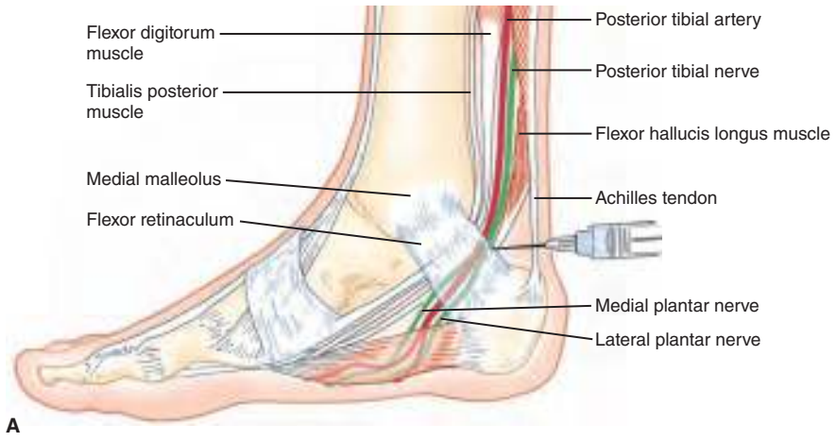
Anatomy: Two dorsal and two volar nerves supply each toe.[60] These nerves branch from the major nerves of the ankle. The dorsal digital nerves are the terminal branches of the deep and superficial peroneal nerves. The volar nerves are branches of the posterior tibial and sural nerves. The nerves lie in the 2, 4, 8, and 10 o'clock positions.

Patient positioning: Place the patient supine with their hip and knee flexed so that the sole of the foot is flat against the examination table or gurney.

Landmarks: Locate the dorsal aspect of the base of the toe to be anesthetized.

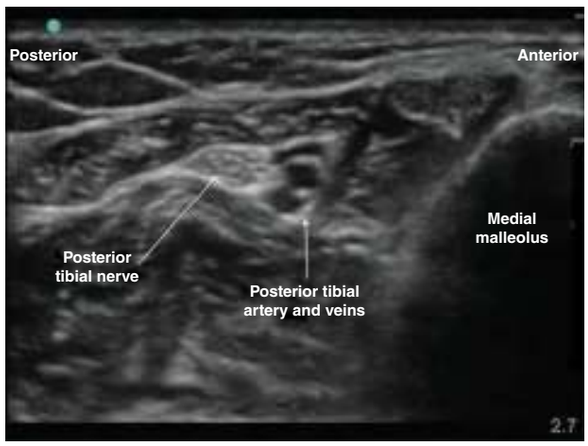
Needle insertion and direction: Place a skin wheal of local anesthetic along the dorsolateral or dorsomedial aspect of the toe. Insert a 25 gauge needle through the skin wheal. Perform the three-sided ring block (**Figure 156-42*A***). Direct the needle across the dorsum of the toe and infiltrate 1.0 to 1.5 mL of local anesthetic solution (**Figure 156-42*A*-*1***). Withdraw the needle and reinsert it on the medial aspect of the toe while infiltrating with 1.0 to 1.5 mL of local anesthetic solution (**Figure 156-42*A*-2**). Repeat the infiltration on the lateral aspect of the toe (**Figure 156-42*A*-3**). Anesthesia should be complete within 10 minutes. The great toe, due to its unique nerve supply, requires an additional anesthetic injection on the plantar aspect (**Figure 156-42*B***).

Remarks: This technique is commonly employed in the Emergency Department. The indications include repair of lacerations,



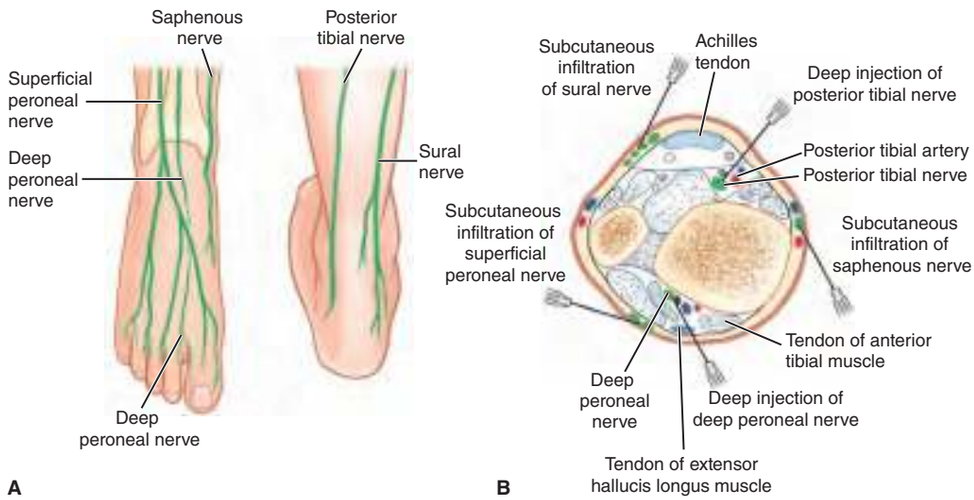
A

B



C

FIGURE 156-40. The posterior tibial nerve block. ***A.*** Artist illustration. ***B.*** US needle and transducer placement. ***C.*** US image of the posterior tibial nerve.



A B

FIGURE 156-41. The ankle block. ***A.*** The five nerves that provide sensory innervation to the foot. ***B.*** Blockade of the five nerves.

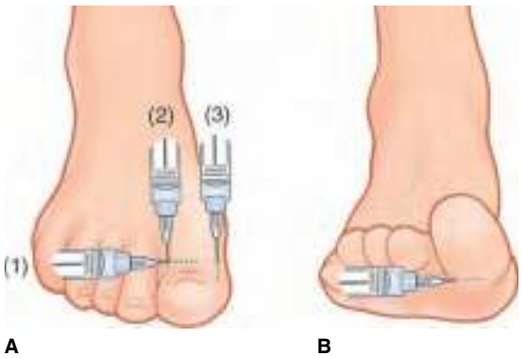


FIGURE 156-42. The digital block of the toe. ***A.*** The three-sided ring block may be used on any of the toes. ***B.*** The great toe requires an additional injection to achieve anesthesia.

incision and drainage of infections, removal of toenails, manipulations of fractures and dislocations, and painful procedures requiring anesthesia. Other techniques to anesthetize the toes are similar to those of anesthetizing the fingers (**Figure 156-24**).

ASSESSMENT

Allow 5 to 10 minutes for most regional anesthesia blocks to take effect. Up to 20 minutes may be required for blockade of major nerves (i.e., axillary, femoral, sciatic, and popliteal fossa). **Incomplete or inadequate anesthesia is the result of not properly identifying the anatomic landmarks or not inserting the needle properly. The procedure may be repeated if the administration of a second dose of local anesthetic solution does not result in the patient receiving a toxic dose of the anesthetic agent (Table 156-2).**

AFTERCARE

Perform frequent neurovascular checks until baseline function has returned. Injury to the anesthetized limb can result if the patient is permitted to use it, to use heat or cold application, or to perform wound care before the anesthesia has worn off.[21] If extensive or major nerve blockade was performed, discharge the patient home only after the sensation and function have returned to baseline. The patient may be discharged home immediately after minor blockade but should be properly cautioned. Avoid compression dressings as they may result in ischemia which is improperly sensed by the anesthetized area or limb.

COMPLICATIONS

Complications can occur from the injection of local anesthetic solution, most of which are minor. Significant complications are rare; yet, they do occur. Complications generally result from poor technique. General precautions include measures to minimize nerve injury, intravascular injection, and systemic toxicity. The Emergency Physician must be prepared to institute quick and immediate abortive care, cardiac monitoring, and airway control if complications do arise.

ALLERGIC REACTIONS

Allergic reactions can occur from hypersensitivity to the local anesthetic solution. Symptoms can range from mild itching and urticaria to circulatory collapse and death. Severe allergic reactions are extremely rare but may occur. The preservative in the local anesthetic solution is often the cause of an allergic reaction. Local anesthetic solutions containing no preservatives are an alternative. One example is "cardiac" lidocaine that is used for Advanced Cardiac Life Support (ACLS) protocols. Topical ice or vapor coolant is an acceptable alternative to nothing if one is concerned about a potential allergic reaction from the local anesthetic solutions. A solution of 1% to 2% diphenhydramine can also be used as an injectable local anesthetic.[8] Refer to Chapters 153 and 154 for the complete details of alternative anesthetic techniques.

HEMORRHAGE

Significant bleeding is extremely rare. Injection of a local anesthetic solution can be performed safely in patients who are anticoagulated or have a bleeding disorder.[8] A hematoma may commonly develop due to an arterial puncture. The application of direct pressure can be helpful if significant external hemorrhage occurs. However, if bleeding continues, treatment may be required to reverse the anticoagulant or replace clotting factors.

OVERDOSAGE

Central nervous system (CNS) complications primarily result from local anesthetic toxicity. These symptoms range from tremors to convulsions. The most severe complication is respiratory compromise resulting in intubation. The maximum dose of local anesthetic solution should not exceed the doses listed in **Table 156-2**.[8,10] Toxicity after intravascular injection requires even less anesthetic than subcutaneous infiltration. Refer to Chapter 153 for the details of local anesthetic toxicity and its management.

INFECTION

Infection can occur when the needle penetrates unclean skin. The risk of infection is significantly reduced if proper aseptic technique is used. The risk of infection is negligible when the skin is properly cleansed, sterile technique is used, puncture through obviously infected skin is avoided, and penetrating the needle through a skin lesion that may harbor microorganisms is avoided.

VASOVAGAL REACTIONS

The patient may experience an increase in vagal tone from apprehension, needle phobias, and/or pain. Vasovagal reactions are relatively common and may be associated with light-headedness and/or fainting. **Always perform regional anesthesia with the patient on a stretcher or in a chair that reclines to prevent secondary injury.** Vasovagal reactions are self-limited and only require reassurance.[8]

NERVE INJURY

Inflammation of the nerve is the most common nerve injury seen after regional anesthesia. Neuritis is a rare complication. Patients may complain of paresthesias, motor deficits, and/or sensory deficits. Most cases are transient and resolve completely, requiring only supportive care and close follow-up. Nerve damage results from direct needle trauma, ischemia due to intraneural injection, and chemical irritation from the anesthetic solution.[60,61] Proper needle style, positioning, and manipulation can minimize direct nerve damage. Intraneural injection can cause nerve ischemia with resultant injury. **Elicitation of paresthesias or severe pain indicates that**

the needle has made contact with the nerve. Withdraw the needle slightly and allow the paresthesias to resolve before injecting the local anesthetic solution. Concentrated anesthetics can produce chemical irritation of the nerve.

COMPARTMENT SYNDROME

A compartment syndrome is more frequently associated with the injury, not the regional nerve block, in most cases.[62,63] A concern is that the regional nerve block will affect the patient recognizing the changes of a developing compartment syndrome. Refer to Chapters 93 and 94 for a more detailed discussion of a compartment syndrome.

INTRAVASCULAR INJECTION

Intravascular injection results in both systemic and limb toxicity. Inadvertent intravascular injection produces high blood levels of the anesthetic agent and resultant toxicity. Particular care must be taken when administering large amounts of local anesthetic solution in close proximity to large blood vessels.

Intraarterial injection of local anesthetic solution containing epinephrine may cause peripheral vasospasm and subsequent ischemia that further compromises injured tissue. The local anesthetic solution is not toxic to the limb itself, although it may produce transient blanching of the skin by displacing blood from the vascular tree. Alpha-adrenergic antagonists have been used with success in relieving arterial vasospasm secondary to intraarterial injection of local anesthetics.[64]

ULTRASOUND-ASSOCIATED COMPLICATIONS

The use of US during the establishment of regional nerve blocks is associated with several complications and pitfalls. The chance of a nerve injury, intraneural injection, or intravascular injection is higher if a regular (i.e., cutting) needle is used. Always use a noncutting needle for nerve blocks. Ensure the identification of blood vessels by routinely turning on the power Doppler window when scanning for nerves. Accidental intravascular injection of large quantities of local anesthetic agents can lead to seizures, arrhythmias, apnea, and death. Failure to "pop" through the nerve sheath will not produce the "donut sign" and will likely lead to a failed block.

There is no standardized training for the Emergency Physician to learn how to perform US-guided nerve blocks. Regional anesthesia has evolved into an Anesthesia subspecialty and brachial plexus blocks can take considerable time to master. The use of phantom and simulation models for training, as well as careful supervision, are highly recommended. Novice sonographers may want to start with forearm, femoral nerve, popliteal, and leg blocks before tackling brachial plexus blocks.

SUMMARY

Regional anesthesia can serve as a valuable adjunct to the Emergency Physician's armamentarium. It is used for wound anesthesia prior to exploration, irrigation, debridement, and repair. Regional anesthesia may also be used to reduce the pain of procedures such as reduction of fractures and dislocations. Nerve blocks are especially useful when pain-sensitive structures such as fingers, toes, hands, and feet are involved. The application of regional anesthesia is frequently overlooked and underutilized in the emergency care of not only adults but even more so in the case of children. The use of regional nerve blocks is simple, safe, and effective for providing analgesia in the Emergency Department.

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157 Intravenous Regional Anesthesia

Christopher Freeman and Emily Cooper

INTRODUCTION

The technique of intravenous regional anesthesia (IVRA) was first introduced by August Bier in 1908.[1] IVRA essentially consists of injecting local anesthetic solution into the venous system of an extremity that has been exsanguinated by compression and/or gravity and isolated from the central circulation by means of a tourniquet. Procaine in concentrations of 0.25% to 0.5% was injected through an intravenous cannula placed between two Esmarch bandages used as tourniquets to divide the arm into proximal and distal compartments in Bier's original technique.[2-4] He noted two distinct types of anesthesia. The first was an almost immediate onset of "direct" anesthesia between the two tourniquets. An "indirect" anesthesia distal to the distally placed tourniquet was noted after a delay of 5 to 7 minutes. This technique was eventually renamed the Bier block.

Bier performed dissections of the venous system of the upper extremity in cadavers after injecting methylene blue. He was able to determine that the "direct" anesthesia was the result of the local anesthetic agent bathing bare nerve endings in the tissues. The "indirect" anesthesia was most probably due to the local anesthetic agent being transported into the substance of the nerves via the vasa nervorum where a conduction block is affected. Bier's conclusion was that there were two mechanisms of anesthesia associated with his technique: a peripheral infiltration block and a conduction block.

The only major modification of Bier's technique in the past 100 years has been the development of the double tourniquet technique in current clinical practice.[5-7] The Bier block is appropriate for brief surgical procedures of the upper or lower extremity. The technique has certainly gained its greatest acceptance for use in the upper extremity as tourniquet problems and safety issues seem to arise more frequently when IVRA is undertaken in the lower extremity.

The Bier block is a procedure that has found utility as a treatment adjunct for patients suffering from complex regional pain syndromes (CRPS; formerly known as reflex sympathetic dystrophy [RSD] or sympathetically maintained pain) as an alternative to repeated sympathetic blocks. Chemical sympathectomy using IVRA with agents such as guanethidine or bretylium may last up to 5 days as compared to local anesthetic blocks that typically provide analgesia lasting only hours.

ANATOMY AND PATHOPHYSIOLOGY

Lidocaine is the most commonly used local anesthetic agent for IVRA in the United States. Prilocaine is more routinely chosen in Europe. Prilocaine is metabolized to orthotoluidine, an oxidizing compound capable of converting hemoglobin to methemoglobin. This is usually only of concern when the dose of prilocaine is greater than 600 mg. Lower extremity IVRA using volumes up to 100 mL does not approach this dosage.

The usual dose of lidocaine to administer is approximately 3 mg/kg. This is a relatively large dose in terms of potential systemic toxicity. Systemic toxic reactions can and do occur due to leakage past the tourniquet, sudden accidental deflation of the tourniquet during the procedure, or intentional deflation following brief surgical procedures.[8,9]

An alternative dose of 1.5 mg/kg of lidocaine to a maximum of 100 mg has been shown to be effective in 95% of patients for reduction of closed fractures.[10] Another study demonstrated that a single forearm tourniquet and 150 mg of lidocaine in adults provided superior analgesia compared with the standard upper arm tourniquet and 300 mg lidocaine dose.[11] The advantage of this decreased dose of lidocaine is the greater therapeutic window and decreased chance of toxicity from accidental or early tourniquet release.

There are multiple studies looking at adjuvant medicine added to local anesthetic agents.[12-36] These adjuvants have the theoretical benefit of improved pain control both during and after the procedure as well as decreased risk of systemic toxicity from the local anesthetic agents as a result of the decreased required dose. Agents studied include acetaminophen, benzodiazepines, bicarbonate, clonidine, dexmedetomidine, dexamethasone, ketorolac, and opiates among others.

Opiate receptors exist in the peripheral nervous system.[12,13] It has been demonstrated that opioids may produce effective, long-lasting analgesia when injected with local anesthetics for brachial plexus blockade.[14-18] Several investigators have attempted to decrease the potential for lidocaine toxicity by adding opioids to reduce the volume of lidocaine. The addition of fentanyl to lidocaine for IVRA results in improved analgesia while reducing the risks of lidocaine toxicity.[19,20] Other investigators have found that adding an opioid and a muscle relaxant to 0.25% lidocaine provides the same analgesia and muscular relaxation as 0.5% lidocaine alone and reduces the likelihood of systemic toxicity.

Adjuvants added to 0.25% lidocaine have included fentanyl plus pancuronium, fentanyl plus rocuronium, and fentanyl plus D-tubocurarine.[21-24] The authors reported outstanding operating conditions in each of these cases. The lidocaine concentration was reduced in half and the potential for systemic toxicity was halved. A small dose of any nondepolarizing muscle relaxant may be chosen as an adjunct to the local anesthetic. **Avoid using D-tubocurarine as it releases histamine.**

Other agents used in an attempt to improve IVRA have included bicarbonate, butorphanol, clonidine, dexmedetomidine, dexamethasone, ketorolac, and ondansetron.[25-30] Adding ketorolac to the IVRA leads to less tourniquet pain and improved postoperative analgesia.[25-27] Clonidine and dexmedetomidine, both α-2-adrenoceptor agonists, have shown some benefit in decreasing tourniquet pain and in improving postoperative analgesia.[31,32] Both have the possible side effects of sedation and hypotension upon cuff deflation which may limit their clinical use. In limited studies, dexamethasone 8 mg intravenously has shown some efficacy in improved postoperative analgesia.[27,33] Alkalization of the local anesthetic with bicarbonate may improve pain upon injection.[34,35] This has not been demonstrated to hasten the onset or prolong the duration of anesthesia. Midazolam (40 to 50 μg/kg) has shown some benefit in decreasing intraoperative as well as postoperative pain.[36] Of all the agents described above, ketorolac 20 to 60 mg intravenously seems to have the largest clinical benefit with the least side effects.

Prilocaine is usually selected for IVRA in countries outside the United States. The addition of opioids to prilocaine has not been shown to improve success with the technique.[37-39] The addition of bicarbonate to prilocaine shortens the onset time of anesthesia and prolongs the duration of anesthesia.[40,41] The addition of clonidine to prilocaine dramatically suppresses tourniquet pain but does not alter postoperative pain following tourniquet deflation.[42]

The addition of long-acting and potent lipophilic opioids with agonist-antagonist activity (e.g., buprenorphine) to local anesthetics administered for brachial plexus blockade has been demonstrated to provide effective anesthesia and long-lasting postblock analgesia. This technique is a single-shot procedure and may supplant other methods of anesthetizing the upper extremity and perhaps the lower extremity. It offers a method of prolonging pain management long into the recovery period which IVRA does not.[18]

INDICATIONS

IVRA is appropriate for procedures, surgeries, and manipulation of the extremities requiring anesthesia of up to 1 hour in duration. The advantages are numerous (**Table 157-1**). It is most suited for laceration repair, reduction of fractures and dislocations, burn care, and minor soft tissue procedures in the Emergency Department.[43-47] The necessity of exsanguinating the extremity is a potentially painful maneuver that may preclude certain procedures from being undertaken with this technique. The Bier block is acceptable in the anticoagulated patient, a feature that distinguishes this technique from other regional anesthesia procedures (e.g., spinal block, epidural block, or plexus blocks).

CONTRAINDICATIONS

The only absolute contraindications for IVRA is patient refusal and allergy to local anesthetic agents. Relative contraindications include crush injuries of an extremity, the inability to obtain peripheral

TABLE 157-1 Advantages of IVRA

Always gets muscular relaxation
Can be used in patients over the age of 2 years (14 kg)
Does not require specific anatomic knowledge
Duration of action is related to on-time of tourniquet
Easy technique
Effective in 96% to 100% of cases
Low cost compared to other sedation techniques
Low toxicity
Minimal adverse effects
Minimal complications
Minimal supplementary drugs required
Neurologic examination possible after procedure
No restrictions on preoperative states
Quick offset
Quick onset
Short Emergency Department stay
Short recovery time allows examination
Well accepted by patients

venous access in the affected extremity, uncontrolled hypertension, local skin infections, cellulitis, and compound fractures. Do not perform IVRA in a patient with prior intravascular thrombosis or with conditions that predispose them to intravascular thrombosis (e.g., malignancies, Raynaud's disease, protein C or S deficiency). Patients with severe vascular injuries to the extremity requiring treatment are not suitable candidates for IVRA. Exclude patients with arteriovenous fistulas from consideration as well as anyone else in whom a tourniquet is unsuitable (e.g., severe peripheral vascular disease or progressive peripheral nerve disorders). The feasibility of using a pneumatic tourniquet in a patient with sickle cell disease must be balanced against the need for performing this type of anesthesia. Tourniquet use in sick patients may induce localized stasis of blood flow, acidosis, and hypoxemia with subsequent formation of sickle cells and a pain crisis. Liver disorders are relative contraindications. The local anesthetic is metabolized by the liver and can cause toxicity if it is metabolized slower.

Never perform IVRA using blood pressure cuffs if a pneumatic tourniquet is not available. Blood pressure cuffs are not designed to stay inflated for prolonged periods of time at high pressures. They can spontaneously deflate from a small leak and result in local anesthetic entering the central circulation and causing toxicity.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Alcohol swabs
- Gauze 4×4 squares
- Local anesthetic solution, 0.25% to 0.50% lidocaine hydrochloride or 0.50% prilocaine
- Penrose drain, 12 to 18 inches long and 7/8 inches wide
- 18 to 20 gauge angiocatheter
- 500 to 1000 mL bag of intravenous solution connected to an infusion set
- Intravenous catheter
- Crystalloid solution
- Intravenous infusion set
- Two single or one dual pneumatic tourniquet, size appropriate for the extremity
- Cotton cast padding
- Esmarch bandages, 60 inches long and 4 inches wide
- 50 mL Luer-lock syringe
- 100 mL sterile graduated measuring cup for mixing of solutions
- Adhesive tape
- Cardiac monitor
- Noninvasive blood pressure cuff
- Pulse oximeter
- Resuscitation equipment
- Adjuvants to local anesthesia (e.g., parenteral analgesics and sedatives)

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Discuss the discomfort that may be experienced during the procedure. Obtain an informed consent to perform the procedure in addition to a consent for the procedure to be performed under the influence of the Bier block. Place the patient supine on a gurney. Alternatively, place the patient in any other position so long as the vein selected for placement is readily accessible. Assemble all of the required equipment (**Figure 157-1**). Test the pneumatic tourniquets to ensure they remain inflated and do not have a leak.

Obtain intravenous access in the affected extremity with a saline-locked angiocatheter. The addition of intravenous extension tubing to the angiocatheter is optional and preferred by some Emergency Physicians. Place the angiocatheter in the dorsum of the hand for procedures involving the upper extremity. Place the angiocatheter in a foot, ankle, or lower leg vein for lower extremity procedures. Obtain a second intravenous access in a nonprocedural extremity. Alternatively, central venous access may be secured if required for other reasons.

Place the patient on the cardiac monitor, noninvasive blood pressure cuff, and pulse oximeter. Obtain, record, and assess baseline vital signs. Administer small aliquots of intravenous analgesics (i.e., 1 to 2 μg/kg fentanyl) if the patient is in severe pain to facilitate the exsanguination procedure. **Total patient cooperation is not essential to be successful. This method of anesthesia is not indicated if the patient is persistently combative and unable to follow commands.** Administer small doses of benzodiazepines (e.g., 15 to 25 μg/kg midazolam) for anxiolysis if needed. An important added benefit to choosing a benzodiazepine is the suppression of the usual convulsant response associated with local anesthetic toxicity, a valid concern in the patient receiving IVRA. **Check to ensure that resuscitative equipment is present and working properly. The time to look for resuscitative equipment is not when it is needed.**

UPPER EXTREMITY TECHNIQUE

APPLICATION OF THE PNEUMATIC TOURNIQUET

Apply two single tourniquets or a double pneumatic tourniquet over cotton cast padding (**Figure 157-2**). Place one single cuff or the double cuff high on the upper arm. Place the second single cuff above the angiocatheter and just below the proximal cuff. Elevate the arm for 1 minute. Tightly apply a rubber Esmarch bandage spirally around the arm starting at the hand and terminating at the distal cuff of the double tourniquet (**Figure 157-3**). The process of arm elevation followed by the application of an Esmarch bandage exsanguinates the arm. Place the tourniquets about the elbow and proximal forearm for procedures of the hand, wrist, and distal forearm (**Figure 157-4**).

Apply digital pressure to occlude the axillary artery. Inflate the proximal cuff of the tourniquet to between 50 and 100 mmHg above the patient's systolic blood pressure. **Wrap tape around the tourniquet to ensure there is not failure of the Velcro or manual ties and the cuff spontaneously opens allowing the local anesthetic solution to access the central circulation. It is important to compress the axillary artery both before and during the inflation of the pneumatic tourniquet.** Venous outflow is prevented before arterial inflow is occluded as the pressure in the tourniquet rises. Exsanguination of the extremity may be incomplete without occlusion of the arterial inflow before the tourniquet is inflated. Remove the Esmarch bandage. The arm should remain pale. Palpate the proximal cuff of the tourniquet to ensure it remains inflated.

It is essential to test for arterial occlusion by palpation of the radial artery before the injection of the local anesthetic solution. Confirm the absence of the radial pulse. Do not proceed with the procedure if the radial pulse is still present. Deflate the tourniquet and reattempt the procedure.

INJECTION OF THE LOCAL ANESTHETIC AGENT

Slowly inject 30 to 50 mL, maximum 3 mg/kg, of 0.5% lidocaine hydrochloride through the IV catheter in the arm were the tourniquet was applied. An alternative is to inject 10 to 15 mL of 1%

FIGURE 157-1. Essential equipment for IVRA. ***A.*** Artist rendition. ***B.*** An example of a pneumatic tourniquet machine. ***C.*** An example of a single cuff. ***D.*** An example of a double cuff. ***E.*** An example of a double cuff.

or 2% lidocaine. The precise volume depends upon the size of the arm being anesthetized and the maximal anesthetic dose based on the patient's weight (**Table 153-1**). **Slow and controlled injection rates are an absolute necessity to avoid the development of elevated venous pressures.** Remove the angiocatheter and apply pressure over the skin puncture site. The onset of anesthesia begins in approximately 5 to 7 minutes. Begin the procedure for which the IVRA was performed once adequate anesthesia is obtained.

If the patient develops tourniquet pain or the procedure duration is longer than 30 minutes, inflate the distal cuff to 100 mmHg above the patient's systolic blood pressure. Palpate the distal cuff of the tourniquet to ensure it remains inflated. **Wrap tape around the distal cuff of the tourniquet to ensure there is not failure of the Velcro or manual ties and it spontaneously opens allowing the local anesthetic solution to access the central circulation.** Remove the tape on the proximal cuff and slowly deflate it. Inflation

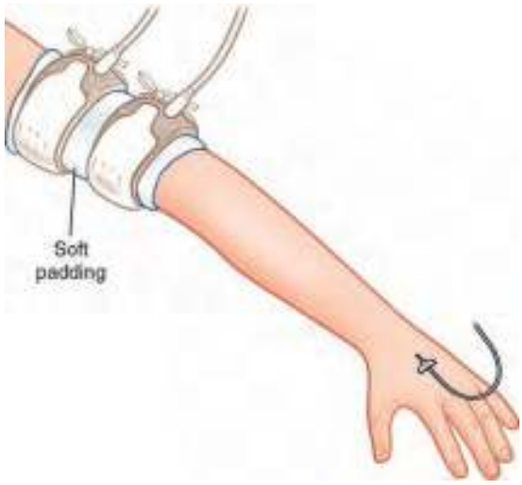

FIGURE 157-2. Preparatory steps. An angiocatheter is inserted into a dorsal hand vein. The double tourniquet is applied to the upper arm after the application of protective padding.

of the distal cuff over the anesthetized area and deflation of the proximal cuff will minimize the development of tourniquet pain.

LOWER EXTREMITY TECHNIQUE

The lower extremity requires double the anesthetic volume for IVRA and is otherwise completely analogous to the upper extremity. Obtain intravenous access in the lower extremity. Place the proximal single or double tourniquet just distal to the femoral pulse. Place the distal single tourniquet just above the site of the procedure. Elevate and exsanguinate the extremity. Apply digital pressure to the femoral artery while inflating the proximal tourniquet. Inject the local anesthetic agent. Inflate the distal tourniquet and deflate the proximal tourniquet. Perform the procedure for which IVRA was performed.

A modification of the above technique can be performed for procedures about the distal leg, ankle, and foot (**Figure 157-5**). Place the proximal tourniquet just above the knee joint and the distal tourniquet just below the knee joint. There is one major advantage of this technique. It allows the same volume of local anesthetic agent to be used as would be used for the upper extremity in the same patient. This is half of the volume required for the entire leg.

FIGURE 157-3. Exsanguination of the arm. The patient's arm is elevated and an Esmarch bandage is tightly applied.

FIGURE 157-4. A modified setup for procedures involving the hand, wrist, and distal forearm.

ALTERNATIVE TECHNIQUES

Some patients, especially those with painful fractures of the upper or lower extremity, may not be able to tolerate the placement of an Esmarch bandage for exsanguination of the extremity. It may

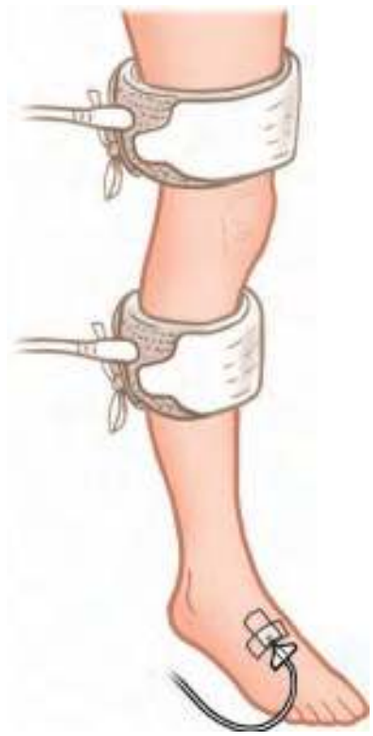

FIGURE 157-5. Placement of the double tourniquet for IVRA of the foot, ankle, and distal leg.

be completely appropriate to forego the Esmarch bandage. Elevate the extremity while occluding the axillary artery for a minimum of 5 minutes to affect the requisite venous drainage of the extremity before inflating the pneumatic tourniquet.

Exsanguination may be painlessly and effectively accomplished using a zippered pneumatic splint if simply elevating the extremity is not sufficient to affect this process and IVRA is still considered the technique of choice.[48] Apply two single tourniquets or the double tourniquet. Place the patient's extremity on the open splint and close the zipper. Inflate the splint to a pressure well above the patient's systolic blood pressure. Inflate the proximal cuff of the tourniquet. Deflate and remove the splint. The gradual inflation of the pneumatic splint is usually more comfortable whereas applying an Esmarch bandage to a painful fracture produces excessive pain. This improves the likelihood of the patient accepting the technique and enhances the chance for success with IVRA.

An alternative method for procedures involving the hand, wrist, and distal forearm is placing a single cuff on the proximal forearm (**Figure 157-4**). This technique has two main benefits. It allows for a lower dose of lidocaine (1.5 mg/kg to a maximum of 150 mg), which decreases the chance of systemic toxicity, it causes less pain at the tourniquet site, and less need for rescue medications during the procedure.[11,49] **This is the preferred method for injuries involving the hand, wrist, and distal forearm.** The remainder of the technique is the same as described for the upper extremity.

Studies have achieved success using a single tourniquet distal to the elbow for forearm IVRA. The smaller area allows for decreased lidocaine requirements, which lessens the concern for systemic toxicity after deflation. The smaller doses of lidocaine allow for decreased inflation time of the tourniquet, resulting in less tourniquet-related pain and faster overall procedure time. The risk of using a single tourniquet lies in the lack of a backup in case of tourniquet failure, leading to systemic dissemination of the anesthetic agent. More research must be done before the single tourniquet technique is appropriate for use in the Emergency Department.[11,49]

DEFLATION OF THE PNEUMATIC TOURNIQUET

Deflation of the tourniquet after the procedure has been performed is a critical step to minimize the possibility of toxicity associated with IVRA. It is absolutely mandatory that the tourniquet not be deflated unless at least 30 minutes have elapsed since the injection of the local anesthetic agent, even if the duration of surgery or manipulation has been very brief. At least one case of cardiac arrest has been reported when the tourniquet was released soon after the injection of the local anesthetic solution.[50]

It is absolutely mandatory to deflate the tourniquet in a cyclical fashion. Remove the tape from the cuff. **Deflate the cuff and immediately reinflate it to 50 to 100 mmHg above the patient's systolic blood pressure.** Observe the patient for 1 or 2 minutes and question them carefully for the occurrence of symptoms associated with local anesthetic toxicity (e.g., tinnitus, lightheadedness, or a metallic taste in their mouth). Signs of central nervous system stimulation may represent local anesthetic toxicity. If there are no signs or symptoms after approximately 1 or 2 minutes, deflate the cuff and once again immediately reinflate the cuff. Observe the patient for a period of approximately 1 to 2 minutes and question them again for the symptoms associated with local anesthetic toxicity. Repeat this process a third time. The tourniquet may be safely deflated and removed if there are no signs and symptoms of local anesthetic toxicity after three cycles of deflation and reinflation of the cuff. The safety of the cycled deflation and reinflation allows only a small fraction of the administered and unbound local anesthetic to enter the systemic circulation each time.[51] This minimizes the possibility of a sudden sustained increase in the blood level of the local anesthetic agent.[51]

ASSESSMENT

Patients must be carefully observed for the signs and symptoms of local anesthetic systemic toxicity (LAST) following injection of local anesthetic agents for IVRA. Remain in verbal contact with the patient. Continually monitor the patient's vital signs (i.e., cardiac monitor, blood pressure, pulse, and oxygen saturation). Adjuvants added to the local anesthetic agent may result in concomitant side effects or toxicities unrelated to the local anesthetic. Muscle relaxants can result in patients developing muscle weakness if they gain access directly into the systemic circulation. This may necessitate assisted ventilation, controlled ventilation, or the establishment of an airway if symptoms are severe. Opioids can result in signs of sedation and respiratory depression. This is quite rare following the small doses of opioid that would normally be administered as supplementation for IVRA.

Ensure that IVRA has been effective in providing appropriate analgesia for the intended surgery or manipulation to proceed uneventfully. The success rate with IVRA has been reported to be as high as 96% in one large series of patients.[52] Supplemental local anesthesia, intravenous sedatives and analgesics, or general anesthesia may be required if IVRA is unsuccessful or only partially effective in preventing nociceptive stimuli (i.e., pain) from being experienced by the patient.

Patients must be treated with standard basic and advanced cardiac and pulmonary life support protocols. Administer oxygen first if toxicity or complications develop following IVRA.

The patient assessment does not end after completing the mechanical portion of IVRA. Tourniquet problems may develop during the procedure and result in leakage of local anesthetic and/or adjuvant into the systemic circulation. **Release and deflation of the tourniquet may be associated with significant morbidity if strict adherence to the guidelines in this text are not followed.**

AFTERCARE

Carefully observe the patient after IVRA has been completed. It is important to keep the extremity relatively quiescent in the immediate postprocedural period. Remove the intravenous cannula and apply a sterile dressing over the injection site. Assess the extremity for signs of venous or arterial insufficiency every 30 minutes or at more frequent intervals if necessary. Peripheral nerve function will rapidly return following the deflation of the tourniquet and should be examined and documented. Continually monitor vital signs at intervals no longer than every 5 minutes for the first 30 minutes and as indicated by the patient's clinical status thereafter. Additional intravenous or intramuscular analgesics may be administered at this time if the patient is experiencing pain as IVRA affords no prolongation of antinociception once the tourniquet has been deflated.

Patients must be able to bear weight and ambulate (if appropriate) prior to being discharged if the lower extremity was chosen for IVRA. Patients may be discharged into the care of a responsible adult or back to their respective units once they meet the established criteria if there have been no untoward effects of the procedure and the IVRA. Instruct the patient regarding the use of the extremity depending, of course, on the nature of the intervention performed upon it.

Perform a detailed follow-up within 24 hours either by telephone for outpatients or in person for inpatients. Place the emphasis on peripheral sensory, motor, and sympathetic nerve function.

COMPLICATIONS

Local anesthetic agents have relatively narrow therapeutic indices. LAST involves primarily the central nervous system and the cardiovascular system. There may be localized neural and skeletal muscle irritation or allergic phenomena, all supporting vigilance following injection for IVRA. LAST usually progresses through several well-defined stages unless a gross overdosage has been directly administered systemically. These include numbness of the tongue and lightheadedness followed by visual and auditory disturbances. This progresses to muscular twitching, unconsciousness, convulsions, coma, respiratory depression, cardiovascular depression, and death in the absence of treatment. Reversal of the effects may be obtained by lipid emulsion infusion. Multiple case reports and animal studies support the use of lipid emulsion in patients with significant overdoses. The standard dose is 1.5 mL/kg of 20% lipid emulsion followed by an infusion of 0.25 to 0.5 mL/kg/min for 20 minutes.[53] Further information on lipid emulsion therapy can be found in Chapter 153 and at www.lipidrescue.org/.

These events progress along plasma concentrations of about 3 to 24 μg/mL of lidocaine for the most severe signs and symptoms. A correlation exists between the convulsive blood level of various local anesthetic agents and their relative anesthetic potencies. Prilocaine and lidocaine are on the lower, least potent, and least toxic end of that spectrum. The rate of injection and the rapidity with which a particular blood level is achieved will influence the toxicity of these agents.

The patient's acid-base status will have a profound influence on the central nervous system activity of the local anesthetic agent. Convulsive thresholds are inversely related to arterial $PaCO_2$. This further emphasizes the importance of continually assessing patients verbally and by noninvasive monitors following the injection of the local anesthetic. Avoid oversedation as it tends to result in respiratory depression and elevation of arterial CO_2.

Lidocaine is well known to depress the maximal rate of cardiac depolarization and cardiac contractility while not significantly altering the resting membrane potential of cardiac muscle. **Continually assess the cardiac monitor during and after the injection of lidocaine for IVRA.** Blood pressure monitoring is mandatory since local anesthetics exert a biphasic action on smooth muscle of peripheral blood vessels. Vasoconstriction is seen early followed by vasodilatation if levels continue to rise. Strict adherence to the recommended doses presented in this chapter will prevent most of the complications due to excessive dosing.

Lidocaine and prilocaine are both amino-amide–type local anesthetic agents having pKa values of 7.7 and are between 55% and 65% protein bound. They react rather similarly when used for IVRA. **Assess the patient for the development of methemoglobinemia if prilocaine is administered for IVRA.** The methemoglobinemia resulting from prilocaine is spontaneously reversible. It can alternatively be corrected by administering intravenous methylene blue. The incidence of allergic reactions associated with the use of lidocaine and prilocaine is not common because amino-amide agents are not derived from para-aminobenzoic acid (PABA). **Patients must be monitored for the development of allergic reactions following injection for IVRA.**

Complications due to IVRA may be classified as drug-related or tourniquet-related.[54] Drug-related complications depend both on the agent being administered directly into the vascular system and the equipment used to isolate the vascular space from the systemic circulation. Inadvertent deflation of the cuff, cuff failure, a sudden increase in venous pressure within the occluded tissue to a level higher than cuff pressure, and an intact interosseous circulation may all contribute to drug-related complications when using IVRA.

Lidocaine is the most commonly used local anesthetic for IVRA and is therefore the agent about which most complications have been reported. Lidocaine does not accumulate to any great extent at sodium channels at therapeutic plasma concentrations.[55,56] Toxic accumulation of the drug at the sodium channels is atypical since it rapidly binds to and dissociates from the channel.[55,56] Excessive plasma concentrations of lidocaine associated with intravenous boluses of large doses with a faulty tourniquet system result in peripheral vasodilatation and diminished cardiac contractility that manifests clinically as hypotension. The onset and termination of lidocaine anesthesia are relatively rapid.[57] The usual onset of IVRA using 0.5% lidocaine is approximately 4.5 ± 0.3 minutes. The termination of anesthesia once the tourniquet has been totally deflated is approximately 5.8 ± 0.5 minutes. There are usually no signs or symptoms of cardiovascular or central nervous system toxicity if the tourniquet is deflated at least 5 minutes after the drug is injected into the venous system, although tinnitus has been noted.[58]

Approximately 70% of the administered lidocaine dose remains within the tissues of the isolated limb after tourniquet deflation.[56] The remaining 30% enters the systemic circulation during the ensuing 45 minutes.[56] **Much more local anesthetic is released from the tissues of the isolated limb into the circulation after tourniquet deflation if the limb is inadvertently exercised. This emphasizes the importance of maintaining the previously anesthetized extremity quiescent following tourniquet deflation.**

The other commonly used local anesthetic agent for IVRA, prilocaine, is associated with the formation of methemoglobin in 4 to 8 hours after administration.[55] Significant methemoglobinemia has not been reported when prilocaine has been used for IVRA. Prilocaine administered for IVRA has an onset of analgesia in approximately 11 ± 6.8 minutes.[59] Termination of analgesia following tourniquet deflation averages 7.2 ± 4.6 minutes.[59] The use of prilocaine for IVRA appears to be extraordinarily safe. There were no serious side effects or deaths reported by using this technique in one survey of 45,000 prilocaine blocks.[60] The effectiveness of prilocaine seems to be equivalent to lidocaine when used for IVRA.

Opioids can be administered in combination with local anesthetic agents for IVRA in an attempt to prolong analgesia following cuff deflation. Occasional side effects typically attributed to opioids given systemically may be noted following cuff deflation. These include nausea, vomiting, and mild sedation.[19,37]

Neuromuscular blocking drugs can be administered in conjunction with local anesthetic agents to improve conditions for patients undergoing fracture reduction. There have been no reports of significant complications due to this adjuvant.

Clonidine and dexmedetomidine, α-2-adrenoceptors agonists, can be administered to decrease tourniquet pain during IVRA. The side effects include sedation and hypotension due to systemic absorption of the medication with cuff deflation which may limit their clinical use.

An intact tourniquet system is absolutely essential for the successful performance of IVRA. Unintentional deflation of the tourniquet or the presence of an arteriovenous communication with an intact tourniquet may result in serious sequelae. The tourniquet may be a source of complications. It may result in discomfort or ischemic pain. Systemic hypertension can occur from prolonged tourniquet inflation. Equipment misuse or malfunction is an important and avoidable source of complications. An intact and functioning tourniquet may be associated with leakage of local anesthetic agents from a supposedly isolated extremity into the systemic circulation.[52,61] Lower limb IVRA has almost a 100% incidence of local anesthetic leakage from beneath the tourniquet versus a 25% incidence for the upper extremity.[62] The use of IVRA for lower extremity analgesia had an associated incidence of a poor-quality block in

almost 40% of patients in one prospective study.[63] Local anesthetic may leak past an apparently fully functioning cuff due to the interosseous circulation that is not affected by occlusion of muscles and soft tissues. The functional significance of this circulation has been recognized for almost 35 years. It does not appear to be a significant factor in the production of complications due to IVRA.[64]

Tourniquet deflation after IVRA is associated with signs and symptoms of systemic local anesthetic toxicity ranging from mild central nervous system-related events (e.g., tinnitus and perioral numbness) to devastating cardiovascular collapse. These symptoms correlate with the local anesthetic concentrations in arterial blood and not to venous concentrations.[51,65] Intermittent cuff deflation may effectively prolong the time to achieve peak local anesthetic arterial concentrations but may not be entirely reliable in minimizing toxicity due to release of local anesthetic into the circulation.[51] **Do not deflate the tourniquet for at least 10 minutes, and for greater safety 30 minutes, from the time the local anesthetic is injected into the isolated venous system.**

Another complication of IVRA is tourniquet pain which commonly occurs.[52] We recommend the use of a tourniquet for any procedure performed under IVRA expected to last longer than 30 minutes.

There are very rare and isolated reports of neurologic complications associated with IVRA that include damage to the median, ulnar, and musculocutaneous nerves.[66] The etiology of such complications appears to be direct pressure from the tourniquet. These nerves subsequently exhibit histologic changes resembling crush injuries. It is recommended that the tourniquet time not exceed 2 hours to reduce the likelihood of capillary and muscle damage secondary to tissue acidosis.[66,67]

A compartment syndrome may rarely occur following IVRA. This is especially true when IVRA is used for reduction of long bone lower extremity fractures. It may be due to the large volume of anesthetic injected to effect analgesia and inadequate (i.e., incomplete) exsanguinations of the limb prior to performing the block.[68,69] A compartment syndrome has resulted from the inadvertent injection of hypertonic saline solution instead of local anesthetic solution.[70] One case report of a compartment syndrome resulted in the amputation of an arm in a patient who experienced thrombosis of the radial and ulnar arteries following IVRA after a relatively brief tourniquet occlusion time.[71] Whether this resulted from unsuspected intraarterial injection of drug, a drug administration error, or perhaps an idiosyncratic drug reaction is purely speculative.

SUMMARY

IVRA is a valuable adjunct to the armamentarium of Emergency Physicians dealing with the acutely injured patient. The simplicity of the technique and the relative safety if strict adherence to the previously listed rules is maintained make it an attractive alternative to a brachial plexus block, spinal block, or epidural block. Simply being able to identify a peripheral vein, secure intravenous access, and use a pneumatic tourniquet makes this one of the most user-friendly regional anesthetic procedures. One of the only potential downsides to IVRA is the very finite duration of anesthesia and the inability to prolong analgesia into the postoperative or postprocedural period. This procedure can safely be performed in the Emergency Department.[8,72,73]

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158 Nitrous Oxide Analgesia

Rene Ramirez and Leann Mainis

INTRODUCTION

Nitrous oxide (N_2O) has been used for more than 150 years in medical practice for its anesthetic, analgesic, and anxiolytic properties. Its ease of use, ability to be combined with or used in conjunction with other medications, relative low cost, rapid onset, and rapid elimination have likely contributed to nitrous oxide remaining one of the most commonly used anesthetics for medical and dental purposes.[1,2]

Joseph Priestly synthesized nitrous oxide in 1772 shortly after his discovery of oxygen. Humphry Davy was the first to identify the analgesic and anesthetic effects of nitrous oxide in 1799 and was the first to coin the term "laughing gas."[1] It remained largely a recreational drug for approximately 40 years despite noting its analgesic effects and predicting surgical applications. An American Dentist by the name of Horace Wells demonstrated clinical anesthesia for the first time in 1845 at Massachusetts General Hospital by using nitrous oxide and having one of his own teeth extracted. It was later recognized that the concentrations needed for anesthesia jeopardized patients because of their potential to cause hypoxia and lead to death.[1] Edmund Andrews added oxygen to the nitrous oxide mixture in 1868 to prevent the hypoxia that was commonly seen with the use of nitrous oxide. The first detailed analysis of nitrous oxide-oxygen mixtures as they apply to pain relief of angina without sedation or hypoxia was published by Stanislav Klikovich in 1881. In 1934, a self-administered apparatus of nitrous oxide with air was introduced to facilitate childbirth.[3] Seward improved on the self-administered apparatus in 1949 by adding oxygen instead of air to the nitrous oxide for a more prolonged analgesia and sedation during childbirth without the possibility of inducing hypoxia.[4] Ruben completed a study in 1969 of more than three million patients receiving nitrous oxide without a mishap.[5] Nitrous oxide has gained widespread acceptance since then and is the most frequently used inhalational anesthetic agent. It is used in conjunction with a volatile anesthetic gas since it only possesses weak anesthetic properties. Nitrous oxide has been used experimentally in animal models after resuscitation. It has been show to offer global neuroprotection due to its blockade of the *N*-methyl-D-aspartate (NMDA) receptor in the brain.[6]

Nitrous oxide-oxygen mixtures were first applied in an ambulatory setting in 1955. Dentists in Denmark used them for office-based procedures. A 50:50 mixture of nitrous oxide with oxygen (Entonox, Linde Healthcare Inc., Worsley, Manchester, United Kingdom) has been used by the British Ambulance Service in a self-administered format since 1970.[7] Nitrous oxide-oxygen mixtures became popular in the United States as a sedative and analgesic for use in the Emergency Department during the late 1970s.[8]

ANATOMY AND PATHOPHYSIOLOGY

Nitrous oxide is a colorless gas at room temperature. It has a pleasant slightly sweet odor and taste. Nitrous oxide is heavier than air and non-inflammable. **It is combustible when mixed with oxygen.** Nitrous oxide is poorly soluble with a high minimum alveolar concentration (MAC) value and is 34 times more soluble in plasma than nitrogen. It diffuses rapidly across biologic membranes into the bloodstream (e.g., lung-blood and blood-central nervous system). It results in a rapid onset in 30 to 60 seconds and has a short duration of action. Maximum effect occurs at approximately 2 minutes. Its duration of action lasts 2 to 5 minutes after administration is discontinued. **Nitrous oxide does not have significant cardiovascular or respiratory depressant effects. Nitrous oxide maintains protective airway reflexes when inhaled at levels below 50% and does not require fasting or postprocedure monitoring.**[9-11] Cortical function is depressed quickly after the start of administration which decreases all sensory input (i.e., hearing, pain, smell, taste, temperature, and touch).

Nitrous oxide displaces nitrogen and increases the volume of gas in body cavities (e.g., the gastrointestinal tract, middle ear, pleural spaces, and sinuses). It will result in an increased pressure within these confined spaces. Nitrous oxide is lipid insoluble resulting in minimal uptake by fat, muscle, and solid organs. **It has no excretion products and is eliminated unchanged through the lungs.** This makes it useful in patients with liver or kidney disorders.

Nitrous oxide acts supraspinally to induce analgesia by activation of opioidergic neurons in the periaqueductal gray matter and nonadrenergic neurons in the locus ceruleus of the brainstem.[12-14] This leads to disinhibition or activation of the descending noradrenergic inhibitory pathways via inhibition of γ-aminobutyric acid (GABA) interneurons. This results in a negative modulation of the nociceptive processes at the spinal cord level.[15] The predominant theory of the molecular mechanism to account for nitrous oxide's anesthetic action is a noncompetitive inhibition of the NMDA subtype of excitatory glutamate receptors.[16]

Nitrous oxide has the five actions of mild sedation, anxiolysis, mild to moderate analgesia, weak anesthesia, and mild dissociative effects.[17] Nitrous oxide has been shown to be effective in 85% of cases involving mild to moderate pain.[18] Nitrous oxide does not require intravenous (IV) access, a feature highly desirable by most patients. **The agent is a more potent anxiolytic than analgesic.** Anxiolysis is obtained by inducing a state of euphoria with a concurrent mild sedating effect. The analgesic effect is a result of an increase in the pain threshold. Inhalation of a 50:50 nitrous oxide-oxygen mixture has been reported to produce analgesia equivalent to 10 to 20 mg of morphine; however, its effects are highly variable.[8,19] **Combination therapy for painful procedures with an analgesic is often required due to its relatively weak analgesic effects.**[20] One example is that the infiltration of a local anesthetic solution for laceration repair is more tolerable under the use of nitrous oxide. Nitrous oxide may be used to decrease the dose of local anesthetic required for the procedure.

Nitrous oxide used in an ambulatory care setting (e.g., Emergency Departments and Dental Offices) is usually mixed with oxygen in a 50:50 mixture. The percentage of nitrous oxide is titrated up or down. Nitrous oxide becomes a more effective general anesthetic when concentrations exceed a 50:50 mixture. **Hypoxemia is a concern at general anesthetic doses.** The nitrous oxide-oxygen ratio must be adjusted for altitude due to a lower atmospheric pressure and a lower partial pressure of the gas. A 70:30 ratio of nitrous oxide to oxygen was effective in Denver with an elevation of 5000 feet.[21] The sex of the patient does not influence the response to nitrous oxide administration.[21]

Nitrous oxide is easy to obtain and usually requires no age limit to purchase. It can be purchased from head shops, kitchen stores, and the Internet. It is also available in balloons. It is highly abused by young individuals.[22-25] Users often binge frequently with nitrous oxide. Its effects are short-lived. Nitrous oxide is used in kitchen gadgets used to produce whipped cream, other whipped agents, and foams (**Figure 158-1*A***). Rechargeable units of nitrous oxide are sold as whippets (**Figure 158-1*B***) for use in kitchen gadgets. The whippets are abused. Nitrous oxide is the propellant in cans of whipped cream and other food substances. These items are purchased for abuse (**Figure 158-1*C***). Automotive kits of nitrous are sold to boost a car's performance (**Figure 158-1*D***). Abuse of nitrous oxide for euphoria can result in permanent neurologic deficits or death.[26]

INDICATIONS

Nitrous oxide is an attractive agent for procedural sedation due to its rapid onset and offset of sedation.[27] Nitrous oxide has been used by many types of medical and dental practices.[2] Nitrous oxide-oxygen mixtures have been shown to be effective in both the Emergency Department and the prehospital setting to alleviate anxiety and control mild to moderate pain. The indications for nitrous oxide use are reserved for patients with mild to moderate pain, who are anxious, or who will undergo a painful procedure. It has been useful in abscess drainage, angina pain, dental procedures, foreign body removal, fracture reduction, laceration repair, lumbar puncture, migraine, urethral catheterization, vascular access, and wound debridement.[19,27-35] A concentration of 70% nitrous oxide for 3 minutes can reduce venipuncture pain.[36] It can be used as an alternative to opioids.[37] A more detailed list of the uses of nitrous oxide is contained in **Table 158-1**.

CONTRAINDICATIONS

There are few contraindications to the use of nitrous oxide (**Table 158-2**).[2] Do not administer nitrous oxide to patients with significant respiratory compromise or heart failure requiring higher oxygen levels. **Normal cognitive function is required for its safe and effective use when using a self-administered nitrous oxide system.** Exclude patients with altered consciousness, head injuries, or the inability to understand instructions for administration. Children younger than 4 years of age cannot properly follow the instructions for the use of the system. Hysterical patients, somnolent patients, and those with altered mental status cannot comprehend and follow the instructions for the proper use of the system.

Nitrous oxide rapidly crosses membranes and exchanges with nitrogen. **Patients with risks for a closed nitrogen-containing space injury are not eligible candidates.**[38] This includes patients with an air embolism, bowel obstruction, emphysema, middle ear surgery, pneumocephalus, pneumoperitoneum, or a pneumothorax. **Rapid diffusion of nitrous oxide into a closed gas-containing space and its inability to leave the space quickly result in an increased volume of the space and an increased pressure within the space.** Patients may have intraocular gas bubbles placed in their eye by an Ophthalmologist in the management of retinal diseases.

FIGURE 158-1. Common nitrous oxide–containing devices are available to the public. ***A.*** Kitchen gadgets. ***B.*** Small charges of nitrous oxide or whippets. ***C.*** Huffing of nitrous oxide from whipped cream cans. (Photo courtesy of Cortland Preventive Services, New York, NY.) ***D.*** An automotive kit to boost performance.

These gas bubbles may persist for several months in the eye. Exposure to nitrous oxide can cause rapid expansion of these gas bubbles and may lead to blindness.[39] Exclude patients with decompression sickness or undergoing hyperbaric oxygen therapy.

Nitrous oxide oxidizes cobalamin and inactivates vitamin B_{12}.[38,40] Vitamin B_{12} is a cofactor of methionine synthetase and is an enzyme in folate and methionine metabolism.[41] Patients with methionine synthetase deficiency will have bad outcomes with nitrous oxide exposure. Inactivation of methionine synthetase is associated with increased plasma homocysteine concentrations which may increase the risk of postprocedural cardiovascular complications.[42] Nitrous oxide irreversibly inhibits deoxyribonucleic acid (DNA) synthesis.[43] Other inborn errors of metabolism that can result in serious outcomes when nitrous oxide is administered include homocystinuria and methylmalonic acidemia.[44] Patients with a vitamin B_{12} deficiency or an acquired vitamin B_{12} deficiency (e.g., resection of the terminal ileum or pernicious anemia) can develop leukopenia, megaloblastic anemia, or myeloneuropathy after receiving nitrous oxide.[38,45] Relative contraindications to the use of nitrous oxide include first-trimester pregnancy, myocardial ischemia, anesthesia over 6 hours, and pulmonary hypertension.[46]

EQUIPMENT

- Nitrous oxide tank
- Oxygen tank
- Mixing valve
- Scavenging device
- Demand valve apparatus
- Nasal mask
- Face mask
- Sensor to measure ambient levels of nitrous oxide

TABLE 158-1 Some of the Indications for Nitrous Oxide Administration

Acute pain	Joint dislocation reduction
Anginal chest pain	Joint injections
Anxiety relief	Labor pain
Biliary colic	Laceration repair
Bladder catheter insertion	Laser procedures
Burn care	Migraine headache
Cerumen impaction removal	Minor gynecologic procedures
Cervical and uterine procedures	Minor surgical procedures
Chronic pain	Musculoskeletal trauma
Cluster headache	Nasal surgery
Colonoscopy/sigmoidoscopy	Pancreatitis
Cystoscopy	Pelvic and physical examinations
Delivery/labor pain	Prehospital analgesia
Dental procedures	Renal colic
Dilation and curettage	Sickle cell pain crisis
Dressing changes	Tube thoracostomy
Foreign body removal	Vaginal examinations
Fracture reduction	Wound care
Incision and drainage of cysts or abscesses	Zipper entrapment
Intravenous access	

- Pulse oximeter
- Cardiac monitor
- Supplemental oxygen source
- IV catheters, tubing, and fluid
- Analgesic agents
- Anxiolytic-sedative agents
- Resuscitation equipment

A typical hospital-based device applicable for Emergency Department use is shown in **Figure 158-2**. The mixing valve ensures that a constant ratio or mixture of oxygen and nitrous oxide is delivered to the patient. The sources for the nitrous oxide-oxygen mixture can be individual cylinders or wall units. The device has a fail-safe mechanism that automatically stops gas flow when either source becomes empty.[47] The unit provides automatic oxygen enrichment at very shallow breathing rates.

TABLE 158-2 Contraindications to the Use of Nitrous Oxide

Abdominal pain of unknown etiology	Immunosuppression
Acute myocardial infarction	Inability to follow or understand instructions
Air embolism	Inner ear pain
Alcohol intoxication	Intoxication (alcohol or drugs)
Altered consciousness	Maxillofacial trauma or injuries
B_{12} deficiency	Methionine synthetase deficiency
Bowel obstruction	Methylmalonic acidemia
Chest trauma (blunt or penetrating)	Middle ear surgery
Chronic asthma	Overdoses
Chronic bronchitis	Pneumocephalus
Chronic obstructive pulmonary disease	Pneumoperitoneum
Congestive heart failure	Pneumothorax
Decompression sickness	Pregnancy, except labor
Drug ingestions	Psychiatric patients
Early pregnancy	Pulmonary edema
Emphysema	Respiratory compromise
Facial trauma	Shock
Folate deficiency	Small bowel obstruction
Head injuries	Treatment with bleomycin sulfate
Hollow viscus perforation	Unable to follow instructions
Homocystinuria	Unable to self-administer gasses
Hypotension	Young children
Hypoxemia	

It is required by the National Institute of Occupational Safety and Health (NIOSH) to have a sensor to monitor ambient levels of nitrous oxide within the treatment area. The scavenging device reduces the ambient levels of nitrous oxide within the treatment area. Elevated levels of nitrous oxide have been associated with a decreased fertility rate, an increased rate of spontaneous abortion, and neurologic disturbances among Dental Assistants.[48] Ambient levels of nitrous oxide were measured and noted to be 500 ppm after 8 minutes of patient administration.[49] The ambient levels in a similarly sized room were 0 when a scavenging device was used.[50]

Portable handheld units are available for use in clinics and ambulances (**Figure 158-3**). Many of these units are being used. Several companies have developed portable units. The nitrous oxide levels will vary in an ambulance with the airflow within the patient compartment. Trace levels of the gas are reduced to safe levels without the use of a scavenging device if the air-conditioning system or fans are operating and the ambulance is in motion.[51]

Entonox (Linde Healthcare Inc., Worsley, Manchester, United Kingdom) is a pre-prepared 50:50 mixture of nitrous oxide and oxygen. It is supplied in blue cylinders so it is not mistaken for another gas. It is quite popular with prehospital care providers due to its compact size and light weight. The main drawback is that only a fixed concentration of nitrous oxide can be administered. Oxygen is heavier than nitrous oxide. Invert the tank several times prior to use to mix the contents as the gasses can separate in cold weather. The patient can receive a higher concentration of oxygen at the start of using the tank and a higher concentration of nitrous oxide toward using the end of the tank if it is not inverted several times before use. This formulation of nitrous oxide is not currently approved for use in the United States by the Food and Drug Administration (FDA).

A face mask or nasal mask connected to a demand valve will help minimize the unwanted release of nitrous oxide into the treatment area (**Figure 158-4**). Use a nasal mask for procedures in and around the mouth. The face mask is more effective for pediatric patients (personal communication from M. Civitello, Porter Instruments). Dental offices usually use nasal masks without demand valves (personal communication from M. Civitello, Porter Instruments). The demand valve allows for the self-administration of the gaseous mixture. A negative inspiratory flow of –1 to –5 cmH_2O must be generated to activate the gas flow. An airtight seal is required between the patient's face and the mask to achieve a negative inspiratory flow. **The demand valve provides for a safe administration of nitrous oxide, minimizes the risk of oversedation, guards against human error, and protects against equipment failure. This demand valve system makes overdose almost impossible. The patient will be unable to hold the mask in position, will drop the mask, and will inhale room air as their level of consciousness declines.**

Many commercially available nitrous oxide administration devices do not have a demand valve and allow the nitrous oxide concentration to be adjusted from 0% to 70%. The advantages of these devices are the ability to control the percentage of nitrous oxide delivered and to make any adjustments based on the clinical situation. These devices have numerous disadvantages. The Emergency Physician must monitor the patient much more closely for the effects of hypoxemia, oversedation, and sedation. This requires the Emergency Physician to adjust the settings during the procedure for which the nitrous oxide is being used. **The lack of a demand valve can result in oversedation. The free flow of the nitrous oxide through the mask when the patient releases it results in increased concentrations in the procedure room which can affect the health care personnel.**

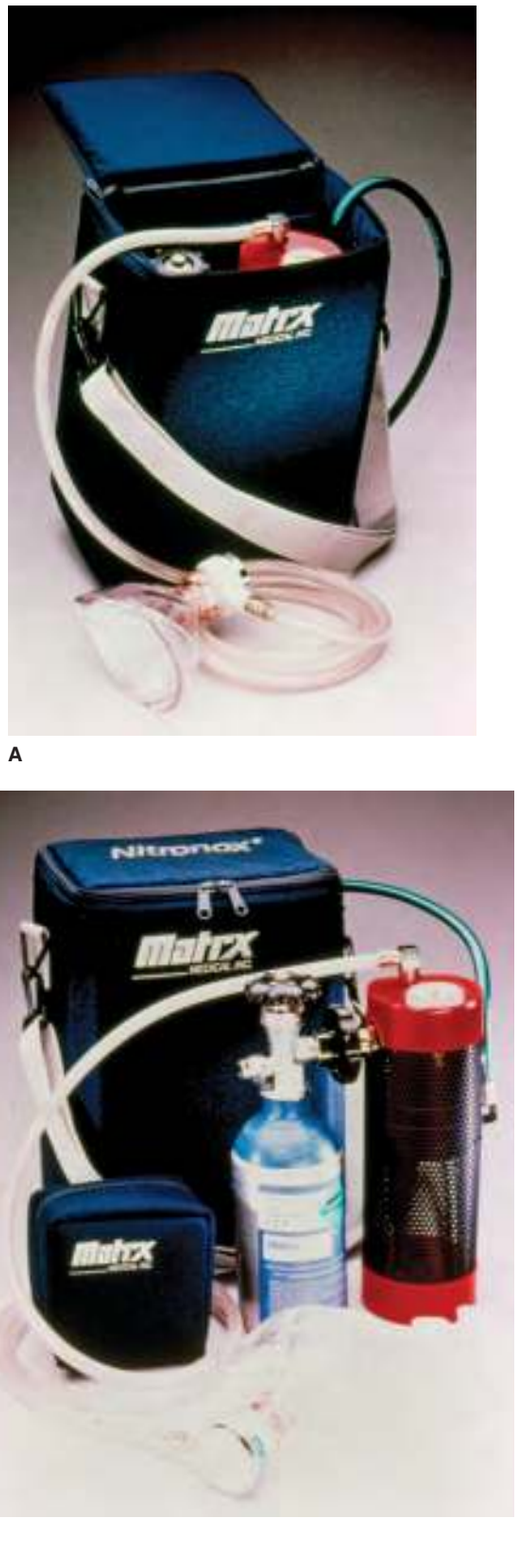
The INOmax DSIR Plus was developed for use in magnetic resonance imaging (MRI) rooms (Mallinckrodt, Dublin, Ireland) (**Figure 158-5**). It is often used in pediatrics to reduce pulmonary

FIGURE 158-2. An example of a nitrous oxide machine. (Photo courtesy of Porter Instruments, Hatfield, PA.)

hypertension. This usually requires the patient to temporarily discontinue nitrous oxide therapy while the they go to be scanned. This can result in an increase in pulmonary artery pressure and hypoxemia. The device can be used with MRI-compatible ventilators to continue the administration of nitrous oxide. It can also be used for the temporary sedation of patients receiving an MRI.

PATIENT PREPARATION

Inform the patient and/or their representative of the risks, benefits, potential complications, and alternatives to nitrous oxide anesthesia. Obtain a signed informed consent for the nitrous oxide anesthesia in addition to the consent for the procedure for which nitrous oxide is being administered. Establishing IV access for nitrous oxide administration is not required but is recommended. An IV can be



A

B

FIGURE 158-3. The portable Nitronox machine. ***A.*** The unit in a case. ***B.*** The unit removed from the case. (Photos courtesy of MDS Matrx, Orchard Park, NY.)

obtained after beginning the nitrous oxide administration to make the pain of catheter insertion much more tolerable. Apply noninvasive cardiac monitoring leads, a noninvasive blood pressure cuff, and pulse oximetry to the patient. Place the patient supine on a gurney

A

B

FIGURE 158-4. Self-administration of nitrous oxide. ***A.*** Use of a face mask. ***B.*** Use of a nasal mask. (Photos courtesy of www.thedrugclassroom.com.)

or sitting in a procedure (e.g., dental) chair that reclines. Preoxygenate the patient for 2 to 3 minutes with 100% oxygen. **Encourage the patient to remain calm and breathe in a controlled manner with special emphasis not to breathe too deeply or rapidly.**

TECHNIQUE

The nitrous oxide-oxygen gas mixture must be administered by appropriately licensed individuals, or under the direct supervision thereof, by state law. The Emergency Physician responsible for the treatment of the patient and/or the administration of the nitrous oxide-oxygen gas mixture must be trained in the use of the agents, the techniques to administer nitrous oxide, and the appropriate emergency response in the rare case of respiratory compromise.

Prepare the machine. A flow rate of 4 to 6 L/min of nitrous oxide is generally appropriate for most patients. Begin with 2 minutes of 100% oxygen followed by a 40% nitrous oxide mixture (i.e., 4 L/min of nitrous oxide and 6 L/min of oxygen). Increase the nitrous oxide in 10% intervals until the desired clinical effect and a maximum of

FIGURE 158-5. The INOmax DSIR Plus system nitrous oxide machine. (Photo courtesy of Mallinckrodt.)

70% nitrous oxide (i.e., 7 L/min of nitrous oxide and 3 L/min of oxygen). Most systems do not allow over 70% nitrous oxide to be administered. The concentration of nitrous oxide should not routinely exceed 50% during the administration. Titrate the nitrous oxide concentration to the clinical situation. It may be decreased during less painful procedures and increased during more painful procedures. **It is important to continuously monitor the patient's respiratory rate and level of consciousness during the procedure.**

The nitrous oxide-oxygen gas mixture can be administered via a nasal mask or a face mask. The nasal mask is used primarily by Dentists. It has been shown to be more effective in pediatric patients and patients undergoing procedures around the mouth and chin. The size and positioning of the nasal mask are important for ensuring a snug fit. Encourage the patient to breathe through their nose as opposed to their mouth in a controlled manner (i.e., not too fast or too deep).

The demand valve in the face mask system prevents gas flow unless a negative inspiratory pressure is applied with a self-administered face mask. The gas flows constantly if a demand valve is not used. The patient applies the face mask, ensuring an airtight seal that covers both the mouth and nose (**Figure 158-4*A***). Do not remove the face mask between breaths unless adverse effects are noted (e.g., dysphoria, headache, lightheadedness, nausea, vertigo, and vomiting). Remove the mask and administer 100% oxygen via a non-rebreather mask for 5 minutes or until the adverse symptoms resolve. The analgesic effects of nitrous oxide are commonly noted within 90 to 120 seconds of the onset of administration.[52] The patient becomes mildly sedated in 3 to 5 minutes. Supplemental analgesics (e.g., wound infiltration of a local anesthetic) may be administered as needed or the procedure can be performed.

It is difficult for a patient to overdose on nitrous oxide if the mask is not secured to their face with tape or a strap. The patient will often remove the hand and the mask from their face as a state of analgesia and euphoria develops. The mask will fall off or loosen if the seal is not maintained. The demand valve will not be triggered if the mask falls off the patient's face. If not using a demand valve, the gas flows into the room and not directly into the patient. **It is extremely important not to secure the mask to the patient's head with straps or tape so that nitrous oxide delivery will cease as the patient falls asleep.** The patient may reapply the mask and self-administer additional nitrous oxide upon awakening or experiencing pain. **It is extremely important to note the need for proper ventilation and/or a nitrous oxide scavenging system.** Nitrous oxide can build up in the treatment area affecting others aside from the intended patient without proper ventilation and/or scavenging.

Remove the face mask upon completion of the procedure. Administer 100% oxygen for 5 minutes. This increases the rate of elimination of nitrous oxide and minimizes the side effects (e.g., dizziness, dysphoria, headache, lightheadedness, nausea, and vomiting).[53] Record the amount of gas and the duration of time used in the patient's chart. Record this information in the log maintained with the nitrous oxide device. Clean the demand valve apparatus, if used, prior to and after each administration.

PEDIATRIC CONSIDERATIONS

The previously described preparation and technique apply to the pediatric patient with a few differences.[2,54-56] Explain how the gas works to the patient. Show the child the equipment and let them handle it to realize it is not scary. Demonstrate the use of the mask on yourself and the parent before demonstrating it on the child. A face mask is preferred over the nasal mask for young children as it is difficult for them to cooperate with not mouth breathing. The young child may have difficulty following instructions or using the face mask. Apply a flavored scent or flavored lip balm to the inside of the mask to make it more inviting for the child to use. A trained health care provider other than the Emergency Physician (e.g., another Emergency Physician, Nurse, Nurse Practitioner, Respiratory Therapist, or Physician Assistant) may be required to administer the nitrous oxide. This second provider can help hold the mask over the child's face and monitor the level of sedation to prevent oversedation. Concentrations up to 70% can be safely used.[57]

ASSESSMENT

Ensure that the patient is experiencing adequate analgesia before performing the procedure for which nitrous oxide is administered. Nitrous oxide analgesia is supposed to be the equivalent of administering 10 to 20 mg of morphine. Real use has shown that patients experience a wide range of pain relief (e.g., none, mild, moderate, or marked). Nitrous oxide administration may require supplementation with parenteral analgesics, parenteral sedatives, or other anesthetic techniques.

AFTERCARE

There is no aftercare related to the administration of nitrous oxide anesthesia other than 5 minutes of supplemental 100% oxygen via a nonrebreather mask. Continue to monitor the patient until they return to their pretreatment level of responsiveness. This often takes less than 10 minutes in almost all patients.

COMPLICATIONS

The side effects of a 50:50 nitrous oxide-oxygen mixture are relatively mild. Lightheadedness is most common with the occasional patient complaining of paresthesias or nausea. Donen and colleagues reported a 19% incidence of dizziness, lightheadedness, or vertigo; 4% incidence of paresthesias, headache, or amnesia; 5% incidence of nausea; and 1% incidence of vomiting.[7] Other reported side effects include abdominal pain, agitation, chest pains, desaturations, fatigue, hallucinations, hiccups, hyperventilation, irritability, and pallor.[27] **There is little to no risk of inducing anesthesia or losing protective reflexes if used in concentrations of 50% or less.** Commonly used procedural sedation medications (e.g., ketamine and propofol) have been associated with laryngospasm. It was previously thought that nitrous oxide did not have the need for advanced airway support in concentrations up to 70% and was not associated with laryngospasm.[27] The first known case report of laryngospasm associated with nitrous oxide was published in 2015.[58] **It is important when using any procedural sedation medication to always have advanced airway equipment readily available.**

The concept of diffusion hypoxia was first described by Fink in 1955.[59] He noted that the rapid diffusibility of nitrous oxide could displace oxygen from the alveoli after discontinuation of the gas.[59] This concept has led to the administration of 100% oxygen for 5 minutes following the nitrous oxide administration. There has been evidence to disprove diffusion hypoxia in low-dose nitrous oxide mixtures (e.g., 50:50 nitrous oxide-oxygen). It is only a legitimate concern at higher concentrations used during general anesthesia. **The use of 100% oxygen following the cessation of nitrous oxide is still suggested.** Caution is advised in patients with chronic obstructive pulmonary disease in whom the increased oxygen concentration found in the nitrous oxide-oxygen mixture can potentially depress the respiratory drive.

There has been growing concern regarding the safety of trace levels of nitrous oxide in the Operating Room, Emergency Department, and Dentist office.[47] Always use nitrous oxide in a well-ventilated treatment room. There is evidence to show that elevated levels of

nitrous oxide are associated with miscarriages, reduced fertility, and increased cervical cancer rates among female Dental Assistants.[1,48] Scavenging devices help reduce the ambient levels of nitrous oxide within treatment areas.[47,60] The demand valve system allows for all exhaled gases to escape into the surrounding environment without a scavenging device. A scavenging device will collect the exhaled gas and remove it from the patient care area.[50] Scavenging devices can maintain ambient levels of nitrous oxide below 1200 ppm by the guidelines proposed by NIOSH.[61] Nitrous oxide levels above 1200 ppm are considered a hazardous condition. Scavenging systems combined with air conditioning are considered the most effective means of lowering ambient nitrous oxide levels.[62]

There has been concern raised about the experimental use (i.e., abuse or diversion) of nitrous oxide gas by medical personnel. Prepare a strict protocol on the use of nitrous oxide and its associated demand valve apparatus prior to the implementation of nitrous oxide-based procedural sedation in the Emergency Department. Record the volume of gas administered in the patient's chart as well as in a log kept with the nitrous oxide device following each use of nitrous oxide. The demand valve apparatus can be safely secured in the narcotic cabinet or within a Pixis system until it is required for patient use.

There has been concern about the effects of nitrous oxide on the hematologic, immunologic, myocardial, neurologic, and reproductive systems. Nitrous oxide has been associated with anemia, birth defects, decreased neutrophil chemotaxis, decreased proliferation of mononuclear cells, increased spontaneous abortions, impaired wound healing, megaloblastic anemia, myocardial ischemia, polyneuropathy, reduced fertility, subacute combined neurodegeneration of the spinal cord, and thrombocytopenia.[24,25,40,44,63-69] Nitrous oxide-induced neurotoxicity has been implicated in the development of long-lasting cognitive deficits when administered to patients of extremes of age.[70]

SUMMARY

The use of a nitrous oxide-oxygen mixture has been shown to be safe, effective, and easy to administer in an ambulatory care setting. The gas provides rapid onset of sedation, hypnosis, and rapid offset. It is a mild anesthetic that helps to facilitate the performance of mild to moderately painful procedures. The adverse effects are minimal. The nitrous oxide gas is usually self-administered. Concerns of hemodynamic compromise are negligible. The use of nitrous oxide in a closed-space environment should be accompanied by a scavenging device to minimize ambient levels of the gas.

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159 Procedural Sedation and Analgesia (Conscious Sedation)

Hagop M. Afarian

INTRODUCTION

Procedural sedation and analgesia (PSA) techniques are an essential skill for any Emergency Physician. The daily practice of Emergency Medicine employs painful and anxiety-provoking measures to perform diagnostic testing or therapeutic interventions. PSA is a skill that may require a credentialing process at some institutions. It probably has evoked a written procedural guide in most hospitals and Emergency Departments, with or without the input from a hospital-wide PSA committee or the Department of Anesthesiology. PSA certification may require annual competency assessments in the form of a written examination or practical scenarios. PSA is a technique that probably receives a great deal of attention from the continuous quality improvement committee because of The Joint Commission's directive. It is a skill that, with proper training and well-designed application principles, will provide the patient and their families with a sense of compassion and caring for their physical and emotional distress.[1,2] PSA is a skill that may result in horrific outcomes when performed without anticipation of complications, appropriate training, knowledge, and risk-benefit analysis.[3]

An extensive spectrum of painful and anxiety-provoking clinical presentations is seen in an Emergency Department on any given day. There may be a dislocation reduction, a fracture reduction, a diagnostic lumbar puncture, a sexual assault examination on a child, or neuroimaging on a combative, head-injured patient. Each presentation has a separate subset of variables to consider prior to PSA. While Anesthesiologists are still considered the "experts" in sedation, they are not readily available to the Emergency Department's beck and call. Multiple guidelines for the use of PSA account for these limitations and are followed to ensure that safe effective care can be rendered to their patient populations.[4-10]

The environment of the Emergency Department is unique in many facets of PSA with situations being nonelective. Procedures are relatively brief and make Operating Room time neither timely nor cost effective. Intrinsic to PSA is the core training and frequent exposure. Who better to handle an untoward cardiopulmonary complication of a procedure than a specialist of airway management and resuscitation?

It is critical to the technique of PSA, including drug selection, to take a variety of parameters into consideration before the first medication is administered.[11] The potency and effectiveness of today's agents are a double-edged sword. They are invaluable with the correct selection and administration.[3] They are a recipe for disaster if risks are not appropriately identified and minimized.

Monitoring techniques (e.g., pulse oximetry and capnography) are effective adjuncts to procedural monitoring. They are no substitute for a trained and dedicated observer.

TERMINOLOGY

Textbooks and review articles use various definitions to define components of PSA, formerly known as conscious sedation.[12] Terms such as light and deep sedation are often applied to the extremes of the sedation continuum. **The important thing to realize is that sedation is a continuum.**[5,13] At the far left of the continuum is the alert and anxious patient. Eye opening, speech, and motor responses diminish as the sedation process proceeds. At the opposite end of the spectrum are CO_2 retention, hypoxia, hypotension, and finally death. These later endpoints are obviously not desirable. Anxiolysis and analgesia are other terms that need definition. The following terminology is representative of current PSA jargon.

Analgesia equates to pain relief without alteration in mental status. Analgesia is required in a variety of procedures in which painful manipulation or instrumentation is anticipated. Most potent pain medications will also have a component of sedation, especially the opioids. **The sedation component needs to be carefully considered during drug selection and dosing calculations to minimize the risk of overshooting the desired sedation endpoint.**

Anxiolysis means to relieve apprehension. Patients may be anxious for any variety of reasons besides the thought of a painful forthcoming procedure. Anxiolysis involves allaying the patient's fears. **The agents used for anxiolysis are pure sedatives and provide no pain relief.** Although some degree of sedation is to be expected, it should be minimized with appropriate drug and dosing selection.

Sedation blunts the patient's perception of the surroundings and pain with a depression in their state of wakefulness. Levels of procedural sedation do not follow a discrete stepwise progression. Sedation proceeds along a continuum ranging from a mild sedation through deep sedation into general anesthesia. The level of sedation is determined by a patient's state of anxiety, mobility, retention of protective reflexes, and wakefulness.

Dissociative sedation implies sedation, analgesia, amnesia, and the induction of a cataleptic state. This state preserves ventilatory drive and maintains protective reflexes while maintaining cardiovascular stability. **Ketamine is the primary agent used to induce this state.**

Neurolepsis is a reduced motor activity state in which the patient has reduced anxiety and indifference to their surroundings. Neurolepsis is best achieved with major tranquilizers (e.g., droperidol or haloperidol). Its application is best suited to the agitated or violent patient whose behavior places their self or others at risk for harm.

General anesthesia represents the extreme right of this continuum. The patient has no awareness of the environment and has lost the ability to self-maintain protective reflexes. This is not a desirable endpoint during PSA in the Emergency Department.

PSA is a technique of administering sedation or dissociative agents with or without analgesics to induce a state that allows the patient to tolerate unpleasant procedures while maintaining cardiorespiratory function. PSA produces a depressed level of consciousness which allows the patient to maintain airway control independently and continuously. The medications, doses, and techniques used are not likely to produce a loss of protective reflexes according to the American College of Emergency Physicians Clinical Policy for PSA in the Emergency Department.[4]

Standardizing levels of sedation along a continuum has been attempted with various scoring systems.[14] The Ramsay scale was specifically devoted to providing objective determinations of sedation during drug-induced sedation practices. Scoring consists of six sequential scoring levels (**Table 159-1**).

TABLE 159-1 The Ramsay Scale for Standardized Levels of Sedation[4]

Level	Clinical status	Sedation equivalent
1	Awake, anxious, agitated	None
2	Awake, cooperative, oriented, and tranquil	Anxiolysis
3	Awake, responds to commands only	Mild sedation
4	Asleep, responds to brisk stimuli	Moderate sedation
5	Asleep, sluggish response to stimuli	Deep sedation
6	Asleep, no response to stimulation	Anesthesia

Another scoring system is the Observer's Assessment of Alertness/Sedation (OAA/S) Scale. It was designed as a research tool for studies incorporating pharmacologic studies with benzodiazepines.[14] The scoring system was based upon the assessment of the patient for facial expressions, ocular appearance, responsiveness, and speech. The OAA/S Scale incorporates the patients' responsiveness to the effects of the agents given in contrast to the similar observational scoring used in the Ramsay scale.

INDICATIONS

A wide variety of clinical presentations and procedures would entail the appropriate use of analgesia, anxiolysis, dissociation, and sedation for case management (**Table 159-2**). PSA is particularly suited to pediatric patients requiring multiple painful procedures in the Emergency Department, especially if they can be done simultaneously or one after another. An example is the young child with a fever undergoing a septic work-up requiring lumbar puncture, urethral catheterization, and vascular access.

There are four goals of PSA. The first and foremost is to assure patient safety with a careful risk-benefit assessment and a well-defined clinical procedure to maximize benefit, limit risk, and foresee potential complications. Second is to appropriately assess and deliver adequate analgesia, anxiolysis, sedation, and amnesia as dictated by the patient's needs. Third is to consider the psychological impact of the forthcoming procedure and minimize the impact of these events. Fourth is to provide a fluid transition to the preprocedural physical and mental status while assuring a safe discharge and postprocedural observation.

TABLE 159-2 The Indications for PSA

Anxiolysis/mild sedation (analgesia minimal)	**Pediatric dissociative sedation**
Painless diagnostic studies, i.e., CT scanning	Multiple trauma procedures
Lumbar puncture	Fracture/dislocation reduction
Posterior nasal packing	Abscess incision and drainage
Pediatric foreign body removal	Paraphimosis reduction
Pediatric slit lamp examination	Complex facial lacerations
Mild sedation and analgesia	Tongue lacerations
Pediatric hand/finger injuries (with local analgesia)	Complex hand lacerations
Disimpaction	Burn debridement
Vaginal/rectal foreign bodies	Sexual assault examination
Moderate sedation and analgesia	**Adult dissociative sedation**
Traction splints for fractures	Asthma intubation
Burn debridement	Trauma resuscitation
Cardioversion	Hemodynamic instability requiring sedation
Fracture/joint reduction	Lengthy and painful procedures
Deep sedation and analgesia	
Incision and drainage perineal/perirectal abscesses	
Complex pediatric lacerations	
Extensive road rash debridement	

CT, computed tomography.

CONTRAINDICATIONS

There are three contraindications to the use of PSA. A known allergy to the individual medication(s) being considered is an absolute contraindication. Ketamine and nitrous oxide have agent-specific contraindications. The lack of experienced or credentialed personnel is essential. PSA requires personnel appropriately trained in airway management. Appropriate monitoring capabilities must be available (e.g., equipment monitors and personnel to observe the monitors, record procedural flow, and monitor postprocedural recovery).

Issues such as the time of the last oral intake should be considered in the risk-benefit analysis. **A full stomach does not constitute an absolute contraindication to PSA.**[15-17] Concomitant drug or alcohol use is a complicating variable that needs to be recognized and accounted for prior to the procedure. Complicated airway anatomy should receive attention in case emergent airway management is necessary.

EQUIPMENT

- Crash cart with resuscitation equipment
- Defibrillator
- Oxygen source
- Oxygen masks
- Nasal oxygen cannulas
- Oral airways
- Nasal airways
- Bag-valve-mask device
- Continuous pulse oximetry
- Capnography, if available
- Continuous cardiac monitoring
- Intravenous access supplies (Chapters 59 and 61)
- Suction source
- Suction catheters
- Pharmaceutical agents
- Reversal agents (i.e., naloxone and flumazenil)
- Succinylcholine
- Procedural supplies (e.g., suturing, splinting, etc.)

It is imperative that resuscitation equipment be immediately available prior to the onset of PSA. Make this requirement part of the preprocedural checklist. Age-appropriate equipment must be immediately available for children. The immediate availability of reversal agents is a prerequisite to the administration of opioids or benzodiazepines. Have succinylcholine readily available in case of laryngospasm or opioid-induced chest wall tightness.

PATIENT PREPARATION

INFORMED CONSENT

The issue of informed consent is institution specific (Chapter 1). Most institutions function under the premise that if a procedure has any significant level of risk inherent in its application, then informed consent regarding the risks and benefits of the procedure should be undertaken with the patient and/or their representative. The benefit to the patient is a careful calculated means at reducing the pain and anxiety associated with a planned diagnostic or therapeutic intervention. The risks are inherent in the medications selected, the patient's current physical condition, and complicating conditions (e.g., last meal, recent drug use, or recent alcohol use).

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Obtain a separate informed consent for the procedure for which PSA is being performed.

PERSONNEL COMPETENCY AND CREDENTIALING

Any Emergency Physician practicing PSA must be competent in airway management and resuscitation.[18] Competency credentialing is institution specific. Emergency Physicians may be granted privileges based on their residency training or current practice. Some institutions may mandate a written competency examination or advanced airway and resuscitation training (e.g., Advanced Cardiac Life Support certification).

Nursing personnel must be proficient in medication profiles, medication administration, and patient monitoring. The staff needs to be aware of policies and procedures. Competency testing may be a prerequisite to providing PSA. All personnel involved must meet these departmental and institutional requirements prior to initiation of PSA. More than one physician may be required for PSA in some complex cases, although there is no evidence to support dual-physician involvement for all cases.[19]

RISK-BENEFIT ASSESSMENT

The preprocedural assessment constitutes a risk assessment and a preprocedural baseline determination. Assessment requires a determination of the patient's current health and an evaluation of potential risk, adverse reactions, and procedural complications. The old adage "an ounce of prevention is worth a pound of cure" certainly applies with PSA. **Thorough preparation is critical to minimize patient risk**. Department procedural flowsheets, complete with preprocedural assessment checklists, can be a valuable adjunct.

Assign patients an American Society of Anesthesiologists (ASA) Physical Status Classification (**Table 159-3**).[13] Emergency Department PSA would normally be limited to class I or class II patients. Class III or class IV patients are best served in consultation with an Anesthesiologist. Never perform PSA on class V patients.

Perform a complete history and physical examination prior to the application of PSA. Direct the emphasis toward allergies to any analgesic or sedation agent. Previous anesthesia and related complications may be critical to drug selection and/or the involvement of an Anesthesiologist. **Assess the patient's airway. Age-appropriate airway management equipment must be immediately available in the event airway control becomes necessary**. A patient's breathing is an important continuous determinate that needs to be observed and recorded before the procedure, throughout the procedure, and during recovery. Hypoventilation or bronchospasm may be clinically evident with observation before an alteration in heart rate or pulse oximetry is detected. **The patient's state of wakefulness and the ability to follow commands are markers for sedation.** A patient's mental status may be minimally depressed whereby they may respond to environmental stimuli whereas deep sedation requires moderate stimulation to arouse the patient.

TABLE 159-3 The American Society of Anesthesiologist's (ASA) Physical Status Classification

I.	Healthy patient
II.	Mild systemic disease—no functional limitation
III.	Severe system disease—definite functional limitation
IV.	Severe systemic disease—constant threat to life
V.	Moribund patient—not expected to survive without the operation

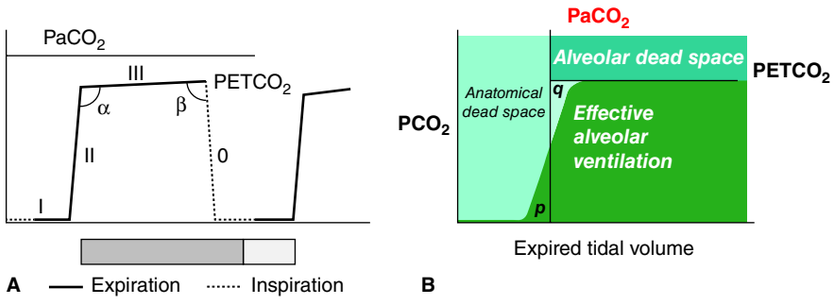

FIGURE 159-1. The capnogram. ***A.*** Segment, phases, and angles identified. ***B.*** The volume capnogram. The two triangles (i.e., p and q) are equal. The line divides the physiologic dead space into the anatomic dead space and alveolar dead space. (Used with permission from reference 119.)

A full meal less than 6 hours or liquids less than 2 to 3 hours prior to the procedure places adults at risk for aspiration if sedation inadvertently results in loss of protective airway reflexes. These time requirements are not a contraindication to the use of PSA. **Procedural sedation need not be delayed based on fasting time as there is no evidence to show decreased aspiration risk with any duration of fasting.**[19] Agents used to promote gastric emptying or altering gastric pH to minimize the effects of aspiration are impractical in the Emergency Department due to their delayed onset for effectiveness. These procedures cannot usually be delayed due to the urgency of the matter.

Obtain baseline measurements of the patient's weight, blood pressure, heart rate, oxygen saturation, and capnography (if available). PSA can result in hypoxia and hypercapnia. The underlying mechanism is opioid-induced and sedative-induced hypoventilation. Pulse oximetry is routinely used during PSA. The degree of desaturation does not correlate alone with poor outcomes.[20] Capnography provides an early warning to a hypoventilatory condition and is the standard care in PSA.[21] Oxygen application is considered routine, yet its value has not been established.[22] The routine application of oxygen may delay recognition of a profound hypoventilatory state if capnography is not used.

The use of capnography is common practice in patients undergoing general anesthesia.[23] It is being used more routinely for PSA in the Emergency Department.[10,24,25] Units come in small handheld models, bigger but portable models, and modules that attach to wall monitors. It numerically displays the exhaled CO_2. The waveform shows the exhaled CO_2 concentration related to volume or time. The y-axis represents the partial pressure of expired carbon dioxide (PCO_2).

There is no evidence that capnography reduces serious adverse events during PSA. Capnography decreases the incidence of hypoxia and respiratory events.[19,26] Capnography can identify respiratory depression early or that is undetected by the Emergency Physician.[22,24,26-30] Evaluate the absolute end tidal CO_2 ($ETCO_2$) level or the $ETCO_2$ waveform to evaluate a patient's respiratory status. The normal range for $ETCO_2$ is 35 to 45 mmHg. **Pulse oximetry measures oxygen saturation but not ventilation. The ventilation is often affected before oxygen saturation in respiratory decompensation.** A patient's oxygen saturation may not drop until late into their respiratory decline. **Direct observation of chest rise and respiratory rate is not sensitive to identify respiratory decompensation during PSA when compared to capnography.**[31,32] **An $ETCO_2$ > 50 mmHg or a change of > 10 mmHg should alert the Emergency Physician that the patient may not be achieving adequate ventilation.** A loss of the $ETCO_2$ waveform implies that the patient is apneic or technical malfunctions are present. **Ensure that the patient is still breathing before looking for an equipment malfunction.** $ETCO_2$ is not perfect in predicting hypoxemia.[25,26]

It is important to note the capnogram waveform and any changes from normal (**Figures 159-1 through 159-4**).[33] Changes can provide information about the patient's respiratory state. Expiration is composed of phases I, II, and III (**Figure 159-1**). Phase I is the baseline inspired gas. This is low in the patient breathing room air or using supplemental oxygen. This is absent of CO_2 in the patient on the ventilator. Phase II is the expired air (i.e., expiratory upstroke) and slopes upward. This represents the transition from dead space to alveolar flow. The α angle is the transition from phase II to phase III. Phase III is a plateau, which is the PCO_2 at the alveoli and is the $ETCO_2$. The β angle is the transition from phase III to inspiration. Phase 0 or IV is the downward deflection (i.e., inspiratory downstroke) of the wave and inspiration. It represents fresh air inflow. The $ETCO_2$ is approximately 5 mmHg lower than the PCO_2 due to the dead space (**Figure 159-1**). The volume capnogram shows the subdivisions of the tidal volume (**Figure 159-1*B***).

There are changes during sedation when compared to presedation waveforms (**Figure 159-2**). The capnogram can show changes in bronchospasm, cuff leaks, $ETCO_2$, obstruction, and respiratory rate (**Figures 159-2 through 159-4**). Increased $ETCO_2$ can be due to increased CO_2 production (e.g., bicarbonate administration, fever, pain, shivering, or tourniquet release), increased

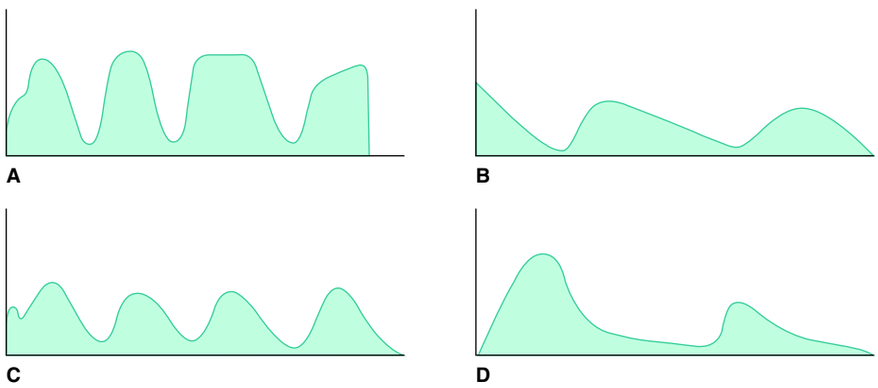

FIGURE 159-2. The capnogram with sedation. ***A.*** Presedation. ***B.*** During sedation the height is decreased. ***C.*** The respiratory rate presedation. ***D.*** During sedation the respiratory rate is decreased. (Used with permission from reference 119.)

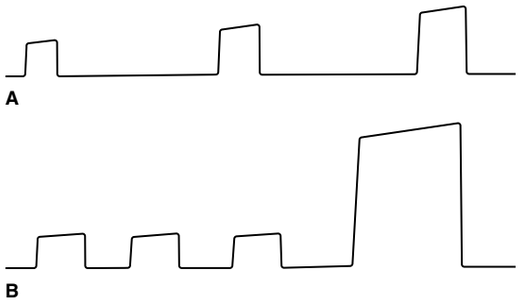

FIGURE 159-3. Examples of capnograms. ***A.*** Hypoventilation. ***B.*** Return of spontaneous circulation after cardiac arrest results in tachycardia and CO_2 elevation from the now perfused tissues. (Used with permission from reference 33.)

pulmonary perfusion (e.g., increased blood pressure or cardiac output), decreased alveolar ventilation (e.g., chronic obstructive pulmonary disease [COPD], hypoventilation, partial airway obstruction, right mainstem intubation, rebreathing, or respiratory insufficiency), or malfunctioning equipment (e.g., exhausted CO_2 absorber, inadequate gas flow of fresh air, machine malfunction, or tubing leaks). Decreased $ETCO_2$ can be due to decreased CO_2 production (e.g., hypothermia), decreased pulmonary perfusion (e.g., cardiac arrest, decreased cardiac output, hypotension, or pulmonary embolism), increased alveolar ventilation (e.g., apnea, extubation, hyperventilation, metabolic acidosis, or total airway obstruction), or malfunctioning equipment (e.g., disconnections, leaks, or machine malfunction).

The changes associated with airway obstruction or bronchospasm are prolongation of phase II, an increased α angle, or a steeper phase III. Increased α angles > 90° are seen with ventilation/perfusion (V/Q) mismatches. Increased β angles suggest rebreathing.

AGENT SELECTION

A patient may present with numerous conditions that may require analgesia, sedation, or a combination of both. The first step is to determine exactly what is needed. Levels of sedation range from light or minimal depression of mental status to deep or heavy sedation where protective airway reflexes or hemodynamic stability may be compromised. **The goal of PSA is to achieve the desired endpoint with minimal risk of cardiorespiratory compromise.**[11]

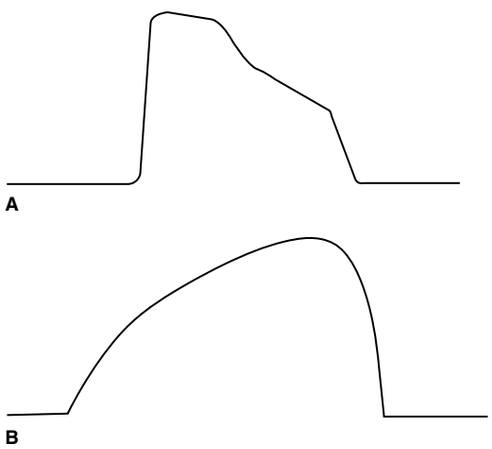

FIGURE 159-4. Examples of capnograms. ***A.*** Cuff leak. An unexpected drop during exhalation indicates that CO_2 is not reaching the detector. ***B.*** Airway obstruction or bronchospasm. The obstruction pattern is formed during exhalation, creating a gradual rise. (Used with permission from reference 33.)

Light sedation is aimed at blunting the patient's level of awareness to environmental stimuli and painful perceptions. Anxiety is alleviated and the patient maintains their responsiveness to verbal and physical stimuli. Protective airway reflexes are maintained. Deep sedation produces profound depression of awareness with the inability to respond to verbal stimuli. Careful monitoring of the cardiorespiratory status is essential in this setting as protective airway reflexes may be lost.

Certain agents, themselves or in combination, produce varied results in different patient subgroups. **Each patient requires individual consideration. Selecting the appropriate agent requires knowledge of the agent's potency, duration to onset, duration of drug effect, titratability, interaction with other drugs, and adverse effects profile.** Increasing dosages of an agent reduces its drug-specific effect in exchange for a nonspecific sedative effect complete with cardiorespiratory compromise. **The most common mistake in sedation is choosing the wrong agent for the specific goal.** For example, sedating a patient does not relieve pain. Sedation must be accompanied by analgesia if a patient is undergoing a painful procedure. This agent should ideally be titratable. **The only reliable and precise means of this is via intravenous administration.** Deep sedation should be provided only via the intravenous route. **Intravenous access is required if the patient undergoes deep sedation in case cardiorespiratory support is required or reversal agents must be administered**. Anxiolysis or mild sedation does not necessarily require intravenous access. Patients may be assessed on an individual basis as to whether intravenous access is deemed appropriate.

Synergistic effects must be considered when choosing a dosing schedule. For example, combining a benzodiazepine with a narcotic analgesic increases the potential effect of either agent alone.[20] It may become very difficult to titrate a sedative and an analgesic concurrently. Some practitioners advocate the use of a sedative initially followed by the addition of a narcotic analgesic as the patient's sedation is resolving. This works well for procedures that are extremely short and where muscle relaxation is key (e.g., simple reductions). The anterograde amnesia provided by most sedative agents is usually adequate to prevent the recollection of pain during the procedure. **The Emergency Physician should reduce the initial doses of multiple agents owing to the additive sedative effects. Small and incremental dosing enables a controlled titration to effect. Physicians must have a fundamental knowledge of the pharmacologic profiles of these agents. Allow the medications to reach peak effect before administering additional medication. Emergency Physicians are best served with a thorough knowledge of a few drugs as opposed to little knowledge over the entire procedural sedation agent spectrum.**

It would behoove an Emergency Physician to develop and practice four PSA drug regimens: sedative plus analgesic, pure sedation, dissociative analgesia and sedation (i.e., ketamine), and inhalation anesthesia (i.e., nitrous oxide). **Become well versed in the applications of these regimens and do not stray from the routine unless circumstances dictate a different regimen.** Identify one's limitations and obtain peer or Anesthesiology backup prior to procedural initiation. Provide adequate analgesia first when performing a painful procedure. Some degree of sedation will have already been established. Provide sedation with a pure sedative to obtain the endpoint of relaxation and sedation required to complete the procedure.

DRUG PROFILES

Table 159-4 provides a summary review of the agents routinely employed during PSA. **It is critical that the Emergency Physician is well versed on the individual agent and its effect in combination**

TABLE 159-4 The Agents Currently Available for PSA

		Pediatric dosing		Adult dosing					
Agent	**Route**	**Initial**	**Maximum**	**Initial**	**Maximum**	**Onset (min)**	**Duration (min)**	**Adverse reactions**	**Contraindications**
Benzodiazepines									
Midazolam (Versed)	IV IM PO PR Nasal	0.05 mg/kg 0.05 mg/kg 0.5 mg/kg 0.25 mg/kg 0.2 mg/kg	0.15 mg/kg 0.20 mg/kg 0.7 mg/kg 0.5 mg/kg 0.5 mg/kg (or 6 mg)	0.025 mg/kg 0.05 mg/kg	0.1 mg/kg 0.15 mg/kg	2–3 10–20 10–30 10–30 10–15	30–60 60–120 60–90 60–90 45–60	Respiratory depression Hallucinations Hypotension Excessive sedation Headache Nausea Vomiting Hiccups Paradoxical reactions	Hypersensitivity Renal impairment Uncompensated acute illness Recent illicit drug use Recent alcohol use
Opioids									
Fentanyl (Sublimaze)	IV Nasal Nebulizer	0.5–1.0 mcg/kg 0.7–1.5 mcg/kg 1.7–3 mcg/kg	2–3 mcg/kg	1-2 mcg/kg		1–2 30 20–30	20–30 30 30	Respiratory depression Pruritus Bradycardia	Hypersensitivity Uncompensated acute illness Recent illicit drug use
Sufentanil (Sufenta)	Nasal	0.7 mcg/kg (0.7 mcg/kg if given with nasal midazolam)				5–15	60–120	Nausea Vomiting Chest wall rigidity Hypotension Serotonin syndrome	Recent alcohol use Coma < 6 months of age
Morphine	IV IM/SQ	0.1 mg/kg 0.1 mg/kg	0.2 mg/kg 0.2 mg/kg	0.1 mg/kg 0.1 mg/kg	0.2 mg/kg 0.2 mg/kg	1–5 30	180–240 240–300	Respiratory depression Hypotension Nausea Vomiting Histamine release Prolonged sedation	Hypersensitivity
Barbiturates									
Methohexital (Brevital)	IV PR	0.5 mg/kg 20 mg/kg	1.0 mg/kg 25 mg/kg	0.5 mg/kg	1.0 mg/kg	0.75 10–15	5–10 60	Nausea Vomiting Apnea Respiratory depression Paradoxical Hyperactivity	Temporal lobe epilepsy Acute intermittent porphyria
Thiopental (Pentothal)	IV PR	20–25 mg/kg		0.5–1.0 mg/kg	4 mg/kg	0.25–0.3 5–8	3–10 60	Apnea Respiratory depression Hypotension Histamine release Decreased myocardial contractility	Hypotension Altered mental status Cardiac ischemia Cardiac conditions

(*Continued*)

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TABLE 159-4 The Agents Currently Available for PSA (*Continued*)

		Pediatric dosing		Adult dosing					
Agent	**Route**	**Initial**	**Maximum**	**Initial**	**Maximum**	**Onset (min)**	**Duration (min)**	**Adverse reactions**	**Contraindications**
Pentobarbital (Nembutal)	IV	2.5 mg/kg (additional increments of 1.25 mg/kg every 30 sec) max 100 mg	6 mg/kg	1.25 mg/kg	2.5 mg/kg	0.5–1.0	15	Nausea Vomiting Apnea Hypotension	Acute intermittent porphyria
	IM	4 mg/kg	6 mg/kg	1.25 mg/kg	2.5 mg/kg	10–20	60–240	Hypoxemia	
	PO	1 mg/kg	6 mg/kg (max 100 mg)	2 mg/kg	6 mg/kg	15–60	60–240	Respiratory depression Paradoxical hyperactivity	
	PR	< 4 years: 3 mg/kg	6 mg/kg (max 100 mg)	2 mg/kg	6 mg/kg	15–60	60–240	Decreased myocardial contractility	
		> 4 years: 1.5 mg/kg	3 mg/kg (max 100 mg)						
Hypnotics									
Chloral hydrate	PO	20 mg/kg	1000 mg			15–60	60–120	Paradoxical	Hepatic impairment
	PR	Not recommended				10–20	60–120	hyperactivity Delirium Residual sedation Nausea Vomiting	Renal impairment Hypersensitivity
Ketamine (Ketalar)	IV	0.5 mg/kg	1.0 mg/kg	0.75 mg/kg (additional 5–10 mg doses as required to effect; 0.01–0.02 mg/kg/min infusion)	1.5 mg/kg	0.5–1.5	15–45	Increase intracranial pressure Increased intraocular pressure Stridor Vomiting	< 3 months Active respiratory infections Increased ICP, head trauma, hydrocephalus Cardiovascular disease
	IM	1 mg/kg (usually 2–4 mg/kg; may repeat every 5–10 min as needed)	6 mg/kg			4–10 10–15 10–15	30–60 30–60 45–75	Hypersalivation Bronchorrhea Hypertonicity Laryngospasm	Glaucoma Psychoses Potential for airway instability (i.e., tracheal stenosis)
	PO PR	5.0 mg/kg 50 mg/kg	10.0 mg/kg					Emergence reactions	Relative contraindications Oral procedures Thyroid disease Acute intermittent porphyria
Propofol (Diprivan)	IV bolus			0.5–1.0 mg/kg		0.5	8–10	Respiratory depression	Hypotension
	IV drip			50–75 mcg/kg/min		0.5	8–10 min after stopped	Apnea Hypotension Similar to thiopental	Respiratory depression

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Inhalation Anesthetics							
Nitrous oxide		50% N_2O–50% O_2 (self-administered by demand valve mask)	50% N_2O–50% O_2 (self-administered by demand valve mask)	0.5–1.0 (peaks in 3–5 min)	3–5 after withdrawal of gas	Nausea, vomiting Disorientation Agitation Air-filled cavity expansion	Altered mental status Intoxication Pregnancy Opioids within past 4 hours Pneumothorax Pneumomediastinum Bowel obstruction Uncooperative patient Facial trauma Relative contraindications Full meal < 1 hour Age < 5 years (cooperation)
Reversing Agents							
Naloxone (Narcan)	IV	< 12 months: 0.1 mg/kg every 2–3 min > 12 months: 1–2 mg/kg 10 mg every 2–3 min	Respiratory arrest: 1–2 mg Respiratory depression: (0.4 g in 9 mL) 1 mL every 2 min	1–2	20–40	May precipitate withdrawal in opioid-dependent patient	None
	IM, SL, SQ	< 12 months: 0.1 mg/kg every 2–3 min > 12 months: 1–2 mg/kg 10 mg every 2–3 min		5–10	60–90		
Flumazenil (Romazicon)	IV	0.02 mg/kg (max 1 mg) 1 mg total (repeat every 1 min to effect)	0.2 mg every 15–45 sec 1 mg/15 min 3 mg/30 min	1–2	20–40	Nausea, vomiting	Concomitant tricyclic antidepressant ingestion Hypersensitivity In patient with chronic benzodiazepine use – may precipitate seizure
	IM	Same as IV		5–10	60–90		

ICP, intracranial pressure; IM, intramuscular; IV, intravenous; PO, oral; PR, per rectum; SL, sublingual; SQ, subcutaneous.

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with other medications. Medication routes, dosing parameters, side effects, contraindications, and anticipated complication management must be well known in advance.

The ideal agent is a single drug that has amnestic, anxiolytic, analgesic, and sedative properties. It should have a predictable and rapid onset of action. It should have a predictable and short duration of action with a rapid recovery. The ideal agent should be inexpensive, easy to administer, and have a wide safety margin to make loss of consciousness extremely unlikely. It should have little or no side effects, especially cardiovascular and respiratory. It must be easily reversible if necessary. There should be no residual effects at the end of the procedure. It is obvious that no agent meets all these criteria. We hope to minimize side effects, maximize benefit, allow quick recovery and dispositions, and produce reliable effects using small doses of multiple agents.

BENZODIAZEPINES

Benzodiazepines produce anterograde amnesia, anxiolysis, muscle relaxation, and sedation. They have no analgesic activity and must be used in conjunction with other agents for painful procedures. The use of a benzodiazepine allows less analgesic agent to be administered and may reduce the severity of adverse reactions. **The major adverse effects of benzodiazepines are respiratory depression and hypotension.** Benzodiazepine-associated respiratory depression is usually transient but can be reversed with flumazenil. Prevent hypotension by ensuring that the patient is euvolemic, using the minimal amount of a narcotic to produce analgesia, and carefully titrating the benzodiazepine.

MIDAZOLAM (VERSED)

Midazolam is an ultra-short-acting benzodiazepine that is metabolized by the liver and excreted by the kidney. Intravenous midazolam in dose ranges from 0.02 to 0.10 mg/kg will produce sedation within 2 to 3 minutes, redistribute rapidly, and provide a 20 to 30 minute duration of action. Most adults have adequate sedation and anxiolysis by a total dose of 5 to 7 mg, administered in 0.05 mg/kg or 1 to 2 mg increments every 3 to 5 minutes. **It is important to note the 2 minute delay in the peak central nervous system (CNS) effect. Allow midazolam time to work before additional dosing.** It may be used intranasally in children at a dose of 0.3 to 0.5 mg/kg as an effective means of sedation.[34] It is water-soluble and does not cause vascular irritation on injection as found in its relative diazepam. Midazolam is 3 to 4 times more potent than diazepam. It has anxiolytic and anticonvulsive properties. It is a potent amnestic agent with the added advantage of providing antegrade and retrograde amnesia. Midazolam alters the response to pain but does not reduce pain perception like all benzodiazepines. The addition of an analgesic agent is required when midazolam is administered for a painful procedure.

Midazolam is a potent respiratory depressant like all sedative agents. Midazolam can cause apnea by depressing the sensitivity of the hypothalamus to hypercapnia. It shifts the CO_2 response curve to the right and depresses its slope. **Its respiratory depressant effects are augmented in patients receiving opioids, with underlying lung disease (e.g., COPD), and with concomitant circulating CNS depressants (e.g., alcohol, barbiturates, and opioids)**. Patients may become hypoxic even with normal respiratory rates.

Pulse oximetry is warranted when administering midazolam. Deaths related to the use of midazolam for sedation during endoscopy prompted the U.S. Food and Drug Administration (FDA) to recommend increased monitoring and caution with elderly or debilitated patients. There is a propensity for apnea when even very small doses of midazolam are given in conjunction with fentanyl.[35] Midazolam may cause hypotension that is related to the dose and to the rate of administration. This effect is more likely to occur in hypovolemic patients or elderly patients. Paradoxical reactions (i.e., agitation, delirium, and excitation) can be produced from using midazolam and reversed with flumazenil.[36-38]

DIAZEPAM (VALIUM) AND LORAZEPAM (ATIVAN)

Diazepam and lorazepam have longer half-lives than midazolam. Either agent may be used for PSA. They are more difficult to titrate and have a longer duration of action. Neither of these characteristics is well suited to PSA. **Midazolam has greater earlier sedation, less recall, less pain upon injection, higher 90 minute alertness scores, and more patients ready for discharge at 90 minutes post-procedure than diazepam**. Diazepam results in more respiratory depression, hypotension, and phlebitis than midazolam. **Diazepam and lorazepam have no advantage over midazolam if the goal is to produce a short and titratable state of anxiolysis and sedation**. These agents are not often used for PSA.

OPIOIDS

Opiates provide analgesia with minimal sedation and no anxiolysis. They have a long track record showing a predictable performance. The respiratory and central nervous system depression can be readily reversed with naloxone and nalmefene. Opiates are relatively inexpensive and used in a balanced approach to PSA. Agents in this class include morphine, fentanyl, meperidine, sufentanil, and alfentanil.

Morphine has been the mainstay narcotic analgesic. The use of potent synthetic short-acting opioids for analgesia and sedation has been common practice for PSA. These drugs are particularly appealing for their short half-lives, rapid onset of action, limited cardiovascular side effects, ease of controlled administration, and the availability of a rapidly acting reversal agent. The use of these agents requires a thorough familiarity with their pharmacologic properties and side effects. A PSA repertoire should include one of the potent synthetic narcotics. It is better to master one drug than dabble in the pharmacology and administration of three different drugs with different dosing regimens.

MORPHINE

Morphine is a "good old" potent analgesic with a lot of clinical experience. It is the prototype opioid to which all others are compared. Incremental intravenous dosing of 0.05 to 0.20 mg/kg every 3 to 5 minutes provides a nice range over which to carefully titrate to an individual analgesia requirement. It begins working in 1 to 3 minutes and peaks within 15 to 20 minutes. Morphine has a duration of activity of 3 to 4 hours. It provides often-needed pain relief after the procedure has been completed. It is metabolized by the liver and excreted by the kidney. Morphine, as with all opioids, decreases the medullary response thereby promoting hypercapnia and hypoxia. Histamine release with hypotension, nausea, vomiting, itching, bronchospasm, and loss of vascular tone are common side effects of morphine administration. Morphine has steadily lost ground to the designer opioids with better cardiovascular stability (e.g., fentanyl, sufentanil, and alfentanil).

FENTANYL (SUBLIMAZE)

Fentanyl is highly lipid soluble and 75 to 125 times more potent than morphine. Peak analgesia is achieved in 2 to 3 minutes after intravenous administration. The terminal half-life is 130 to 220 minutes.

The clinical effectiveness is limited to approximately 30 minutes due to rapid tissue redistribution. Fentanyl is a "20 minute drug for a 20 minute procedure." It is metabolized in the liver with renal and hepatic excretion. Approximately 20% is excreted as the unmetabolized parent compound. The major side effect of fentanyl is respiratory depression. **Hypotension is less common than with morphine because fentanyl does not induce histamine release.**

Administer fentanyl very slowly and in small increments. Patients may experience rigidity of their chest muscles (i.e., the "rigid chest syndrome") when it is administered intravenously too rapidly.[39] This syndrome can severely impact ventilation to the point that the patient must be paralyzed to facilitate adequate ventilation. Administer incremental doses of 0.5 to 1.0 μg/kg or 10 to 100 μg boluses slowly intravenously until adequate analgesia is achieved.

Fentanyl may be administered intranasally at a dose of 0.7 to 1.5 μg/kg.[40] Concentrated fentanyl at a dose of 1.7 μg/kg is equivalent to 0.1 mg/kg of morphine.[41] Its advantages include simple rapid administration, limited training required to administer it intranasally, and faster analgesia when compared to establishing intravenous access and administering an intravenous opioid.[40] The disadvantages include having to restrain the child's head for the administration and the time it takes to administer the medication. Atomizer devices can minimize or eliminate these issues.

An alternative method is to administer fentanyl through a nebulizer mask.[42,43] Nebulized fentanyl at a dose of 3 μg/kg through a breath-actuated mask is effective.[42] This study only used children over the age of 3 years because younger children have difficulty triggering the mask. Another study used a standardized nebulizer system to deliver 4 μg/kg of fentanyl, a dose equivalent to 0.1 mg/kg of morphine, to children between 4 and 13 years of age.[43] Appropriate analgesia was achieved with this dose and administration system.

Fentanyl is contraindicated in children less than 6 months of age. It stimulates the central vagus nucleus in the brainstem. This can result in a prolongation of the refractory period of the atrioventricular (AV) node and significant bradycardia.

The rigid chest syndrome may occur to the point at which the patient may not be ventilating and attempts at assisted ventilation with a bag-valve-mask device are unsuccessful.[39] The patient may then become hypoxic, bradycardic, and eventually die. This phenomenon has been well documented in children. The rigid chest syndrome is the reason many are reluctant to administer fentanyl. **It occurs during rapid intravenous administration, so give it slowly**. It only occurs at high doses (e.g., > 15 μg/kg) to anesthetic doses (e.g., 50 to 100 μg/kg). **The doses used for PSA are safe and usually do not result in the rigid chest syndrome**. Immediately administer naloxone intravenously if the rigid chest syndrome develops. The use of naloxone does not often work to overcome the rigid chest syndrome.[39] The patient may require paralysis and orotracheal intubation to overcome the rigid chest syndrome.[39]

SUFENTANIL (SUFENTA)

Sufentanil is an extremely potent narcotic analgesic. It is 5 to 10 times more potent than fentanyl and 500 to 1000 times more potent than morphine. It is metabolized and eliminated more rapidly than fentanyl. There is no significant advantage to using sufentanil over fentanyl for most PSA applications.

The one advantage is that its potency and small-volume dosing enable it to be delivered intranasally in the pediatric population. The American College of Emergency Physicians's Guideline to Pediatric Sedation is a proponent of this application.[44] Sufentanil has an onset of action in 5 to 15 minutes and a duration of action lasting 1 to 2 hours when administered intranasally. It provides exceptional cardiovascular stability but maintains the profound respiratory depressive effects inherent in the opioid class of agents. Careful patient selection is well advised when considering sufentanil.

ALFENTANIL (ALFENTA)

Alfentanil is less lipid soluble than fentanyl and less likely to accumulate if multiple doses are required. It is 10 to 20 times more potent than morphine. It is one-tenth to one-fifth as potent as fentanyl. The duration of analgesia is ultrashort. Its onset of action is within seconds and lasts only 2 minutes. **Alfentanil is too short-acting to use for PSA.** Total dosing is 8 to 10 μg/kg delivered in small repeated dosing aliquots. Its half-life is only 80 minutes. The adverse effect profile is like fentanyl except it may cause more respiratory depression.[45] The rigid chest syndrome may be seen with alfentanil. Alfentanil is more appropriate to use for the induction of general anesthesia.

REMIFENTANIL

Remifentanil is a synthetic short-acting opioid. It is commonly used by Anesthesiologists for analgesia that is deep and rapid. It causes little central nervous system depression. One advantage is that it is metabolized by blood esterases and does not rely on the kidney or liver. The half-life is 3 to 8 minutes, often too short for the Emergency Department unless redosed. There has been little use in the Emergency Department.[46,47]

MEPERIDINE (DEMEROL)

Meperidine is one-tenth as potent as morphine. It was the most commonly administered opioid agent for pain in the United States. It is less often used due to its euphoric effects. It is not used for PSA because it is hard to titrate and takes a long time to reach peak activity.

DISSOCIATIVE AGENTS

KETAMINE (KETALAR)

Ketamine is a unique pharmaceutical agent that provides amnesia, analgesia, anxiolysis, and sedation.[48,49] Its safe and effective use in pediatric sedation and analgesia is well studied.[50] It is the most commonly used anesthetic agent throughout the world. It has the best safety profile in terms of cardiorespiratory complications of any agent. Ketamine is a derivative of phencyclidine (PCP) that generates a functional and electrophysical dissociation between the brain's cortical and limbic systems. This results in a dissociative state whereby the patient is in a trance-like cataleptic condition in which sensory perceptions and memory are blunted. The patient appears awake yet is dissociated from their environment. Random tonic movements will occur and gentle physical restraint may be required.

Ketamine is a positive inotrope. It increases the heart rate, blood pressure, cardiac output, and intracranial pressure.[51] The blood pressure and heart rate may slightly increase with the use of ketamine. The systolic and diastolic blood pressure may increase up to 30 mmHg with an average of 15 mmHg. The heart rate may increase up to 30 beats per minute with an average of 15 beats per minute. These effects are believed to be due to decreased uptake of catecholamines at neural endplates.

Ketamine's effect on the respiratory system includes bronchodilation, a slight increased respiratory rate, increased secretions, and potential laryngospasm. Ketamine is a potent bronchodilator. **Ketamine does not depress airway reflexes like narcotics or benzodiazepines.** The side effects of increased respiratory secretions can be blocked with atropine or glycopyrrolate.[52,53] Administer atropine

prior to or concurrently with ketamine in a dose of 0.01 mg/kg to a maximum of 0.3 to 0.5 mg. Administer glycopyrrolate before ketamine in a dose of 0.005 mg/kg to a maximum of 0.25 mg. Atropine administration is associated with less adverse respiratory events, less recovery agitation, and less vomiting during recovery than glycopyrrolate.[54] Atropine causes more side effects compared to glycopyrrolate because it crosses the blood-brain barrier.[55,56] A recent trend is to not use an antisialogue because excessive respiratory secretions are uncommon and not associated with adverse respiratory events.[57]

Ketamine may be administered by several routes. Rapid predictable effects are best seen with parenteral administration.[58] Intravenous dosing is 0.5 to 1.0 mg/kg increased slowly up to 2 mg/kg.[59] This is approximately one-fourth of the intramuscular dosing of 1 to 6 mg/kg, usually 2 to 4 mg/kg. A dissociative state is produced in less than a minute with intravenous administration and 2 to 10 minutes via the intramuscular route. Intravenous dosing may be repeated at 5 minutes with an additional 1 to 2 mg/kg. Intramuscular dosing may be repeated at 10 minutes with an additional 2 to 4 mg/kg if adequate sedation has not been achieved. Ketamine may be given orally at 5 to 6 mg/kg or rectally at 5 to 10 mg/kg. Its titratability is poor orally and rectally. This makes its effectiveness much less predictable. Intravascular dosing lasts 20 to 25 minutes with a single administration.[60] Intramuscular dosing may be more commonly associated with laryngospasm when compared to intravenous administration.[61]

Ketamine may be administered intravenously or intramuscularly due to its safety profile.[62] Intramuscular administration is preferable when intravenous access is difficult to establish, intravenous access is not required for another reason, and for procedures lasting 15 to 25 minutes due to its longer effects. Intravenous administration is preferable if intravenous access is required for another reason, if intravenous access is easily obtained, for procedures lasting less than 10 minutes, and to decrease recovery time.[63]

The lack of cardiorespiratory compromise of ketamine and the effectiveness of intramuscular administration make it an excellent agent for pediatric PSA. Patients tend to remain hemodynamically stable when using ketamine. This reduces the need for intravenous fluid boluses and reversal agents. This is especially true in children in whom intravenous access can be a difficult procedure. A medication that can be safely administered intramuscularly not only simplifies PSA but also improves patient and family satisfaction.

Contraindications to the use of ketamine include age less than 3 months, procedures involving pharyngeal stimulation, known cardiovascular disease, concurrent head trauma with altered mental status, airway compromise including previous tracheal surgery or stenosis, glaucoma, known central nervous system mass lesions, active upper or lower respiratory disease, and porphyria. Ketamine can rarely cause acute pulmonary edema.[64,65] Evidence now shows it does not increase intraocular pressure or intracranial pressure significantly.[66-69] Allergic reactions to ketamine are rare.[70] It can cause cardiac arrhythmias.[71] Information suggests ketamine is safe to administer in head injury patients in the pediatric intensive care unit.[72]

Patients will exhibit a slow emergence from the effects of ketamine over the course of 1 to 2 hours. Ketamine is metabolized and excreted by hepatic mechanisms. Place the patient in a quiet room that is free of excessive external stimuli to minimize the possibility of them becoming hyperactive or overstimulated by their surroundings. Ketamine-associated emesis is a commonly seen side effect.[61,73-75] The rate of emesis is higher with intramuscular administration, with initial intravenous doses over 2.5 mg/kg, and with total doses ≥ 5.0 mg/kg.[61,73] However, a study of over 1000 children refuted the theory that emesis is dose related.[74] The use of intravenous ondansetron at 0.15 mg/kg to a maximum 4 mg decreased the rate of ketamine-associated emesis.[75]

Emergence reactions are hallucinations that occur as the ketamine wears off and the patient awakens. They may be seen in up to 50% of adults and up to 10% of children given ketamine. The etiology of these emergence reactions is unknown. There is an association of emergence reactions with an age over 10 years, female sex, rapid intravenous administration, stimulation during the recovery period, and personality disorders. Rarely will a child less than 10 years of age develop an emergence reaction with hallucinations.

The incidence of emergence reactions can be decreased. Administer the ketamine slowly intravenously. Place the patient in a dark, quiet room with minimal sensory stimulation during the recovery period. Treatment with benzodiazepines is indicated if an emergence reaction develops. The concurrent administration of midazolam in a dose of 0.025 to 0.050 mg/kg can reduce emergence reactions. There is no advantage of combining midazolam with ketamine in preventing an emergence reaction.[76] Midazolam can significantly reduce emergence reactions in adults.[77] A review noted no benefit and no harm in coadministering benzodiazepines.[73] The concurrent use of midazolam is Physician-dependent, has no serious sequelae, and may reduce emergence reactions.

KETAMINE-PROPOFOL COMBINATION (KETOFOL)

The use of ketamine and propofol in combination has been coined "ketofol." The reason these agents were combined was to provide appropriate sedation and analgesia while decreasing adverse events by using less of each individual agent. Ketamine potentiates the sedation of propofol without cardiac or respiratory depression. The combination of ketamine and propofol is effective for PSA, although the evidence does not show a clear benefit over ketamine alone.[21,78-86] The combination has similar complication rates, similar adverse effects, and similar satisfaction scores when compared to using ketamine as a single agent.[21,86]

There is no established standard dosing. Prepare both the ketamine and the propofol as a 10 mg/mL solution. These agents can then be administered intravenously in a 1:1 ratio in aliquots of 1 to 3 mL every 2 minutes until the desired effect is achieved. Another option is to mix equal parts of both solutions into one syringe and administer the appropriate volume to deliver 0.5 mg/kg of each agent. Additional boluses can be administered to deliver 0.25 mg/kg of each agent every 2 to 3 minutes until the desired effect is achieved.

SEDATIVE-HYPNOTICS

The sedative-hypnotics are a diverse category of agents that include thiopental, methohexital, etomidate, and propofol. **Sedative-hypnotics provide anxiolysis and sedation with various degrees of amnesia. They provide no analgesia.**

BARBITURATES

Short-acting barbiturates (i.e., thiopental, pentobarbital, and methohexital) are effective PSA agents. They provide good titratability with a rapid onset of action. Barbiturates provide amnesia, hypnosis, and sedation. They are highly lipophilic and cross the blood-brain barrier readily resulting in an onset of action in less than 1 minute. They have short half-lives that promote a rapid recovery and minimize the risks inherent in prolonged sedation. **Their major drawbacks are respiratory depression, apnea, transient hypotension, and lack of a reversing agent. The window between PSA, deep sedation, and general anesthesia is extremely narrow**. These agents are rarely used for PSA in the Emergency Department.

THIOPENTAL (PENTOTHAL)

Thiopental is one of the more commonly used barbiturates. It has an extremely rapid onset of action of 10 to 20 seconds with a peak activity in 1 minute. Its sedative effect lasts 3 to 10 minutes. Thiopental is titratable in doses of 0.5 to 1.0 mg/kg intravenously. Rarely is more than 2 mg/kg required.

It may be administered rectally at 20 to 25 mg/kg in children who require sedation without intravenous access. Sedation is achieved within 5 to 8 minutes. Rectal administration does not result in apnea or respiratory depression. Thiopental is often administered rectally for laceration repairs in children.

METHOHEXITAL (BREVITAL)

Methohexital has a profile like thiopental with a few noted exceptions. It is twice as potent as thiopental. It has much less of an effect on decreasing myocardial contractility and decreasing vascular tone. A single study has convinced most people not to use this drug in the Emergency Department.[87] One hundred and two patients were given methohexital with a mean cumulative dose of 1.6 mg/kg. Three patients developed hypotension. Twenty-two patients developed respiratory depression requiring bag-valve-mask assistance. Five of the 22 patients with respiratory depression developed transient apnea.

Methohexital may be administered as repeated incremental bolus dosing or via a titratable continuous infusion. Begin with a 0.5 to 1.0 mg/kg bolus followed by an infusion at 50 μg/kg/min. Titrate infusion rates upward to effect with a maximum rate of 75 μg/kg/min. Bolus dosing is best delivered as 0.5 to 1.0 mg/kg to a maximum of 20 mg every 45 to 60 seconds until the desired endpoint is attained. Maximum sedation is achieved at 40 seconds.

Respiratory depression is common and independent of the dose or concomitant administration of a narcotic or benzodiazepine. Patients may require assisted ventilation with a bag-valve-mask device or ventilator assistance until the effect of the drug clears. **Respiratory depression is minimized by first administering the analgesic agent to control pain followed by methohexital for sedation to effect**. Do not use methohexital in children younger than 12 years of age.

PENTOBARBITAL (NEMBUTAL)

Pentobarbital is a short-acting barbiturate that is effective for pediatric sedation during nonpainful diagnostic procedures (e.g., computed tomography or magnetic resonance imaging). **Controlled sedation levels are best achieved with titrated small incremental intravenous dosing.** It may also be administered intramuscularly, orally, and rectally. Intravenous administration will induce sleepiness within 30 seconds with a duration of action up to 15 minutes. Other dosing methods take longer to work with a duration of action ranging from 1 to 4 hours.

Pentobarbital is primarily administered intravenously or rectally. Initial intravenous dosing at 2.5 mg/kg may be sufficient. Subsequent doses of 1.25 mg/kg every 30 seconds to effect will minimize oversedation and limit hypoxia. Other side effects are nausea, vomiting, and paradoxical hyperactivity. A maximum total dose of 6 mg/kg or 100 mg should mark the dosing endpoint. Older children may exceed this limit with due caution. Rectal dosing is age-dependent. Administer 3 to 6 mg/kg to a maximum of 100 mg in children younger than 4 years of age and 1.5 to 3 mg/kg to a maximum of 100 mg in children older than 4 years of age.

ETOMIDATE (AMIDATE)

Etomidate is an imidazole derivative that is commonly used for the induction of anesthesia or rapid sequence induction. It produces amnesia, anxiolysis, and sedation equal to that of barbiturates but with fewer hemodynamic affects.[88] It is ultra-short-acting and in a class of its own. It may produce unconscious sedation like the barbiturates. There is a narrow window for the use of etomidate in PSA. It can be used for an emergent procedure in a patient with borderline low blood pressure and ketamine is contraindicated. It is often used when there may be contraindications to other sedating agents due to hypotension and or other injuries.

There is evidence showing that intensive care unit patients given etomidate are at greater risk for the development of adrenal insufficiency.[89] Etomidate inhibits 11-beta-hydroxylase causing a decrease in serum cortisol levels. This was initially found in patients who were receiving continuous intravenous administration of etomidate. There is evidence that adrenal insufficiency may occur even after a single dose of etomidate.[90] These studies were conducted on patients receiving etomidate for induction prior to intubation. The study populations had higher injury severity scores and were receiving higher doses of etomidate when compared to PSA doses. This effect on the adrenal gland is usually transient and resolves after 24 hours. The actual effect on morbidity and mortality is still not clear.[91,92] Adrenal suppression in the Emergency Department patient undergoing PSA for a short procedure and then being discharged is probably of little to no consequence.

Etomidate is one of the more frequently used agents for sedation and PSA. It has a shorter onset and faster recovery time than midazolam.[93,94] Myoclonus is a well-described adverse effect that is unique to etomidate.[95-97] Myoclonus can be severe enough to result in a decreased oxygen saturation and full-body rigidity requiring a brief period of respiratory support. A rare adverse event is agitation or an "emergence-type reaction" as the effects of etomidate wear off.[98] These few adverse effects do not outweigh its benefit of a minimal effect on the cardiovascular system.

PROPOFOL (DIPRIVAN)

Propofol is a highly lipid-soluble compound with ultra-short sedative hypnotic properties. **It can produce amnesia, anxiolysis, hypnosis, and profound sedation.[48,99,100] Propofol has no analgesic properties.** It is unrelated to the barbiturate or benzodiazepine classes. The onset of action is extremely rapid and within 15 to 30 seconds. The maximal effect is seen within 30 to 45 seconds. The duration of effect is typically only 8 to 15 minutes, even after prolonged administration. Propofol is contraindicated if the patient has a known sensitivity or allergy to egg or soy products.

Dosing can be delivered intravenously via repeated small 20 mg doses at 2 minute intervals or initial loading of 0.5 to 2.0 mg/kg followed by infusion rates of 25 to 130 μg/kg/min. A simple method of titrating propofol for short procedures is to administer 1 mg/kg initially with repeat boluses of 0.5 mg/kg every 1 to 2 minutes as needed to achieve or maintain adequate sedation. Effective total doses may range from 20 to 150 mg. A continuous infusion of 50 to 70 μg/kg/min can be used to produce a sleep-like state with minimal respiratory depression. Continuous infusions allow the patient to be easily aroused by verbal stimuli and recover within 2 to 3 minutes of stopping the infusion. Induction dosing or rapid bolus injection can result in profound respiratory depression and hypotension, especially in the hypovolemic patient. High-dose oxygen can decrease the hypoxemia.[101] It can be associated with bronchospasm, laryngospasm, and pulmonary edema in rare cases.[102] Propofol can be coadministered with fentanyl or ketamine (i.e., ketofol) for painful procedures requiring a combination of sedation and analgesia. Propofol can turn the urine green.[103]

The high lipid content of the propofol solution causes pain upon injection. The administration of intravenous lidocaine immediately prior to the propofol may decrease the injection pain. Inject

0.5 mg/kg up to a maximum of 40 mg of lidocaine.[104-106] Be cautious to not exceed the maximum toxic dose of 4.5 mg/kg in small children. An alternative is to add 30 mg of ephedrine to 20 mL of 1% propofol solution to decrease the injection pain.[107] An additional advantage of ephedrine is that it counteracts the potential propofol-associated hypotension.

FOSPROPOFOL

Fospropofol is a water-soluble prodrug of propofol.[108,109] This formulation is not associated with many of the lipid emulsion effects. This formulation results in decreased injection pain, has a wider therapeutic window, and allows long-term sedation without a large lipid load. Fospropofol is currently FDA approved for monitored anesthesia care. Future studies and safety data are required before this agent can be recommended for PSA.

DEXMEDETOMIDINE

Dexmedetomidine is an alpha-2 agonist with analgesic, anxiolytic, and sedative properties that works as well as midazolam.[110,111] It decreases central adrenergic tone by blocking norepinephrine release from presynaptic and postsynaptic central nervous system receptors. Dexmedetomidine causes a minimal respiratory depression. It has a delayed onset of action in 15 to 30 minutes and its effects persist up to 3 hours after the infusion is discontinued. It is administered intravenously at 2 μg/kg over 10 minutes followed by a maintenance dose of 1 μg/kg/h. The side effects include bradycardia, cardiac arrhythmias, hypertension with loading, and hypotension with maintenance infusions. This agent is often used in the outpatient setting for elective computed tomography and magnetic resonance imaging scans.[112,113] Its delayed onset, prolonged effects, and adverse effect profile make it an agent not recommended for PSA.

MISCELLANEOUS AGENTS

Several agents can be used for the sedation of children in the Emergency Department and outpatient setting. **These agents are not used for PSA.** These agents include chloral hydrate and DPT (meperidine [Demerol], promethazine [Phenergan], and chlorpromazine [Thorazine]). Chloral hydrate is not often used in the Emergency Department. **Never use the DPT cocktail in any setting**.

CHLORAL HYDRATE

Chloral hydrate is a time-tested, effective, and pure sedative-hypnotic. It has no analgesic properties. Its primary usage has been for sedation during nonpainful diagnostic testing. The oral dose is 20 to 100 mg/kg to a maximum of 2000 mg. Most children require 50 to 75 mg/kg to achieve proper sedation. It has an onset of action of 15 to 60 minutes and a duration of effect of 1 to 2 hours. Its effects may persist for up to 24 hours. Rectal dosing is not recommended due to erratic absorption. Contraindications include renal impairment, hepatic impairment, or hypersensitivity to the agent itself. The wide dosing range and variability of onset and duration make chloral hydrate a far-from-perfect sedative for use in the Emergency Department. Its primary usage has been with outpatient testing.

DPT

DPT, also known as the "lytic cocktail," was popularized for the induction of general anesthesia. It was expanded to use in the outpatient setting and had many adverse events. It consists of Demerol, Phenergan, and Thorazine in a 2:1:1 or 4:1:1 mixture. It is administered intramuscularly at a dose of 1 mg/kg based upon the Demerol component. Intramuscular absorption is erratic with up to one-third of children never obtaining moderate to adequate sedation. It produces too deep a level of sedation that is difficult to reverse in the other two-thirds of the children. The mean time to Emergency Department discharge is 5 hours due to the prolonged sedation. The mean time for the child's behavior to return to normal is 19 ± 15 hours.

Many children have suffered respiratory depression, hypoxemia, and apnea due to this agent. DPT is an agent of the past due to its prolonged action and significant potential for respiratory depression. The DPT agent has no use in the Emergency Department. Agents now exist that are safer, are easier to use, and have a more rapid recovery. Its use is described here only for completeness. Do not use this drug combination for PSA.

INHALATION AGENTS

NITROUS OXIDE

Nitrous oxide is an anesthetic gas that has been used in the outpatient procedural arena since the 1950s (Chapter 158). It produces amnesia, anxiolysis, and sedation with a variable degree of analgesia.[114] It dissociates a patient from pain and their surroundings. Nitrous oxide is self-delivered via a handheld demand valve mask in a 50/50 mixture with oxygen and can be administered safely in concentrations of up to 70%.[115] The risk of oversedation is minimized with this mode of administration. The patient will drop the mask once they are too sedated to coordinate self-administration.

The onset of action of nitrous oxide is 30 to 60 seconds with a peak in 3 to 5 minutes. It has a similar washout period once delivery is ceased. Nitrous oxide is eliminated unchanged via the lungs thereby minimizing any drug-drug interactions. Nausea and vomiting are common.[116] The feelings of disorientation and agitation are common. Nitrous oxide can rarely produce laryngospasm.[117]

Contraindications to the use of nitrous oxide include impaired mental status, concomitant intoxication, pregnancy, potential to expand air-filled cavities (e.g., pneumothorax or bowel obstruction), and coadministration of narcotics within 4 hours as general anesthesia may be induced. Relative contraindications include a full meal within 1 hour of its use and children less than 5 years of age due to delivery system compliance.

REVERSAL AGENTS

It should be well recognized that a desired endpoint can be overshot resulting in an apneic and hypotensive patient despite good preprocedural assessments, appropriate agent selection, appropriate dosing, and titration. There are two reversal agents (i.e., flumazenil and naloxone) that can be of help in these situations. **Reversal agents are not routinely required following PSA. They are reserved for the patient who develops apnea, hypoxia, and/or hypoventilation**.

FLUMAZENIL (ROMAZICON)

Flumazenil is a benzodiazepine antagonist that works by competitive inhibition at the gamma-aminobutyric acid (GABA) receptor. **Intravenous administration will rapidly reverse central nervous system depression and respiratory depression from benzodiazepines within 2 minutes.** Begin by administering a dose of 0.02 mg/kg to a maximum of 0.2 mg over 15 seconds. Administer additional 0.2 mg doses at 1 minute intervals until the desired state of consciousness is achieved. Maximum dosing is 1.0 mg over 15 minutes or 3.0 mg over 60 minutes. **The benzodiazepine metabolites are active longer than the reversal agent. Carefully monitor a patient receiving flumazenil for resedation**. A safe recommendation would require a minimum observation of 2 hours after the last dose is given.

Flumazenil is contraindicated in patients taking benzodiazepines for an extended amount of time or patients with an underlying seizure disorder. These patients are prone to seizure activity with the administration of flumazenil. It is also contraindicated in patients taking tricyclic antidepressants.

NALOXONE (NARCAN)

Naloxone is a pure opioid antagonist that works by competitively inhibiting narcotics at the opioid receptor. **Intravenous administration reverses the respiratory depressive effects of opioids within 1 to 2 minutes. Its clinical duration of effect is approximately 20 to 30 minutes.** Long-acting narcotics may cause resedation. **The opioids and their metabolites are active longer than the reversal agent. Carefully monitor a patient receiving naloxone for resedation and respiratory depression**. The drug can be delivered via multiple routes (i.e., endotracheally, intramuscularly, intravenously, subcutaneously, and sublingually). The administration of 1 to 2 mg intravenously in adults and 0.1 mg/kg in children will reverse most respiratory arrest situations. Administer additional doses every 2 to 3 minutes to a total of 10 mg. Actively seek another etiology of the sedation and respiratory depression, other than narcotics, if the respiratory depression is not reversed after 10 mg of naloxone. **Use caution as naloxone can result in opioid withdrawal in those with physical dependence or intoxicated with narcotics.**

Small aliquots of 40 μg titrated to effect may be delivered in a situation where the patient is slightly oversedated and rapid full reversal of the narcotic is not desired. Mix 0.4 mg of naloxone with 9 mL of normal saline to produce a concentration of 40 μg/mL. Administer 1 to 2 mL aliquots every 1 to 3 minutes to alleviate respiratory depression yet maintain the narcotic analgesic effect.

TECHNIQUE

The technique of PSA can be quite variable depending on the institution and the agents chosen to administer. A sample PSA protocol can be found in **Table 159-5**. **PSA requires two persons at a minimum.** One person must monitor the patient while the other administers the medication and performs the procedure. **Take the time to do it right!** The right time to perform PSA is not when the Emergency Department is at capacity and the acuity of other patients is high. The medications must be titrated to effect which can take up to 15 minutes.

TABLE 159-5 A Sample PSA Protocol

1. Place the patient supine in bed with the rails up.
2. Obtain intravenous access with a large-bore angiocatheter and hang a 1 L bag of normal saline.
3. Apply the cardiac monitor to record pulse, respiratory rate, and blood pressure at start of procedure and every 3–5 minutes during the procedure.
4. The nurse must monitor the patient's level of consciousness at the start of the procedure and every 3–5 minutes during the procedure.
5. Apply continuous pulse oximetry to monitor and maintain the oxygen saturation > 95%, or no less than 3–5% below baseline.
6. Apply supplemental oxygen by nasal cannula at 2–4 L/min.
7. Place the resuscitation cart at the bedside.
8. Set up suction equipment and ensure that it is working properly.
9. Have the required medicines at the bedside and drawn up into labeled syringes.
10. Have the reversal agents (naloxone and flumazenil) at the bedside, not drawn up unless needed.
11. Administer and titrate the medications to effect.
12. Administer local or regional anesthesia if indicated.
13. Perform the procedure for which PSA was performed.
14. Administer additional doses of sedatives and analgesics as needed.
15. Closely observe and monitor the patient until they are awake, alert, and back to baseline.

ASSESSMENT

Nursing personnel managing the care of the patient receiving PSA must continuously monitor the patient's airway, breathing, circulation, and mental status. It is imperative to observe the rise and fall of the chest wall with the focus upon the work of breathing. **Relying solely on pulse oximetry may give a false sense of security**. **Observation of a progressive slowing in the patient's respiratory effort is a sign of impending hypercapnia or hypoxia that is seen well ahead of any monitoring alerts or cutoffs.** Avoid sterile wraps that cover the entire face or chest. Observe the patient for a decrease in respiratory rate or depth of breathing. Notice any abnormal airway sounds indicative of partial airway obstruction, bronchospasm, laryngospasm, or stridor. Visually monitor the peripheral perfusion of assessments of skin and mucosa color, moisture, and temperature. Assess pulse quality and rate in addition to frequent blood pressure determinations. Blood pressure monitoring may awaken the patient and forgo the ability to complete the diagnostic or therapeutic interventions in cases of mild sedation (e.g., pediatric sedation for neuroimaging). Continually assess the patient's level of consciousness throughout the procedure and recovery period. The previously mentioned scoring systems offer one means of determining the level of sedation. Record the patient's responses to verbal stimuli, if applicable.

AFTERCARE

The patient must meet several criteria to ensure their safety before leaving the Emergency Department. Vital signs must be appropriate to the age of the patient and comparable to preprocedural parameters. The respiratory effort must return to baseline. Mobility must be equal to or better than that before the procedure. The patient must be able to follow commands and discharge instructions. Preprocedural levels of consciousness and mental status must be present. The patient must be able to tolerate oral fluids. The pain of the procedure for which PSA was performed must be controlled. The patient must be discharged in the care of a responsible adult who understands the discharge instructions. All these criteria must be documented in the medical record and timed prior to discharge.

Patients who have received multiple medications or a reversal agent will require a longer recovery period. A 2 hour observation window is prudent. Provide written discharge instructions and explain them to the patient and the responsible adult who will accompany the patient home. Examples of preprinted pediatric and adult discharge sheets are presented in **Tables 159-6 and 159-7**.

COMPLICATIONS

Respiratory depression and hypoxia are inherent risks of PSA despite efforts to minimize risk with prudent patient risk-benefit assessment, appropriate drug selection, and appropriate dosing.[3] Individual patient responses to analgesic, hypnotic, or sedative agents can be very unpredictable. Part of the preparation includes bedside availability of reversal agents in anticipation of these complications.

The first course of action is simply to provide patient stimulation in the event of respiratory depression. Painful stimulation is most easily applied by continued traction or manipulation of the injured extremity. Patients undergoing PSA will frequently not become apneic until after the procedure is complete and the noxious stimulus is no longer present. The jaw-thrust maneuver is another

TABLE 159-6 Pediatric PSA Discharge Instructions

In order to best care for your child, they were given medications that can cause drowsiness and clumsiness over the next few hours. While most of the effect has worn off by this time, their coordination and judgment may still be affected. During this time it is very important to directly watch your child's activity to assure their safety.

Diet . . . Do not let them eat or drink for the next 2 hours. At that time, start with sips of water or juice before giving them solid foods. Children younger than 1 year may start feeding in 1 hour.

Activity . . . For the next 6–8 hours, do not allow them to participate in any activity where lack of coordination could cause injury. Examples would include biking, climbing, running, playing on swing sets or monkey bars, swimming, or even stair climbing without your assistance. Your direct observation of their activities is very important during this recovery time. You need to directly watch your child if they are bathing, showering, cooking, or using any tool or device where injury could result due to poor judgment or coordination.

Sleep . . . Your child may wish to nap or sleep for the duration of the night. You need to awaken them in 2 hours. If they should appear as they normally would after being awoken from sleep, you can let them finish their nap or sleep through the night.

Contact or return to the Emergency Department immediately if:

1. You notice anything you feel is unusual about your child.
2. You feel your child's breathing is abnormally fast, slow, or shallow.
3. Your child's skin appears pale or grayish in color.
4. You have more difficulty awakening your child.
5. Your child has repeated vomiting (3 or more times).

Emergency Department phone number: _____

method of providing significant stimulation while placing yourself at the patient's head and assisting ventilation by opening the airway. Support the patient's ventilation with a bag-valve-mask device while the appropriate reversal agent(s) are being drawn up if stimulation is inadequate. Naloxone will rapidly reverse the respiratory depression if a narcotic was administered. Flumazenil will reverse the respiratory depression if a benzodiazepine was administered.

There is no way to determine which reversal agent is best suited to reverse the respiratory depression when both a narcotic and benzodiazepine have been administered. A rational approach is to first administer flumazenil. This maintains the needed analgesia while, hopefully, improving the respiratory drive. Administer naloxone if the flumazenil does not reverse the respiratory depression.

Laryngospasm is a real but relatively rare event during PSA provided the appropriate patient and drug selection was employed.[3] Fentanyl, ketamine, midazolam, and phenobarbital may all cause laryngospasm. This tends to be a brief and self-limited event that can usually be supported with the use of positive-pressure ventilation. Administer succinylcholine at a dose of 1 to 2 mg/kg intravenously or 4 to 5 mg/kg intramuscularly. Support the patient's airway and provide airway management if laryngospasm is severe or persistent.

TABLE 159-7 Adult PSA Discharge Instructions

To best manage your condition, you were given medications that can cause drowsiness and clumsiness over the next several hours. While most of the effect has worn off by this time, your coordination and judgment may still be affected.

1. You should avoid all activities that could result in injury due to drowsiness or impaired coordination or judgment. Examples would include driving a motorized vehicle, operating machinery, biking, swimming, skating, or any activity at height where a fall could result.
2. The effects of the medications could cause you to feel weak, shaky, or even nauseated. You should rest during this time and wait 1–2 hours before trying small sips of liquids. You can increase to solid foods once you start feeling better.
3. Take additional pain medications as directed by the doctor.
4. For the next 24 hours, avoid taking anything that could make you drowsy. Examples would include alcohol, sleeping pills, and antihistamine medications.

Please call the Emergency Department if you have any questions or feel anything is unusual about your recovery.

Emergency Department phone number: _____

TABLE 159-8 Pregnancy Categories for PSA Agents

Agent	Safety in pregnancy	Safety in lactation
Morphine	C	?
Meperidine	C but +	?
Fentanyl	C	?
Sufentanil	C	?
Alfentanil	C	?
Diazepam	D	–
Midazolam	D	–
Ketamine	?	?
Thiopental	C	?
Methohexital	B	?
Etomidate	C	?
Propofol	B	–
Nitrous oxide	–	?
Chloral hydrate	C	?
Naloxone	B	?
Nalmefene	B	?
Flumazenil	C	?
Atropine	C	–
Glycopyrrolate	B	–

Legend:
(A) Safety established in human studies
(B) Presumed safe based on animal studies
(C) Uncertain safety, animal studies show adverse effects, no human studies
(D) Unsafe
(+) Generally accepted as safe
(–) Generally regarded as safe
(?) Safety unknown or controversial

Virtually all agents can induce nausea and vomiting.[3,118] Patients with recent oral intake are at a higher level of risk for aspiration.[118] **Always have suction available in case it is required.** Hypotension induced by opiates or benzodiazepines is responsive to fluid management and the corresponding reversal agent. Chest wall rigidity from short-acting opioids that cannot be reversed with naloxone requires succinylcholine and airway management to support ventilation and oxygenation.

Allergic reactions can result from the administration of any of the medications. Management is like other allergic reactions in terms of treatment with epinephrine, diphenhydramine (i.e., an H_1 antagonist), an H_2 antagonist, and methylprednisolone.

The use of PSA in pregnant and lactating patients must be undertaken with great caution. The safety of many of the agents is unknown or of concern (**Table 159-8**). Consult an Anesthesiologist, Gynecologist, or Obstetrician prior to performing PSA on pregnant and lactating women.

SUMMARY

PSA is a necessary part of Emergency Medicine practice. Skillful application in the appropriate clinical scenarios will result in relief of patients' anxiety and pain. We have the necessary pharmaceuticals to control patients' pain and anxiety. It is essential to possess the knowledge and training to understand each patient's needs and satisfy this requirement with a careful risk-benefit assessment and appropriate drug selection. It is essential to maintain the necessary high standards to safely get the patient through these procedures with a minimum of risk and complications. Competency extends beyond the Emergency Physician and includes nursing and support personnel. They too must be knowledgeable as to the content of procedural sedation and analgesia with an uneventful recovery.

Everyone must know their limitations. Become very comfortable with a limited set of medications that will enable one to address

95% of all PSA requirements. Secure the assistance of a colleague or backup from an Anesthesiologist in difficult situations. PSA will never be a substitute for a little patience and a soft, gentle manner. Reserve PSA use for the patient who truly needs the intervention.

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SECTION 10

Obstetrical and Gynecologic Procedures

160 Ultrasound in Early Pregnancy

Srikar Adhikari, Wes Zeger, and Lori Stolz

INTRODUCTION

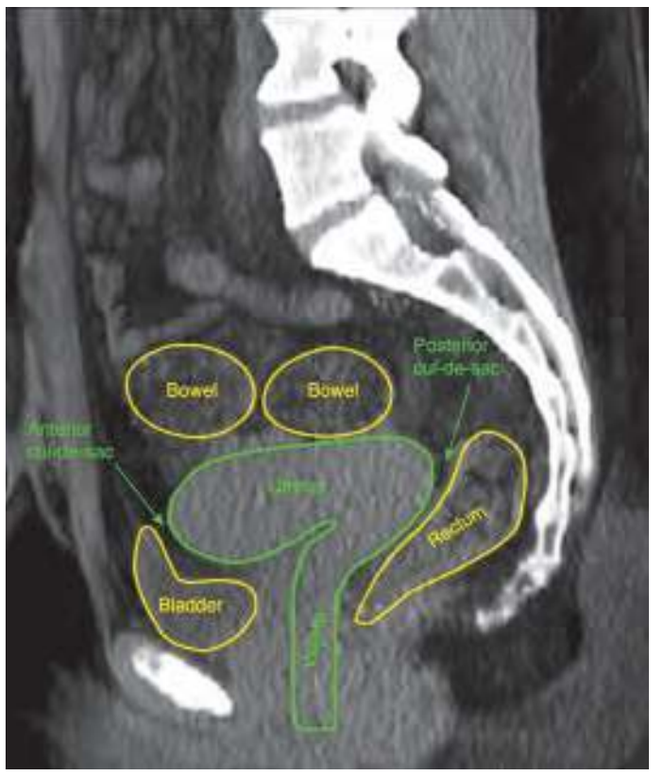
Abdominal pain and vaginal bleeding are common complaints of patients presenting to the Emergency Department (ED) in the first 20 weeks of pregnancy. Ectopic pregnancy is the most common life-threatening emergency during the first trimester of pregnancy. The current incidence of ectopic pregnancy is difficult to estimate from existing data.[1-3] The overall incidence of ectopic pregnancy has increased during the mid-twentieth century due to increased occurrence of pelvic inflammatory disease.[1-3] History and physical examination have proven to be unreliable in excluding the presence of an ectopic pregnancy. Pelvic ultrasound (US) is the diagnostic test of choice in the initial evaluation of these patients with possible ectopic pregnancy.

Emergency Physicians have been using bedside pelvic US in the evaluation of first-trimester pregnancy symptoms for more than a decade.[4] Prior studies have demonstrated that ED pelvic US decreases cost, patient length of stay, and morbidity.[5-7] **The main goal of ED pelvic US is to identify an intrauterine pregnancy (IUP).** An IUP is identified 60% to 70% of the time on bedside US in women presenting with abdominal pain and/or vaginal bleeding to ED.[8] The accuracy of pelvic US performed by Emergency Physicians for identifying an IUP has been well established.[9-11] The scanning skills of Emergency Physicians have extended beyond just identifying an IUP to diagnosing ectopic pregnancy using bedside US with increasing experience.[12]

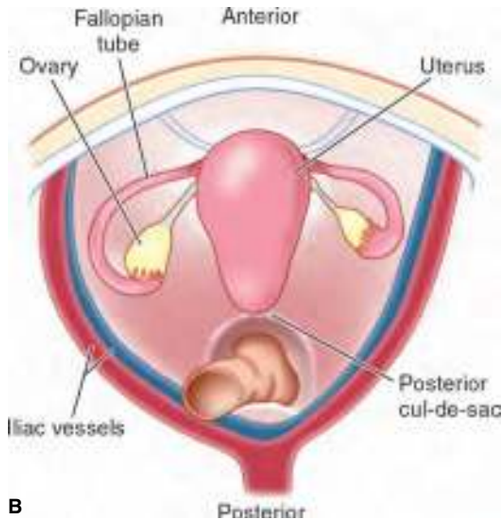
ANATOMY AND PATHOPHYSIOLOGY

The pelvic cavity extends from the iliac crests superiorly to the pelvic diaphragm inferiorly. The female pelvis consists of the genital tract (i.e., vagina, uterus, and uterine tubes), ovaries, urinary bladder, a portion of the ureters, lower intestinal tract, pelvic musculature, ligaments, and peritoneal spaces.

The uterus is an oval, hollow, muscular, and pear-shaped extraperitoneal organ located in the pelvis between the urinary bladder anteriorly and the rectum posteriorly (**Figure 160-1**). It is divided anatomically into the fundus, the body, the isthmus, and the cervix. The central cavity of uterus opens into a fallopian tube on either side and into the vagina below. The uterus is usually anteverted



A



B

FIGURE 160-1. Anatomy of the normal female pelvis. ***A.*** Midline sagittal CT view. ***B.*** Transverse view.

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(i.e., pointing forward toward the anterior abdomen) and anteflexed (i.e., flexed forward over the bladder). It may be retroverted (i.e., pointing back toward the spine) and retroflexed (i.e., flexed away from the bladder) in some patients. The long axis of the uterus rarely lines up exactly with the long axis of the body. Most often it is found angled to one side. The size of the uterus is variable depending on the patient's parity, pubertal stage, and age. A postpubertal adult female uterus is approximately 8 cm long, 5 cm wide, and 3 cm deep. The endometrial canal is formed by the central, linear, and opposing surfaces of the walls of the endometrial cavity. The endometrial canal continues as the endocervical canal inferiorly. The vagina extends from the cervix to the external genitalia. It lies between the urinary bladder and the rectum (**Figure 160-1*A***).

The fallopian tubes are bilateral coiled muscular tubes located lateral to the uterus (**Figure 160-1*B***). They are anteromedial to the ovaries and posterior to the urinary bladder. Each fallopian tube is approximately 10 cm in length and 3 mm in diameter. The fallopian tube is divided into four anatomic segments from proximal to distal (i.e., interstitial, isthmus, ampulla, and infundibulum).

The ovaries are bilateral almond-shaped structures that measure approximately 4 cm in length, 3 cm in width, and 2 cm in height. The size of the ovaries varies depending on the patient's age, menstrual status, and menstrual cycle phase. They are quite mobile and their position is variable. They are most often identified posterolateral to the uterus against the pelvic side walls and just anteromedial to the internal iliac vessels within the adnexa (**Figure 160-1*B***).

The female pelvis has an anterior cul-de-sac and a posterior cul-de-sac (**Figures 160-1*A* and 160-1*B***). The anterior cul-de-sac or vesicouterine pouch is the peritoneal space between the anterior wall of the uterus and urinary bladder. The posterior cul-de-sac or pouch of Douglas is the area between the uterus and the rectum. **The posterior cul-de-sac is the most dependent area of the peritoneal sac. A small amount of free fluid in the posterior cul-de-sac is a normal physiologic finding. Physiologic free fluid in the pelvis of a pregnant patient is rare. A large amount of fluid in the posterior cul-de-sac is considered abnormal. Any amount of fluid identified in the anterior cul-de-sac is also considered abnormal.**

INDICATIONS

The indications to perform a pelvic US examination during early pregnancy include abdominal pain, back pain, dizziness, hypotension, a pelvic mass, pelvic pain, syncope, tachycardia, trauma, or vaginal bleeding on physical examination.

CONTRAINDICATIONS

There are no absolute contraindications for a pelvic US during early pregnancy. A recent pelvic surgical procedure is a relative contraindication. Always perform a bimanual or speculum examination before performing an endovaginal US examination. Use caution when performing an endovaginal US examination if the cervical os is open. **The US transducer should never enter the endocervical canal or the uterus.**

EQUIPMENT

- US machine
- Low-frequency 5-2 MHz curvilinear transducer (**Figure 160-2*A***)
- High-frequency 9-4 MHz endocavity transducer (**Figure 160-2*B***)
- Condom or US transducer sheath, nonlatex preferable
- US gel

A

B

FIGURE 160-2. US transducers or probes used in pelvic ultrasonography. ***A.*** Curvilinear transducer. ***B.*** Endocavitary transducer.

PATIENT PREPARATION

Obtain a relevant history from the patient prior to performing an US examination. It should include current symptoms, date of last menstrual period, use of fertility agents, assisted reproductive technology, gravidity, parity, complications of prior pregnancies, and prior pelvic surgeries. Use a nonlatex condom or transducer sheath for the US examination if the patient is allergic to latex. Discuss with the patient what the US examination entails and obtain verbal consent. **A sonographer should always be accompanied by a chaperone when performing a transvaginal US examination.**

A full urinary bladder is essential for a transabdominal US examination. The full bladder displaces bowel loops from the pelvis and provides an acoustic window to visualize the underlying pelvic organs. An excessively distended bladder can compress or displace the pelvic structures out of view. If this happens, instruct the patient to partially empty their bladder. **Transvaginal US requires an empty urinary bladder.** A distended bladder displaces the uterus and ovaries, creates artifacts, and makes it difficult to visualize the pelvic structures.

TECHNIQUES

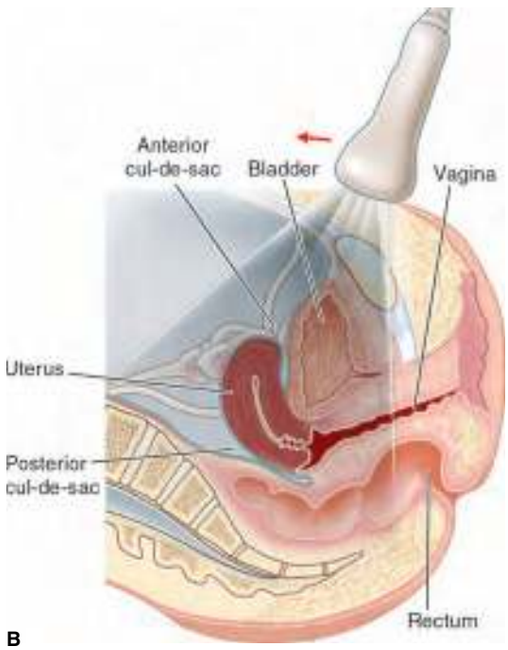
TRANSABDOMINAL SONOGRAPHY

Two approaches are used to perform a pelvic US examination of an early pregnancy.[4] Transvaginal sonography (TVS) is used for most cases in conjunction with transabdominal sonography (TAS). Both techniques provide complimentary information. TAS usually precedes TVS while the patient has a full bladder. TAS helps define the spatial orientation of the pelvis. The field of view is larger with TAS compared to TVS. TAS allows better visualization of the relationship of the uterus with adjacent pelvic and abdominal structures. Hemoperitoneum and large pelvic masses that extend outside of the true pelvis are better visualized with TAS. **TVS is not required if the TAS provides adequate information to make a diagnosis.**

TAS OF THE UTERUS AND VAGINA IN THE SAGITTAL PLANE

A low-frequency 5-2 MHz curvilinear transducer is typically used for TAS. It can be performed using a low-frequency 5-1 MHz phased array transducer if a curvilinear transducer is not available. Visualize the uterus and adnexal structures in both the sagittal (i.e., longitudinal) and transverse (i.e., cross-section or short) planes. Place the patient supine. Apply US gel to the suprapubic area and to the footprint of the transducer. Begin scanning in a sagittal or longitudinal plane by placing the transducer just above the pubic symphysis. Aim the transducer indicator toward the patient's head (**Figure 160-3**). The indicator on the transducer corresponds with the left side of the screen and cephalad structures are seen on the left side of the image in the sagittal plane (**Figure 160-4**).

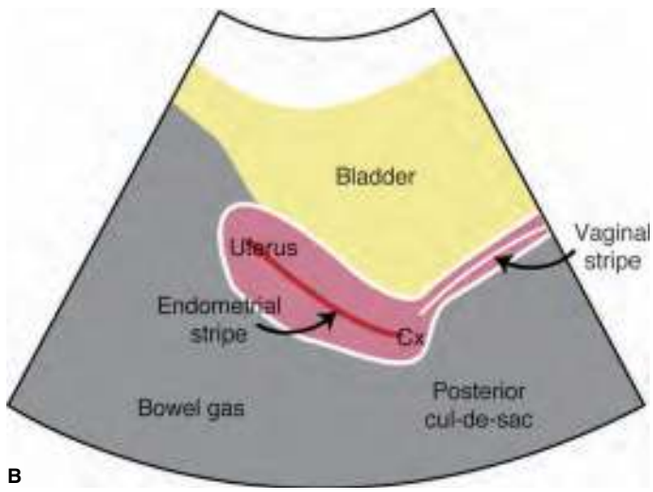
A



B

FIGURE 160-3. Sagittal view of TAS. ***A.*** Transducer positioning. The arrow indicates the direction of the indicator. ***B.*** The US beam is directed through the anatomic structures in the midsagittal plane. (Courtesy of Stephen Leech, MD.)

A



B

FIGURE 160-4. Sagittal view of a nonpregnant uterus by TAS. ***A.*** The US image. ***B.*** Corresponding anatomic diagram. (Courtesy of Stephen Leech, MD.)

Identify the uterus and vagina in the sagittal or long axis (**Figure 160-4**). Note the position of the uterus and whether it is anteverted or retroverted. The shape and position of the uterus are extremely variable. The position changes depending on the amount of urine in the urinary bladder. Obesity and a retroverted position can make it difficult to identify the uterus. Identify each of the anatomic areas of the uterus (i.e., the fundus, the body, and the endometrial stripe).

The normal uterus has a sonographically low-gray homogenous texture (**Figure 160-4*A***). The appearance of the uterine endometrium changes with the different phases of the menstrual cycle. The normal endometrial stripe or endometrial canal is visible as a thin and bright echogenic line in the middle of the uterus (**Figure 160-4**). The sonographic appearance of the endometrial stripe changes as the thickness of the endometrium changes cyclically with the menstrual cycle. Use the direction of the endometrial stripe to find the uterine position and determine if it is anteverted or retroverted. The cervix has a similar appearance to that of the uterus.

Adjust the transducer to visualize the uterine body and fundus in the same scanning plane with the vagina and cervix. Slide the transducer to the right and/or left of midline, angle the transducer toward the patient's feet, or combine both maneuvers until the vagina and cervix come into view. The vagina is visualized between the anechoic bladder anteriorly and the echogenic rectum

posteriorly (**Figure 160-4**). The muscular vaginal walls appear low gray and isoechoic to the uterine myometrium. The central mucosal lining of the collapsed vaginal canal walls appears thin, linear, and hyperechoic. The vagina is visible as a tubular extension of the uterus in the sagittal plane. It is flattened and oval shaped in the transverse plane. The rectum usually has air and fecal material casting a posterior acoustic shadow in the far field of the image. The bladder is always visualized in the right upper corner of the image in transabdominal view. A fully distended bladder is visible as an anechoic structure with hyperechoic walls anterior to the uterus in the near field of the image (**Figure 160-4**). The bladder cavity will not be visible if it is empty and collapsed.

Twist the transducer varying degrees to align the long axes of the uterus, vagina, endometrial canal, endocervical canal, and vaginal canal. Move the transducer from side-to-side through the entire width of the uterus in the transverse plane looking for evidence of an IUP. Determine the long axis of the uterus and vagina. Sweep the transducer from left-to-right and right-to-left to scan through and beyond the lateral boundaries of the uterus, adnexa, and pelvic side walls on both sides. Note the location of the ovaries if they are visualized. **The uterus serves as the reference point for other pelvic structures.** It might be necessary to apply firm pressure with the US transducer to the patient's abdominal wall to displace bowel gas and improve image quality.

TAS OF THE UTERUS AND VAGINA IN THE TRANSVERSE PLANE

Rotate the US transducer 90° counterclockwise to scan in the transverse plane (**Figure 160-5**). The transducer indicator will be aimed toward the patient's right side. The transducer indicator and the marker on the screen are aimed in the same direction. Angle the transducer inferiorly to identify the transverse section of the vagina between the bladder anteriorly and the rectum posteriorly. Adjust the transducer as necessary to identify the vaginal canal. Angle the transducer inferiorly and scan through and beyond the margins of the vagina and pelvic cavity. Slowly straighten the transducer back to the perpendicular position. Continue scanning superiorly through and beyond the margins of the vagina and onto the cervix. The transverse section of the cervix will appear slightly larger than the vagina. Adjust the transducer to visualize the endocervical canal.

Keep the transducer perpendicular and angle it further superiorly to scan through and beyond the cervix and onto the uterine body (**Figure 160-6**). The body of the uterus will appear larger than the cervix. Twist the transducer gently to visualize the centrally located endocervical canal. Continue to scan superiorly through the body of the uterus and onto the fundus. The transverse section of the uterine fundus will appear slightly larger than the body. Adjust the transducer as necessary to visualize the centrally located endometrial canal. Angle the US transducer further superiorly through the fundus and urinary bladder walls up to the level of the umbilicus.

TAS OF THE OVARIES AND ADNEXA

Normal ovaries are often not visualized with TAS. They are relatively small and hidden by bowel gas or other pelvic structures with similar echogenicity. The location and lie of the ovaries are quite variable. The long axis of the ovary can lie within either scanning plane, transverse or sagittal. The ovaries are usually found lateral to the body or fundus of the uterus. Sweep laterally from the uterus in both planes to find the ovaries. Look for the greatest dimension of the ovary regardless of the transducer position. The greatest dimension may be in the transverse, sagittal, or oblique plane. Determine the volume of the ovary by measuring the length, width, and height using views obtained in two orthogonal planes. Blood vessels can be confused with ovarian follicles. Rotate the transducer 90° and determine if the structure becomes "tube-like." If so, it is most likely a blood vessel. Doppler can help to make this distinction.

It is difficult to thoroughly evaluate the adnexa on TAS. The ovaries can serve as landmarks for assessing adnexal pathology. Survey the adnexa for abnormalities such as masses and dilated tubular structures. If an abnormality is found in the adnexa, note its size, sonographic appearance, and relationship to the uterus and ovaries.

Evaluate the cul-de-sac for free fluid, a hematoma, or a mass in the sagittal and transverse planes. Fluid in the cul-de-sac is visible as an anechoic area. Note the echogenicity of the fluid. If a mass is visualized, note its size, shape, location, sonographic appearance, and relationship to the ovaries and uterus. Bowel can look like a pelvic

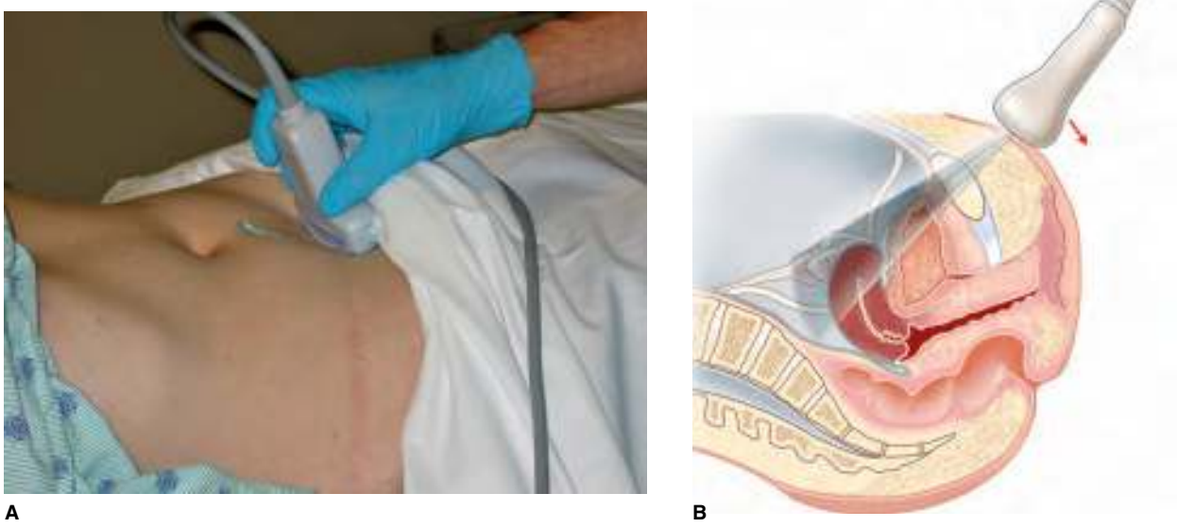
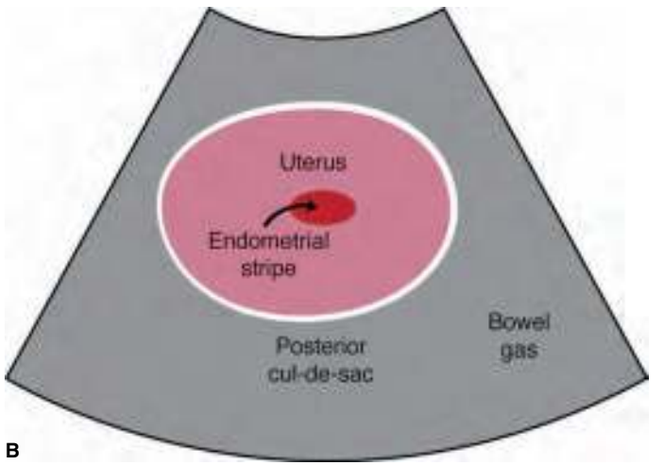


FIGURE 160-5. Transverse view of a nonpregnant uterus by TAS. ***A.*** Transducer positioning. ***B.*** The US beam is directed through the anatomic structures in the transverse plane. The arrow indicates the direction of the indicator. (Courtesy of Stephen Leech, MD.)

A



B

FIGURE 160-6. Transverse view of a nonpregnant uterus by TAS. ***A.*** The US image. ***B.*** Corresponding anatomic diagram. (Courtesy of Stephen Leech, MD.)

mass, an ovary, or a dilated fallopian tube. Hold the US transducer still over the structure and observe it for signs of peristalsis to distinguish a bowel loop from other structures. Differentiation of normal bowel loops from a mass may be difficult by TAS. TVS might help to differentiate a suspected mass from fluid and fecal material within the normal rectosigmoid colon.

TRANSVAGINAL SONOGRAPHY

TVS is necessary after TAS when pelvic structures require further evaluation, structures are not visible on TAS, or the TAS is inconclusive.13 Instruct the patient to void after completing the TAS and then perform the TVS. The endocavity transducer is placed closer to pelvic structures when compared to TAS. TVS provides more anatomic detail of pelvic organs than TAS, especially adnexal structures. It provides images of pelvic structures with higher resolution, allowing the early identification of intrauterine contents and adnexal abnormalities.

Place the patient in the lithotomy position. This position allows for a full range of movement of the endocavity US transducer. TVS is best performed with an empty bladder following the speculum and bimanual examination to minimize patient discomfort. Remove any tampons in the vagina during the speculum examination. Note any vaginal or cervical lesions, vaginal wounds, bulging membranes through the cervix, or if the cervical os is open. Explain the procedure and verbally consent the patient regarding the examination.

FIGURE 160-7. Endocavity transducer preparation. US gel is first applied to the footprint of the transducer before it is inserted into the sheath cover. Additional US gel is then applied to the distal end of the sheath cover.

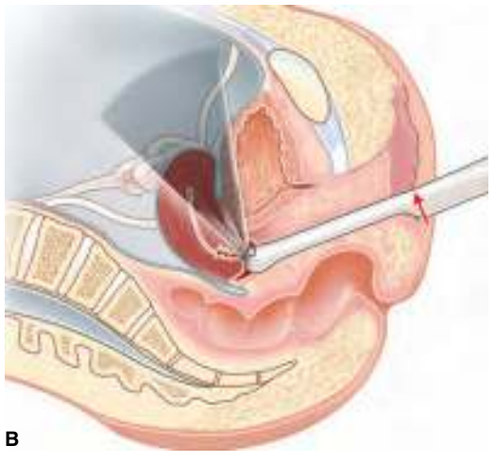
A high-frequency 9-4 MHz endocavity transducer is used for TVS. Apply US gel to the footprint of the transducer and cover it with a condom or transducer sheath (**Figure 160-7**). Smooth all the air bubbles away from the footprint of the transducer by running a finger over the condom or transducer sheath covering the distal end of the transducer. This ensures smooth transmission of the US beam and prevents imaging artifacts. Apply additional US gel to the outside of the condom or transducer sheath before inserting the transducer into the patient's vagina.

TVS requires viewing the uterus and other pelvic structures in the sagittal and transverse planes. Each structure must be scanned left to right in the sagittal plane and up and down in the transverse plane. Image orientation in TVS can be challenging due to the narrow field of view, the inferior scanning approach, and variations in the position of the pelvic organs. **It is important to determine the proper position of the US transducer before inserting it into the vagina.** Aim the transducer indicator toward the ceiling to scan in the sagittal plane. Rotate the transducer 90° counterclockwise with the indicator aimed toward the patient's right side to scan in the transverse plane.

TVS OF THE UTERUS AND VAGINA IN THE SAGITTAL PLANE

Gently insert the US transducer downward and backward into the lower third of the vagina with the indicator directed toward the ceiling (**Figure 160-8**). This will position the US transducer adjacent to the cervix (**Figure 160-8*B***). Start scanning by slowly lowering the handle toward the floor to view the fundus of the uterus in the sagittal plane. The fundus of an anteverted uterus will be on the left side of the screen pointing toward the anterior abdominal wall (**Figure 160-9**). A retroverted uterine fundus will be on the right side of the screen pointing in the direction of the posterior abdominal wall (**Figure 160-10**). To completely visualize the retroverted

A



B

FIGURE 160-8. Sagittal view of a nonpregnant uterus by TVS. ***A.*** Transducer positioning. The arrow indicates the direction of the indicator. ***B.*** The US beam is directed through the anatomic structures in the sagittal plane. (Courtesy of Stephen Leech, MD.)

uterus, lift the US transducer handle toward the ceiling, moving the US transducer tip posteriorly and inferiorly.

Move the transducer side to side and vice versa to evaluate the centrally located endometrial canal and lateral margins. Any urine

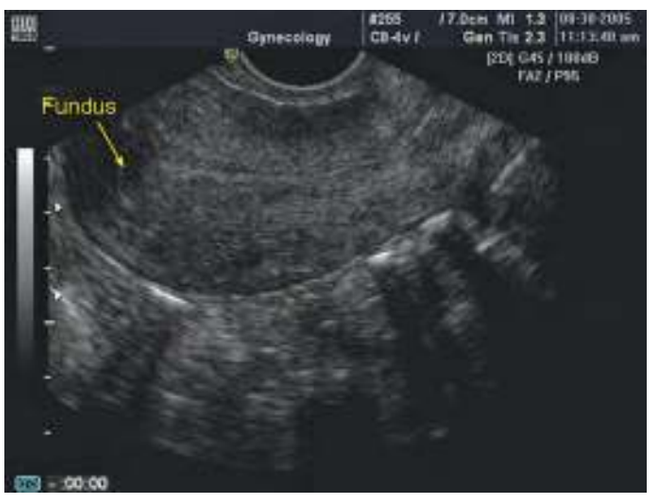


FIGURE 160-9. TVS of an anteverted uterus in the sagittal plane.

FIGURE 160-10. TVS of a retroverted uterus in the sagittal plane.

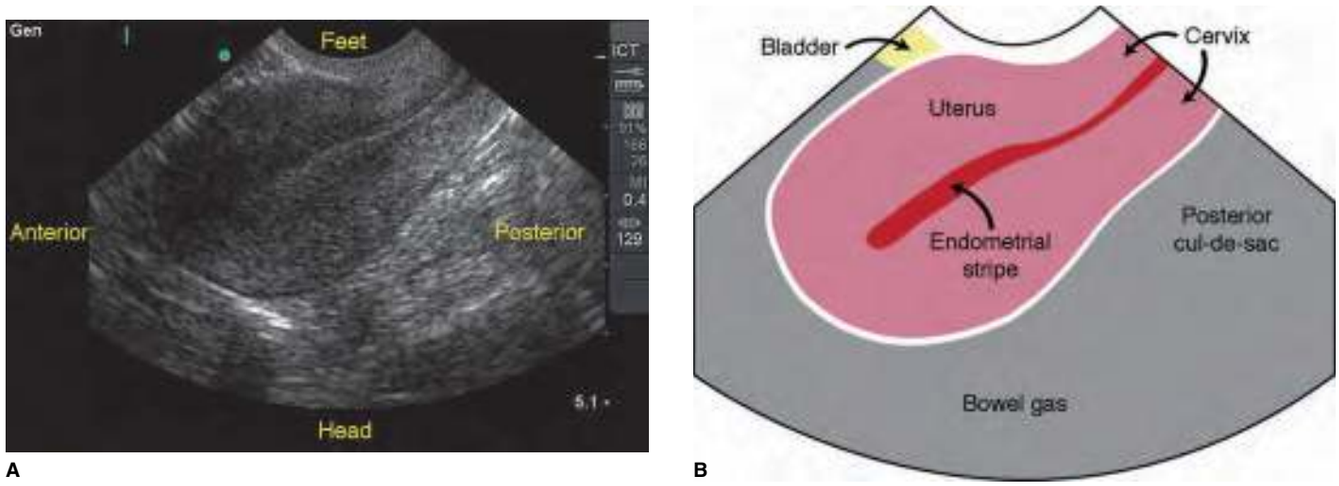
in the bladder will be visible in the left upper corner of the screen (**Figure 160-11**). Slowly lift the US transducer handle toward the ceiling to visualize the body of the uterus and the cervix. Continue moving the US transducer side to side to evaluate the lateral margins, the centrally located endometrial canal, and the endocervical canal. It may be necessary to slightly rotate or twist the US transducer to fully visualize the long axis of the uterus and the entire endometrial stripe (**Figure 160-11**). Thoroughly screen for any intrauterine contents. Note the contents, if any, of the posterior cul-de-sac at the level of uterine fundus, uterine body, and the cervix. **The goal is to scan the entire uterus from right to left through its entire length.**

TVS OF THE ADNEXA IN THE SAGITTAL PLANE

Continue scanning sagittally to the adnexal areas after scanning the uterus in the sagittal plane. Lower the US transducer handle and identify the fundus of the uterus. Move the US transducer handle toward the patient's left thigh to scan through the right adnexa. This movement directs the US beam toward the right adnexal region. Return the US transducer to the midline and reidentify the fundus. Move the US transducer handle toward the patient's right thigh to scan through the left adnexa. This movement directs the US beam toward the left adnexal region. Repeat these lateral sweeps through the adnexal areas at the levels of the uterine body and cervix. The uterus serves as the reference point for other pelvic structures while performing the pelvic US examination.

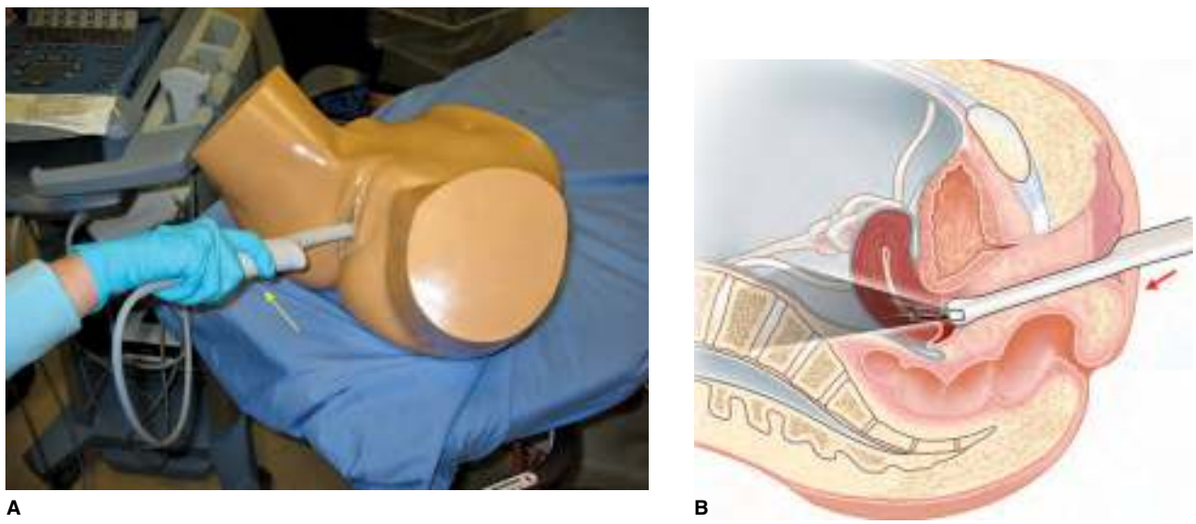
TVS OF THE UTERUS AND VAGINA IN THE TRANSVERSE PLANE

Scan in the transverse plane after completing TVS in the sagittal plane. Turn the US transducer 90° counterclockwise from the sagittal plane (**Figure 160-12**). The US transducer indicator will be directed toward the patient's right side. The orientation in this plane is like a computed tomography (CT) scan image. The left side of the US image corresponds to patient's right side (**Figure 160-13**). Begin scanning by slowly lowering the handle of the US transducer toward the floor. Identify the uterine fundus and the endometrial stripe. Continue to scan until completely through the uterus. Slowly lift the US transducer handle toward the ceiling to scan the entire length of the uterus, the cervix, and the posterior cul-de-sac.



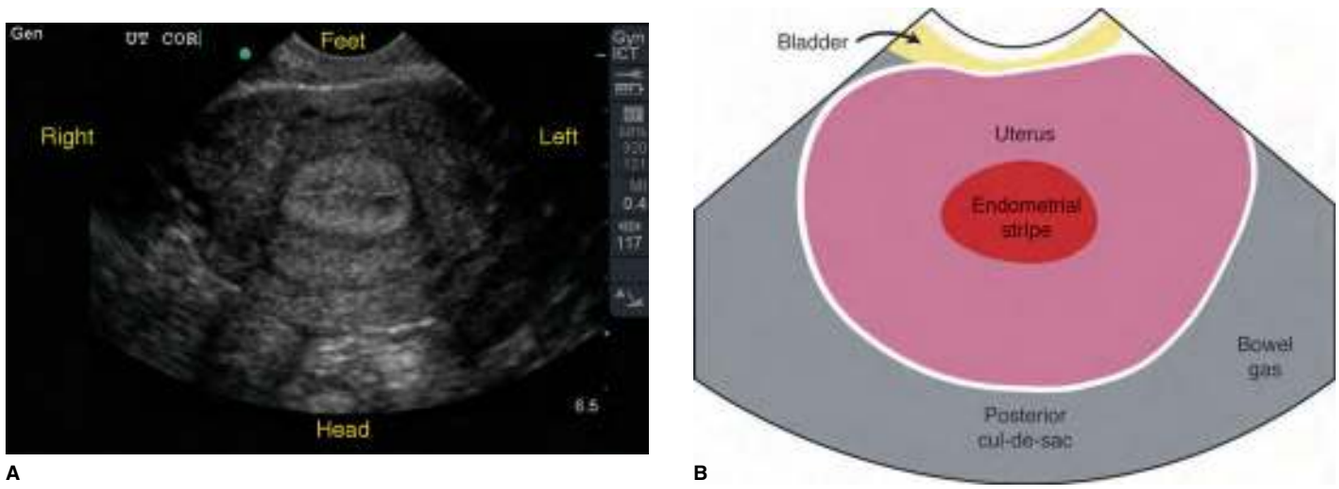
A B

FIGURE 160-11. Sagittal view of a nonpregnant uterus by TVS. ***A.*** The US image. ***B.*** Corresponding anatomic diagram. (Courtesy of Stephen Leech, MD.)



A B

FIGURE 160-12. Transverse view of a nonpregnant uterus by TVS. ***A.*** Transducer positioning. The arrow indicates the direction of the indicator. ***B.*** The US beam is directed through the anatomic structures in the transverse plane. (Courtesy of Stephen Leech, MD.)



A B

FIGURE 160-13. Transverse view of a nonpregnant uterus by TVS. ***A.*** The US image. ***B.*** Corresponding anatomic diagram. (Courtesy of Stephen Leech, MD.)

TVS OF THE OVARIES IN THE TRANSVERSE PLANE

The ovaries are best visualized in the transverse plane while scanning the adnexal regions transvaginally. Keep the uterus to one side of the image and scan up and down on each side of the uterus to locate the ovaries. The ovaries appear hypoechoic to the uterine myometrium with a mid-to-low gray echogenicity. The periphery of the normal ovary represents the tunica and is hypoechoic. The stroma is visualized as a low-gray echogenic center. Ovarian follicles in the periphery are small, round, and anechoic structures that vary in size and number. The sonographic appearance, size, and location of the ovaries vary greatly depending on the patient's age, menstrual phase, and pregnancy status. Identify iliac blood vessels. The ovaries are usually located lateral to the uterus and anteromedial to the iliac vessels (**Figure 160-14**). When the ovary is identified, scan through the margins by moving the US transducer handle up and down as necessary.

Rotate the US transducer 90° and sweep the beam anterior to posterior to visualize the ovaries and obtain the longest dimension of the ovary. The iliac vessel can serve as landmark to obtain the longest length of the ovary. Measure the size of the ovary by obtaining the three longest dimensions of the ovary (i.e., width, length, and height) in two orthogonal planes. Note any abnormalities of the ovary. The ovaries may not be identifiable in the presence of a large leiomyoma, in prepubertal females, and in postmenopausal females. The fallopian tubes are not routinely visible on US unless filled with fluid, filled with pus, or outlined by free intraperitoneal fluid.

A

B

FIGURE 160-14. Transverse view of a normal ovary by TVS. ***A.*** The ovary is seen medial to the iliac vessel. ***B.*** Follicles are seen in the periphery of the ovary (arrows).

TVS OF THE ADNEXA IN THE TRANSVERSE PLANE

After scanning the uterus and ovaries, continue the transverse scanning through the left and right adnexal regions. Identify the uterine fundus. Sweep the US transducer handle toward the patient's left thigh to visualize the right adnexa. Slowly move the US transducer handle from the floor toward the ceiling to sweep through the entire right adnexal area. Return the US transducer to the midline and reidentify the uterine fundus. Sweep the transducer handle toward the patient's right thigh to visualize the left adnexa. Survey the adnexal regions for any masses or dilated tubular structures. If an abnormality is found in the adnexa, note its size, sonographic appearance, and relationship to the ovaries and uterus. Doppler mode may be useful to evaluate any adnexal abnormalities and to help determine if the structure is vascular or if there is a heartbeat.

Survey the anterior and posterior cul-de-sacs thoroughly for free fluid, hematomas, or masses in the sagittal and longitudinal planes. Note the echogenicity of the fluid. If a mass is visualized, scan it in two orthogonal planes. Note its size, shape, location, sonographic appearance, and relationship to the ovaries and uterus. TVS is very helpful to differentiate a suspected mass from fluid and fecal material within the normal rectosigmoid colon. Distinguish bowel loops from other structures. Posterior cul-de-sac fluid can be quantified into small, moderate, and large in volume.[14] Free fluid seen less than one-third of the way up the posterior wall of the uterus is classified as small (**Figure 160-15**). Free fluid seen two-thirds of the way up the posterior wall of the uterus is classified as moderate (**Figure 160-16**). Free fluid seen over two-thirds of the way up the posterior wall of the uterus is classified as large (**Figure 160-17**).

FIRST-TRIMESTER ULTRASOUND

Approximately 70% of first-trimester patients seen in the ED will have an IUP visualized with US.[15] **First determine if the patient has an IUP when evaluating a patient with pain or bleeding in the first trimester of pregnancy.** A high-frequency 12-3 MHz linear transducer can be used for transabdominal sonography to identify an IUP after a failed examination with a low-frequency 5-2 MHz curvilinear transducer. It provides greater resolution of smaller structures and may identify a yolk sac or embryo not detected with

FIGURE 160-15. A small amount of free fluid in pelvis (arrows).

FIGURE 160-16. A moderate amount of free fluid in pelvis (arrows).

a curvilinear transducer. Determine the number of fetuses, their viability, and the gestational age if an IUP is visualized. **If an IUP is not visualized, scan the pelvis thoroughly and look for any signs of an ectopic pregnancy.** The introduction of fertility treatments has increased the incidence of heterotopic pregnancy (i.e., simultaneous intrauterine and ectopic pregnancy) and multiple intrauterine gestations.[16] TVS is generally not performed if an IUP is found on TAS and the patient is not undergoing any fertility treatment. **Perform TVS if an IUP cannot be confirmed using TAS or evaluate other causes of pelvic pain including ovarian torsion.**

NORMAL IUP

Early gestational structures are generally visualized 7 to 10 days earlier using TVS compared to TAS. **Systematically look for the sonographic findings of an IUP as discussed in the following sections.**

THE GESTATIONAL SAC

A gestational sac is an anechoic fluid collection within the uterus surrounded by a ring of bright, thick, and symmetric echogenic tissue (**Figure 160-18**). It is clearly visible at 5 weeks of gestation by TVS. The gestational sac is approximately 5 mm in diameter at 5 weeks and grows at a rate of 1 mm/day. **A gestational sac is not a definite sign of an IUP.** Determine the number and location of the gestational sac(s). Evaluate the gestational sac(s) in two orthogonal planes. Note the outline, shape, and contents of the gestational sac(s). The normal gestational sac is a round, centrally located, smooth-walled structure (**Figure 160-18**). Suspect a hematoma formation if the structure is irregular in outline and collapsing. The echogenic tissue surrounding the gestational sac represents developing chorionic villi and the adjacent endometrium. It should be thicker and echogenically brighter than the myometrium.

The size of the gestational sac can be used for gestational dating. The most commonly used method is the mean sac diameter (MSD) method. Obtain the longest three measurements in millimeters of the length, width, and height of the gestational sac in two orthogonal planes. Do not include the echogenic rim of trophoblastic and decidual tissue surrounding the gestational sac. Obtain the measurements from the inner margin to the inner margin of the gestational sac. The MSD is calculated by the following equation: MSD = (length + width + height) ÷ 3. The gestational age is calculated by the following equation: gestational age (days) = MSD (mm) + 30.

FIGURE 160-17. A large amount of free fluid in pelvis (arrows).

THE DOUBLE DECIDUAL SIGN

Two concentric echogenic rings surrounding a gestational sac form the double decidual sign (**Figure 160-19**).[17,18] The outer bright ring is called the decidua vera and is formed by the endometrial lining of the uterus (i.e., the decidua). The inner echogenic ring is called decidua capsularis and is formed by the chorion surrounding the gestational sac. The two bright layers are separated by a hypoechoic or anechoic layer of fluid which is the endometrial canal. **The double decidual sign is highly suggestive of an IUP but is not 100% reliable.[19] It occurs in only about half of all intrauterine pregnancies.**

THE YOLK SAC

The yolk sac is the first embryonic structure seen within the gestational sac (**Figures 160-18 and 160-19**). **It is the earliest reliable US sign of an IUP. The presence of a yolk sac within the gestational sac in the uterus is diagnostic for an IUP.** The normal yolk sac is round with an anechoic fluid-filled center and measures less than 7 mm in diameter. The yolk sac is visualized as a bright, spherical, thin-walled structure within the gestational sac with a balloon on a string appearance (**Figures 160-18 and 160-19**). It is visible when the gestational sac is > 8 mm by TVS and > 20 mm by TAS. The yolk sac is usually visible 5 to 6 weeks after the last menstrual period by TVS and at 7 weeks by TAS. Measure the yolk sac from its inner margin to its opposite inner margin. It generally disappears after 10 to 12 weeks but can persist until 20 weeks.

THE EMBRYO

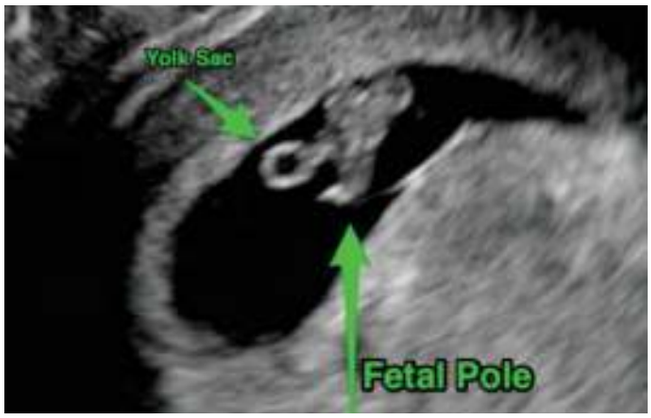
The embryonic or fetal pole is visible at 5 to 6 gestational weeks by TVS. It is approximately 1 to 2 mm in length at 5 to 6 weeks of gestation. The normal embryo grows at a rate of 1 mm/day. It appears as a highly echogenic focal thickening adjacent to the yolk sac, hugging the wall of the gestational sac surrounded by anechoic fluid (**Figure 160-18*A***). The embryo is visible within the gestational sac when the MSD is between 5 and 12 mm by TVS. **The presence of a fetal pole within the gestational sac in the uterus is diagnostic for an IUP.** Measure the fetal pole along its longest axis.

FETAL CARDIAC ACTIVITY

Fetal cardiac activity is visible by 6.5 weeks of gestation and a fetal pole size of 6 mm by TVS. Fetal cardiac activity is generally visible by TAS in embryos > 10 mm long. A faint fluttering motion within the fetal pole adjacent to the yolk sac represents the neurologically

A

B



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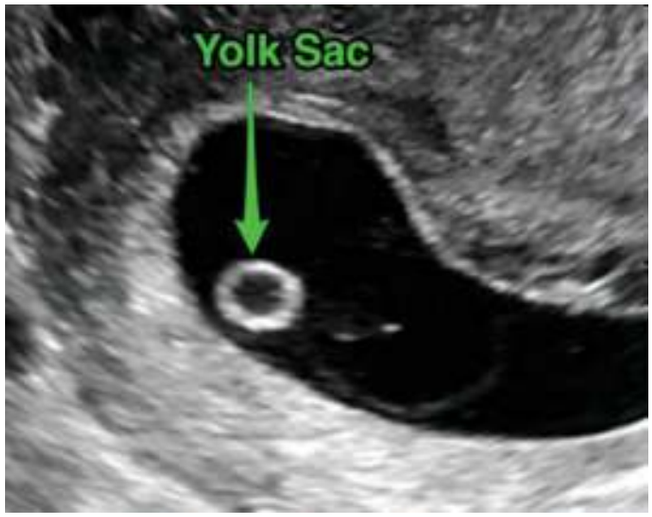


D

FIGURE 160-18. Sagittal view of a uterus in early pregnancy. ***A.*** TVS. ***B.*** TAS. ***C.*** Normal early IUP demonstrating the yolk sac and fetal pole. (Photo used with permission from www.aliem.com.) ***D.*** Close-up view of normal IUP demonstrating the yolk sac within the gestational sac. (Photo used with permission from www.aliem.com.)

active heart tissue. **The presence of fetal cardiac activity inside the uterus is diagnostic for a live IUP.** The incidence of a miscarriage, or spontaneous abortion, before 20 weeks of gestation with a live IUP diagnosed on US in the ED is 9.2%.[20] Use M-mode to determine the fetal heart rate (FHR). It is calculated by measuring the length of one cycle and then determining the cycles per second from that measurement (**Figure 160-20**). The normal FHR for an embryo > 6 mm in length is over 120 beats/min. FHR serves as an important prognostic indicator. Rates less than 90 beats/min are associated with a high risk of spontaneous abortion.

A

B

FIGURE 160-19. Sagittal view of an early pregnancy by TVS. ***A.*** Double decidual sign. ***B.*** A gestational sac (yellow arrows) contains a yolk sac (red arrows) and a fetal pole (FP) within the endometrium (green arrows). (Photo used with permission from www.aliem.com.)

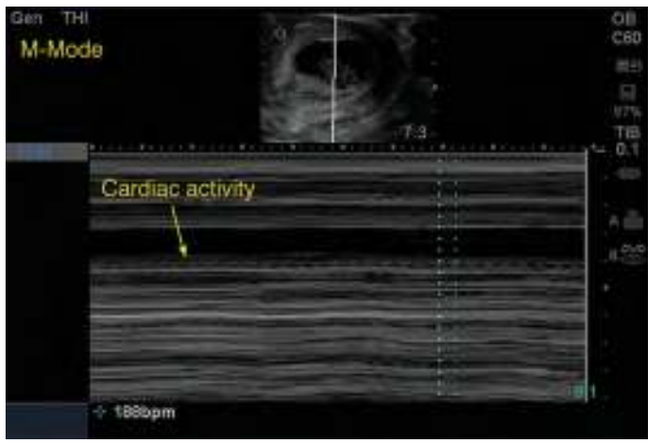


FIGURE 160-20. Fetal heart rate measurement using M-mode.

ESTIMATING FETAL AGE

The crown-rump length (CRL) is the most accurate sonographic measurement for gestational dating between 7 and 10 weeks.[21] It is accurate within ±3 days for gestational dating. The embryonic pole length is used for the CRL measurement. By 8 weeks, a true CRL measurement can be determined. Obtain the longest measurement of an unflexed fetus and do not include the yolk sac (**Figure 160-21*A***). The gestational age is calculated by the following equation when CRL is < 25 mm: gestational age (days) = CRL (mm) + 42.

The CRL cannot be seen and measured accurately during some US scans. The biparietal diameter (**Figure 106-21*B***) and/or femur length (**Figure 160-21*C***) can be measured. Measure the biparietal diameter perpendicular to the central fissure at the level of the thalamic nuclei. Measure the femur length from the two blunted ends. Do not include the extension to the greater trochanter and the femoral head. The measurement is considered inaccurate when the femur image is at an angle of over 30° to the horizontal. Compare these values to standardized tables to determine the fetal age. Some US machines have these tables built in. A click of a button or two is all it takes.

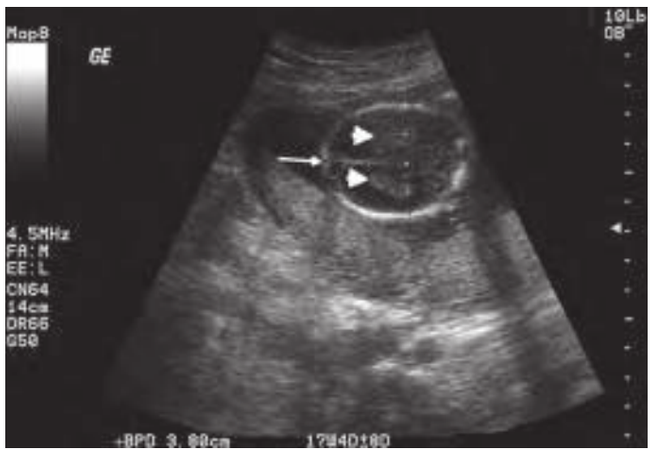
CORPUS LUTEAL CYST

The corpus luteum is a normal physiologic structure found within one of the ovaries during pregnancy. It is unilocular and thin-walled. The sonographic appearance of a corpus luteum is highly variable. It can appear as a hypoechoic, hyperechoic, or isoechoic structure. Power Doppler mode shows the corpus luteum has increased blood flow at the periphery that is described as a "ring of fire" (**Figure 160-22**). The ring of fire is visible with an ectopic pregnancy.

EMBRYONIC DEMISE OR EARLY PREGNANCY FAILURE

Definitive diagnosis of an early pregnancy failure requires serial US examinations. A blighted ovum is a failed pregnancy in which the embryo failed to develop within a gestational sac of sufficient size that an embryo should be visible. There are several sonographic findings that can suggest early pregnancy failure. The earliest sign of early pregnancy failure is a gestational sac without a yolk sac or embryo. An empty gestational sac with a MSD ≥ 25 mm and no evidence of an embryo or yolk sac by TVS is diagnostic of pregnancy failure (**Figure 160-23*A***).[22] A CRL of ≥ 7 mm without a fetal heart rate is also diagnostic of pregnancy failure (**Figure 160-24**). Other findings that are suggestive but not diagnostic of embryonic demise include a MSD between 16 and 24 mm by TVS without an embryo, a CRL of < 7 mm by TVS without a fetal heartbeat, an enlarged yolk sac (i.e., > 7 mm), and a small gestational sac in relation to the size of the embryo (i.e., < 5 mm difference between mean sac diameter and CRL).[22]

A



B

C

FIGURE 160-21. Determining the age of an embryo. ***A.*** Crown-rump length. Maximal embryo length is measured (dotted line) and excludes the yolk sac. ***B.*** Biparietal diameter measured perpendicular to the central fissure (arrow) and through the thalamic nuclei (arrowheads). (Photos used with permission from reference 4.) ***C.*** Femur length measurement (between +'s).

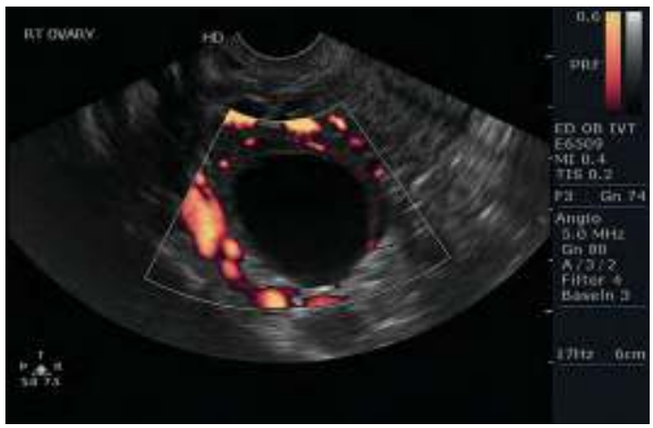
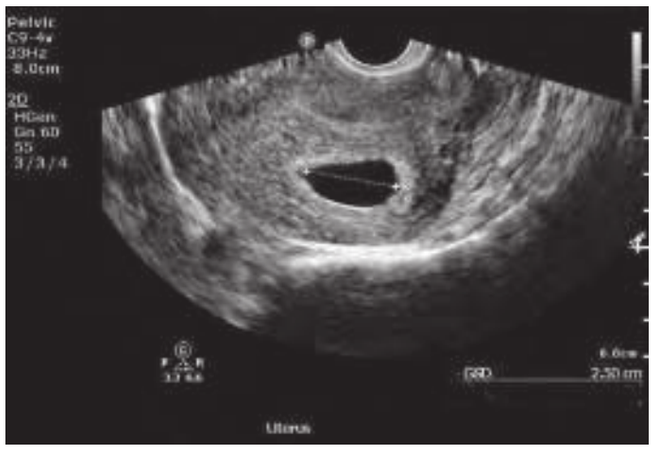


FIGURE 160-22. Corpus luteum cyst with the ring of fire visualized on power Doppler mode.

Additional signs suggestive of early pregnancy failure include a gestational sac that is irregular in shape, distorted, or collapsed (**Figure 160-23*B***). A gestational sac positioned low in the uterine cavity or surrounded by a thin (i.e., < 2 mm) wide decidual reaction indicates embryonic demise. Absence of a yolk sac when an embryo is visualized prior to 12 weeks is indicative of embryonic demise. A calcified yolk sac is associated with an adverse pregnancy outcome.



A

B

FIGURE 160-23. Sagittal view of an early pregnancy failure by TVS. ***A.*** The gestational sac is 25 mm in diameter (dotted line) without a visible yolk sac or embryo. ***B.*** An irregular gestational sac.

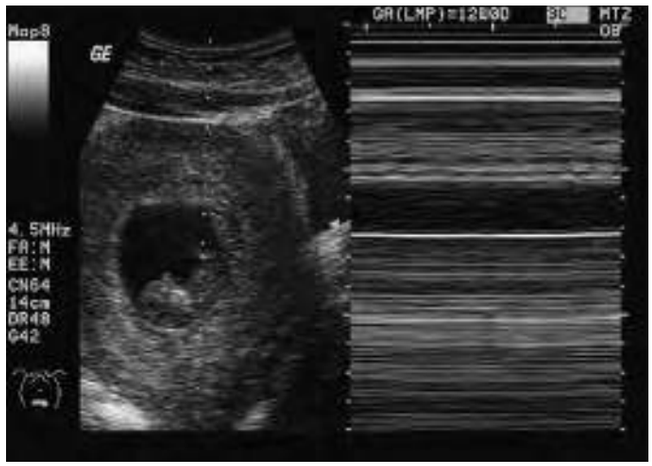


FIGURE 160-24. Embryonic demise. Note the absence of fetal cardiac activity on M-mode. (Courtesy of Sam Hsu, MD).

Embryonic bradycardia (i.e., < 90 beats/min) is associated with a poor prognosis.

SPONTANEOUS ABORTION

A spontaneous abortion is the most common complication of first-trimester pregnancy and occurs in approximately 20% of clinically apparent pregnancies.[23] An empty uterus with a clear midline echo occurs on US after a complete spontaneous abortion. An incomplete abortion is visible on US as irregular echogenic material within the uterine cavity (**Figure 160-25*A***) or a thickened midline stripe ≥ 10 mm wide within the uterine cavity (**Figure 160-25*B***). These both represent retained products of conception.

SUBCHORIONIC HEMORRHAGE

A subchorionic hemorrhage is bleeding between the chorion and the uterine endometrium. It can cause sudden pelvic pain or vaginal bleeding. The overall incidence in pregnancy is 1.3%. It results in the chorionic membrane separating and elevating from the decidua vera (i.e., endometrium) by a hematoma or clot. The acute hemorrhage appears hyperechoic or isoechoic. The hematoma becomes hypoechoic or anechoic as it undergoes liquefaction during the subsequent 1 to 2 weeks (**Figure 160-26**).

There is an increased risk of miscarriage, stillbirth, abruption, preterm premature rupture of membranes, and preterm labor in patients with a subchorionic hemorrhage. In a retrospective study done by Bennet et al, the overall pregnancy loss rate was 9.3%.[24] A large meta-analysis demonstrated that the odds ratio of spontaneous abortion in pregnant women found to have a subchorionic hematoma was 2.18.[25] This was found to increase with increasing maternal age and decreasing gestational age. The prognosis depends upon the size of the subchorionic hemorrhage. Small and medium-sized hemorrhages, up to approximately 50% of the gestational sac circumference, have a spontaneous abortion rate of 9%. Hemorrhages larger than 50% of the gestational sac circumference have a spontaneous abortion rate of 18.8%. Hemorrhages > 40% of the volume of the gestational sac are associated with spontaneous abortion rates of 50%.

ECTOPIC PREGNANCY

The prevalence of ectopic pregnancy among patients presenting to the ED with bleeding, pain, or both during first trimester is 6% to 16%.[26] **Clinical criteria alone are not sufficient to distinguish**

A

B

FIGURE 160-25. Sagittal view of an incomplete spontaneous abortion by TVS. ***A.*** Irregular echogenic material in the endometrial canal without evidence of a fetus. ***B.*** Thickened endometrial stripe (dotted line) without evidence of a fetus.

between a patient having a spontaneous abortion and an ectopic pregnancy. Pelvic US is the diagnostic test of choice in the evaluation of a patient with a possible ectopic pregnancy. The risk of heterotopic pregnancy in the general population is 1 in 4000 to 1 in 30,000 pregnancies. The risk increases to 1% in women undergoing in vitro fertilization.[23,27,28]

The discriminatory zone is the β-human chorionic gonadotropin (β-hCG) level at which an IUP should be seen by pelvic US. This level generally is between 1000 and 2000 mIU/mL by TVS and between 4000 and 6500 mIU/mL by TAS. Approximately 38% of patients with a confirmed ectopic pregnancy had a β-hCG level < 1000 mIU/mL. Approximately 39% of ectopic pregnancies with β-hCG levels less than 1000 mIU/mL can be identified on US.[29] The concept of a discriminatory zone becomes more significant if the US is indeterminate.

There is no β-hCG level that rules out an ectopic pregnancy in a pregnant patient. Correlate the serum β-hCG values with the US findings. Have a high suspicion for an ectopic pregnancy if the US findings are indeterminate and the serum β-hCG levels are above the discriminatory zone. Any pregnancy in which there is a detectable β-hCG value and no visualized IUP on US is considered a pregnancy of unknown location (PUL).

FIGURE 160-26. Sagittal (left) and transverse (right) views of a pregnant uterus by TAS showing a subchorionic hemorrhage (SCH). (Courtesy of Sam Hsu, MD.)

Approximately 95% of ectopic pregnancies occur in the fallopian tube. This is split into 80% in the ampulla, 10% to 15% in the isthmus, and < 2% in the interstitial region. The remaining 5% are found in the cervix, on the ovary, and in the abdomen. Approximately 31% of pregnant patients presenting to the ED have no definite signs of either an IUP or an ectopic pregnancy on US. The diagnosis of an ectopic pregnancy is often reached through a combination of abnormal US findings. The spectrum of sonographic findings of an ectopic pregnancy is broad and includes an empty uterus, a pseudogestational sac, an adnexal mass, an extrauterine empty gestational sac, an extrauterine gestational sac with a yolk sac, an embryo with or without cardiac activity, a tubal ring, and fluid in the cul-de-sac.

EMPTY UTERUS

An ectopic pregnancy should be suspected in any pregnant patient presenting with abdominal pain or vaginal bleeding. Perform TVS to determine if an IUP is present. The empty uterus is the lack of an IUP in a pregnant patient. This is referred to as a PUL. **An empty uterus is a sign of an ectopic pregnancy unless proven otherwise.**

PSEUDOGESTATIONAL SAC

A pseudogestational sac is the decidualization of the endometrium from hormonal stimulation by the ectopic pregnancy. It occurs in 10% to 20% of ectopic pregnancies. The pseudogestational sac is often < 10 mm in diameter and consists of endometrial fluid. The pseudogestational sac is distinguished from a normal gestational sac by its central location in the endometrial cavity, lack of a double decidual sign, an ovoid shape, and poorly defined margins (**Figure 160-27**). A normal gestational sac is eccentrically placed within the endometrial cavity.

EXTRAUTERINE GESTATIONAL SAC

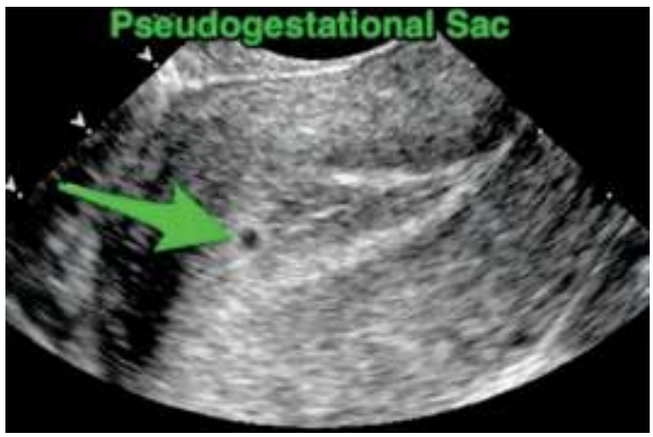
An intact gestational sac with a yolk sac outside the uterus confirms an ectopic pregnancy (Figure 160-28). The most specific finding of an ectopic pregnancy is the presence of a live extrauterine pregnancy. This occurs in 3% to 26% of ectopic pregnancies and is characterized by fetal cardiac activity outside of the uterus.[30]

ADNEXAL MASSES

Adnexal abnormalities occur in approximately 70% to 90% of ectopic pregnancies. **The sonographic appearances of these abnormalities are variable, often only suggestive, and not**

A

FIGURE 160-29. An adnexal mass adjacent to the ovary by TVS. (Courtesy of Michael Blaivas, MD.)



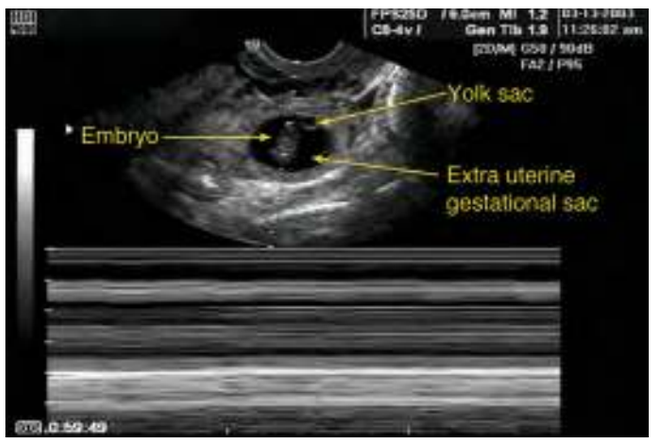
B

FIGURE 160-27. Examples of a pseudogestational sac. ***A.*** Sagittal view of a pseudogestational sac (arrows) by TVS. ***B.**** Empty fluid-filled sac within the endometrium. (Photo used with permission from www.aliem.com.)

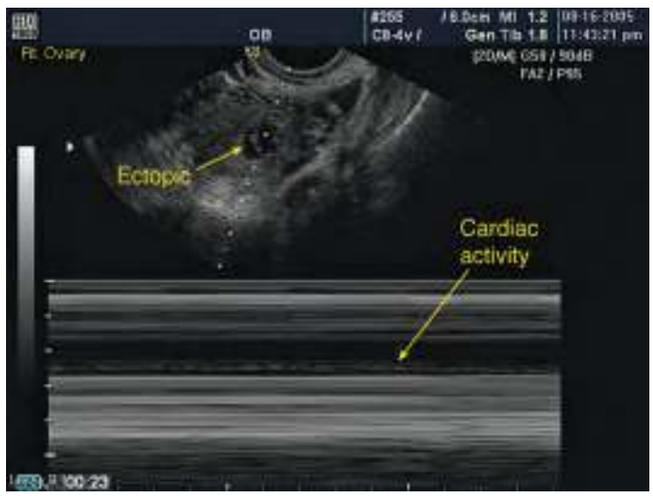
diagnostic of an ectopic pregnancy. Abnormalities are frequently visible as spherical structures with increased blood flow on power Doppler, such as the ring of fire seen with a normal corpus luteum. The most common finding is a small noncystic mass with mixed echoes adjacent to the ovary (**Figure 160-29**). The corpus luteum is located on the same side as the ectopic pregnancy in 80% of cases (**Figure 160-30**). Adnexal masses next to the ovary can be difficult to distinguish from a normal corpus luteum. Apply pressure with the endocavitary US transducer to help separate the ectopic mass from the ovary. The Emergency Physician's hand can be used to palpate the abdomen and apply additional pressure to separate an adnexal mass from the ovary for improved visualization.[31]

TUBAL RING

The tubal ring is an extrauterine hypoechoic concentric mass with a thick-walled echogenic rim and separate from the ovary (**Figures 160-31 and 160-32**). It represents a gestational sac and the surrounding trophoblastic reaction. It is found in 68% to 78% of ectopic pregnancies. The presence of a tubal ring has a 95% positive predictive value for an ectopic pregnancy.



A



B

FIGURE 160-28. Ectopic pregnancy. ***A.*** An extrauterine gestational sac containing a yolk sac and an embryo. ***B.*** Cardiac activity is identified on M-mode. (Courtesy of Michael Blaivas, MD.)

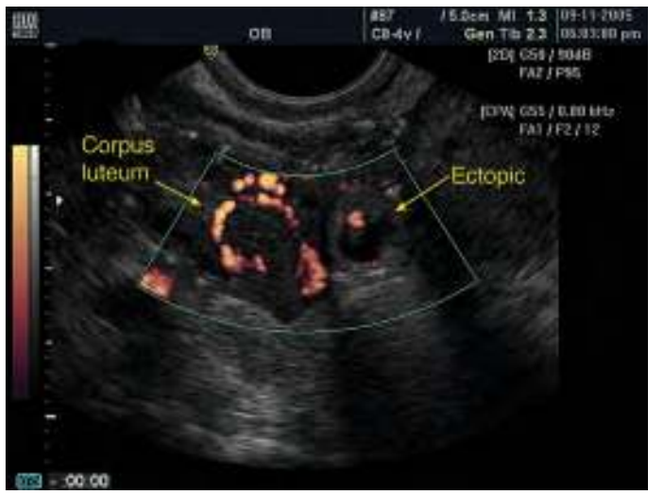


FIGURE 160-30. An ectopic pregnancy adjacent to a corpus luteum with its ring of fire by TVS. (Courtesy of Michael Blaivas, MD.)

A

B

FIGURE 160-31. The tubal ring representing an unruptured ectopic pregnancy adjacent to the ovary by TVS. ***A.*** The tubal ring. ***B.*** The tubal ring containing an embryo. (Courtesy of Michael Blaivas, MD.)

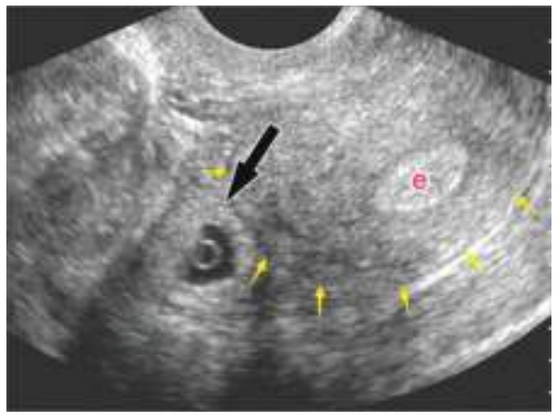


FIGURE 160-32. Transverse view of an ectopic pregnancy by TVS. The yolk sac is visible within a gestational sac (black arrow) and outside the uterus (yellow arrows) with an empty endometrium (E). (Photo used with permission from www.aliem.com.)

FREE FLUID IN THE CUL-DE-SAC

A hemoperitoneum is found in approximately 40% to 83% of patients with a complicated ectopic pregnancy. This has a 90% positive predictive value for an ectopic pregnancy. **Anything more than a small amount of pelvic fluid is abnormal. Free fluid in the cul-de-sac and an empty uterus on TVS is an ectopic pregnancy until proven otherwise (Figure 160-33).** The greater the quantity of free fluid in the pelvis, the higher is the likelihood of an ectopic pregnancy. As the amount of pelvic fluid increases, it spreads from the posterior aspect of the cervix to the posterior aspect of the uterus then to above the fundus or around the ovaries. The type of free fluid in the pelvis determines the risk of an ectopic pregnancy. The risk is dependent on the quantity of the free fluid if the free fluid is echo free. **Any amount of echogenic pelvic fluid or complex free fluid represents blood and a high risk for the possibility of an ectopic pregnancy. Hyperechoic masses floating within the echogenic pelvic fluid suggest clotted blood.**

Free fluid in the pelvis occurs frequently with a ruptured ectopic pregnancy. Free fluid may be found with an unruptured ectopic pregnancy. The likelihood of a ruptured ectopic pregnancy increases as the amount of free fluid increases. Clotted blood in the cul-de-sac after a ruptured ectopic pregnancy can distort anatomic landmarks and obscure the US image. Scanning through the hepatorenal space can often lead to identification of an active

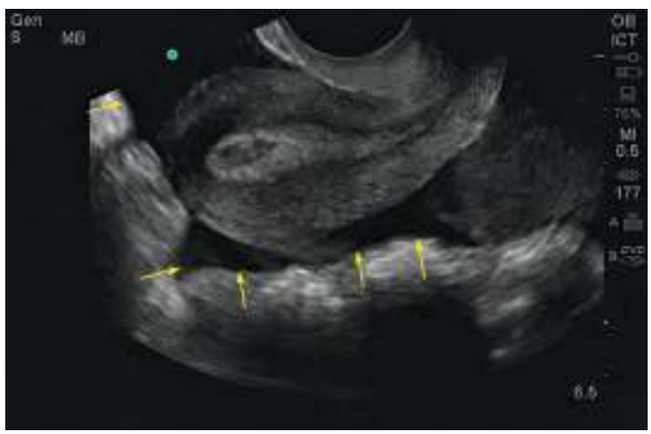


FIGURE 160-33. Sagittal view of a ruptured ectopic pregnancy. The uterus is empty with a large amount of free fluid (arrows) in the pelvis.

hemorrhage. The presence of free fluid in the Morison's pouch of a first-trimester patient with a PUL is highly suggestive of a ruptured ectopic pregnancy.

ASSESSMENT

Perform a complete ED US examination. Classify the US findings into one of three categories: IUP, ectopic pregnancy, or PUL. **Determine the patient disposition based upon their symptoms, the US results, and the β-hCG level.**

Patient disposition is based upon their vital signs, physical examination findings, and bleeding status when the definitive diagnosis of an IUP is made and the patient did not undergo any fertility treatments. If the patient is hemodynamically stable with no significant abdominal tenderness and is not actively bleeding, discuss the risks and symptoms of a spontaneous abortion and instruct the patient to follow-up with an Obstetrician. Consider other causes of abdominal pain (e.g., appendicitis, cholelithiasis, ovarian torsion, and renal colic) if the patient has severe abdominal pain. A complete spontaneous abortion can be managed expectantly with follow-up by an Obstetrician. An incomplete abortion requires an Obstetrician consultation for possible dilatation and curettage. Consult an Obstetrician if there are definite signs of an ectopic pregnancy on the US. The Obstetrician will determine the need for chemical or surgical treatment.

An indeterminate US study demonstrates no signs of an IUP or an ectopic pregnancy and is considered a PUL. Approximately 20% of all ED first-trimester pelvic US examinations are categorized as indeterminate.[32] Approximately 20% of patients with an indeterminate US study and β-hCG level > 1000 mIU/mL are found to have an ectopic pregnancy.[33,34] The possible outcomes with indeterminate first-trimester US studies are early pregnancy, ectopic pregnancy, missed abortion, or incomplete abortion. The patient can be safely discharged with follow-up in 48 hours for reevaluation if they have a PUL and are hemodynamically stable with no significant abdominal or adnexal tenderness, no free fluid on US, and a β-hCG level < 1000 mIU/mL.

AFTERCARE

There is no specific aftercare for the patient based on the US examination itself. All disposition and management decisions are described in the assessment section above. Dispose of the condom or transducer sheath after completing the transvaginal US examination. Soak the endocavity US transducer in an antimicrobial solution. Follow the manufacturer's specifications and the hospital's Infectious Disease Department recommendation for the type of antimicrobial solution to be used and the soak time.

COMPLICATIONS

There are no complications to performing a pelvic US examination during early pregnancy except for mild discomfort during the procedure. US examination of the fetus is generally considered to be safe during pregnancy. Prior studies have reported no association of US with any adverse fetal outcomes. There is a theoretic risk of thermal injury to a fetus with US. Do not perform a Doppler examination in early pregnancy without clinical indications.

SUMMARY

Women with abdominal pain and bleeding during the first trimester of pregnancy are commonly seen in the ED. Pelvic US is the primary imaging modality used in the evaluation of first-trimester symptoms. ED pelvic US has revolutionized the assessment of these patients. The goals of the first-trimester US are to find an IUP and rule out an ectopic pregnancy. Knowledge of sonographic anatomy, technique, and limitations is essential to perform this procedure. Understanding the principles discussed in this chapter and following a systematic approach to performing a pelvic US examination will maximize the amount of useful information obtained in first-trimester patients.

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161 Uterine Bleeding

Leah W. Antoniewicz, Beth R. Davis, Kara N. Purdy, Alexis R. Taylor, Lindsay K. Grubish, and Sarah Christian-Kopp

INTRODUCTION

Uterine bleeding may occur in the absence of current or recent pregnancy, during pregnancy, or in the postpartum period.[1] Abnormal uterine bleeding (AUB) is a term that defines menstrual bleeding that occurs on an abnormal schedule, for an abnormal duration of time, or with an abnormal quantity of blood loss.[2-6] The term dysfunctional uterine bleeding is no longer used.[7,8] Dysfunctional uterine bleeding is a general term that was used to describe irregular bleeding in the nonpregnant patient. AUB can manifest as heavy menstrual bleeding (HMB). HMB is cyclical or ovulatory bleeding and was previously termed menorrhagia. Intermenstrual bleeding (IMB) refers to bleeding that occurs between menses. This was previously termed metrorrhagia. IMB often has a cervical etiology. Approximately 3% of women have physiologic IMB associated with ovulation.[9] Approximately 66% of women who have had one or more cesarean births have a defect in the uterus. This is reported as regular postmenstrual spotting.[10] Irregular AUB is caused by ovulatory dysfunction (AUB-O) and is often reported as no cycle for 2 to 3 months. When cycles occur, they are often heavy.

A normal menstrual cycle frequency is 21 to 35 days with a duration of 5 to 7 bleeding days. The International Federation of Gynecology and Obstetrics (FIGO) created the PALM-COEIN classification system in 2011 to help categorize bleeding abnormalities based on the underlying etiology and the bleeding pattern.[11,12] The acronym divides the causes into structural (PALM) and nonstructural (COIEN). The most common causes of AUB in women of reproductive age include uterine pathology or the "PALM" portion of the classification system.

This chapter will focus on nonpregnant reproductive-age women. Postpartum uterine bleeding or postpartum hemorrhage is discussed in Chapter 166. Perimenopausal bleeding is generally secondary to hormonal imbalances. Postmenopausal bleeding is a concern for benign etiologies (e.g., fibroids) and malignant etiologies (e.g., cancers). **Postmenopausal bleeding is considered cancer until proven otherwise.** An endometrial biopsy and transvaginal ultrasound (US) should be performed if a uterine source for bleeding is suspected in a postmenopausal woman. Lower genital tract atrophy is the most common benign cause of postmenopausal bleeding and is easily treated with estrogen cream. **All postmenopausal bleeding requires follow-up with a Gynecologist.**

The most important interventions in AUB are to obtain hemostasis and provide volume repletion if needed (e.g., intravenous fluids or blood products). It is essential to determine if the bleeding is occurring in the presence or absence of a pregnancy, the source of the bleeding, and the most appropriate method for hemostasis.

Acute and more conservative management measures (e.g., conjugated equine estrogen, oral contraceptives, medroxyprogesterone acetate, minor surgical interventions, and tranexamic acid) are warranted when uterine bleeding occurs in a stable patient (**Figure 161-1**). Stabilization with more invasive measures is warranted when hypovolemia and hemodynamic instability exist (**Figure 161-1**).

The evaluation and intervention will largely be guided by the patient's hemodynamics and should always start with a pregnancy test. Stable uterine bleeding or continuous bleeding in a stable patient can be comfortably managed as an outpatient. Acute blood loss anemia, hemodynamic instability, massive hemorrhage, and symptomatic anemia warrant urgent Gynecology consultation and intervention. This chapter discusses medical interventions, fluid or blood resuscitation, medication administration, and bedside procedures to slow, stop, or treat the symptoms of acute hemorrhage.

ANATOMY AND PATHOPHYSIOLOGY

The uterus is a highly vascular structure and there are multiple sources of bleeding to consider. The uterus is a unique organ in that some form of bleeding is normal, and therefore, it is sometimes difficult to determine whether a true emergency condition exists. AUB as defined by FIGO is bleeding that is abnormal in duration, frequency, regularity, or volume in the absence of pregnancy.[12,13] The FIGO approved a new classification system in 2011. They named this using the acronym PALM-COEIN for causes of abnormal uterine bleeding in nonpregnant women of reproductive age. The acronym stands for **P**olyp, **A**denomyosis. **L**eiomyoma, **M**alignancy and hyperplasia, **C**oagulopathy, **O**varian dysfunction, **E**ndometrial etiologies, **I**atrogenic etiologies, and **N**ot yet classified.[7,12,14]

POLYPS (AUB-P)

Polyps are a common and usually benign etiology of AUB. This is especially true in women who are premenopausal or menopausal, although polyps can occur in younger women. Polyps are epithelial growths of the uterine endometrium. They primarily cause IMB. Studies estimate that intrauterine abnormalities account for almost 50% of patients presenting with AUB.[11] Endometrial polyps are one of the common causes of AUB in premenopausal and postmenopausal patients. Polyps are derived from hyperplastic overgrowth of endometrial glands and stroma usually from estrogen stimulation. Some risk factors for polyp formation include hormone replacement therapy, obesity, tamoxifen therapy, and unopposed estrogen therapy.

Common presenting symptoms include IMB, postcoital bleeding if the polyp is prolapsed, and postmenopausal bleeding. Polyp-related symptoms may be controlled with medical therapy in the

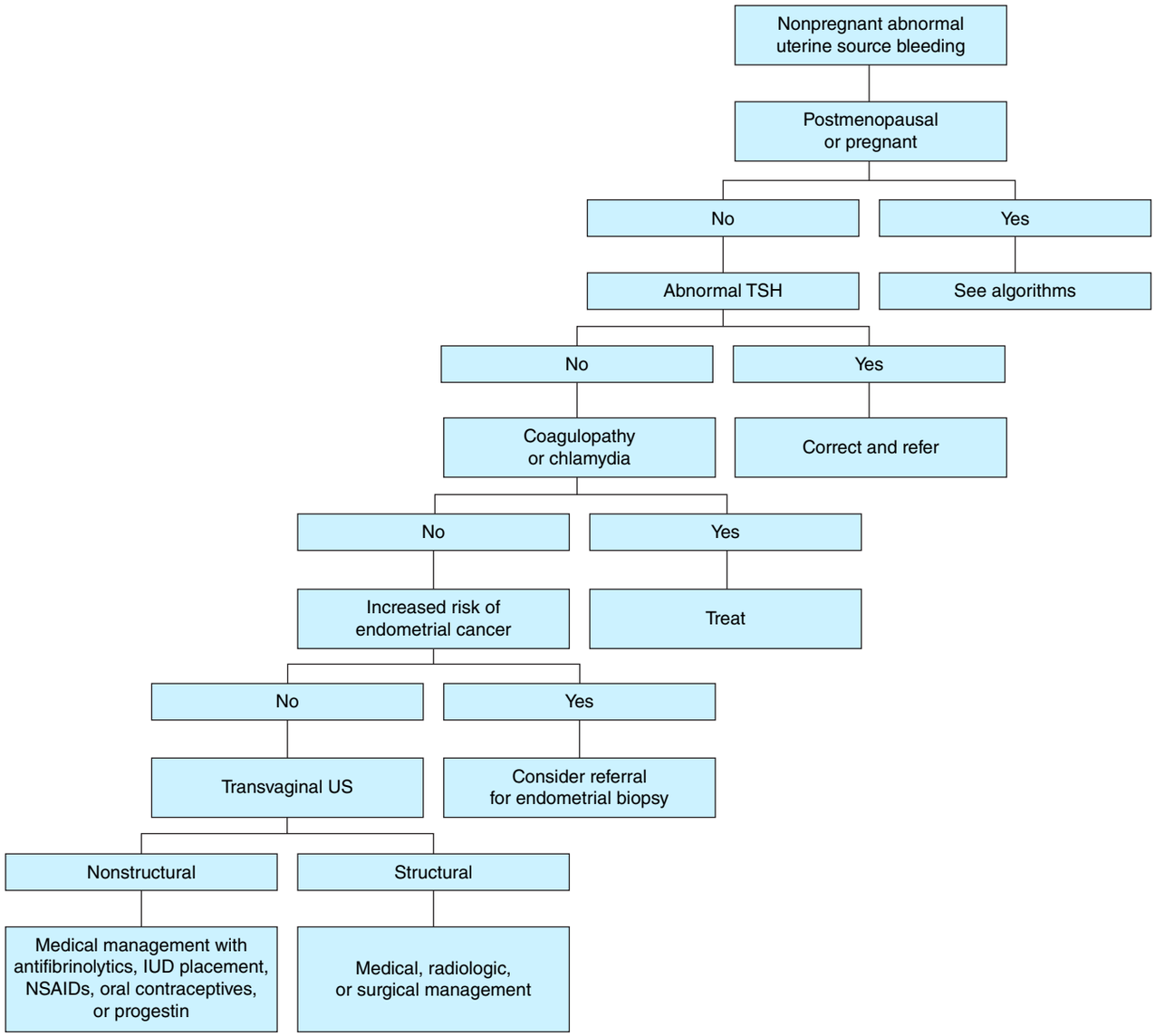

FIGURE 161-1. Algorithm for the management of AUB.

short-term. Transvaginal US may assist in the diagnosis if a thickened or heterogeneous endometrial stripe is noted. Referral to a Gynecologist for outpatient management is appropriate if there is concern for an underlying structural cause in the stabile patient. Sonohysterogram or hysteroscopy is the preferred imaging for suspected polyps. Definitive diagnosis is with histologic examination via endometrial biopsy or polypectomy. Definitive management is with hysteroscopic polypectomy.

ADENOMYOSIS (AUB-A)

Adenomyosis occurs when there is ectopic endometrial glands and stroma within the myometrium. The ectopic tissue is stimulated by endogenous hormones that induce hypertrophy of the myometrial tissue. This often results in a "globular" appearance radiographically and a palpable boggy texture of the uterus. The uterus may remain a normal size in some patients but adenomyomas, which appear and act like leiomyomas, may develop.

AUB and dysmenorrhea are the most common presenting symptoms that may worsen with age. Clinical symptoms and physical examination typically raise suspicion for adenomyosis. Transvaginal US and magnetic resonance imaging (MRI) may assist in the diagnosis with findings of asymmetric myometrium, myometrial cysts, and increased heterogeneity. Definitive diagnosis requires pathologic confirmation after hysterectomy. Medical management of symptoms is similar to that of leiomyomas (i.e., gonadotropin-releasing hormone [GnRH] analogues, nonsteroidal anti-inflammatory drugs [NSAIDs] for pain, and oral contraceptives). Definitive treatment is with a hysterectomy.

LEIOMYOMA (AUB-L)

Leiomyomas (i.e., fibroids) are benign tumors of muscle cell origin and are the most commonly occurring pelvic tumor.[15] Leiomyoma presents in nearly 70% of Caucasian women and 80% of African American women by age 50.[16] These numbers increase with age but decrease with menopause.[17] Leiomyomas are likely related to a combination of hormones and genetic factors, although the exact cause is unknown. Up to 30% of women with leiomyomas will have AUB and pelvic pain or pressure. Large leiomyomas may be palpable on examination.

Leiomyomas may be in multiple locations including intramural, pedunculated, submucosal, or subserosal. Symptoms typically vary based on size and location of the leiomyomas. Subserosal and pedunculated leiomyomas typically cause bulk symptoms (e.g., pelvic pain and pressure) whereas intramural and submucosal leiomyomas cause heavy menstruation. Patients who are asymptomatic do not require any intervention. Medical treatments for leiomyomas include medications (e.g., antifibrinolytic agents, aromatase inhibitors (currently experimental), GnRH agonists, and oral contraceptives).[15,18]

Oral contraceptives are typically the first-line treatment for AUB-L. Both combined and progesterone-only contraceptive pills have been shown to decrease bleeding secondary to leiomyomas without stimulating growth of the leiomyomas. The levonorgestrel intrauterine device (IUD; e.g., Mirena) has been effective in reducing the bleeding and cramping associated with leiomyomas and adenomyosis. There may be a higher risk of IUD expulsion depending on the location of the leiomyomas.

Leuprolide acetate (i.e., Lupron) is a GnRH agonist approved by the U.S. Food and Drug Administration (FDA) that can be used for the management of leiomyomas. Lupron effectively induces a menopausal state resulting in amenorrhea in most patients and a decrease in leiomyoma volume within 3 months of treatment.[16] Leuprolide acetate therapy is temporary because of long-term side effects from estrogen deprivation. Prescribe daily add-back therapy with 5 mg of norethindrone acetate to reduce side effects. Lupron is typically used to correct underlying anemia and shrink leiomyoma size in anticipation of a surgical intervention.

There are procedures that may be performed for symptomatic management with uterine preservation. One option that is becoming more readily available is uterine artery embolization (UAE) where an Interventional Radiologist uses gelatin microspheres to embolize the uterine artery via cannulation of the femoral artery. The resulting vascular occlusion results in a decrease of bulk symptoms.[16] The rates of complications are typically lower than those associated with surgical interventions and postprocedure recovery is faster. This treatment is not recommended for patients who desire future fertility.

MRI-guided focused US surgery (MRgFUS) is a noninvasive approach to managing leiomyomas. It uses high-intensity US waves directed into a leiomyoma that was localized with MRI. The US waves are focused to small areas of the leiomyoma to raise the temperature and cause cellular death. A water bath is used between the skin and the US transducer to maximize acoustic contact. The benefits of MRgFUS include an alternative to hormonal therapy, an alternative to surgery, few side effects, outpatient basis that does not require staying in the hospital, preservation of future fertility, relative noninvasiveness, treatment in approximately 3 hours, and return to work in a few days. Contraindications include extensive abdominal skin scarring, implanted devices (e.g., neurostimulators and pacemakers), inability to lie still for 3 hours, pregnancy, and poor health. This procedure is not available at all facilities. Long-term data regarding efficacy are currently lacking.

Endometrial ablation may be an option for some patients who desire uterine preservation depending on the size and location of leiomyomas. Ablation involves use of energy (e.g., microwave, radiofrequency, or thermal) to destruct the uterine lining. The procedure is typically done in the Operating Room although office procedures are becoming more common. **Patients need to have completed childbearing and the possibility of cancer needs to be excluded before considering an ablation procedure.** A decrease in menstrual flow is the most common outcome of endometrial ablation while many patients achieve amenorrhea.

Myomectomy, or removal of the leiomyoma, is an alternative to hysterectomy that preserves fertility in patients who desire future childbearing. There are various approaches to myomectomy and the mode depends on the location of leiomyomas, size of leiomyomas, and the skill level of the Gynecologist. An abdominal (i.e., laparotomy or mini-laparotomy) or laparoscopic approach is appropriate for subserosal or intramural leiomyomas whereas a hysteroscopic approach is used for submucosal leiomyomas. Myomectomy carries risks associated with the surgical procedure and the risk of recurrence of leiomyoma growth which is higher if there are multiple leiomyomas present. Hysterectomy is the definitive treatment for leiomyomas and may be used if medical management fails. Hysterectomy may be accomplished via various routes (e.g., abdominal, laparoscopic [single-site, robotic-assisted], or vaginal). The approach to hysterectomy will depend on Gynecologist skill, patient risk factors, uterine mobility, and uterine size.

MALIGNANCY AND HYPERPLASIA (AUB-M)

Endometrial hyperplasia and malignancy is the most concerning cause of AUB. This should be considered in any female with AUB who is menopausal or postmenopausal or who is younger with risk factors for genital tract malignancy.[19-21] Risk factors for endometrial hyperplasia and malignancy include estrogen-secreting tumors, increasing age, Lynch syndrome, obesity, nulliparity, and unopposed estrogen (e.g., anovulatory cycles, hormone replacement therapy, and tamoxifen therapy). Initial evaluation should include a thorough history and physical examination. Pelvic US may be useful for evaluation, particularly for measurement of the endometrial stripe in postmenopausal patients. Definitive diagnosis requires endometrial sampling with an office endometrial biopsy or a hysteroscopy with dilation and curettage in the Operating Room.

Treatment for hyperplasia may be conservative if the patient desires preservation of fertility and typically involves progesterone therapy. Definitive treatment for hyperplasia or malignancy is achieved with a hysterectomy. **Arrange a prompt outpatient referral of stabile patients with a possibility of endometrial hyperplasia or malignancy to a Gynecologist or Gynecologic Oncologist for an endometrial biopsy.**

COAGULOPATHY (AUB-C)

Bleeding disorders are common causes of AUB, especially in adolescents.[22,23] von Willebrand disease is the most common cause. However, any acquired coagulopathy (e.g., thrombocytopenia from cirrhosis), autoimmune disorder, coagulopathy, or myeloproliferative disorder can be the cause. Family and patient history of bleeding disorders, easy bruising, hemarthrosis, menorrhagia, or other symptoms suggesting coagulopathy are an integral portion of the work-up.

Approximately 20% of women presenting with HMB have an underlying bleeding disorder.[24] All adolescents with HMB and all adults with a coagulopathy-positive screen (**Table 161-1**) should

TABLE 161-1 Coagulopathy Screening for Underlying Bleeding Disorders[9]

Heavy menstrual bleeding since menarche and 1 of the following:
- Postpartum hemorrhage
- Surgery-related bleeding
- Dental work-related bleeding

Or 2 or more of the following:
- Bruising 1–2 times per month
- Epistaxis 1–2 times per month
- Frequent gum bleeding
- Family history of bleeding symptoms

have a complete blood count (CBC), partial thromboplastin time (PTT), and prothrombin time (PT). Specific tests for other coagulopathies (e.g., factor VII deficiency, von Willebrand factor antigen, and von Willebrand-ristocetin cofactor activity) may be indicated depending on the history and initial laboratory testing.[11] AUB-C includes those patients on anticoagulants because the management is similar to inherited disorders.

OVARIAN DYSFUNCTION (AUB-O)

Although most AUB occurs during ovulatory cycles, anovulation is the most common cause of nonstructural bleeding. This can lead to anemia and diminished quality of life. Normally the endometrium is exposed to estrogen during the development of the ovarian follicle then both estrogen and progesterone after ovulation. Normal bleeding occurs after these hormones are withdrawn. The endometrium is exposed to unopposed estrogen if ovulation does not occur that can cause abnormal bleeding. This can be physiologic or pathologic. Physiologic causes include adolescence, lactation, menopause, and perimenopause. Pathologic causes include hyperandrogenic anovulation (e.g., polycystic ovarian syndrome, congenital adrenal hyperplasia, androgen-secreting tumors), hyperprolactinemia, hypothalamic dysfunction (e.g., anorexia, exercise, or stress), iatrogenic (e.g., chemotherapy or radiation), medications, pituitary disease, premature ovarian failure, or thyroid disease.[25]

Ovarian dysfunction has many potential causes and can affect any age group.[8] It usually occurs during perimenarche in adolescent females and is often due to an immature hormonal axis. Ovarian dysfunction in this age group produces minimal bleeding in most cases. Heavy bleeding is a concern for a coagulopathy. Follicular degradation and hormonal fluctuations are the primary causes of anovulatory bleeding in the perimenopausal woman. Hypothyroidism may be associated with ovarian dysfunction outside of these age groups. Other medical causes of ovarian dysfunction and AUB-O include adrenal disorders, eating disorders, exogenous hormones, hormonal disorders, intense exercise, or steroid use.

Structural and endometrial pathology must be ruled out before a diagnosis of AUB-O can be made. Cycles that vary in length by more than 10 days are likely anovulatory. Anovulatory cycles are common for the first 3 years after menarche in the adolescent patient (i.e., 13 to 18 years). Examine adolescent women for a coagulopathy, pregnancy, sexually transmitted diseases, and sexual trauma. Women age 19 to 39 most commonly have polycystic ovarian system whereas those over age 40 most commonly have AUB-O due to the menopausal transition. **All women 45 and older who have suspected AUB-O must have an endometrial biopsy because of the increased risk of cancer. Refer patients with prolonged anovulatory bleeding or other risk factors for increased estrogen exposure (e.g., obesity) for an endometrial biopsy.** Laboratory testing of suspected AUB-O patients includes a pregnancy test, prolactin level, and thyroid-stimulating hormone (TSH). Refer the patient to a Gynecologist for an endometrial biopsy or sonohysterography.

Treatment is aimed at the underlying endocrine disorder which includes exogenous steroid use. This can be progesterone alone or combined estrogen and progesterone. Use progesterone only if contraindications to estrogen exist (e.g., coagulopathies, hypertension, or smokers over 35 years of age). Otherwise, combined contraceptives are a better choice because they not only protect the patient from unwanted pregnancy but also allow for predictable bleeding. Combined contraceptives can be used in an extended fashion (i.e., skipping the placebo week) to help recover from anemia. Estrogen will decrease menopausal symptoms for women in the menopause transition and maximize bone density. See **Table 161-2** for a list of therapies. Weight loss and exercise may be prescribed in overweight women. Failing medical therapy requires further investigation by a Gynecologist and includes endometrial biopsy, hysteroscopy, imaging, or uterine ablation.

TABLE 161-2 Hormonal Therapy for AUB-O

Progesterone only	Combined estrogen and progesterone
Medroxyprogesterone acetate 10–20 mg (Provera)	Oral contraceptive pill
Norethindrone 10 mg	Transdermal patch
Megestrol acetate 40 mg (Megace)	Vaginal ring
Depot medroxyprogesterone (Depo-Provera)	Oral contraceptive taper for stable active bleeding and anemia
Levonorgestrel intrauterine device (IUD) (Mirena)	IV estrogen for unstable active bleeding and anemia

ENDOMETRIAL CAUSES (AUB-E)

AUB sometimes occurs in the setting of a normal ovulatory cycle, normal uterine tissue, and no other medical cause. Menstruation is often heavy in these situations. This is due to abnormal prostaglandin synthesis and receptor upregulation, increased local fibrinolytic activity, and increased tissue plasminogen activator activity.[11] It is often treated with oral contraceptives and NSAIDs for symptomatic treatment of pain. Other treatment includes tranexamic acid or levonorgestrel-releasing IUDs. More intensive treatments of hysterectomy and uterine ablation may be considered by the Gynecologist in severe cases.

Inflammation and infection can cause IMB. Acute endometritis is seen in the settings of pelvic inflammatory disease, postabortion, postpartum hemorrhage, and after procedures. Refer to the current Centers for Disease Control and Prevention recommendations for inpatient and outpatient treatment.

IATROGENIC CAUSES (AUB-I)

Medications are common causes of AUB (**Table 161-3**). Many antipsychotics cause hyperprolactinemia by blocking dopamine receptors. Oral contraceptives and other gonadal steroid medications can cause unscheduled or "breakthrough bleeding." This is more common with initiation of the hormones or prolonged use. IUDs can cause both HMB and IMB. The levonorgestrel-releasing IUD can cause HMB or IMB that usually resolves with time. It can eventually cause amenorrhea. The copper IUD often causes HMB that can be treated with perimenstrual NSAIDs. It may be easy to attribute AUB to these common medications. Do not rule out other potential causes of AUB without a thorough evaluation and Gynecologist referral.

TABLE 161-3 Medications and Medical Devices That Can Cause AUB

Antiemetics, especially metoclopramide
Antihypertensives, especially methyldopa and verapamil
Antipsychotics, especially first generation
Contraceptive rings
Douches
Exogenous hormones
Glucocorticoids
Hormone replacement therapy
Intrauterine devices
Medications that affect the hepatic P450 system
Medications that affect the hormonal axis
Oral contraceptives
Pessaries
Tricyclic antidepressants, especially clomipramine
Vaginal irritants

OTHER CAUSES (NOT YET DETERMINED) (AUB-N)

Intravaginal devices (e.g., pessaries or hormonal contraceptive rings), douching, or other irritants may cause vaginitis and bleeding. Sexually transmitted infections, sexual trauma, and vaginal infections are a common cause of AUB, especially in younger sexually active women. Other causes include arteriovenous malformations (AVMs), chronic endometritis, and etiologies that do not fit into the other categories. Bleeding from an AVM will often present as excessive bleeding after a uterine procedure (e.g., dilation and curettage).[26,27]

HISTORY AND PHYSICAL EXAMINATION

The patient's history and physical examination may steer the Emergency Physician toward a cause. Determining the exact cause of a patient's AUB is not the goal for the Emergency Physician. The work-up done by the Emergency Physician will likely be an integral part of a consultant's evaluation of the patient.

Important aspects of the history include the last Pap smear and its results, familial bleeding problems or disorders, patient bleeding problems or disorders, uterine pathology, cancer history, and recently starting or stopping medications. Key points to obtain from the medical history include a thorough menstrual history (e.g., onset of menarche, typical cycle frequency, days of bleeding, clots, number of pads/tampons used, and presence or absence of pain with cycles) and screening for an underlying bleeding disorder. Family history should focus on any bleeding or clotting disorders. Review any medications and include any herbal supplements. Inquire what treatments have controlled bleeding in the past. Women with HMB tend to be anemic, change sanitary products at night, change sanitary products at < 3 hour intervals, pass clots > 1 inch in diameter, and use > 21 sanitary products in a cycle.[28]

The physical examination can help determine the source of bleeding (e.g., cervical, rectal, urethral, uterine, vaginal, or vulvar). The examination includes a speculum examination to evaluate for abnormalities (e.g., cervical or vaginal) and a bimanual examination (e.g., consistency, mobility, shape, size, and tenderness) of the pelvic organs. A fibroid uterus may feel enlarged with an irregular contour. A uterus with adenomyosis may feel slightly enlarged, globular, and boggy (i.e., soft).

Corporeal signs are helpful in diagnosis. Extremes of weight cause hypothalamic dysfunction. Hirsutism and acne are signs of increased androgen production that can be seen with congenital adrenal hyperplasia and polycystic ovarian syndrome. Acanthosis nigricans is a sign of insulin resistance and is also seen in polycystic ovarian syndrome. Exophthalmos, lid lag, and myxedema are signs of thyroid dysfunction. Petechiae, ecchymosis, skin pallor, and swollen joints may be indicative of a bleeding disorder.

The initial laboratory evaluation should include a pregnancy test, CBC, and TSH. Perform chlamydia testing, coagulation studies (e.g., international normalized ratio [INR], PT, and PTT), iron studies, and a type and screen if indicated. Pelvic US is the first-line imaging to assess for structural causes of AUB (e.g., adenomyosis or leiomyomas). Refer the patient to a Gynecologist for sonohysterography or diagnostic hysteroscopy if an intrauterine polyp or submucosal leiomyoma is suspected on initial imaging. Both methods use fluid to distend the endometrial cavity which allows visualization of protruding structures.

EQUIPMENT

- Speculum, various sizes
- Water-soluble lubricant
- Balloon catheters to tamponade the uterus
- Cardiac monitor
- Pulse oximeter
- Noninvasive blood pressure monitor
- Oxygen source
- Nasal cannula and face masks
- IV access supplies (Chapters 59, 61 to 64, and 70)
- Resuscitation equipment
- US machine
- US transducers
- US transducer cover
- US gel

At least two transducers are required to obtain a good US examination (Chapter 160). Perform an abdominal US with a 3.0 to 5.0 MHz curvilinear transducer. The lower frequency of this transducer allows imaging of the pelvic organs through the abdominal wall. Perform the intravaginal US with a 8.0 to 13.0 MHz endocavitary transducer. This high-frequency and long-shaped transducer provides a wide field of view and details of the pelvic organs.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Place the patient on the cardiac monitor, noninvasive blood pressure cuff, pulse oximetry, and supplemental oxygen to monitor them during and after the procedure. Obtain IV access. Resuscitate the patient with fluids and blood products as necessary. Have resuscitation medications at the bedside if needed. Don personal protective equipment to protect from contact with the patient's blood and body fluids.

Prepare the US transducer if using US (Chapter 160). Apply US gel over the footprint of the transducer. Place the gel-coated US transducer in the sterile cover. Place US gel on the transducer. Place the patient supine. Explain the transabdominal and transvaginal US examination.

Perform a transabdominal US examination. This should be performed first to scan the pelvis in the transverse and sagittal planes (Chapter 160). Place the transducer in the midline and just below the pubic symphysis. Make sure the marker is facing cephalad. The filled bladder is a sonographic window when scanning the pelvis. Do not wait until the bladder fills if not filled with fluid. Find the bladder and vaginal stripe as landmarks. The bladder is fluid filled with a black anechoic center. The vaginal stripe is a bright white line. The uterus is posterior to the bladder. Follow the vaginal stripe upward into the cervix and uterus. Scan with the transducer sagittally in the left and right of midline to look for normal anatomy and pathology. Turn the transducer 90°. Scan with the transducer transversely up and down to look for normal and pathology.

Perform an endovaginal US (Chapter 160). Ask the patient to empty their bladder or catheterize the bladder (Chapter 173). Place the patient supine with their legs abducted and knees bent 45° or the frog-leg position. Carefully insert the endocavitary transducer. Scan the pelvic organs sagittally and transversely (Chapter 160).

TECHNIQUES

MEDICAL MANAGEMENT

Management of acute AUB depends on clinical stability and the suspected etiology.[5,29-32] First-line therapies for stable bleeding include gonadal steroids (more effective) and NSAIDs (less effective).

NSAIDs block the formation of prostacyclin, a thromboxane antagonist, and decrease uterine blood flow. Steroid hormone treatment includes progesterone alone (less effective) and combined estrogen and progesterone oral contraceptives (more effective). Combined oral contraceptives containing 35 mcg of estrogen may be used in multidose regimens or pill tapers. One dosing regimen includes taking a combined oral contraceptive three times a day for 7 days. An example of a pill taper is one pill three times a day for 3 days, one pill twice a day for 3 days, then one pill daily to finish the pack. Skip the placebo pills and start a new pack to prevent bleeding. Consider prescribing an antiemetic for possible nausea and vomiting.

Contraindications to combined oral contraceptives include heavy cigarette smoking in a patient older than 35 years, history of venous or arterial thromboembolism, hypertension, migraine with aura, past or present breast cancer, prolonged immobilization, severe liver disease, or valvular heart disease. See U.S. medical eligibility criteria for contraceptive use (www.cdc.gov/reproductivehealth/unintendedpregnancy/usmec.htm).

Medroxyprogesterone acetate (Provera) is a progesterone-only alternative to help with acute AUB in patients who have contraindications to estrogen. This synthetic progestin has an antimitotic effect and allows the endometrium to atrophy. A regimen that has shown to be effective in decreasing bleeding is oral Provera 20 mg three times a day for 7 days followed by 10 mg daily for maintenance. Megestrol acetate (Megace) is another progesterone agent that may be used for acute AUB in doses as high as 80 mg twice a day until the patient can be seen by a Gynecologist. Contraindications to progesterone therapy include active liver disease, active venous thromboembolism, and breast cancer.

Tranexamic acid (Lysteda) is a synthetic analogue of the amino acid lysine. It is an antifibrinolytic agent that works by displacing plasmin from fibrin. Both intravenous (IV) and oral forms are available for use.[31,33] Oral tranexamic acid can be used for outpatient treatment of uterine bleeding in a hemodynamically stable patient. Consider IV tranexamic acid for uterine bleeding in a hemodynamically unstable patient. However, massive uterine bleeding is an off-label use. Oral tranexamic acid is an alternative to hormone therapy for the management of AUB at a dose of 1300 mg every 8 hours for 5 days. An alternative is 300 to 1000 mg orally three times a day for 3 days. Administer 1 gm IV over 10 minutes and an additional 1 gm 8 hours later for HMB. This therapy has been highly effective at treating chronic AUB with a reduction in bleeding of nearly 50% but currently has limited data for use in acute AUB.[10] **Use tranexamic acid with caution in patients at high risk for thrombosis.**

IV conjugated equine estrogen or Premarin (25 mg every 4 to 6 hours for 24 hours) has been shown to successfully stop bleeding in approximately 75% of patients within 8 hours of initiation for unstable bleeding or severe anemia.[13] Contraindications to IV estrogen therapy include breast cancer, history of venous or arterial thrombosis, or active liver disease.

Other medical treatments include GnRH analogues or levonorgestrel-releasing IUD systems.[34] GnRH analogues downregulate pituitary receptors and suppress GnRH release. It is a form of medical castration. Prolonged treatment with GnRH analogues is associated with osteoporosis and postmenopausal symptoms. These medications should be administered and managed by a Gynecologist.

INTRAUTERINE BALLOON PLACEMENT

Another option for stabilization is uterine tamponade.[21,35-45] This can be used in conjunction with medical therapies. There are various methods that provide intrauterine compression including 4 inch gauze packing with the tail extending through the cervix and balloon catheters. The uterine packing or balloon can remain in place for 24 hours while the patient is resuscitated with fluids and blood products. Antibiotic prophylaxis with a cephalosporin is recommended after uterine packing.

There are multiple balloon catheters that may be used for uterine tamponade including Foley catheters (Chapter 173), Bakri (**Figure 161-2*A***) or Ebb balloons (**Figure 161-2*B***), Sengstaken-Blakemore balloons (Chapter 81), Rusch catheters (**Figure 161-2*C***), and condom catheters. The Bakri tamponade balloon catheter and

A

B

C

FIGURE 161-2. Balloon catheters. ***A.*** Bakri. ***B.*** Ebb. ***C.*** Rusch.

the Ebb complete tamponade system are large-volume balloon catheters specifically designed for placement in the uterus for control of uterine hemorrhage. The Rusch urologic balloon is a large-volume urinary catheter originally developed with the intent to control vesicular hemorrhage. All three devices can be used to treat uterine bleeding as a tamponade system. The pressure applied by the inflated balloon against the uterine wall causes hemostasis. Balloon catheters, except Rusch and condom catheters, have an outflow component that allows for continued measuring of blood loss and monitoring of hemostasis. The balloons typically remain in place for 12 to 24 hours with antibiotic prophylaxis while hemodynamic stability is achieved. The balloons may accommodate up to 500 mL of saline except Foley catheters. Multiple Foley catheters may be tied together and inflated with 30 to 60 mL each. Dilation and curettage may be necessary for definitive care.

INDICATIONS

Placement of an intrauterine balloon catheter may be successful in stopping uterine hemorrhage and result in hemostasis. **It is a temporary measure while awaiting more definitive surgical procedures (e.g., arterial embolization, arterial ligation, or hysterectomy).** An intrauterine balloon can be used for suspected AUB when ectopic pregnancy and urogenital tract lacerations have been ruled out. The use of a balloon is indicated before more invasive surgical approaches. It is also placed for postpartum hemorrhage.

CONTRAINDICATIONS

Known infection (i.e., vagina, cervix, or uterus) and uterine abnormalities are a contraindication to balloon placement. The patient can become more septic when the balloon is inflated and the infection leaves the female genital tract. Balloon inflation would be unlikely to apply effective tamponade for hemorrhaging uterine cancer and uterine rupture. Intrauterine balloon placement is contraindicated when the source of bleeding is suspected to be extrauterine (e.g., vaginal or vulvar injuries leading to bleeding distal to the uterus or in ectopic pregnancy). Avoid intrauterine balloons if the patient has any known allergies to device materials. Arterial bleeding requiring surgical exploration or angiographic embolization is a contraindication to inserting a balloon. Disseminated intravascular coagulation and other coagulopathies are a relative contraindication. Surgery of the uterus prohibits device inflation and bleeding tamponade.

EQUIPMENT

- Internal antiseptic solution (e.g., povidone iodine)
- 60 mL syringe
- Balloon catheter device
- IV extension tubing with attached bag spike
- 500 mL sterile liquid (e.g., saline or water)
- Iodine gauze or antibiotic-soaked gauze
- Ring forceps

The Bakri balloon catheter is a common device that is for one-time use. It is made of silicone and contains no latex. A large-bore drainage channel is rarely blocked by blood clots. The balloon contains up to 500 mL of fluid. The catheter measures 43.5 cm in length and is a size 24 French (i.e., 8 mm).

PATIENT PREPARATION

Consider premedicating the patient with pain and/or nausea medications. Intrauterine balloon tamponade can cause significant pain. Determine the uterine volume by either direct examination or US measurements. This prevents balloon overdistension and rupture

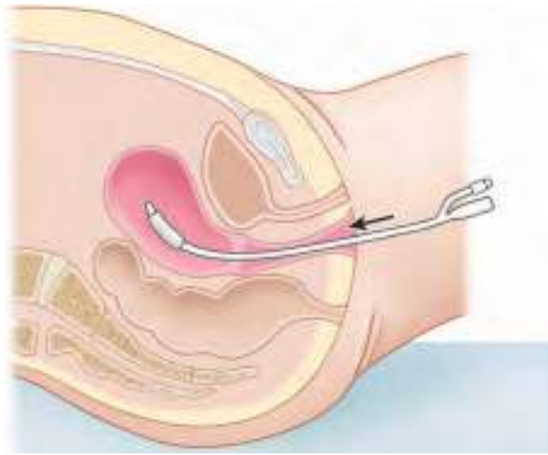

FIGURE 161-3. Transvaginal placement of a balloon catheter into the uterus.

of the uterus. Evaluate the genital tract for lacerations or trauma. Clear the uterus of placental fragments if the patient is postpartum. Clean the vulva, vagina, and cervix with povidone iodine or other internal body safe solution. Place a Foley catheter into the bladder to monitor urine output.

TECHNIQUE

Place the patient in the lithotomy position. The description is for the Bakri balloon. Another balloon catheter can be used and inflated within the uterus. Carefully and gently grasp the cervix with a ring forceps to stabilize its position. Carefully and gently insert the balloon portion of the catheter fully through the vagina and cervical canal and past the internal ostium and into the uterus (**Figure 161-3**). Use US guidance if possible during the insertion. Pull the catheter until the base of the balloon is at the internal cervical ostium (**Figure 161-4**).

Attach the IV extension tubing connected to the sterile fluid to the stopcock. Attach the 60 mL syringe to the stopcock. Slowly inflate the balloon with sterile liquid (e.g., Ringer's lactate, saline, or water) to a maximum of 500 mL (**Figure 161-4**). **Do not inject the balloon with air or any gas. Do not overinflate the balloon and cause a uterine rupture.** Consider placing the sterile liquid in a separate container when measured to ensure proper volume instillation to avoid losing count of the syringes used. Confirm that the balloon is properly placed using transabdominal US.

Apply light tension to seat balloon against the internal ostium of the cervix. Secure the catheter to the patient's thigh or hang a weight from the catheter. **Do not exceed 500 gm of tension weights.** This tension is essential for tamponade of bleeding vessels. Consider packing the vagina to apply counter pressure and tamponade

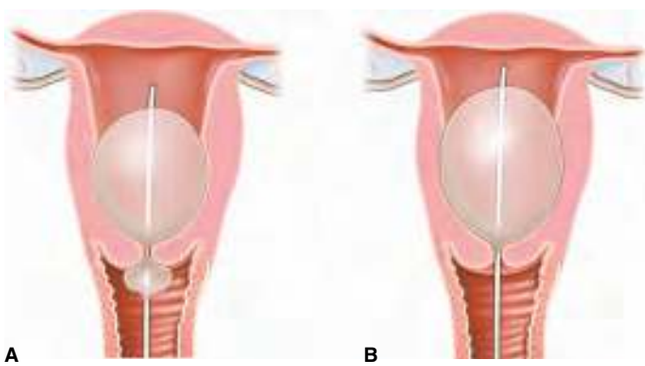

FIGURE 161-4. Placement and inflation of a balloon catheter. ***A.*** Improper placement. ***B.*** Proper placement.

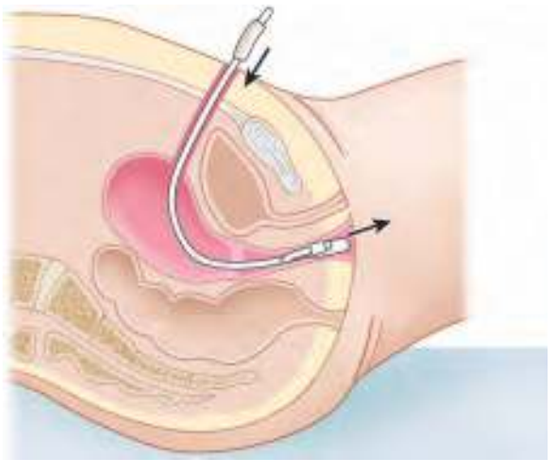

FIGURE 161-5. Transabdominal placement of a balloon catheter into the uterus.

the bleeding. Use iodine gauze or antibiotic-soaked gauze for the vaginal packing. Connect the drainage port to a collection bag to monitor any continued bleeding. Flush the drainage port and tubing periodically with sterile saline to remove any clots.

An alternative technique is used by the Gynecologist if placing the balloon catheter through an abdominal incision (e.g., after cesarean section) (**Figure 161-5**). Insert the inflation port through the skin incision and into the uterus. Thread the proximal end of the catheter through the internal ostium from within the uterus to properly seat the balloon within the uterine cavity. Close the uterus and skin in the usual fashion with care to avoid puncturing the balloon and/or catheter.

BALLOON REMOVAL

Removal of the catheter is simple. Relieve the tension on the catheter by removing any weights and detaching the catheter from the patient's thigh. Attach a 60 mL syringe to the balloon inflation port. Completely deflate the balloon in increments to allow patient observation throughout the removal procedure. Remove any vaginal packing, if present. Gently remove the catheter. Inspect the balloon to assess for damage or possible retained balloon fragments. Discard the balloon catheter. Monitor patient for signs of bleeding.

COMPLICATIONS

The catheter and balloon may be inappropriately placed. The balloon can be overinflated and risk uterine rupture. Underinflation of the balloon will result in lack of tamponade and continued bleeding. Bleeding can persist around the balloon. Insufficient tension on the catheter may not provide effective tamponade.

SUMMARY

Place the Bakri balloon for non–life-threatening uterine bleeding. The balloon must be completely within the uterus, inflated to a maximum of 500 mL, and placed under tension to provide a source for tamponade. Remove the balloon within 24 hours. Closely monitor patients requiring uterine balloon placement for signs of continued hemorrhage.

REFERRAL TO A GYNECOLOGIST

Some patients require a Gynecologist consult in the Emergency Department. Heavy uterine bleeding may require uterine ablation. Symptomatic leiomyomas may require a myomectomy. Long-term bleeding, bleeding resulting in acute blood loss anemia, or hemodynamic instability may require a hysterectomy.

ASSESSMENT

The balloon and/or packing can be left in place for up to 24 hours. Resuscitate the patient with fluids and blood products as necessary. Frequently reassess the patient for continued bleeding and hemodynamic stability. Monitor any continued hemorrhage from the vagina. Consult a Gynecologist if bleeding stops and the patient is stable. Emergently consult a Gynecologist for continued bleeding. The continuously bleeding patient must be taken to Invasive Radiology or the Operating Room to stop the bleeding.[46]

AFTERCARE

Stable patients with minimal bleeding and minimal symptoms can be discharged from the Emergency Department with Gynecology follow-up in 24 to 48 hours. Prescribe oral iron and multivitamins. NSAIDs will help manage minor bleeding and pain. These patients may receive elective dilation and curettage, endometrial ablation, and outpatient hormonal therapy.

Severe bleeding requires multiple interventions after placement of the balloon catheter to tamponade uterine bleeding. Reverse any bleeding disorders. Consider administering fresh frozen plasma and 10 mg of vitamin K IV to patients taking warfarin (Coumadin), patients with end-stage renal disease, or patients with a high INR. Consider administrating 0.4 mcg/kg desmopressin IV over 10 minutes if the patient has a platelet disorder or renal disease. Administer IV estrogen if not contraindicated. Consult a Gynecologist. Do not reinflate the balloon and do not reinsert the balloon if bleeding recurs during deflation or removal. Use a new balloon catheter. Continual bleeding or recurrent bleeding requires more invasive treatments in the Operating Room or Invasive Radiology.[46]

COMPLICATIONS

The indications and contraindications to some medications have already been discussed. The catheter and balloon may be inappropriately placed. The balloon can be overinflated and risk uterine rupture. Underinflation of the balloon will result in lack of tamponade and continued bleeding. Bleeding can persist around the balloon. Insufficient tension on the catheter may not provide effective tamponade. Insertion and inflation of a balloon catheter in an infected uterus can result in sepsis. Uterine abnormalities can result in difficult balloon inflation, difficult insertion, rupture, and traumatic lacerations. Continued bleeding can result from balloon ineffectiveness, hemorrhage from cancer necrosis, extrauterine bleeding, and uterine rupture. Only inflate the balloon with liquid to prevent a gas embolism.

Complications can arise from placing a Foley catheter (Chapter 173). The catheter can allow bacteria into the bladder and cause a fever, bladder infection, hematuria, and a kidney infection. This can partially be prevented by using strict sterile technique.

SUMMARY

Abnormal uterine bleeding is a very common complaint in the Emergency Department. Nonpregnant uterine bleeding can be due to many causes and result in rapid large-volume blood loss. Most AUB has a structural cause. Rule out coagulopathy, endocrinopathy, malignancy, and pregnancy. Bleeding is often due to ovulatory dysfunction if a structural cause is not found and can be medically treated in most cases. Any suspicion of cancer requires a Gynecologist referral and possible biopsy. Consult a Gynecologist immediately if a patient is actively bleeding and is hemodynamically unstable. Medical therapies include tranexamic acid and blood product transfusion while awaiting Gynecology consultation.

Administer hormonal therapies for lower volume bleeding. Numerous procedures are available for bleeding (e.g., balloon catheter placement, myomectomy, and uterine ablation).

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162 Normal Spontaneous Vaginal Delivery

Stephen Dunay and Simeon Ashworth

INTRODUCTION

Emergency Physicians must be prepared to manage the precipitous delivery of a newborn. It is a rare occurrence and requires a good amount of preparation. The Emergency Department is not the ideal environment in which to deliver a newborn.[1] A Labor and Delivery suite with a specialist trained in handling potential complications (e.g., malpresentation, prematurity, or prolapsed cord) is preferred. The Emergency Physician will need to deliver the infant when transfer to a facility with a trained Obstetrician, Family Physician,

or Certified Nurse Midwife is not feasible.[2] A thorough understanding of the physiology of pregnancy and labor, the evaluation of the pregnant female, and the proper technique of delivery will help to ensure the safety of both mother and fetus.

Labor involves the repetitive uterine contractions that lead to cervical changes (i.e., dilation and effacement). The mechanisms of labor, or cardinal movements of labor, describe the changes in the position of the fetal head as it travels through the birth canal. **The safe delivery of the infant is the goal of labor while decreasing maternal morbidity.**

ANATOMY AND PATHOPHYSIOLOGY

A successful vaginal delivery depends on the adequacy of the female pelvis. Pelvimetry may be used during prenatal visits to evaluate the adequacy of the pelvic space for fetal passage. Inadequate space may result in fetal entrapment, prolonged labor, and shoulder dystocia.[3] The most useful planes for measurement are the pelvic inlet and the midplane (**Figures 162-1*A* and 162-1*B***). The pelvic inlet is through measurement of the diagonal conjugate. The midplane is through measurement of the ischial interspinous or bi-ischial diameter.[3]

The diagonal conjugate refers to the distance from the inferior border of the pubic symphysis to the sacral promontory (**Figure 162-1*A***). The normal diagonal conjugate measures approximately 12.5 cm, with the critical distance being 10 cm. Place the tip of the middle finger at the sacral promontory and note the point on the hand that contacts the pubic symphysis (**Figure 162-1*B***). The true conjugate, a radiographic measurement of the pelvic inlet, represents the smallest diameter of the inlet from the sacral promontory to the superior aspect of the pubic symphysis (**Figure 162-1*A***). It can be estimated by subtracting 1.5 to 2 cm from the diagonal conjugate and normally measures 11 cm or more (**Figure 162-1*A***). The obstetric conjugate is the distance from the sacral promontory to a point on the inner surface of the pubic symphysis that is a few millimeters from the upper margin of the pubic symphysis (**Figure 162-1*A***). This corresponds with the true conjugate and is approximately 11 cm.[3] The midpelvis can be evaluated through measurement of the ischial interspinous diameter. Measure the distance between the ischial tuberosities (**Figure 162-1*C***). A value greater than 10 cm is considered adequate.[3]

The assessment of maternal pelvic shape is generally not of clinical significance in the Emergency Department where patients will be delivering precipitously. Current clinical practice guidelines recommend a trial of labor as the fundamental way to determine whether a fetus will be able to pass through the maternal pelvis. Studies have suggested that the routine practice of pelvimetry is not effective in predicting the occurrence of cephalopelvic disproportion and is associated with an increase in rates of cesarean section.[4-6]

FETAL POSITIONING

Consider the anatomic position of the fetus in preparing for the successful delivery of an infant. This includes the fetal lie, fetal presentation, fetal station, and fetal position. Fetal lie may be a longitudinal, oblique, or transverse relationship between the longitudinal axis of the fetus in relation to the long axis of the mother. Fetal presentation defines the fetal part that can be felt during delivery. Infants will present in vertex or cephalic presentation most commonly. The Emergency Physician should be able to palpate the skull. An infant in a breech presentation will be in the longitudinal lie with the buttocks or feet first.

Fetal station refers to the relationship between the presenting part and the maternal ischial spines. Each number represents a centimeter in distance from the ischial spines. It is reported as a negative (i.e., from 0 to –5) if the presenting part lies above the ischial spines and positive (i.e., from 0 to +5) if it lies below the ischial spines. The fetus is considered "engaged" in the pelvis when it reaches the level of the ischial spines or 0 station.

The fetal position describes the relationship of the fetal presenting part (e.g., buttocks, foot, leg, or occiput) to the maternal pelvis (**Figure 162-2**). The position is best assessed by a sterile vaginal exam after the mother is completely dilated. The fetal occiput in the vertex presentation is used to determine the fetal position. Certain positioning (e.g., occiput posterior and occiput transverse) can make delivery more difficult. The different fetal vertex positions are noted in **Figure 162-2**.

CLASSIFICATION OF LABOR

The process of labor is divided into three stages and involves the repetitive uterine contractions that lead to cervical dilation and effacement. The first stage is the period between the onset of labor and complete cervical dilation. The second stage begins with complete cervical dilation and ends with the delivery of the infant. The third stage consists of the time between delivery of the infant and delivery of the placenta.

FIRST STAGE OF LABOR

The first stage of labor is further divided into three phases. The first or latent phase is the period between the onset of labor to cervical dilation of 3 to 4 cm. The second phase leads up to almost complete cervical dilation. During the third phase, maximum cervical dilation is achieved. The first stage of labor lasts approximately 8 hours in nulliparous women and 5 hours in multiparous women.

The American College of Obstetricians and Gynecologists (ACOG) recommends monitoring the fetal heart rate (FHR) during active labor by Doppler immediately following a contraction, every 30 minutes in low-risk pregnancies, and continuously

FIGURE 162-1. Measure pelvic distances to determine if there may be difficulties during the delivery. ***A.*** The pelvic conjugate diameters. ***B.*** Measuring the diagonal conjugate. ***C.*** The ischial interspinous distance.

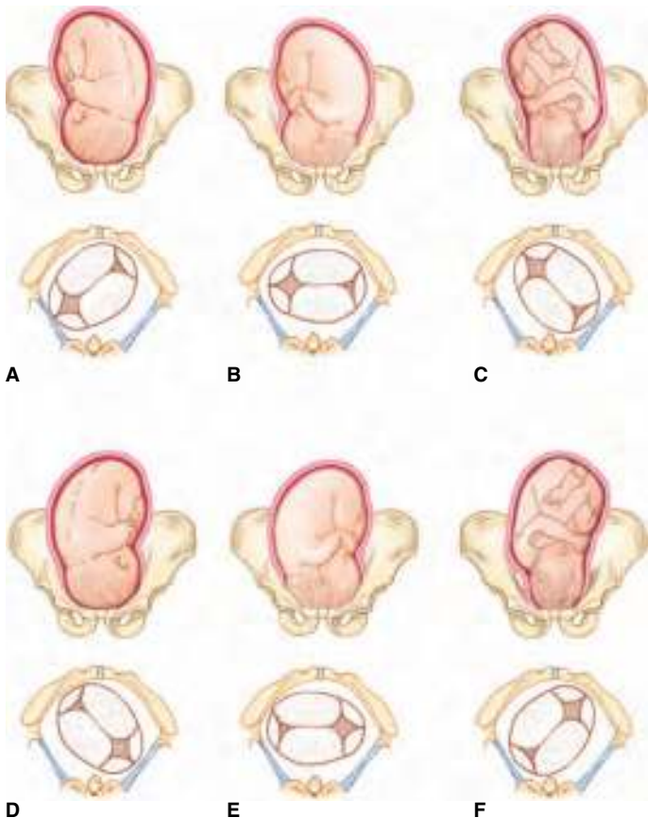

FIGURE 162-2. Fetal presentations and positions in labor. ***A.*** Left occiput anterior (LOA). ***B.*** Left occiput transverse (LOT). ***C.*** Left occiput posterior (LOP). ***D.*** Right occiput anterior (ROA). ***E.*** Right occiput transverse (ROT). ***F.*** Right occiput posterior (ROP).

with high-risk pregnancies.[7] Suspect fetal distress if the heart rate following a contraction is repeatedly below 120 beats per minute.[8] Monitor the FHR following the contraction after the rupture of the membranes (ROM) to assess for occult umbilical cord compression.[8]

Contraction intensity is determined by the degree of firmness that the uterus achieves. Contractions may be palpated or monitored by an external transducer (i.e., tocodynamometer) placed upon the maternal abdomen. Monitor the maternal vital signs during active labor. Obtain a temperature at least every 2 hours and a blood pressure every 30 minutes.[9] Antibiotic coverage for group B streptococcal infection is recommended in the case of prolonged ROM or membrane rupture lasting longer than 18 to 24 hours prior to the onset of labor.[8,10,11] Perform a vaginal examination immediately after ROM if the fetal head is not engaged because of the possibility of a prolapsed umbilical cord.

SECOND STAGE OF LABOR

The second stage of labor begins with complete dilatation of the cervix and ends with delivery of the infant. It lasts approximately 50 minutes in nulliparous women and 20 minutes in multiparous women in an uncomplicated vaginal delivery. It is recommended that the FHR be auscultated at least every 15 minutes in low-risk infants and at least every 5 minutes in high-risk infants.[7] FHR decelerations may occur with contractions as the head descends. Allow labor to progress if prompt recovery occurs as the contraction diminishes.[7] Decelerations may occur from progressive compression of an umbilical cord around the fetus, premature placental separation, or reduction in uterine blood flow.[12] Prolonged FHR decelerations are an indication for immediate cesarean section.[7-9,11,12]

Bearing down is a natural reflex during the second stage of labor. Coaching may be helpful to optimize pushing efforts, especially in the presence of epidural anesthesia. Other techniques to optimize pushing efforts include flexing the maternal legs to 90° at the hips and knees, encouraging the patient to take a deep breath at the onset of a contraction and then push downward or Valsalva, and encouraging rest between contractions. Rest between contractions is needed for the mother and the fetus to recover from the effects of breath holding, muscular effort, and uterine contractions.[8]

The perineum will bulge as the fetus descends into the maternal pelvis. Sponge any expelled stool downward with a sterile sponge and diluted soap solution. Delivery is eminent when the scalp of the infant becomes visible at the introitus.

MECHANISM OF LABOR

The mechanism of labor or the cardinal movements of labor describe the process by which the fetal head optimally descends in relational to the maternal pelvis (**Figure 162-3**). They consist of engagement, descent, flexion, internal rotation, extension, external rotation, and expulsion (**Figure 162-4**).

The fetal head floats freely in the amniotic fluid before it engages the maternal pelvis (**Figure 162-4*A***). The head is engaged when the widest diameter of the presenting part of the fetus (i.e., the biparietal diameter) passes below the plane of the pelvic inlet (**Figure 162-4*B***). This can be confirmed clinically by palpation of the presenting part abdominally and vaginally. Bimanual examination will reveal the presenting part at station 0 or at the level of the maternal ischial spines. The head then descends into and through the maternal pelvis (**Figures 162-4*B* and 162-4*C***). The greatest rate of descent occurs near the end of the active phase of labor.

The head flexes as it meets resistance from the cervix, pelvic floor, and pelvic walls during passage (**Figure 162-4*B***). The smallest diameter of the fetal head (i.e., the suboccipitobregmatic diameter) passes passively through the maternal pelvis.

The fetal head then internally rotates causing the head to move anteriorly toward the pubic symphysis to assume the occiput anterior position (**Figures 162-4*C* and 162-4*D***). It may rotate less commonly posteriorly toward the sacral hollow to assume the occiput posterior position. This too is a passive movement resulting from the contours of the maternal pelvis.

The fetal head then descends to the level of the introitus and begins to extend until the head is delivered (**Figures 162-4*D* and 162-4*E***). A combination of the downward force of the contracting uterus and the upward force exerted by the maternal pelvic floor muscles rotates the fetal head in extension around the pubic symphysis. Extension continues until the fetal head is delivered.

The body externally rotates once the head is delivered. This brings the occiput and spine back into the same plane with the shoulders (**Figure 162-4*F***). The body rotation can occur to either side depending upon the orientation of the fetus. The infant is then expelled or delivered. Anterior shoulder delivery precedes the delivery of the posterior shoulder in most cases (**Figures 162-4*G* and 162-4*H***).

INDICATIONS

The indications for the performance of a vaginal delivery in the Emergency Department include a patient in active labor without the ability or time to transfer the patient to a delivery unit. The lack of an Obstetrician, Family Physician, Certified Nurse Midwife, or other obstetric practitioner will require the Emergency Physician to perform the delivery.

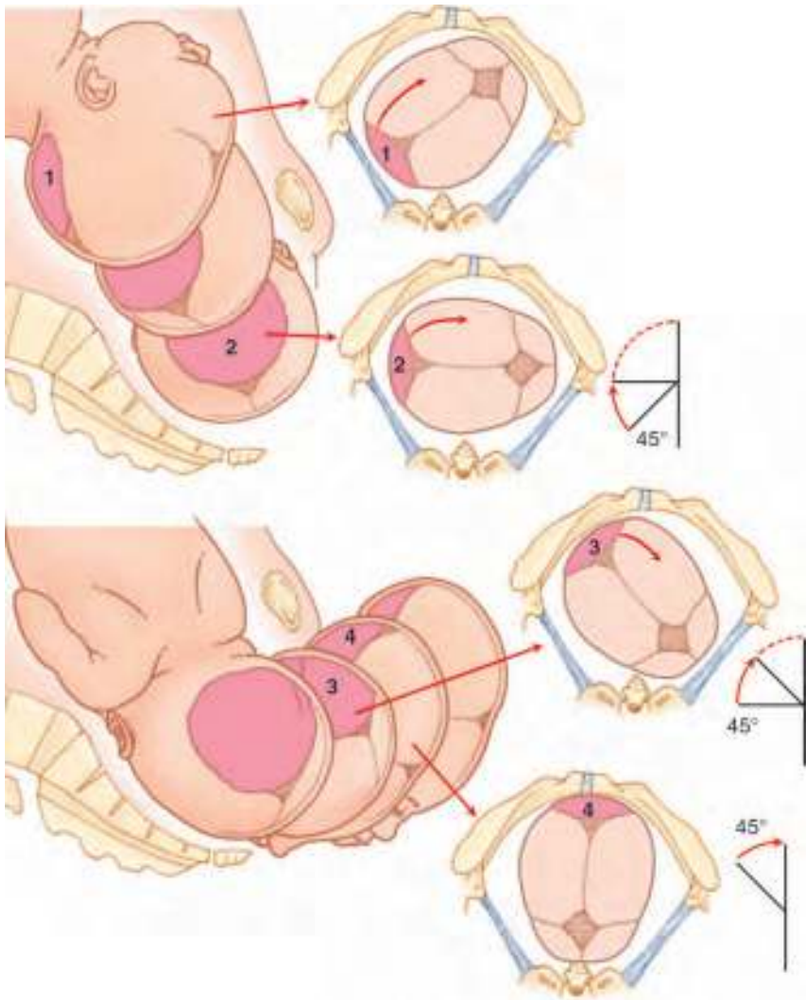

FIGURE 162-3. The mechanism of labor.

CONTRAINDICATIONS

There are no absolute contraindications to the emergent delivery of the fetus in the Emergency Department. Delivery is preferred in a well-staffed Obstetrics Department, especially when the delivery is complicated (e.g., breech presentations, prematurity, or a prolapsed umbilical cord).

EQUIPMENT

GENERAL SUPPLIES

- Electronic fetal monitor
- Sterile towels
- Povidone iodine or chlorhexidine solution
- Clock or timer watch
- Sterile perineal drapes
- Sterile gloves
- Needles, various gauges
- Syringes, various sizes
- Chromic suture, 2–0 and 4–0
- Vicryl suture, 2–0 and 3–0
- Bulb syringe
- Local anesthetic solution
- Clean towels or blanket for baby
- Umbilical cord clamp (**Figure 162-5**)
- Sterile scissors
- Infant warmer
- Infant resuscitation equipment

DELIVERY INSTRUMENT PACK

- Bandage scissors
- 4 towel clips
- 2 Allis forceps
- 4 ring forceps
- 6 straight Kocher clamps
- Straight Mayo scissors
- 2 suture scissors
- Adson forceps, or other forceps with teeth
- Russian forceps, 5.5 inches and 8 inches
- Gelpi retractor
- 2 small or medium Richardson retractors
- 2 Army-Navy retractors

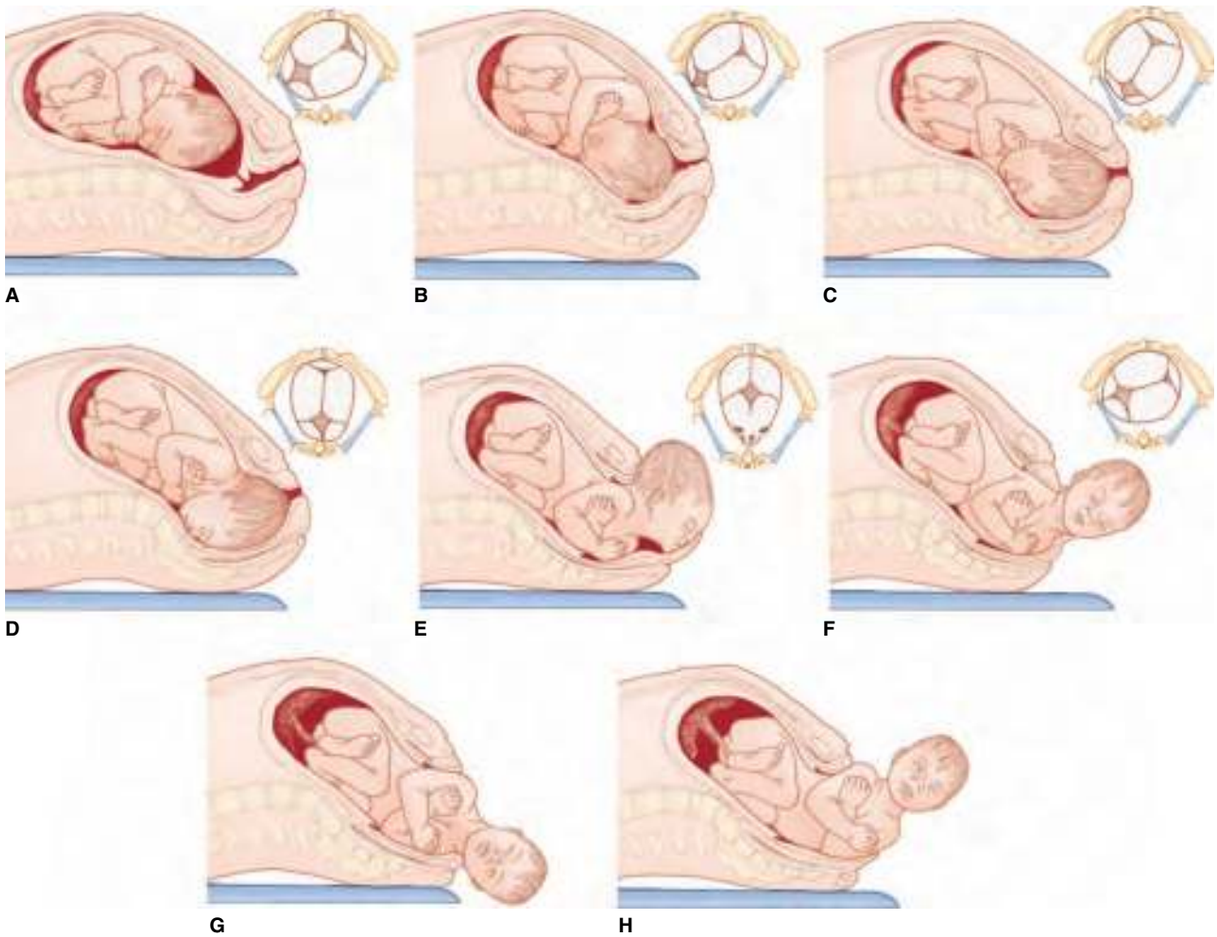

FIGURE 162-4. The fetal movements associated with the mechanism of labor. ***A.*** The fetal head is floating free. ***B.*** The head has descended and flexed to engage in the pelvis. ***C.*** The head descends further and internally rotates. ***D.*** The head has completely rotated into the direct occiput anterior position. The head begins to extend. ***E.*** The head completely extends to allow it to be delivered. ***F.*** External rotation of the head to bring it back to its natural position. ***G.*** Delivery of the anterior shoulder. ***H.*** Delivery of the posterior shoulder.

- 2 needle holders, 6 inches
- Placenta basin

Prepackaged obstetric kits are readily available and should be stocked in the Emergency Department. They contain all the required equipment and supplies. These kits may be prepared and packaged by the hospital. Commercially produced, disposable kits are available from several manufacturers.

FIGURE 162-5. An umbilical clamp.

INITIAL ASSESSMENT

Obtaining as thorough a maternal history as possible. **It is important as part of the initial assessment to identify whether the gravid patient is truly in labor, the imminence of delivery, and any potential associated problems.** Perform Leopold maneuvers, bedside ultrasonography, a sterile vaginal examination, an evaluation to detect whether or not spontaneous ROM has occurred, and fetal monitoring.

TRUE LABOR VERSUS FALSE LABOR

True labor consists of contractions at regular intervals and that gradually increase with intensity. The interval between contractions decreases as labor progresses. Contractions cause discomfort in the back and upper abdomen. Physical examination reveals that the fetus has descended and the cervix is actively dilating and effacing.

False labor is common in late pregnancy. It is characterized by contractions that are brief, occur at irregular intervals, and with constant intensity. These are often referred to as Braxton-Hicks contractions. These contractions primarily cause discomfort in the lower abdomen. Physical examination often reveals the fetus has not descended and the cervix is not dilated. False labor contractions can be relieved by hydration and sedation.

HISTORY AND PHYSICAL EXAMINATION

Obtain a thorough history if the circumstance allows. Include gravity and parity, estimated gestational age, route of delivery for previous pregnancies (i.e., vaginal or surgical), the length of previous labor, and description of the contractions (i.e., frequency, onset, and strength). Estimate the number of weeks pregnant or estimated date of confinement. Did the patient experience bloody show, ROM, or any problems during this pregnancy? Did the patient have access to prenatal care? Does the patient have any general surgical and medical history? Inquire about allergies, blood type, and infectious disease history. Include prior herpes infections and human immunodeficiency virus (HIV) status. It is important to send a rapid HIV test if the HIV status is unknown.

The Centers for Disease Control and Prevention guidelines suggest treating for group B streptococcal (GBS) infection if the GBS status of the laboring mother is unknown and she is ≤ 37 weeks of gestation, has ROM ≥ 18 hours, or has a temperature ≥ 38.0°C.[13] Treat mothers who have tested positive by GBS culture within 5 weeks of delivery, have had a previous newborn with GBS-invasive disease, or have a history of GBS bacteriuria during pregnancy.[13]

LEOPOLD MANEUVERS

Determine the fetal position and presenting part (i.e., vertex or breech) using the Leopold maneuvers (**Figure 162-6**). The fetus bends in late pregnancy so that its back is convex, the extremities and neck are sharply flexed, and the fetal arms are crossed across the chest (**Figure 162-6*A***). This posture is assumed so that the growing fetus can fit within the uterine cavity. The lie of the fetus, or its long axis in comparison to the mother, is either longitudinal or transverse. Most fetuses lie in the longitudinal plane at term. Presentation describes the fetal part that is closest to the vagina as felt through the cervix. This is usually the head, but possibly the buttocks or feet if in a longitudinal lie.

Approximately 97% of fetuses will present in the vertex position at full term. The Leopold maneuvers can help to determine the position of the fetus by identifying specific fetal landmarks or the spatial relationship between the fetus and the mother (**Figure 162-6**). These maneuvers can be imprecise, especially in the hands of the unfamiliar. These maneuvers are only 28% sensitive when compared with ultrasonography for the verification of the fetal position.[14] The use of the Leopold maneuvers has been largely replaced by bedside ultrasonography. They may still be useful when an Emergency Department ultrasound unit is not available.

The procedure involves multiple maneuvers, starting with the fundal grip to determine presentation. Stand at the patient's side facing her (**Figure 162-6*A***). Gently palpate the abdomen with the fingertips of both hands to determine which fetal part occupies the uterine fundus. The fetal head is firm, freely mobile, moves independent of the body, and is round and smooth. The fetal buttocks are less well-defined and can be felt as an irregular, large, and soft structure in the uterine fundus.

Perform the second maneuver or the umbilical grip. Stand at the patient's side facing her (**Figure 162-6*B***). Place both hands on either side of the abdomen. Palpate deeply, firmly, and gently to identify the back as a smooth hard surface and the extremities (i.e., the small parts) as small nodular parts. Note the lie of the fetus and the position of the back.

Perform the third maneuver or Pawlik's grip. Stand at the patient's side facing her (**Figure 162-6*C***). Place the thumb and middle finger of the dominant hand just above the pubic symphysis. Palpate to determine the presenting part and its relation to the fetal spine. The fetus will be in a vertex or occiput presentation if the cephalic prominence is palpable on the same side as the small parts (i.e., the head is flexed). The fetus will be in a forehead presentation if the cephalic prominence is palpable on the same side as the spine (i.e., the head is extended). The fourth maneuver must be performed to determine the presentation if the presenting part is fixed and deep within the pelvis.

The fourth maneuver or pelvic grip can determine the degree of flexion of the fetal head. Stand at the patient's side facing her feet (**Figure 162-6*D***). Place the tips of the thumb, index, and middle fingers over both sides of the lower abdomen. Palpate deeply, firmly, and gently toward the pelvic inlet with both hands. Determine the head position in relation to the fetal spine and small parts.

ULTRASOUND FOR FETAL PRESENTATION

Confirmation of vertex position by bedside ultrasound can be performed by placing the abdominal transducer transversely in the suprapubic region. Orient the transducer indicator directed to the maternal right side. Fan the transducer caudally to identify the presenting part. The fetal head will be low in the pelvis. The fetal skull will be visible as a hyperechoic, bright white oval image (**Figure 162-7**). Ensure that this is the fetal head rather than a cross-section of abdomen which can also appear as a bright white circular to oval-shaped image. Rotate the ultrasound transducer 90° to visualize the fetal skull in a second plane and visualize the fetal head articulating with the trunk.

STERILE VAGINAL EXAMINATION

A sterile vaginal examination is crucial to accurately assess a woman in labor. It will evaluate dilation, effacement, station, fetal position, and the presence of blood. **Always use sterile gloves to decrease the**

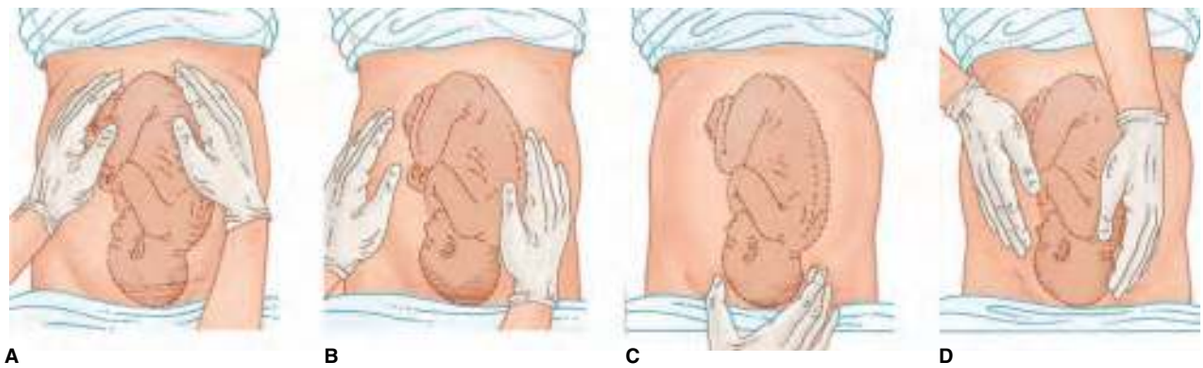

FIGURE 162-6. Leopold's maneuvers. ***A.*** The first maneuver. ***B.*** The second maneuver. ***C.*** The third maneuver. ***D.*** The fourth maneuver.

FIGURE 162-7. Transabdominal ultrasound of the lower abdomen of a gravid woman. The brightly echogenic fetal skull bones are visualized indicating a vertex presentation.

risk of infection. Vaginal bleeding that exceeds a bloody show is a contraindication to a digital vaginal examination.

Examine the cervix for dilatation and effacement. **Effacement is said to be 50% when the length of the cervix is one-half (i.e., approximately 2 cm long) that of an uneffaced cervix (i.e., approximately 4 cm long). It is said to be completely or 100% effaced when the cervix becomes as thin as the lower uterine segment. Dilation is an estimate of the cervical opening expressed in centimeters.** Place two fingers in the vagina and spread them apart until the walls of the cervix are appreciated. Use the distance between the fingers to determine the dilation of the cervix in centimeters. The cervix is completely dilated (i.e., approximately 10 cm) when no cervix can be palpated around the presenting part. Use caution in declaring the patient fully dilated once the cervix has dilated beyond the ability of the fingers to spread apart. **Take extra care to palpate the circumference of the presenting part.** Ensure that no cervix is appreciated by palpating the circumference of the presenting part (i.e., the occiput) to avoid falsely declaring a patient fully dilated.

Assessment of the station of the fetal head is vital for understanding how far the fetus has descended. It measures how many centimeters the fetal occiput is above or below the maternal ischial spines. The classification system ranges from −5 cm to +5 cm. Zero station is when the occiput reaches the level of the maternal ischial spines, negative station (i.e., −1 cm to −5 cm) when the fetal head is above the level of the maternal ischial spines, and positive station (i.e., +1 cm to +5 cm) when the fetal head is below the level of the maternal ischial spines.

DETECTION OF RUPTURED MEMBRANES

ROM can be determined by the speculum exam and specific testing. Look for fluid accumulation or pooling in the posterior vaginal fornix during the sterile speculum examination. Swab the pooled fluid and examine it for ferning, Nitrazine positivity, and the presence of meconium.

Determine the pH of the pooled fluid by touching a sample of the pooled fluid to pH or Nitrazine paper. Amniotic fluid is basic (i.e., pH 7.0 to 7.5), whereas the vagina and its secretions are acidic (i.e., pH 4.5 to 5.5). The pooled fluid is presumed to be amniotic fluid if the color of the Nitrazine paper changes from yellow to blue. Blood is also basic and may give a false-positive result if the sample is contaminated.

The final diagnostic test for ROM and the most specific way to identify ROM is the ferning test. Place a sample of the pooled fluid on a slide and allow it to air dry. Examine the slide under a microscope. The crystals that make up the amniotic fluid will arborize giving the appearance of fern leaves and thus a positive test if the fluid is amniotic fluid.

FETAL ASSESSMENT

The FHR is between 120 and 160 beats per minute. It can be assessed by auscultation, Doppler, or electronic fetal monitoring (EFM). The ACOG has issued guidelines regarding continuous fetal monitoring.[7] The false-positive rate of EFM for predicting an adverse outcome is high.[7] The use of EFM is associated with an increase in the rate of operative interventions (i.e., cesarean, forceps, and vacuum delivery) due to a high false-positive rate of EFM for predicting an adverse outcome.

EFM is unnecessary for most straightforward and low-risk deliveries but is standard practice.[7] An understanding of how to interpret the FHR is important.[7] The first step is interpreting the tracing for variability which reflects intact neuromodulation of the heart rate and that the cardiovascular system is robust. **Variability is the beat to beat irregularity in the tracing that gives the appearance of a wavy baseline.** Variability can be further characterized as absent, minimal, moderate, or marked. Absent variability shows no changes in amplitude from the baseline. Minimal variability shows ≤ 5 beats per minute (bpm) change. Moderate variability shows 6 to 25 bpm change. Marked variability shows ≥ 25 bpm change. Decreased variability is a sensitive finding and may represent benign processes (e.g., fetal sleep) or more concerning etiologies (e.g., acidosis and hypoxia).[11]

Evaluate the EFM tracing for any accelerations (**Figure 162-8**). These represent fetal movement and are defined as an increase above the baseline of 15 bpm lasting for 15 seconds. The presence

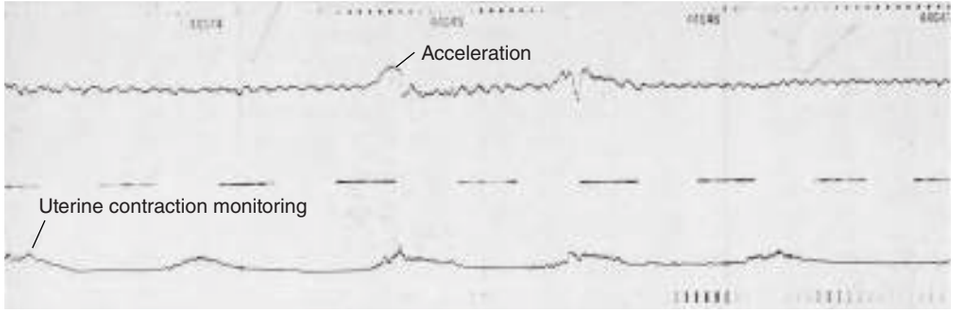

FIGURE 162-8. Acceleration recorded on the electronic fetal monitoring strip. (Reproduced with permission from Pearlman MD et al: *Obstetric & Gynecologic Emergencies: Diagnosis and Management*. New York: McGraw-Hill; 2004.)

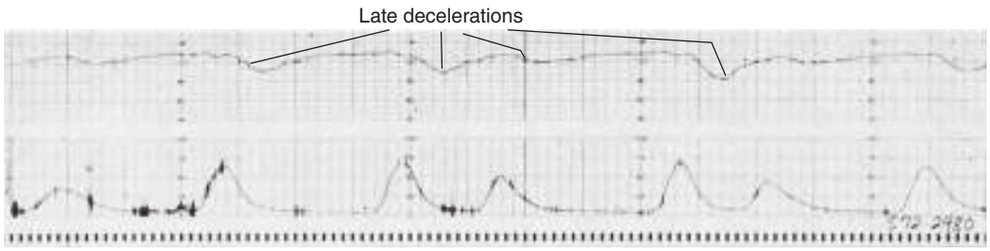

FIGURE 162-9. Late deceleration recorded on the electronic fetal monitoring strip. (Reproduced with permission from Pearlman MD et al: *Obstetric & Gynecologic Emergencies: Diagnosis and Management*. New York: McGraw-Hill; 2004.)

of accelerations provides reassurance that the fetus is healthy and withstanding the stress of labor.[11]

Evaluate the EFM tracing for decelerations. The two main types are late and variable decelerations. Late decelerations are a sign of fetal hypoxia. They are characterized as a gradual fall and rise, shallow, symmetric, and U-shaped (**Figure 162-9**). They usually begin 30 seconds after the onset of a contraction. The nadir of the deceleration occurs after the peak of the contraction. Variable decelerations are the most common type of deceleration encountered during labor and signify compression of the umbilical cord. They are variable in duration, shape, size, and timing relative to the contraction (**Figure 162-10**). Their onset and resolution are generally abrupt and sharp. Variable decelerations are usually preceded and followed by an acceleration.[11]

PATIENT PREPARATION

An important first step is to obtain the vital signs of the mother if time allows during a precipitous delivery. Assess FHR if fetal monitoring is available. Explain to the mother the indication for emergent delivery in the Emergency Department including the risks and benefits (**Table 162-1**). Explain what to expect, the procedures that may be performed, and attempt to incorporate maternal cooperation to accomplish a controlled delivery.[15,16] Obtain intravenous access. Send samples of blood and urine to the laboratory to determine a hematocrit, urine protein, blood type, Rh status, antibody screen, Venereal Disease Research Laboratory (VDRL), rapid HIV, and hepatitis screen. Do not give the patient anything to eat or drink once she enters the Emergency Department since gastric emptying is delayed in pregnancy.

The most commonly used maternal position for delivery is the dorsal lithotomy position (**Figure 162-11**). This position increases the diameter of the pelvic outlet. Place the patient supine with her legs in stirrups or leg holders. Be sure not to strap the legs so that quick flexion of the thighs can occur if shoulder dystocia presents. Cleanse the vulvar and perineal area with sterile soap, povidone iodine solution, or chlorhexidine solution if time permits. Apply sterile drapes to only expose the vulvar area (**Figure 162-11**). Full sterile technique is not required. Don sterile gloves, a sterile gown, and a mask with eye protection.

Perineal infiltration with a local anesthetic can safely and effectively provide pain relief during vaginal delivery. A pudendal nerve block is a minor regional block that provides adequate analgesia (**Figure 162-12*A***). Apply a needle guard onto the middle finger. Palpate the ischial spine through the vagina. Insert a 20 gauge needle between the index and middle finger (**Figure 162-12*B***). Advance the needle and pierce the sacrospinous ligament (**Figure 162-12*B***). Aspirate to ensure that the tip of the needle is not within a blood vessel. Inject 5 to 10 mL of local anesthetic solution. Repeat this procedure on the contralateral side.

The pudendal nerve block does not eliminate sensation from the anterior perineum. This area is supplied from branches of the genitofemoral nerve and ilioinguinal nerve. The pudendal nerve block does not eliminate uterine contraction pain. The uterus is supplied by sympathetic fibers from T12-L2. The uterus does not relax with a pudendal nerve block. Allow 5 to 20 minutes for the block to take effect.

A paracervical block can also be used to provide analgesia. It anesthetizes the Frankenhäuser ganglions that contain all the visceral sensory nerve fibers from the uterus, cervix, and upper vagina. It can only be used during the first stage of labor. Apply a needle guard onto the middle finger. Palpate the lateral vaginal fornix (**Figure 162-13**). Insert a 20 gauge needle along the middle finger (**Figure 162-13**). Advance the needle into the submucosa of the lateral vaginal fornix. Aspirate to ensure that the tip of the needle is not within a blood vessel. Inject 5 to 7 mL of local anesthetic solution. Repeat this procedure on the contralateral side. Paracervical block anesthesia has been associated with a relatively high

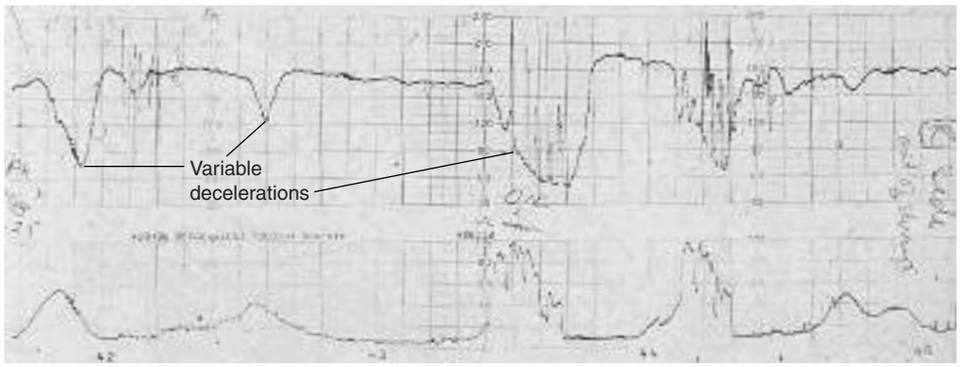

FIGURE 162-10. Variable decelerations recorded on the electronic fetal monitoring strip. (Reproduced with permission from Pearlman MD et al: *Obstetric & Gynecologic Emergencies: Diagnosis and Management*. New York: McGraw-Hill; 2004.)

TABLE 162-1 Some of the Complications Associated with Delivery in the Emergency Department

Allergic reaction to medications used
Blood loss
Brain damage
Cardiac arrest
Damage to adjacent structures
Death
Disfigurement
Fistula formation
Infection
Lacerations
Need for hysterectomy
Need for organ removal
Need for surgery
Paralysis
Paraplegia
Pelvic relaxation
Painful intercourse
Scarring
Shock
Sterility
Urinary incontinence
Uterine rupture
Vascular emboli

FIGURE 162-11. Patient positioning and sterile drape placement.

FIGURE 162-13. The paracervical nerve block.

incidence of fetal bradycardia, usually developing 2 to 10 minutes after the block.

TECHNIQUE

DELIVERY OF THE INFANT

Coaching may be required for optimal pushing efforts. Flex the patient's legs at the hips and knees to increase and improve pushing efficacy. Encourage the patient to take a deep breath at the onset of a contraction and then push downward or Valsalva. Advise rest between contractions as she and the fetus need to recover

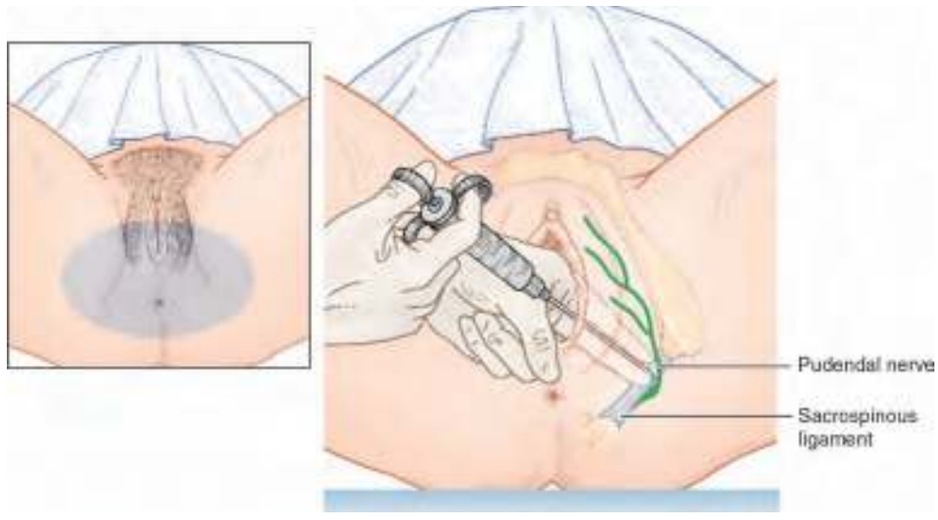

FIGURE 162-12. The pudendal nerve block. ***A.*** The area of sensory innervation of the pudendal nerve. ***B.*** Infiltration of local anesthetic solution.

from the effects of breath holding, muscular effort, and uterine contractions. The perineum will bulge as the fetus descends into the maternal pelvis. Sponge downward any expelled stool with a sterile towel or piece of gauze. The patient and the fetus are prepared for delivery when the scalp of the fetus becomes visible at the introitus.

The fetal head becomes increasingly visible at the introitus as labor progresses. Apply gentle downward pressure on the occiput to aid in the controlled delivery of the head once the bony occiput can be seen and palpated at the vaginal introitus. The fetal head stretches the vaginal outlet and the vulva until they encircle the largest diameter of the fetal head, known as crowning. Gentle digital stretching of the inferior portion of the perineum may aide delivery. The routine use of an episiotomy for delivery has been discouraged due to increases in third-degree and fourth-degree lacerations.[17] **Perform an episiotomy when the baby is crowning if there appears to be inadequate stretching of the perineum (Chapter 163).**

Apply manual perineal support and control the head by performing the modified Ritgen maneuver as the fetal head emerges from the introitus (**Figure 162-14**). Apply a towel-draped hand to protect against fecal contamination. Apply pressure on the fetal chin through the perineum anterior to the maternal coccyx. Place the other hand on the fetal occiput to hold the suboccipital region against the maternal pubic symphysis. It was thought that this maneuver helps to extend the fetal neck, ease the delivery of the fetus, and reduce the incidence of third-degree and fourth-degree perineal lacerations. A study did not show a decrease in perineal lacerations with this technique.[18] **It is important to control the speed at which the fetal head delivers and ease the pressure on the perineum to decrease perineal tears.**

Encourage less forceful pushing after crowning occurs to control delivery of the head and to minimize perineal trauma. Slowly deliver the fetal head using the modified Ritgen maneuver.[8,11] The base of the occiput will rotate, or restitute, toward the posterior aspect of the maternal pubic symphysis while the fetal brow, face, and chin pass over the maternal perineum. Instruct the mother to stop pushing once the head is delivered. Gently rotate the infant's head slightly if it does not spontaneously rotate. **The routine intrapartum and peripartum suctioning of the nasal cavities and oropharynx of infants with or without meconium-stained amniotic fluid is no longer recommended.**[19-22]

FIGURE 162-14. The modified Ritgen maneuver. A hand covered with a sterile towel applies moderate upward pressure on the perineum between the anus and the introitus. The other hand extends the fetal head to maintain the suboccipital region against the maternal pubic symphysis. This maneuver assists in the delivery of the fetal head.

FIGURE 162-15. Slip any loops of umbilical cord that are wrapped around the fetal neck over the head.

Pass one hand around the neck of the fetus to assess for a nuchal cord which occurs in approximately 25% of deliveries.[8,11,12] **Unwind the umbilical cord from the fetal neck prior to continuing with the delivery.**[12] Slip it over the infant's head if possible (**Figure 162-15**). The umbilical cord can occasionally be wrapped so tightly around the fetal neck that it cannot be slipped over the head. **Carefully grasp and clamp the umbilical cord between two hemostats or umbilical clamps placed 1 to 2 cm apart. Carefully cut the umbilical cord between the two clamps with sterile scissors. Unwind the umbilical cord from around the fetal neck. Immediately deliver the fetus as it no longer can rely on the maternal circulation once the umbilical cord is clamped and cut.**

Deliver the body of the fetus (**Figure 162-16**). Gently grasp the sides of the fetal head with two hands (**Figure 162-16**). Apply steady and gentle downward traction until the anterior shoulder appears under the maternal pubic symphysis (**Figure 162-16*A***). Apply steady and gentle upward traction until the posterior shoulder is delivered (**Figure 162-16*B***). **The use of gentle traction with both of these maneuvers is important to avoid brachial plexus injuries.**

The rest of the infant will spontaneously deliver once the shoulders are delivered. **It is still very important to control the delivery of the body to prevent maternal perineal lacerations.** A combination of amniotic fluid, blood, and vernix makes delivery of the infant very slippery. Proper hand positioning is important during the delivery of the body. Position the posterior or left hand underneath the infant's axilla prior to delivering the rest of the body. Use the anterior or right hand to grasp the infant's ankles as they are delivered. This ensures a firm grip on the infant.

Place two hemostats or umbilical clamps on the umbilical cord approximately 4 to 5 cm from the infant and 1 cm apart. Cut the

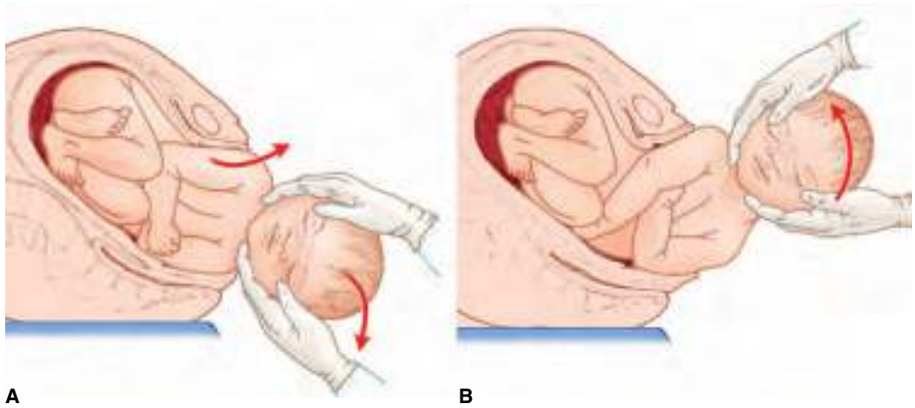

FIGURE 162-16. Delivery of the body. Grasp the head with both hands. ***A.*** Apply gentle and steady downward traction to deliver the anterior shoulder. ***B.*** Apply gentle and steady upward traction to deliver the posterior shoulder.

umbilical cord between the two clamps using sterile scissors. Obtain a 10 to 20 mL sample of umbilical cord blood from the placental end for cord blood pH to determine fetal acid-base status and other required tests.[19]

It is now recommended to delay umbilical cord clamping for at least 30 to 60 seconds after birth in term and preterm uncomplicated deliveries.[20,23] This has been found to increase hemoglobin levels at birth and improve iron stores in the first several months of life. This may have a favorable effect on developmental outcomes. It is associated with significant neonatal benefits in preterm infants including improved transitional circulation, better establishment of red blood cell volume, decreased need for blood transfusion, and lower incidence of necrotizing enterocolitis and intraventricular hemorrhage. There is no increased risk of postpartum hemorrhage. There is a small increase in the incidence of jaundice that requires phototherapy in term infants undergoing delayed umbilical cord clamping. This requires that the Emergency Physician ensure that mechanisms are in place to monitor and treat neonatal jaundice.[20]

Dry the infant while simultaneously stimulating and assessing for appropriate physiologic responses. The mother may immediately hold the infant and breastfeed, if desired, while the umbilical cord is being cut in an uncomplicated birth. The infant should respond well to initial stimulation and have an adequately clear airway and good respiratory effort. The infant can be further dried and stimulated in a warm incubator if necessary. **Calculate the APGAR scores at 1 minute and 5 minutes after delivery.** Repeat the APGAR score every 5 minutes in the immediate postpartum period if the APGAR score is low at 5 minutes. The acronym **APGAR** was coined as a mnemonic learning aid. It stands for **A**ppearance (skin color), **P**ulse (heart rate), **G**rimace (reflex irritability), **A**ctivity (muscle tone), and **R**espiration (**Table 162-2**).

TABLE 162-2 The APGAR Score

Sign	Score = 0	Score = 1	Score = 2
Skin color (**A**ppearance)	Blue or pale	Pink body and blue extremities	Completely pink
Heart rate (**P**ulse rate)	Absent	Slow (< 100)	> 100
Reflex irritability (**G**rimace)	No response	Grimace	Vigorous crying
Muscle tone (**A**ctivity)	Flaccid	Some extremity flexion	Active motion
Respiratory effort (**R**espiration)	Absent	Slow or irregular	Good and crying

Note: Add the scores for each of the five signs to obtain the infant's APGAR score. The maximal possible score is 10. Scores less than 7 are considered low.

MATERNAL AND FETAL MONITORING

The ACOG recommends that providers evaluate and record the FHR at least every 30 minutes in the active phase of the first stage of labor and every 15 minutes in the second stage in patients without complications or risk factors.[7] The ACOG recommends continuous FHR monitoring if the delivery is complicated (e.g., suspected fetal growth retardation, preeclampsia, and type 1 diabetes).[7] Evaluate and record the FHR at least every 15 minutes in the active phase of the first stage of labor and every 5 minutes in the second stage if continuous monitoring is not possible.[7]

Suspect fetal distress if the FHR following a contraction is repeatedly below 120 bpm. Place the mother on oxygen, move her to the left lateral decubitus position, and check for umbilical cord prolapse in the absence of vaginal bleeding if fetal distress is appreciated. FHR decelerations may occur with contractions as the head descends. Allow labor to continue if prompt recovery occurs as the contraction diminishes. Decelerations may also occur from progressive compression of the umbilical cord around the fetus, premature placental separation, or reduction in uterine blood flow.[12] These can lead to fetal compromise.[12] Prolonged FHR decelerations are worrisome and warrant action (e.g., supplemental maternal oxygen, repositioning of the mother, and immediate delivery of the baby by cesarean section).[7]

The intensity of a contraction is determined by the degree of firmness that the uterus achieves. The frequency of contractions may be palpated or monitored by an external transducer (i.e., tocodynamometer) placed upon the mother's abdomen. **Remember to monitor the maternal vital signs during active labor and intervene as needed when abnormal vital signs present themselves.**

DELIVERY OF THE PLACENTA

Watchful waiting until the placenta separates from the uterus is an acceptable practice if there is no unusual bleeding after delivery of the infant. Watch for the signs of placental separation during expectant management. These include a firm and globular uterus, a sudden gush of blood, uterine elevation in the abdomen, and a lengthening of the umbilical cord. This usually occurs within 5 to 10 minutes after delivery of the infant. It may take as long as 30 minutes. Times longer than 30 minutes raise concern for a retained placenta.

Some may choose to actively manage the third stage of labor by clamping the umbilical cord, applying controlled umbilical cord

traction, and administering a uterotonic drug (e.g., oxytocin) to stem postpartum hemorrhage.[24,25] The literature is mixed with respect to whether active management of labor is beneficial when compared to expectant management.[24-26] **Pulling too hard on the umbilical cord can invert the uterus and make postpartum hemorrhage worse.**[27]

Instruct the patient to bear down once the placenta has separated. This results in increased intraabdominal pressure that aids in delivery of the placenta. Apply suprapubic pressure (**Figure 162-17*A***). Apply gentle traction on the umbilical cord to keep it taut without the use of excessive traction (**Figure 162-17*A***). **Aggressive traction on the umbilical cord can result in disruption of the placenta, uterine inversion, or tearing of the umbilical cord.**[27] **All these can result in excessive bleeding.** Applying suprapubic pressure as the placenta exits the uterus helps to prevent uterine inversion (**Figure 162-17*B***). Deliver the placenta from the uterus (**Figure 162-17*C***). Take care to prevent tearing of the placenta and its membranes as it passes through the introitus. Grasp the placenta and membranes with hands or ring forceps and guide them out the vagina with gentle twisting traction (**Figure 162-17*D***). Gently massage the uterus through the anterior abdominal wall.[17,28] **Carefully examine the maternal surface of the placenta for completeness. Any missing pieces represent retained products and must be removed.**

ALTERNATIVE TECHNIQUES

Operative vaginal delivery involves the use of forceps or vacuum extractors. This mode of delivery is sometimes controversial but it can be a safe and effective technique for delivery.[8,11] The use of forceps and vacuums is beyond the scope of this chapter.

ASSESSMENT

Vaginal lacerations are classified as first, second, third, or fourth degree.[8,11] First-degree lacerations involve the fourchette, perineal skin, and vaginal mucous membrane but not the underlying fascia or muscle. Second-degree lacerations involve the above plus the fascia and muscles of the perineal body but not the anal sphincter. Third-degree lacerations involve the anal sphincter and fourth-degree lacerations extend through the anal mucosa. The repair of vaginal lacerations is beyond the scope of this chapter. Refer to Chapters 163 and 166 for the complete details regarding laceration repair. Resuscitate the mother if necessary. This includes positioning on the left side in Trendelenburg position.[29]

Assess the infant. Determine the APGAR score (**Table 162-2**). Repeat this every 5 minutes until the infant is stable. Place the infant in a warmer. Warm, dry, and stimulate the infant, and resuscitate as necessary.[1,2,30,31] Resuscitate the infant immediately and do not stop

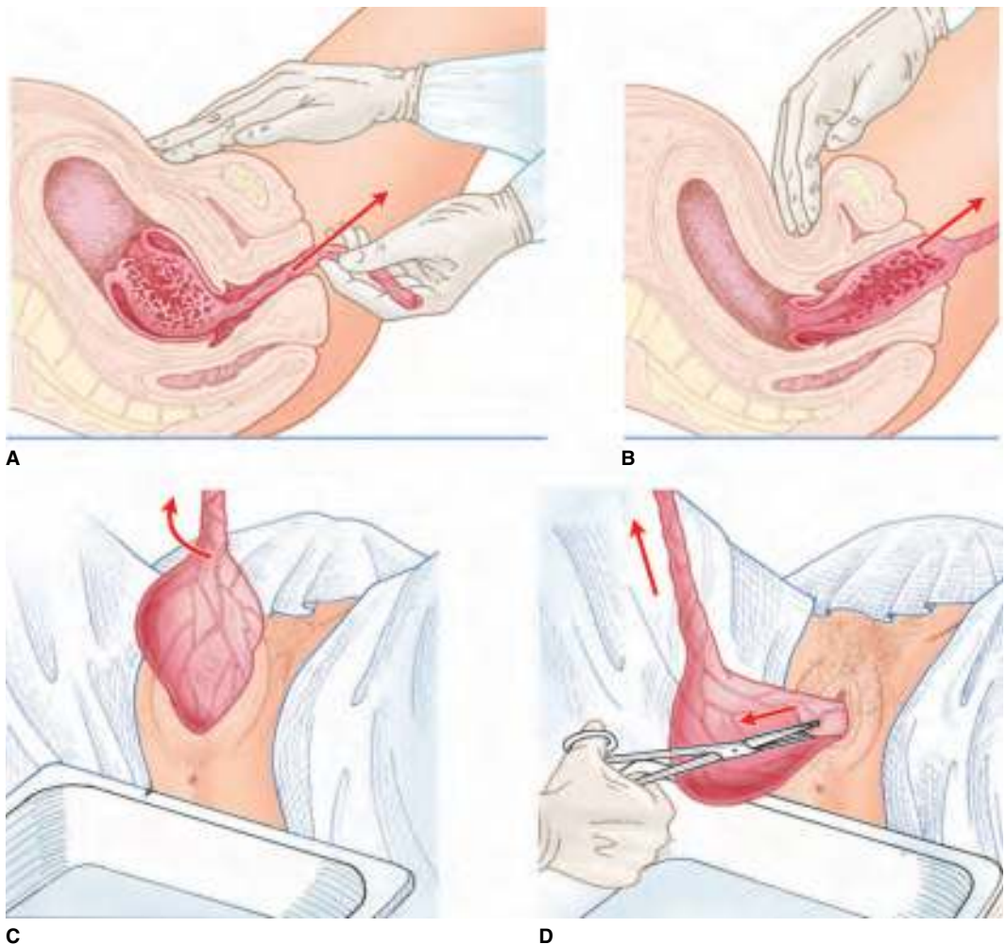

FIGURE 162-17. Delivery of the placenta. ***A.*** Apply gentle traction on the umbilical cord. ***B.*** Apply suprapubic pressure to help expel the placenta and prevent inversion of the uterus. ***C.*** Delivery of the placenta. ***D.*** Torn membranes or placenta are grasped with a ring forceps and delivered.

until the decision is made that the infant is not viable. This may include suction, intubation, chest compression, vascular access, and tube thoracotomy.

AFTERCARE

The hour immediately following delivery is a critical time and often referred to as the fourth stage of labor. The mother is at risk for hemorrhage from uterine atony or lacerations of the birth canal. **Investigate any unusual bleeding immediately**. Administer 20 units of oxytocin in 1 L of normal saline intravenously to prevent uterine atony and postpartum hemorrhage. Persistent vaginal bleeding can be managed with additional intravenous oxytocin, 0.2 mg of methylergonovine (Methergine) intramuscularly, or 0.2 mg of prostaglandin F2-alpha (Hemabate) intramuscularly (Chapter 166).

COMPLICATIONS

Numerous complications can result during the delivery of an infant. These may affect the infant, the mother, or both.

UMBILICAL CORD PROLAPSE

Do not remove the hand if the initial vaginal examination reveals a prolapsed umbilical cord. Elevate the fetal presenting part away from the prolapsed umbilical cord and place the patient in the Trendelenburg position. This reduces compression of the umbilical cord and optimizes blood flow to the fetus. **Immediately transport and prepare the patient for an emergent cesarean section while keeping the hand in place.**

SHOULDER DYSTOCIA

The shoulders may occasionally impact at the pelvic outlet after delivery of the head. This is more likely to occur in large infants who have larger shoulders compared to their head circumference. Complications of shoulder dystocia include brachial plexus injuries, fetal hypoxia, and umbilical cord compression. Place the mother in the extreme lithotomy position with her legs sharply flexed up to the abdomen (i.e., McRoberts maneuver). An episiotomy may be made to assist in the delivery (Chapter 163). Instruct an assistant to apply suprapubic pressure to help disimpact the anterior shoulder from the pubic symphysis. Refer to Chapter 164 for the complete details regarding the management of shoulder dystocia.

BREECH PRESENTATION

The breech presentation is associated with a higher incidence of fetal distress and umbilical cord prolapse. It usually occurs in premature infants as the final rotation in the uterus may have not yet occurred. The major concern of breech deliveries is entrapment of the head because of an incompletely dilated cervix. **Do not pull on the fetus. This may put additional pressure on the head or further entrap an extremity. Obtain immediate Obstetrical assistance**. Refer to Chapter 165 for the complete details regarding breech deliveries.

PRETERM DELIVERY

Delivery of the preterm fetus must be controlled and slow to reduce the likelihood of trauma to the fragile infant. Immediately dry and warm the preterm infant while making an initial assessment. Begin resuscitation even for extreme prematurity until the determination of viability is made.

POSTPARTUM HEMORRHAGE

Significant bleeding can occur from cervical lacerations, uterine atony, retained products, or first-degree through fourth-degree lacerations.[17] Perform a thorough search for the cause of the postpartum hemorrhage. Refer to Chapter 166 for the complete details regarding the evaluation and management of postpartum hemorrhage.

INJURIES TO THE INFANT

A variety of injuries can occur to the fetus during the delivery process. These include abrasions, brachial plexopathies, bruising, clavicle fractures, cephalohematomas, femur fractures, humerus fractures, intracranial hemorrhage, lacerations, nerve injuries, skull fractures, spinal cord injuries, and visceral injuries. Most of these can be prevented by using appropriate techniques and care when delivering the infant.

SUMMARY

The Emergency Physician will occasionally be required to perform a delivery of a baby when the Obstetrician or Family Physician is not available or when delivery is imminent. Fortunately, births in the Emergency Department are rare and most proceed with good outcomes. Knowledge of the normal labor and delivery mechanics aids in a safe delivery and helps to identify complications. The Emergency Physician must develop strategies to treat potential complications and must be prepared to intervene. It is important to remember there are two patients.

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163 Episiotomy

Francisco Orejuela and Padraic Chisholm

INTRODUCTION

An episiotomy is a surgical incision of the female perineum performed at the time of delivery to increase the diameter of the soft tissue pelvic outlet and facilitate a vaginal delivery. It is one of the most commonly performed surgical procedures in women in the United States yet it is controversial.

The episiotomy was thought to provide protection to the female genital tract and to facilitate an easier vaginal repair compared to a spontaneous laceration.[1,2] It is thought to prevent perineal tearing by substituting a straight surgical incision for a ragged spontaneous laceration that may have a worse outcome after repair. It was also believed that an episiotomy resulted in decreased postoperative pain and improved healing when compared to a tear. These original beliefs have been challenged. There is a growing body of evidence demonstrating increased injury to the pelvic floor with the routine use of an episiotomy.[3-6] A Cochrane database review of eight randomized controlled trials concluded that a restrictive use of an episiotomy lowers the incidence of posterior perineal trauma, decreases the amount of suturing, and results in fewer complications as compared to routine episiotomy.[7] Current recommendations favor a restrictive use of episiotomy.[8,9] The rate of episiotomy has steadily declined in the recent decades. As an example, the rates of episiotomy between 2006 and 2012 decreased from 17.3% to 11.6% in a cohort of 2.3 million women.[10]

There is a place for the episiotomy in modern obstetrics. The indications for an episiotomy today are based primarily on the clinical situation at the time of delivery (e.g., need to expedite delivery for suspected non-reassuring fetal status). Another important reason to make an episiotomy is the prevention of a long and irregular spontaneous perineal laceration. The repair of a controlled surgical incision might be easier and the anatomic planes easier to recognize. Good clinical judgment is still the best guide to use to determine whether or not to proceed with an episiotomy.[11]

ANATOMY AND PATHOPHYSIOLOGY

ANATOMY OF THE PERINEUM

The perineum is a diamond-shaped region bounded by the bony structures of the pelvic outlet (i.e., the pubic symphysis, inferior pubic rami, and coccyx). An important anatomic area in obstetrics is the perineal body. It is located toward the center of the perineum between the lower vagina and the anus. The most critical area of the perineum is the distance from the vestibular fossa to the anus. It is usually 3 to 4 cm in length in the nonpregnant woman.[11,12] The perineal body is a complex fibromuscular mass into which many structures insert.

The perineal body is the center of the hub of a wheel that includes the transverse perineal muscles, the capsule of the external anal sphincter muscle, and the bulbospongiosus muscle (**Figure 163-1**). The perineal body attaches to the ischial tuberosities and to the inferior pubic rami through the perineal membrane and superficial transverse perineal muscles. The bulbospongiosus muscles are located laterally to the introitus and deep to the labia majora. They insert into the inferior limit of the perineal body with the superficial transverse muscles and the subcutaneous portion of the external anal sphincter. The bulbospongiosus muscles are attached laterally to the muscles of the pelvic diaphragm. The perineal body is anchored posteriorly to the coccyx by the anal sphincter and anococcygeal ligament. The mediolateral episiotomy transects the superficial muscles of the perineum whereas the midline episiotomy does not. The pudendal nerve supplies most of the innervation to the perineal body. This nerve is formed from the ventral rami of sacral spinal nerves two through four. The arterial supply is primarily derived from the internal pudendal artery, one of the branches of the anterior trunk of the internal iliac artery.[11]

TYPES OF EPISIOTOMY

An episiotomy is a surgical enlargement of the posterior vagina by an incision into the perineum and may be performed in the midline or mediolaterally (**Figure 163-2**).[13-17] The choice between the two types of episiotomy is largely dependent upon the experience of the Emergency Physician. Factors that influence type of episiotomy include the site of prior episiotomies, position of the presenting fetal part, the thickness or rigidity of the patient's perineum, and the increased likelihood of a severe perineal laceration (e.g., in patients with a short perineal body and the use of forceps for vaginal delivery). A mediolateral incision may be prudent when an extended

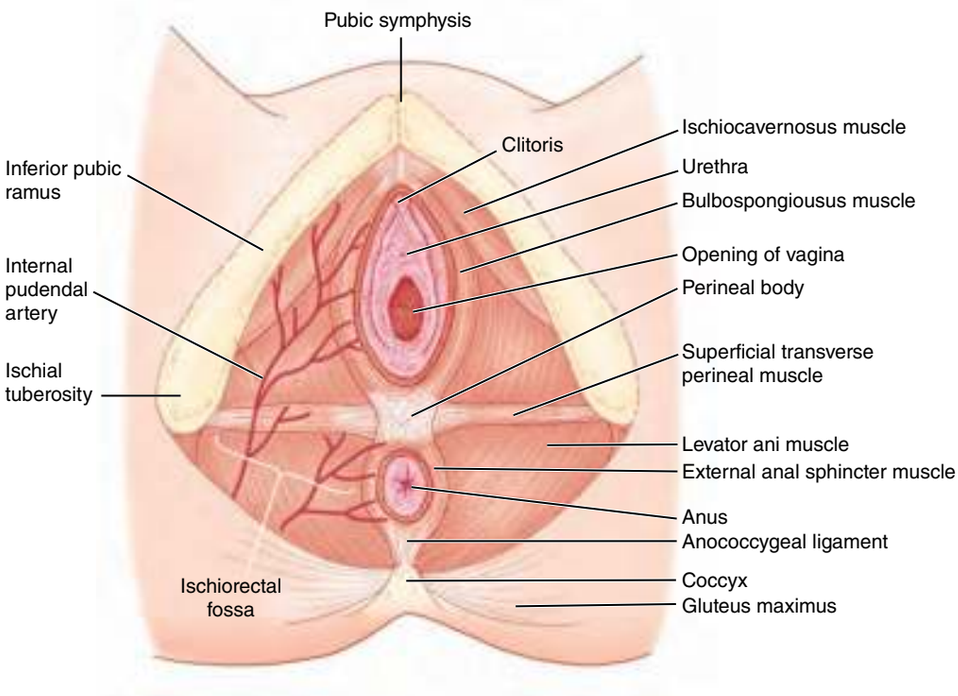


FIGURE 163-1. The anatomy of the perineum. The skin and subcutaneous tissues have been removed.

episiotomy is required or when the risk of a fourth-degree laceration is significant.[18-20] **Never perform any other episiotomy incision in the Emergency Department. These are associated with significant complications.**[14] Reserve the numerous other episiotomy incisions for the Obstetrician.[21]

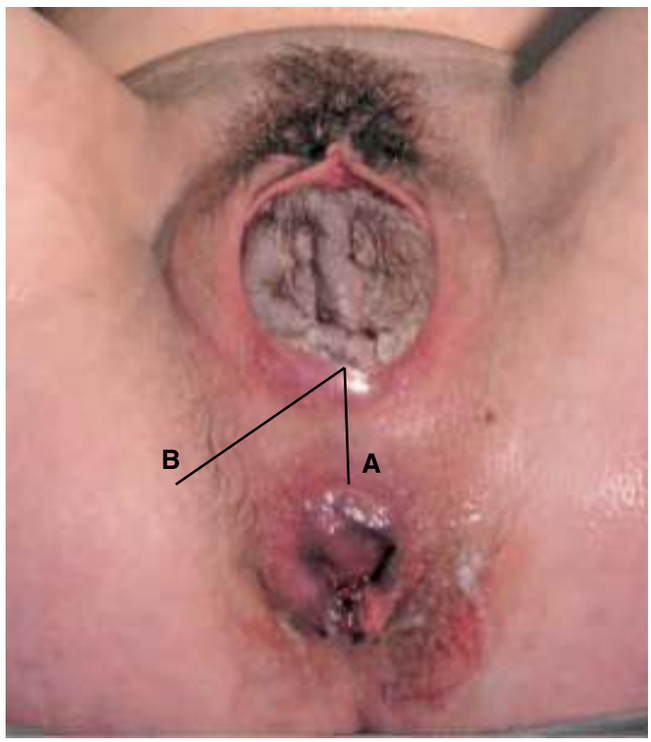


FIGURE 163-2. The types of episiotomies for the Emergency Physician. ***A.*** Midline. ***B.*** Mediolateral. (Photo used with permission from reference 13.)

MIDLINE EPISIOTOMY

The midline episiotomy is a surgical incision made in the midline of the perineal body starting from the vaginal orifice toward the anus (**Figure 163-2*A***). The incision transects the central tendinous portion of the perineal body and usually extends to the level of the transverse perineal muscle (**Figure 163-1**). The incision should run halfway between the posterior fourchette and anus. This is the most common type of episiotomy performed in the United States. The midline episiotomy is easy to perform and to repair. It is associated with quicker healing, less pain, less postpartum discomfort since no muscle belly is transected, quicker return to sexual intercourse, and less blood loss. There is an increased risk of third-degree and fourth-degree extensions with the midline episiotomy when compared to mediolateral episiotomies.

MEDIOLATERAL EPISIOTOMY

The mediolateral episiotomy is the preferred technique outside the United States.[22] The mediolateral episiotomy is a surgical incision made from the midline of the posterior vaginal orifice obliquely toward the ischial tuberosity (**Figure 163-2*B***). The anatomic structures transected from superficial to deep are the skin, the subcutaneous tissues, the bulbospongiosus muscle, the superficial transverse perineal muscle, and a portion of the levator ani muscle and fascia.

The mediolateral episiotomy is the preferred technique when considering an operative vaginal delivery. It is associated with a decreased risk of extension to the anal sphincter which is associated with fecal incontinence.[23-25] The disadvantages of the mediolateral episiotomy include increased blood loss, increased postpartum discomfort, faulty healing, anatomic deformities, and dyspareunia.[6,18-20] The adequate surgical repair of these structures requires an understanding of the perineal anatomy and represents a higher degree of difficulty to repair.

PERINEAL LACERATIONS

The classification of perineal lacerations most often used is quite simple.[26] A first-degree laceration is a superficial laceration of the vaginal epithelium not requiring suturing. A second-degree laceration or episiotomy involves the vaginal epithelium and the perineal body but does not include the anal sphincter complex (i.e., external anal sphincter and internal anal sphincter). It typically requires suture closure. The third-degree laceration or episiotomy is an extension of the laceration to involve any part of the anal sphincter complex. The fourth-degree laceration or episiotomy is an extension to the anal sphincter complex and anal mucosa.

INDICATIONS

The rate of episiotomy decreases as one travels the continuum from Obstetrician to Family Practitioner to Certified Nurse Midwife to lay Midwife. The indications for episiotomy are more largely based on expert consensus than evidence-based criteria. The indications depend on the clinical picture at the time of delivery.[27] Most practitioners consider the performance of an episiotomy to be appropriate in situations of fetal distress or maternal disease requiring urgent delivery.[8] Deliveries necessitating greater space accommodation for effective delivery are also indications for an episiotomy. These situations include shoulder dystocia, breech delivery, or operative vaginal deliveries.[27-29] Mediolateral episiotomies are the preferred method to avoid extension into the anal sphincter for operative deliveries.

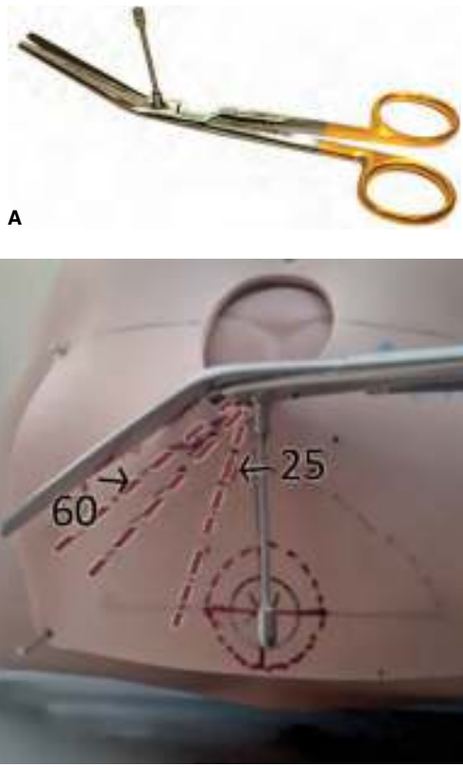
CONTRAINDICATIONS

Contraindications to performing an episiotomy include rectal or perineal lesions, prior or concurrent fistulae, inflammatory bowel disease, or prior anorectal surgery.[30] Maternal diseases that impair healing such as autoimmune disorders (e.g., systemic lupus erythematosus, rheumatoid arthritis), HIV, pregestational diabetes mellitus, and immunosuppression are relative contraindications to an episiotomy. Some authors suggest that coagulation disorders may be contraindications; however, a vaginal delivery with an episiotomy is preferable to a cesarean delivery.[14]

EQUIPMENT

- Local anesthetic solution (e.g., lidocaine or bupivacaine)
- Alcohol wipes
- 16 to 18 gauge needle
- 10 mL syringe
- 22 to 25 gauge needle
- Mayo scissors
- #10 scalpel blade on a handle
- Povidone iodine or chlorhexidine solution
- Sterile drapes
- Sterile gown and gloves
- 2–0 polyglactin suture (i.e., Vicryl)
- 2–0 or 3–0 polydioxanone (i.e., PDS) and 3–0 Vicryl
- Needle driver
- 23 gauge spinal needle for possible pudendal nerve block

The incision of 60° is required for the achievement of a 45° postdelivery angle due to the perineal distention from crowning.[24] The recommended safest cutting angle is 60°.[9] Midwifes and doctors perform a true mediolateral incision in most cases.[31,32] The Emergency Physician does not have much experience making an episiotomy incision. It can be expected that the Emergency Physician will not make an incision at the correct angle. Approximation of or eyeballing the incision does not work in most cases. The Episcissors-60 (Medinvent Ltd, United Kingdom) solves this problem (**Figure 163-3**).[33-35] The guide limb is positioned at the anus. The blades are fixed at a 60° angle from the guide limb. Sutured angles after delivery are between 40° and 52°.[33-35] These special scissors are not often found in the Emergency Department. Hopefully, they will soon be contained in the hospital delivery kit.



A

B

FIGURE 163-3. The Episcissors-60. ***A.*** The scissors. ***B.*** Use on a manikin. (Photos courtesy of D. Kapoor, MD.)

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Clean the perineum of any dirt, debris, stool, and urine. Apply povidone iodine or chlorhexidine solution and allow it to dry. Place sterile drapes beneath the buttocks, over the legs, and on the abdomen to prevent contamination from nonsterile areas.

Anesthetize the perineal body (**Figure 163-4*A***). Infiltrate 10 to 20 mL of local anesthetic solution directly into the perineal body from the posterior base of the vaginal fourchette to the anus (**Figure 163-4*A***). This provides safe and effective anesthesia. It may be carefully performed if the baby's head is within the vagina or crowning. The application of EMLA cream provides equal analgesia to the injection of lidocaine.[36] The time required for EMLA cream to work (Chapter 154) is usually not available for deliveries in the Emergency Department.

Alternatively, a bilateral pudendal nerve block will provide excellent pain control (**Figure 163-4*B***). It requires more time to perform and cannot be done if the baby's head is in the vagina. Arm a 10 mL syringe with a 23 gauge spinal needle and filled with local anesthetic

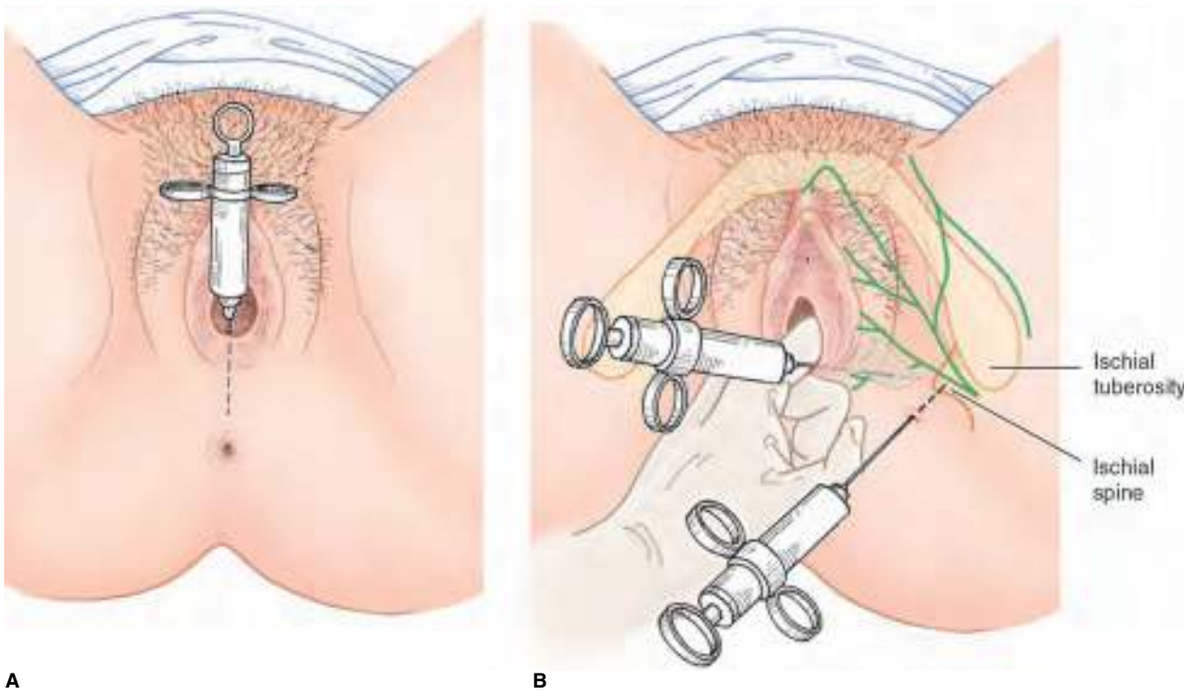


FIGURE 163-4. Anesthesia of the perineum. ***A.*** Local anesthetic infiltration subcutaneously from the posterior fourchette to the anus. ***B.*** The pudendal nerve block.

solution. Place the nondominant index finger in the vagina and palpate the ischial spine. The ischial spines are identified as the bony protrusions located in the posterolateral region of the vaginal side wall. The sacrospinous ligament is a firm band that runs from the ischial spine to the sacrum. The pudendal nerve and artery lie between the ischial spine and ischial tuberosity. Palpate the ischial tuberosity with the nondominant thumb.

To perform the block on the maternal right side, use the right index and middle fingers to palpate the right ischial spine and guide the spinal needle. Use the left hand to introduce the needle. Insert the tip of the needle two finger breadths medial to the ischial spine (**Figure 163-4*B***). Advance the needle through the sacrospinous ligament. Aspirate to ensure that the injection is not intravascular. Inject 3 to 4 mL of local anesthetic solution. Advance the needle a few millimeters, aspirate, and then inject another 3 to 5 mL of local anesthetic solution. Repeat the procedure on the contralateral side. Occasionally, a cutaneous branch of the inferior anal nerve can innervate the area surrounding the anus. The infiltration of a local anesthetic solution will be required.[11]

A pudendal nerve block is usually performed intravaginally by the Obstetrician (**Figure 163-4*B***). This increases the risk of an Emergency Physician sticking themselves with the needle. It may be easier to perform the block subcutaneously (**Figure 163-4*B***). The decision is physician dependent.

TECHNIQUES

MIDLINE EPISIOTOMY

Make the incision vertically in the midline of the perineal body from the midpoint of the posterior vaginal orifice to halfway toward the anus (**Figure 163-5**). The incision length depends upon the perineal length. **Make it of sufficient length to increase the area of the introitus for successful delivery of the presenting part without**

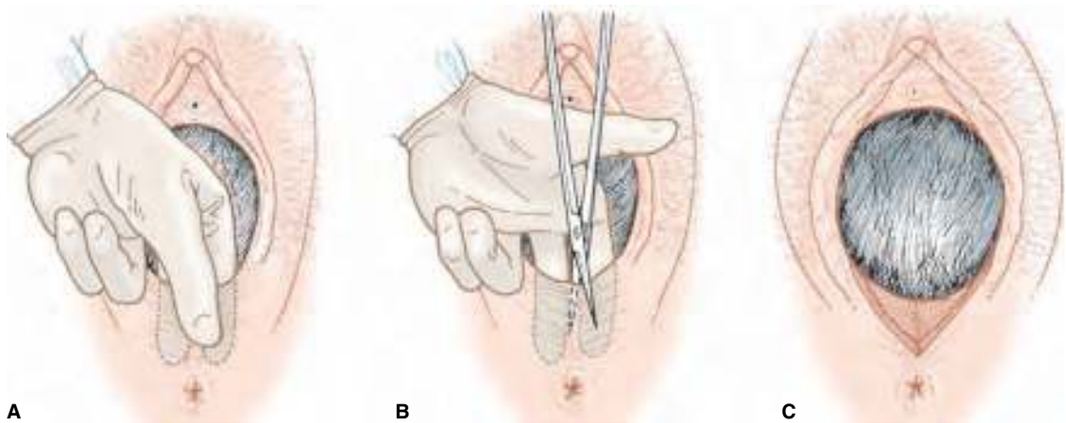


FIGURE 163-5. Performing a midline episiotomy. ***A.*** Place the nondominant index and middle fingers inside the introitus with the palm facing outward. ***B.*** Pull the perineal body away from the fetus and insert the straight Mayo scissors. Note that the blades of the Mayo scissors are positioned between the fingers. ***C.*** The introitus readily opens after making the episiotomy.

compromising the anal sphincter. The incision must include the tendinous central portion of the perineal body. The most important aspect of the midline episiotomy is extension of the incision upward into the vaginal epithelium and past the hymenal ring. This releases any constriction and allows maximal room for the presenting part of the fetus.

Place the index and middle fingers of the nondominant hand into the patient's introitus with the palm of the hand facing outward (**Figure 163-5*A***). Pull the perineal body away from the fetal presenting part to protect the fetus from injury (**Figure 163-5*B***). Use a Mayo scissors to make the incision (**Figures 163-5*B* and 163-5*C***). **A scalpel can cause significant injury to the fetus, the mother, and/or the Emergency Physician.**

Insert the Mayo scissors so that one blade lies along the skin and the other is inside the introitus between the index and middle fingers and against the vaginal epithelium (Figure 163-5*B*). Allow the maximum descent of the fetal presenting part and moderate distension of the perineum before making the incision to avoid a third-degree or fourth-degree laceration. **Make the incision when the perineum is bulging and when approximately 4 cm of fetal scalp is visible during a contraction unless early delivery is indicated.**[1,19] Extend the incision 2 to 3 cm vertically up the vaginal epithelium past the hymenal ring. The introitus will readily open after making the episiotomy (**Figure 163-5*C***).

MEDIOLATERAL EPISIOTOMY

Make an incision at a 45° angle to the midline of the posterior vaginal orifice, from the inferior portion of the hymenal ring toward the ischial tuberosity (**Figure 163-2*B***). **The angle must be increased to 60° to obtain the correct angle of incision during pushing or distention of the perineum.** The length of the incision is less critical than that for a median episiotomy. Longer incisions require a more extensive repair. The side to which the episiotomy is performed is generally the same as the handedness of the operator.[1] Make the incision at approximately 5 o'clock for left-handed dominants or at 7 o'clock for right-handed dominants. If the incision is not deep enough, there will be too little tissue relaxation and a second incision will be necessary. This second incision increases the risk of a zigzag line after healing.[11]

The anatomic structures incised and requiring repair (in progression from superficial to deep) are the skin and subcutaneous tissues, the bulbospongiosus muscle, the superficial transverse perinei muscle, and a portion of the levator ani muscle and its fascia.

ASSESSMENT

Carefully examine the vagina, cervix, and lower uterine segment for any signs of injury or laceration immediately following delivery of the infant and placenta. Do not begin repairing the episiotomy until after the delivery of the placenta. The repair may be compromised if manual removal of the placenta or intravaginal procedures must be performed after the episiotomy is reconstructed. Assess the incision for extension into a third-degree laceration or fourth-degree laceration with a rectal exam. A rectal exam will also identify a button hole (i.e., a laceration through to the rectal mucosa that is not contiguous with the episiotomy). Identify any site of excessive bleeding and immediately control it with Vicryl suture.

MIDLINE EPISIOTOMY REPAIR TECHNIQUES

A midline episiotomy is easier to repair. The incision is symmetrically situated in the perineal body.[23,37,38] There are several options for episiotomy closure with limited data to suggest the superiority of one single type of suture material. An absorbable synthetic suture (e.g., polyglactin) is recommended, although they frequently require removal during the puerperium. Vicryl Rapide and absorbable monofilament have gained popularity for repair of episiotomies and perineal lacerations to avoid removal of persistent suture material. The use of chromic gut is associated with the rapid loss of tensile strength, increased wound breakdown, and increased initial pain.[26] Adhesive glue can be used for closure of perineal skin to decrease the operative time.[39] The traditional repair methods using suture techniques are described below.

ONE-SUTURE REPAIR TECHNIQUE

The one-suture technique uses one strand of suture to close the entire episiotomy. Repair the vaginal mucosa first (**Figure 163-6**). Use 3–0

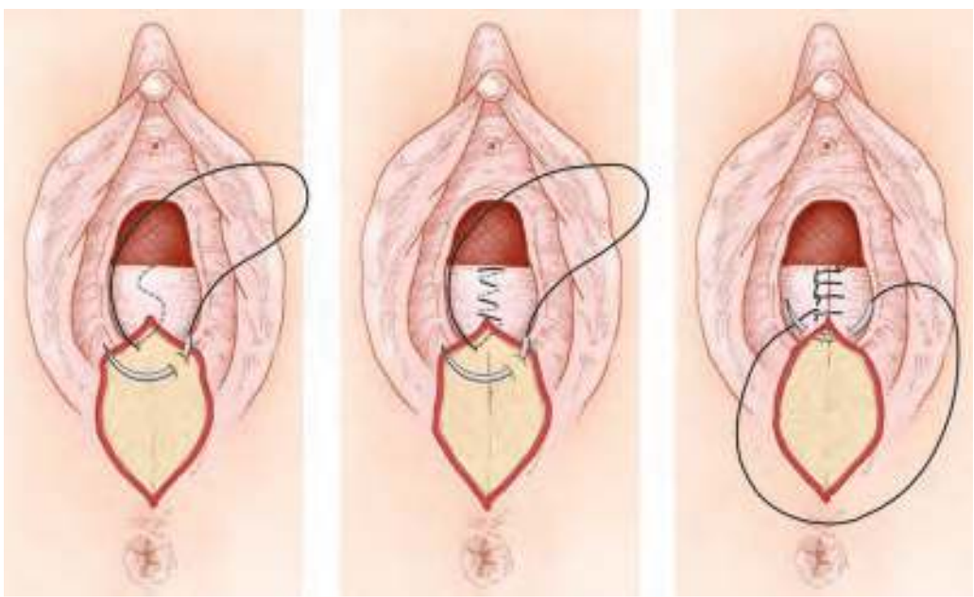


FIGURE 163-6. Methods of closing the vaginal mucosa. ***A.*** The running subcuticular stitch. ***B.*** The running simple stitch. ***C.*** The running locked stitch.

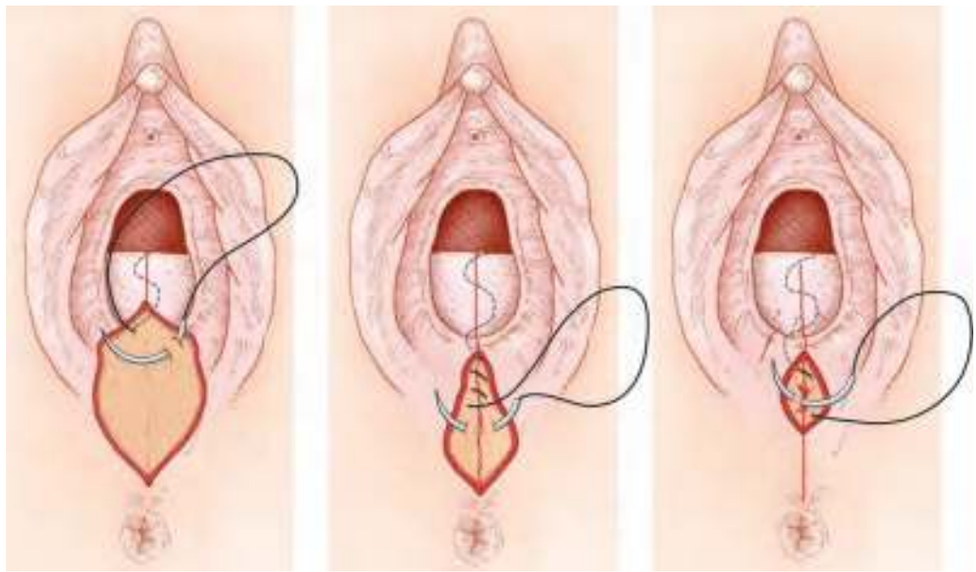


FIGURE 163-7. The one-suture technique to close an episiotomy. ***A.*** Approximate the vaginal mucosa with a running stitch. ***B.*** Approximate the hymenal ring followed by a running stitch to approximate the perineal body. ***C.*** Reverse the suture direction with a running subcuticular stitch to approximate the skin.

Vicryl on a noncutting needle. Place the initial suture superior to the apex of the vaginal incision to incorporate any retracted vessels. Tie the suture at the apex of the incision securely. Approximate the wound edges with a running subcuticular stitch (**Figure 163-6*A***), a running simple stitch (**Figure 163-6*B***), or a running locked stitch (**Figure 163-6*C***) if there are any concerns about hemostasis.[40] Continue the suture to the hymenal ring. Each suture should be of sufficient depth to approximate the vaginal epithelium and underlying muscularis layers to the midline thereby assuring closure of dead space. **The stitch should not be so deep as to cause penetration of the rectal mucosa. Do not allow the needle to enter through the rectal mucosa as this can result in the formation of a rectovaginal fistula.**

This method of suturing provides for hemostasis, eliminates dead space, and decreases the risk of subsequent infection. Once the hymenal ring is reached, throw the next stitch under the hymen exiting from the midline of the perineal body. The crown stitch is next and performed by reapproximating the bulbocavernosus muscles to the midline (**Figure 163-7*A***). This is carried down in a running fashion to approximate the remaining superficial muscles of the perineum and subcutaneous fat toward the inferior angle of the episiotomy (**Figure 163-7*B***). The repair is completed with a subcuticular closure of the perineal skin to the apex (**Figure 163-7*C***). The suture is directed from the midline into the vagina and placed through both sides of vaginal epithelium and tied upon itself ideally behind the hymenal ring.[26]

TWO-SUTURE REPAIR TECHNIQUE

This technique uses two strands of suture to close the episiotomy (**Figure 163-8**). Close the vaginal mucosa with a running stitch as described previously (**Figure 163-8*A***). Approximate the hymenal ring with this first strand of suture (**Figure 163-8*B***). Perform the crown stitch and remaining perineal body stitches with single interrupted stitches using a second strand of suture (**Figure 163-8*B***). Use the first strand of suture to place a subcuticular running stitch to approximate the skin (**Figure 163-8*C***). Finish approximating the skin incision with a buried knot.

ALTERNATIVE TECHNIQUE

Approximation of an episiotomy with one or two sutures can be time consuming and difficult. An alternative is to close the episiotomy using simple interrupted stitches (**Figure 163-9**). Take deep bites of tissue with the needle to ensure that the stitches close the subcutaneous tissues, perineal body, vaginal mucosa, and skin.

EPISIOTOMY EXTENSIONS

During a vaginal delivery, there is a small risk of lacerations extending into the muscular anal sphincter complex and the rectal mucosa, termed third-degree and fourth-degree lacerations, respectively. These lacerations may occur spontaneously or as a direct extension of an episiotomy. Visually inspect the perineum after every delivery and perform a digital rectal examination to assess the integrity of the external anal sphincter and the rectal mucosa. The repair of third-degree and fourth-degree lacerations is described below.

THIRD-DEGREE LACERATION REPAIR

A third-degree laceration involves the anal sphincter muscle and spares the anal mucosa. Visually inspect the anal sphincter muscle for partial lacerations. **A patient can have normal anal sphincter tone and a partial anal sphincter muscle laceration.** Repair any anal sphincter muscle lacerations as described in the following section on fourth-degree laceration repair.

FOURTH-DEGREE LACERATION REPAIR

Fourth-degree lacerations involve the anal sphincter muscle and the anal mucosa. They must be repaired to restore the normal anatomy and decrease the chance of fecal incontinence and a rectovaginal fistula.

A suture is placed 1 cm above the apex of the laceration and extended through the submucosal tissue but not through the rectal mucosa. Close the rectal mucosa in a continuous running or subcuticular fashion. Use a delayed absorbable suture such as 3–0 polyglactin. The suture should not penetrate the rectal mucosa and is

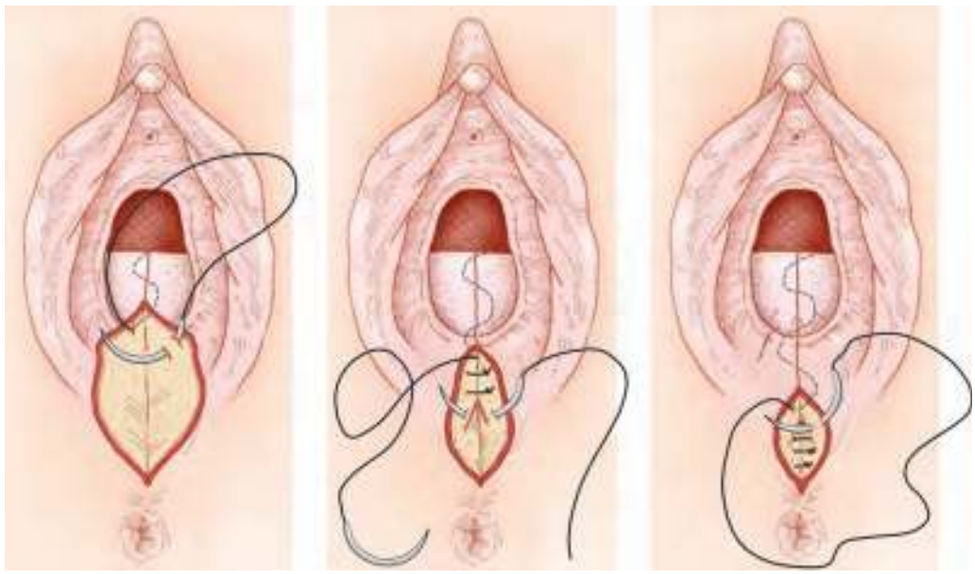


FIGURE 163-8. The two-suture technique to close an episiotomy. ***A.*** Approximate the vaginal mucosa with a running stitch. ***B.*** The hymenal ring is approximated with the first suture. The perineal body is approximated with a second suture utilizing an interrupted stitch. ***C.*** The first suture is continued subcutaneously to approximate the skin.

carried down to the anal verge. Place a second layer of sutures over the rectal mucosa to reinforce the initial sutures. Additionally, this suture will close the dead space between the vaginal mucosa and the rectum.

Identify the internal anal sphincter muscle. This is a thin band of smooth muscle encircling the anal mucosa. It appears whitish in color to the naked eye. Repair it with single interrupted stitches using 3–0 polyglactin or 3–0 polydioxanone suture material.

Identify both ends of the external anal sphincter. They are usually retracted laterally. Use Allis clamps to grasp the edges and pull them together in the midline (**Figure 163-10*A***). Repair the sphincter with four single interrupted stitches using 2–0 polyglactin or 3–0 polydioxanone on a tapered needle.[41] **Incorporate the capsule of the external anal sphincter in the repair and not just the muscle fibers (Figure 163-10*B*).** The authors recommend the use of the pneumonic "PISA" when repairing the external anal sphincter (**Figure 163-10*C***). Initiate the repair with the **P**osterior aspect of the sphincter which has the structure of a cylinder at the 9 o'clock position. Place the most **I**nferior suture at the 6 o'clock position. Place a stitch in the **S**uperior aspect at the 12 o'clock position. Finish the repair with a suture at the 3 o'clock position on the **A**nterior aspect of the sphincter. **Perform a rectal exam after the repair to test for adequate sphincter tone or penetrating suture.**

Alternatively, perform an overlap repair of the external anal sphincter.[41] Mobilize both ends to allow interposition of the ends of the sphincter. This requires a complete transection of the sphincter and expertise with a more extensive dissection of the sphincter. Reserve this type of repair for the Obstetrician.

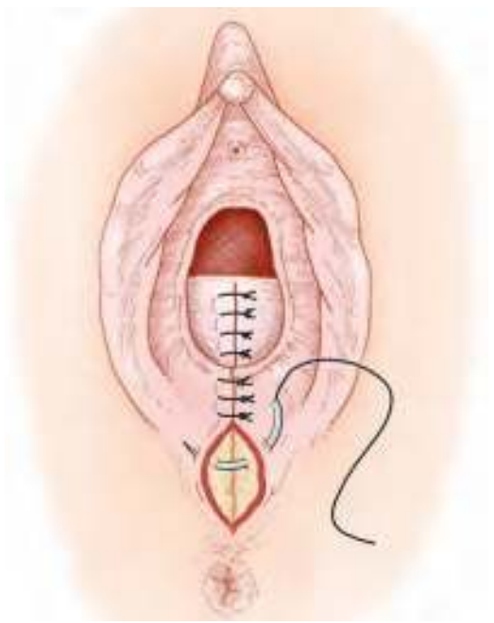


FIGURE 163-9. An alternative technique to close an episiotomy. Place simple interrupted stitches to approximate the mucosa, skin, and perineal body.

MEDIOLATERAL EPISIOTOMY REPAIR TECHNIQUE

The mediolateral episiotomy repair requires more surgical skill and anatomic knowledge. Perform the repair of the vaginal epithelium as one would with a midline episiotomy to the level of the hymenal ring. Repair the pelvic floor muscle (i.e., levator ani) with single interrupted stitches using delayed absorbable suture (e.g., 2–0 polyglactin). The medial edge of this muscular layer lies close to the anterior rectum. Care must be taken to avoid passing the needle into the rectum.[26]

It is extremely important to locate and repair the transected bulbospongiosus muscle. Find and grasp the transected ends of the bulbospongiosus muscle with Allis clamps. The lateral edge of this muscle may have retracted superiorly into the labia majora requiring adequate exposure of the underlying tissue and deep suture placement to ensure successful closure.[26] Approximate the bulbospongiosus muscle using single interrupted stitches with 2–0 or 3–0 polyglactin suture (**Figure 163-11*A***). Approximate the remaining subcutaneous tissue and perineal skin using the one-suture or the two-suture technique as a midline episiotomy repair (**Figure 163-11*B***). Approximate the skin of the perineum using a running subcuticular stitch (**Figure 163-11*C***).

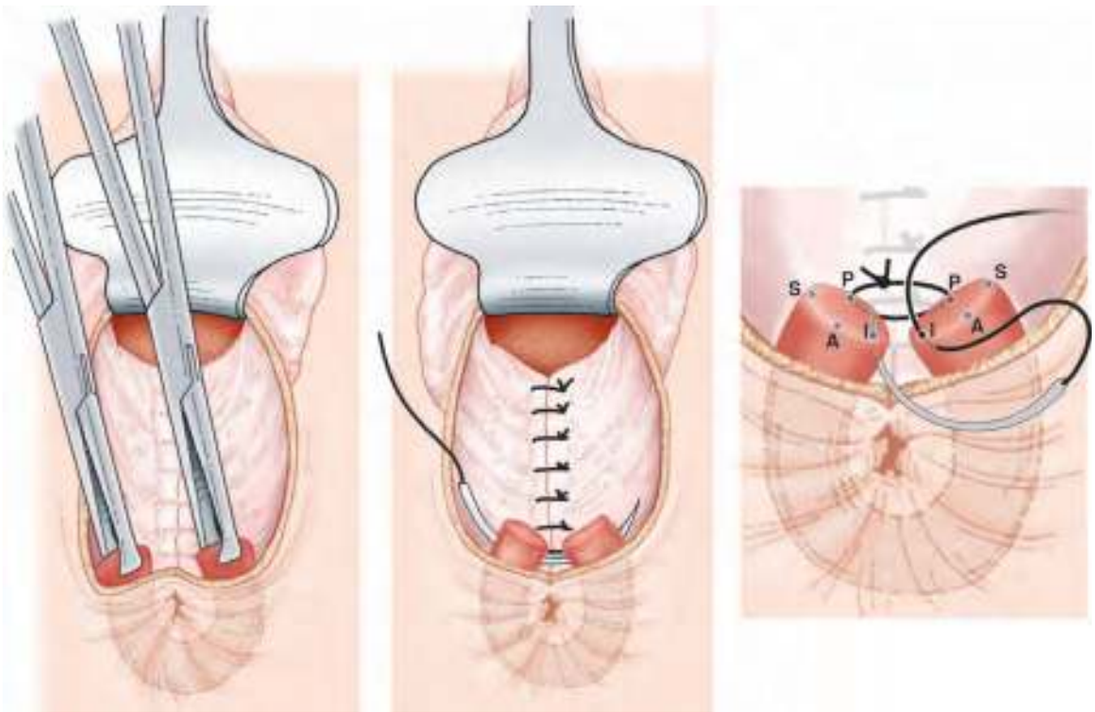


FIGURE 163-10. Repair of a fourth-degree laceration or episiotomy. ***A.*** Approximate the rectal submucosa and muscularis with simple interrupted sutures. Grasp the cut edges of the anal sphincter muscle with Allis clamps. ***B.*** Approximate the ends of the anal sphincter muscle with transfixion stitches. ***C.*** Repairing the anal sphincter muscle. (A, anterior; I, inferior; P, posterior; S, superior.)

AFTERCARE

The aftercare for a perineal incision involves perineal hygiene and adequate analgesia. Routine use of prophylactic antibiotics has never been proven effective to prevent infections after an episiotomy. A single intravenous dose of a second-generation cephalosporin (e.g., cefazolin) during or immediately after the repair of a third-degree or fourth-degree laceration is recommended.[9] The use of warm sitz baths three to four times a day in concert with the use of ice packs to the perineum will decrease inflammation. Prescribe stool softeners to decrease the pain of defecation and the risk of episiotomy disruption, especially if it was associated with a third-degree or fourth-degree extension. Nonsteroidal anti-inflammatory drugs or acetaminophen provides adequate analgesia in most patients.

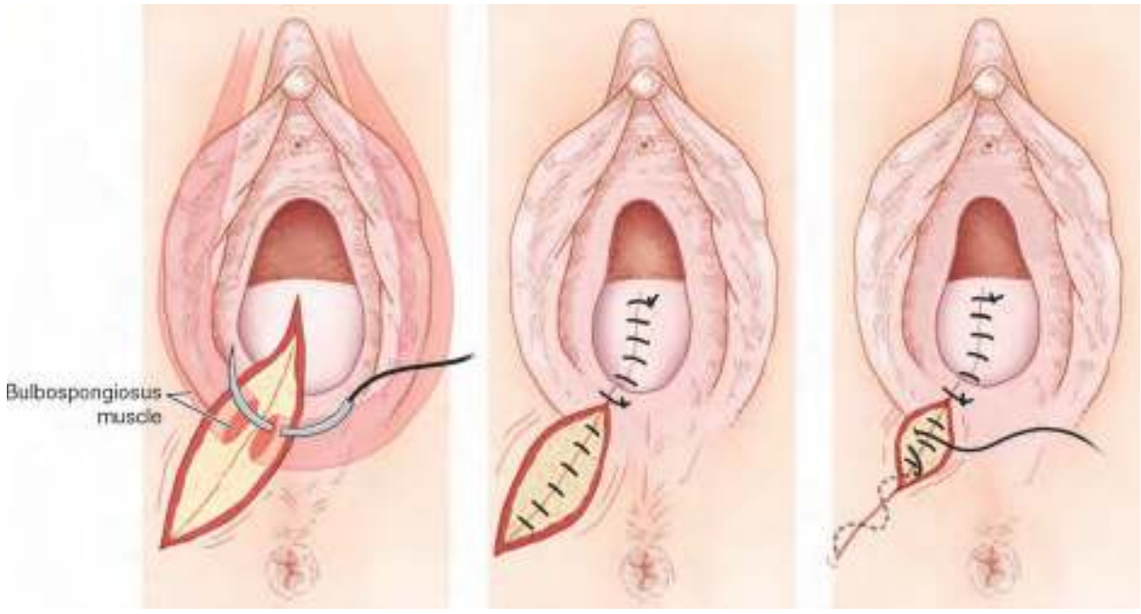


FIGURE 163-11. Repair of a mediolateral episiotomy. ***A.*** Find and approximate the ends of the transected bulbospongiosus muscle. ***B.*** Approximate the vagina and deep peroneal tissues using the one-suture or two-suture technique. ***C.*** Approximate the skin with running subcuticular stitches.

Narcotic analgesics may be required for 24 to 72 hours, especially in cases of mediolateral episiotomies. Many Obstetricians prefer oxycodone or acetaminophen with codeine as they are not as constipating as the other narcotic analgesics. This is especially important in patients with third-degree or fourth-degree lacerations.

COMPLICATIONS

The most common complications are hemorrhage, infection, and dehiscence. Most hemorrhage can be controlled by the application of direct pressure with sterile gauze. Bleeding vessels may be secured with single interrupted stitches or incorporation into the repair using a running locked technique. The mediolateral episiotomy has an increased risk of hemorrhage compared to the midline episiotomy. Adequate hemostasis must be assured during the repair. Vaginal hematomas can easily form. Large or expanding vaginal hematomas usually require the incision to be opened, the hematoma drained, and the hemorrhage controlled. Small hematomas can be managed with analgesics, vaginal packing, and ice packs.

Infection usually presents 6 to 8 days after delivery. The patient usually complains of a fever, perineal pain, and a purulent discharge that may be foul smelling. Management includes local perineal care and exploration with debridement under adequate anesthesia to drain a potential abscess. Carefully examine the patient and explore the area to rule out necrotizing fasciitis.[28] Begin intravenous antibiotics after consulting with the Obstetrician-Gynecologist and obtaining wound cultures.

Pain at the episiotomy site is common. It usually resolves in 3 to 5 days. The pain usually responds to acetaminophen and nonsteroidal anti-inflammatory drugs. A hematoma or an infection must be ruled out if the patient is complaining of intense pain. Dyspareunia is more common with a mediolateral episiotomy.[11] Prospective studies have not found differences in the resumption of intercourse at 3 months when compared to patients without an episiotomy.[8] No difference in pain was noted in one study between continuous and interrupted suture closure.[40] A Cochrane review noted less short-term pain, the decreased need for analgesics, the lack of suture removal, and the use of less suture material with continuous sutures.[42] The decision to use continuous or interrupted sutures is up to the Emergency Physician and depends on several factors (e.g., time constraints and the ability to place).

Dehiscence is reported in 0.1% to 2% of episiotomies. Early closure within 1 week is preferable to the delayed closure at 2 to 3 months. It is necessary to remove all necrotic tissue and copiously irrigate the area with a diluted povidone iodine solution. This is followed with twice-daily scrubbings of the wound with a scrub brush and povidone iodine. The wound can be repaired once it is free of exudates and begins to show granulation tissue. Intravenous antibiotics might be necessary in the presence of an infection.[43] The repair procedure should be performed by a Gynecologist or a Colorectal Surgeon if the dehiscence involves the anal sphincter.

The use of a midline episiotomy may increase the risk of third-degree and fourth-degree lacerations which may result in incontinence of feces, incontinence of flatus and/or rectovaginal fistula formation if not properly repaired.[44-47] Use a mediolateral episiotomy for patients at increased risk for third-degree or fourth-degree lacerations (e.g., short perineal body or during forceps delivery).

SUMMARY

Episiotomies should be performed selectively for appropriate indications. Clinical judgment, patient assessment, expertise level, and common sense should be employed. The midline episiotomy is the preferred technique for non-Obstetricians. It is easier to perform and easier to repair when compared to the mediolateral episiotomy. It carries a higher risk for extension into the anal sphincter.

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164 Shoulder Dystocia Management

Christopher Freeman and Adi Abramovici

INTRODUCTION

Shoulder dystocia is a rare obstetric emergency placing the mother and the fetus at risk for significant morbidity and mortality. It is diagnosed when after delivery of the fetal head further expulsion of the fetus is prevented by impaction of the fetal shoulders within the maternal pelvis (**Figure 164-1**).[1] **Shoulder dystocia is considered an emergent situation. The Emergency Physician must be able to recognize the condition and quickly respond. The goal is to deliver the fetus as quickly as possible using safe maneuvers and documenting the chain of events.**

The incidence of shoulder dystocia varies due to the subjective nature of the diagnosis and the dependency on the documentation.

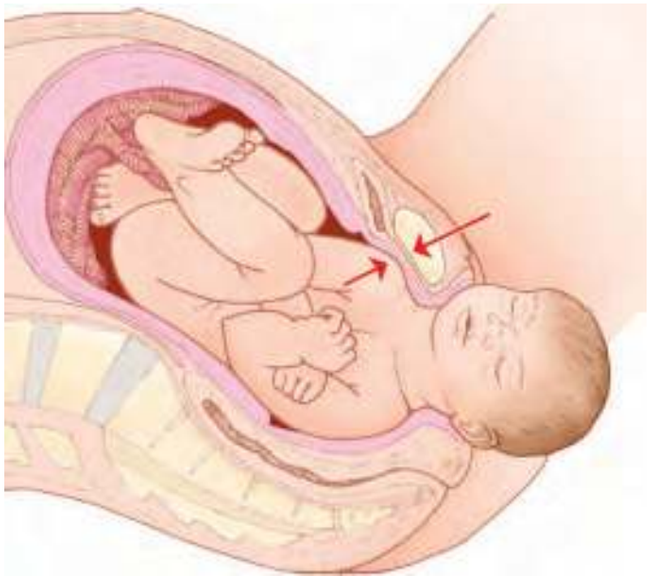

FIGURE 164-1. The head may retract toward the perineum (i.e., turtle sign) when delivery of the fetal head is not followed by delivery of the shoulders.

It has been reported in up to 4% of cephalic spontaneous vaginal deliveries.[2-7] Differing definitions of shoulder dystocia may account for some of this variability. Some reports require that maneuvers for shoulder release be documented on the chart whereas others accept the clinical diagnosis of shoulder dystocia. Other definitions look at the timing of the delivery of the head in relation to the delivery of the shoulders or the completion of the birth. The rare occurrences of shoulder dystocia make designing prospective studies difficult in describing the incidence and in evaluating the efficacy of various release maneuvers.[8]

Shoulder dystocia is most often unpredictable and unanticipated because accurate methods of predicting do not exist. Risk factors that increase the risk for shoulder dystocia are documented in the literature (**Table 164-1**). Many patients with shoulder dystocia do not have risk factors. Emergency Physicians may not have access to the patient's prenatal history and/or ultrasound reports that may highlight the presence of risk factors for shoulder dystocia. It is therefore imperative for the Emergency Physician to be knowledgeable and comfortable with the various release maneuvers if shoulder dystocia is encountered during a precipitous delivery.[9]

TABLE 164-1 The Risk Factors for Shoulder Dystocia

Antepartum	Intrapartum
Abnormal pelvic anatomy	Arrested dilation
Advanced maternal age	Assisted delivery (forceps or vacuum)
Diabetes (actual or gestational)	Descent arrested, failed, or protracted
Excessive weight gain	Labor augmentation (e.g., oxytocin)
Fetal macrosomia	Pelvimetry concerning for cephalopelvic disproportion
Male fetus	Prolonged labor of first stage
Maternal obesity	Prolonged labor of second stage
Multiparity	
Previous large baby	
Previous shoulder dystocia	
Prolonged pregnancy	
Short maternal stature	

ANATOMY AND PATHOPHYSIOLOGY

Shoulder dystocia is usually diagnosed after delivery of the fetal head when the fetal shoulders fail to deliver despite standard gentle traction on the fetal head. It results from impingement of the biacromial diameter of the fetus against the maternal pubic symphysis anteriorly and the maternal sacral promontory posteriorly.[10] Additional definitions are a delivery in which maneuvers are required to deliver the fetus after normal gentle downward traction the head delivers but external rotation does not occur and the head recoils tightly against the perineum (i.e., turtle sign) or a head-to-body delivery completion of greater than 60 seconds.[11,12] Shoulder dystocia can frighten the Emergency Physician and the patient.

Shoulder dystocia is a rare and potentially catastrophic obstetrical emergency. The Emergency Physician must deliver the fetus quickly and without applying excessive forces that may result in fetal injury. Associated risk factors include fetal macrosomia, maternal diabetes, multiparity, obesity, operative vaginal delivery, prior history of shoulder dystocia or macrosomia, prolonged second stage of labor, and postterm pregnancy (**Table 164-1**).[10]

Fetal macrosomia is defined as fetal growth beyond a specific weight, usually 4000 to 4500 gm, regardless of the fetal gestational age. The risk of shoulder dystocia is 9.2% to 24% in nondiabetic pregnant women and 19.9% to 50% in diabetic women when birth weight is greater than 4500 gm.[13] Dystocia can occur unexpectedly in infants of normal birth weights.[13] A simple algorithm to help determine if shoulder dystocia or fetal macrosomia may be present is shown in **Figure 164-2**.

INDICATIONS

The presence of shoulder dystocia requires immediate recognition and action. Most cases will respond to commonly used maneuvers. Utilize the maneuvers in the approximate order described in this

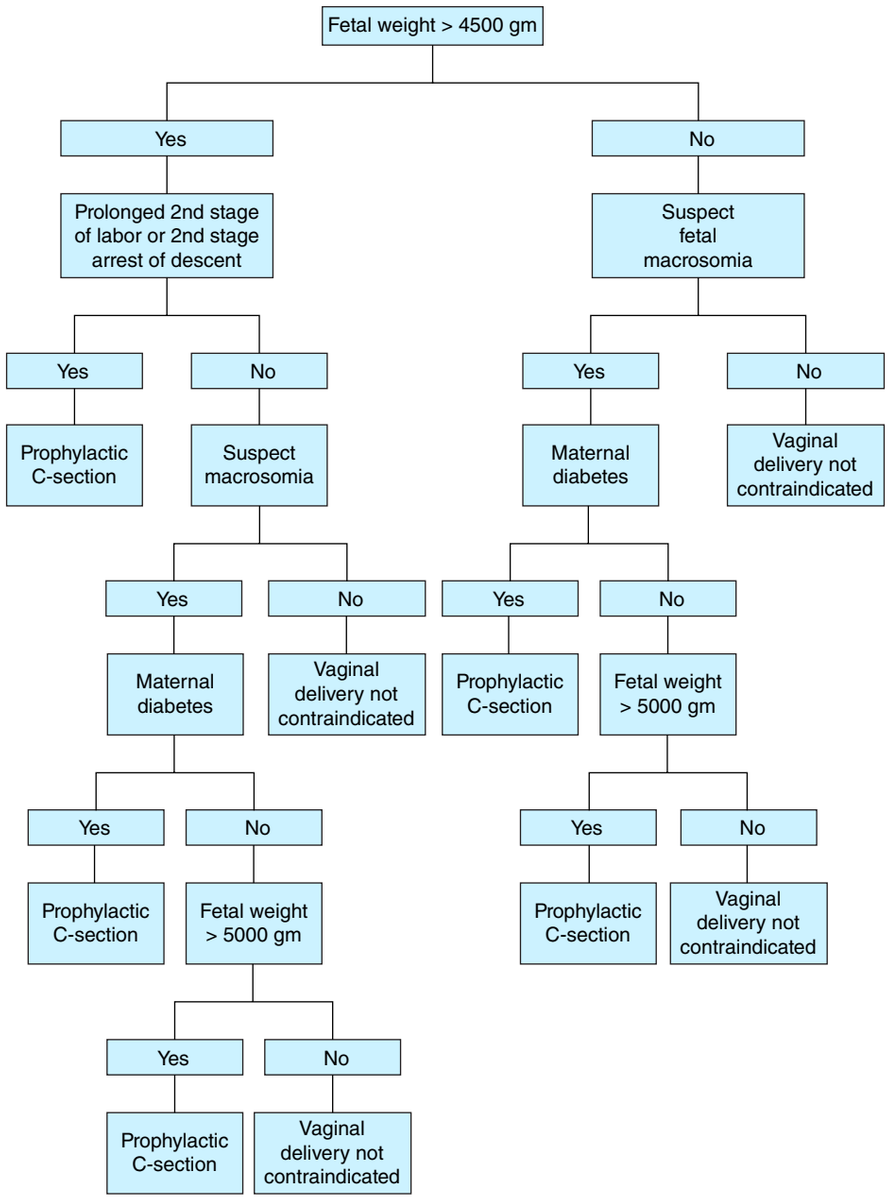

FIGURE 164-2. An algorithm providing a sequence of decisions for evaluation and anticipation of shoulder dystocia.[13]

chapter from the least invasive and easiest to perform to the most invasive and difficult to perform (**Figure 164-3 and Table 164-2**). More aggressive approaches may be indicated in cases that are refractory to successful delivery of the neonate.

Documenting shoulder dystocia is imperative and the American College of Obstetrics and Gynecology (ACOG) has a standardized checklist to be used in the documentation process if a patient has experienced shoulder dystocia (**Figure 164-3 and Table 164-2**).[14] Call for help from Anesthesiologists, Neonatologists or Pediatricians, and Neonatal Intensive Care Unit Staff. Assign a timekeeper. Perform the maneuvers. Reevaluate the course of action including using other maneuvers or repeating maneuvers. Consider abdominal delivery (i.e., cesarean section). Document the events using a checklist.

A symphysiotomy (Chapter 168) is indicated for shoulder dystocia unresponsive to less invasive techniques and for fetal head entrapment by presumed cephalopelvic disproportion. It is an alternative to the cesarean section when a qualified Obstetrician or Surgeon is unavailable.[13]

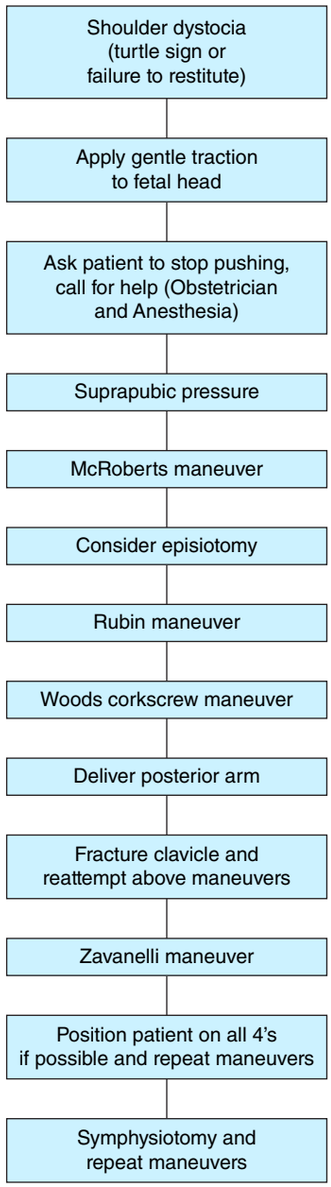

FIGURE 164-3. Algorithm for the treatment of shoulder dystocia. Perform in the order listed from least to most invasive.

TABLE 164-2 Common Mnemonics for the Treatment of Shoulder Dystocia

ALARMER
A – Ask for help
L – Lift and hyperflex legs (McRoberts maneuver)
A – Anterior shoulder disimpaction
R – Rotation
M – Manually remove posterior arm
E – Episiotomy
R – Roll over onto all fours
BE CALM
B – Breathe and stop pushing
E – Elevate the legs into McRoberts maneuver
C – Call for help
A – Apply suprapubic pressure and not fundal pressure
L – Enlarge the vaginal opening (i.e., episiotomy)
M – Maneuvers to deliver posterior arm or rotational maneuvers
HELPERR
H – Help (call for assistance)
E – Evaluate for episiotomy
L – Legs (McRoberts position)
P – Pressure (apply suprapubic pressure)
E – Enter maneuvers (perform rotational maneuvers)
R – Remove the posterior arm
R – Roll the patient onto all fours

CONTRAINDICATIONS

The only absolute contraindication is if the procedure might endanger the mother. Consider an emergency cesarean section if an Obstetrician is available when shoulder dystocia is of high clinical concern prior to fetal crowning (**Figure 164-2**). The Emergency Physician is usually only managing precipitous deliveries where there is no other personnel trained in the management of laboring patients.

The indications to perform a cesarean delivery are relative contraindications for release maneuvers. ACOG supported the recommendation that planned cesarean delivery may be a reasonable strategy for diabetic pregnant women with estimated fetal weights exceeding 4250 to 4500 gm (**Figure 164-2**).[8] ACOG issued guidelines on fetal macrosomia in 2001.[8] The guidelines were based upon limited or inconsistent scientific evidence. ACOG recommended that an estimated fetal weight of more than 4500 gm, a prolonged second stage of labor, or arrest of descent in the second stage of labor are indications for cesarean delivery.[8,13] ACOG noted that the diagnosis of fetal macrosomia is imprecise and recommended prophylactic cesarean delivery be considered with estimated fetal weights of more than 5000 gm in nondiabetic pregnant women and more than 4500 gm in diabetic pregnant women.[13]

EQUIPMENT

GENERAL SUPPLIES

- Electronic fetal monitor
- Sterile towels
- Clock or timer watch
- Sterile perineal drapes
- Sterile gloves

- Chromic suture, 2–0 and 4–0
- Vicryl suture, 2–0 and 3–0
- Bulb syringe
- Clean towels/blanket for baby
- Umbilical cord clamp
- Sterile scissors
- Infant warmer
- Step stool to be placed next to the patient's bedside
- Neonatal resuscitation equipment and medications

DELIVERY INSTRUMENT PACK

- Sterile bandage scissors
- Towel clips
- 2 Allis forceps
- 4 ring forceps
- 6 straight Kocher clamps
- Straight Mayo scissors
- 2 suture scissors
- Adson forceps, or other forceps with teeth
- Russian forceps, 5½ inches and 8 inches
- Gelpi retractor
- Richardson retractors, small and medium
- Army-Navy retractors
- 6 inch needle driver

SYMPHYSIOTOMY

- #10 or #15 scalpel blade on a handle
- Finger guard
- Foley catheter
- Povidone iodine or chlorhexidine solution

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Include fetal distress, fetal death, fetal hypoxia, and hysterectomy in the discussion. Obtain an informed consent for the procedure and place this in the medical record. There is often not enough time to obtain a written consent in the Emergency Department. Carefully and completely document this discussion in the medical record.

Pain associated with the first stage of labor can be relieved with a paracervical block or pudendal nerve block.[15] Inject 5 mL of local anesthetic solution into the submucosa of the lateral vaginal fornix. Repeat the injection in the contralateral lateral vaginal fornix. Pain transmission is interrupted for all visceral sensory nerve fibers from the cervix, upper vagina, and uterus. The somatosensory fibers from the perineum are not blocked. This technique is effective only during the first stage of labor and before shoulder dystocia occurs.[15,16] **These anesthetic techniques must be performed prior to the occurrence of shoulder dystocia.**

Place the patient in the lithotomy position on a bed with stirrups. Preparation begins with recognition of fetal macrosomia by clinical examination or fetal ultrasound. Retraction of the fetal head immediately after its delivery and the fetal chin against the maternal thigh (i.e., turtle sign) with difficulty delivering the anterior shoulder signals the beginning of action toward resolving the shoulder dystocia (**Figure 164-1**).

FIGURE 164-4. Restitution (i.e., external rotation) of the fetal head normally results in a natural perpendicular relationship of the head to the shoulders.

The fetal sagittal suture generally lies oblique to the maternal anteroposterior diameter with the fetal shoulders occupying the opposite oblique diameter after delivery of the fetal head (**Figure 164-4**). The shoulder may become impacted behind the pubic symphysis and impede delivery if the anterior shoulder descends in the anteroposterior diameter (**Figure 164-1**). Apply gentle and downward pressure on the fetal head to move the posterior shoulder into the hollow of the sacrum and deliver the anterior shoulder (**Figure 164-5**). **Resist applying excessive downward or lateral traction on the fetal head and neck.**

TECHNIQUES

Perform attempts at gentle traction coordinated with maternal expulsive efforts before attempting maneuvers to relieve shoulder dystocia. Initiate a planned sequence of events if delivery is impeded (**Figure 164-3**). **Avoid applying fundal pressure and discontinue maternal pushing efforts until disimpaction has occurred.** Fundal pressure and maternal pushing may further impact the fetal shoulders, increase the risk of uterine rupture, and should never be performed. Notify the medical personal in the room of the situation and summon extra personnel for help with one person designated as a timekeeper. Notify a Neonatologist of the impending delivery and contact an Anesthesiologist for pain control. Maneuvers for shoulder dystocia disimpaction will be described in order of ease of implementation and from least invasive to more invasive (**Figure 164-3**).

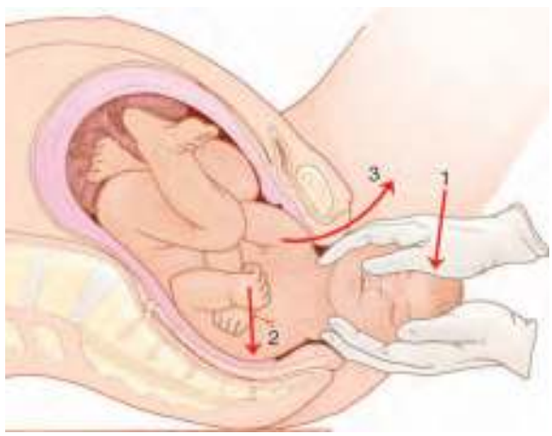

FIGURE 164-5. Apply gentle and downward pressure on the fetal head (1) to move the posterior shoulder into the hollow of the sacrum (2) and deliver the anterior shoulder (3).

FIGURE 164-6. Moderate suprapubic pressure is applied to disimpact the fetal shoulder while gentle downward traction is applied to the fetal head.

There is no indication that any one of the described techniques is superior to another to relieve the shoulder dystocia. The use of these techniques should relieve the dystocia. The use of uterine relaxant medications may be necessary to overcome the expulsive forces of the uterine contraction.

SUPRAPUBIC PRESSURE

Use suprapubic pressure alone or in combination with the McRoberts maneuver. Apply gentle downward traction to the fetal head while an assistant simultaneously applies moderate suprapubic pressure with the heel of their hand. The suprapubic pressure can disengage the anterior shoulder from the pubic symphysis. Use a step stool placed on the side of the patient's bed for easier application of suprapubic pressure (**Figure 164-6**). **Do not apply heavy pressure to prevent injury to the fetus's brachial plexus, neck, and spinal cord.** Start with continuous pressure. Use a rocking motion of pressure to dislodge the anterior shoulder if continuous pressure does not release the shoulder.

McROBERTS MANEUVER

The McRoberts maneuver is easy to perform and has proven to be effective (**Figure 164-7**). It should be used after suprapubic pressure to try to relieve the shoulder dystocia. Place an assistant on each side of the patient. Hyperflex and abduct the mother's thighs onto her abdomen (**Figure 164-7*A***). Instruct the assistants to maintain support of the hyperflexion while simultaneously applying suprapubic pressure (**Figure 164-7*B***). This results in a flattening of the maternal lumbosacral curve and a rotation of the pubic symphysis cephalad (**Figure 164-7*C***). The suprapubic pressure disengages the anterior shoulder from the pubic symphysis. Rotation of the maternal pelvis may free the impacted anterior fetal shoulder.[7,17] This maneuver has the advantages of reducing shoulder extraction forces, brachial plexus stretching, and the incidence of clavicular fractures.[7,17] **The McRoberts maneuver is effective in disimpacting the fetal shoulders in 50% to 90% of cases of shoulder dystocia and should be a first-line treatment.[7,17-19]**

EPISIOTOMY

Episiotomy is a surgical incision at the time of delivery to increase the diameter of the maternal soft tissue of the pelvis and facilitate vaginal delivery (Chapter 163). Consider performing an episiotomy in shoulder dystocia patients when it has not resolved with suprapubic pressure and the McRoberts maneuver. The episiotomy creates extra space for the Emergency Physician's hand. **An episiotomy will not disimpact the anterior shoulder and relieve shoulder dystocia but does allow for increased access of rotational maneuvers.**

The two most commonly used episiotomies are median and mediolateral. The median (i.e., midline) is the easier to perform but is associated with an increased risk of extension toward the anal

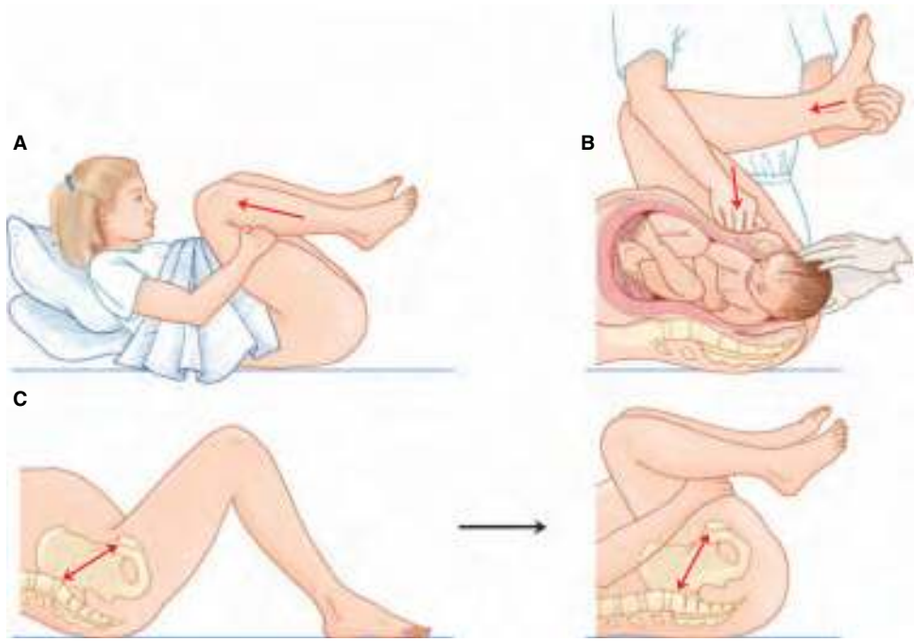

FIGURE 164-7. The McRoberts maneuver. ***A.*** Hyperflex the maternal thighs upon the abdomen. ***B.*** An assistant applies suprapubic pressure while maintaining flexion of the legs. ***C.*** This maneuver results in cephalad rotation of the maternal pelvis and an increase in the size of the pelvic outlet.

FIGURE 164-8. The Rubin maneuver. Rotation of the anterior shoulder counterclockwise through a small arc to the oblique position.

sphincter (i.e., third-degree tear) and rectum (i.e., fourth-degree tear). The mediolateral episiotomy has a decreased risk of extension but is less commonly used because it can be more difficult to repair, increases the risk of bleeding, and causes more postpartum discomfort.[20]

RUBIN MANEUVER

The Rubin maneuver is simple and may lead to the descent and delivery of the anterior fetal shoulder (Figure 164-8). Insert the dominant hand into the vagina. Place the fingers of the hand against the posterior aspect of the anterior fetal shoulder. Rotate the fetal shoulder in a small arc toward the fetal chest. **The Rubin maneuver compresses and diminishes the size of the fetal shoulder girdle to disimpact the anterior shoulder.** Rotation of the anterior fetal shoulder in the opposite direction (i.e., toward fetal back) will open the shoulder girdle, increase the size of the shoulder girdle, and further impact the fetus.

WOODS CORKSCREW MANEUVER

The Woods corkscrew maneuver (Figure 164-9) is an alternative to the Rubin maneuver and is the release of the posterior shoulder. Insert the dominant hand into the vagina. Place the fingers of the hand against the posterior aspect of the posterior fetal shoulder. Gently rotate the posterior shoulder toward the chest (**Figure 164-9**).[21] **This maneuver compresses and diminishes the size of the fetal shoulder girdle to disimpact the anterior shoulder.** Rotation in the opposite direction (i.e., toward fetal back) will open the shoulder girdle, increase the size of the shoulder girdle, and further impact the fetus.

FIGURE 164-9. The Woods corkscrew maneuver. Rotation of the posterior shoulder through a 180° arc.

DELIVERY OF THE POSTERIOR ARM

Delivery of the posterior arm is also known as the Barnum maneuver or Jacquemier maneuver. Attempt to deliver the posterior arm if the previous maneuvers are unsuccessful (**Figure 164-10**). Insert the dominant hand into the vagina. Place the fingers of the hand against the posterior fetal humerus (**Figure 164-10*A***). Sweep the fetal arm across the chest (**Figure 164-10*B***). Palpate for and grasp the fetal hand (**Figure 164-10*C***). Gently pull the hand along the side of the face. Continue to gently pull the hand to deliver the posterior arm and shoulder (**Figure 164-10*D***). Apply gentle downward traction on the fetal head and arm while an assistant simultaneously applies suprapubic pressure to release and deliver the anterior shoulder (**Figure 164-10*E***). **Do not pull directly on the fetal arm.** Rotate the shoulder girdle into the oblique diameter if traction on the fetal head and arm does not deliver the anterior shoulder.[10] This will usually disimpact the anterior shoulder and allow it to be delivered. **The major disadvantage of this maneuver is that it may result in a clavicle fracture or a humerus fracture.**

DELIBERATE FRACTURE OF THE CLAVICLE

Fracture the fetal clavicle by pressing the anterior clavicle against the maternal pubic symphysis if the above maneuvers fail. This will decrease the rigidity and the size of the fetal shoulder girdle. Exert the pressure in a direction away from the lungs to avoid a pneumothorax. **Never use an instrument to fracture the clavicle.** The instrument may penetrate the thoracic cavity and cause a pneumothorax or result in subsequent osteomyelitis if the skin is punctured.[22] The fracture will heal quickly and is much less serious than asphyxia, a brachial plexus injury, or death. This maneuver is difficult to perform.[21] **It is physically and mentally difficult to deliberately fracture the clavicle of an infant.**

ZAVANELLI MANEUVER

The Zavanelli maneuver involves replacement of the fetal head followed by a cesarean section (Figure 164-11). Use this maneuver when other maneuvers fail or as a last resort. This procedure is reserved for catastrophic situations due to the increased risk of perinatal and maternal morbidity and mortality. Consider using uterine relaxant medications to stop the uterine contractions. **The expulsed head must undergo two maneuvers to reverse the mechanisms of labor.** Manually rotate the fetal head into the pre-restitution position (**Figure 164-11*A***). This is usually the direct occiput anterior position with full extension of the neck (**Figure 164-11*B***). The second maneuver is flexion of the fetal head followed by gentle upward pressure on the head to replace it into the maternal vagina (**Figure 164-11*B***).

The Emergency Physician must maintain their hand in the vagina and maintain gentle pressure on the fetal head to prevent re-expulsion. Keep the hand in the vagina and hold the fetal head during the entire transport to the Operating Room and until a cesarean section is performed. The Emergency Physician can quickly switch their hand with that of an assistant at any time.

This series of maneuvers decompresses the fetus. Immediately transport the mother to the Operating Room for a cesarean section. The Zavanelli maneuver was found to be successful in 84% of initial attempts and 91% of attempts when uterine-relaxing anesthesia or uterine-relaxing medication (e.g., usually 0.25 mg terbutaline subcutaneously) was administered.[23,24] It was successful on the first attempt by untrained practitioners in 69% of the cases.

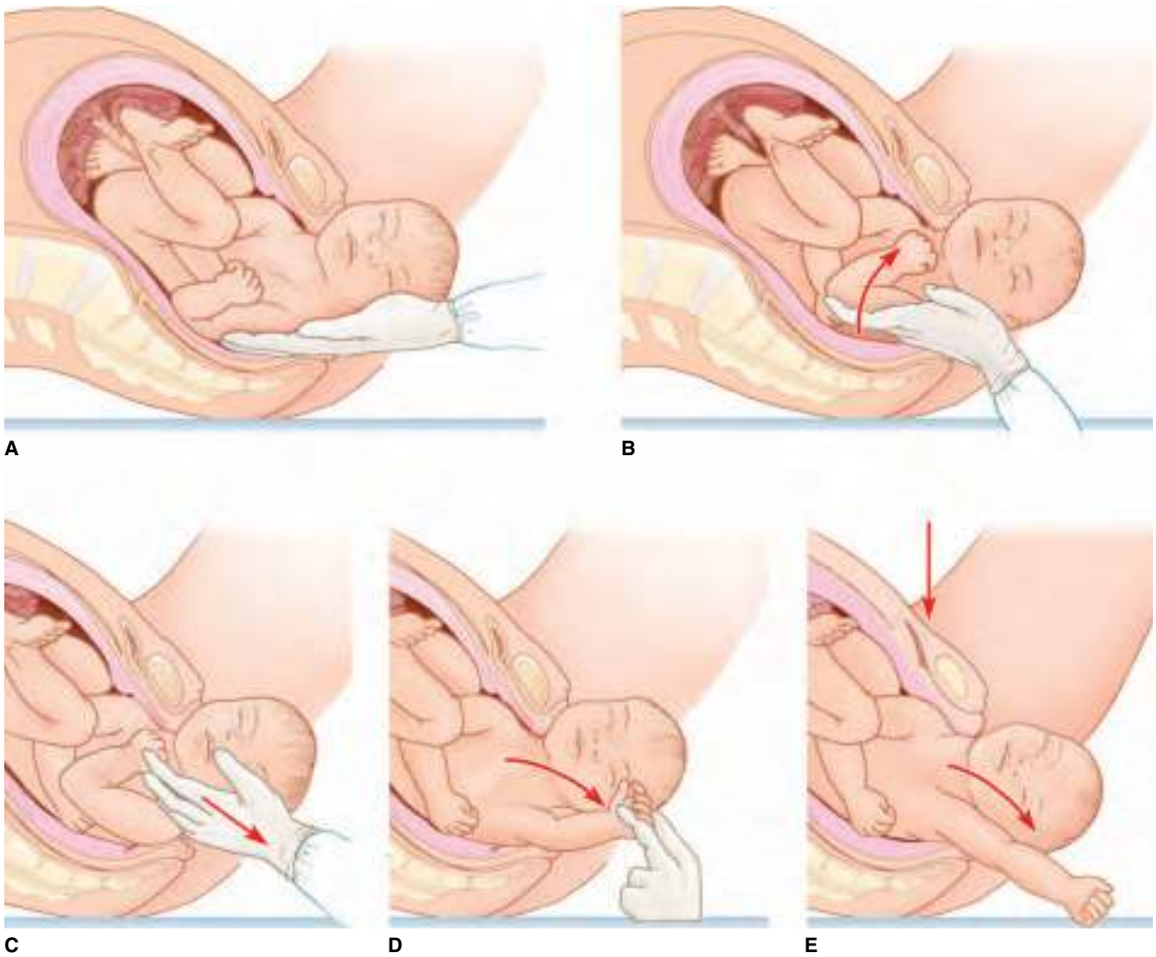

FIGURE 164-10. Delivery of the posterior arm. ***A.*** Insert a hand into the vagina and along the posterior fetal humerus. ***B.*** Sweep the fetal arm across the chest. ***C.*** Grasp the hand and extend the arm along the side of the face. ***D.*** Deliver the posterior arm and shoulder from the vagina. ***E.*** Apply gentle downward traction on the fetal head and arm while an assistant simultaneously applies suprapubic pressure to deliver the anterior shoulder.

GASKIN MANEUVER

The Gaskin maneuver is also known as the "all-fours" maneuver and named after the midwife Ina May Gaskin (**Figure 164-12**). It is old and not familiar to many. It is considered an effective, rapid, and safe technique to relieve a shoulder dystocia.[25] This increases the pelvic diameters and allows better access to the posterior shoulder. Roll the patient prone then have her get up on all four extremities (**Figure 164-12**). Use gentle downward traction to deliver the

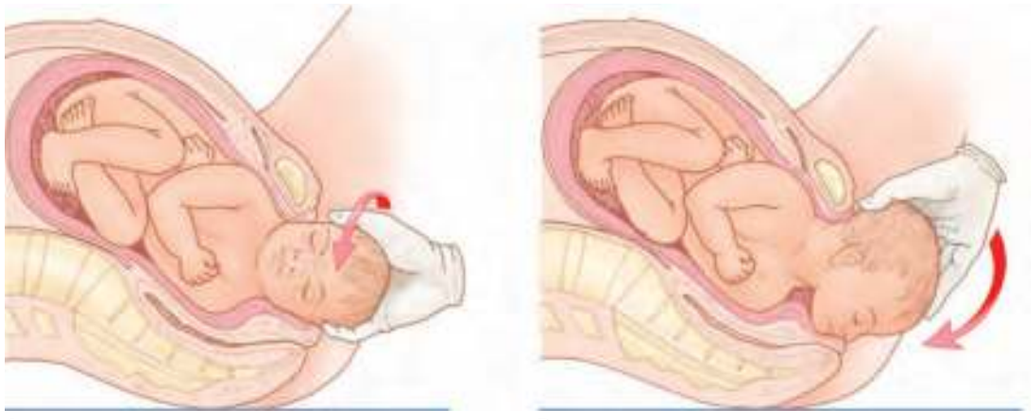

FIGURE 164-11. The Zavanelli maneuver. ***A.*** Manually return the fetal head to the pre-restitution position if restitution has occurred. This position is usually the direct occiput anterior position. ***B.*** Flex the fetal head and apply gentle upward pressure to place the fetal head back into the vagina.

FIGURE 164-12. The Gaskin or all-fours maneuver.

posterior shoulder with the help of gravity. Reattemptthe above maneuvers in this position if the shoulder dystocia is not relieved.

There are problems associated with this position. It may disorient the Emergency Physician while performing the maneuvers and the posterior shoulder is delivered first. Traction is still applied toward the floor but to deliver the posterior shoulder. The position is difficult to maintain in the exhausted patient.

SYMPHYSIOTOMY

A symphysiotomy is an uncommon procedure and a last effort used primarily in two situations (Chapter 168). The first is shoulder dystocia unresponsive to less invasive techniques. The second is when the head of a breech delivery is trapped by presumed cephalopelvic disproportion. It serves as an alternative to the more invasive cesarean section. It is especially useful in situations where an Obstetrician or Surgeon is unavailable.[26]

ASSESSMENT

Hand the infant to others to resuscitate if a Neonatologist or Neonatal Nurse is present so the Emergency Physician can attend to the mother. Let a Gynecologist attend to the mother while the Emergency Physician attends to the infant if a Neonatologist is not available. Resuscitate both patients as necessary. Determine if the mother has any hemorrhage and deliver the placenta.

AFTERCARE

Disimpaction of the shoulder girdle is usually followed by delivery of the infant. Clamp and cut the umbilical cord. Immediately assess and implement any resuscitative measures for the infant without delay. Deliver the placenta. Repair any lacerations, Dührssen's incisions, episiotomy incisions, perineal lacerations, and vaginal lacerations. Refer to Chapter 163 for the complete details regarding episiotomy repair. Refer to Chapter 166 for the complete details regarding postpartum hemorrhage management. Initiate uterine massage and administer Pitocin following delivery of the placenta to prevent postpartum hemorrhage.

Accurately, clearly, and completely document the series of events in the medical record. **Do this for good medical management and in case the infant has complications and the family litigates.** Standard forms provide consistent and complete documentation.[14] Make sure the documentation includes all personnel present, fetal head position, sequence of maneuvers and any complications from them, the time spent with each maneuver, the time between delivery of the head and body, resuscitation required and what was used, and the condition of both patients. Include which shoulder was impacted against the pubic symphysis and infant arm movement after delivery in case nerve palsy develops. Avoid the terms mild, moderate, and severe shoulder dystocia. They offer little information and all refer to shoulder dystocia requiring relief. Notify risk management of the issue and delivery. Review the events with the family, explain any nerve palsy present or possible, and answer any questions.

TABLE 164-3 The Complications Associated with Shoulder Dystocia and the Maneuvers to Relieve It

Fetal	Maternal
Brachial plexus injury	Episiotomy tears
Cerebral palsy	Femoral neuropathy
Clavicle fracture	Lacerations
Death	Postpartum hemorrhage
Humeral fracture	Pubic symphysis separation
Hypoxia	Rectovaginal fistula

COMPLICATIONS

Complications can occur for the mother and the fetus from shoulder dystocia (**Table 164-3**).[27] The most common maternal complications are lower genital tract lacerations, postpartum hemorrhage (e.g., secondary to uterine atony or lacerations), and infection.[2,28]

Significant fetal morbidity and mortality are attributable to asphyxia from delayed delivery or trauma sustained during delivery.[10] Perinatal mortality ranges from 2% to 29% in shoulder dystocia. Neonatal morbidity is immediately apparent in 20% of the affected infants.[2] Neonatal trauma may occur in utero secondary to chronic nerve compression from malposition and during the delivery.[10] Birth trauma occurring during shoulder dystocia may include brachial plexus injuries or Erb palsy (6% to 16%), Klumpke palsy, clavicular fractures (5% to 13%), or humeral fractures.[3,5,6,21,22,28,29] Many palsies resolve within 6 to 12 months.

SUMMARY

The successful management of shoulder dystocia requires considerable judgment by the Emergency Physician in a timely fashion. Warning signals (e.g., fetal macrosomia based on clinical estimate, maternal diabetes, and labor disorders) should alert the Emergency Physician to be prepared for possible shoulder dystocia. Any of the maneuvers described will usually resolve shoulder dystocia if performed in a methodical fashion. Begin with the simplest and least invasive maneuver and work toward the more invasive maneuvers. Reduction of the time interval from delivery of the head to delivery of the body is crucial to fetal survival.

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165 Breech Delivery

Sarah Christian-Kopp

INTRODUCTION

The breech presentation exists when the cephalic pole of the fetus is positioned in a longitudinal lie and the buttocks or feet of the fetus enter the maternal pelvis before the head.[1] Management of the breech presentation in labor is an area of much trepidation and controversy, even among seasoned clinicians. **A breech delivery is considered a high-risk obstetric complication that is best handled by an Obstetrician.** There are unavoidable situations when a pregnant woman will present to the Emergency Department in active labor with a fetus in the breech position.[2]

A vaginal breech delivery may be the best delivery option in situations such as advanced labor with eminent delivery, the absence of obstetrical assistance, fetal distress, or umbilical cord prolapse. **Knowledge and preparedness facilitate comfort and promote success in approaching emergent procedures.** The breech delivery is no exception to this rule.

ANATOMY AND PATHOPHYSIOLOGY

The breech presentation may be associated with a variety of maternal and fetal conditions.[1,3,4] Maternal variants that increase the risk of a breech presentation include a small pelvis and uterine anomalies (e.g., bicornuate or septate uterus and uterine fibroids). Fetal conditions associated with a breech presentation include fetal malformations (e.g., hydrocephalus, anencephaly, and fetal masses), genetic abnormalities, low birth weight, neurologic disorders, oligohydramnios, prematurity, and polyhydramnios. Other associated factors include multiparity, multiple gestations, placental abnormalities (e.g., placenta previa), and previous breech presentations.

Prematurity is a risk factor for a breech presentation. The incidence of breech presentations is inversely related to the fetal gestational age.[1,3] Approximately 24% of fetuses are in the breech presentation at 28 weeks of gestation. The fetus usually turns spontaneously to a cephalic presentation so that at term only 3% to 4% are in the breech presentation.[1,3,5]

There are three main types of breech presentation (**Figure 165-1**).[4,5] The most common is the frank breech which accounts for 50% to 73% of breech presentations. The fetus is flexed at the hips and extended at the knees (**Figure 165-1*A***). The fetus is in the "pike" position. The complete breech is the least common type and accounts for approximately 5% to 11% of breech presentations. The fetus is flexed at both the hips and the knees (**Figure 165-1*B***). The footling or incomplete breech accounts for approximately 12% to 38% of breech presentations. The fetus is not completely flexed at one or both knees or hips (**Figure 165-1*C***). This results in one or both feet presenting before the buttocks. The risks of umbilical cord prolapse and prematurity associated with the breech presentation are listed in **Table 165-1**.

A breech presentation may be suspected upon clinical examination. The abdominal examination may reveal the hardness of the fetal head palpable in the fundus rather than above the pelvic inlet. A vaginal examination can confirm this suspicion. An ultrasound examination is recommended to confirm presentation when a breech presentation in labor is suspected and can exclude a fetal abnormality when time permits.[1]

Breech delivery is divided into three categories. These include unassisted or spontaneous expulsion, partial breech extraction, and total breech extraction.[6] Unassisted or spontaneous expulsion of the fetus occurs when there is no assistance from the provider in the delivery of the infant. This generally occurs only with very premature infants or in precipitous deliveries where the baby delivers so rapidly as not to allow the provider time to arrive.

Partial breech extraction is spontaneous delivery of the infant to the level of the umbilicus followed by assistance from the provider. This is the usual manner of breech delivery. **Allowing the fetus to descend naturally into the pelvis avoids deflexion of the fetal head, decreases the incidence of head entrapment, decreases the incidence of nuchal arms, and decreases the incidence of umbilical cord prolapse. This is the preferred method of delivery for the Emergency Physician confronted with an actively laboring breech presentation and little hope of obtaining an Obstetrician before delivery.**

FIGURE 165-1. The main types of breech presentations. ***A.*** The frank breech. ***B.*** The complete breech. ***C.*** The incomplete breech.

Total breech extraction occurs when the provider reaches into the uterus and pulls or extracts the fetal feet into the vagina and through the vulva followed by the assisted delivery of the remainder of the infant. Total breech extraction is indicated when expedient delivery is preferred in the absence of an experienced Obstetrician to perform a cesarean section and if performed for the breech presentation of a second twin, acute and profound fetal distress, and/or umbilical cord prolapse.[3]

The breech presentation has been associated with an increased risk of cerebral palsy and perinatal mortality regardless of the mode of delivery. Failure to adopt the cephalic presentation may in some cases be a marker for preexisting fetal impairment or a result of fetal distress during the birthing process.[7] The cesarean section has been suggested as the way to reduce perinatal problems associated with the breech presentation.[8] Cesarean section has become the normal mode of breech delivery in many European countries and in North America. The rate of cesarean section for the breech presentation in the United States has increased from 12% in 1970 to 87% in 2002.[9] Prospective and retrospective outcome studies over the past several decades have disagreed with this practice.[10-13]

The American College of Obstetricians and Gynecologists (ACOG) recommends that the decision regarding the mode of delivery should be based on the experience of the provider. It is recognized that cesarean delivery will be the preferred mode of delivery. The Term Breech Trial in 2000 compared planned cesarean deliveries with planned vaginal deliveries.[14] It noted that neonatal mortality, perinatal mortality, and serious neonatal morbidity were significantly lower among the planned cesarean delivery group compared with the planned vaginal delivery group. There are circumstances in which a vaginal breech delivery will be preferable (e.g., an obstetric indication, patient preference, or a precipitous delivery).[14]

Obstetricians will often perform external cephalic version prior to the delivery date if a breech presentation is identified during prenatal visits.[1,3,13] **This technique is reserved for the experienced Obstetrician as it can be associated with significant complications. External cephalic version cannot be performed if the mother is in active labor and the fetus is engaged in the maternal pelvis.**

TABLE 165-1 Breech Presentations and Associated Complications[1]

Breech presentation	Breech deliveries (%)	Umbilical cord prolapse (%)	Prematurity (%)
Frank	50–73	0.5	38
Complete	5–11	5	12
Incomplete	12–38	14	50

INDICATIONS

Vaginal breech delivery by the Emergency Physician is indicated when the mother is in active labor and an Obstetrician is not available to perform the delivery or a cesarean section.

CONTRAINDICATIONS

There are no agreed upon absolute contraindications to a vaginal breech delivery. Recommendations have been made based upon factors that increase morbidity and mortality in vaginal and cesarean breech deliveries.[1,3,6,11] Maternal risk factors for increased morbidity and mortality with a vaginal breech delivery include arrest of labor, pelvic anomalies, a small pelvis, uterine malformations, and uterine masses. Fetal risk factors for increased morbidity and mortality with a vaginal breech delivery include extremes of fetal weight, fetal head extension, prematurity, non-frank breech, and a nonreassuring fetal heart rate pattern.

There are factors regarded as unfavorable for a vaginal breech birth.[15-17] Contraindications include arrest of active labor, a clinically inadequate maternal pelvis, a compromised fetal condition, and placenta previa. Do not vaginally deliver large infants (i.e., larger than 4000 gm) and low birth weight infants (i.e., smaller than 2500 gm) in the breech position. The footling breech presentation can result in more complications than other breech presentations when delivered vaginally. A hyperextended fetal neck makes delivery more complicated and risks injury to the fetus. Do not vaginally deliver a breech fetus unless necessary if inexperienced with the breech delivery technique.

Many of these conditions are not known in an emergent breech presentation. The Emergency Physician may not have the time or experience to assess these conditions. Emergent cesarean delivery is indicated if any of the conditions listed above are known and the setting permits it.

The normal progress of labor with breech presentations has not been extensively evaluated. Poor progress in the active phase of labor may be a sign of fetopelvic disproportion. Failure of the breech to descend once the cervix is completely dilated should be managed with a cesarean section.[15-17]

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Electronic fetal monitor
- Delivery instrument pack (Chapter 162)
- Piper forceps
- Towels
- Clock or timer watch
- Sterile gowns and gloves
- Chromic or Vicryl suture, 2–0 or 3–0
- Richardson or right-angle retractors
- Ring forceps
- Straight Mayo scissors
- Umbilical cord clamps
- 8 inch needle driver
- Sterile drapes

PERSONNEL

- Two medical practitioners if available
- Nursing personnel
- Timekeeper
- Anesthesiologist or Anesthetist if available
- Pediatric or neonatal team if available

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain a verbal informed consent for the procedure and document this in the medical record. Perform an abdominal and vaginal examination to assess the fetal position.[5] Apply a fetal monitor and tocodynamometer to the maternal abdomen. Perform ultrasonography if available to confirm the fetal position, to estimate fetal weight, and to evaluate for any gross fetal anomalies.

Epidural anesthesia or a spinal block by an Anesthesiologist is recommended if available and time permits. A pudendal nerve block with local perineal infiltration may be used as an alternative (Chapter 163). Place the mother in the lithotomy position. Scrub the perineum with povidone iodine solution, chlorhexidine solution, or antibacterial soap. Apply a mask with eye protection or goggles, a sterile gown, and sterile gloves. Apply sterile drapes to isolate the patient's perineum (**Figure 162-10**).

ASSISTED FRANK BREECH DELIVERY

This method is appropriate for all breech presentations. The fetus enters the maternal pelvis with the bitrochanteric diameter in an oblique position (**Figure 165-2*A***). The sacrum is the point of designation for these presentations. The fetus rotates with labor and descent so that the bitrochanteric diameter is in the anteroposterior axis and the sacrum is in the transverse axis (**Figure 165-2*B***).

Encourage maternal "pushing" to expel the fetal buttocks (**Figure 165-3*A***). Support the fetal buttocks with a toweled hand. **Do not assist with the delivery of the buttocks as they emerge (Figure 165-3*B*). Allow the fetus to deliver spontaneously to the level of the umbilicus with only maternal uterine propulsive efforts.** Early extraction of the buttocks increases the risk of head entrapment in a partially dilated cervix, results in deflexion of the fetal head, and increases the risk of nuchal arm entrapment. Perform a midline episiotomy when the buttocks crown (Chapter 163). **Do not perform the episiotomy too early. This may lead to excessive maternal blood loss.**

Proceed with the delivery of the fetal legs when the umbilicus emerges (**Figure 165-4**). The legs will likely have delivered themselves by this time if the fetus is in the complete or incomplete breech position. Insert the fingers of the dominant hand along the long axis of the medial fetal thigh (**Figure 165-4*A***). Apply laterally directed pressure on the fetal thigh to flex the knee and externally rotate the leg out and down through the vulva (**Figure 165-4*A***). Apply simultaneous opposite rotation on the fetal hip and pelvis. Deliver the other leg in a similar manner (**Figure 165-4*B***). **Rotation of the hip in a direction opposite the direction of knee flexion facilitates the delivery of the distal extremities. Therefore, rotate the left leg clockwise and the right leg counterclockwise.**

Prepare to deliver the fetal arms (**Figure 165-5**). Place a sterile towel around the legs and trunk of the fetus for support (**Figure 165-5*A***). Continue to support the fetal body and encourage maternal pushing efforts. **Do not actively assist in the delivery until the fetal scapulae are visible. Aggressive extraction of the fetus following the delivery of the legs may cause deflexion of the vertex or nuchal entrapment of the arms.**

Proceed with active delivery of the shoulders and arms as the scapulae emerge from the vagina (**Figure 165-5**). Rotate the fetal trunk to present the anterior arm and shoulder (**Figure 165-5*A***). Insert the fingers of the dominant hand longitudinally along the humerus of the presenting arm (**Figure 165-5*A***). Apply traction to the humerus to flex the elbow. Sweep the arm across the chest and deliver it through the mother's vulva (**Figure 165-5*B***). Rotate the fetus in a manner to bring the alternate shoulder into the anterior position (**Figure 165-5*C***). Gently rotate the fetus clockwise to deliver the left arm and counterclockwise to deliver the right arm. This direction of motion prevents the arm from becoming entrapped on the neck. Repeat the procedure to deliver the second arm.

The fetal shoulders and arms may not easily deliver using the above technique. Perform this alternate technique if the arms and shoulders do not deliver. Place the thumbs of both hands over the fetal posterior iliac spines and sacroiliac area. Place the palms and fingers over the fetal hips. **Apply pressure only over the iliac spines. Never grasp the fetal abdomen. The adrenal glands, kidney, liver, and spleen may be injured with excessive pressure during the delivery process.** Elevate the fetal body to deliver the posterior shoulder over the more pliable posterior perineum. Deliver the posterior arm as described above. Lower the fetal trunk. Deliver the anterior shoulder from under the maternal pubic symphysis as described above.

The fetal vertex will generally rotate into the anteroposterior orientation after delivery of the arms and shoulders. The fetal vertex will lie against the maternal pubic symphysis. The fetal chin will lie in the posterior aspect of the vagina and/or lower uterine segment. Wrap the fetal arms and trunk in a towel. Encourage maternal pushing efforts. The fetal head may deliver spontaneously. Note when the fetal chin and mouth appear at the posterior perineum. **Instruct the mother to stop pushing.** Suction the fetal mouth. Gently lift the infant upward to deliver the head in a controlled manner (**Figure 165-6**). **Avoid hyperextending the fetal back.**

Complete the delivery of the infant. Clamp and cut the umbilical cord. Place the infant in the warmer for examination and resuscitation. The Apgar scores for infants delivered from a breech position are generally slightly lower than from a vertex delivery. The infant may show some initial signs of short-term and generally clinically insignificant hypoxia.

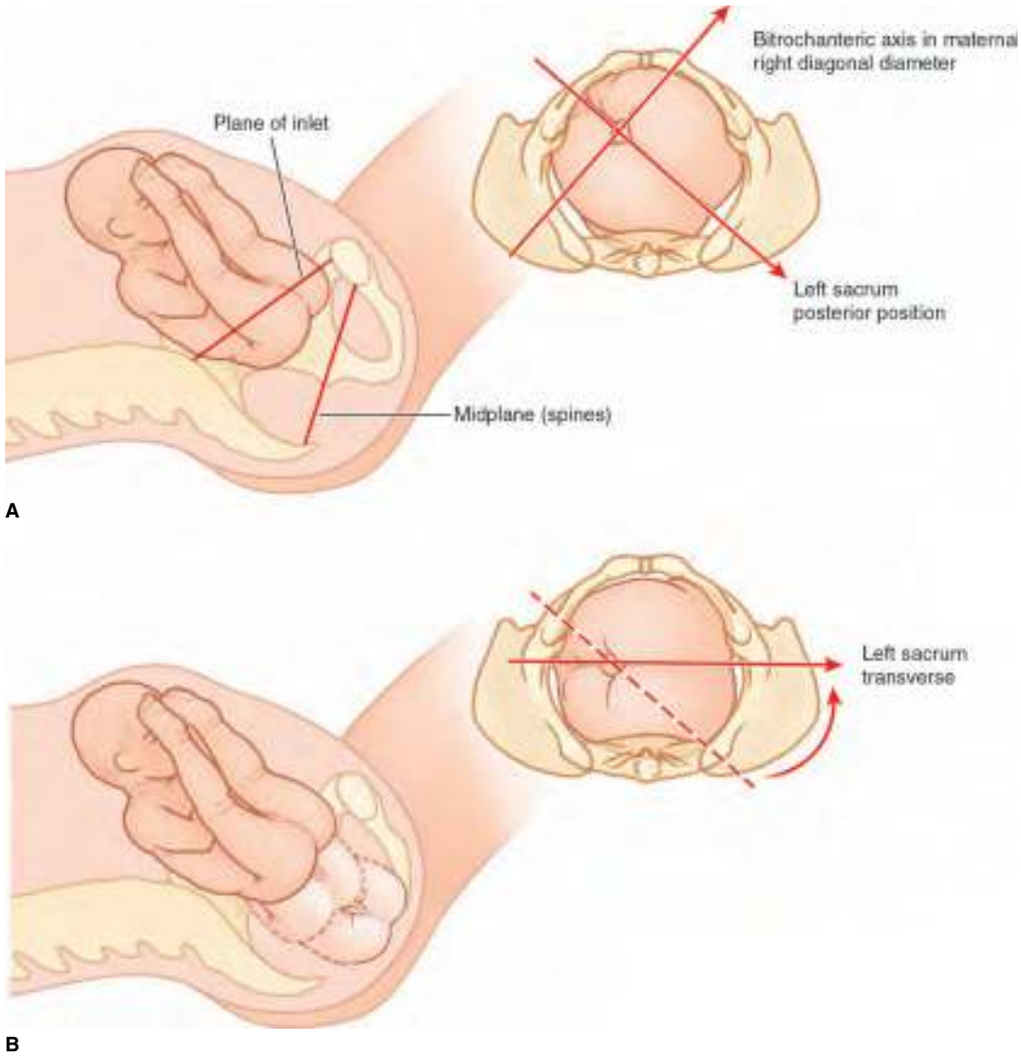


FIGURE 165-2. Breech engagement of the maternal pelvis. ***A.*** The bitrochanteric diameter is aligned with one of the diagonal diameters. ***B.*** Descent causes the bitrochanteric diameter to rotate into the anteroposterior axis and the sacrum to rotate into the transverse axis.

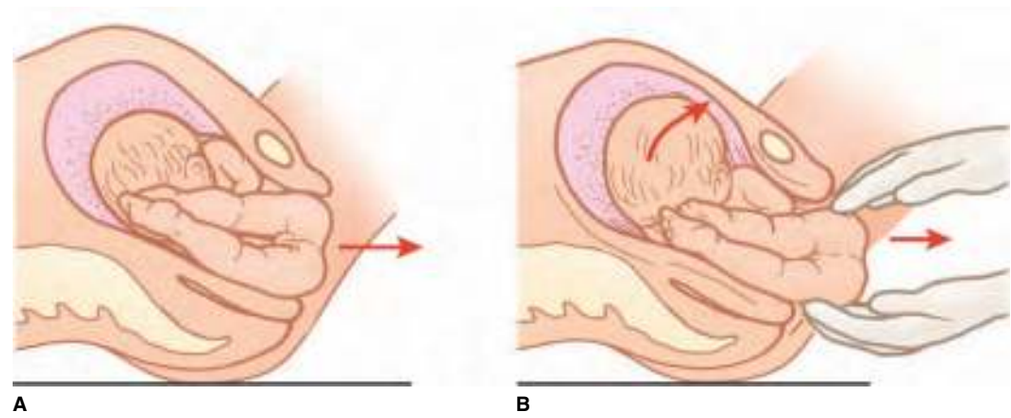


FIGURE 165-3. Delivery of the buttocks. ***A.*** Uterine contractions result in spontaneous emergence of the buttocks while maintaining cephalic flexion. ***B.*** Premature traction can result in deflexion of the vertex, head entrapment, or nuchal arms.

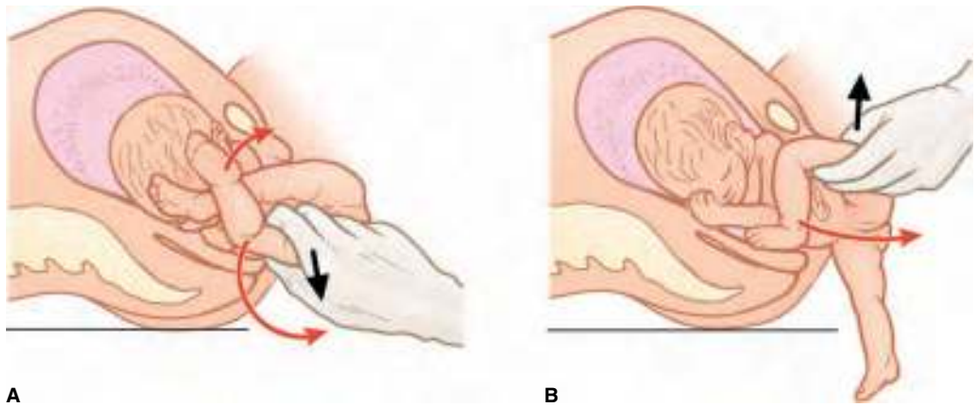


FIGURE 165-4. Delivery of the legs. ***A.*** Apply laterally directed pressure on the medial thigh with opposite rotation of the pelvis to deliver the leg. ***B.*** Repeat the procedure to deliver the other leg.

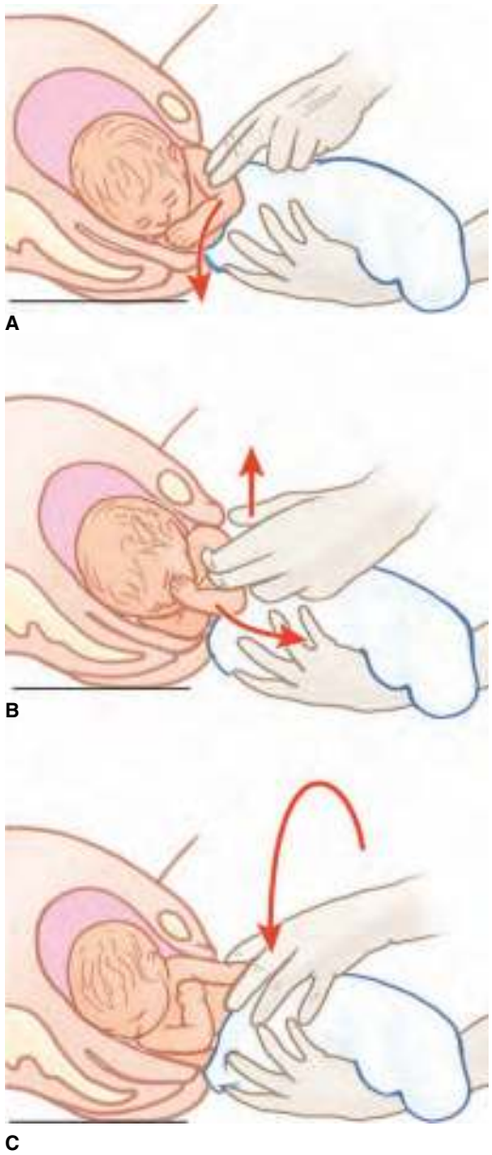


FIGURE 165-5. Delivery of the arms. ***A.*** Insert the dominant hand along the humerus of the anterior arm. Apply traction (*arrow*) to flex the elbow. ***B.*** Deliver the arm. ***C.*** Gently rotate the fetus so that the other arm presents anteriorly.

DELIVERY OF THE FETAL HEAD

Cervical entrapment (i.e., inadequate cervical dilation) is a more common occurrence in the delivery of a fetus in the breech presentation. It is most frequently observed when rapid labor results in the fetal trunk being delivered through a partially dilated cervix or during the delivery of a premature breech when the fetal head is relatively larger than the fetal trunk.

The fetal head often delivers spontaneously with maternal pushing efforts but may occasionally fail to do so. Several options exist to aid in the delivery of the fetal head. These include manual extraction, manual extraction with the McRoberts maneuver or the Mauriceau-Smellie-Viet maneuver, forceps-assisted delivery, and Dührssen's incisions.

Adequate anesthesia is vital to the extraction of the fetal head. A pudendal block and peroneal infiltration should have been performed previously. If not, consider the administration of parenteral sedation and analgesia (Chapter 159) and local infiltration. Inhaled halogenated agents, intravenous beta-mimetics (i.e., terbutaline or ritodrine), and intravenous nitroglycerin are all useful for uterine relaxation in the event of entrapment of the fetal head or if intrauterine manipulation is necessary. Use assistants as needed to offer adequate visualization. Richardson or Pratt retractors may be placed inside of the vagina to maximize the view.

McROBERTS MANEUVER

Apply the McRoberts maneuver if the fetal head does not deliver. The McRoberts maneuver is easy to perform (**Figure 164-7**). Place an assistant on each side of the patient. Hyperflex the mother's thighs onto her abdomen (**Figure 164-7*A***). Instruct the assistants

FIGURE 165-6. Delivery of the fetal head.

FIGURE 165-7. The Mauriceau-Smellie-Viet maneuver.

to maintain support of the hyperflexion while applying suprapubic pressure (**Figure 164-7*B***). This results in flattening of the lumbosacral curve with rotation of the pubic symphysis cephalad (**Figure 164-7*C***). Perform the manual extraction.

MAURICEAU-SMELLIE-VIET MANEUVER

Perform the Mauriceau-Smellie-Viet maneuver if the McRoberts maneuver is unsuccessful in the spontaneous delivery of the head (**Figure 165-7**). Rest the body of the infant on the dominant arm. Place the index and middle finger of the dominant hand on the fetal maxilla. Apply pressure to the maxilla to maintain the fetal head in flexion. **Do not place the fingers on the fetal mandible or in the fetal mouth.** Place the nondominant hand on the posterior aspect of the fetal neck and shoulders. Slightly elevate the fetal body from the horizontal plane.

Exert continued and gentle downward traction with both hands until the occiput moves under the mother's pubic symphysis. **Never hyperextend the fetal trunk to avoid spinal cord injuries. Never apply pressure to the mandible or the fetal mouth during this maneuver. The force applied to the mandible or mouth can dislocate the mandible.** Instruct an assistant to apply firm and gentle suprapubic pressure in conjunction with gentle downward traction by the Emergency Physician. Continue this process until the head delivers.

FORCEPS-ASSISTED DELIVERY OF THE HEAD

The utilization of forceps may be warranted if none of these maneuvers is successful.[16,18] The Piper forceps have a minimal pelvic curve and allow direct application to the fetal head. They are used solely for the delivery of the head in a breech delivery. **Forceps-assisted delivery can result in significant injury to the fetus and the mother. The use of the Piper forceps is not recommended unless the Emergency Physician has training and experience in their proper use.**

Apply the Piper forceps. Instruct an assistant to hold and elevate the fetal trunk slightly above the horizontal plane (**Figure 165-8*A***). Kneel below the fetus. Insert the forceps directly into the vagina and along the fetal head. Apply the right forceps blade. Grasp the blade in the left hand. Place the right hand along the left fetal parietal bone between the fetal head and the right maternal pelvic side wall. Insert and apply the forceps blade. Instruct the assistant to hold the blade in this position. Apply the left forceps blade. Grasp the blade in the right hand. Place the left hand along the right fetal parietal bone between the fetal head and the left maternal pelvic side wall. Insert and apply the forceps blade. Gently join the forceps handles while avoiding excessive pressure.

Apply gentle downward and outward traction to the forceps blades to deliver the fetal head (**Figure 165-8*B***). Slowly elevate the plane of the fetal trunk toward the maternal abdomen as the fetal face delivers. **Do not hyperextend the fetal neck.** Stop the extraction as the mouth appears. Gently suction the fetal mouth. Deliver the head.

DÜHRSSEN'S INCISIONS

Perform Dührssen's incisions if the fetal head is entrapped, if other methods of extraction have failed, or if you are unfamiliar with the use of forceps (**Figure 165-9**). Dührssen's incisions consist of two to four incisions placed circumferentially around the cervix. **The two required incisions are at the 10 and 2 o'clock positions.** Additional incisions can be made at the 5, 6, and/or 7 o'clock positions. **The cervix must be more than 70% effaced and dilated more than 6 cm for the procedure to be successful and to prevent significant hemorrhage.**

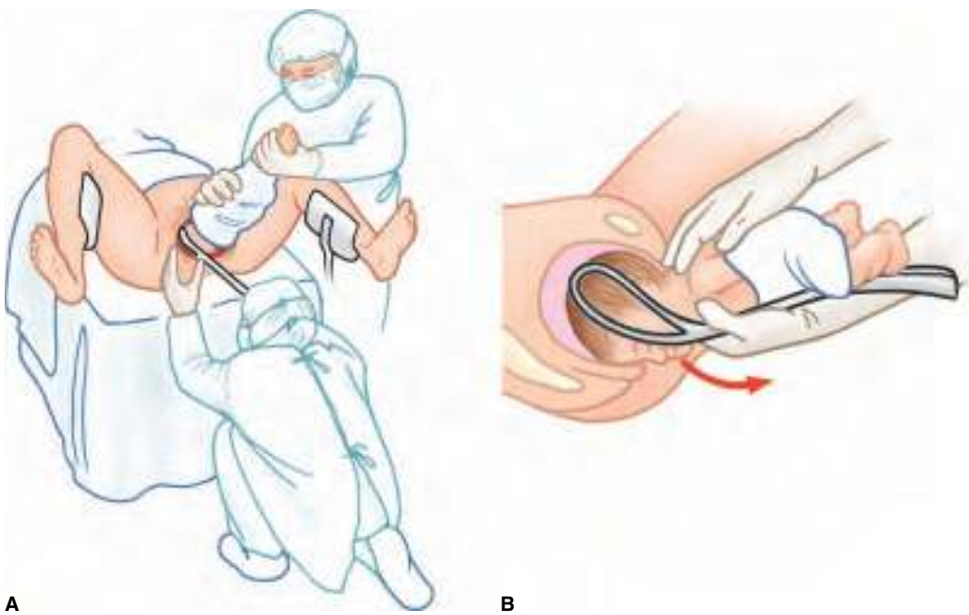
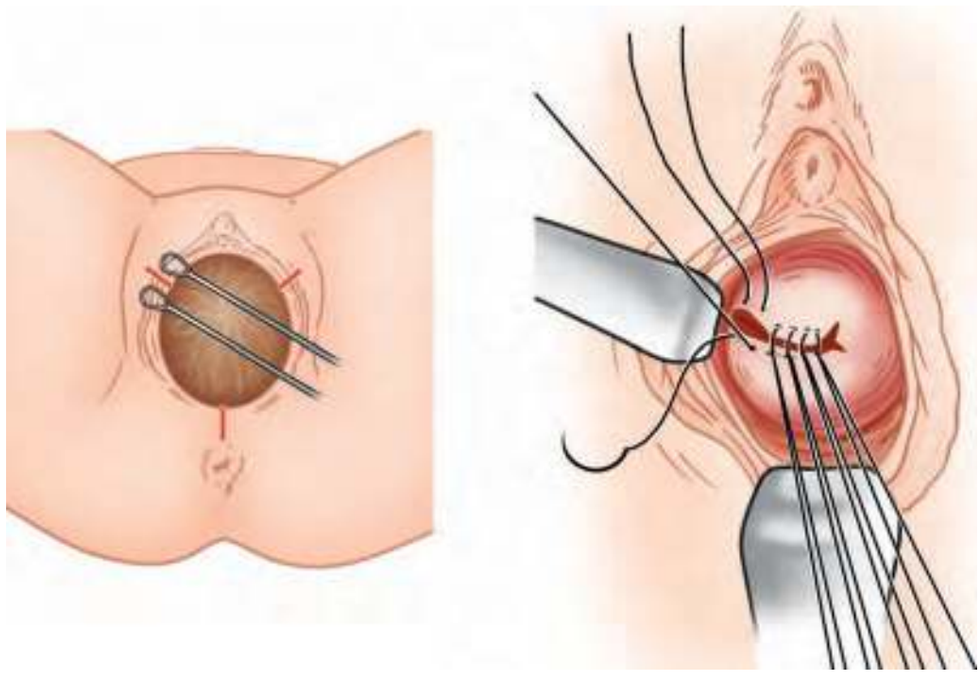


FIGURE 165-8. Forceps-assisted delivery of the head. ***A.*** Application of the forceps. ***B.*** Delivery of the fetal head.



A B

FIGURE 165-9. Dührssen's incisions. ***A.*** The incisions. ***B.*** Repair of the incisions.

Grasp two pairs of ring forceps. Grasp the cervix around the point of the incision. For example, place ring forceps at the 9 and 11 o'clock positions to make the 10 o'clock incision (**Figure 165-9*A***). Make the incisions 2 to 3 cm in length with straight Mayo scissors. Deliver the fetal head.

PERSISTENT FETAL HEAD ENTRAPMENT

The fetal head may fail to deliver despite the maneuvers described above. True cephalopelvic disproportion must be differentiated from cervical entrapment (i.e., inadequate cervical dilation). The options are limited in the rare case of a true cephalopelvic disproportion. The Emergency Physician can perform a symphysiotomy (Chapter 168), the fetal body can be pushed back into the uterus and a cesarean section performed, or an experienced Obstetrician may perform a destructive procedure if the fetus is nonviable.

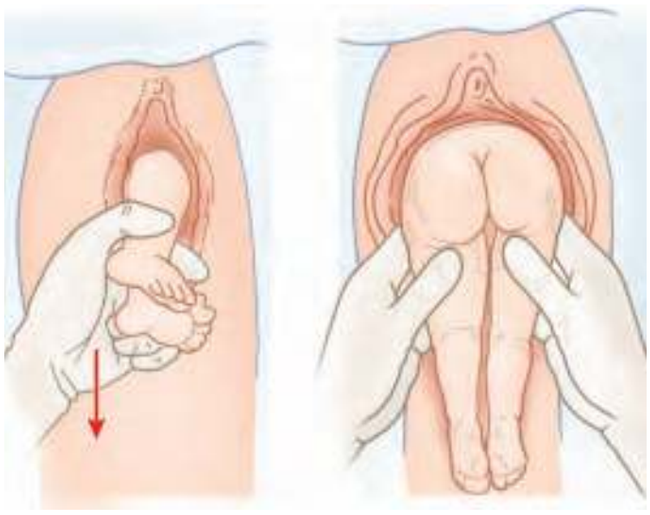
TOTAL BREECH EXTRACTION

Breech extraction is to be performed only in the rare instances of a second twin presenting in a breech position, extreme fetal distress without the capability of performing a cesarean section, and/or umbilical cord prolapse that does not allow time for setup or performance of a cesarean section.[3,18-20]

Use ultrasound if available to identify the fetal legs and feet. This will ensure grasping of the appropriate extremity. Reach into the lower uterine segment with the dominant hand and firmly grasp the fetal feet. Place the fingers around the fetus's ankles with the index finger between the two ankles (**Figure 165-10*A***). Apply continuous and firm traction in a downward and outward direction. Deliver the fetal feet and legs through the vaginal opening (**Figure 165-10*A***). Continue to apply traction to deliver the fetus to the level of the buttocks (**Figure 165-10*B***). Instruct an assistant to support the fetus. Perform a midline episiotomy if necessary. Deliver the fetus to the level of the umbilicus. Grasp the bony sacrum and pelvis. The remainder of the technique is as described previously.

COMPLETE AND INCOMPLETE BREECH DELIVERIES

Complete and incomplete breech deliveries may occur in the same fashion as the frank vaginal breech delivery. The one exception is that one or both feet may already be extended and not require attention.[3] There is an increased risk of head entrapment, umbilical cord entanglement, and umbilical cord prolapse.[3,10,13] This is the basis of the recommendation for cesarean delivery in these cases.[3,10,13] One randomized trial reported non-frank vaginal breech delivery to be relatively safe.[3,13]



A B

FIGURE 165-10. Breech extraction. Reach into the uterus and grasp the fetal ankles. ***A.*** Apply traction to deliver the feet and ankles. ***B.*** Delivery of the buttocks.

ASSESSMENT

The two patients must now be assessed and treated.[21] A second Physician or neonatal team can evaluate and resuscitate the infant if available.[21] Determine the infant's Apgar score. Examine the infant for birth trauma and signs of prematurity (e.g., fusing of eyelids), as both correlate with perinatal morbidity and mortality.

Perform the maternal assessment simultaneously with that of the neonate. Offering reassurance to the mother while delivering the placenta (Chapter 162) and evaluating for postpartum hemorrhage (Chapter 166) is of the utmost importance.

AFTERCARE

The Apgar scores for infants delivered from a breech position are generally slightly lower than from a vertex delivery. The infant may show some initial signs of short-term and generally clinically insignificant hypoxia.

Postpartum recovery for the mother is generally uneventful. Observe her for evidence of infection or symptomatic anemia. Monitor urinary output for the presence of urinary retention if regional anesthesia is used or if there is significant perineal trauma.

Deliver the placenta and repair the cervix if there are any lacerations or Dührssen's incisions. An episiotomy, perineal laceration, and/or vaginal laceration must be repaired (Chapter 163). Initiate uterine massage. Administer Pitocin intramuscularly following delivery of the placenta.

It is vital that postpartum hemorrhage be managed to prevent maternal morbidity and mortality. Grasp the base of each cervical incision or laceration to decrease the bleeding. Do not waste time attempting to locate it if the distal angle is difficult to visualize. Apply traction to the proximal edges of the incision or laceration with ring forceps. Alternatively, place 2–0 chromic, Dexon, or Vicryl sutures along the edges of the cervical incision or laceration that can be visualized. Place progressively proximal interrupted sutures until the base is completely visualized (**Figure 165-9*B***). Apply traction to the sutures to better visualize the cervical incision or laceration.

COMPLICATIONS

Maternal complications associated with the performance of a vaginal breech delivery include episiotomy extension, hematomas (i.e., vaginal or pelvic), infection, lacerations (i.e., cervical, perineal, and/or vaginal), and postpartum hemorrhage.

Neonatal complications include anoxia (i.e., perinatal asphyxia), arrest of head decent, brachial nerve palsy, cephalohematomas, cerebral hemorrhage, cerebral palsy, death, fractures (e.g., clavicular, cranial, and femoral), hypoxia, lacerations, spinal cord injuries from head hyperextension, and umbilical cord prolapse.[1,3,10,11,13,22-27] Studies suggest that the occurrence of these complications may be greatly influenced by the urgency of the delivery rather than solely the method of delivery.[3] An adequately prepared Emergency Physician may not be able to prevent these complications but will less likely contribute to them.

There are complications that are unique to the breech presentation. Head entrapment is a potentially serious complication of breech delivery. The head is often the largest part of the fetus. There is no time for molding of the breech head. Umbilical cord compressions can contribute to fetal acidosis, hypoxia, and death if the fetal head does not deliver easily after delivery of the torso. Attempts to expedite delivery can result in maternal and fetal trauma. The risk of head entrapment is especially high in the preterm infant before 30 weeks as the head is large in relation to the body.[28]

Non-frank breech presentation has an increased incidence of umbilical cord prolapse as the presenting part may not completely occlude the cervix. The risks of umbilical cord prolapse and prematurity associated with the breech presentation are listed in **Table 165-1**.

SUMMARY

Perinatal morbidity and mortality are improved with a planned cesarean section when compared to a planned vaginal delivery for the delivery of a breech infant. A cesarean delivery may not be an option in the emergency setting. Increased morbidity and mortality may be seen with estimated fetal weights of less than 1500 gm or greater than 4000 gm, single or double footling presentation, a diminished maternal pelvis, cephalic hyperextension, or in the hands of an inexperienced Emergency Physician. Cesarean section is preferable in these instances if available. The Emergency Physician managing an imminent breech delivery should be well aware of the potential complications, the relative indications for cesarean delivery, and the techniques and tools for vaginal delivery to lessen the inherent risks to the infant and mother.

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166 Postpartum Hemorrhage Management

Leah W. Antoniewicz

INTRODUCTION

Postpartum hemorrhage, or excessive blood loss following delivery, is the leading cause of maternal death worldwide. It is traditionally defined as blood loss greater than 500 mL after vaginal delivery and 1000 mL after a cesarean section.[1,2] This is impractical because the normal blood loss is believed to be 300 to 600 mL following a vaginal delivery and 900 to 1200 mL following a cesarean section.[3,4] Postpartum hemorrhage has also been defined as blood loss that results in a decrease in the hematocrit of greater than 10 points between admission and the postpartum period which corresponds to the 97th percentile of vaginal and 92nd percentile of cesarean deliveries.[1,3] A clinically useful definition is excessive bleeding that results in signs and/or symptoms of hypovolemia (e.g., dizziness, hypotension, oliguria, palpitations, syncope, shortness of breath, and tachycardia), which corresponds to a 10% or more loss in total blood volume.[4] Postpartum hemorrhage can occur at sites within or external to the genitourinary tract (**Table 166-1**).

The incidence of postpartum hemorrhage ranges from 2% to 3% of pregnancies in the United States.[5] The risk factors for postpartum hemorrhage are listed in **Table 166-2**. The etiologies are varied (**Table 166-3 and Figure 166-1**). Primary postpartum hemorrhage accounts for greater than 90% of all cases and occurs within 24 hours of delivery. Uterine atony (i.e., lack of uterine contraction) is by far the most common cause. This is often the result of excessive bleeding from the placental implantation site. Trauma (e.g., genital tract lacerations, surgical incisions, or uterine rupture) is the second most common etiology. Hemorrhage can be associated with a considerable drop in hematocrit before clinical symptoms occur and result in significant maternal complications.

Secondary postpartum hemorrhage occurs more than 24 hours after delivery and up to 12 weeks postpartum. It is usually the result of excessive bleeding from the placental implantation site or retained products of conception but can also be caused by an infection or coagulation defects.[6,7] This chapter reviews the pathophysiology of early postpartum hemorrhage, discusses the diagnosis and assessment of postpartum hemorrhage, and concludes with strategies for treatment.

TABLE 166-1 Anatomic Sites of Postpartum Hemorrhage

Anus
Broad ligament
Cervix
Contractile tissue from previous uterine incision
Episiotomy incisions
Lower urinary tract (i.e., periurethral area, urethra, bladder)
Noncontractile or poorly contractile tissue (i.e., lower uterine segment)
Perineum (e.g., perineal body or rectum)
Placental implantation site
Rectum
Tissue tears
Vagina (e.g., anomalous septa, fornices, hymen, and side walls)

TABLE 166-2 Risk Factors for Postpartum Hemorrhage

Advanced age
African American
Anemia
Hypertension
Lack of prenatal care
Liver disease
Obesity
Precipitous delivery
Prolonged labor
Uterine distension

ANATOMY AND PATHOPHYSIOLOGY

The most common causes of postpartum hemorrhage are uterine atony (70% to 90%), genital tract trauma (5% to 8%), retained products of conception (3% to 5%), and hematologic or coagulopathic abnormalities (< 2%). Uterine inversion is a rare cause of postpartum hemorrhage. Risk factors for uterine atony include anesthesia, augmented or induced labor, chorioamnionitis, high parity, overdistention of the uterus, previous postpartum hemorrhage, prolonged third stage of labor, and use of uterine relaxing agents.[1] Genital tract

TABLE 166-3 Etiologic Associations for Postpartum Hemorrhage

Altered maternal anatomy	Pitocin use, prolonged
Altered uterine contractility	Placental abruption
Asian ethnicity	Placenta previa
Cesarean section dehiscence or rupture	Postpartum hemorrhage history
Chorioamnionitis	Precipitous delivery
Coagulopathy	Preeclampsia
Compound delivery	Prolonged or tumultuous labor
Disseminated intravascular coagulation	Reproductive tract anomalies
Dührssen's incisions	Retained placental fragments
Endometritis	Shoulder dystocia
Episiotomy	Tissue lacerations
Fetal scalp electrode injury	Tocolytic use
General anesthesia	Trauma
Grand multiparity	Uterine anomalies
Hispanic ethnicity	Uterine atony
Infection	Uterine inversion
Intrauterine pressure catheter injury	Uterine leiomyomata
Myomectomy dehiscence or rupture	Uterine pressure monitor injury
Occiput posterior delivery	Uterine rupture
Operative delivery	von Willebrand's coagulopathy

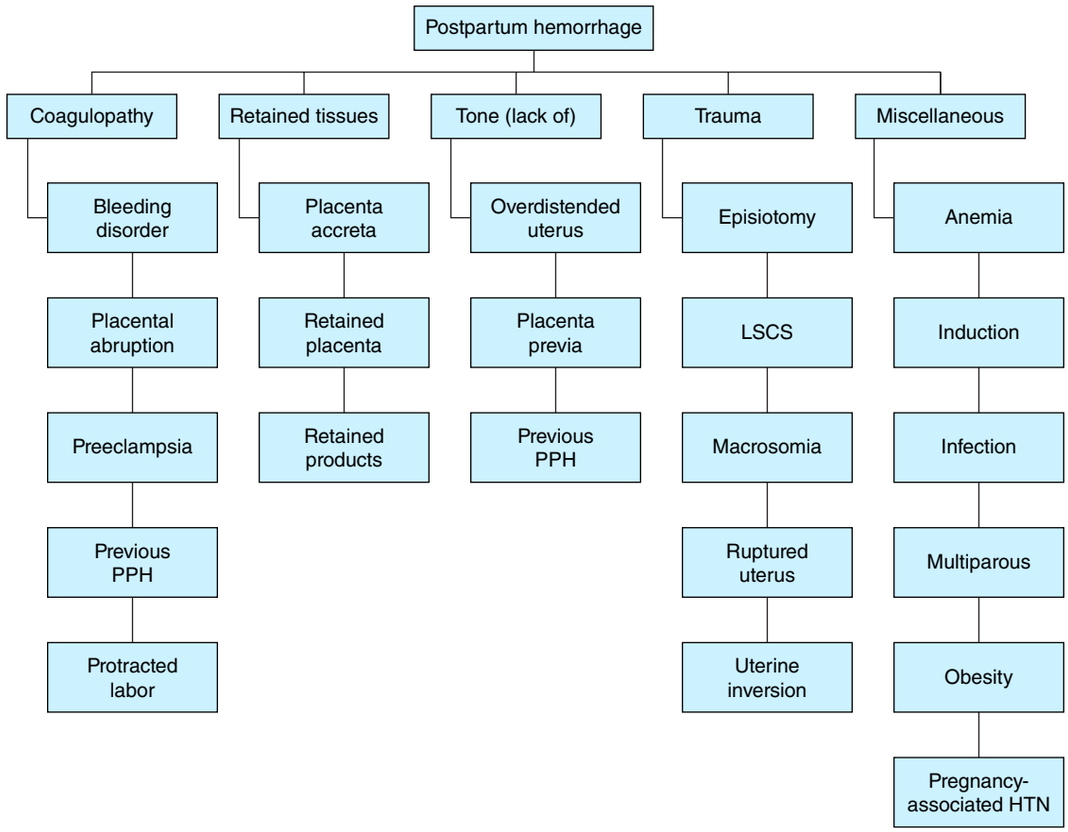

FIGURE 166-1. Some of the more common etiologies of postpartum hemorrhage. HTN, hypertension; LSCS, lower segment cesarean section; PPH, postpartum hemorrhage.

injuries include episiotomies, hematomas (e.g., broad ligament, ischiorectal fossa, vaginal, and vulvar), lacerations (e.g., uterine or vaginal), and uterine rupture. Retention of all or part of the placenta interferes with uterine contraction and results in continued bleeding from the placental implantation site. Hematologic abnormalities include disseminated intravascular coagulation, von Willebrand's disease, and other less common disorders (i.e., acquired, congenital, or inherited).

The plasma volume increases in a normal pregnancy by approximately 40% and the red cell mass by 25%, resulting in a hypervolemia of approximately 1500 to 2000 mL.[6,8] This compensates for typical blood losses. **Visual estimates of blood loss are inaccurate, especially if blood is mixed with amniotic fluid. Weigh all pads for an accurate assessment and use 1 gm = 1 mL of blood.** The signs and symptoms of postpartum hemorrhage include diaphoresis, dizziness, fatigue, hypotension, measurable blood loss greater than 500 mL, oliguria, pallor, palpitations, shortness of breath, syncope, and tachycardia. Suspect a concealed hemorrhage from a broad ligament hematoma, uterine rupture, or vaginal hematoma if the patient is symptomatic in the absence of external blood loss.

Patients at risk for postpartum hemorrhage require blood typed and crossmatched on admission. Obtain a blood chemistry, coagulation studies (e.g., fibrinogen, international normalized ratio, prothrombin time, partial thromboplastin time, and thromboelastography). Most women who hemorrhage, however, have no risk factors. Leading risk factors associated with early postpartum hemorrhage in vaginal birth include arrest of descent, chorioamnionitis, mediolateral episiotomy, multiple gestations, preeclampsia, previous postpartum hemorrhage, prolonged third stage of labor, and soft tissue lacerations.[1,3] Other risk factors are listed in **Tables 166-2 and 166-3**. Surgical deliveries are often associated with increased blood loss when compared with vaginal deliveries. Inhalation anesthesia, especially with halogenated agents, increases the risk for postpartum hemorrhage and should be used sparingly in high-risk cases.[8,9]

INDICATIONS

All postpartum hemorrhage must be controlled as soon as possible to prevent maternal morbidity and mortality (Figure 166-2).[2] Injuries and lacerations are most common with difficult deliveries (e.g., operative vaginal deliveries and use of an episiotomy). Carefully examine the cervix and vagina for injury immediately following delivery of the infant and placenta. Note these injuries and anticipate the possibility of postpartum hemorrhage.

CONTRAINDICATIONS

There are no absolute contraindications to the management of postpartum hemorrhage. Medication therapies for postpartum hemorrhage for atony may have significant side effects. Avoid undiluted rapid intravenous (IV) infusions of oxytocin which can cause hypotension. Ergots (e.g., methylergonovine) are potent vasoconstrictors and should be administered intramuscularly. They are contraindicated in the presence of cardiac disorders, coronary artery disease, hypertensive disorders, preeclampsia, Raynaud's phenomenon, and scleroderma. Prostaglandin F (PGF) is contraindicated in patients with active asthma as it can incite severe bronchospasm in sensitive individuals.[7] PGF may result in vasoconstriction of the pulmonary bed.[7] The side effects include diarrhea, fever, and tachycardia. Avoid prostaglandin E_2 (PGE_2; i.e., Prostin) in hypotensive patients. Human recombinant factor VIIa (NovoSeven) is an expensive

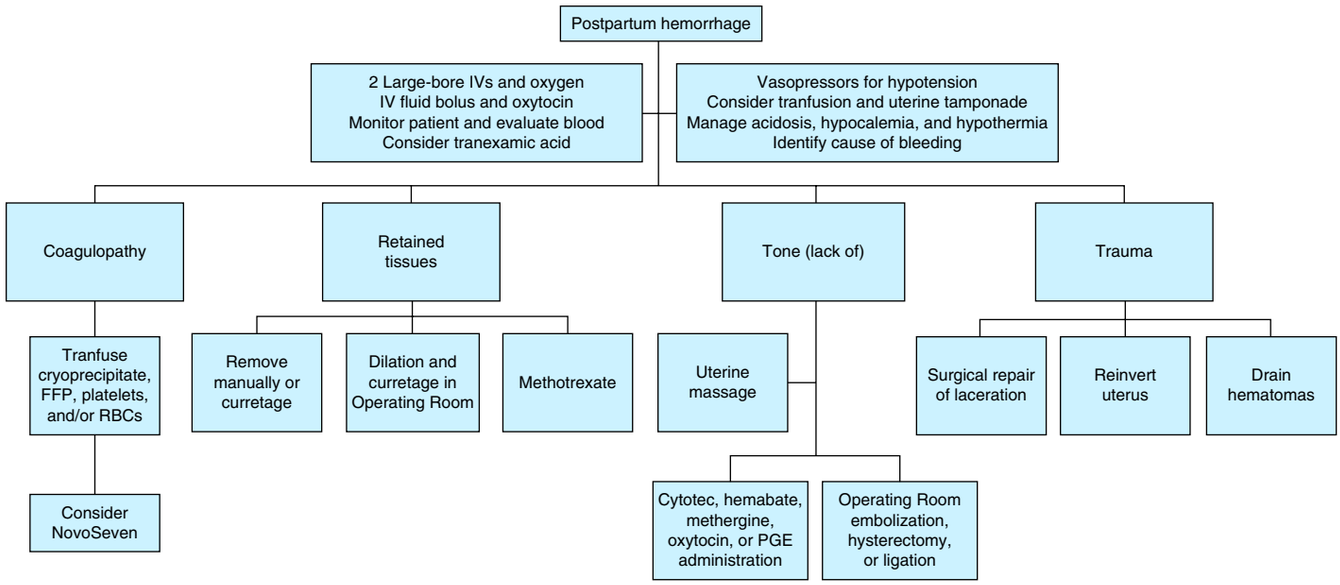

FIGURE 166-2. Treatment of postpartum hemorrhage. FFP, fresh frozen plasma; RBCs, red blood cells.

treatment for life-threatening hemorrhage and can cause subsequent thromboembolism. Ensure the balloons used do not contain latex if allergic and only contain silicone.

Indications for sending the patient directly to the Operating Room are uncommon. **Significant arterial bleeding requires surgery or embolization and not temporizing measures.** Congenital anomalies of the uterus, uterine distortion (e.g., leiomyomas), and uterine rupture (i.e., actual or suspected) require surgical treatment. Purulent infections of the genital tract might be spread by uterine manipulation.

EQUIPMENT

- Sterile gown and gloves
- Face mask with eye shield or goggles
- Surgical hat
- Chlorhexidine or povidone iodine solution
- Foley catheter
- Gauze sponges
- Sponge-tipped applicators, large
- Sterile delivery drapes
- Alcohol swabs
- 16 to 18 gauge needle for IV access
- 10 mL syringe
- 22 to 25 gauge needle for injection of local anesthetic
- Weighted vaginal speculum or Sims retractor
- 2 right angle (Heaney) retractors
- 4 ring forceps
- Straight Mayo scissors or sterile scalpel
- 2–0 or 3–0 suture: monofilament polydioxanone (Monocryl) or polyglactin (Vicryl)
- PPH Butterfly (**Figure 166-3**)
- Bakri Postpartum Balloon, optional (**Figure 166-4**)
- ebb Complete Tamponade System, optional (**Figure 166-5**)
- BT-Cath (**Figure 166-6**)
- Sengstaken-Blakemore tube, optional (Chapter 81)
- 3 Foley catheters if balloon is not available, optional (Chapter 173)
- Poiesis Duette Dual-Balloon (**Figure 166-7**)
- Packing forceps

MEDICATIONS

- Local anesthetic solution (e.g., bupivacaine, lidocaine, or mepivacaine)
- Oxytocin (i.e., Pitocin)
- 15-methyl $PGF_{2\alpha}$ (i.e., Hemabate, Carboprost)
- Methylergonovine maleate (i.e., Methergine)
- Misoprostol or PGE_1 (i.e., Cytotec)
- Dinoprostone or PGE_2 (i.e., Prostin)
- 5000 U of thrombin in 5 mL of sterile saline
- Factor VIIa (i.e., NovoSeven)

PATIENT PREPARATION

Obtain an informed consent if possible and time allows. Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Include the possibility of the Operating Room and a hysterectomy. Obtain a signed informed consent for the procedure and place this in the medical record. Verbal consent can be documented in the medical record if the patient cannot sign for some reason.

The initial management is aimed at stabilizing the mother and identifying the bleeding source.[2] Apply bimanual compression (**Figure 166-8**) which will control most hemorrhage and obtain help. Establish IV access at two sites with 16 to 18 gauge angiocatheters. Type and crossmatch the patient for any required blood components. Obtain a complete blood count, chemistry, and coagulation profile.[10] Place a Foley catheter to monitor urine output and to allow the uterus to contract. Regulate and monitor fluid intake carefully to avoid acute respiratory distress syndrome or dilutional disseminated intravascular coagulation (DIC).

Identification of the origin or site of bleeding is critical.[2] Uterine hypotonia or atony is the most frequent source of hemorrhage. Uterotonics (e.g., oxytocin) are usually administered concurrently with a

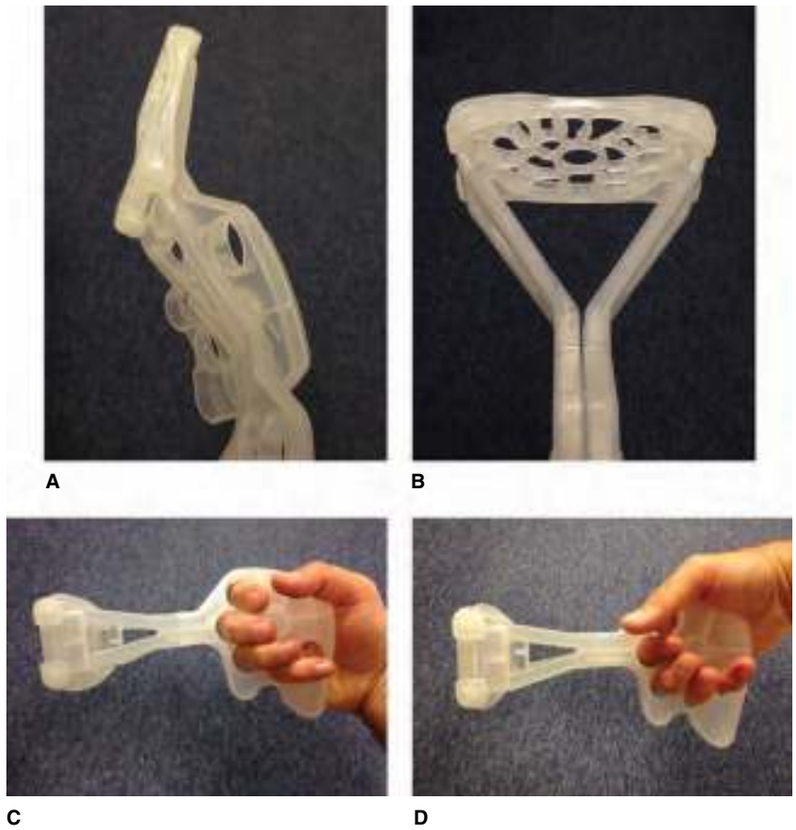

FIGURE 166-3. The PPH Butterfly. ***A.*** The device is folder flat for insertion. ***B.*** The handles are brought together to create the triangular platform. ***C.*** Holding the device longitudinally. ***D.*** The device can be held from above if the patient is on a bed. (Used with permission from reference 23.)

complete examination of the uterus. Palpation will often exhibit a soft and spongy (i.e., boggy) uterus that may be increasing in size due to the accumulation of clots and blood within the endometrial cavity. Clear these to allow for adequate uterine contractions. Continued bleeding from the endometrial cavity through the cervical os is always present. **A well-contracted uterus does not bleed significantly in the absence of a severe coagulopathy.** Examination of the uterus is performed by palpation of the fundus with the nondominant hand and manual exploration of the uterine cavity with the other. Use ultrasound to identify the presence of any clots or placental tissue that may require manual removal and/or curettage.[11]

Continued hemorrhage with a firm uterus indicates bleeding from another source (e.g., cervix, lower uterine segment, the cervix, or the vagina). **Examine all areas carefully, even after hemorrhage is noted at one site, as there may be more than one source of hemorrhage.** Begin at the most distal aspects of the genital tract and work proximally.

FIGURE 166-4. The Bakri Balloon (Cook Medical, Bloomington, IN).

TECHNIQUES

TRANEXAMIC ACID

Tranexamic acid (TXA) is a synthetic derivative of lysine. It is an antifibrinolytic that prevents the conversion of plasminogen to plasmin and inhibits the degradation of fibrin clots by plasmin. TXA can be used prophylactically to prevent postpartum hemorrhage.[12-14] The studies so far have limited information on its prophylactic use. Prophylactic use reduces blood loss, postpartum hemorrhage, and the need for transfusion.[14] It can be used for postpartum hemorrhage management when bleeding is noted.[15-19] The best evidence so far is from the WOMAN trial.[19] TXA decreased deaths and had no adverse effects. Safety regarding long-term effects is not well studied.

UTERINE ATONY

Administer oxytocin and fundal (i.e., transabdominal) massage just after delivery of the placenta. These can usually result in a

FIGURE 166-5. The ebb Complete Tamponade System (Clinical Innovations, Murray, UT).

FIGURE 166-7. The Poiesis Duette (Poiesis Medical LLC, Jupiter, FL).

contracted, firm and rock-like fundus. However, the uterus may never become firm or may relax again.

Begin transabdominal uterine massage for uterine atony to promote uterine muscle contractions. Use one or two hands to palpate the uterus through the abdominal wall and rhythmically press downward in a circular motion. Massage in a firm but gentle manner without pushing the uterus through the birth canal (i.e., inversion or prolapse). **Avoid overly vigorous massage which can injure the broad ligament vasculature.**[8]

Administer an oxytocin infusion at the same time as fundal massage. Inject 40 U of oxytocin into 1 L of sterile IV normal saline

Administer the oxytocin-saline solution over 10 minutes. Alternatively, administer 10 U of oxytocin intramuscularly. Repeat the oxytocin dose if atony persists.

Continued bleeding requires the initiation of bimanual uterine compression. Massage the posterior aspect of the uterus with the

FIGURE 166-6. The BT-Cath (Utah Medical Products Inc., Midvale, UT).

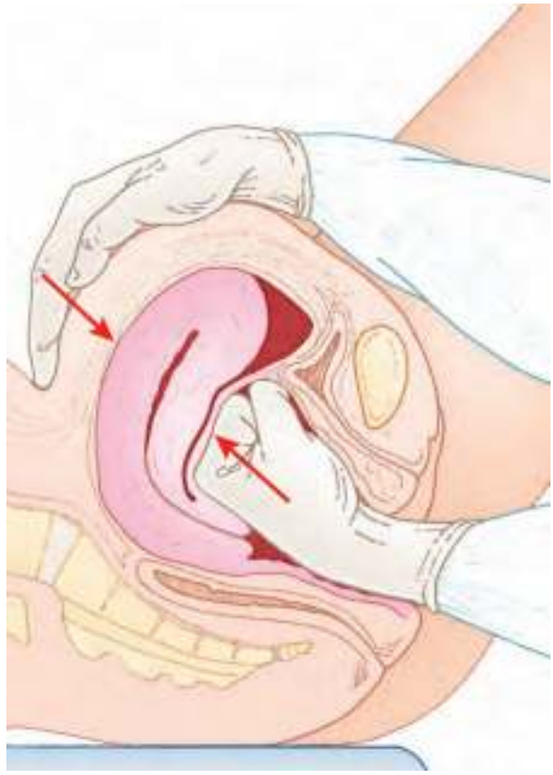

FIGURE 166-8. Bimanual uterine compression.

abdominal hand and the anterior aspect of the uterus through the vagina with the other hand clenched into a fist (**Figure 166-8**).[8] Use the intravaginal hand to massage the uterus against the external pressure applied by the abdominal hand. **This allows for more effective uterine massage.** Perform manual uterine exploration if the massage fails to control the hemorrhage. Manual uterine exploration can localize and extract any placental fragments remaining within the uterus.

Begin second-line medications to contract the uterus if bimanual massage is ineffective and consult an Obstetrician. Several options are available depending on what medication is readily available.[20] Administer 250 µg of Hemabate (i.e., PGF_2) intramuscularly or transabdominally into the uterine musculature every 15 to 90 minutes to a maximum total dose of 2 mg. Only use Hemabate in nonasthmatic patients. It is a potent stimulator of uterine contraction.[21] Administer 0.2 mg of methylergonovine (i.e., Methergine) intramuscularly in the nonhypertensive patient. This is an ergot derivative that causes uterine contraction. The dose may be repeated every 2 to 4 hours. Misoprostol (i.e., Cytotec) is quickly absorbed, inexpensive, and safe to use in patients with hypertension or asthma. Administer 400 µg in tablets buccally for faster peak absorption or rectally. This dose may be repeated every 4 to 6 hours. Monitor the patient's temperature as pyrexia can occur. Administer dinoprostone (e.g., Prostin) 20 mg rectal suppository as continued vaginal bleeding may expel the suppository. This may be repeated every 2 hours. Consider using 16.7 to 200 µg/kg IV of factor VIIa concentrate (i.e., NovoSeven). This agent has the potential to cause intravascular clot formation with subsequent end organ damage.[22]

Sustained bleeding requires an Obstetrician for curettage or other operative management. A discussion of these techniques is beyond the scope of this chapter. Tamponade the uterus while waiting for the Obstetrician as described below. Consult an Interventional Radiologist and consider uterine artery embolization.

The PPH Butterfly was developed to stop postpartum hemorrhage (**Figure 166-3**).[23] The Butterfly achieves the benefits of bimanual compression but is less invasive (i.e., a hand is not inserted into the patient). The size and shape of the compression platform are based on a pessary. It is slim, easy to insert, and acceptable to women. The Butterfly is held in place by the physician with a handle on it. It can also be held in place by an assistant or wedged on the bed. This device is not yet available commercially.

BALLOON TAMPONADE

A balloon within the uterus can tamponade the hemorrhage.[24,25] Balloons are safe and effective. They are incorporated into postpartum hemorrhage pathways.[25] Their use may decrease morbidity and mortality. Balloons are easy to insert and use. The side effects are minimal. This is only a temporizing measure to control hemorrhage.

The Bakri Balloon is a dual-channel and single-use device that was designed to tamponade the uterus for postpartum bleeding (**Figure 166-4**).[26-29] Advantages include conformation to the shape of the endometrial cavity, low risk of uterine perforation, ready availability to use, and ease of use. The Bakri Balloon is made of silicone and contains no latex. Ensure that no placental fragments remain in the uterus (e.g., palpation or ultrasound).

Clean the cervix and vagina with antiseptic solution (e.g., povidone iodine). Gently grasp the balloon with ringed forceps. Insert it into the uterus transcervically, under ultrasound guidance if possible or by palpation, to the fundus. **Ensure that the entire balloon is within the uterus.** Hold the tubing in place at the cervical os to ensure the balloon remains above the cervix. Instill 300 to 500 mL of sterile saline to inflate the balloon. **Use only sterile fluid and not air or another gas. Using a gas can result in an air embolism if the balloon is perforated.** Clamp the tubing. Palpate the cervix or use ultrasound to ensure uterine placement. Apply gentle traction to maximize lower uterine segment tamponade. Secure the tube to the patient's thigh or attach a 500 mL saline bag to the tubing as a weight. Blood will drain from sites proximal to the tube. Consider packing the vagina to prevent the balloon from migrating downward into the vagina (**Figure 166-9**). Tamponade has failed if the bleeding is excessive and continues. Bleeding is occurring above the balloon if the fundal height increases. Flush the drainage tube with 10 mL of sterile saline. The Bakri Balloon can remain in place for up to 24 hours.

FIGURE 166-9. Vaginal packing with a balloon in the uterus.

The ebb Complete Tamponade System (Clinical Innovations, Murray, UT) can also be used up to 24 hours (**Figure 166-5**). It is a dual-balloon that conforms to the endometrial cavity.[30] The 750 mL intrauterine balloon is larger than most others. The uterine balloon has three lumens to enable balloon inflation/deflation, uterine irrigation, and uterine drainage. The dual-balloon anchors the device and eliminates the vaginal packing with the inflated vaginal balloon. The vaginal balloon has one lumen to inflate and deflate the balloon. The ebb is made of polyurethane and contains no latex.

The ebb catheter is used similar to the Bakri Balloon. Collapse the uterine balloon loosely. Insert the device through the cervical os and to the uterine fundus. Attach the fluid bag to the infusion port spike. Inflate the uterine balloon by squeezing the fluid bag. This transfers the fluid from the bag to the intrauterine balloon. Transfer approximately 250 mL to the balloon. Continue to fill the balloon gradually until the desired fluid volume is reached and tamponade is achieved. **Rapid infusion can rupture the uterus.** Unlock the vaginal balloon and slide it to the desired location. Lock the vaginal balloon in place. Fill the vaginal balloon up to 300 mL maximum. Apply traction if desired and secure the catheter as above.

The BT-Cath (Utah Medical products LLC, Midvale, UT) is a silicone catheter that contours to the shape of the endometrial cavity (**Figure 166-6**). It is made of silicone. The soft balloon is easy to insert. The dual-lumen catheter allows infusion of fluid to fill the balloon and monitor uterine drainage. The set includes the catheter, stopcock, syringes, and valves. Assemble the system (**Figure 166-6**). Insert the catheter into the uterus. Spike the IV bag and draw fluid into the syringe. Pump fluid into the balloon.

Use a Sengstaken-Blakemore tube if a tamponade balloon is not available (Chapter 81). These are often available in the Emergency Department. Insert the Sengstaken-Blakemore tube into the uterine cavity. Inflate the gastric balloon with 250 mL of sterile saline. Check placement and apply slight traction. Secure the tube as described above. Flush the lumen of the Sengstaken-Blakemore tube with 10 mL of sterile saline to clear any clots or blood collected behind the balloon.

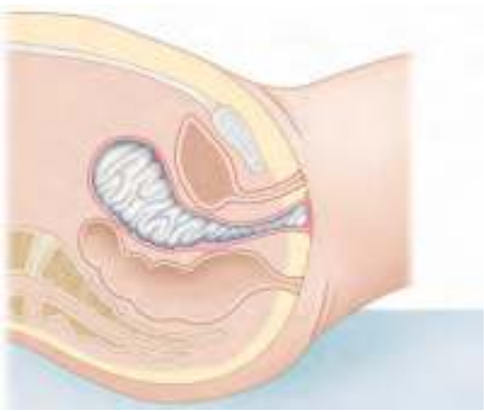

FIGURE 166-10. The uterus and vagina are packed with Kerlix gauze.

Use Foley catheters (Chapter 173) with a 30 mL bulb if a Bakri Balloon or a Sengstaken-Blakemore tube is not available. Gently grasp the end of the Foley catheter with ring forceps just below the bulb. Insert it completely into the uterus. Securely hold the catheter in place. Instruct an assistant to instill 60 mL of sterile saline into the bulb. Repeat this procedure with up to two more Foley catheters to completely fill the endometrial cavity and tamponade the hemorrhage.

The Duette (Poiesis Medical LLC, Jupiter, FL) was developed as an alternative to the Foley (**Figure 166-7**). The dual-balloon catheter was meant for bladder catheterization. Drainage holes are located between the two balloons. It has been placed in the endometrial cavity for tamponade of postpartum hemorrhage.

UTERINE PACKING

Consider packing the uterus if none of the previously mentioned devices are available or if they fail (**Figure 166-10**).[31] Packing is effective, quick, and safe. Tightly pack the uterus starting at the fundus and working outward. Use a 4 inch wide gauze roll (e.g., Kerlix) soaked in 5 mL sterile saline and 5000 U of thrombin. Administer IV broad-spectrum antibiotics if the uterus is packed with gauze. Carefully monitor hematocrit, urine output, and uterine size. Blood may collect in the fundus with all forms of tamponade.

RETAINED PLACENTA

Postpartum hemorrhage, whether primary or secondary, may be caused by retained placental tissue within the uterine cavity. The retained placental tissue may be completely attached, partially separated, or completely separated from the uterine wall. Placental fragments completely attached to the uterine wall may not be removable manually and may require a curettage or laparotomy. Completely or partially separated placental fragments may remain due to a closed cervix entrapping them or inadequate uterine contractions. Manual extraction will remove the retained placental fragments. **Administer adequate anesthesia. Administer a uterine relaxant to relax the lower uterine segment in the absence of uterine atony. This must be performed aseptically.**

Insert a sterile gloved hand through the open cervix and into the uterine cavity (**Figure 166-11**). Place the other hand on the abdominal wall and over the fundus of the uterus. Gently and carefully sweep the fingers around the circumference of the uterus to determine if any fragments of placenta remain. Identify the edge of the fragment if it has not separated from the uterine wall. Gently place the fingertips under the edge of the fragment. Gently remove any placental fragments from the uterus by alternating abducting, adducting, and advancing the fingers in a scissors-like motion until

FIGURE 166-11. Manual removal of the placenta. The fingers are abducted, adducted, and advanced continually (in a scissor-like motion) in this sequence of movements until the placenta is completely detached.

the entire fragment is separated from the uterus wall. Grasp and gently remove the placental fragments when separated from the uterine wall. **Ensure that the entire placenta is removed. Reinsert a hand into the uterine cavity and palpate for any remaining placental fragments. Examine the placenta carefully to make sure that no cotyledons are missing.**

Determine if any placental membranes are retained. Reinsert the gloved hand covered with a gauze sponge (**Figure 166-12**). Wipe the uterine walls with the sponge to collect any retained membranes. Remove the gauze sponge with any adherent membranes. Initiate IV oxytocin following removal of the placenta.

The placenta is likely embedded into the wall of the uterus if it does not manually separate. This is known as placenta accreta, percreta, or increta depending on the degree of myometrial penetration. Consult an Obstetrician as a laparotomy, hysterotomy, and possibly a hysterectomy will be required.

GENITAL TRACT INJURIES

Bleeding from the lower uterine segment, cervix, or upper vagina is difficult to diagnose and manage. The anatomic locations are awkward and difficult to visualize. Excessive bleeding makes visualization even more problematic. It may be almost impossible to see a small laceration or an individual bleeding vessel. **Do not attempt to repair a laceration that cannot be completely visualized as this could result in damage to other structures.** The uterotonic medications (e.g., oxytocin and the prostaglandins) are less effective due to the relative paucity of contractile muscle in these tissues. Consider packing the uterus with gauze or a vaginal pack if available. Management of excessive bleeding from this area may require a laparotomy with uterine artery embolization or a hysterectomy. Discussion of these techniques is beyond the scope of this chapter.

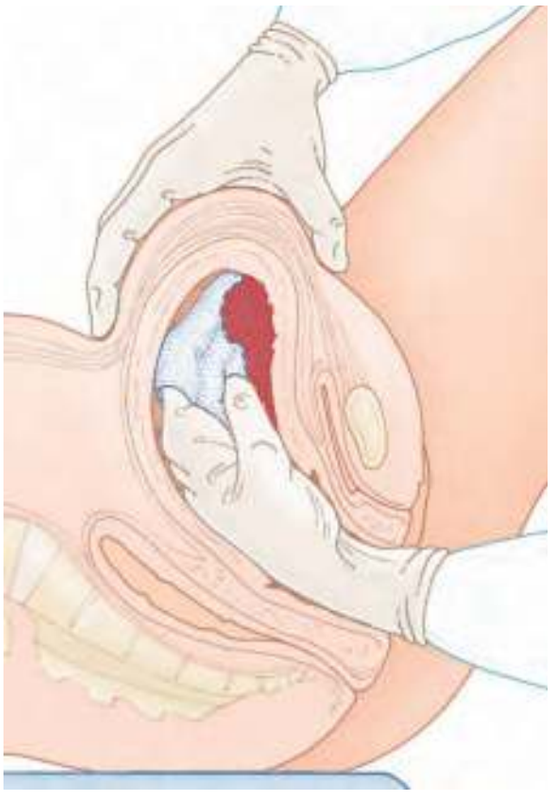

FIGURE 166-12. Manual removal of retained placental membranes.

LACERATION REPAIR

All bleeding lacerations must be repaired. Always make sure there is adequate exposure and visualization of the laceration. Insert a Foley catheter if the laceration is in proximity to the urethra. This ensures urethral patency and helps preclude urethral inclusion when placing sutures. Scrub the perineum with chlorhexidine or povidone iodine solution. Apply sterile drapes beneath the patient's buttocks, on the legs, and on the abdomen to prevent contamination from nonsterile areas. Provide anesthesia with the injection of local anesthetic solution directly into the laceration or with a nerve block (Chapter 163).

Thorough knowledge of the anatomy and awareness of where sutures are being placed are necessary to avoid perforation of any proximate viscera. Always use absorbable suture. Refer to Chapter 163 for complete details regarding the repair of an episiotomy. A brief description of the repair procedure is provided below.

FIRST-DEGREE LACERATIONS

First-degree lacerations involve the fourchette, the perineal skin, and/or the vaginal mucous membrane. They spare the underlying fascia and muscle. If the lacerations are small and hemostatic, they do not need to be repaired. Use continuous running 2–0 or 3–0 monofilament polydioxanone (i.e., Monocryl) or polyglactin (i.e., Vicryl) suture to close the vaginal mucosa and submucosa. Chromic gut suture is an alternative but causes more postprocedural pain until it is absorbed. Interrupted stitches may better approximate the laceration if it is very irregular. Approximate the cut margins of the hymenal ring with a stitch. Repair skin lacerations with subcuticular 3–0 stitches as they cause less perineal pain.[8] An alternative is to place interrupted 3–0 stitches.[8]

SECOND-DEGREE LACERATIONS

Second-degree lacerations involve the perineal skin, vaginal mucous membrane, subcutaneous tissue, fascia, and muscles of the perineum but not the anal sphincter muscle. Repair is essentially the same as for an episiotomy but complicated by the irregularity of the laceration. Begin as in a first-degree laceration by repairing the vaginal mucosa and submucosa. Do not place the stitches too deep above the vaginal fornices to avoid injuring the ureters. Approximate the hymenal ring. Place interrupted 2–0 or 3–0 stitches to close the fascia and muscles of the lacerated perineum. Carry a continuous running stitch downward to unite the superficial fascia and then upward to close the subcutaneous tissue. Close the skin with a running subcuticular stitch, which is preferred, or interrupted stitches.

THIRD-DEGREE LACERATIONS

Third-degree lacerations involve a second-degree laceration that extends into the anal sphincter but not the rectal mucosa. Isolate, approximate, and stitch together the cut ends of the anal sphincter muscle with interrupted 2–0 Vicryl suture at the anterior, inferior, posterior, and superior portions of the muscle. The remainder of the repair is the same as that for second-degree lacerations.[8]

FOURTH-DEGREE LACERATIONS

Fourth-degree lacerations extend through the rectal mucosa to expose the rectal lumen. Approximate the torn rectal mucosa with running or interrupted 3–0 or 4–0 chromic gut sutures placed approximately 0.5 cm apart. Isolate and approximate the internal anal sphincter with 3–0 or 4–0 Vicryl or Monocryl suture. Proceed as with the repair of third-degree lacerations.[8]

CERVICAL LACERATIONS

Gently grasp each side of the cervical laceration with ringed forceps. Start at the apex of the laceration using 2–0 suture and close the laceration in a running pattern. **Do not stitch closed the endocervical canal.** Start at the highest point that can be visualized if the apex cannot be visualized and place the first stitch as a single interrupted stitch. Use the suture to apply gentle traction to identify the upper part of the laceration. Place interrupted 2–0 stitches to close the apex. Use a running stitch to close the lower part of the laceration.

ASSESSMENT

Thoroughly examine the patient to ensure the cessation of bleeding. **Any additional bleeding sites must be found and repaired.** Carefully monitor the patient's vital signs and urinary output. Manage any hypotension with fluid boluses and blood products. Follow serial complete blood counts if the patient has lost a significant amount of blood. A hematocrit less than 21% or persistent hypotension and tachycardia may require a transfusion. Manage hemodynamically stable patients expectantly. Continue to monitor blood parameters every 30 minutes and use these to guide treatment.[10] Consult an Obstetrician for all patients with postpartum hemorrhage for repair in the Emergency Department or the Operating Room and patient admission.[32,33] Consider consulting an Invasive Radiologist, after consulting the Obstetrician, for embolization and to avoid surgery.[34]

Do not deflate the balloon catheter in the Emergency Department. This is best done by the Obstetrician after the tamponade of the hemorrhage. Deflate the balloon if it is no longer needed (i.e., the hemorrhage is stopped). Deflate the balloon slowly in increments in case bleeding resumes. Observe the patient between deflation increments until the balloon is empty. Cut the inflation lumen with a scissors for quick deflation.

AFTERCARE

The use of antibiotics has not been studied in comparison to not using them. Expert consensus is that administration of parenteral antibiotics decreases the rate of infection (i.e., cervicitis, endometritis, and vaginitis). All agree that antibiotics should be used if the vagina or uterus is packed.

The aftercare for postpartum hemorrhage is targeted toward the etiology of the bleeding episode. Warm sitz baths alternated with ice packs applied to the perineum three to four times a day will decrease inflammation and the risk of infection in patients with lacerations or episiotomies. Stool softeners will decrease the pain of defecation and the risk of wound dehiscence, especially with third-degree or fourth-degree lacerations. A high-fiber, low-residue diet may be helpful if the anal sphincter was involved. Prescribe oral analgesics for pain relief. Nonsteroidal anti-inflammatory drugs or acetaminophen provides adequate analgesia for most patients. Narcotic analgesics (e.g., acetaminophen with codeine, hydrocodone, or tramadol) may be necessary initially. Avoid formulations that increase constipation in patients with third-degree or fourth-degree lacerations.

Follow complete blood count values until the patient is asymptomatic and stable. Consider a transfusion if the patient's hematocrit is less than 25 or if the patient is symptomatic from anemia. Prescribe 325 mg of iron sulfate orally two to three times daily.

Follow-up therapy for uterine atony often includes the use of oxytocin (e.g., 20 to 40 U/L of IV fluid) for 12 to 24 hours. An alternative is oral Methergine or other uterotonic medications. There are no studies comparing the individual medications. Initiate therapy after consultation with an Obstetrician.

COMPLICATIONS

Maternal complications resulting from postpartum hemorrhage include acute renal failure, acute respiratory distress syndrome, coagulopathy (i.e., consumptive or dilutional), extreme fatigue, postpartum endometritis, Sheehan's syndrome, shock, the need for blood transfusion, and the need for surgical intervention (e.g., dilatation and curettage or laparotomy) and its associated complications. Complications associated with pharmacologic therapy can be avoided by carefully selecting the appropriate agent for each patient as noted previously.

Intrauterine balloons can migrate or slip into the vagina (**Figure 166-13**). This risks rupture of the cervix or vagina. Prevent migration by using a dual-balloon system, frequently checking balloon location, not applying traction, or packing the vagina.

FIGURE 166-13. Improper uterine balloon placement or migration.

SUMMARY

Postpartum hemorrhage is a serious and potentially lethal condition. Early identification of risk factors and a prompt response to the early signs and symptoms of postpartum bleeding will decrease the morbidity and mortality. Always consult an Obstetrician immediately if the patient experiences postpartum hemorrhage.

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167 Perimortem Cesarean Section (Perimortem Cesarean Delivery)

Jeanne A. Noble

INTRODUCTION

The term "perimortem cesarean delivery" (PMCD) was introduced in 1986 to describe a cesarean section performed during cardiopulmonary resuscitation (CPR) of the mother and initiated within the first 4 minutes following maternal arrest.[1] **The goals of this procedure are to improve the effectiveness of maternal resuscitation as well as to increase the chances of fetal survival.**[2,3] For more than two decades, the PMCD has been included as part of the algorithm for management of cardiac arrest in pregnant patients.[4-7]

The incidence of cardiac arrest during pregnancy is estimated to be about 1 in 30,000 pregnancies.[8,9] According to a review of reported PMCDs from 1985 to 2004, the most frequent causes are anesthesia, cardiogenic, eclampsia, emboli (e.g., amniotic fluid and air), intracranial hemorrhage, magnesium overdose, sepsis, spontaneous uterine rupture, and trauma.[2]

Trauma is the leading cause of death in women of reproductive age and accounts for 25% to 50% of maternal morbidity. Major maternal injury is associated with a fetal loss rate of 45% to 50%. **The primary goal in the management of the severely injured pregnant patient is maternal assessment and stabilization. Prompt attention to the needs of the gravid patient can save the life of both the fetus and the mother.** There are occasions when emergent cesarean delivery is the intervention most likely to save the life of the mother, the fetus, or both.

This procedure is best performed by a qualified Surgeon in the Operating Room. However, in the case of maternal arrest out of hospital or in the Emergency Department, valuable minutes should not be wasted transferring the patient to an Operating Room. Rather, the Emergency Physician should proceed with PMCD with the goal of fetal delivery by minute 5 after maternal arrest. It may also be performed for imminent maternal demise. PMCD is a key procedure that all Emergency Physicians need to be able to perform in the rare instance that it is required.

There are two basic conditions that must be met when considering PMCD. First, confirm that the uterine fundus reaches the mother's umbilicus (or higher) correlating with a gestational age of 20 weeks (or more). This is the point at which aortocaval compression by the uterus has a significant impact on maternal hemodynamics. Second, verify that the mother is pulseless and that noninvasive manual displacement of the uterus to her left side does not lead to return of spontaneous circulation (ROSC). **Proceed quickly to PMCD unless there is another obvious intervention that is likely to lead to maternal ROSC.**

ANATOMY AND PATHOPHYSIOLOGY

Important physiologic changes during pregnancy should be kept in mind in the event of maternal cardiac arrest.[10] Cardiac output, blood volume, and heart rate all increase during pregnancy. Pulmonary and systemic vascular resistance decreases. Uteroplacental blood flow increases with the uterus receiving up to 30% of cardiac output. Aortocaval compression during the second half of pregnancy can result in decreased venous return to the heart, decreased cardiac output, and systemic hypotension. The pregnant woman is also predisposed to a more rapid decrease in arterial and venous oxygen tension during episodes of hypoxia. **These physiologic changes occurring to support the fetus complicate resuscitation efforts of the mother.**[3,8,11,12]

In general, the same algorithms used for the nonpregnant patient should be used for the pregnant patient during cardiac arrest with some modifications.[11] The stroke volume of a term pregnant patient lying supine is only 30% of normal. Lateral displacement of the uterus will increase the stroke volume and cardiac output by at least 25%.[13] Pregnancy increases the risk of aspiration. Early intubation to protect airway is indicated. Be cautious of using sodium bicarbonate to reverse metabolic acidosis. Rapid correction of maternal, but not fetal, acidosis could lead to reduced compensatory hyperventilation and normalization of fetal $PaCO_2$ with worsening of the fetal acidosis.[8] The performance of adequate CPR in the gravid patient at or near term is extremely difficult. CPR produces a cardiac output equivalent to only 30% of normal under ideal circumstances. The enlarged uterus lies anterior to the inferior vena cava and suppresses venous return in the gravid patient. Uterine compression of the aorta increases afterload.

Consider early intubation. Oxygen consumption is higher in the pregnant patient due to an increased maternal metabolic rate plus the fetal demands. Functional residual capacity is lower. These factors lead to rapid hypoxemia. The patient's risk of aspiration is also higher due to decreased tone of the lower esophageal sphincter and delayed gastric emptying.[8]

The Emergency Physician must give early consideration to PMCD in the critically ill pregnant patient. Evacuation of the uterus will enhance maternal resuscitative efforts and may lead to higher rates of fetal survival as well.

INDICATIONS

The major indication for performing PMCD is to optimize maternal CPR. The rescue of a viable fetus greater than 20 weeks of gestation is an important consideration, but such rescue is always secondary

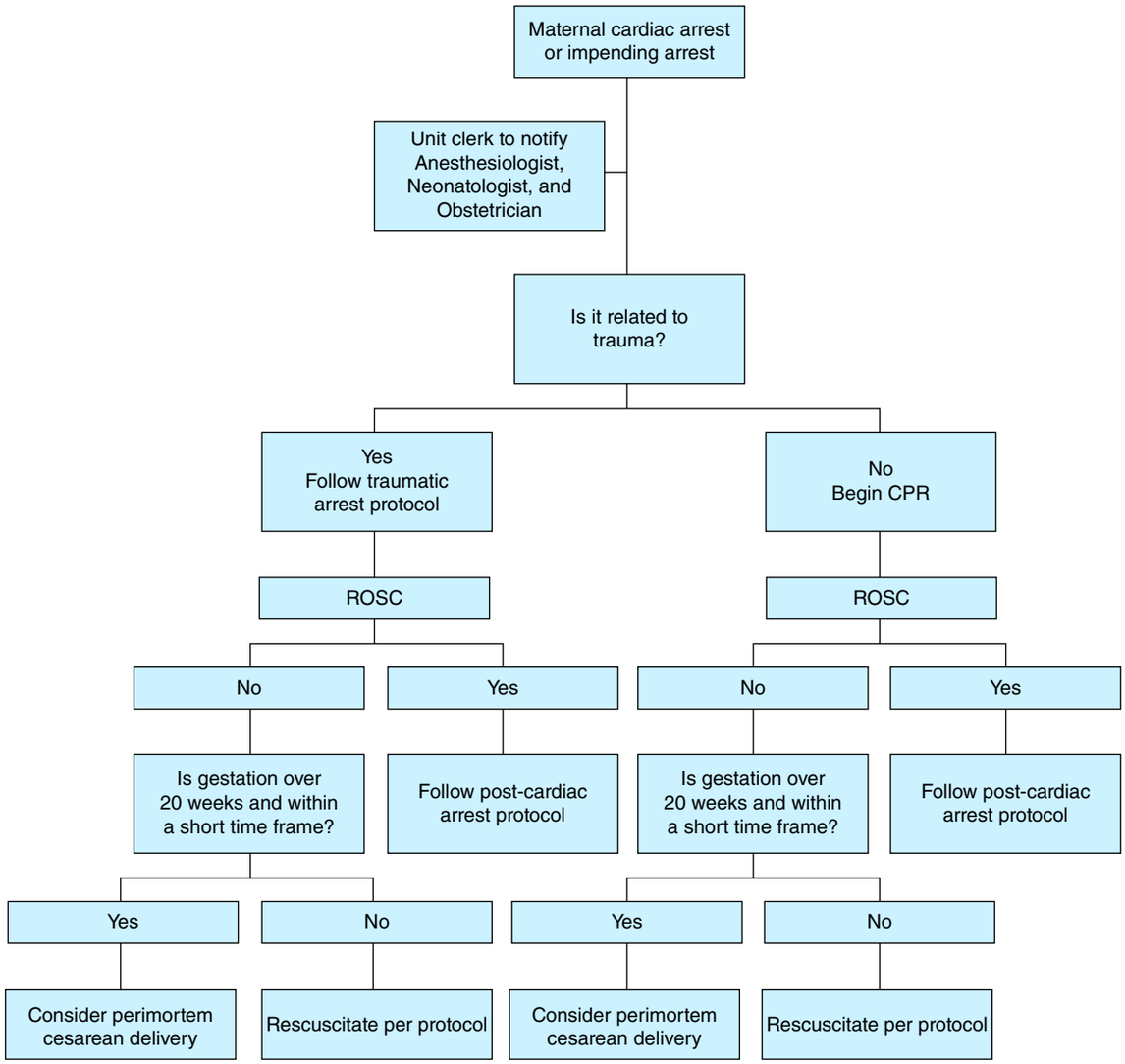

FIGURE 167-1. Algorithm for PMCD. (Modified from www.foamshed.wordpress.com/.)

to the safety and life of the mother. Consider performing PMCD if CPR does not lead to maternal ROSC within 4 minutes of her arrest.[4,14-21] Waiting for the support of Obstetrical and Pediatric teams is contraindicated.[2,13] Consider performing a PMCD, or having an Obstetrician perform it, for imminent maternal demise or as soon as CPR is begun.[22] **Figure 167-1** provides an algorithm for performing a PMCD.

CONTRAINDICATIONS

There are few contraindications to performing a PMCD. The best fetal outcomes are reported when cesarean delivery is completed within 5 minutes of maternal arrest. However, a PMCD should be considered when the time from maternal arrest to delivery is no greater than 25 minutes.[23] It is contraindicated if the mother has a serious brain injury but is otherwise hemodynamically stable and the fetus shows no signs of distress. PMCD is contraindicated when the gestational age of the fetus is estimated to be less than 20 weeks.

Attempt to obtain consent for the procedure only if it does not introduce additional delay. There is no documentation of physician liability in these situations. The unanimous consensus in the medical literature and of legal authorities is that a civil suit for performing a PMCD, regardless of the outcome, would not result in a judgment against the Emergency Physician.

EQUIPMENT

- 10 towel clips
- 16 Kelly clamps
- 16 hemostats
- 10 Peon or Pennington clamps
- 8 Allis forceps
- 6 Babcock forceps
- 6 ring forceps
- 6 straight Kocher clamps
- #10 surgical scalpel blade and handle
- #15 surgical scalpel blade and handle
- Straight Mayo scissors
- Curved Mayo scissors
- Metzenbaum scissors
- Bandage scissors

- Bladder retractor
- Pack of sponges
- Ring forceps
- Suture scissors
- 2 Adson forceps, or other forceps with teeth
- 2 Russian forceps, 5 1/2 inches and 8 inches
- Dressing forceps
- Skin stapler
- 2 medium Richardson retractors
- 2 small Richardson retractors
- 2 malleable retractors
- 2 Army-Navy retractors
- Needle drivers, 8 inches and 6 inches long
- 2 suction tips
- Suction tubing set(s)
- Wall suction
- Electrocautery unit with disposable tips
- Povidone iodine or chlorhexidine solution
- Sterile drapes
- Sterile gloves
- 2 skin staplers
- 3 packs 0 chromic suture with CTX taper needle
- 1 pack 2–0 chromic suture with taper SH needle
- 2 packs of 1–0 Vicryl suture with a CT taper needle
- Chromic, Polysorb, or Vicryl suture (2–0 or 1–0)
- Nylon suture, 3–0
- Bulb syringe
- Clean towels or blanket for baby
- Umbilical cord clamp
- Sterile scissors to trim umbilical cord
- Infant warmer
- Neonatal resuscitation kit
- Neonatal resuscitation equipment (including an infant warmer)

A lot of equipment is required to perform a PMCD. Ideally, all of the equipment required should be prepackaged in a sterile cesarean section instrument tray prepared by the hospital. A prepackaged sterile instrument tray is commercially produced but may not be available as this procedure is rarely performed in the Emergency Department. A standard thoracotomy tray or tube thoracostomy tray will contain all the required equipment except for the bulb syringe and umbilical cord clamp.

PATIENT PREPARATION

Quickly determine the gestational age using fundal height. A fundal height at the umbilicus correlates with a gestational age of approximately 20 weeks, at which time aortocaval compression by the uterus is likely to significantly hinder resuscitative efforts. A quick rule to remember is that if you can place at least four fingers above the umbilicus to the top of the fundus, the gestational age is likely to be equal to or more than 24 weeks.[2,24] This estimate can be distorted by overweight patients, abdominal distention, and a history of multiple pregnancies. Unfortunately, no other quick rule has been yet described. If the mother has not yet arrested, bedside ultrasound may be used to provide a more accurate estimation of gestational age.[25] **However, performance of the PMCD should never be delayed in order to obtain more accurate measurements or to assess for fetal heart tones.**

Note the time of maternal arrest and appoint a timekeeper. Maintain left uterine displacement (LUD) throughout the resuscitation. Plan to proceed with the PMCD if the resuscitation of a pregnant patient with an estimated gestational age of 20 weeks or greater has not regained spontaneous circulation within 4 minutes of her arrest.[4,14-21]

The single most important prognostic factor for neonatal outcome is the time from maternal arrest to delivery. Delivery of the fetus can also maximize maternal resuscitation efforts and minimize the risk of maternal brain injury. Other resuscitative efforts, such as volume expansion and vasopressors, may be initiated simultaneously but should not delay the PMCD.

Time is of the essence. Establishing intravenous access and endotracheal intubation takes several minutes. Thus, it is very important to activate a protocol for a PMCD as soon as cardiac arrest occurs.[12,13] Every attempt should be made to contact an Obstetrician and Neonatologist if PMCD is anticipated. Alert the covering General Surgeon if an Obstetrician is not available. Notify the neonatal resuscitation team of the impending procedure. Anesthesia will not be required if the patient is deceased. **The procedure should not be delayed in an attempt to obtain consent or the consultants.**[1,12,26] The medicolegal risks are few since the Emergency Physician is acting under the principle of beneficence.

The patient will be clinically deceased in most cases if this procedure is performed in the Emergency Department. Patient preparation is not necessary and wastes valuable time when trying to salvage the fetus. Administer broad-spectrum intravenous antibiotics if the mother survives the procedure. Continue CPR throughout the PMCD.

LUD should be performed to reduce aortocaval compression. Stand on the patient's left side. Cup the uterus between two hands and lift it upward and leftward off the maternal vessels.[22,27,28] **If the rescuer must be on the patient's right side, two hands are used to push the uterus upward and to the left.** LUD is preferred over maternal 15° lateral tilt maneuvers as the latter may lead to less effective chest compressions.[29] Prop the patient up if no assistant is available to maintain the patient in 15° of lateral displacement. This can be accomplished by putting pillows or blankets beneath the patient's right hip or with a Cardiff wedge. Lateral displacement of the uterus will increase maternal stroke volume and cardiac output by at least 25%.[13]

Surgical preparation in an emergency is minimal.[1,26,30] Prepare the abdomen with povidone iodine or chlorhexidine solution. If time permits prior to maternal arrest, it is appropriate to employ the same sterile barrier methods as used during central line placement (i.e., a hat, mask, booties, sterile gloves, and a sterile gown). Have a nurse place a Foley catheter to drain the bladder and reduce the risk of bladder injury during the prearrest phase. Once the patient has become pulseless, bladder decompression is of minor importance and needle aspiration can always be performed by an assistant if a distended bladder interferes with the procedure.

TECHNIQUE

A long vertical incision is the most appropriate approach for this emergency procedure.[31] It provides better exposure, easy access, and a larger opening for the delivery. Identify the patient's umbilicus and pubic symphysis. Make a long vertical midline skin incision with a #10 scalpel blade beginning at the top of the uterine fundus and

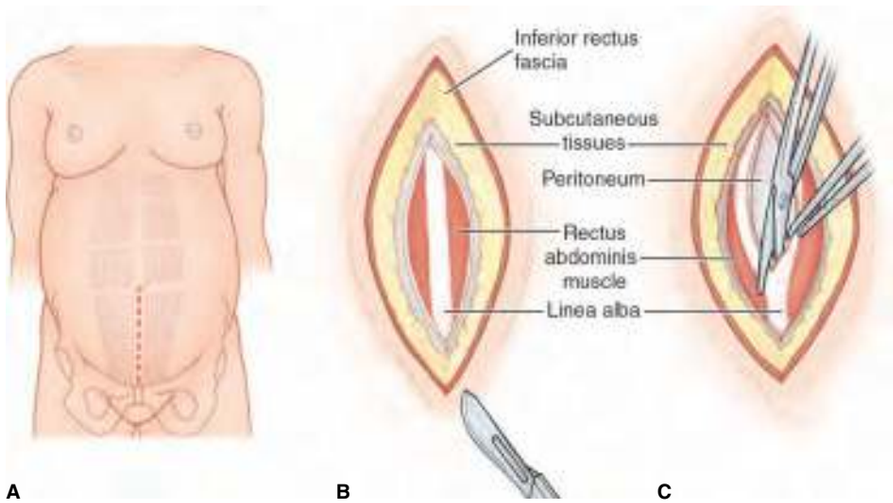

FIGURE 167-2. Accessing the peritoneal cavity. ***A.*** Make a midline skin incision from just below the umbilicus to just above the pubic symphysis. ***B.*** Extend the incision through the subcutaneous tissues and down to the linea alba. ***C.*** Grasp and elevate the rectus abdominis muscle while opening the linea alba with a Mayo scissors.

extending down to just above the pubic symphysis (**Figure 167-2*A***). This incision is long enough to rapidly deliver a full-term infant.[13,26,32] An alternative is to make the incision from just below the xiphoid process to the pubic symphysis. Ignore any subcutaneous bleeding unless it is arterial. Have an assistant clamp any bleeding arteries.

Extend the incision through the subcutaneous fat to the rectus sheath (**Figure 167-2*B***). **Do not be overzealous and cut through the rectus sheath, peritoneum, uterus, abdominal organs, or bladder.** Grasp and elevate the rectus sheath using toothed forceps (**Figure 167-2*C***). Make an incision in the rectus sheath with a Mayo scissors. Extend the rectus sheath incision superiorly and inferiorly with the Mayo scissors (**Figure 167-2*C***). **Be cautious not to cut any abdominal or pelvic organs.**

Expose the uterus (**Figure 167-3**). The underlying peritoneal membrane (i.e., peritoneum) should be visible (**Figure 167-3*A***). It may occasionally be attached to the rectus sheath and opened simultaneously. Instruct assistants to retract both sides of the rectus sheath laterally, or insert retractors if available, to fully expose the peritoneal membrane (**Figure 167-3*A***). Grasp and elevate the peritoneal membrane with toothed forceps. Incise the peritoneal membrane with a Mayo or Metzenbaum scissors in a manner similar to that used to open the rectus sheath (**Figure 167-3*A***).[32]

Make reasonable attempts to protect the bowel and bladder from injury. If possible, elevate the bowel off the field and cover it with a saline-soaked towel. Place a bladder retractor over the pubic symphysis to retract the rectus sheath and bladder (**Figure 167-4*A***).[32] Alternatively, grasp and elevate the bladder from the pelvis with a saline-soaked gauze or towel (**Figure 167-3*B***). This will allow visualization of the uterus and prevent injury to the bladder.

Identify the position of the fetal head by palpating the uterus. **Carefully make a 2 to 4 cm midline vertical incision in the uterus with a #10 scalpel (Figure 167-4*A*).** The amniotic sac will bulge through the incision if the membranes are intact. **Place the index and/or middle fingers into the uterine cavity at the superior aspect of the incision to lift the uterine wall off of the fetus (Figure 167-4*B*). Insert one blade of a bandage scissors and extend the uterine incision upward toward the fundus, cutting above the inserted finger(s) while lifting up on the uterine wall (Figure 167-4*B*).** Repeat this procedure inferiorly to open the uterine wall in the direction of the bladder (**Figure 167-4*C***).

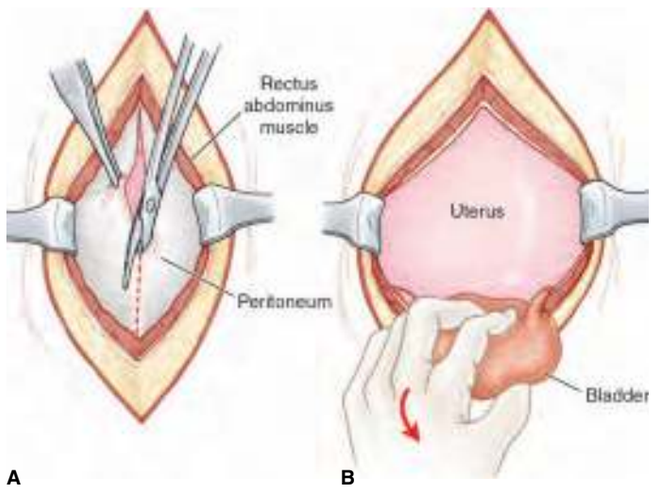

FIGURE 167-3. Exposure of the uterus. ***A.*** Apply retractors to hold the abdominal wall open. Grasp, elevate, and incise the peritoneal membrane in the midline. ***B.*** Grasp and elevate the bladder out of the pelvis.

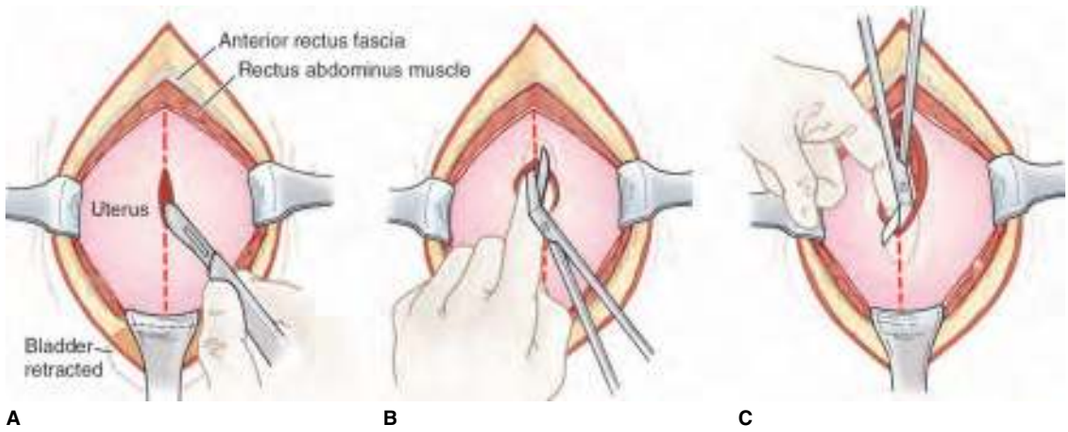

FIGURE 167-4. The vertical uterine incision. ***A.*** Make a 2 to 4 cm midline vertical incision with a scalpel blade. ***B.*** Place a finger into the incision to protect the fetus. Extend the uterine wall incision superiorly. ***C.*** Extension of the uterine wall incision inferiorly.

Rupture the amniotic membranes with a clamp or other blunt instrument. The placenta will often be embedded in the anterior wall of the uterus. It is imperative to expedite the delivery of the fetus by cutting through the placenta. Carefully transect the placenta. There is an urgency to deliver the fetus and clamp the umbilical cord to prevent significant fetal hemorrhage. Insert a hand between the pubic symphysis and the fetal occiput if the fetus is in a cephalic presentation (**Figure 167-5*A***). Advance the hand to the base of the occiput. Gently flex the fetal head with the fingers and palm (**Figure 167-5A**). Apply gentle anteriorly and superiorly directed traction to elevate and deliver the head (**Figure 167-5*B***). Deliver the entire fetal head (**Figure 167-5*C***). Consider suctioning the mouth and nose with a bulb syringe (**Figure 167-5*D***).[32] Deliver the shoulders in a manner similar to that of a vaginal delivery (**Figure 167-5*E***). Apply gentle upward traction on the head while an assistant applies pressure on the uterine fundus. First deliver the anterior shoulder. Deliver the other shoulder followed by the torso and lower extremities. Once the shoulders are delivered, the remainder of the fetus readily follows.

If the fetus is in a breech presentation or transverse lie, feet first delivery is easiest. During the delivery, grasp the fetus by the pelvic bones and not by the abdomen to protect the delicate viscera.[26,32] Deliver the fetus.

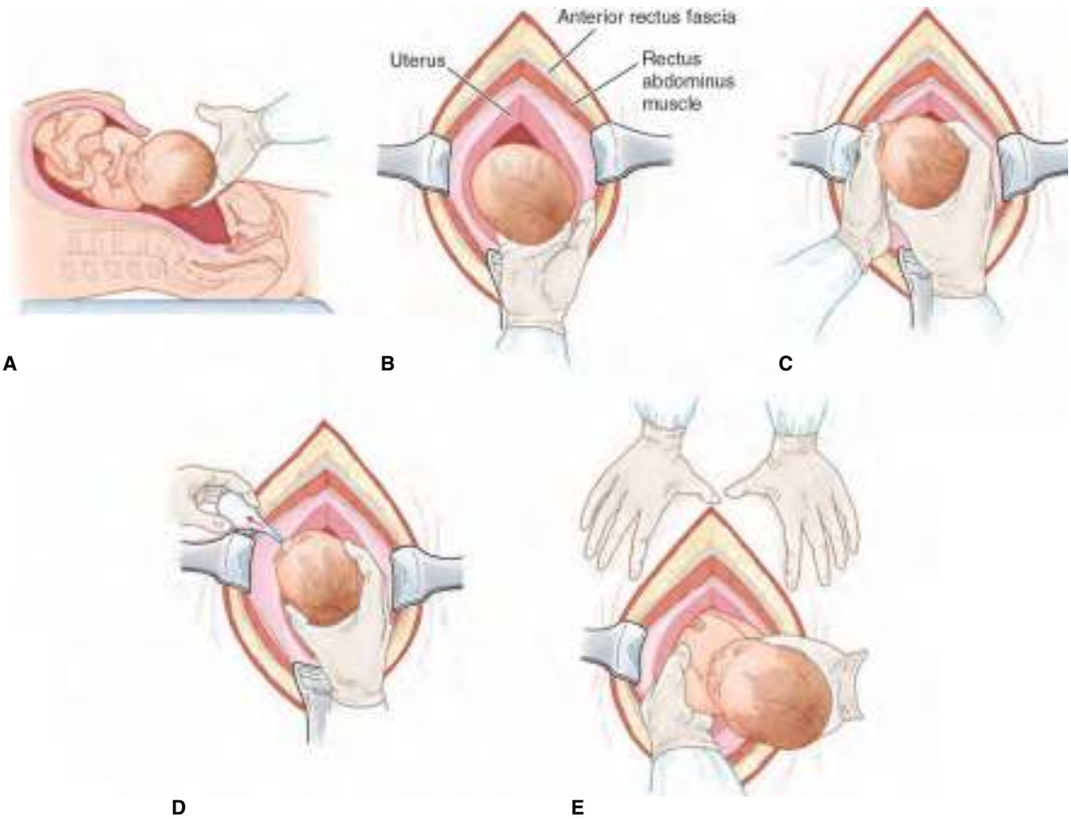

FIGURE 167-5. Delivery of the infant. ***A.*** Midsagittal section through the abdomen and pelvis. A hand is inserted between the pubic symphysis and the fetal head. ***B.*** Flex the fetal head while applying anterior and superior traction. ***C.*** Delivery of the entire fetal head. ***D.*** Suction the mouth and nose. ***E.*** Deliver the fetus while an assistant applies pressure to the uterine fundus.

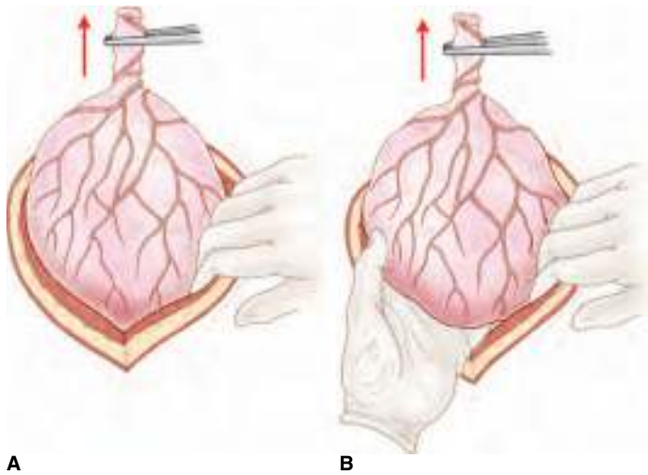

FIGURE 167-6. Delivery of the placenta. ***A.*** Apply gentle upward traction on the umbilical cord while holding the uterine wall open. ***B.*** Insert a hand between the placenta and the uterine wall to separate the placenta from the uterus.

Clamp the umbilical cord with a hemostat or umbilical cord clamp approximately 10 cm from the fetus. Attach a second hemostat or clamp 2 to 3 cm distal to the first. Cut the umbilical cord between the clamps with a scissors. Hand the neonate to waiting personnel for resuscitation.[7,26,32] Resuscitate the neonate yourself if additional help is not available.

AFTERCARE

Palpate inside the uterus for another fetus to rule out an unknown twin pregnancy. Deliver the placenta (**Figure 167-6**). Apply gentle upward traction on the umbilical cord while holding the uterine wall open (**Figure 167-6*A***). Insert the other hand between the placenta and uterine wall (**Figure 167-6*B***). Apply gentle pressure to separate the placenta from the uterus and sweep the uterus clean. Begin an oxytocin infusion of 20 U in 1 L of normal saline at a rate of 10 mL/min to help the uterus contract if the mother shows signs of life.[32]

Close the patient if they are still alive or if they regain vital signs. Apply ring forceps to the uterine incision. Close the uterus in two layers with 0 or 1–0 absorbable chromic gut, Polysorb, or Vicryl suture. Close the first layer in a running locked fashion (**Figure 167-7*A***). This will provide hemostasis. A second running layer is necessary to ensure hemostasis and reapproximate the uterine wall. Closure of the serosa of the uterus (**Figure 167-7*B***) and the peritoneal membrane (**Figure 167-7*C***) is recommended but not required. Close the serosa and peritoneum with running 2–0 absorbable chromic gut, Polysorb, or Vicryl suture. Identify and grasp the cut ends of the rectus sheath with hemostats. Close the rectus sheath in a running pattern with 2–0 absorbable suture (**Figure 167-7*D***). Close the skin with running 3–0 nylon (**Figure 167-7*E***) or staples for speed.[13,26,32]

The only closure required if there is no hope of maternal ROSC is a minimal closure of the skin. Use either a running baseball stitch or skin staples.

COMPLICATIONS

The major complications associated with PMCD include maternal sepsis, maternal visceral injury, maternal hemorrhage, and fetal injury secondary to delivery.[33] The possible benefits of maternal and/or fetal survival far outweigh these considerations.[1]

SUMMARY

The perimortem cesarean section or delivery is an emergency procedure that should be considered in any maternal arrest with an estimated fetal gestational age of 20 weeks or greater. This is a high-stress

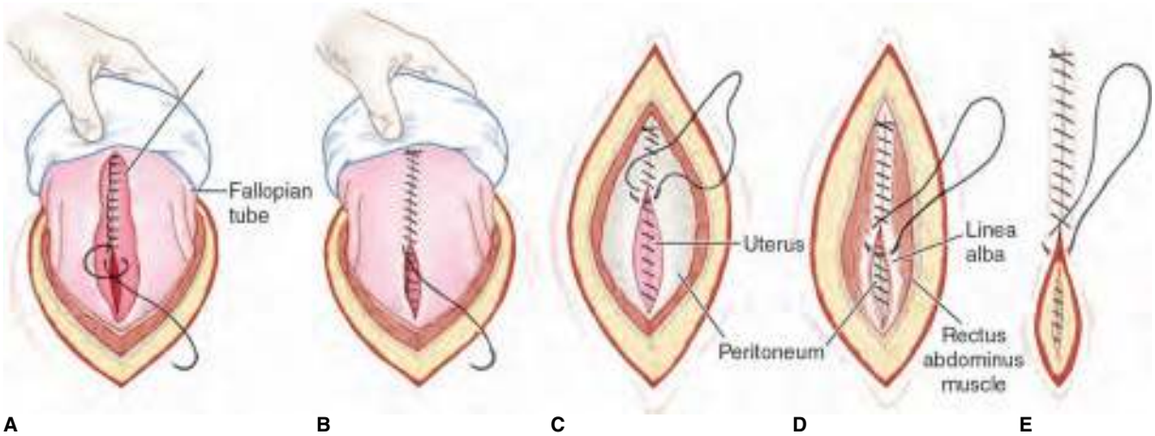

FIGURE 167-7. Closure of the incisions. ***A.*** Running locked closure of the uterine wall. ***B.*** Closure of the serosa of the uterus. ***C.*** Running closure of the peritoneal membrane. ***D.*** Running closure of the rectus sheath. ***E.*** Running closure of the skin.

procedure requiring fast decision making. Emergent delivery of the fetus may provide the mother with her greatest chance of survival. It also provides the fetus with a chance for survival in the face of maternal death. The medicolegal risks are few. Whenever possible, it should be completed by minute 5 after maternal arrest, timing that confers the greatest potential for maternal and fetal survival.

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168 Symphysiotomy

Ikem Ajaelo

INTRODUCTION

Symphysiotomy is the artificial division and separation of the pubic symphysis to facilitate vaginal delivery. This is not to be confused with a pubiotomy or the severance of the pubic bone a few centimeters lateral to the symphysis for the same purpose. A symphysiotomy was first performed in the seventeenth century. It is indicated in cases of cephalopelvic disproportion and may be a lifesaving alternative to cesarean delivery.[1-5] The reported success rate of this procedure is approximately 80% when performed appropriately.[6]

The procedure requires minimal equipment and can be accomplished under local anesthesia in as little as 2 to 3 minutes.[6,7] The increased availability of cesarean sections combined with the known urologic and orthopedic complications have led to its decline and lack of acceptance in modern obstetrical practice.[8,9] However, symphysiotomies are performed regularly and successfully in developing countries where resources and trained medical professionals are often limited or unavailable.[2,3,10]

Indications for a symphysiotomy include breech delivery, cephalopelvic disproportion, and relief of shoulder dystocia.[1,11,12] Each of these conditions poses significant potential risks to mother and child, even under optimal conditions. Emergency Physicians should be familiar with the indications and technique of symphysiotomy as expeditious delivery is vital in these scenarios.

ANATOMY AND PATHOPHYSIOLOGY

The pelvis is composed of four bones: the sacrum, the coccyx, and the innominate bones. Each innominate bone is made up of the fused ischium, ilium, and pubis. The innominate bones are connected at the sacrum by the sacroiliac ligaments and at the pubic symphysis by the superior and arcuate pubic ligaments.[12] Together they determine the size and shape of the pelvis. The fetus assumes positions during labor that are primarily determined by the conformation of the mother's pelvis.[13]

The pelvis is divided into a true pelvis and a false pelvis. They are separated by the linea terminalis, an anatomic boundary formed by the pelvic brim (i.e., superior pelvic aperture). The true pelvis lies below the linea terminalis and is the more relevant portion in delivery.[14] Dense ligaments hold the walls of the true pelvis together. The posterior wall is the anterior surface of the sacrum and coccyx. The lateral boundaries are formed by the inner surface of the ischial bones as well as the sacrosciatic notches and ligaments. The ischial spines can be readily palpated during the vaginal or rectal

examination. They serve as important landmarks in determining to which level the presenting part has descended into the true pelvis. The true pelvis is bounded anteriorly by the pubic bones, the ascending superior rami of the ischial bones, and the obturator foramina.

The ligaments of the pubic symphysis and the sacroiliac ligaments allow mobility and contribute to the increase in pelvic diameter during pregnancy.[13] The sacral nerves, the coccygeal nerves, and the pelvic portion of the autonomic nervous system innervate the pelvis. The important pudendal nerve arises from the sacral plexus and accompanies the internal pudendal artery. It enters the perineal region via the lesser sciatic foramen and around the sacrospinous ligament to supply the muscles of the perineum.[12] Anesthesia to the perineal region can be accomplished by performing a pudendal nerve block.

INDICATIONS

Symphysiotomy is indicated in any difficult delivery in the presence of fetal distress or obstructed labor or when an Obstetrician is not immediately available.[1-3] It has traditionally been reserved for complicated breech deliveries. It has also been reserved for cases of cephalopelvic disproportion in which the fetal head is presenting vertex, at least one-third of the fetal head has entered the pelvic brim, and cervical dilatation is no more than 7 cm.[1] Symphysiotomy has been advocated more recently for the relief of intractable shoulder dystocia.[11,15]

CONTRAINDICATIONS

Contraindications to performing a symphysiotomy include fetal demise and incomplete dilatation of the cervix.[8] Some authors have recommended limiting symphysiotomy to mothers weighing between 50 and 80 kg and a fetus with an estimated mass of 2700 to 3700 gm.[15,16] Other relative contraindications include the presence of maternal spinal deformities, hip deformities, pelvic deformities, and gross obesity.[15] Symphysiotomy has been performed after a prior cesarean section without added complications despite concerns regarding the safety of symphysiotomy in the presence of a previous cesarean scar.[15]

EQUIPMENT

- Local anesthetic solution
- Povidone iodine or chlorhexidine solution
- Sterile gloves and gown
- Face mask and cap
- Urethral catheter set
- Sterile drapes
- #10 scalpel blade on a handle
- Mayo scissors
- Finger guard
- Hemostats
- Sterile gloves
- Neonatal resuscitation equipment
- Infant warmer

PATIENT PREPARATION

Explain the risks, benefits, and potential complications to the patient and/or their representative. Obtain an informed consent to perform this procedure if time allows. Place the patient in the lithotomy position with their thighs separated no more than 90°. This position is necessary to prevent undue strain on the sacroiliac joints. Two assistants should be available to maintain proper maternal leg positioning. Insert a Foley catheter to drain the bladder (Chapter 173).

Prepare the surgical area. Shaving the mons pubis is optional. Apply povidone iodine or chlorhexidine solution onto the skin overlying the pubic symphysis and surrounding area. Allow this to dry. **The Emergency Physician must perform this procedure using aseptic technique.** This includes wearing sterile gloves, a sterile gown, a cap, and a face mask. Apply sterile drapes to form a sterile field. Infiltrate the anterior and inferior aspects of the pubic symphysis and the surrounding skin with 5 to 10 mL of local anesthetic solution to anesthetize the pubic symphysis. Leaving the needle in place may be useful in identifying the pubic symphysis joint. Equipment for infant resuscitation should be readily available, as with all deliveries. Notify the neonatal resuscitation team and a Neonatologist of the impending procedure and delivery.

TECHNIQUE

Apply the finger guard to the nondominant index finger. Place the nondominant index finger into the vagina and against the posterior aspect of the pubic symphysis (**Figure 168-1**). Advance the finger approximately 2 to 3 cm beyond the superior aspect of the pubic symphysis to displace the bladder and urethra. **Do not move this finger until the procedure is complete.**

Grasp the scalpel in the dominant hand with the cutting edge of the blade facing outward (**Figure 168-2**). **The scalpel blade must be kept in a strict midsagittal plane and perpendicular to the skin overlying the pubic symphysis.** Resting the hypothenar eminence of the dominant hand against the patient's pubic ramus will help maintain fine control of hand and finger movements.[6]

Make a midline stab incision 1 cm below the upper edge of the pubic symphysis (**Figure 168-3**). **Very little resistance should be met as the scalpel blade pierces the hyaline cartilage of the pubic symphysis if the scalpel is in the midline.** Carefully and slowly advance the scalpel blade until the tip is just felt through the anterior vaginal wall. Resistance is usually a result of lateral deviation of the scalpel blade against the pubic bones. Withdraw the scalpel blade 2 to 3 mm and readvance it in the midline. **Use extreme caution so that the scalpel does not lacerate the vagina, the urethra, or the finger.**

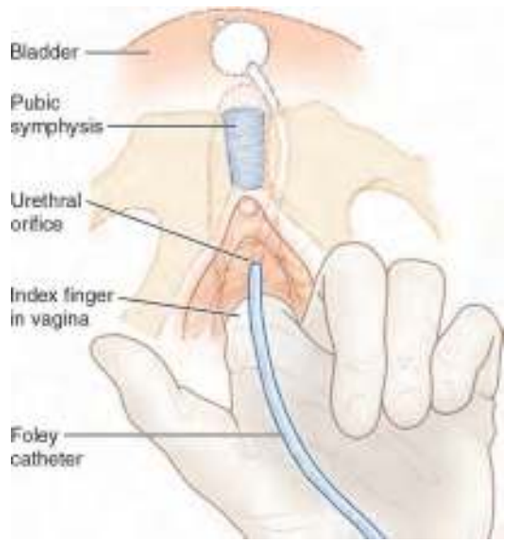

FIGURE 168-1. Frontal view demonstrating the nondominant index finger inserted into the vagina and pushing anteriorly to palpate the pubic symphysis.

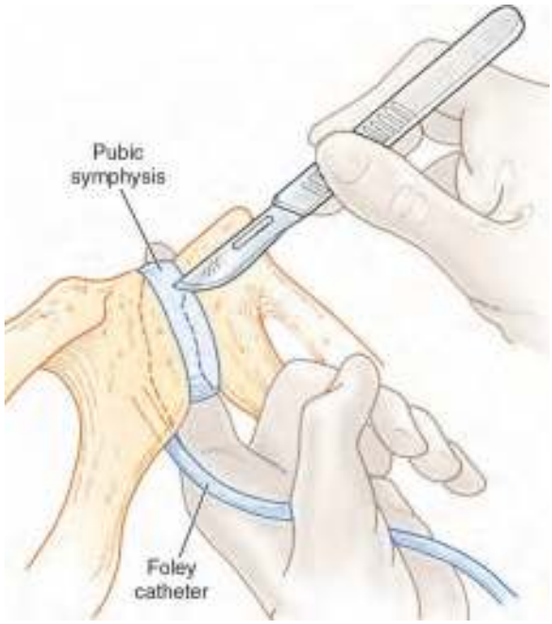

FIGURE 168-2. Skeletal outline demonstrating the position of the scalpel blade in relation to the pubic symphysis.

Lower the handle of the scalpel blade cephalad, toward the abdomen, using the upper portion of the pubic symphysis as a fulcrum. Cut through the lower half of the pubic symphysis dividing the cartilage and associated ligaments. Remove the scalpel blade and reinsert it with the cutting edge in the opposite direction and facing cephalad. Repeat the same maneuver but with the scalpel handle lowered caudally to divide the upper portion of the pubic symphysis and completing the separation.

Use the nondominant index finger to confirm complete separation of the pubic symphysis. **The symphysiotomy is complete when the index finger pressing through the vaginal wall can fit into the space between the pubic symphysis.** Reinsert the scalpel to complete the division if any ligaments or cartilage remains. The entire procedure should take 2 to 3 minutes. A variation of the above technique involves simply dividing the pubic symphysis in a single step by a repeated slow stabbing motion.

FIGURE 168-3. Sagittal view of a symphysiotomy using the scalpel blade to divide the pubic symphysis.

Delivery of the infant should follow once the symphysiotomy is completed. Delivery of the infant usually requires more downward traction than is usually necessary.

AFTERCARE

Deliver the placenta and control any postpartum hemorrhage. Carefully return the mother to the supine position with their thighs in normal anatomic position. Close the skin and subcutaneous tissue with simple interrupted 4–0 nylon sutures. Some authors have recommended binding the knees for 12 to 48 hours to prevent inadvertent abduction or external rotation of the hips.[17] Admit the patient to the hospital for observation and monitoring of any potential complications. Leave the Foley catheter in place until any bleeding resolves or the patient begins to walk. Patients may begin ambulation with assistance between the second and fifth days. They should be warned against any unusual straining or heavy lifting.

COMPLICATIONS

Experience and careful case selection appear to be the most important factors in determining outcome and morbidity associated with this procedure.[6] Symphysiotomies do not cause maternal death. Immediate complications include bladder lacerations, urethral lacerations, subcutaneous bleeding, and urinary incontinence.[15,17] Some patients develop gait instability immediately after the operation that tends to be transient and does not appear to affect subsequent pelvic stability.[18]

Lacerations to the Emergency Physician's fingers are a significant concern.[19,20] Prevent lacerations by using the utmost of care and slowly transecting the cartilage and ligaments of the pubic symphysis. The nondominant hand should be double-gloved at a minimum.[19] The use of a Kevlar glove that is resistant to scalpel injury may also be used.[20] A third option is to apply a malleable splint over the palmar aspect of the index finger before double gloving.[19] A final option is to use a finger guard.

Long-term complications include, but are not limited to, stress urinary incontinence, recurrent urinary tract infections, sepsis, and vesicovaginal fistulas.[21] Vaginal fistulas are frequently due to nonplacement of a Foley catheter during the procedure.[8] **This important step should never be omitted.** The effect of a symphysiotomy on subsequent pregnancies has not been studied in detail. The limited data available suggest that a symphysiotomy permanently enlarges the pelvis so that subsequent vaginal deliveries are easier.[3,18] Late-onset osteoarthritis of the sacroiliac joints is possible.[22]

SUMMARY

Modern surgical techniques and obstetrical advances have diminished the practice of a symphysiotomy.[9] The symphysiotomy still remains a simple and safe method to overcome the common and lethal problems of cephalopelvic disproportion, shoulder dystocia, and breech deliveries when an Obstetrician is unavailable or in a resource-limited setting.[2,3,15] The procedure itself can be mastered by any clinician responsible for the care of a woman in active labor.[19] A symphysiotomy is a viable and potentially lifesaving option for anyone who may be working outside the confines of a well-staffed and equipped hospital.

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169 Bartholin Gland Abscess or Cyst Incision and Drainage

Alison Uyemura and Charlie C. Kilpatrick

INTRODUCTION

Bartholin gland cysts and abscesses are common problems for women of reproductive age with an incidence of 2% in this population.[1-4] A Bartholin gland and its duct may enlarge to form a Bartholin cyst or become infected to form a Bartholin abscess. A number of different techniques have been developed for the treatment of Bartholin cysts and abscesses.

ANATOMY AND PATHOPHYSIOLOGY

The Bartholin glands are named after Caspar Bartholin, a Danish anatomist.[1,5] They are located on each side of the vulvar vestibule beneath the fascia, posterolaterally to the vaginal orifice, and at the 4 and 8 o'clock positions.[6,7] The glands are approximately 1 cm in diameter, the size of a pea, and drain through a duct approximately 2.5 cm in length. The ducts of the glands open into the vulvar vestibule at the 5 and 7 o'clock positions, existing between the hymenal ring and the labia minora. The cells of the gland produce mucin which is secreted during sexual excitement and contributes to vaginal lubrication.[5,8] The Bartholin glands are not normally palpable. The gland has an extensive blood supply from branches of the internal pudendal artery. The neural supply is provided from branches of the pudendal nerve.

A cyst of the Bartholin gland may develop most often secondary to obstruction of the duct. This leads to ductal dilation and cyst formation. Noninfectious etiologies of cyst formation include inspissated gland secretions, trauma, tumors of the vulva, or scarring of the duct from repeated bouts of cyst formation.[3] Cysts may grow as large as 3 cm. Bartholin cysts present as painless unilateral swellings in the labial area. A patient will become symptomatic if they become large enough or infected. Infected cysts contain purulent material and are known as a Bartholin abscess, although it is more akin to a pseudoabscess unless the surrounding tissue appears erythematous, tender, and inflamed.[3] A Bartholin cyst may present with pain, dyspareunia, pressure, difficulty with walking, or may be completely asymptomatic.[5,8-12] The diagnosis is made on visual inspection of the vulva. Common signs are a mass near the inferior labia minora, drainage, and erythema.

Primary carcinoma of the Bartholin gland is a rare occurrence but should be considered in the differential diagnosis. Most cancers occurring in the Bartholin gland are metastatic primary vulvar cancers. They can present as a Bartholin gland cyst when the vulvar cancer obstructs the ductal outflow of the gland. Adenocarcinoma (40%), squamous cell carcinoma (40%), adenoid cystic carcinoma (15%), and transitional cell carcinoma (5%) of the Bartholin gland have all been documented.[7,13-20] Carcinoma can easily mimic a Bartholin gland cyst or abscess.

The majority of Bartholin gland cysts appear to be sterile or contain bacteria common to the vaginal flora.[21-23] Studies of Bartholin gland abscesses have shown no bacterial growth in up to 30% of specimens cultured.[23-25] The causative organisms are multiple in the cultures that do grow bacteria. The most prevalent organisms isolated are anaerobes, with *Bacteroides* and *Peptostreptococcus* being the most common species.[24] The remainder of the cultures demonstrated either aerobic/facultative isolates, with *Escherichia coli* being the most common species, or a mixture of both aerobic and anaerobic organisms.[22] *Neisseria gonorrhea* and *Chlamydia trachomatis* have also been implicated as causative agents and have been isolated in up to 16% of cultures.[23,26]

INDICATIONS

Small and asymptomatic cysts in women less than 40 years of age can be managed expectantly. All others should be treated. Any cyst that becomes large and symptomatic (e.g., painful, interfering with physical or sexual activity, or interfering with the flow of urine) should be treated. Any cyst that appears tender, red, hot, or cellulitic represents a Bartholin abscess and requires treatment.

CONTRAINDICATIONS

There is some controversy about the treatment of Bartholin cysts and abscesses in women over the age of 40. Previously, the recommendation was to excise the gland in women over the age of 40 for concern of the possibility of carcinoma. Carcinoma of the Bartholin gland is rare, comprising less than 1% of female genital tract neoplasms.[12] It is felt that women over the age of 40 are at an increased risk for having carcinoma of the Bartholin gland, Bartholin duct, or

adjacent structures; and thus all of these should be properly biopsied or completely excised and sent for pathologic evaluation.[1,7,8,13-15] Another study reported an incidence of Bartholin gland cancer in postmenopausal women of 0.114 per 100,000 women and suggests that selective biopsy be performed to reduce the number of total excisions.[16] Given the rarity of malignancies in this area, this view has been challenged in the recent literature. Currently, drainage and selective biopsy seems to be a reasonable alternative.[8] In general, these women should be referred to a Gynecologist for diagnosis and further treatment.

Patients who have had multiple recurrences and previous treatments are best served with a referral to their Gynecologist for more definitive measures.

Consider draining Bartholin abscesses that are large or extremely tender in the Operating Room. Other indications for drainage in the Operating Room include patients with a contraindication to procedural sedation, those who cannot be adequately anesthetized using procedural sedation and local anesthetics, and those who cannot be safely sedated in the Emergency Department.

EQUIPMENT

SIMPLE INCISION AND DRAINAGE

- Povidone iodine or chlorhexidine solution
- Gloves and gown
- Face mask with an eye shield or goggles
- Drapes
- Lidocaine, 1% or 2%
- 3 mL syringe
- 18, 24, and 25 gauge needles
- Gauze squares, 2×2 and 4×4
- #11 scalpel blade and scalpel handle
- Culture medium for routine bacteria, gonorrhea, and chlamydia
- Hemostat
- Small wick, 2 inches long

INCISION AND INSERTION OF A WORD CATHETER

- Equipment for simple incision and drainage
- Word catheter
- 5 mL sterile saline or water

MARSUPIALIZATION

- Equipment for simple incision and drainage
- 2 small retractors
- Specimen container for excised tissue
- Silver nitrate sticks or electrocautery
- Vicryl, 3–0 or 4–0 on a cutting needle
- Scissors
- Small toothed forceps

WINDOW OPERATION

- Same as equipment for marsupialization

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. The postprocedural care should also be discussed. Obtain a signed consent for the procedure. Some Emergency Physicians may omit the signed consent and place in the procedure note a statement saying: "The risks, benefits, and complications were described and discussed with the patient. They understood this and gave verbal consent for the procedure."

Place the patient in the lithotomy position. Apply povidone iodine or chlorhexidine solution to the labia and allow it to dry. **The surface of the vagina must be anesthetized as the procedure is extremely painful.** The use of ice packs, topical refrigerant spray (Chapter 154), parenteral sedation, parenteral analgesics, or procedural sedation (Chapter 159) should be considered for patient comfort prior to infiltrating local anesthetic solution. Infiltrate 0.25 to 1.0 mL of local anesthetic solution subcutaneously under the mucosal surface of the labia minora for simple incision and drainage with or without the use of a Word Catheter. When injecting local anesthetics, ensure that you do not inject purulent material after aspiration as this can cause extensive inflammation. Infiltrate 2 to 3 mL of local anesthetic solution if a marsupialization will be performed.

The Emergency Physician should be wearing a gown, gloves, and a face mask with an eye shield or goggles. The cyst contents are usually under pressure and can spray out. A face mask and eye protection will prevent a mucous membrane exposure if the contents spray out of the incision. The gown will protect clothing from contamination.

TECHNIQUES

A number of different treatments and procedures have been developed to manage a Bartholin cyst or abscess.[4,20] These are largely influenced by its size, patient symptoms, the age of the patient, the suspected etiology, and any previous treatment in the same patient. The techniques include simple incision and drainage, incision and insertion of a Word catheter, marsupialization, a window operation, and complete excision.

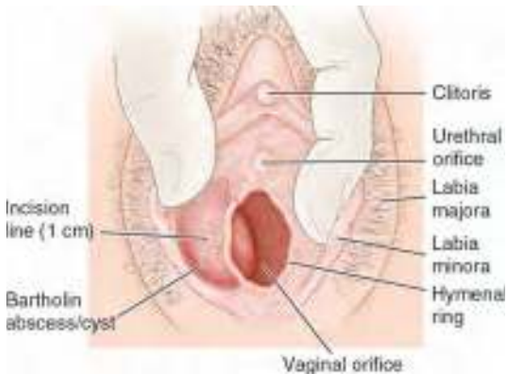
SIMPLE INCISION AND DRAINAGE

The standard treatment for most abscesses is simple incision and drainage.[8,10,11,27,28] Simple incision and drainage of a Bartholin cyst or abscess provides immediate pain relief but is often complicated by chronic recurrences. This technique is not recommended but described for the sake of completeness.

Spread open the labia to visualize the area (**Figures 169-1*A* and 169-1*B***). Make a 1 cm vertical incision on the mucosal surface of the labia minora in the vaginal vestibule. It should be parallel to the border of the hymenal ring, between the hymen and labia minora (**Figure 169-1**). **The incision should ideally be external to the hymenal ring, if possible, and in the area where the duct normally exits.** Extend the incision into the abscess/cyst cavity. **Do not make incisions along the skin surface of the labia minora.** Skin incisions along the labia minora can lead to the formation of a fistula tract, more postprocedural pain, and are fraught with complications. Culture the contents of the sac. Manually express the contents of the sac. Insert a hemostat and break any adhesions. There is no need to irrigate the cavity as this will cause additional pain for the patient and does not affect the outcome.[29] Place a small wick within the cavity and exiting the incision to allow for complete drainage and prevent premature closure of the skin incision.

INCISION AND INSERTION OF A WORD CATHETER

Dr. B. Word first described this procedure in 1964.[5] It is a relatively simple procedure that can be accomplished in the Emergency Department.[5,8,10,11,30] The catheter is approximately 5 cm long and



A

B

FIGURE 169-1. Simple incision and drainage of a Bartholin cyst or abscess. Spread open the labia to expose the area. ***A.*** The dotted line represents the incision line over the cyst or abscess. ***B.*** Scalpel incision results in a purulent fluid. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill, 2016. Photo contributor: Medical Photography Department, Naval Medical Center, San Diego, CA.)

made of soft pliable latex with a 10 French tip. The tip contains a balloon that inflates up to a volume of 5 mL (**Figure 169-2**). These catheters cost approximately $15 each (Berkeley Medevices Inc., Berkeley, CA).

Verify that the patient is not allergic to latex.[9] Fill a 5 mL syringe armed with a 24 or 25 gauge needle with saline or water. Insert the

FIGURE 169-2. The Word catheter. The plastic catheter has a port at its proximal end to insert a needle. The distal end contains a balloon that can be inflated.

needle into the self-sealing port on the distal end of the Word catheter. Inject 3 to 4 mL of the fluid to inflate the balloon. Observe the balloon for any leaks. If no leaks are visible, aspirate the fluid back into the syringe to deflate the balloon. Remove the needle from the self-sealing port.

Spread open the labia to completely visualize the area (**Figure 169-3*A***). Make a 0.5 cm long puncture on the mucosal surface of the vulvar vestibule with a #11 scalpel blade into the cyst or abscess cavity (**Figure 169-3*A***). **This puncture should ideally be located just outside the hymenal ring and where the duct normally drains into the vulvar vestibule, between the hymen and labia minora at the 5 or 7 o'clock position.** The incision should be just large enough to allow the passage of the Word catheter. Culture the contents of the sac. Manually express the contents of the sac. Insert a small hemostat and break any adhesions. There is no need to irrigate the cavity as this will cause additional pain for the patient and does not affect the outcome.[29]

Insert the tip of the Word catheter deep within the cavity (**Figure 169-3*B***). Inject 2 to 4 mL of sterile saline into the free end of the catheter to inflate the balloon (**Figures 169-3*C* and 169-4**). **Pain upon inflation of the balloon or persistent pain after the procedure indicates overinflation of the balloon. Do not use air to inflate the balloon.** The catheter will stay in place because the inflated balloon is larger than the puncture incision. Tuck the free

A B C

FIGURE 169-3. Incision and insertion of the Word catheter. ***A.*** Make a 0.5 cm long stab incision on the mucosal surface of the labia minora. ***B.*** The cavity has been evacuated and the Word catheter inserted. ***C.*** The balloon is inflated with saline.

FIGURE 169-4. The Word catheter is inserted and inflated with saline. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill, 2016. (Photo contributor: Medical Photography Department, Naval Medical Center, San Diego, CA.)

end of the catheter within the vaginal canal. The Word catheter should ideally stay in place for up to 4 weeks. This allows the tract to become epithelialized and prevent a recurrence. The patient should abstain from sexual activity while the catheter is in place.

MARSUPIALIZATION

Marsupialization is an alternative treatment choice initially described in 1950.[1,8,10,11,28,31-34] It is performed once and, in some case series, has been shown to be curative in greater than 90% of patients.[33,35] Advantages are that it can be done as an outpatient, is associated with little postoperative pain, has minimal postoperative scarring, and preserves the gland function.[9] Marsupialization can be easily and safely performed in the Emergency Department using local anesthesia. **Do not attempt to marsupialize a Bartholin abscess with an accompanying cellulitis.** Some Obstetricians prefer to perform this procedure in the Operating Room under general anesthesia.

Make an oval-shaped or elliptical incision approximately 1.5 cm long and 1 cm wide through the vestibular mucosa over the cyst/abscess and just outside the hymenal ring (**Figure 169-5*A***). Remove this piece of mucosa to visualize the anterior wall of the cyst/abscess cavity. Insert a retractor to pull the skin edges open and better visualize the sac (**Figure 169-5*B***). Make a similar incision through the anterior wall of the cyst/abscess cavity (**Figure 169-5*B***). Most times the initial incision will pierce both the vaginal mucosa and cyst wall. Remove this piece of tissue, leaving the cavity exposed. Culture the contents of the sac. Express the contents of the sac. Insert a small hemostat and break any adhesions. There is no need to irrigate the cavity as this will cause additional pain for the patient and does not affect the outcome.[29]

Control any bleeding with electrocautery, silver nitrate application, or manual pressure. Closing the cavity with interrupted sutures, as a final option, will control the bleeding. Suture closure is only temporary for hemostasis as this pocket will become reinfected and require another procedure. Remove any sutures placed for hemostasis in 20 to 30 minutes. Identify the former bleeding site and treat it with electrocautery or silver nitrate to prevent rebleeding after the patient leaves the Emergency Department.

Evert the cyst/abscess wall. Approximate the wall of the cyst/abscess to the vestibular mucosa. Sew the wall of the cavity to the mucosa of the vulvar vestibule with interrupted 3–0 or 4–0 Vicryl sutures (**Figure 169-5*C***). The orifice will reduce in size, epithelialize, and form a new duct in time. **Send all excised tissue to the Pathology Department to confirm the diagnosis and to rule out neoplasm.[18,19]**

WINDOW OPERATION

This technique is almost identical to marsupialization.[36] It differs only in the size of the incision. One study claimed this procedure had no long-term complications or recurrences thus making it a more desirable technique.[36] However, the total patient population was small and the study has not been duplicated and republished. In essence, one follows the exact procedure for marsupialization with the exception of making a larger incision (2 to 3 cm long and 1.5 cm wide). At the completion of the procedure, a wide opening is observed. The opening will be approximately one-half its original size at the 1 year follow-up. This much larger and radical incision is not recommended for the Emergency Physician.

COMPLETE EXCISION

This technique is reserved for the treatment of recurrent cysts and abscesses unresponsive to less invasive techniques.[8,10-13,16,28,36] It is also indicated for patients in whom carcinoma is suspected. Even though the function of the gland is to provide lubrication for sexual activity, in a well-estrogenized woman removal of the gland poses

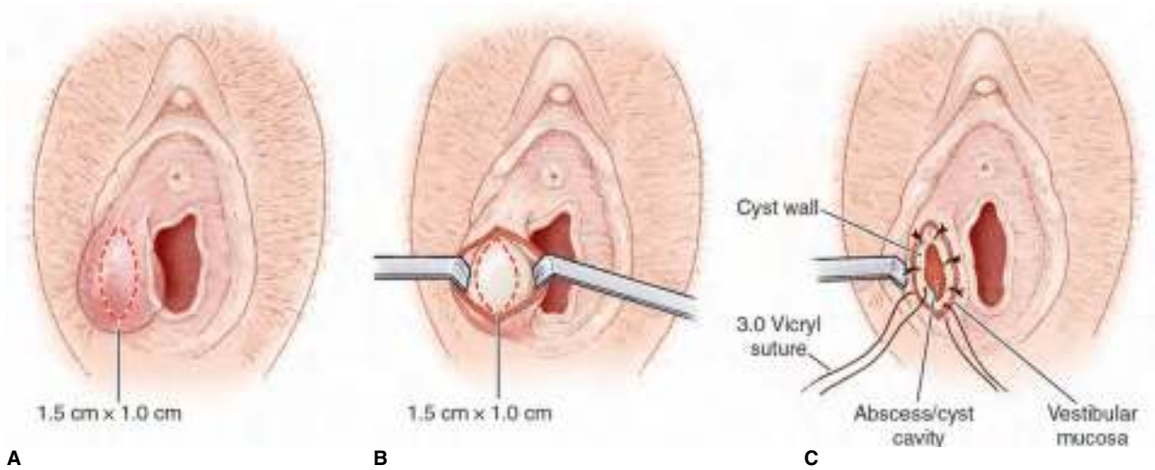


FIGURE 169-5. The marsupialization of a Bartholin cyst or abscess. ***A.*** An elliptical incision, 1.5 cm in length and 1.0 cm in width, is made through the vulvar mucosa and just outside the hymenal ring. The ellipse of mucosal tissue is then removed. ***B.*** An elliptical incision (dotted line) is then made through the anterior wall of the cyst or abscess. ***C.*** Approximation of the cyst or abscess wall to the vestibular mucosa with 3–0 or 4–0 interrupted Vicryl suture.

little clinical problem. In the past, excision of the Bartholin gland in all women over the age of 40 for fear of carcinoma was championed. This has been challenged given the low incidence of carcinoma of the Bartholin gland in favor of drainage and selective biopsy.[8] Significant bleeding may complicate dissection into this area due to the underlying anastomosing venous plexus. If hemostasis is not achieved, a vulvar hematoma will result. Excision of the gland is often referred to as the "bloodiest little operation in gynecology." This procedure should only be performed by a Gynecologist in the controlled setting of an Operating Room under general anesthesia.

ALTERNATIVE TECHNIQUE

An alternative to the Word catheter has been described in the literature.[37-39] The advantages of this technique are that the materials are available in every Emergency Department, the device does not prematurely fall out as often happens with the Word catheter, and it is easily placed. Unfortunately, these reports had few patients. Larger studies comparing this technique to the Word catheter are required.

Prepare the equipment. Obtain a 5 to 7 cm long piece of tubing. Suggestions include cutting a piece from an 8 French T-tube or a butterfly intravenous catheter.[37-39] Insert a strand of 2–0 silk suture, without a needle or the needle cut off, through the tubing (**Figure 169-6*A***).

The procedure is quite similar to that previously described. Make a stab incision in the lower half of the abscess and outside the hymenal ring with a #11 scalpel blade (**Figure 169-6*B***). Insert a hemostat and break any loculations (**Figure 169-6*C***). With the hemostat in the abscess cavity for support, make a second stab incision in the upper half of the abscess (**Figure 169-6*D***). Insert one end of the silk suture into the upper or second incision and grasp it with the hemostat (**Figure 169-6*E***). Pull the suture and tubing out through the first or lower incision (**Figure 169-6*F***). Tie the ends of the suture to form a ring with the tubing (**Figure 169-6*G***). Place four to five knots in the suture. Cut off the excess suture.

AFTERCARE

All abscess and cyst contents should be cultured. The initiation of antibiotics can be delayed pending the culture results at the discretion of the physician. Administer antibiotics if the abscess is accompanied by cellulitis. Keep in mind the prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) in soft tissue infections and specifically in labial abscesses when choosing antibiotics.[40-42]

All patients should begin sitz baths in 2 days. Prescribe appropriate oral analgesics. Nonsteroidal anti-inflammatory drugs will provide most patients with adequate analgesia. Some patients may

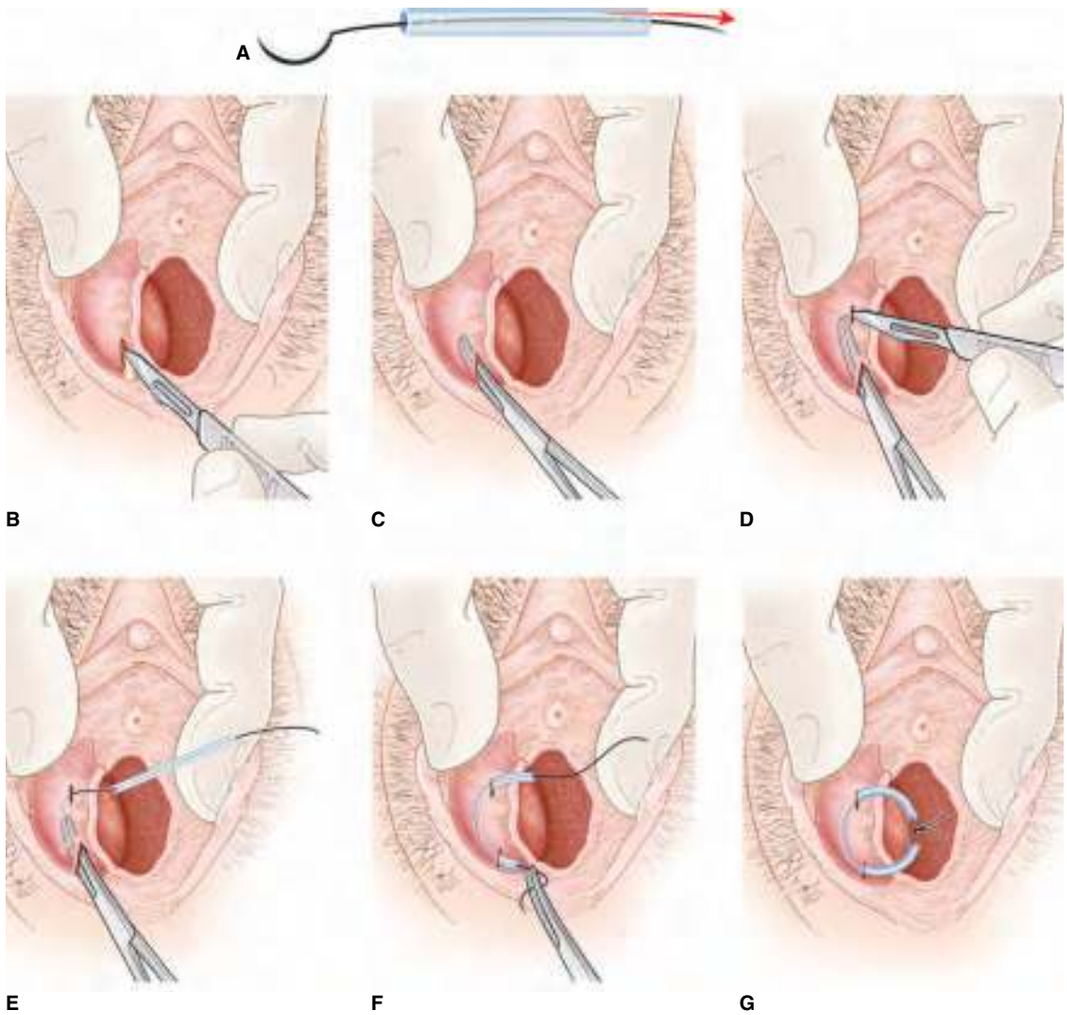


FIGURE 169-6. An alternative technique to incise and drain a Bartholin cyst or abscess. ***A.*** A silk suture has been inserted through the tubing. ***B.*** A stab incision is made in the lower half of the abscess and outside the hymenal ring. ***C.*** A hemostat is inserted to break any loculations. ***D.*** The hemostat provides support as a second stab incision is made in the upper half of the abscess. ***E.*** One end of the silk suture is grasped with the hemostat. ***F.*** The suture and tubing are pulled out through the lower incision. ***G.*** The suture ends are tied to form a ring with the tubing.

require a brief course (1 to 3 days) of narcotic analgesics. Give written discharge instructions to each patient explaining the aftercare, the complications, and the actions to take if a complication does arise. All patients should abstain from sexual activity until the inflammation and pain resolve or until the Word catheter is removed.[43]

COMPLICATIONS

The most common complication with the incision and drainage technique is frequent recurrence. The most common complication of Word catheter insertion is premature loss of the catheter resulting in early incision closure and recurrence. This can be due to a large incision, underinflation of the balloon, injection through or piercing with the needle other than the injection site, or more than two needle punctures to the injection port. The Word catheter can also be associated with significant discomfort if the catheter is inserted improperly. Marsupialization has few complications and a low recurrence rate. With any of these procedures, one can miss a carcinoma of the Bartholin duct or Bartholin gland if a biopsy or excision is not performed. Bleeding and progressive infection, including sepsis and toxic shock syndrome, are rare but possible complications.[44,45]

The immunocompromised patient (e.g., diabetic, steroid dependent, or human immunodeficiency virus positive) needs particular attention paid to cleanliness during and after the procedure. If the abscess is accompanied by cellulitis and/or involves the labia majora, consider inpatient management and gynecologic consultation. Initial intravenous antibiotics should cover MRSA. A large Bartholin/labial abscess in a diabetic patient requires close surveillance and a high suspicion for necrotizing fasciitis. Daily follow-up visits are recommended for these patients during the initial postprocedural period. Early hospitalization and administration of intravenous antibiotics are warranted if there is any sign of progressive infection.

SUMMARY

Bartholin cysts and abscesses will be encountered by the Emergency Physician and can easily be treated. They present as a painful unilateral swelling of the inferior aspect of the labia. There are many treatments option. Culture all cyst or abscess contents. Send any tissue removed to the Pathology Department to rule out carcinoma. Refer patients with previously treated cysts or abscesses, those suspicious for carcinoma, and those over the age of 40 to a Gynecologist for definitive care. Give all patients written discharge instructions at the end of the procedure. All immunocompromised patients require very careful follow-up.

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170 Sexual Assault Examination

Monique A. Mayo and Christopher J. Russo

INTRODUCTION

Sexual assault is defined as forced sexual contact without consent. Sexual assault involves the threat of force, the use of force, or a person's inability or refusal to give consent (e.g., children, dementia, and unconsciousness).[1] The exact legal definition of sexual assault varies from state to state. Nonconsensual sexual contact involves a continuum ranging from unwanted touching and fondling to forced penetration (i.e., anal, oral, or vaginal). Fingers or objects (e.g., bottles, broomsticks, or knives) could be used instead of or in addition to a penis as a weapon of choice. Drugs (e.g., alcohol, gamma-hydroxybutyric acid [GHB], ketamine, and Rohypnol [flunitrazepam]) are commonly used as "date rape" drugs.[2-4] These agents can be used to disable the victim prior to a sexual assault. **Alcohol remains the most common intoxicant involved in a sexual assault.**[5]

Approximately 300,000 to 700,000 adult women are victims of sexual assault annually in the United States.[6] The lifetime prevalence of sexual assault for women is 18% to 19% and 2% to 3% for men.[6,7] The prevalence may be grossly underestimated with only 10% to 15% of all assaults being reported.[6] Vulnerable populations (e.g., homeless, institutionalized, or sheltered) are not included in most large studies looking at the incidence or prevalence.[8]

Sexual assault is not generally a crime committed by strangers. Most women (78% to 82%) are sexually assaulted by someone known to them.[6,9] The assailant may be an acquaintance, coworker, family member, neighbor, significant other, or spouse. More than 50% of rape victims over the age of 30 are sexually assaulted by an intimate partner.[9] Even fewer women report assault when it is committed by someone they know.

Victims do not fit a specific profile. Adolescent girls and young women face particularly high rates of sexual assault.[10] Victims have been reported in all age groups. Sexual assault occurs across all ethnic backgrounds, racial backgrounds, and socioeconomic groups. Up to 39% of women will be raped more than once during their lifetime.[6]

Some victims will not identify themselves as victims of sexual assault.[1] They may be ashamed or fearful to disclose what happened. They may be experiencing the rape trauma syndrome which is a special category of posttraumatic stress disorder. The use of screening questions and a high index of suspicion are necessary to identify these patients. It is clear there is a significant increase in the utilization of medical resources after a sexual assault and early identification of potential victims may be useful.[11]

The Emergency Department visit of a sexual assault victim is vital in assuring evidence collection and proper medical treatment.[5,12] Proper follow-up plans, medical appointments, and referrals to local rape crisis centers and/or hotlines are crucial for the mental and physical recovery of victims. The likelihood that the patient will seek follow-up care and pursue the case through legal avenues is strongly influenced by their initial experience in the Emergency Department.[13] It is critical to not revictimize the patient or make them feel responsible for the sexual assault.[13]

INDICATIONS

Perform a sexual assault examination, including evidence collection, on all patients who present within 5 days of a sexual assault. Evidence collection has the highest yield if collected within 72 hours after the sexual assault. Some states may recommend evidence collection up to 14 days from the alleged sexual assault. Proceed with the examination if the victim is unsure of when the assault occurred or when the patient is unsure if a sexual assault occurred. Perform a thorough history and physical examination with extra attention to the areas violated to evaluate for injuries, even when the patient presents more than 5 days after the sexual assault. Evidence collection requirements vary from state to state. Samples collected prior to the assignment of a case number cannot be admitted as evidence in some states. Be familiar with local laws.

Perform a complete history regardless of when the assault took place.[1,14] Patients must consent to the evidence collection process (Figure 170-1). Proceed with the evidence collection if a patient is unable to consent due to a medical condition, with local statutes and hospital policy determining the release of evidence. Minors do not require parental consent for the initial evaluation of any potential life-threatening injuries. State laws mandate the age with which a minor can be examined and treated for medical complaints without parental consent related to sexual activity and pregnancy. Many states require parental permission for the release of evidence.

CONTRAINDICATIONS

Manage life-threatening injuries and unstable vital signs according to usual recommendations prior to evidence collection.[1] Sexual assault victims may present with major nongenital physical injuries that must be addressed prior to any assault examination or evidence collection. Up to 5% of sexual assault victims have major nongenital physical injuries.[15] **Do not perform a sexual assault examination if a patient does not consent to the examination unless unconscious or due to a medical condition.** Victims must be able to consent to the examination and evidence collection unless there is a medical reason they cannot consent. Document this reason in the medical record.

EQUIPMENT

- Separate paper bags for clothing and undergarments
- Clean white sheet and/or large paper sheet to place on floor when patient disrobes
- 4 sterile cotton-tipped swabs for oropharynx evidence collection
- Pharyngeal culturettes or nucleic acid amplification test (NAAT) swabs for chlamydia or gonorrhea
- 4 sterile cotton-tipped swabs for vaginal, cervical, or penile evidence collection
- Culturettes or NAAT swabs for vaginal or penile gonorrhea and chlamydia
- Slides for wet mount *Trichomonas* and/or NAAT swabs for vaginal or penile *Trichomonas*

PLEASE SEE INSIDE BOX COVER

STEP 1

Illinois State Police
Division of Forensic Services

Please print, type or use a patient information stamp.

(patient stamp)

PATIENT CONSENT/AUTHORIZATION TO RELEASE EVIDENCE TO LAW ENFORCEMENT AGENCY

Patient _____

DOB _____ Hospital Patient No. _____

CONSENT

I understand that in addition to consent for medical evaluation and treatment given to the hospital and physician, I also give consent to a medical examination for evidence of sexual assault. I understand that the evidence collected in the State Police Evidence Collection Kit will be sent to the Illinois State Police Forensic Sciences Command for the sole purpose of analysis to determine sexual activity relating to the investigation of sexual assault. I understand that this exam may include reference samples. I also understand that I may withdraw consent at any time for any portion of the exam.

_____
Patient, Parent, Guardian (please circle)

I understand that collection of evidence may include photographing injuries and that these photographs may include the genital area. Knowing this I consent to having photographs taken.

_____
Patient, Parent, Guardian (please circle)

RELEASE

I hereby authorize _____ (Name of hospital) _____ (City) to release to _____ (Law enforcement agency name)

the following information covering treatment given to me on _____ (Month) (Day) (Year).

	AUTHORIZED FOR RELEASE (check those which apply)	NOT AUTHORIZED FOR RELEASE (check those which apply)
1. Copies of Forensic Laboratory Report Form	☐	☐
2. State Police Evidence Collection Kit	☐	☐
3. Photographs	☐	☐
4. X-rays or copies of x-rays taken in connection with exam	☐	☐
5. Clothing	☐	☐

Authorized for release (list clothing or miscellaneous items)

Article	Description
_____	_____
_____	_____
_____	_____

Signature of person authorizing release of information _____
Patient, Parent, Guardian (please circle)

STEP 13

RECEIPT OF INFORMATION

I certify that I have received the following items (check those which apply):

☐ One sealed evidence collection kit ☐ X-rays ☐ Photographs ☐ Swabs/specimens (if no evidence collection kit is used)

☐ Sealed paper clothing bag(s) (if more than one sealed clothing bag, please note) ☐ Other _____

Signature of person receiving information and/or articles _____ Date _____ Time _____

Officer ID# and rank _____ Representative of _____

Name of hospital representative who is releasing articles _____
(Printed name) (Signature)

ILSP501

(White copy to hospital; yellow copy to law enforcement agency)

FIGURE 170-1. Sample form for patient consent and release of information to a law enforcement agency. It includes the receipt of information form to be signed by the law enforcement officer when the evidence is transferred to their care. (Courtesy of the Illinois State Police.)

- 4 sterile cotton-tipped swabs for rectal evidence collection
- Culturettes or NAAT swabs for rectal chlamydia and gonorrhea
- Extra sterile cotton-tipped applicator swabs for stains and bite marks
- Comb and a piece of clean white paper for loose pubic hairs
- Comb and a piece of clean white paper for loose head hairs
- 2 sticks for fingernail scrapings
- Filter paper for blood and/or saliva collection, depending on your state requirement
- Urine collection equipment for chlamydia and gonorrhea testing as an alternative to swabs or culturettes
- Urine or blood collection equipment to test for pregnancy
- Equipment for serum collection to test for hepatitis, human immunodeficiency virus (HIV), and syphilis
- Digital camera or forensic photographer for documentation
- Colposcope, as applicable
- Sterile water to moisten speculum and cotton-tipped swabs
- Change of clothing for the patient
- Separate envelopes to place evidence within for each body site
- Tape to seal all evidence that can be written upon with ink

Most states have prepackaged evidence collection kits with most of the necessary equipment included (**Figure 170-2**). These kits usually do not contain paper bags for the clothes, culturettes, NAAT swabs, blood drawing equipment (i.e., needles, Vacutainers), and blood collection tubes. All these are readily available in any Emergency Department.

A

B

FIGURE 170-2. Examples of the sexual assault evidence collection kits. ***A.*** The kit. ***B.*** Contents of the kit.

A

B

FIGURE 170-3. Examples of toxicology and drug testing kits. ***A.*** Urine kit. ***B.*** Blood kit.

Use only paper bags and not plastic bags for the collection. Plastic bags retain moisture and do not breathe. This can promote mold formation and destroy DNA evidence.

A separate kit is available for toxicology and drug testing (**Figure 170-3**). Use these items and not hospital supplies for specimen collection and analysis. Do not use the supplies available in the Emergency Department. Kits can be used for urine or blood.

Use a colposcope if trained in its use. This is a routine part of the sexual assault examination and used by some practitioners. The colposcope provides a magnified view of the area and allows one to distinguish any abnormalities. It consists of a monocular or binocular microscope that takes photographs and/or video (**Figure 170-4**). The colposcope picks up details not seen by the unaided eye (e.g., abrasions, bruising, lacerations, and tears). The application of a dye to the tissues allows abnormalities to be easier visualized through the colposcope. The colposcope enhances the examination by being noninvasive, examining internal and external structures, providing light, providing magnification, measuring accurately, and recording images (i.e., photographs and video).

PATIENT PREPARATION

Address any life-threatening injuries and unstable vital signs before proceeding to the formal sexual assault examination. Do not to cut or rip any clothes to best preserve evidence. Cut or torn clothing for the appropriate care of the patient often supersedes

FIGURE 170-4. Examples of digital colposcopes. ***A.*** Seiler 955 (Seiler Instruments Inc., St. Louis, MO). ***B.*** SW-3306. (Jiangsu Sanwe Medical Technology Inc., Jiangsu Province, China.)

any evidence collection. Make a note of any clothing that is cut or ripped for patient care in the medical record.

Conduct the examination in a quiet and private room.[1] The room should ideally have the facilities to perform a pelvic examination so that the patient does not have to be moved multiple times. Assign a designated nurse and physician who are trained in the sexual assault protocol.[5,16] Keep the number of providers to a minimum. **Be aware of the multiple emotional manifestations that the patient may experience. Safety and privacy must be ensured.** Notify a community-based or hospital-based advocate upon the patient's arrival to the Emergency Department. Advocates can best define their own role. Allow the patient to decide if the advocate should stay for the examination and evidence collection. Contact the Police in the jurisdiction of the sexual assault and the Department of Children and Family Services (DCFS) according to local laws and patient request. All providers can be subpoenaed to testify in the event of legal action. Alert hospital security to the possibility that the assailant may come to the hospital.

The patient, or their guardian for a minor, must give written consent prior to the examination (**Figure 170-1**). **Obtaining consent has important legal and psychological implications for the patient. It is important to let the victim guide the process as much as possible to help them restore control of their life. Let the victim know that you are their advocate. The sexual assault victim has the right to refuse the sexual assault examination, any medical treatment, and any interviews by advocates or social workers.** Note in the medical record any portions of the sexual assault examination and treatment that the patient refuses. **Encourage the patient to at least have the examination without the use of the sexual assault kit to provide proper medical treatment. Encourage the patient to allow the collection of evidence with the sexual assault kit if they later change their mind and decide to prosecute the assailant.**

Discourage the patient from changing clothes, defecating, drinking, eating or urinating prior to the examination. This is to best preserve evidence, especially if the patient is seen in close time proximity to the sexual assault. Perform examinations specific to the affected area earlier to accommodate the patient if they need to drink or void.

EXAMINATION OF FEMALE PATIENTS

Exact details of the examination will vary depending upon state and local evidence collection requirements.[5] Some states have provisions for Sexual Assault Nurse Examiners (SANEs) who are specially trained to perform the complete sexual assault examination and testify if the case is prosecuted.[17] Other regions have a sexual assault response team (SART) with representatives from law enforcement, forensics, health care, the prosecutor's office, and rape crisis centers.[18] SANE and SART programs increase compliance with recommended medical care, quality of evidence collection, and the likelihood that charges will be filed and successfully prosecuted.[8]

The order of the examination may vary depending upon the patient's needs. The Emergency Department is often the first official system to which the victim reports an assault. **Patients may be reluctant to share their story with personnel if they are met with judgmental attitudes or insensitive treatment.[19]**

Collect a urine sample early if there is any possibility of the use of a "date rape" drug. The decision to process the urine for toxicologic analysis can be determined later. The window to detect drugs (e.g., GHB or Rohypnol [flunitrazepam]) can be as little as 8 hours after ingestion.[2,4] Most toxicology screens will detect prescription drugs and recreational drugs that the patient may have taken. For this reason, some patients may decide not to have their urine tested.

HISTORY

Proceed with the history and physical examination once the patient is in a private room, the patient has consented to the examination, and an advocate is made available. Obtain a complete

STEP 2

FORENSIC LABORATORY REPORT
(4 PAGES)

Illinois State Police
Division of Forensic Services

Please print, type or use a patient information stamp.

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Patient Name		DOB	Age
Race	Sex	Arrival Date	Arrival Time
Address		City	
County	State	Zip	Phone
Hospital			ER#
For Children: Name of Guardian			Relationship
Person providing history		Relationship to patient	

For children: Avoid multiple interviews. Take time to establish rapport. Avoid leading or yes/no questions. Use direct quotes. Avoid surprise or negative emotions, while still showing concern and support.

1. Chief complaint of person providing history to include physical injuries and methods employed by perpetrator, i.e., weapons, restraint, biting, threats.

2. Chief complaint in child's words to include physical injuries and methods employed by perpetrator, i.e., weapons, restraint, biting, threats.

3. Date of Assault	Time of Assault	4. Location & geographical surroundings of assault

5. Name(s), number, & race of assailant(s) if known.

6. Sexual acts described by patient/historian

Acts Described	Yes	No	Attempted	Unsure
Penetration of vagina by:				
penis				
finger				
foreign object				
describe object				
Penetration of rectum by:				
penis				
finger				
foreign object				
describe object				
Oral copulation of genitals:				
of victim by assailant				
of assailant by victim				

Acts Described	Yes	No	Attempted	Unsure
Masturbation:				
of victim by assailant				
of assailant by victim				
Did ejaculation occur?				
inside body orifice				
outside body orifice				
If outside body orifice, describe location				
Other sexual acts?				
If yes, describe				
Did assailant use condom?				
Did you bite your assailant?				
Were you bitten by your assailant?				
Victim was licked/sucked?				
If yes, where_____				
Victim licked/sucked assailant?				
If yes, where_____				

7. Post-assault hygiene/activity	Yes	No
urinated	☐	☐
defecated	☐	☐
genital wipe/wash	☐	☐
bath/shower	☐	☐
douche	☐	☐
tampon, sponge, diaphragm removed/inserted (circle)	☐	☐
oral hygiene/intake/vomit	☐	☐
changed clothes	☐	☐

8. Pertinent medical history
   a. Date of LMP
   b. Sexual activity within 72 hrs. of assault?
      Yes No
   c. Contraceptive used at time of assault?
   d. Communicable diseases of risk to lab personnel
      (e.g., hepatitis, TB, HIV, lice, etc.)

ILSP502A

(White copy to hospital; yellow copy in kit)

FIGURE 170-5. Sample history form. (Courtesy of the Illinois State Police.)

history to guide the medical examination and help with possible legal matters (**Figure 170-5**). **Avoid judgmental questions that may feed into the victim's feeling of self-blame. Focus on asking the patient open-ended questions.** Inquire about the time and place of the assault, the race of the assailant(s), their gender, and the number of assailants. Obtain and document a brief description of the assault including whether there was oral penetration, rectal penetration, vaginal penetration, and/or ejaculation. Elicit

any use of foreign objects, force, lubricants, or restraints. **Ask the patient and document what they have done since the assault.** Simple things (e.g., bathing, changing clothes, defecating, douching, urinating, or using a tampon) may change the ability to collect forensic evidence.

The past medical history should emphasize the gynecologic history and include last menstrual period, type of birth control used if any, last consensual intercourse, previous sexually transmitted infections (STI), previous pregnancies, and previous gynecologic surgeries. **The time of the last voluntary intercourse is important as mobile sperm may be found for up to 72 hours in the cervix.** Determine the tetanus immune status and provide immunoglobulin and boosters if needed. Try to determine if "date rape" drugs might have been given, especially if there seems to be a lapse of time.[2]

PHYSICAL EXAMINATION

The physical examination serves to detect injuries and document them for future legal prosecution. Perform a complete physical examination, even if the patient does not want to pursue legal matters. Although up to 70% of rape victims reported no physical injuries, 4% to 5% sustained serious physical injury and 24% sustained minor physical injuries.[6,20] **It is important to help patients guide the examination and allow them to stop at any point if they are not ready to proceed. Many patients will need encouragement through the examination.**

Note the patient's general appearance, affect, and emotional status (**Figure 170-6**). Instruct the patient to disrobe over a clean paper and sheet if they are wearing the same clothes they wore prior to the assault. Place all clothes in paper bags, with the underwear in a separate paper bag. Fold the paper sheet, containing any debris, and place it in a collection envelope. Examine the entire body for abrasions, bites, ecchymoses, foreign bodies, lacerations, and scratches. **Closely examine every laceration to ensure it is not a stab wound.** Use a body diagram to document any injuries (**Figure 170-6**). Look for any signs of strangulation and document the negative or positive findings.[21]

Genital trauma after consensual sex is possible. Genital injuries can occur from the sexual assault. Most common injuries occur at the fossa navicularis anterior to the fourchette, hymen, labia minora, and posterior fourchette.[22] Commonly injured nongenital areas due to the assault include the breasts, mouth, thighs, throat, and wrist.[19] Oral cavity injuries can include broken teeth and a torn frenulum. Bite marks on the breast and genitalia are common.[22] Use an alternative light source (e.g., Wood's lamp) to examine the skin for fluorescent stains that may represent saliva, semen, or urine. Use a digital camera or a forensic photographer to document any abrasions, bites, lacerations, scratches, or any other injuries. Use a measuring tape to show the size of each injury that is photographed. Take close-up pictures of the injury for detail and pictures further away to show the body location of the injury. **Photographs are much more informative than body diagrams.**

Collection of forensic evidence usually precedes the gynecologic examination unless the patient is bleeding, has severe lower abdominal pain, or has pelvic pain. Most states have set evidence collection kits and the elements required will vary. Evidence is most useful up to 3 days after an assault. Collection of evidence can be performed up to a week after an assault as patients may not recall the dates exactly. Most sexual assault kits require fingernail scraping, head hair combing, saliva specimens, and blood type screening. Obtain samples of the patient's saliva. Follow the instructions on the state kit for this collection. Obtain a sample of the patient's blood and again follow the instructions of the state kit which may include the use of filter paper. Allow any specimen on filter paper to air-dry before placing it in the envelope.

Swab any stain on the patient's body that fluoresces under the Wood's light or an alternative light source.[23] Swab all orifices (i.e., anal, oral, and vaginal) even if not penetrated as the recollection of events may change and a negative examination in a nonpenetrated area helps to validate the patient's story. **Use only sterile water or sterile normal saline to moisten a swab. Allow all specimens to air-dry before placing them in envelopes.** Use a wooden stick to scrape under all the patient's fingernails over a piece of white paper to collect the scrapings.

Collect hair samples from the victim. Pluck two or three hairs from the scalp hair and place them in a labeled envelope. Comb the patient's pubic hair over a white piece of paper. Place the comb, paper, and any hair or debris in a labeled envelope. Pluck two or three hairs from the pubic region and place them in a labeled envelope. The process of plucking hairs can be painful and somewhat insensitive to the patient. **Consider asking the patient to pluck the hairs for the evidence collection.** This may be better tolerated by the victim and allows them to actively participate in the process. Hair samples can be obtained later if the case is prosecuted as the patient's hair samples will not change. **Cutting off pieces of hair is of no value as the roots of the hairs are required for the forensic evidence process.**

Inspect the mouth and perioral structures for any signs of trauma. Carefully inspect the frenulum of the lower lip and tongue for bruising or tears. Examine the tonsils, the tonsillar pillars, and the oropharynx for bruising or lacerations. Document and photograph any injuries. Swab the oral cavity thoroughly and allow the swabs to air-dry. Obtain additional swabs for chlamydia and gonorrhea testing in the hospital laboratory. Prepare a wet mount to look for spermatozoa.

Thoroughly examine the anorectal area for signs of trauma (e.g., abrasions, ecchymoses, and lacerations). Document and photograph any injuries. Swab the rectum and anal canal. Send one swab to the hospital laboratory for chlamydia and gonorrhea testing. Place the remainder of the air-dried swabs in envelopes for evidence collection and testing.

The gynecologic examination is usually the most traumatic aspect of the examination for the patient. It may remind them of the assault. **Explain all procedures in simple terms prior to beginning. Allow the patient to help guide the examination.** Do all the forensic evidence collection at the same time as the gynecologic examination. Examine the external genitalia (**Figure 170-7**).[24] Document and photograph any injuries. Examine the hymen as it is one of the most common sites injured.[25] Document in writing and photographs its shape and any lesions, notches, or injuries. Up to 8% of patients have vulvar trauma.[26] Examine the internal vaginal vault and the cervix. **Only use sterile water to lubricate the speculum. Lubricants interfere with forensic evidence collection.**

Collect baseline chlamydia and gonorrhea swabs at the time of the pelvic examination from pooled vaginal secretions and the endocervical canal. This testing can be done via NAAT testing in the urine. Complete the studies based on the policies and procedures of the individual hospital. Obtain additional swabs in the vagina and of the cervix to test for evidence. Allow these to air-dry before placing them in an envelope. Colposcopy is used to detect and document more subtle injuries of the cervix and vagina (**Figure 170-8**).[27] Colposcopy increases the detection of genital trauma from 6% to 53% of victims.[28]

Toluidine blue can be used to identify small abrasions and lacerations not easily detected by looking. It can increase the chances

FORENSIC LABORATORY REPORT — PAGE 2

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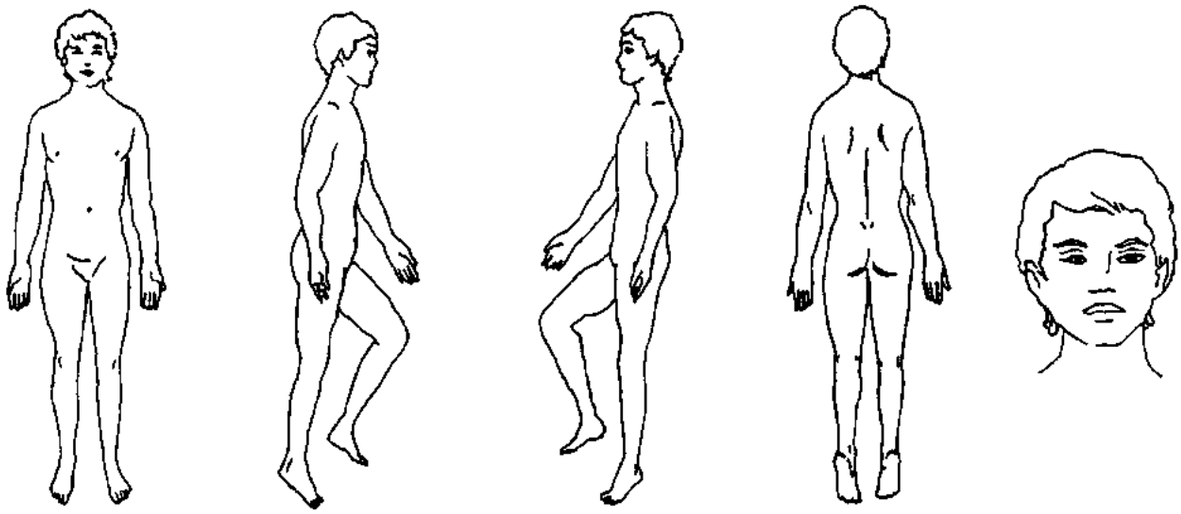
COMPLETE EVIDENCE COLLECTION STEPS 3, 4, AND 5

After STEP 5, obtain appropriate medical specimens from the mouth for clinical lab testing. Patient may rinse out his/her mouth after specimens are obtained. (DO NOT INCLUDE MEDICAL SPECIMENS IN KIT.)

GENERAL EXAM

Trauma should be recorded on the diagrams below which may be used in a criminal proceeding. These include: lacerations, scratches, bruises (detail size, shape, color), erythema, bites, patterned injury, burns, fractures and stains/foreign materials on body, swelling, tenderness. Be sure to note even the most minor signs of trauma. In children: include anogenital or behavioral symptoms. Note general appearance.

Note abnormalities in diagram and/or text.



TEXT

GENITAL EXAM

- For children: Take time to establish rapport and proceed slowly. Extent of examination, including physical as well as specimens, must be decided on a case-by-case basis by the attending physician. If the examination would be too physically or emotionally traumatic for the child, then specimens may need to be obtained by gently using a moist swab on the external vaginal and rectal areas, especially with small hymenal openings.
- Note all discharge, stains, and foreign materials. Note any bleeding.
- Draw shape of hymen and anus in diagrams.
- Use sterile, non-bacteriostatic water or saline for lubrication of speculum.
- Record all acute trauma (lacerations, scratches, bruises [detail size, shape and color], erythema, bites, patterned injury, burns, swelling, tenderness) and chronic trauma (scarring, notching, pigmentary changes) on below diagrams.

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FIGURE 170-6. Sample general physical examination form with body diagrams. (Courtesy of the Illinois State Police.)

for detection of abrasions and lacerations in sexual assault victims. Apply the dye with a cotton-tipped applicator to the external genitalia and wipe off the excess. Document and photograph any areas of uptake of the toluidine blue. Use the toluidine blue on the external genitalia prior to the speculum examination as the speculum can result in small lacerations that result in dye uptake.[29] **The dye is spermicidal and can interfere with wet mount examinations.**

FORENSIC LABORATORY REPORT — PAGE 3

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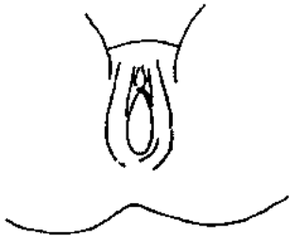
(patient stamp)

EXTERNAL GENITALIA

• Note abnormalities in diagrams and/or text.

Vulva/Hymen



• position during exam	lithotomy	knee chest	supine
• labia majora maneuver?	If yes, outward traction		lateral-down separation
• horizontal hymenal opening (with above position and maneuver)			mm
• description of hymen			
• draw picture of hymen on diagram			

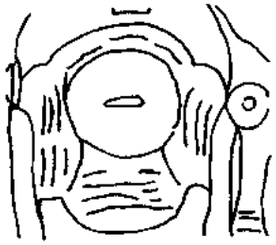
Penis/Scrotum

• circumcised? Yes ☐ No ☐

INTERNAL EXAM

• As noted above, most children require only vaginal introital specimens without speculum or bimanual exams. Please individualize.

Vagina and Cervix



Uterus, Adnexa

• Note abnormalities in diagram and/or text.

• Note abnormalities on bimanual exam, if indicated.

COMPLETE EVIDENCE COLLECTION STEP 6

After Step 6, obtain appropriate medical specimens from the vagina/cervix or male urethra for clinical lab testing (DO NOT INCLUDE MEDICAL SPECIMENS IN KIT.)

RECTAL EXAM

• Digital or anoscopic exam at discretion of physician.

• Note abnormalities on above diagrams and/or text.

COMPLETE EVIDENCE COLLECTION STEPS 7 - 12

After Step 7, obtain appropriate medical specimens from the rectum for clinical lab testing. (DO NOT INCLUDE MEDICAL SPECIMENS IN KIT.) After Step 11, obtain appropriate blood specimens for clinical lab testing. (DO NOT INCLUDE MEDICAL SPECIMENS IN KIT.)

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FIGURE 170-7. Sample physical examination form with diagrams for the genital examination. (Courtesy of the Illinois State Police.)

FIGURE 170-8. Colposcopy. (Used with permission from www.superstock.com.)

EXAMINATION OF MALE PATIENTS

Perform a complete urogenital examination in male victims looking for abrasions, bites, and lesions (**Figure 170-7**). Observe the same disrobing, specimens, and precautions in male sexual assault victims. Obtain oral, rectal, and urethral swabs for chlamydia and gonorrhea. Chlamydia and gonorrhea can be detected in urine of males eliminating the need for most urethral swabs.[30] Swab the penis and scrotum with separate sterile cotton-tipped applicators.

EXAMINATION OF CHILDREN

The procedure for an adolescent or postpubertal child is similar to that of an adult.[24] Children are often referred to pediatric hospitals or specially trained physicians to perform the sexual assault examination.[24] **Consent from the parent of a minor or from the minor depending on state law is needed for the examination. Assent from a child is necessary to complete the examination. Defer the sexual assault examination of a child to a specialist if you do not have experience in this area.[16,24,25] The anatomy and examination of a child are different from that of an adult.[31] Do not restrain an uncooperative or combative child as this can result in significant psychological trauma.** Some children may require an examination under procedural sedation or general anesthesia. This is reserved for those with more significant or internal trauma and performed by a pediatric subspecialty physician.

LABORATORY INVESTIGATIONS

Decisions regarding STI testing should be individualized given the potential for these diagnoses to be accessed later despite limitations in all 50 states on the evidentiary use of prior sexual history. Perform testing based on guidelines created and regularly updated by the Centers for Disease Control and Prevention (CDC).[32] Test serum for hepatitis B, HIV, and syphilis.[32] These are baseline tests and will be repeated in later follow-up testing. Process the chlamydia and gonorrhea tests at the hospital laboratory. **NAATs are the preferred tests for *Chlamydia trachomatis* and *Neisseria gonorrhoeae* regardless of the site. NAAT vaginal testing is the preferred test for *Trichomonas vaginalis*. Perform a wet mount for *T. vaginalis* if there is no access to NAAT swabs.** Perform toxicology screens at the hospital laboratory only if there is a medically necessary reason for the test (e.g., altered mental status). **Individual states have protocols for urine testing at a crime labs to detect "date rape" drugs.**

CHAIN OF EVIDENCE

The work does not stop after the evidence is collected. Each state has different rules for evidence collection. Verify the proper procedure in your current setting. No one should leave or enter the examination room until all evidence is properly tagged and secured. Seal all paper bags and envelopes with evidence tape. Place your signature across the sealed ends of each bag or envelope and the evidence tape. This will make it easier to determine if they have been opened and tampered with. Clearly label each bag or envelope with the patient's name and hospital identification number, the date of collection, the time of collection, the source of the sample, and the printed and signed name of the nurse or assistant. Secure all the evidence in a locked room or cabinet until it can be turned over to a law enforcement officer. Have the officer sign a form stating the officer's name, badge number, the date and time the evidence was transferred, and the name of the physician or nurse releasing the evidence to the officer (Figures 170-1 and 170-9, Step 13). Both individuals must sign this form.

AFTERCARE

Referral to a Social Worker, Psychologist, or Psychiatrist may be necessary, especially if a patient advocate is not available. Treat any injuries in the standard manner. Update the tetanus immune status as necessary. Make a written or electronic summary of the complete history and physical examination findings for the medical record (**Figure 170-9**).[14] Place or upload any photographs taken in the medical record.

Provide all follow-up instructions in writing (**Figure 170-10**). Arrange medical follow-up with a primary care physician in 1 to 2 weeks and again in 2 to 4 months.[33] The follow-up appointment should include an examination for STIs, HIV testing, human papillomavirus immunizations, and hepatitis immunizations.

Give all patients a 24-hour crisis phone number as well as a follow-up in 1 to 2 days with the local rape crisis center if the community has one.[34] The Rape, Abuse, and Incest National Network (RAINN) offers a 24-hour hotline (1-800-656-HOPE) as well as a website (www.rainn.org) with helpful information for the patient.

Arrange, with the help of the advocate or Social Worker, a safe place for the patient to stay. This is especially important if the offender is not in custody. Encourage the patient not to stay by themselves. A patient may need to be hospitalized in rare instances for psychiatric issues from the assault or for the patient's safety. Children may need placement at the discretion of DCFS.

PREGNANCY PROPHYLAXIS

The risk of pregnancy after a sexual assault is estimated to be approximately 5% if the patient is of reproductive age.[35] The American Academy of Pediatrics (AAP), American College of Emergency Physicians (ACEP), and American Congress of Obstetricians and Gynecologists (ACOG) support the provision of emergency contraception to victims of sexual assault.[1,3,36] There are two categories of emergency contraceptives: emergency contraceptive pills and the Copper-T380A intrauterine device (IUD). The only contraindication to emergency contraception is pregnancy. **Emergency contraceptive pills will not harm an existing pregnancy. IUD placement may disrupt a pregnancy or increase the risk of a serious infection and septic abortion.** Educate the patient that emergency contraception does not protect against HIV or STI.

Early emergency contraceptive pills consisted of high doses of common oral contraceptive pills (Yuzpe method).[37] Emergency contraceptives that contain only the progestin component (e.g., levonorgestrel) are better tolerated and more effective than the

FORENSIC LABORATORY REPORT — PAGE 4

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(patient stamp)

Photographs — may be taken for evidentiary purposes with the written consent of the patient or the patient's guardian if the patient is a minor. If the patient is a minor and the parent/guardian is not immediately available, photographs may be taken but shall be released to law enforcement personnel and state's attorneys only with the written consent of the parent/guardian. If consent is refused, all photographs and negatives shall be given, without charge, to the parent/guardian.

SUMMARY OF FINDINGS

SIGNATURES

(Attending Physician Signature) (date)

(Attending Nurse/Assistant Signature) (date)

(Please print)

(Please print)

COMPLETE EVIDENCE COLLECTION STEP 13

FINAL INSTRUCTIONS

1. Make sure all information requested on all sample envelopes and bag labels have been filled out completely.
2. Separate all forms (Steps 1, 2, and 14) and follow distribution requirements on the bottom of each form.
3. With the exception of the large sealed and labeled clothing bags, return all other evidence envelopes/bags to the kit box.
4. Initial and affix red evidence tape on box top.
5. Fill out information required on kit box top.
6. Hand the sealed kit and sealed bags to investigating officer.

NOTE: If officer is not present at this time, place sealed kit and sealed bags in secure area, and hold for pickup by investigating officer.

COMPLETE EVIDENCE COLLECTION STEP 14

ILSP502D

(White copy to hospital; yellow copy in kit)

FIGURE 170-9. Sample form for summary of physical examination findings. Note that the form is signed by the attending physician and the nurse, or assistant, who was present during the examination. (Courtesy of the Illinois State Police.)

Illinois State Police
Division of Forensic Services

STEP 14

Please print, type or use a patient information stamp.

(patient stamp)

PATIENT DISCHARGE MATERIALS

Patient name	DOB/Age
Hospital name/phone	
Date of examination	ER#
Examining physician	

With your consent, a number of specimens were collected from you to provide evidence in court should your attacker be caught and you decide to prosecute. Additional tests were conducted as follows: ☐ A blood test for syphilis, ☐ A culture for gonorrhea, ☐ Tests for chlamydia, ☐ Other tests_____ _____, and ☐ A pregnancy test to determine pre-existing pregnancy only, not to determine a possible pregnancy as a result of the assault.

VENEREAL DISEASE TREATMENT

☐ You were given an antibiotic to prevent gonorrhea. However, you must return in six weeks following this treatment for another test to be sure you do not have syphilis. Return for this test and possible treatment the week of _____ name of drug _____ dosage _____

☐ You were *not* given treatment to prevent gonorrhea or any other venereal disease because _____

☐ If you wish to obtain follow-up testing or treatment for venereal disease, please visit your local health clinic or contact _____ _____ (name, address, and telephone)

☐ Patient declines

PREGNANCY PREVENTION

☐ You were given medication to prevent pregnancy as a result of the sexual assault. If you should become pregnant despite having been given treatment, you should return immediately to the hospital emergency room or go to your private physician because the drug you were given may cause damage to the fetus. Name of drug _____ Dosage_____

☐ You were *not* given medication to prevent pregnancy because _____
If you want counseling, referrals and/or follow-up pregnancy testing information, consult the social service department of this hospital or call one of the agencies listed below for assistance.

Agency _____ Agency _____
Address _____ Address _____
Telephone _____ Telephone _____

☐ Patient declines

FOLLOW-UP

☐ An appointment was made for you at this hospital for follow-up *medical* treatment on (date) _____

☐ No appointment was made for follow-up medical treatment because _____

☐ An appointment was made for you at this hospital for follow-up *counseling* on (date) _____

☐ No appointment was made for follow-up counseling because _____

PSYCHOLOGICAL SUPPORT referral to

Was written and verbal information given to patient? ☐ Yes ☐ No

DOCUMENTATION If the patient is less than 18 years of age, was DCFS notified, if appropriate? ☐ Yes ☐ No
Were police notified? ☐ Yes ☐ No Was Patient Consent and Authorization Form completed? ☐ Yes ☐ No

INFORMATION

I have received the following written information:

☐ Illinois Department of Public Health brochure, "After Sexual Assault" ☐ Attorney General's Office flyer "Illinois Crime Victims Compensation Act"
☐ Patient Medication information sheets listing anticipated effects and side effects of medicines given to me.

☐ This patient information sheet _____
Patient Signature Date

☐ I do not wish to receive this sheet _____
Patient Signature Date

ILSP514

(White copy to patient; yellow copy to hospital)

FIGURE 170-10. Sample patient discharge instruction form. (Courtesy of the Illinois State Police.)

Yuzpe method.[38] Data have shown that a single 1.5 mg dose of levonorgestrel is equally effective up to 5 days after unprotected intercourse.[39,40]

The U.S. Food and Drug Administration has approved the emergency contraceptive pill ulipristal acetate (Ella). Studies have shown pregnancy rates 42% lower than with levonorgestrel when used up to 72 hours after unprotected intercourse and 65% lower when used between 72 and 120 hours after unprotected intercourse.[41,42]

The Copper-T380A IUD (ParaGard) is the most effective form of emergency contraception and can be inserted up to 5 days after unprotected intercourse.[43] The copper IUD is 99% effective in reducing the risk of pregnancy and can be used for up to 10 to 12 years. There is no evidence to support the routine use of prophylactic antibiotics prior to the placement of an IUD.[44]

The ACOG believes that emergency contraception should be provided and immediately available where victims of sexual assault are treated.[36] Emergency contraception should be available in hospitals and facilities where victims of sexual assault at risk of pregnancy are treated.[45] There are hospitals that may still prohibit contraception and practitioners who morally object to it. Promptly refer patients to a center or provider where they can receive the appropriate treatment.

ANTIMICROBIAL PROPHYLAXIS

Postexposure prophylaxis for STI is recommended since compliance with follow-up visits remains poor. The risk of acquiring an STI is relatively high and the side effects from the medications are relatively low. The most common infections diagnosed during sexual assault exams are bacterial vaginosis, chlamydia, gonorrhea, and trichomoniasis.[46] The current CDC recommendation is to provide prophylaxis for chlamydia, gonorrhea, and trichomoniasis. The current CDC treatment regimen consists of 250 mg of intramuscular ceftriaxone, 2 gm of oral metronidazole or oral tinidazole, and 1000 mg of oral azithromycin in one-time single doses.[32] The current guidelines do not recommend prophylaxis for syphilis. The side effects of metronidazole/tinidazole include significant nausea that may interfere or complicate pregnancy prophylaxis. Recent alcohol consumption is a contraindication to immediate oral treatment and can be deferred until 24 or 72 hours after the alcohol. Patients can take the treatment at home. **The most updated guidelines for STI treatment can be found at www.cdc.gov or on their application called "STD Tx (treatment)" available as a download.**

Hepatitis B vaccination, without hepatitis B immunoglobulin, is recommended if the patient has not been previously vaccinated.[32] This is especially important in high-risk exposures (e.g., unprotected penetration, contact with the assailant's blood, or contact with the assailant's bodily fluid). Provide the first dose of the hepatitis B vaccine in the Emergency Department. Boosters are recommended at 1 to 2 months and 4 to 6 months.

Human papillomavirus vaccine is recommended for female victims from age 9 to 26 years old who have not been previously vaccinated. It is recommended for men from age 9 to 21 years or 9 to 26 years for men who have sex with men.[32] Boosters are recommended at 1 to 2 months and at 6 months.

There are no clear guidelines for postexposure prophylaxis (PEP) for HIV.[47] The guidelines are individualized according to factors increasing the risk of HIV infection. These include the site of penetration (i.e., oral is lowest versus vaginal versus rectal being highest), trauma, and the presence of lesions from other infections or ulcers in the assailant or the victim.[48] Discuss the possible benefits of PEP with the patient, noting the specific risks and benefits of HIV prophylaxis. Have a specific discussion regarding the importance of adherence, close follow-up, and early initiation.[32] Document this discussion in the medical record. The current recommendations for PEP include a 28-day course of highly active antiretroviral therapy (HAART). **Consult the CDC website at www.cdc.gov/hiv for specific medication regimens.**

Treatment for the male sexual assault victim is the same except for pregnancy prophylaxis.

COMPLICATIONS

There are no physical complications to performing the sexual assault examination. **Be aware of potential psychological complications.[13] Treat the patient with the utmost privacy and dignity to help them through this stressful situation. Do not further victimize the victim. Do not add to the abuse they have already suffered. Allow the patient to make decisions and have control during the history and physical examination.** The STI medications can cause allergic reactions, disulfiram reactions with alcohol, and gastrointestinal upset.

SUMMARY

The Emergency Department plays a key role in providing quality treatment to the sexual assault victim. Providing compassionate care strongly influences the healing process from being a victim to becoming a survivor. It is important to perform a complete examination for trauma and not just focus on evidence collection. Careful collection and documentation of the elements of the evidence collection kit will impact successful prosecution. Refer to the CDC website for the latest treatment recommendations. Proper follow-up care and a strong link to a local rape crisis center are important.

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171 Culdocentesis

JoAnna Leuck and Jennalee Cizenski

INTRODUCTION

Culdocentesis is a procedure used to sample peritoneal fluid to help confirm a diagnosis or to obtain a culture. It has mainly been used for diagnosing a ruptured ectopic pregnancy or ruptured ovarian cyst.[1-11] Culdocentesis involves introducing a hollow needle through the posterior vaginal cuff and into the peritoneal space.[1-11] This is a relatively simple and fast procedure. **Ultrasound (US) has virtually replaced culdocentesis as the test of choice.** US is minimally invasive, has high sensitivity, has a high specificity, and involves no ionizing radiation. Culdocentesis may still prove to be an important skill for the Emergency Physician to consider using for unstable patients and in low-resource settings that do not have US capabilities.

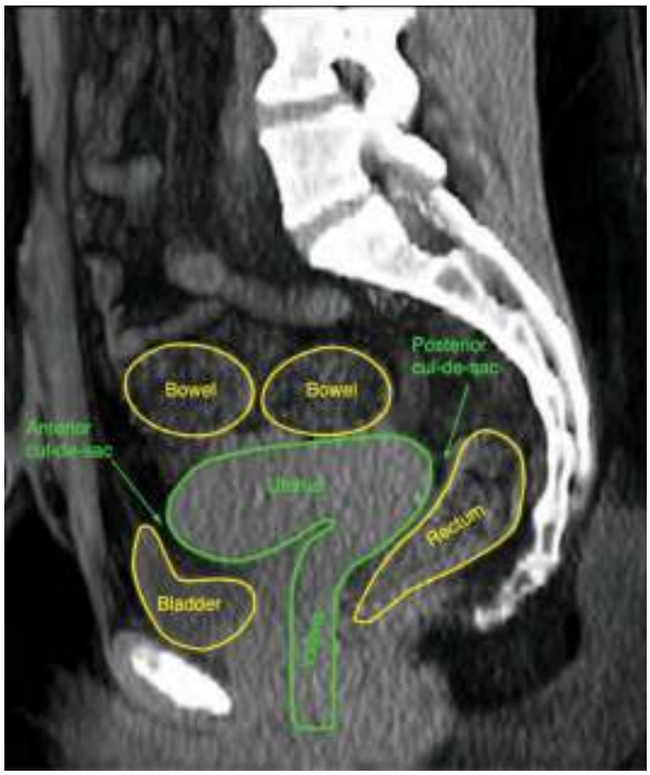
ANATOMY AND PATHOPHYSIOLOGY

The key anatomy to be familiar with is the vagina and the posterior cul-de-sac (i.e., the rectouterine pouch or the pouch of Douglas). The posterior cul-de-sac is formed by reflections of the peritoneum between the posterior surface of the uterus and the anterior surface of the rectum (**Figure 171-1**). It is the most dependent intraperitoneal space in both the upright and supine positions. This allows blood, pus, and other free fluids to pool in this space. The posterior cul-de-sac separates the upper portion of the rectum from the uterus and the upper portion of the vagina. The small intestine and a small amount of peritoneal fluid often lie within the posterior cul-de-sac. The sensory innervation of the vagina is greatest near the introitus. There is minimal sensory innervation in the posterior vaginal fornix adjacent to the posterior cul-de-sac.

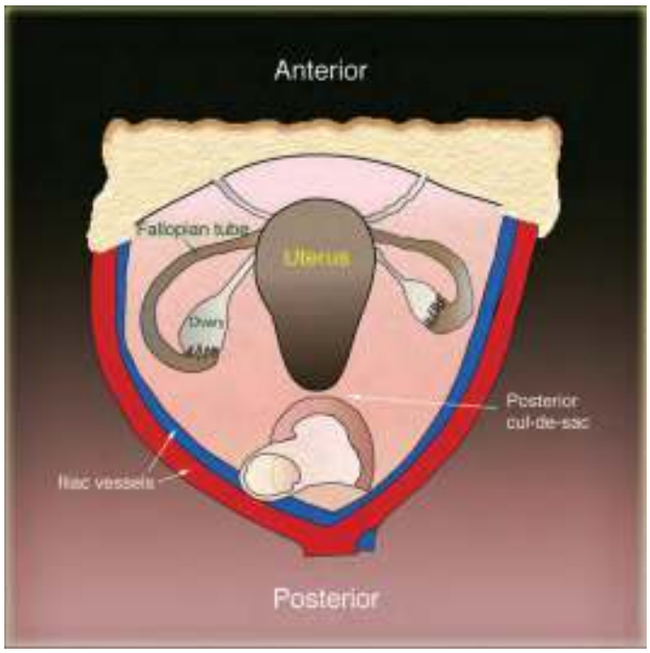
INDICATIONS

Culdocentesis has been used in the Emergency Department in the past to diagnose a ruptured viscus, particularly an ectopic pregnancy. The use of culdocentesis has decreased significantly with the emergence of improved serum and urine tests for pregnancy, increased accessibility to US, and increased resolution of US. Recent studies have clearly shown US to be more sensitive and noninvasive in detecting a hemoperitoneum.[1]

The primary indication for this procedure is a hemodynamically unstable female patient of reproductive age with evidence of peritoneal irritation in the pelvic region when bedside US is not available.



A



B

FIGURE 171-1. Anatomy of the female pelvis. ***A.*** Midline sagittal view. ***B.*** View from above inside the pelvis.

This patient most likely has a ruptured ectopic pregnancy and needs emergent surgery. A diagnostic test is usually not necessary to take the patient directly to the Operating Room if a rapid pregnancy test is positive. **An unstable patient cannot be sent to the Radiology Department for an US if there is any diagnostic uncertainty.** A culdocentesis may be performed if bedside US is not available. Approximately 85% to 90% of patients with ruptured ectopic pregnancies have a positive culdocentesis.[2]

Culdocentesis could also be considered in a stable pregnant patient with US evidence of free fluid in the pelvis or posterior cul-de-sac. A culdocentesis can confirm if the fluid is blood. Approximately 65% to 70% of patients who have a stable presentation and unruptured ectopic pregnancy have a positive culdocentesis.

A culdocentesis is indicated if US or a laparoscopy is not readily available. A negative culdocentesis may be used to reassure the Emergency Physician that following serial quantitative beta-human chorionic gonadotropin levels can be performed before committing a stable patient to an operative procedure. A positive culdocentesis would indicate intraabdominal bleeding that requires immediate operative intervention.[3]

Other indications for a culdocentesis include the evaluation of ascites, blunt abdominal trauma, or patients with pelvic inflammatory disease. It can be performed to obtain ascitic fluid to evaluate for infection, malignancy, and/or type of fluid (i.e., an exudate or transudate). Culdocentesis has been used in place of a diagnostic peritoneal lavage (Chapter 84) to detect hemoperitoneum in blunt abdominal trauma because small amounts of blood tend to collect in the posterior cul-de-sac. Aspiration of clear peritoneal fluid makes significant hemoperitoneum very unlikely. US and computed tomography (CT) scanning have significantly reduced the usefulness of culdocentesis for this indication. Intraperitoneal fluid from patients with pelvic inflammatory disease can be cultured to guide treatment, especially in treatment-resistant cases.

CONTRAINDICATIONS

There are a few contraindications to performing a culdocentesis. An US is the preferred diagnostic method to confirm intraperitoneal bleeding if it is available in a timely manner. A pelvic mass detected on bimanual pelvic examination is a contraindication. A pelvic mass may be a tubo-ovarian abscess, an appendiceal abscess, an ovarian mass, or a pelvic kidney. There is a concern of rupturing an abscess into the peritoneal cavity with the culdocentesis needle. Other contraindications include a nonmobile uterus and patients with a coagulopathy. The procedure is limited to patients beyond puberty on the basis of anatomy. It is difficult to perform the procedure through a small prepubertal vagina. **A patient with unstable vital signs and a positive bedside pregnancy test should be immediately taken to the Operating Room and does not require a culdocentesis. They will require fluid and blood resuscitation until the surgical team and Operating Room are available.**

Culdocentesis may be unsatisfactory in women with previous salpingitis and pelvic peritonitis because the posterior cul-de-sac may have been obliterated. **Failure to obtain blood does not exclude the diagnosis of a hemoperitoneum. The lack of blood on culdocentesis cannot exclude an ectopic pregnancy as the false-negative rates can be as high as 15%.**[4,5]

EQUIPMENT

- Exam table with stirrups
- Water-soluble lubricant
- Vaginal speculum
- Cervical tenaculum
- 19 gauge butterfly needle or 18 gauge spinal needle
- 25 and 27 gauge needles
- Ring forceps
- 20 mL aspirating syringe
- Povidone iodine or chlorhexidine solution

FIGURE 171-2. The sizes of the ER-SPEC. (Photo courtesy of OBP Medical.)

- Sterile water
- Cotton balls
- 4×4 gauze squares
- Local anesthetic solution with epinephrine
- 4% cocaine (optional)
- 20% benzocaine (optional)
- Red top test tube for laboratory analysis
- Purple top test tube for laboratory analysis
- Culture tubes
- Light source

The ER-SPEC is stocked and used in some Emergency Departments (OBP Medical, Lawrence, MA). The single-use speculum is available in three sizes and comes with a built-in light source (**Figure 171-2**). The speculums are available in all three sizes as sterile or nonsterile models. The light source works for 30 minutes and produces no heat. The lack of a cord to the light source or an overhead light is advantageous. No sterilization is needed as with metal speculums which reduces the risk of cross-contamination.

PATIENT PREPARATION

Explain the risks, benefits, potential complications, and alternatives (e.g., US or immediate laparoscopy) to culdocentesis to the patient and/or their representative. Obtain a signed consent for the procedure. Some Emergency Physicians omit the written informed consent for verbal consent. A statement in the chart should state the following: "The risks, benefits, alternatives, and complications were described and discussed in detail. The patient has a clear understanding of these and all questions were answered." A witness should be noted in the record.

Perform a bimanual pelvic examination to rule out a fixed pelvic mass and to assess the position of the uterus prior to the culdocentesis. Place the patient in the lithotomy position with the head of the table slightly elevated in reverse Trendelenburg position so that the intraperitoneal fluid gravitates into the posterior cul-de-sac. Place the patient's feet in stirrups. Procedural sedation (Chapter 159) or premedication with intravenous narcotics or sedatives is recommended and may make the procedure more tolerable for the patient. Premedication is not required in an unstable patient.

Some Emergency Physicians obtain stat upright plain radiographs in stable patients prior to the procedure. These are performed to assess for a pneumoperitoneum. This will help in determining if an iatrogenic pneumoperitoneum was secondary to the procedure upon obtaining postprocedural radiographs.

TECHNIQUE

Insert a lubricated speculum as deep into the vagina as possible without causing the patient discomfort. Open the speculum widely so the blades are above and below the cervix. Adjust the light if using an overhead light source. Grasp the posterior lip of the cervix with a toothed tenaculum or ring forceps (**Figure 171-3*A***). **Forewarn the patient that grasping of the cervix with a tenaculum may be painful.** Elevate the cervix to lift the uterus and to stabilize the posterior wall of the uterus during the needle puncture. This causes a tightening of the vaginal wall adjacent to the posterior cul-de-sac to expose the puncture site and keeps it from moving away from the needle as the vaginal wall is punctured.

Prepare the area. Swab any secretions out of the vagina. Swab the vaginal wall in the area of the posterior cul-de-sac with povidone iodine or chlorhexidine followed by sterile water. Some Emergency Physicians may optionally choose to topically anesthetize the area with a cotton ball soaked in 4% cocaine or 20% benzocaine prior to infiltrating with local anesthetic solution. Insert the cocaine- or benzocaine-soaked cotton ball into the posterior vaginal fornix area

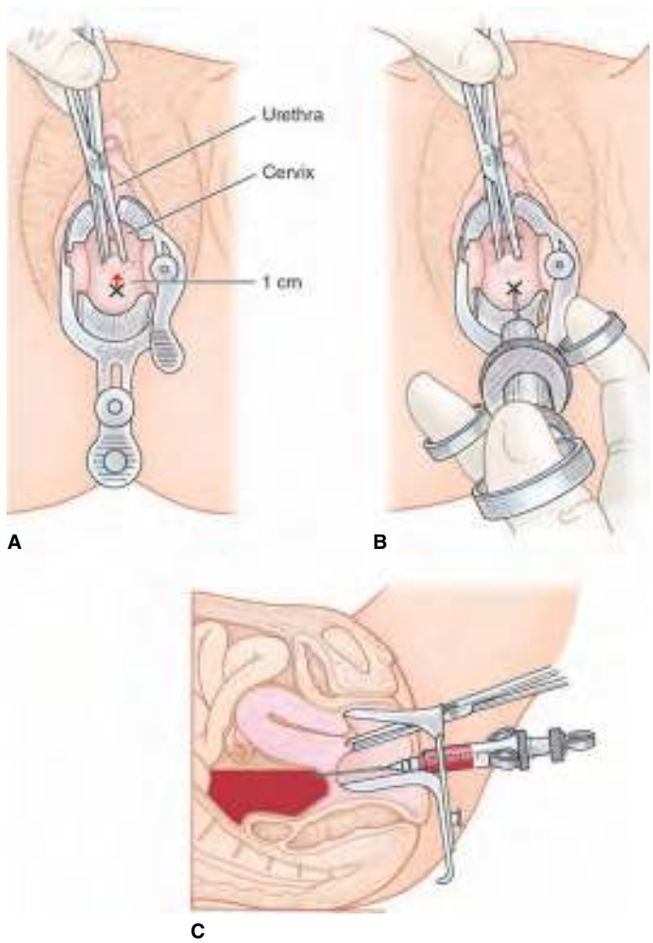


FIGURE 171-3. Culdocentesis. A speculum has been inserted in the vagina and opened. ***A.*** Grasp the posterior lip of the cervix with a toothed tenaculum and elevate it. The "X" marks a spot 1 cm (red arrow) from the junction of the cervix and posterior vaginal wall where the needle will be inserted. ***B.*** The needles used for anesthesia and culdocentesis are inserted. ***C.*** Midsagittal section demonstrating the needle is inserted and aimed slightly posterior to access the fluid.

and allow it to remain for 3 minutes. The maximum dose of cocaine to apply to the cotton ball is 3 mg/kg.

Arm a 5 mL syringe containing local anesthetic solution with epinephrine with a 25 gauge needle. Insert the needle in the midline and 1.0 cm posterior (i.e., inferior) to the point at which the posterior vaginal wall joins the cervix (**Figure 171-3*B***). Inject 2 mL of local anesthetic solution containing epinephrine into the tissues of the vaginal wall.

Fill a 20 mL aspirating syringe with 2 to 3 mL of sterile saline. Local anesthetic solution can be used instead of saline but it is bacteriostatic and any aspirated fluid cannot be cultured. Attach an 18 gauge spinal needle to the 20 mL syringe. Insert the spinal needle parallel to the lower blade of the speculum (**Figure 171-3*B***). Penetrate the vaginal wall in the midline and 1.0 cm posterior (i.e., inferior) to the point at which the vaginal wall joins the cervix (**Figures 171-3*B* and 171-3*C***). Advance the needle 2.0 to 2.5 cm.[4,6] **Slowly inject 0.5 to 1.0 mL of saline.** It should flow freely and without resistance if the needle tip is within the posterior cul-de-sac (**Figure 171-3*C***). **It may be within the wall of the uterus or intestines if there is resistance to saline injection.** Withdraw and redirect the tip of the needle until the saline flows freely upon injection. Aspirate any pelvic fluid and remove the needle. Apply negative pressure to the syringe while slowly withdrawing the needle. **It is important to avoid aspirating any blood that has accumulated in the vagina from previous needle punctures or cervical bleeding**.

Fluid may not be aspirated. Withdraw the needle and reintroduce it slightly to the left or right of the midline. Avoid directing the needle too far laterally. This can result in puncture of a mesenteric or pelvic vessel. Always repeat the procedure once if no fluid is obtained on the first attempt.

Place some of the peritoneal fluid into both a red top and purple top test tube. Place some of the fluid into aerobic and anaerobic culture tubes. Observe the fluid for clotting. Transport the samples to the laboratory for Gram stain, culture, cell count, and hematocrit.

ALTERNATIVE TECHNIQUE

Slight alterations in the technique described above have been used successfully. Longitudinal traction on the cervix instead of grasping and elevating the posterior lip of the cervix is sometimes used to elevate the uterus. A 19 gauge butterfly needle held with ring forceps can be used instead of the spinal needle (**Figure 171-4**).

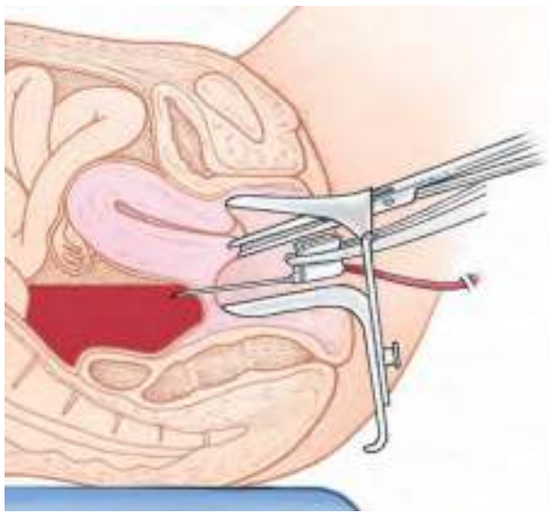


FIGURE 171-4. The alternative technique of using a butterfly needle and a ring forceps to perform a culdocentesis.

This allows for good control of the needle during the puncture and also offers a built-in guide for needle depth. An assistant is required for this alternative technique to aspirate the tubing while the Emergency Physician positions and withdraws the butterfly needle.

ASSESSMENT

A normal culdocentesis when there is no pathology should yield 2 to 4 mL of clear-to-yellowish peritoneal fluid. There is no diagnostic value when there is no return of fluid of any type, also known as a dry tap. The culdocentesis is considered nondiagnostic if less than 2 mL of clotting blood is obtained. This may have come from a puncture site on the vaginal wall.

A hemoperitoneum is signified by the aspiration of more than 2 mL of nonclotting blood. It has been suggested that as little as 0.3 mL of nonclotting blood equates with a positive tap.[7] The finding of nonclotting blood with a hematocrit greater than 15% has been 70% to 97% predictive of a significant bleeding source (e.g., an ectopic pregnancy).[3] Approximately 70% to 83% of ectopic pregnancies will have nonclotting blood on culdocentesis. Approximately 10% to 20% of ectopic pregnancies will have a negative or nondiagnostic culdocentesis. False positives will occur in 2% of cases.[8]

Culdocentesis is less sensitive with unruptured ectopic pregnancies. It is estimated to be 70% positive with an unruptured ectopic pregnancy versus 85% to 90% positive in a ruptured ectopic pregnancy.[9] A quantity of blood greater than 2 mL has no further significance as it may be related to needle position or rate of bleeding. Blood remains nonclotted for days in the syringe due to defibrination activity of the peritoneum. A ruptured ectopic pregnancy, a hemorrhagic ovarian cyst, or a ruptured spleen can all result in a hemoperitoneum. Peritoneal bleeding usually corresponds to a hematocrit of greater than 10%. One study showed that 97% of cases with an ectopic pregnancy had a hematocrit of at least 15%.[10]

A positive culdocentesis with a positive pregnancy test is not always an ectopic pregnancy. A ruptured corpus luteum cyst is the most common cause of a false-positive result. The false-positive rate is estimated at 9%.

A culdocentesis is considered negative when the aspirated fluid is pus, cystic, or straw-colored and performed for the purpose of diagnosing an ectopic pregnancy. Greater than 10 mL of clear fluid most likely indicates a ruptured ovarian cyst, aspiration of an intact corpus luteal cyst, ascites, or possibly carcinoma. **Obtaining greater than 10 mL of clear fluid does not automatically rule out an ectopic pregnancy since it may coexist with other pathology.[2]**

US was far superior in recent studies comparing US versus culdocentesis.[1] The sensitivity and specificity of echogenic fluid for establishing a hemoperitoneum were 100% and 100%, respectively, for an US compared with 66% and 80%, respectively, for a culdocentesis. The negative predictive value of a nondiagnostic culdocentesis was 25% compared to 100% for echogenic fluid seen on US in the ectopic pregnancy subgroup of patients.

AFTERCARE

Aftercare is limited to local wound care of the puncture site. Proper postsedation monitoring and precautions are necessary. Bleeding from the tenaculum puncture sites on the cervix and/or the vaginal wall needle puncture site is usually self-limited. Apply manual pressure with a gauze square to provide hemostasis. Obtain an upright plain radiograph to rule out an iatrogenic pneumoperitoneum from the procedure. Compare this film to any preprocedural films.

COMPLICATIONS

Complications associated with performing a culdocentesis are rare. The most common and serious complication reported includes the rupture of an unsuspected tubo-ovarian abscess.[4] Other complications include puncture of the gravid uterus, bowel perforation, perforation of a pelvic kidney, and bleeding from the puncture site in patients with bleeding disorders. Bowel and uterine wall punctures usually do not result in serious morbidity. Most complications are eliminated by a careful bimanual examination to detect a pelvic mass prior to the procedure.

SUMMARY

Culdocentesis can be very useful in evaluating for a ruptured ectopic pregnancy. It is easy to perform, rapid, and relatively safe. Its utility has decreased as the test of choice with the improved capability and availability of US. It is indicated when US is not available or in unstable patients where bedside US is unavailable.

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172 Prolapsed Uterus Reduction

Andrea Dreyfuss and Eric R. Snoey

INTRODUCTION

Symptomatically important uterine prolapse can result from relaxation of pelvic support with age in susceptible women. Uterine prolapse can significantly impact a woman's daily activities, sexual function, ability to exercise, and body image. Population-based studies note that approximately 10% of women report symptoms of pelvic organ prolapse.[1] Approximately 14% of 16,616 participants possessing a uterus were found to have uterine prolapse in the Women's Health Initiative Hormone Replacement Therapy Clinical Trial.[2] Pelvic organ prolapse was the etiology for 15% to 18% of hysterectomies.[3] The most common etiology in postmenopausal women for a hysterectomy is uterovaginal prolapse.[3] Manual reduction of the prolapsed uterus and placement of a pessary represent a safe and temporizing measure that may be performed in the Emergency Department. Surgical correction may ultimately be necessary. It is estimated that pelvic organ prolapse is responsible for more than 200,000 surgical repair procedures each year (22.7 per 10,000 women). This chapter will address the nonsurgical management of a prolapsed uterus.[2,4-7]

ANATOMY AND PATHOPHYSIOLOGY

The structural support of the female pelvis is subject to a number of identifiable stresses that may predispose certain women to uterine prolapse later in life. Multiparity is the most commonly shared trait, suggesting that birth trauma has a primary role to play. Alternative mechanisms relate to increased intraabdominal pressure (e.g., heavy lifting, ascites, obesity, large intraabdominal tumors, chronic constipation, or pelvic tumors). A congenital form of uterine prolapse seen in newborns has been attributed to vigorous crying.[8] Two cases of acute uterine prolapse after restrained motor vehicle collisions were described in 1997.[9] It was hypothesized that the sudden increase in intraabdominal pressure from the lap belt was the cause of the prolapse.[9] Chronic respiratory disorders (e.g., asthma, bronchitis, or emphysema) may put undue tension on the pelvic floor musculature and contribute to the increased risk of prolapse.[10] The integrity of the pelvic connective tissues may have a role as suggested by the increased incidence of uterine prolapse in women with Marfan syndrome and other connective tissue disorders.[11]

Uterine prolapse is defined as the descent of the uterus and cervix down the vaginal canal toward the vaginal introitus. All forms of uterine prolapse are described in reference to the hymen. The uterine displacement is typically graded on a scale of 0 to 4, with 0 referring to no prolapse, 1 halfway to the hymen, 2 at the hymen, 3 halfway out of the hymen, and 4 referring to total prolapse (**Figure 172-1**).[2,12] A first-degree or mild prolapse is defined with the cervix palpable as a firm mass in the lower third of the vagina. Patients with grades 0 or 1 prolapse are usually asymptomatic. Grade 3, or moderate prolapse, is characterized by the cervix being visible and projecting into or through the vaginal introitus. The patient may experience a falling-out sensation or may report the feeling of sitting on a ball.[12] Additional symptoms include heaviness in the pelvis, low backache, lower abdominal discomfort, and inguinal discomfort. Grade 4, also known as severe prolapse or procidentia, involves the cervix and entire uterus projecting through the introitus, completely inverting the vaginal vault (**Figure 172-2**).[13] The uterine mass frequently has one or more areas of easily bleeding atrophic lesions secondary to exposure and local pressure effects. It may result in leukorrhea, abnormal uterine bleeding, or spontaneous abortions.[7]

The pelvic diaphragm, the endopelvic fascia, and the vagina provide the primary support for the pelvic organs. The superficial muscles of the perineum lie below the pelvic diaphragm and indirectly support the pelvic organs by inserting centrally into the perineal body. The perineal body serves to fix the distal vagina and anus. The bony pelvis is the ultimate support for all the soft tissues of the pelvis.[11]

The pelvic diaphragm is made up of a bilaterally paired group of striated muscles (i.e., the pubococcygeus and puborectalis, the iliococcygeus, and the coccygeus). The pelvic diaphragm is innervated by fibers originating from sacral segments 4 and 5. The pelvic diaphragm is normally in a state of tonic contraction. It increases its tone reflexively in response to increases in intraabdominal pressure.[11]

The endopelvic fascia is the layer of fibrous connective tissue that envelops all pelvic organs. The endopelvic fascia develops supportive

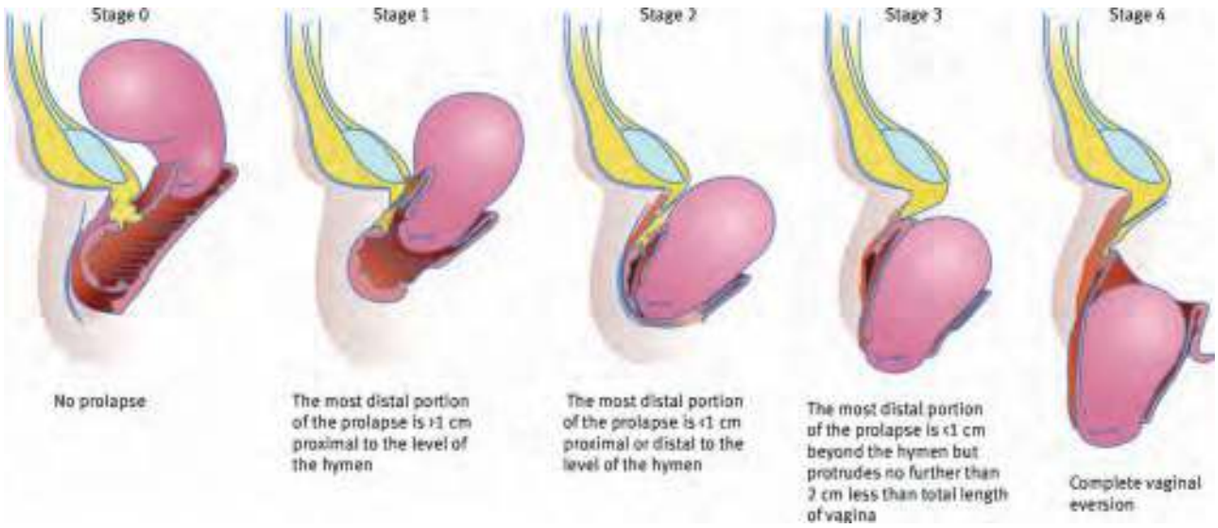

FIGURE 172-1. Stages of uterine prolapse. (Used with permission from reference 12.)

thickenings along lines of tension that are referred to as ligaments (i.e., uterosacral and cardinal ligament complex). These ligaments provide further support for the pelvic structures. The uterosacral and cardinal ligaments suspend the uterus and are simple extensions of the endopelvic fascia.[11]

The vagina has supportive functions. The uterosacral and cardinal ligaments shift the upper one-third of the vagina posteriorly and laterally. This provides support for the cervix. The middle third of the vagina is attached bilaterally to the fascia overlying the pelvic sidewall musculature and supports the bladder. The lower third of the vagina is fused to the tissues about the vaginal outlet.

There are numerous contributing factors in the development of a prolapsed uterus.[12] Prolonged labor can lead to significant pelvic floor damage suggesting that women who delivered vaginally or who later required cesarean section because of failure to progress are more at risk.[11] No clinical indicator has been identified as of yet that allows reliable prediction of pelvic floor dysfunction in apparently normal individuals following vaginal delivery.[11] The quantity and quality of collagen appear to deteriorate in women after menopause, likely attributable to the resultant estrogen deficiency.[14,15] It is thought that defective connective tissue contributes significantly to uterine prolapse in the absence of other risk factors. One study of premenopausal women with uterine prolapse found a 25% reduction in the collagen content of tissues.[15] Conditions that affect spinal cord pathways and pelvic nerve roots (e.g., muscular dystrophy, trauma, myelodysplasia, meningomyelocele) can result in paralysis of the pelvic floor with the subsequent prolapse of pelvic structures. Spina bifida has been associated with the majority of cases of uterine prolapse in newborn girls and nulliparous women.[11]

Increased intraabdominal pressure is a major factor in the development of pelvic organ prolapse. Obesity contributes to pelvic prolapse by increasing intraabdominal pressure that is transmitted to the pelvic organs. Chronic respiratory conditions that are associated with forceful coughing are also thought to predispose women to uterine prolapse. Occupational and recreational activities (e.g., heavy lifting or repeated jumping) result in repeated and prolonged increases in intraabdominal pressure. A review of 1.6 million women found an increased risk of uterine prolapse in a group of nurses whose jobs required heavy lifting.[11] The deterioration in muscle function and connective tissue associated with aging together with gravity, childbirth, neurologic deterioration, and hormonal status all combine to lead to a progressive decline in pelvic floor function. This may be compounded by factors that increase intraabdominal pressure leading to the development of symptomatic pelvic floor prolapse.[14]

All aspects of the vaginal support should be carefully surveyed when examining a patient with a uterine prolapse. Look for the presence of a cystocele or rectocele. Examine the patient in the standing position and the dorsal lithotomy position. The prolapse is almost invariably worse when the patient is upright.[14] It may be necessary to have the patient strain to assess the full extent of the prolapse.[15] The examination may be facilitated by having the patient stand with one foot on a well-supported stool. Some degree of a cystocele, urethrocele, enterocele, or rectocele may develop or may be associated with a uterine prolapse as the uterus progressively descends. Some authors recommend expanding the traditional speculum examination by using a single blade speculum (e.g., Sims speculum) or a bivalve speculum to selectively visualize each portion of the vaginal vault for defects in pelvic support. Perform a rectovaginal examination with the patient in the standing position to detect an occult enterocele suggested by the presence of small bowel easily palpable in the cul-de-sac using the thumb and forefinger.[16,17]

FIGURE 172-2. Uterine procidentia. (From Cunningham GF et al: *Williams Obstetrics*, 23rd ed. New York: McGraw-Hill; 2010. Used with permission from Dr. Charles P. Read.)

Asymptomatic uterine prolapse requires no treatment in the Emergency Department.[12] The patient with only intermittent symptoms can prevent against prolapse by simply inserting a tampon or a diaphragm. This additional support may be used in anticipation of increased activity or prolonged periods of standing. Inform the patient that she is losing some aspects of pelvic support and should be referred to a Gynecologist for evaluation of future treatment options.[8] Lifestyle changes can prevent the progression or recurrence of prolapse and preclude the need for future surgical evaluation and complications. Most of these changes focus on limiting unnecessary increases in intraabdominal pressure (e.g., losing weight, removing girdles, avoiding heavy lifting, stopping smoking, treating allergies, and treating pulmonary disease).[5] Kegel proposed a series of pelvic muscle exercises for the treatment of urinary incontinence that can accompany uterine prolapse. These exercises strengthen the pelvic muscles and may be helpful early. Kegel exercises were associated with improvement of symptoms in a meta-analysis including over 2300 women with uterine prolapse. They are of limited value in patients with significant prolapse since fascial attachments have already been disrupted.[8,18,19]

Symptomatic women can be treated conservatively or surgically.[12] Pelvic organ prolapse is frequently not recognized until advanced disease is present. Prolapse does not become symptomatic until the descending segment is at or through the introitus.[20] Therapeutic options are variable and relate to the age and health of the patient, the severity of the symptoms, and the degree of prolapse. The options for uterine prolapse include observation, reduction with or without supportive pessary therapy, vaginal hysterectomy with corrective therapy for pelvic relaxation (i.e., hysteropexy), and some form of colpocleisis (i.e., surgical obliteration of the vagina).[21]

INDICATIONS

Conservative management of symptomatic uterine prolapse in the Emergency Department involves reduction and subsequent fitting of the patient with a pessary. The pessary is largely used as an alternative to surgery or as a temporizing measure in patients awaiting surgery.[22] Preoperative use of a pessary in advanced degrees of prolapse may aid in decongesting the mucosa, improving circulation, and reestablishing vaginal tonicity. Patients desiring surgical management may be treated conservatively in the Emergency Department and referred to a Gynecologist. Symptomatic women who wish to complete childbearing before having a surgical repair may be candidates for a vaginal pessary.[5] The gynecologic and obstetric indications for pessary support after reduction of the prolapsed uterus are listed in **Table 172-1**.[10]

TABLE 172-1 The Indications for the Placement of a Pessary[10]

Gynecologic Indications
Aid in the healing of cervical stasis ulceration associated with uterine prolapse
Nonsurgical management of uterine prolapse
Reduction of a cystocele, enterocele, or rectocele
To alleviate the complications of free uterine retroposition and adnexal prolapse
To control stress urinary incontinence
To determine if future hysteropexy will relieve backache associated with uterine malposition
To facilitate hysteropexy by holding the uterus in position for operation
To reduce infertility due to cervical retroposition
Obstetric Indications
To avoid threatened abortion due to uterine retroposition and chronic passive congestion
To prevent postpartum subinvolution or retroversion of the uterus
To prevent and protect against abortion in cervical incompetence
To relieve urinary retention and/or pain that can accompany uterine retroposition in pregnancy

CONTRAINDICATIONS

There are no absolute contraindications to the manual reduction of a prolapsed uterus. Pessaries are contraindicated in patients with acute genital tract infections and in those with adherent retroposition of the uterus.[10] Any vaginal inflammation, ulceration, infection, or atrophy should be treated before pessary fitting to decrease the likelihood of vaginal erosions and granulation tissue formation. These latter conditions can be mitigated by oral or topical estrogen supplementation. The use of large amounts of lubricants (e.g., K-Y Jelly) should be encouraged with the pessary in the event that estrogen is contraindicated.[22] Other contraindications include impaired mental capacity, lack of patient dexterity, a history of noncompliance, severe atrophic mucosal changes, exposed foreign body (e.g., mesh), or sexually active women unable to remove and reinsert the pessary.

EQUIPMENT

- Pessaries (**Figures 172-3 and 172-4**)
- Standard pelvic bed
- Vaginal speculum
- Water-soluble lubricant
- Culture swabs
- Biopsy and Pap smear brush, optional
- Uterine forceps
- Sterile gloves
- Procedural sedation supplies and medications (Chapter 159), as needed

FIGURE 172-3. Examples of different types of pessaries. (Photo courtesy of Cooper Surgical, Inc., Trumbull, CT.)

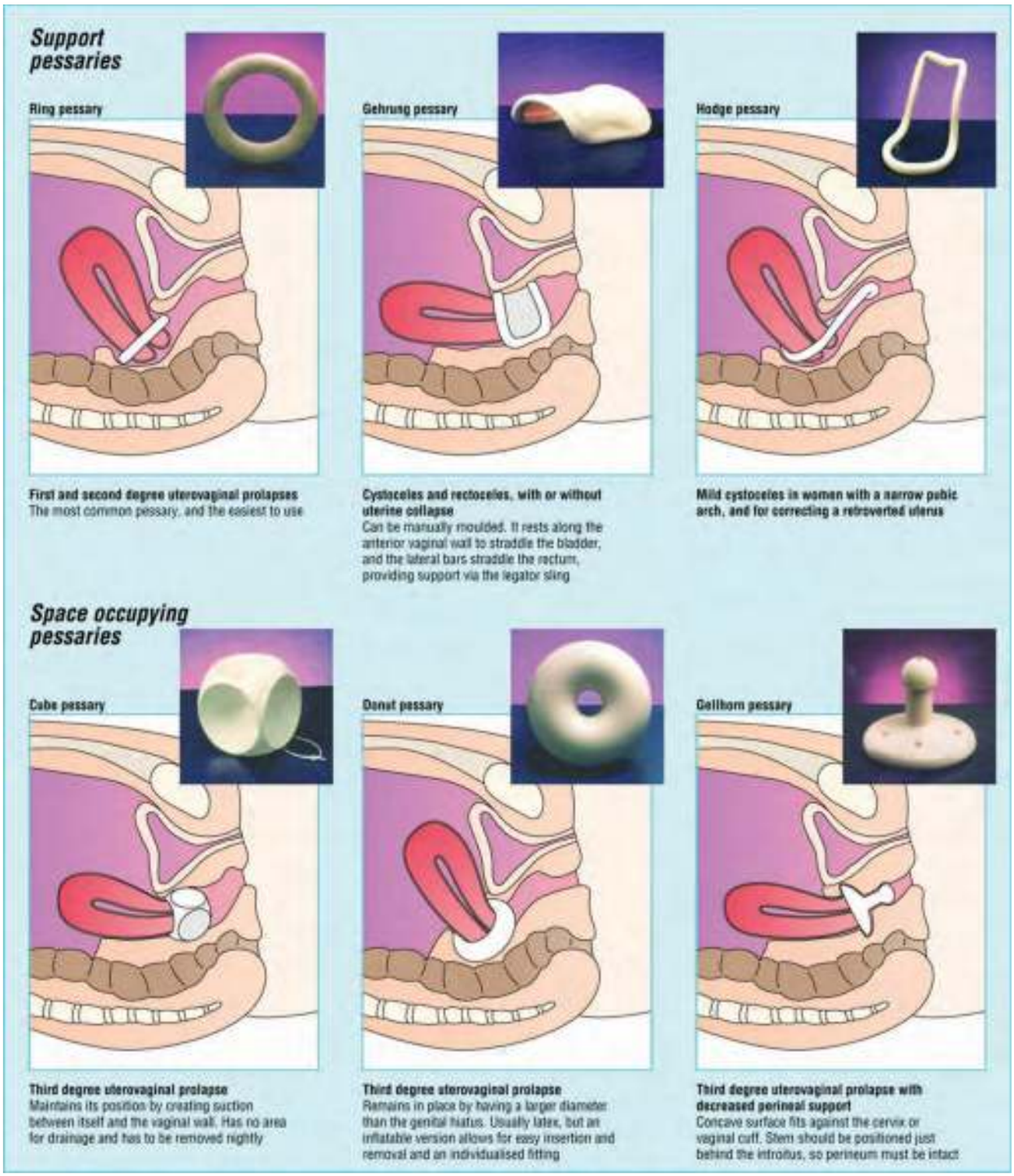

FIGURE 172-4. The use of different types of pessaries. (Photo courtesy of Cooper Surgical, Inc., Trumbull, CT.)

The vaginal pessary is of ancient lineage. They are now made of plastic, rubber, or silicone. The silicone variety has the advantage of being flexible and pliable while causing less vaginal discharge and odor than rubber.[21] Pessaries are available in numerous sizes and shapes (**Figures 172-3 and 172-4**). A well-placed pessary supports prolapsing structures against the perineal body and pubic bone (e.g., the uterus, cervical stump, or hernias of the pelvic floor).[10] The pessary is inserted into the vagina to reduce prolapsed tissue inside the vagina, provide support to pelvic structures, and relieve pressure on adjacent structures (e.g., the bladder and bowel).[23] They elevate the vagina and maintain normal anatomic relations thereby reducing vaginal relaxation and increasing the tautness of the pelvic floor structures. Usually all that is needed is adequate support anteriorly and a reasonably good perineal body. Otherwise, the pessary may slip from behind the pubic symphysis and extrude from the vagina.[10] Patients who are sexually active are unable to remove certain pessaries (e.g., Gellhorn and the donut). The education of Emergency Physicians is somewhat compromised in the use of pessaries.

Placement of a pessary is often a trial and error experience. Review the accompanying pessary brochures to obtain the most satisfactory results from the pessary.[21]

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Place the patient in the lithotomy position with both feet comfortably resting in stirrups. An assistant should be available to assist with positioning.

TECHNIQUES

The management of complete uterine prolapse in the Emergency Department depends upon successful replacement of the uterine fundus into the pelvis and subsequent fitting of the appropriate pessary. Refer the patient for surgical management upon discharge from the Emergency Department. An incarcerated uterus in the pregnant patient is an obstetrical emergency requiring immediate surgical elevation. Other obstetric emergencies include any pregnant patient who develops acute urinary retention or is at risk of aborting.[10]

UTERINE REDUCTION

Insert one gloved and lubricated hand into the vagina with the fingers extended to identify the margins of the cervix. Allow the uterine corpus to rest in the palm of the hand. Apply firm and gentle pressure to elevate the uterine fundus with the gloved hand by pressing against the cervix. Use the fingers on the edges of the uterus closest to the cervix to gently manipulate the uterus in the direction of the umbilicus. Gradually replace the uterus into the pelvis.

If this maneuver is not successful, insert a pessary into the posterior vaginal fornix while the patient is in the lithotomy position. Instruct the patient to sit up and assume the knee-chest position after insertion of the pessary. Instruct the patient to slip slowly into the prone position and then into the lithotomy position. The pessary will maintain the uterus in anteposition.

It may be necessary to pack the vagina following manual reduction of a procidentia to maintain the uterine position as a preoperative management decision of an ulcerated or infected prolapse.[10,24] This should be performed in consultation with a Gynecologist.

FITTING OF THE PESSARY

Measure the distance from the introitus to the posterior vaginal vault using uterine forceps after reduction of the uterus. This measurement minus 1 cm is the approximate length of the pessary. If an ovoid rather than a round pessary is required, place the forceps into the introitus to about the level of the cervix and separate the handles until the blades touch the walls of the vagina. This represents the greatest diameter or approximate width of the pessary.[10]

Liberally lubricate the pessary. Insert it with its widest dimension in the oblique diameter of the vagina to avoid painful distension of the introitus. Use the fingers of the nondominant hand to support the perineum. Slip the posterior bar behind the cervix using a finger on the dominant hand. The forefinger should pass easily between the sides of the frame and the vaginal wall at any point to ensure adequate fitting. The pessary is too large if this is not possible.[10]

ASSESSMENT

Ask the patient initially to walk around the examination room to confirm comfort and to exclude renewed prolapse or any expulsion of the pessary. Replace the pessary with one of a different size or style to prevent any further prolapse.

AFTERCARE

Instruct the patient to remove the pessary each night and clean it with warm soapy water during the first few weeks of use. The soap should not contain any deodorants, detergents, or perfumes. These agents can irritate the vaginal mucosa and result in a chemical vaginitis. Weekly cleanings and overnight removal are adequate after the patient has become comfortable with its use. Apply vaginal cream once a week if the patient is not receiving systemic supplemental estrogen replacement. Warm, low-pressure acetic acid douches or acidic vaginal creams (e.g., Aci-Jel) may relieve irritation and prevent any discharge caused by the vaginal pessary.[10]

Refer the patient to a Gynecologist 1 to 2 weeks after the procedure. Routine follow-up at 1 to 2 months and every 12 months thereafter is recommended after several weeks of a well-managed and appropriately sized pessary. Additional care of these devices includes biannual pelvic examinations and replacement of pessaries every 12 to 18 months.[5] Instruct patients on the hazards of leaving the pessary in for prolonged periods of time and the importance of complying with recommended follow-up. It is not uncommon to have to change the pessary size and/or type. Patients are sometimes unable to manage the responsibilities of a pessary. A family member or a nurse trained in the insertion, removal, and care of the pessary is an appropriate alternative.

Advise all patients to return to the Emergency Department for difficulty urinating or defecating, signs of infection, or any problems with insertion and removal of the pessary.

COMPLICATIONS

Attempts at reducing the symptomatic uterine prolapse may be unsuccessful. Review the surgical and medical options with the patient and arrange the appropriate follow-up. Consult a Gynecologist before the patient is discharged from the Emergency Department.

A loose pessary is ineffective and usually will be expelled. The patient may experience symptoms if the pessary is too large or not removed and cleaned periodically (e.g., pelvic pain, vaginal bleeding, vaginal ulcers, urinary retention, urinary fistulas, bowel fistulas, vaginal discharge, and/or dyspareunia). Proper initial fitting and appropriate follow-up are important to mitigate these issues.[21]

Minor complications include ulceration and abrasions of the vaginal mucosa secondary to local pressure effects. Pessaries act as foreign bodies and if colonized by bacteria can lead to a vaginitis. Many patients experience a physiologic watery discharge with pessary use. This should not be confused with an infectious process that usually is accompanied by itching, burning, or odor. Severe vaginitis is more common in the elderly and debilitated patients because of the inability to remove and cleanse the pessary.[25,26] Treat these conditions with discontinuation of the pessary until the infection has cleared and local care with estrogen or antibiotic creams.[22]

Serious complications are a result of prolonged (e.g., often decades) uninterrupted use and neglect rather than directly a result of the pessary itself.[22] Case reports described in the literature include vesicovaginal fistulas, rectovaginal fistulas, urosepsis, and malignancy.[27] Cervical and vaginal cancer is a rare complication and may be related to prolonged irritation or the chemical constituents found in older pessary models.[27] Pessary-related infections can occasionally develop local complications (e.g., abscess, sinus tract, pelvic cellulitis) or spread to other systems and cause systemic manifestations (e.g., pyelonephritis or peritonitis). Patient counseling and adequate follow-up are important.[22] All of the complications are preventable and point to the need for periodic gynecologic examinations in pessary users.[25]

SUMMARY

Reduction of the prolapsed uterus with subsequent pessary placement is a safe and acceptable mechanism for management of women who present to the Emergency Department with symptomatic prolapse. Emergency Physicians should be familiar with this technique and recognize women who are most at risk in order to avoid related complications.

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SECTION 11

Genitourinary Procedures

173 Urethral Catheterization

Richard Dean Robinson and Caleb Andrew Rees

INTRODUCTION

The first recorded bladder catheterization was by Oribasius of Pergamon (325–403 AD) in a description of a fistula surgery. Philip Syng Physick tied Chief Justice Marshall to the bed and introduced instruments through a bladder catheter to break up bladder stones. August Mercer developed the coudé catheter in 1863. The bend near the tip, known as an elbow in French, made passage in a stenosed urethra easier. The current Foley catheter was developed in the 1930s by Urologist Frederick Foley. There have been no major changes in the Foley catheter in over 90 years.

Urethral catheterization is the most frequent retrograde manipulation of the urinary tract. It is routinely performed for diagnostic and therapeutic reasons in both urologic and nonurologic diseases.[1-11] Catheters may be inserted as an in-and-out procedure for immediate drainage, left in place with a self-retaining device for short-term drainage, or left indwelling for long-term drainage.[6,7] **Although this is one of the more routinely performed procedures in the Emergency Department, great care must be taken to avoid lower urinary tract injury, reduce the introduction of infection, and minimize patient discomfort.** The basic principles underlying urethral catheterization are gender neutral.[6,7] **It is important to respect the patient's need for modesty and privacy as much as possible.**

ANATOMY AND PATHOPHYSIOLOGY

The genitourinary system is frequently divided into upper and lower urinary tracts. The former refers to the kidneys and ureters, or those structures above the bony pelvis. The lower urinary tract includes the bladder and urethra, or those structures contained within or below the bony pelvis. Although the entire urinary tract may be catheterized, it is the lower tract, namely the urethra, that will be the focus of this chapter.

Averaging 4 cm in length, the female urethra is rarely a focus of difficulty. Most of the confusion related to urethral catheterization in the female results from poor anatomic knowledge of the external genitalia (**Figure 173-1**). The clitoris is often mistaken for the urethral meatus. This can result in catheter-related trauma, bleeding, patient discomfort, and frustration on the part of the patient and the person inserting the catheter. After lateral retraction of the labia minora and exposure of the vaginal vault, the cephalad-most structure is the clitoris. Traveling in a caudal direction, the orifices encountered are the urethra, the vagina, and then the anus.

The male urethra is most often the site of catheter-associated difficulty.[3] The male urethra may extend upward of 20 cm in length and follows a tortuous course. The male urethra is named based on the anatomic structure with which it traverses or travels. The distalmost portion of the male urethra is the meatus, followed proximally by the penile, bulbar, membranous, and prostatic portions (**Figure 173-2**). Resistance to the advancement of a catheter may occur at any point along the course of the urethra as a result of meatal stenosis, urethral strictures, urethral valves, false urethral passages, an enlarged prostate, inflammatory processes, malignant processes, bladder neck constrictions, urethral disruptions, and/or bladder neck disruptions. A careful clinical history and thorough physical examination will, in most cases, uncover these causes. The two most common sites that may be difficult for the catheter to pass are the junction between the bulbar and membranous urethra and the bladder neck.

INDICATIONS

Urinary catheterization can be performed for diagnostic and/or therapeutic reasons.[2,5,12,13] Urethral catheterization is often performed in females to collect urine for culture and avoid contamination from skin and vaginal flora. This is usually not necessary in males. Measurement of residual (i.e., postvoid) urine, urinary output monitoring in critically ill patients, treatment of urinary incontinence, and facilitation of postsurgical healing are all indications for urethral catheterization. It can be performed to facilitate diagnostic studies such as retrograde cystography and urodynamic evaluations. The main therapeutic indication for urethral catheterization is to relieve acute or chronic urinary retention (**Figure 173-3**).[14-18] Patients with neurogenic bladders are often taught self-catheterization so they may perform this procedure at home. Other indications include bladder irrigation, extensive burn wounds, instillation of medications into the bladder, intraoperative monitoring of urine output, perineal pressure sores with incontinence, prolonged immobilization (e.g., lower extremity fractures, multiple traumatic injuries, pelvic fractures, unstable spine injury), surgery of the urinary tract or nearby structures, the intubated patient, and to improve end-of-life care.

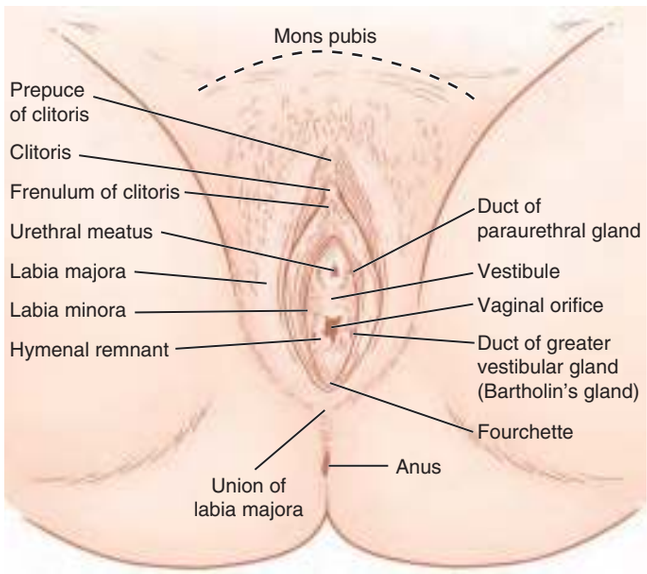

FIGURE 173-1. External anatomy of the female genitourinary tract.

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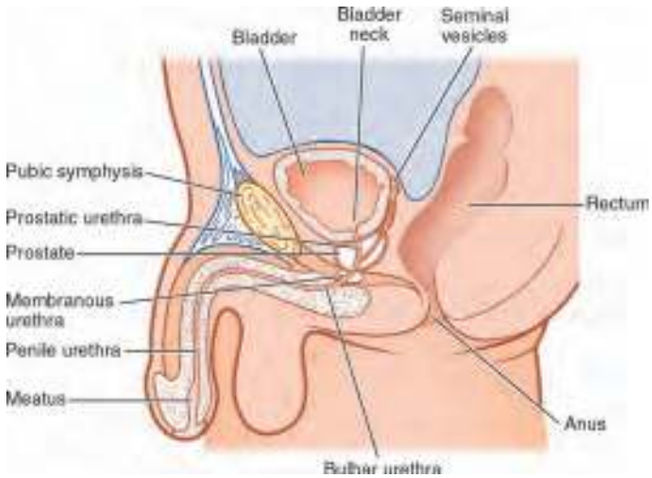


FIGURE 173-2. Anatomy of the male genitourinary tract.

CONTRAINDICATIONS

The only absolute contraindication to urethral catheterization is in patients with known or suspected traumatic injury to the lower urinary tract.[2,4,5,19] They should not undergo urethral catheterization until urethral continuity has been confirmed radiographically. Physical signs on examination that might suggest urethral trauma include blood at the urethral meatus, a perineal hematoma, or a high-riding prostate. **Attempts at urethral catheterization may convert a partial urethral disruption into a complete disruption.** Refer to Chapter 176 for the complete details regarding retrograde urethrography and cystography.

There are a few relative contraindications to urethral catheterization. Microscopic or gross hematuria in the absence of lower urinary tract trauma is not a contraindication to urethral catheterization. Patients with grossly bloody urine are at risk for urinary retention secondary to obstructing clots and require urethral catheterization for bladder irrigation and continuous drainage. Although hypocoagulable states are not contraindications to urethral catheterization, great care must be exercised to avoid iatrogenic trauma and uncontrollable bleeding of the urethra. Prior placement of a penile prosthesis or an artificial urinary sphincter is not a contraindication to catheterization. However, vigorous attempts at inserting the catheter are discouraged. Urethral catheterization should not be performed in an uncooperative or combative patient unless they are sedated and/or restrained. A Urologist should be consulted prior to urethral catheterization in patients with known urethral strictures or recent surgery of the urethra or bladder neck. Children who have a screening urine analysis can use a bag or one of the other techniques to noninvasively obtain urine.[20-24]

The Choosing Wisely Campaign recently published results. Each specialty chose tests or procedures that were not necessary, low yield, or caused more harm than benefit. Emergency Medicine chose, among others, to avoid using bladder catheters routinely in stable patients to decrease the number of catheter-associated urinary tract infections.[19] This included catheterization of the bladder for convenience of the patient, convenience of the staff, morbid obesity, patient refusal, patients with dementia, patients with delirium, patients who are intoxicated, and urine output monitoring when the patient can use a urinal.

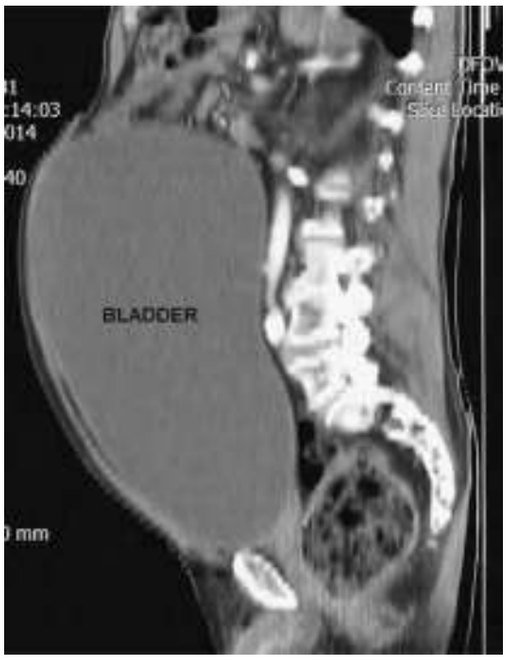


FIGURE 173-3. Sagittal view of the enlarged bladder on an abdominal computed tomography scan.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile gloves
- Commercially available kit containing the components listed below
- Water-soluble lubrication gel
- Lidocaine jelly in penile applicator (i.e., Uro-jet)
- Sterile drapes
- Urethral catheters (Foley, coudé, filiform, and followers)
- Christmas tree adapter
- Pediatric feeding tubes, sizes 5 to 8 French
- Sterile saline
- 10 mL syringe
- Urine meter, if catheter is to be retained
- Urine collection system
- Urethral catheter leg strap, if catheter is to be retained
- 1 inch tape
- Benzoin solution
- Ultrasound (optional)
- Ultrasound gel (optional)
- Ultrasound transducer cover (optional)
- High-frequency linear array or micro-convex transducer (optional)

Foley catheters are the most commonly placed urethral catheters. A Foley catheter is a dual lumen tube that contains an inflatable cuff near the distal end (**Figures 173-4 and 173-5**). The distal end has two holes for urine to enter the catheter. Urine traverses the large inner lumen of the catheter to exit the proximal port. The second lumen is extremely small and allows air or fluid to flow into the inflatable cuff. The proximal end contains two ports. One port allows egress of urine from the catheter. The second port is an inflation port. An air-filled or saline-filled syringe attaches to this port and is used to inflate the cuff. When inflated, the cuff prevents the distal end of the catheter from slipping out of the bladder.

Foley catheters come in a variety of sizes and styles. A 14 or 16 French catheter is the size most commonly used in adolescent or

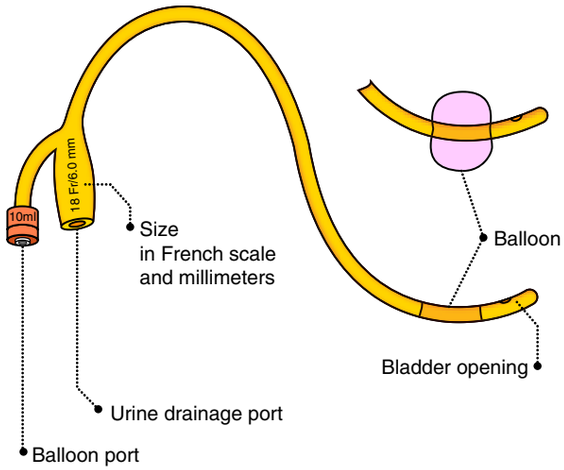


FIGURE 173-4. The schematic of a urethral catheter. (Used from www:commons.wikimedia.org/wiki/File:Foley_catheter_inflated_and_deflated_EN.svg.)

adult patients. The two-way catheter is most commonly used. It is designed for urinary drainage. Three-way catheters are employed when bladder irrigation, in addition to urinary drainage, is required. These catheters have a small lumen to inflate the cuff, an intermediate-sized lumen to instill irrigation solution, and a large lumen to drain urine from the bladder. In the Emergency Department, they are placed in patients with gross hematuria and the passage of blood clots that may have caused acute urinary retention.

There are many advantages to the use of bedside ultrasound. It is quick, uses no radiation, shows if the bladder is enlarged, and obviates the need for additional routine workup of abdominal pain until the distended bladder is drained and symptoms are not relieved. Bladder ultrasound increases the successful catheterization procedure.[24,25] Bedside ultrasound is more accurate than dedicated bladder ultrasound devices.[26]

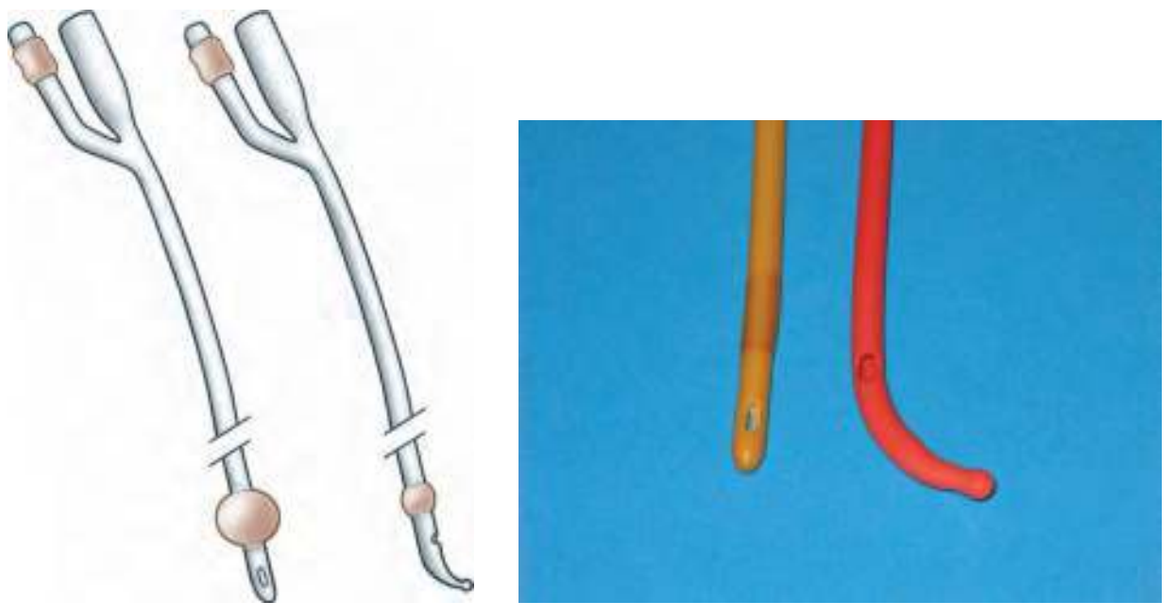
PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. **Urethral catheterization must be performed using strict sterile technique.** All equipment needed to perform the procedure should be assembled at the bedside prior to beginning the procedure. The preparation for a male patient will be described below. The preparation for a female patient will be described in the techniques section.

Place the male patient in a bed or gurney. In uncircumcised patients, the foreskin must be retracted to expose the glans penis and the urethra. If a phimosis is encountered, it should be approached accordingly. In the pediatric population, male infants and children often have a physiologic phimosis. **Attempts at aggressive foreskin retraction should be avoided.** Clean any dirt and debris from the penis. Apply povidone iodine or chlorhexidine solution, with cotton balls or swabs and allow it to dry. Place sterile drapes around the penis to isolate a sterile field.

Generous lubrication and anesthesia of the male urethra is one of the most important aspects of urethral catheterization.[9,10,27-30] A water-soluble lubricant (e.g., K-Y Jelly) or local anesthetic lubricant (e.g., 2% lidocaine jelly) can be applied to the tip of the urethral catheter before it is inserted. Unfortunately, this provides little to no anesthesia. Ideally, commercially packaged blunt-tip syringes of lidocaine jelly (e.g., the Uro-jet, Amphastar Pharmaceuticals Inc., Rancho Cucamonga, CA) should be used. Insert the blunt tip of the syringe into the urethral meatus. Firmly squeeze the glans penis to form a seal around the syringe tip. Inject the anesthetic jelly into the urethra. Maintain the syringe tip in the urethral meatus and manual pressure on the glans penis for 10 seconds to prevent egress of the anesthetic jelly. Allow several minutes for the lidocaine jelly to anesthetize the urethra.

Prophylactic antibiotic coverage is generally not indicated for urethral catheter placement. Patients with penile prostheses, artificial urinary sphincters, prosthetic heart valves, vascular grafts, and other indwelling foreign bodies may benefit from prophylactic antibiotics. An intravenous dose of a cephalosporin, quinolone, or other



A B

FIGURE 173-5. Commonly used urethral catheters. ***A.*** Illustration of the Foley catheter (left) and the coudé catheter (right). ***B.*** Photograph of the distal ends of the Foley catheter (left) and the coudé catheter (right).

antibiotic that covers skin and perineal flora may be administered just prior to inserting the urethral catheter in these patients.

MALE CATHETERIZATION TECHNIQUES

FOLEY CATHETER INSERTION

Clean and prep the penis, anesthetize the urethra, and set up a sterile field as mentioned previously. Select an appropriately sized Foley catheter. Inflate the cuff to check its integrity. Deflate the cuff. Grasp the penis with the nondominant hand. Pull the penis taut and upright to straighten out the penile urethra (**Figure 173-6*A***). Retract the foreskin if the patient is uncircumcised. **Do not forget to reduce the foreskin after inserting the catheter to prevent the formation of an iatrogenic paraphimosis.** Grasp the Foley catheter with the dominant hand. Dip the tip of the catheter in, or an assistant can apply, water-soluble lubricant or anesthetic jelly. Insert the catheter into the penile urethra via the meatus (**Figure 173-6*A***). Gentle and continuous pressure helps open the urogenital sphincter to let the Foley catheter pass. Continue to gently but firmly advance the Foley

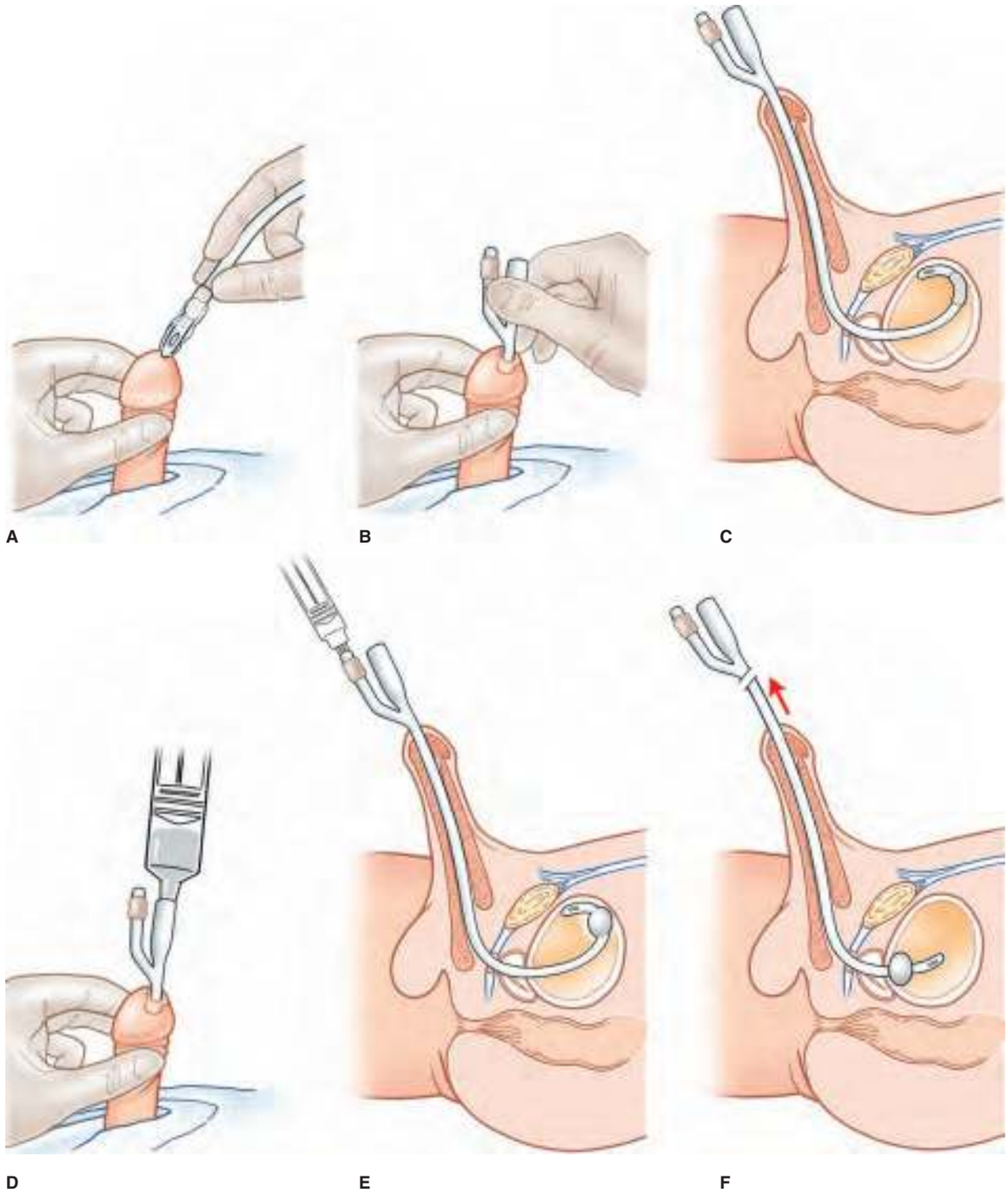


FIGURE 173-6. Foley catheter insertion. ***A.*** The lubricated catheter is inserted into the urethra. ***B.*** The catheter is advanced until the ports are at the meatus. ***C.*** Cross-section of the male pelvis showing the distal catheter positioned within the bladder. ***D.*** Urine aspiration confirms proper placement of the catheter. ***E.*** The cuff at the tip of the catheter is inflated. ***F.*** The catheter is gently withdrawn to lodge the cuff against the bladder neck.

catheter into the urethra until the proximal ports are at the urethral meatus (**Figure 173-6*B***). This ensures that the distal tip of the catheter and the cuff will reside within the bladder (**Figure 173-6*C***). If the distal end of the catheter is not completely within the bladder, inflation of the cuff inside the urethra will result in severe pain, hematuria, and possible urethral rupture.

Urine should begin to spontaneously flow out of the large port. Insert the proximal end of the catheter into a sterile container to collect urine. If urine does not spontaneously flow out of the large port, attach a 60 mL syringe to the port and aspirate urine to confirm proper placement of the catheter (**Figure 173-6*D***). Consider using transabdominal ultrasonography to confirm proper catheter placement and the presence of urine within the bladder. Deflate the cuff if no urine is aspirated and/or ultrasound imaging does not confirm appropriate placement. Remove the catheter from the urethra and reattempt the procedure.

Attach an air-filled or saline-filled syringe to the cuff inflation port (**Figure 173-6*E***). Inject air or saline to inflate the cuff (**Figure 173-6*E***). Inject only the volume of air or sterile saline recommended by the catheter manufacturer. This can be found on the catheter package and is often printed on the cuff inflation port. Remove the syringe from the cuff inflation port. Gently withdraw the Foley catheter until resistance is met. This signifies that the cuff is lodged against the bladder neck (**Figure 173-6*F***). Attach an adapter and urine collection system to the urine port of the Foley catheter. Reduce the previously retracted foreskin, if present and retracted, once the catheter placement is confirmed.

Secure the catheter. Wrap tape around the urine port of the catheter and continue it onto the adapter and first 3 to 5 cm of the collection tubing. This will prevent the system from disconnecting. Tape the collection tubing to the patient's thigh to prevent it from pulling out the adapter or the catheter when the patient moves. Some authors also secure the catheter as it exits the penile urethra (**Figure 173-7**).[5] This is especially important if a red rubber catheter or uncuffed coudé catheter is used as these cuffless catheters are not self-retaining. Place three thin strips of tape along the length of the penis and attach them to the catheter. Benzoin solution can be applied to the penis to aid in adhesion of the tape. Place a piece of tape circumferentially around the tape ends attached to the catheter to secure the tape strips. **Never apply tape circumferentially around the penis as it may cause ischemia.**

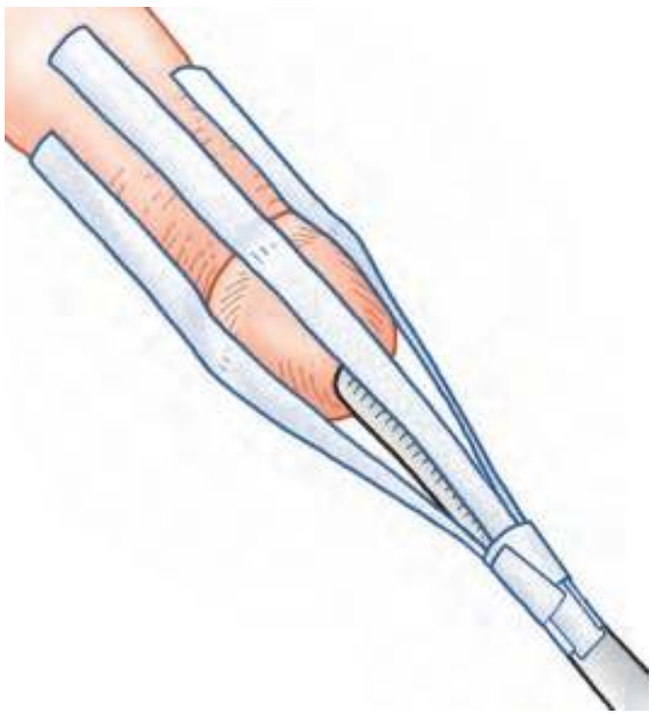


FIGURE 173-7. A method to secure the urethral catheter as it exits the penis.[5]

DIFFICULT URETHRAL CATHETERIZATION

Difficulty is commonly encountered when inserting a urethral catheter. Urethral pathology (e.g., valves, strictures, or folds) or prostate enlargement can often be overcome by switching from a Foley to a coudé catheter and the application of digital assistance. If unsuccessful, filiforms and followers can often be employed to successfully insert the catheter. Foreskin edema from anasarca, pelvic lymphatic blockage, a paraphimosis, or penile trauma can often cover the glans penis and urethral meatus. Attempt to reduce the edema manually or with an elastic bandage (Chapter 179). Significant edema may require penile anesthesia and needle decompression (Chapters 177 and 179) or a dorsal slit of the foreskin (Chapter 181). A phimosis may be physiologic or acquired in children and the result of scarring in adults from repeated bouts of inflammation, infection, or sexually transmitted diseases. A phimosis may require a dorsal slit of the foreskin (Chapter 181) to allow for urethral catheterization or a dilation of the phimotic opening.

The DirectVision™ microendoscope (PercuVision LLC, Columbus, OH) consists of a rolling cart, fiberoptic endoscopic bundle, and tri-lumen silicone catheter. The cart houses a color monitor and the fiberoptic light source (**Figure 173-8*A***). A disposable fiberoptic bundle is attached to the light source and is inserted through the tri-lumen catheter (**Figures 173-8*B* and 173-8*C***). The fiberoptic bundle transmits light to the tip of the catheter and images back to the monitor. The system allows visually guided urethral catheterization in the male patient. It may decrease urinary tract trauma during catheterization, improve catheterization success rates, and simplify the catheterization process. The major limitation of this product for routine urethral catheterization is due to the initial expense of the system and the cost of the single-use disposable endoscopic bundles and catheters. This device may be commonly used in the future as costs decrease and clinical information becomes more readily available.

COUDÉ CATHETER INSERTION

The coudé catheter is similar to a Foley catheter except that the distal end is curved and the tip has a small rounded ball (**Figure 173-5**). The catheter was designed to bypass the areas of the urethra that a straight catheter could not negotiate. A coudé catheter may be used if a Foley catheter cannot be passed into the bladder. It may also be used if the patient has a known urethral stricture, urethral valve, narrow urethra, or enlarged prostate.

The coudé catheter comes in various sizes and styles. Some models contain a cuff while others do not. The catheter is inserted into the urethra with the elbow on the tip of the catheter facing anteriorly. The procedure for placement of the catheter into the bladder is the same as that for a Foley catheter.

DIGITAL ASSISTANCE

Occasionally, the Foley or coudé catheter tip will become caught in a posterior fold of the urethra just distal to the urogenital diaphragm (**Figure 173-9*A***).[31] Place the fingers of the nondominant hand on the perineum between the scrotum and anus (**Figure 173-9*B***). Apply upward pressure on the perineum to direct the catheter tip upward while simultaneously advancing the catheter with the

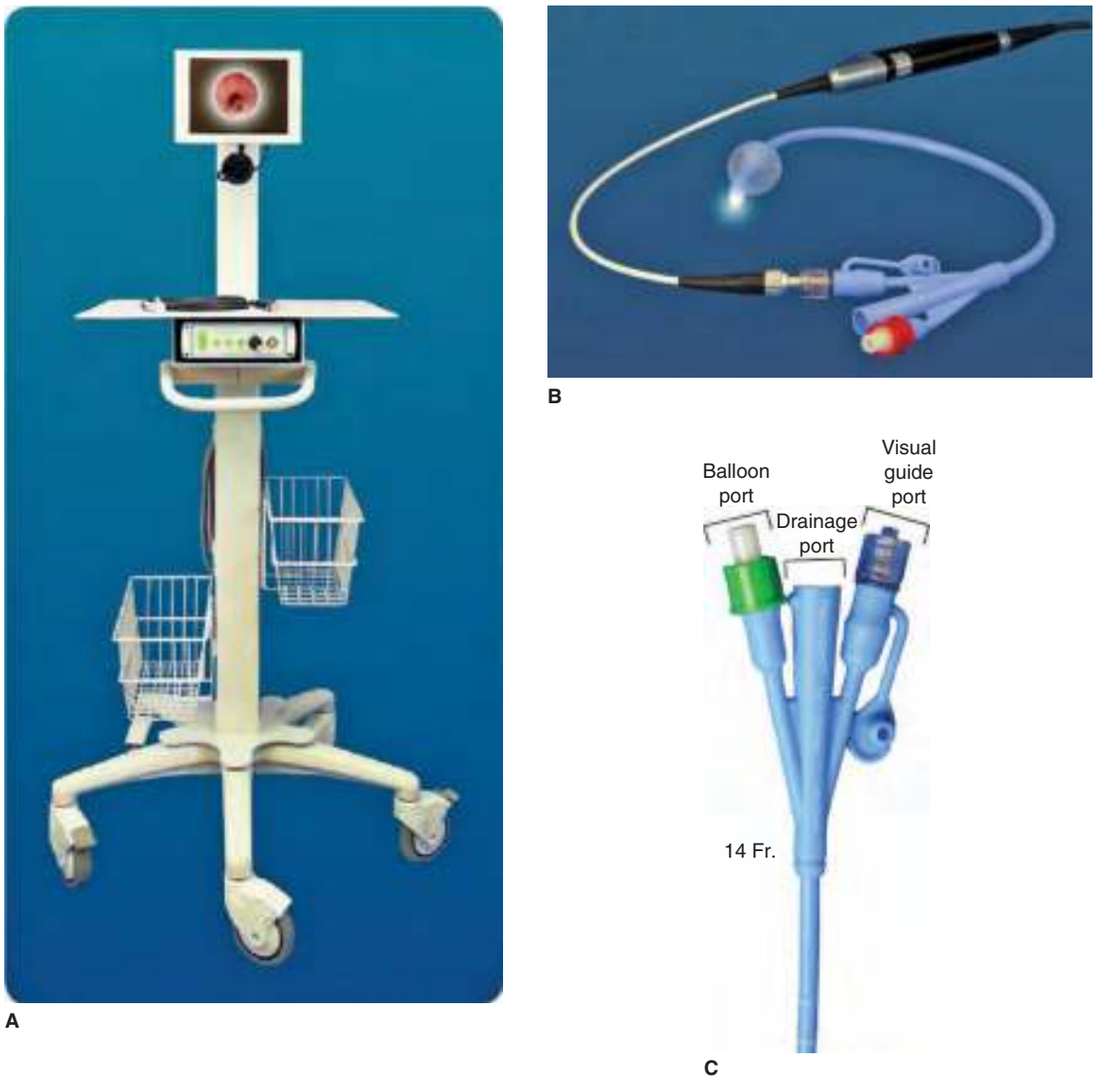


FIGURE 173-8. The PercuVision DirectVision microendoscope. ***A.*** The cart. ***B.*** The catheter with the light source inserted. ***C.*** The proximal catheter. (Photos courtesy of PercuVision.)

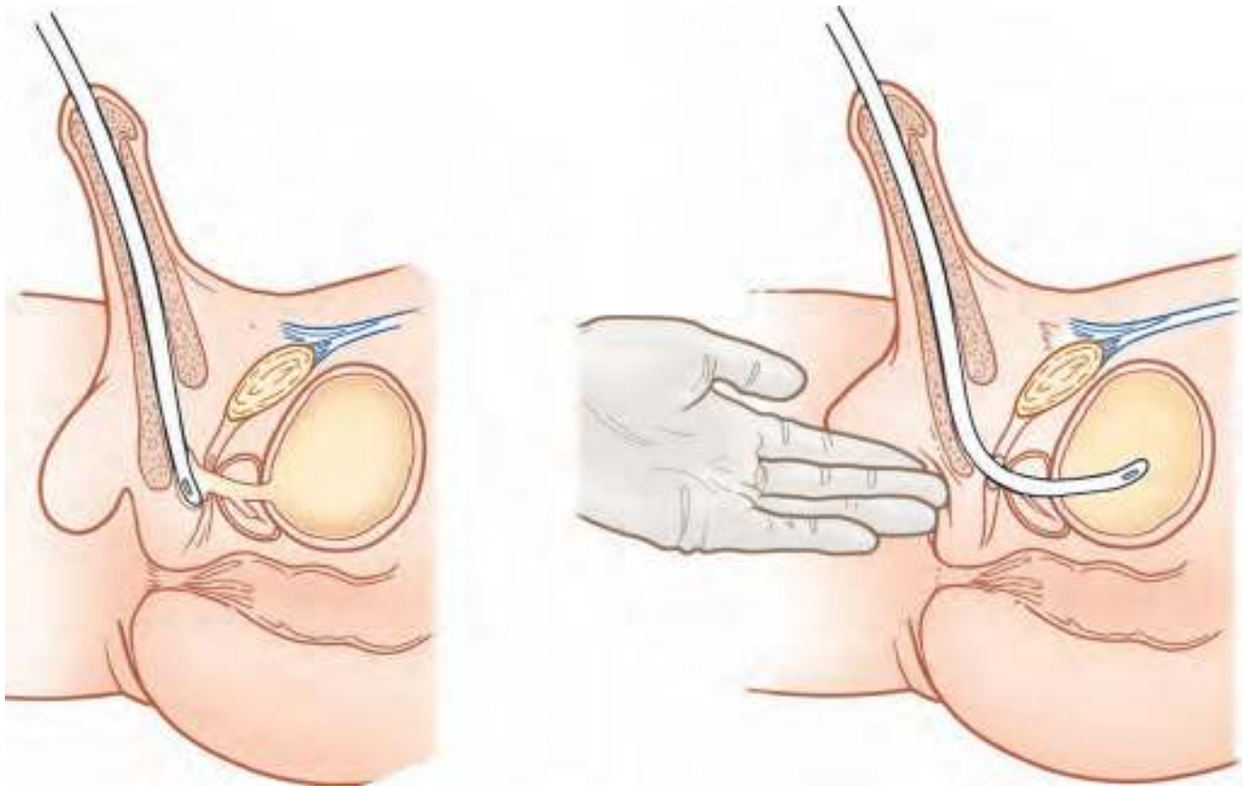
dominant hand through the urogenital diaphragm and into the bladder. An assistant is often required to hold and stabilize the penis while the Emergency Physician uses their hands to manipulate the catheter and perineum.

If the patient has an enlarged prostate, the bladder neck is often elevated superiorly and anteriorly. Digital assistance on the skin of the perineum may be unsuccessful. A finger in the rectum may be used to move the catheter tip anteriorly so that it can be advanced into the bladder.

FILIFORM AND FOLLOWER CATHETERS

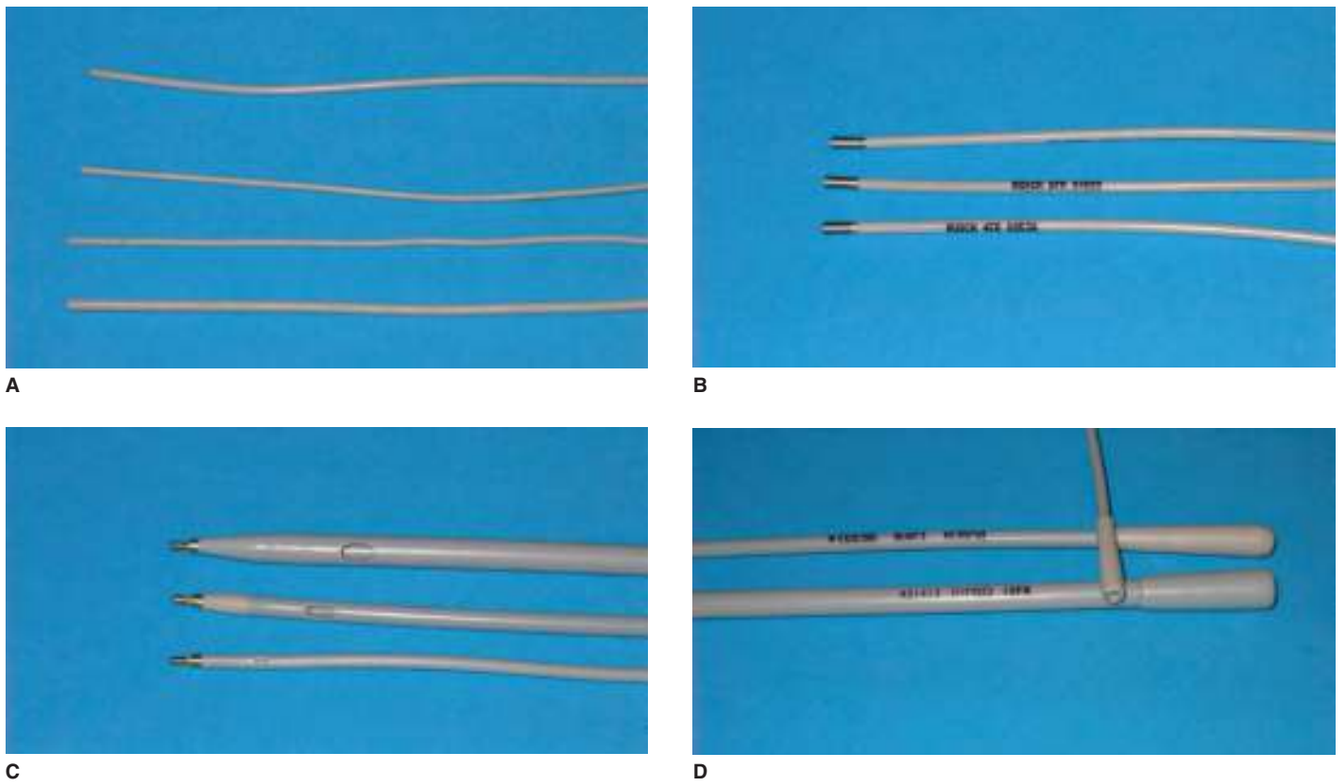
In patients with severe urethral strictures or urethral folds, it may be impossible to pass a Foley catheter or a coudé catheter. The next step in the progression to catheterize the bladder is to use filiform and follower catheters. Filiforms are very narrow, flexible, and solid catheters. Their sole function is to successfully negotiate a strictured urethral segment and enter the bladder. The distal end of the filiform catheter may be straight or pig-tailed and is available in a variety of sizes (**Figure 173-10*A***). The proximal end of the filiform catheter is a standard size and contains a metal female connector (**Figure 173-10*B***). The followers are flexible, hollow catheters that attach to the filiform catheters. The distal end of the follower catheter contains a metal male connector that attaches to the proximal end of the filiform catheter and a hole to allow urine to enter the catheter (**Figure 173-10*C***). Follower catheters come in a variety of sizes and allow the Emergency Physician to dilate the urethra and catheterize the bladder. The proximal end of the follower catheter is open and accepts a Christmas tree adaptor (**Figure 173-10*D***).

A filiform and follower should be used only after unsuccessful catheterization attempts with a Foley catheter and/or coudé catheter. Do not use them if there is contrast extravasation on a retrograde urethrogram, a urethral disruption (real or potential), or urethral trauma. The patient has already been prepped and draped for the prior catheterization attempts. Re-instill anesthetic jelly into the urethra to ensure adequate anesthesia. Numerous sizes and shapes of filiform and follower catheters should be available at the bedside. An assistant will be required to open each sterile packet and hand the filiforms and followers to the Emergency Physician as needed.



A B

FIGURE 173-9. Digital assistance. ***A.*** The Foley catheter is caught in a posterior urethral fold. ***B.*** Digital upward pressure on the perineum will direct the catheter tip upward and through the urogenital diaphragm.



A B C D

FIGURE 173-10. Photographs of the ends of the filiform and follower catheters. ***A.*** Distal ends of the filiform catheter. ***B.*** Proximal ends of the filiform catheter. ***C.*** Distal ends of the follower catheter. ***D.*** Proximal ends of the follower catheter.

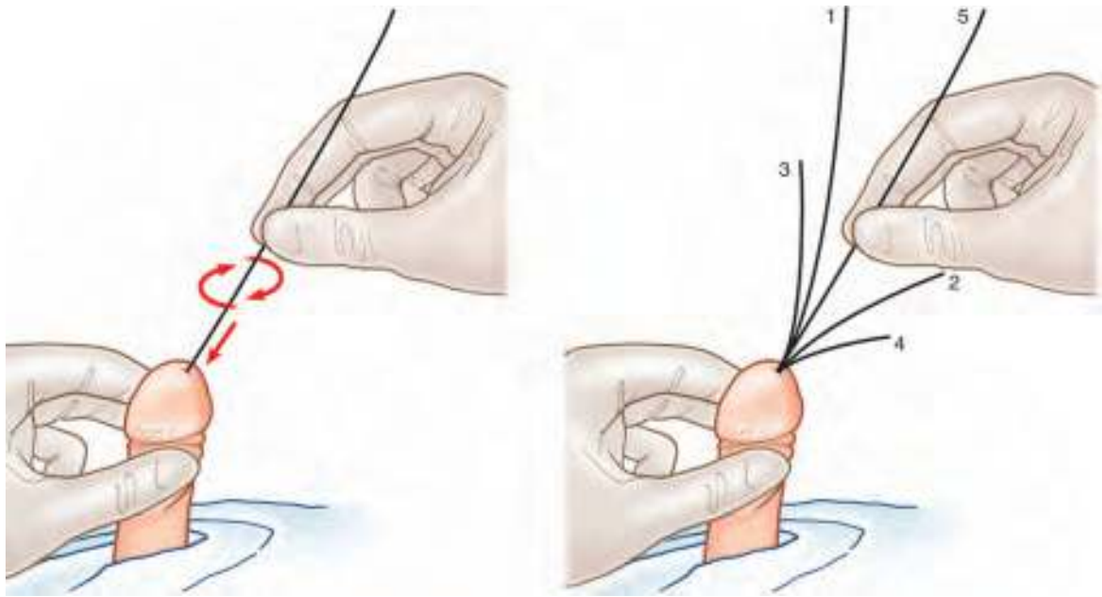


FIGURE 173-11. Insertion of the filiform catheters. ***A.*** The catheter is inserted and advanced into the urethra (straight arrow) with a twisting motion (curved arrows). ***B.*** Additional catheters are inserted until one advances into the bladder. The numbers represent the order of insertion of the filiform catheters.

Grasp the cleaned, prepped, and anesthetized penis with the nondominant hand. Pull the penis taut and upright to straighten out the penile urethra (**Figure 173-11*A***). Grasp a filiform catheter and dip the tip in water-soluble lubricant or anesthetic jelly. Gently insert and advance the filiform catheter into the urethra with a twisting motion (**Figure 173-11*A***). Stop advancing the filiform catheter when resistance is met (**Figure 173-12*A***). Insert a second well-lubricated filiform catheter into the urethra with a twisting motion until it meets resistance (**Figure 173-12*B***). Gently attempt to advance the first filiform catheter. If it will not advance, insert a third filiform catheter (**Figure 173-12*C***). Continue to insert filiform catheters and gently manipulate the previously inserted filiform catheters (**Figure 173-11*B***). Continue this process until one filiform catheter advances into the bladder so that its proximal end is 2 cm from the tip of the penis (**Figure 173-12*C***). Remove all the filiform catheters except the one that entered the bladder (**Figure 173-12*D***).

Choose a follower catheter to insert into the bladder. Liberally lubricate the tip of the follower catheter and attach it to the filiform catheter (**Figure 173-13*A***). Gently advance the follower catheter until its proximal end is 3 to 4 cm from the tip of the penis (**Figure 173-13*B***). **If the follower catheter meets resistance during its advancement, do not force it into the urethra.** Instead, withdraw the follower catheter until the tip is 2 to 3 cm outside the penis. Remove the follower catheter from the filiform catheter. Attach a 1 or 2 French smaller well-lubricated follower catheter onto the filiform catheter and attempt to advance it into the bladder. Continue this process until a follower catheter can be completely advanced into the bladder. The filiform catheter will be completely curled up inside the bladder (**Figure 173-13*B***).

Urine should flow spontaneously from the properly positioned follower catheter. If not, attach a 60 mL syringe and Christmas tree adaptor to the follower catheter (**Figure 173-13*C***). Apply negative pressure to the syringe to aspirate urine and confirm the proper

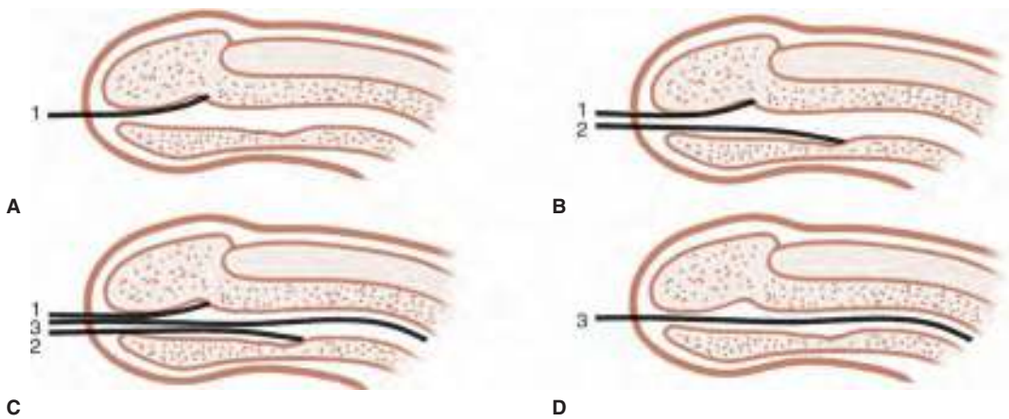


FIGURE 173-12. Midsagittal section of the penis demonstrating insertion of the filiform catheter. ***A.*** The filiform catheter is inserted until resistance is encountered. ***B.*** A second filiform catheter is inserted until it encounters resistance. ***C.*** A third filiform catheter is inserted and advances into the bladder. ***D.*** Filiform catheters #1 and #2 have been removed.

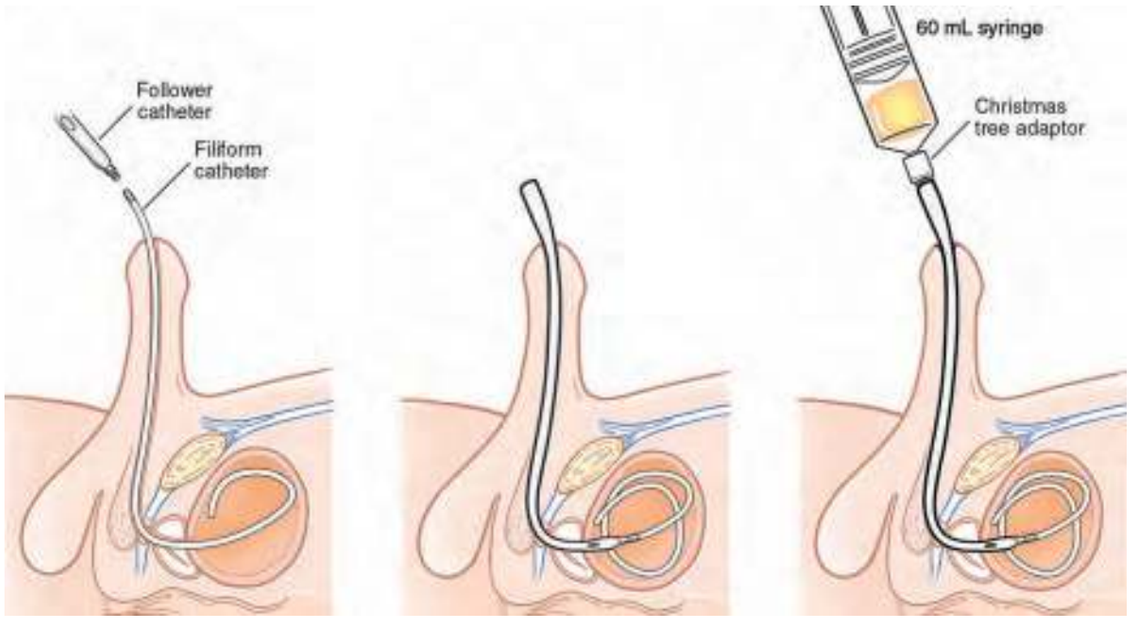


FIGURE 173-13. Insertion of the follower catheter. The filiform catheter has been previously inserted into the bladder. ***A.*** The follower catheter is screwed into the filiform catheter. ***B.*** The follower catheter is advanced into the bladder. ***C.*** Urine aspiration confirms proper placement.

placement of the tip of the follower catheter. Attach a urinary collection system to the follower catheter and secure the catheter as described previously. Inadequate local anesthesia may require procedural sedation and anesthesia (Chapter 159) or general anesthesia.

The use of filiforms and followers has many complications. The creation of a false passage, urethral injury, and urethral perforation are possibilities. Bleeding can be the result of any of the latter. The use of multiple instruments increases the risk of infection.

The patient with urinary obstruction cannot be discharged home with a filiform and follower catheter inserted inside the bladder. If the follower catheter is a size 16 or 18 French, completely withdraw it and the filiform catheter and insert a 16 French Foley catheter. If the follower catheter is smaller than size 16 French, the urethra must be dilated. Withdraw the follower catheter until the distal tip is 2 to 3 cm outside the penis. Remove the follower catheter from the filiform catheter. Attach a 1 to 2 French larger well-lubricated follower catheter onto the filiform catheter and gently advance it into the bladder. Continue this process until a follower catheter that is size 16 French easily passes into the bladder. Remove the filiform and follower catheters. Insert a size 16 French Foley catheter. Secure the catheter as described previously.

FEMALE CATHETERIZATION TECHNIQUES

Place the female patient supine in an examination bed. Place the patient in the frog-legged position. If an examination table equipped with stirrups is available, the patient can be placed in the lithotomy position. Separate the labia with the nondominant thumb and index

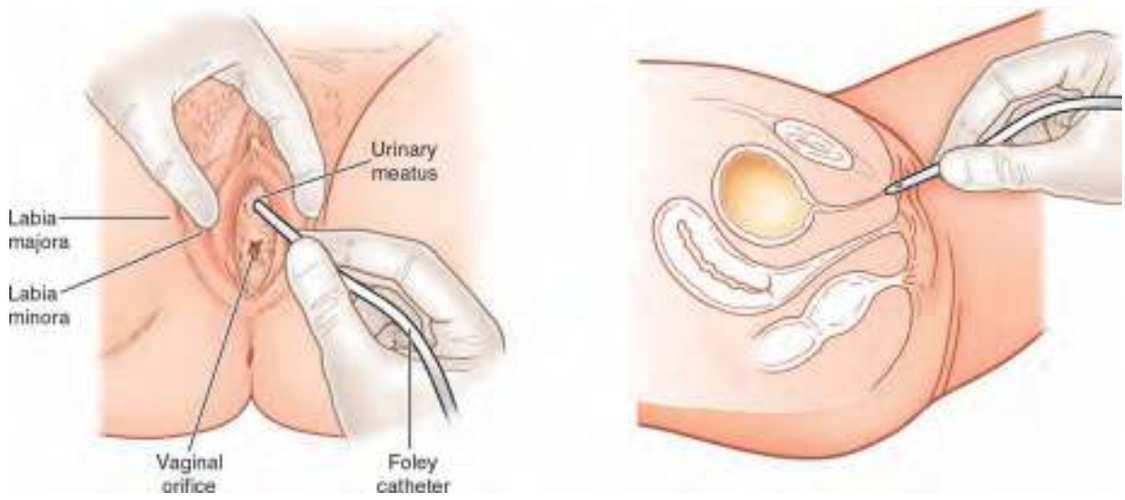


FIGURE 173-14. Female urethral catheterization. ***A.*** External view of the genitalia. The catheter is inserted into the urethral meatus and advanced. ***B.*** Midsagittal section of the female pelvis demonstrating catheter insertion.

finger to expose the vulva (**Figure 173-14*A***). Identify the urethral meatus. Apply povidone iodine or chlorhexidine solution to the urethral meatus and surrounding vulva. Without moving the nondominant hand, apply sterile drapes to isolate the vulva. A lesser amount of anesthetic jelly is needed to lubricate the female urethra. Place anesthetic jelly on a sterile cotton-tipped applicator. Insert the cotton-tipped applicator just into the tip of the urethral meatus for 8 to 10 seconds. Remove and discard the cotton-tipped applicator. This method of anesthesia is painful and avoided by most.

Grasp the Foley catheter with the dominant hand. Dip the tip of the catheter in, or an assistant can apply, water-soluble lubricant or anesthetic jelly. The use of anesthetic jelly on the catheter provides no anesthesia.[32,33] Insert the catheter into the urethral meatus (**Figures 173-14*A* and 173-14*B***). Advance the catheter 6 to 8 cm to ensure the distal end and cuff are within the bladder. Urine should flow spontaneously out of the large port. Insert the proximal end of the catheter into a sterile container to collect urine. If urine does not flow spontaneously from the large port, attach a 60 mL syringe to the port and aspirate urine to confirm proper placement of the catheter. The remainder of the procedure is exactly the same as previously described for the male patient.

Some women may present with a cystocele, cystourethrocele, or urethrocele. These abnormalities result in the urethra running posteriorly and inferiorly making urethral catheterization difficult. Insert your gloved and lubricated index and middle fingers into the vagina and gently push superiorly and anteriorly on the anterior vaginal wall. This maneuver will reposition the bladder and urethra to its normal position. Insert the urethral catheter in the usual manner while maintaining anteriorly directed pressure on the anterior vaginal wall.

INFANT AND CHILDREN CATHETERIZATION TECHNIQUES

The indications and contraindications for catheterization of infants and children are the same as for adults. The anatomy of the prepubertal child is similar to the adult except for the difference in size and lack of secondary sexual characteristics. Identifying the urethral meatus can be difficult in uncircumcised boys. An assistant is often required to help position children during the procedure. Gently manipulate and retract the foreskin just enough to expose the meatus. It is worth noting that the urethral orifice may be difficult to identify in a young female due to abundant hymenal tissue covering the vaginal introitus. **This can be circumvented by gentle lateral traction of the labia and downward pressure on the vaginal introital fold with a cotton-tipped applicator which will reveal the urethral opening immediately anterior to the vaginal orifice**. The catheterization of newborns and infants is accomplished with an appropriately sized pediatric feeding tube, usually size 5 French in the neonate up to 8 French in newborns and infants. Small Foley catheters or feeding tubes (e.g., size 10 or 12 French) may be used in children aged 1 to 6 years. Children over 12 years of age can accommodate a 14 French catheter. **The same techniques and cautions followed in the adult patient also apply to the pediatric patient. Advancing the catheter a few centimeters longer than the length of the penis in boys and just a few centimeters in girls is a reasonable approach.[34] Children may be more prone to attempting to try and remove the catheter. Ensuring the catheter is adequately secured after placement is paramount.**

REMOVAL OF THE CATHETER

Consider if a urethral catheter is really needed. If so, remove it as soon as possible. Removal is much easier than insertion and does not require anesthesia. Apply gloves. To be safe, also use a mask, eye protection, and a gown. Drain the bladder. Deflate the catheter cuff with a syringe. Loosely hold the gauze around the catheter near where it exits the body. Pull out the catheter through the gauze to prevent splashing urine. Culturing the catheter tip is on an individual patient basis.

ASSESSMENT

Urine drainage is the sign of appropriate catheter placement. Lubricating gel may plug the outflow port of the catheter. If no urine is seen, apply gentle pressure to the suprapubic area to force the flow of urine. Should this maneuver fail to initiate urine flow, irrigate the catheter with a small volume of sterile saline. The catheter will flush and withdraw fluid with ease if properly positioned in the urinary bladder. Use of an irrigation tray, which includes a 60 mL piston tipped syringe, is recommended. It may require greater than one fill (i.e., more than 60 mL of saline) to produce two-way flow. The inability to successfully irrigate denotes obstruction or misplacement of the catheter. Ensure that the catheter is inserted completely to the hub in males to prevent inadvertent inflation of the cuff in the membranous or prostatic urethra.

AFTERCARE

Always remember to reduce the foreskin after catheter placement to avoid the complication of a paraphimosis in uncircumcised males. Indwelling catheters should be secured to a closed gravity drainage system and attached to the anterior medial thigh. For long-term requirements in males, the catheter should be secured to the anterior abdominal wall to decrease the likelihood of stricture formation.

The patient may be discharged from the Emergency Department with a Foley catheter or coudé catheter. Instruct the patient on the proper care and emptying of the leg bag (**Table 173-1**). The patient should immediately return to the Emergency Department if they develop a fever, pain, hematuria, inability to void through the catheter, or abdominal pain. Consider prescribing a 3 day supply of broad-spectrum antibiotics if the patient has valvular heart disease, mitral valve prolapse, cardiac valve replacements, a penile prosthesis, or an artificial joint.

COMPLICATIONS

GENERAL COMPLICATIONS

Creation of false passages, urethral perforations, bleeding, infection, and catheter misplacement are complications associated with urethral catheterization. Urethritis is fairly common following catheterization, especially in patients with urethral strictures or prostatic enlargement. Epididymitis, cystitis, and pyelonephritis are uncommon complications and often seen with prolonged catheterization. Bacteremia can occur following the procedure. High-risk patients should receive antibiotic prophylaxis prior to catheterization.

TABLE 173-1 Patient Instructions for the Proper Care of a Urinary Drainage Bag at Home

1. Always thoroughly wash your hands with soap and warm water before putting on gloves and emptying the drainage bag.
2. Never place the drainage bag on the floor to prevent contamination.
3. Always keep the drainage bag below the level of the genitals to prevent backflow of urine.
4. Clean the drainage port with an alcohol swab before emptying the bag.
5. Always empty the drainage bag into a clean container.
6. Do not touch the drainage port or allow it to touch the container.
7. Close the drainage port after emptying the bag and wipe it with an alcohol swab.
8. Discard the urine in the container and thoroughly wash the container with hot soapy water.
9. Discard the gloves and do not reuse them.
10. Always thoroughly wash your hands with soap and warm water after taking off your gloves.

Urinary tract infection (UTI) is also a common complication after urethral catheterization. It is especially common with a prolonged duration of an indwelling catheter which can lead to susceptibility to a variety of organisms including those that produce biofilms that can adhere to the catheter and external balloon. A recent Cochrane review evaluated the difference between urethral and suprapubic catheterization.[35] It found that suprapubic catheters reduced the risk of re-catheterization, pain, and asymptomatic bacteriuria. There was incomplete evidence to comment on whether either method was superior with regard to symptomatic UTI. The evidence was also inconclusive for symptomatic UTI when comparing indwelling and intermittent urethral catheterization. A silver-coated or antibiotic-coated catheter may decrease microbial colonization of the catheter.[36]

Iatrogenic trauma to the urethra can lead to strictures, hemorrhage, hematuria, or fistulas.[37,38] Creation of a false passage can occur by inserting the catheter alongside the urethra. Attempts at catheterization in a trauma patient with a urethral injury can convert a partial urethral tear to a complete tear. Failure to reduce the foreskin after catheter placement may result in a paraphimosis.

LEAKAGE AROUND THE CATHETER

Urine may occasionally leak out between the catheter and the walls of the urethra. Make sure that an appropriately size catheter has been inserted. Check the balloon to make sure it is properly inflated. Gently pull on the catheter to seat the balloon at the neck of the bladder. Too large of a catheter or balloon can irritate the urinary tract or bladder and result in bladder spasm and urine leakage. Check the catheter, tubing, and drainage bag for kinks. An occlusion distal to the catheter can result in retained urine, an overdistended bladder, and bladder spasm. Evaluate a urine sample for the presence of an infection. A UTI can result in bladder spasm.

DIFFICULTY REMOVING A CATHETER

A less common complication is difficulty removing an indwelling urethral catheter that has an inflated cuff. Ensure that the catheter is not clamped or kinked. This can compress the cuff inflation lumen. A vacuum may have formed in the inflation lumen causing it to collapse. Attach a syringe without the plunger to the inflation port and release the vacuum followed by aspiration to deflate the balloon. The use of a syringe without the plunger will sometimes deflate the cuff without aspirating. Gently turn the catheter clockwise and counterclockwise as the catheter may be compressed, kinked, or twisted. Gently advance the catheter further into the bladder and attempt to deflate the balloon. Gently inject 2 to 3 mL of sterile saline through the inflation port to open the inflation lumen or unclog it. Inspection of the valve sometimes reveals the problem. One may attempt to cut proximal to the valve in hopes of evacuating the cuff contents but this is not always successful. **Do not do this unless other methods have failed.**

In patients with a Foley catheter balloon that will not deflate, real-time ultrasound (US) can facilitate percutaneous balloon puncture and removal of the catheter.[39] Prepare the skin and apply sterile US gel to the patient's skin. Apply a sterile US transducer cover over the transducer. Stand to the side of the patient so that your dominant hand is cephalad (**Figure 174-6**). Position the US monitor on the opposite side of the patient so that it is easy to see (**Figure 174-7**). A longitudinal-oriented US transducer is preferred for the dynamic approach since it allows visualization of the entire needle as it courses toward the bladder. Place the US transducer marker to your left. This will ensure that the left-right orientation on the US screen matches the real-world orientation.

Place the longitudinally oriented US transducer in the suprapubic area and move it from side to side to locate the largest bladder dimension. To deflate a Foley catheter balloon, make sure there is sufficient fluid in the bladder. If necessary, fill the bladder with enough sterile saline through the Foley catheter to make the bladder easily visible on the US monitor. Clamp the Foley catheter to retain the saline within the bladder. Instruct an assistant to apply gentle traction to the external portion of the Foley catheter to fix the balloon against the bladder neck.

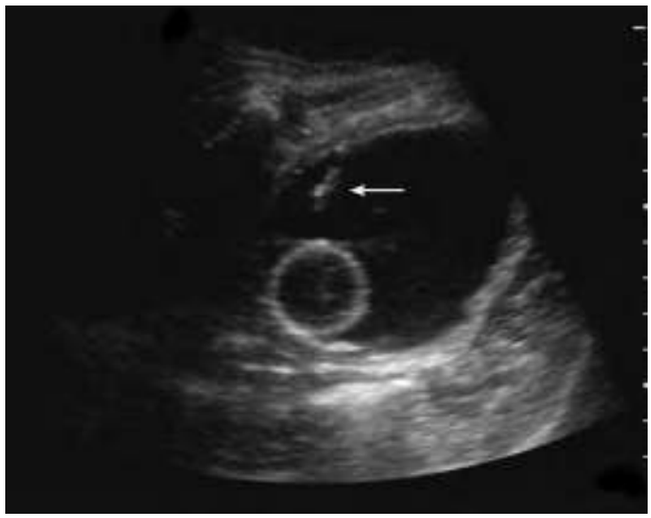


FIGURE 173-15. Longitudinal US view of the transabdominal spinal needle (arrow) approaching the Foley balloon.

Place a 5 inch, 22 or 24 gauge spinal needle on a 10 mL syringe. Position the spinal needle on the cephalad side of the US probe and angle it caudally (**Figure 174-6**). Insert and slowly advance the spinal needle. Look for the needle on the US screen as it is inserted and advanced. If the needle is a narrow gauge and the US transducer is low frequency, the spinal needle will not necessarily be visible in the tissue. Rather, look for movements in the tissue as the spinal needle is advanced (**Figure 174-11**). Adjust the needle and the US transducer side to side as needed to keep the needle in the same plane as the US beam. Continue to advance the spinal needle into the bladder (**Figures 173-15** and **174-7**). Continue to advance the spinal needle toward and into the inflated balloon. Puncture the balloon with the spinal needle. The balloon will deflate with a dramatic "pop." Remove the spinal needle. Remove the Foley catheter. Inspect the balloon to make sure there are no obvious missing fragments that are retained in the bladder.

Other options include transperineal cuff puncture, blind transabdominal cuff puncture, or injection of an organic agent (e.g., ether, acetone, mineral oil, or toluene) through the inflation port to dissolve the cuff. Occasionally, a narrow gauge guidewire may be placed through the cut balloon port to loosen any concretions that may have formed and allow the cuff to deflate. Obtain a plain radiograph of the pelvis to determine if the catheter is knotted within the bladder.[40,41] This will require cystoscopic manipulation to unknot the catheter. These techniques to reduce the cuff should be performed in consultation with a Urologist.

INTERMITTENT SELF-CATHETERIZATION

Some patients use self-catheterization at home to empty their bladder. The PerfIC Cath (Adapta Medical, Colorado Springs, CO) is better than the red rubber catheter or reusable catheters. It was designed for patients with limited dexterity including patients with diabetes, multiple sclerosis, spina bifida, spinal cord injury, and stroke. It can also be used by patients with neurogenic bladders,

prostate disorders, or normal dexterity. It is in easy-to-open packaging. The closed system was designed to reduce the risk of infection. Regardless of the system used for intermittent self-catheterization, make sure the patient does it correctly (**Table 173-1**).

SUMMARY

Although urethral catheterization is one of the most routinely performed urologic procedures, it is not a benign process and should be attempted with mindfulness. A working knowledge of genital and perineal anatomy along with a thorough clinical history and physical examination is paramount to successful catheter placement and avoidance of complications. Liberal amounts of lubrication and topical local anesthetic should always be used when inserting the catheter. Patients requiring chronic indwelling catheters should be versed in catheter care and have routine medical evaluations for catheter change and follow-up.

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174 Suprapubic Bladder Aspiration

Richard Dean Robinson, Teresa Proietti, and Andrew Shedd

INTRODUCTION

Suprapubic bladder aspiration is the insertion of a needle through the anterior abdominal wall and into the bladder to obtain a urine specimen. It is performed to either temporarily relieve urinary retention when the bladder outlet is obstructed and one is unable to place a transurethral catheter or to obtain a sterile urine sample for urinalysis.[1-10] It is most commonly performed in children under the age of 2 years as part of the septic work-up.[7] The procedure is quick,

simple to perform, safe, and has a low rate of complications. The main advantage of suprapubic bladder aspiration is that it bypasses the urethra and minimizes the risk of obtaining a contaminated urine specimen.

Urinary sampling remains the cornerstone for the diagnosis of many disease processes. Suprapubic bladder aspiration is a viable option, both therapeutically and diagnostically, when the usual means of urine collection or bladder drainage is not possible or preferable. The technique can yield an uncontaminated urine sample without urethral or skin flora contamination.

ANATOMY AND PATHOPHYSIOLOGY

The urinary bladder of the neonate and infant begins as an abdominal organ (**Figure 174-1*A***). As the child grows, the pelvis enlarges and the bladder migrates down into the bony pelvis. The bladder eventually assumes a retropubic position that is maintained throughout life (**Figure 174-1*B***).

The anatomic knowledge required to perform this procedure is minimal. The pubic symphysis is in the midline and forms the anterior border of the bony pelvis. The bladder resides posterior and superior to the pubic symphysis in the young child. The needle will pass through the skin and subcutaneous tissue of the lower abdominal wall, the rectus sheath, the peritoneum, and the bladder wall.

The adult urinary bladder resides behind the pubic symphysis and has both retroperitoneal and intraperitoneal attachments (**Figure 174-1*B***). A working knowledge of this anatomy makes percutaneous bladder manipulation both safe and possible. The rectum lies just inferior and posterior to the urinary bladder. The bladder is attached to the anterior abdominal wall by the urachus which is a fibrous chord.[11] The posterior surface of the bladder is lined with the parietal peritoneum.[11] The bladder dome has peritoneal attachments and access in this area carries the potential for bowel injury and intraperitoneal bladder perforation. These relationships must be kept in mind when attempting percutaneous access of the bladder.

Multiple major vascular structures, including the common iliac and hypogastric vessels, reside in the bony pelvis alongside the bladder. These structures are lateral to the bladder and eccentric (i.e., off the midline) percutaneous access may result in iatrogenic injury and hemorrhage.

INDICATIONS

Suprapubic bladder aspiration is the preferred method of urinary sampling and drainage in instances where voided specimens are undesirable or unattainable and when urethral catheterization is not technically possible or is contraindicated.[1-9,12,13] It offers a means of obtaining an uncontaminated urine sample from the bladder. Obtaining a urine sample by urethral catheterization in the neonate or young child may be technically difficult in which case suprapubic aspiration is an alternative. Although urethral catheterization may be an easier method of urine collection in the child or adult, suprapubic aspiration may be required to isolate intravesicular infections, to rule out contamination with asymptomatic bacteriuria, or in cases of urinary retention from a phimosis. The procedure may also be performed to temporarily relieve acute urinary retention of a nonphimotic etiology.

CONTRAINDICATIONS

The single most important aspect of suprapubic bladder manipulation is the presence of an identifiable and distended urinary bladder. The bladder can be identified by palpation, percussion, transillumination, and ultrasonography. Under no circumstances should "blind" percutaneous access be attempted if the bladder is not palpable or visualized with the aid of ultrasonography.

Patients with a coagulopathy are at an increased risk for significant hemorrhage from any percutaneous procedure including suprapubic bladder aspiration. Any coagulopathy, bleeding diathesis, platelet dysfunction, and/or thrombocytopenia should be corrected prior to performing this procedure.

Other contraindications to bladder aspiration exist. The peritoneal cavity has been violated and the bowel may be displaced more caudally and to the level of the urinary bladder in individuals with prior lower abdominal surgery or traumatic injury. The bowel may be adhesed to the anterior abdominal wall. This heightens the risk of inadvertent entry into the peritoneal cavity and bowel injury. It should not be performed in patients with bladder cancer or subcutaneous vascular grafts in the suprapubic region. The return of ascitic fluid may lead the Emergency Physician to a false sense of security when the catheter is actually intraperitoneal. Urine leakage in patients with a urinary tract infection may result in bacteremia, peritonitis, and/or sepsis. An uncooperative patient will require

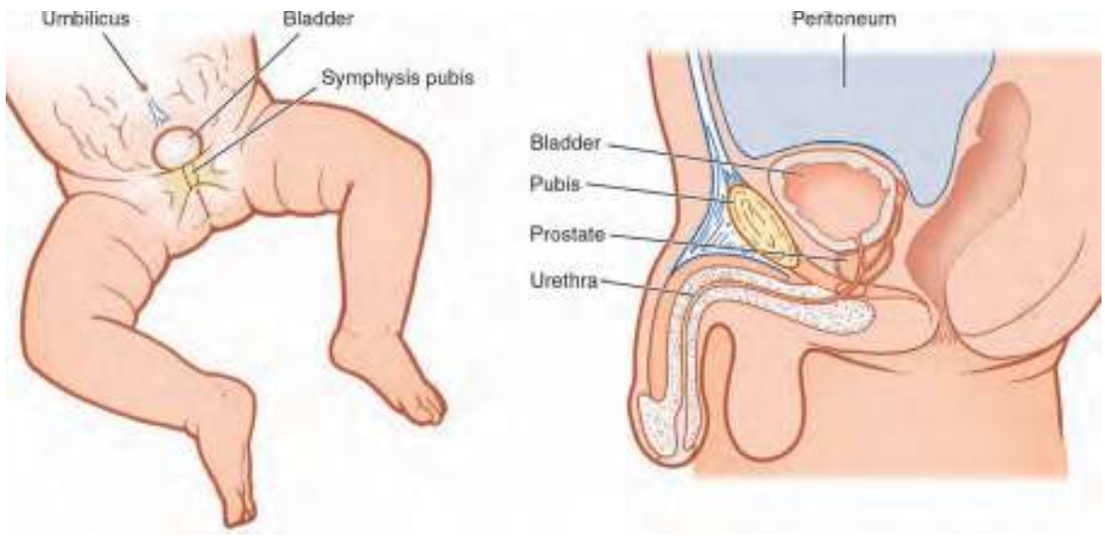


FIGURE 174-1. Position of the bladder. ***A.*** The bladder is an abdominal organ in the neonate and infant. ***B.*** The bladder is a pelvic organ in the older child, adolescent, and adult.

parenteral sedation or procedural sedation (Chapter 159) prior to performing this procedure. Any extremity contractures, physical alterations, spinal deformities, truncal obesity, or other conditions that would preclude the patient from lying supine and inhibiting bladder palpation are also relative contraindications to performing a percutaneous cystostomy.

Skin infections of the abdominal wall overlying the puncture site are a contraindication to bladder aspiration. Abnormalities in genitourinary anatomy or enlargement of pelvic structures (e.g., ovarian cysts or uterine fibroids) increase the chance of complications with bladder aspiration. Known neoplastic processes of the lower genitourinary tract heighten the possibility of complications. Distention or enlargement of the abdominal viscera can increase complication rates. The use of ultrasound (US) guidance may decrease associated risks in these situations. US directly visualizes the needle tip in the bladder.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile gloves
- 22 to 24 gauge needle or spinal needle, 1½ to 3 inches long for neonates, infants, and young children
- 22 to 24 gauge needle or spinal needle, 3 inches long for older children, adolescents, and adults
- 10 mL syringe
- Injectable local anesthetic solution, most commonly 1% lidocaine
- 4×4 gauze squares
- 25 gauge needle and 3 mL syringe for anesthetic administration
- Sterile towels or drapes
- Specimen containers for urine analysis and culture
- Bandage
- US machine (recommended)
- Sterile US gel
- Sterile US transducer covers
- 5 to 10 MHz linear US transducer for neonates, infants, and children
- 2 to 5 MHz curvilinear abdominal US transducer for adolescents and adults

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

It is important to identify the distended bladder by palpation, percussion, transillumination, or ultrasonography. Transillumination of the full bladder may be conducted in the neonate. Place the patient supine (**Figure 174-2**). Ultrasonography should be used even when the bladder is readily palpable. US can identify the bladder, quantify the amount of urine in the bladder, guide needle placement, and increase the success of the procedure.[13-16] If the bladder is distended, it appears on US as an anechoic (i.e., black) rectangular or triangular structure just below the abdominal musculature depending on the transducer orientation.[6] Identify and mark the skin location immediately above the bladder where it is closest to the skin.

FIGURE 174-2. The frog-leg position. ***A.*** The infant. ***B.*** The older child.

Prepare and drape the abdomen in a sterile fashion from the umbilicus to the pubis. Clean the skin of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. Apply sterile drapes. Prepare the US by placing the sterile US transducer cover on the selected transducer. Inject a local anesthetic agent, usually 1% lidocaine, to create a subcutaneous wheal in the area of the intended skin puncture site. Prior teaching was that infants did not need local anesthesia. Recent research has found suprapubic bladder aspiration to be a painful procedure for neonates.[11,17]

TECHNIQUES

NEONATES & INFANTS

Have an assistant place and hold the neonate or infant supine in the frog-leg position (**Figure 174-2*A***). Identify the needle insertion site in the midline and 2 cm cephalad to the pubic symphysis (**Figure 174-3**). The use of US is recommended to assist in determining the proper needle insertion site. Inject 1 mL of local anesthetic solution subcutaneously and into the abdominal wall musculature at the needle insertion site.

Place a 22 or 24 gauge spinal needle onto a 10 mL syringe. Occlude the urethra to prevent reflexive micturition by applying manual

FIGURE 174-3. Anatomic landmarks for suprapubic bladder aspiration in the neonate and infant. A line is drawn from the umbilicus to the pubic symphysis (dotted line). The intersection of the line with the suprapubic crease is the landmark for insertion of the needle.

pressure to the urethral meatus of the female (**Figure 174-4*A***) or the glans penis of the male (**Figure 174-4*B***). Insert the needle through the anesthetized skin and at a 70° angle from the skin (**Figure 174-4**). Advance the needle cephalad while applying negative pressure to the syringe. Stop advancing the needle when urine is aspirated. On US, the bladder will appear to tent as the needle pierces its anterior wall.[6] If no urine is aspirated, withdraw the needle to the subcutaneous tissue and redirect it to an 80° angle from the skin. If the procedure is unsuccessful on the second attempt, delay further attempts until the bladder is more distended, consult a Urologist, or obtain urine through another method.[18-22] After urine is obtained, remove the needle and apply a bandage to the skin puncture site.

OLDER CHILDREN

The positioning of the older child is similar to that of the neonate or infant (**Figure 174-2*B***). The procedure is similar to that of the infant although it is more important to identify the location of the distended bladder to assure successful aspiration. The use of US is recommended to assist in determining the proper needle insertion site. **The urinary bladder of the older child may be in the abdomen or may have migrated into the pelvis.** The technique is similar to neonates and infants if the bladder is in the abdomen or that of adolescents and adults if the bladder is in the pelvis.

ADOLESCENTS & ADULTS

Place the patient supine. Identify the bladder by palpation or ultrasonography. Identify the needle insertion site in the midline and 2 to 4 cm cephalad of the pubic symphysis. Inject 1 to 3 mL of local anesthetic solution subcutaneously and into the abdominal wall musculature at the needle insertion site.

Place a 3 inch, 22 to 24 gauge spinal needle onto a 10 mL syringe. Insert the needle through the anesthetized skin and at a 60° angle to the skin of the abdominal wall (**Figure 174-5**). Advance the needle caudally while applying negative pressure to the syringe. Stop advancing the needle when urine is aspirated. If no urine is aspirated, withdraw the needle to the subcutaneous tissue and readvance it in a slightly different direction (e.g., 50° to the skin of the abdominal wall). After urine is obtained, remove the needle and apply a bandage to the skin puncture site.

ULTRASOUND GUIDANCE

Bedside US imaging can ensure there is sufficient urine to aspirate, confirm the anatomic location of the bladder, help guide needle placement, and decrease complications.[23-29] It is especially helpful

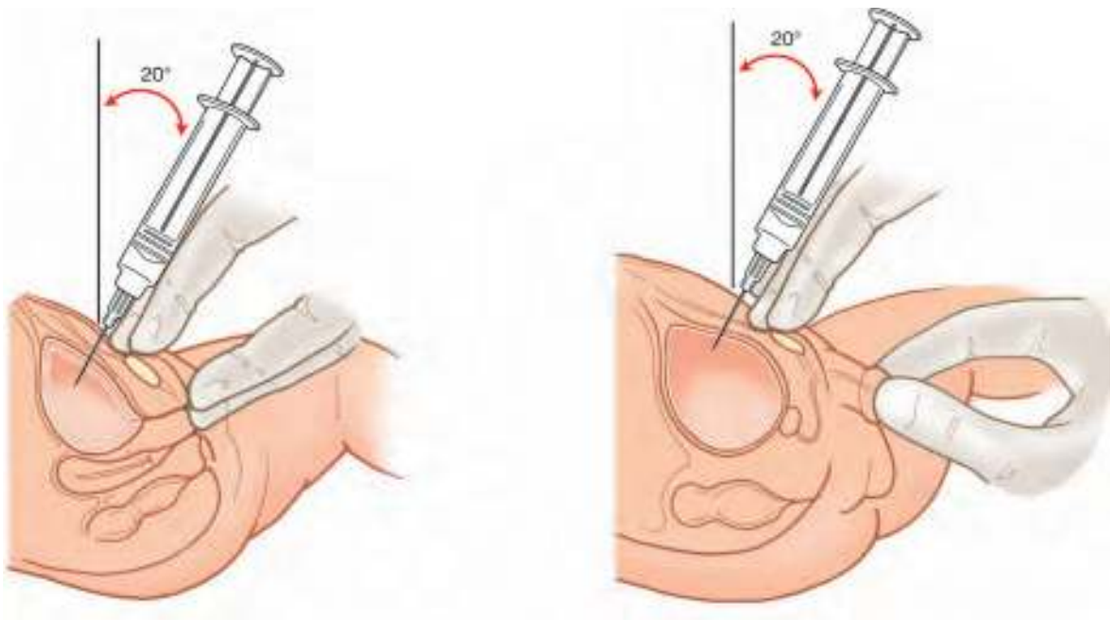


FIGURE 174-4. Suprapubic bladder aspiration in the neonate and infant. ***A.*** Digital pressure on the urethral meatus will prevent micturition in the female. ***B.*** Digital pressure on the glans penis will prevent micturition in the male.

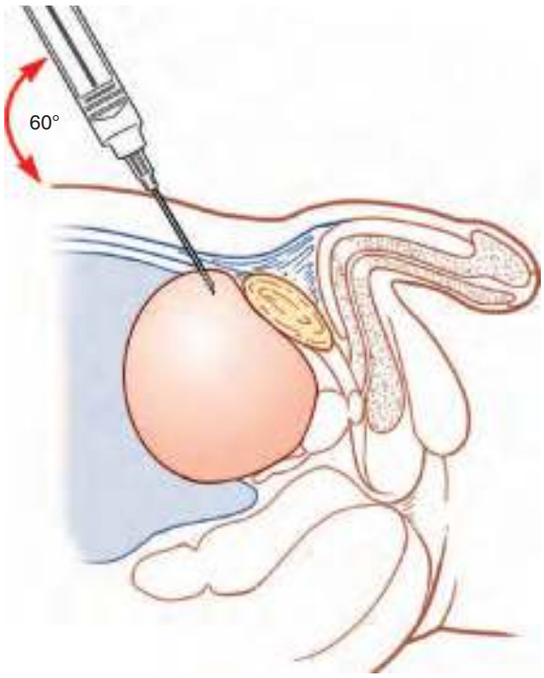


FIGURE 174-5. Suprapubic bladder aspiration in the adolescent and adult.

in infants and toddlers who have smaller bladders. There are no contraindications to using ultrasound other than those for the procedure itself. For adults, a general-purpose curvilinear abdominal or phased-array transducer provides the best combination of penetration and field of view. A linear transducer could be used for children.

Either a static or dynamic (i.e., real-time) technique can be used. The static technique involves visualizing the bladder to confirm its size, bladder location, and needle insertion site. However, the actual needle insertion is then done blindly. The dynamic technique is preferred as it allows real-time visualization of the urinary bladder, needle, and surrounding structures while the needle is advanced. The dynamic technique is not only safer, but also increases the success rate for small bladders with minimal urine. The dynamic technique can be further divided into two approaches, out-of-plane and in-plane, depending on the orientation of the US transducer to the needle.

OUT-OF-PLANE US TECHNIQUE

The out-of-plane US technique involves obtaining a transverse view of the bladder while advancing the needle in the sagittal plane of the patient's body (**Figure 174-6**). Because it is perpendicular to the transducer, the needle will be seen in its short axis on the US monitor and appear as a hyperechoic dot. The benefit of this technique is that the needle does not have to be perfectly aligned with the transducer and you are able to see structures adjacent to the bladder bilaterally. The downside to this technique is that it cannot always reliably determine where the tip of the needle is because the needle will appear as a "dot" on the US monitor regardless of which part of the needle is in view (**Figure 174-7**).

To perform the out-of-plane technique, first image the bladder to ensure there is sufficient urine to aspirate. The bladder will appear black or anechoic (**Figure 174-8**). Prepare the skin, apply sterile US gel to the patient's skin, and apply a sterile US transducer cover. Stand to the side of the patient so that your dominant hand is cephalad (**Figure 174-9**). Position the US monitor on the

FIGURE 174-6. The spinal needle is inserted under the cephalic side of the transversely oriented US transducer and angled caudally. Note, the US transducer cover and sterile gloves have been removed for clarity and should be used during the procedure.

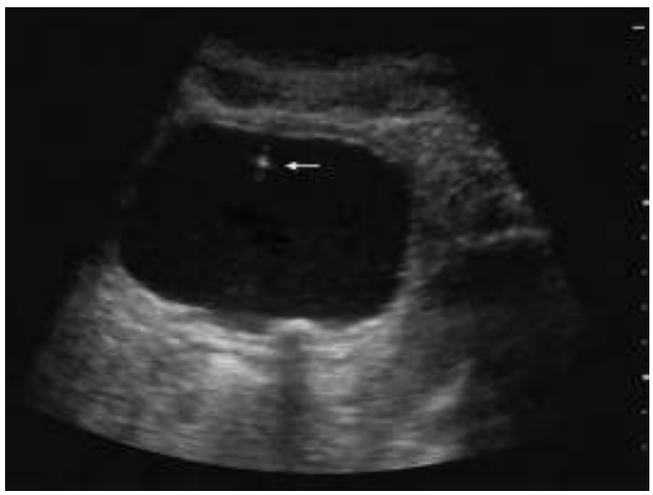


FIGURE 174-7. Transverse US appearance of the spinal needle. The hyperechoic needle tip (arrow) is visible with a small ring-down (bright shadow) artifact immediately below it.

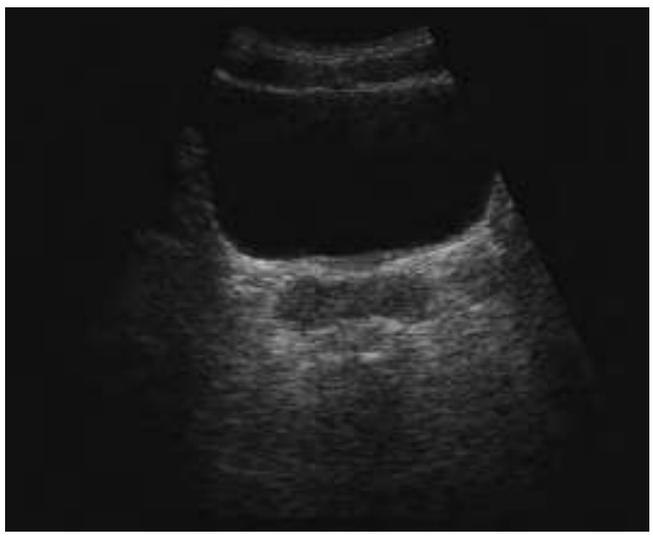


FIGURE 174-8. Transverse US view of the bladder.

FIGURE 174-9. The US machine is positioned in the same sight-line as the procedure. The physician's dominant (right) hand is cephalad and controls the needle. Note, the US transducer cover and sterile gloves have been removed for clarity and should be used during the procedure.

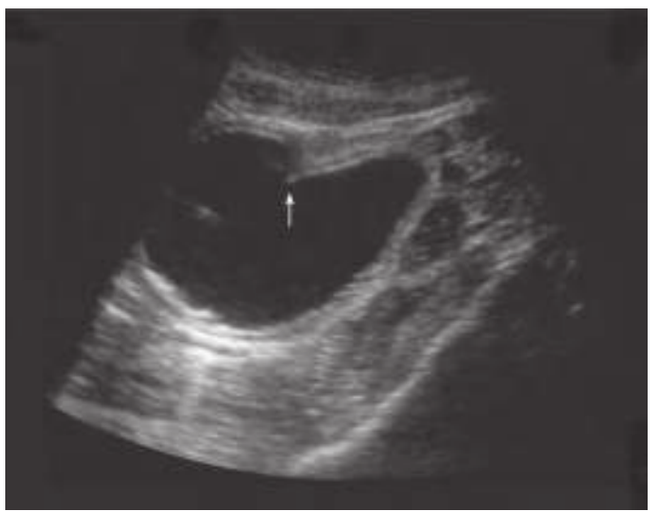
opposite side of the patient so that it is easy to see (**Figure 174-9**). Place the transducer in a transverse orientation just above the pubic symphysis. Angle the transducer caudally to view the largest anterior-posterior diameter of the bladder. Move the transducer left and right on the patient as needed to center the bladder on the US monitor.

Place the procedure needle with a 10 mL syringe in the midline of the transducer and angled caudally. The needle entry site may need to be slightly above the transducer, depending on the body habitus of the patient and depth of soft tissues superficial to the bladder, so that as it enters the bladder it traverses the plane of the US beam. Insert the needle slowly and advance it while monitoring its trajectory on the US monitor. If the needle is a small gauge and the US transducer is low frequency, the needle will not necessarily be visible in the superficial tissue planes. Look for movements in the tissue as the needle is advanced. Once the needle crosses the plane of the US beam, it will appear as an echogenic dot with a posterior repeating artifact or shadow (**Figure 174-7**). Aspirate as the needle is advanced. Aspiration should result in urine return when the needle enters the bladder. Remove the US transducer once urine is aspirated. The remainder of the procedure can proceed as previously described.

FIGURE 174-10. The spinal needle is inserted under the cephalic side of the longitudinally oriented US transducer and angled caudally. Note, the US transducer cover and sterile gloves have been removed for clarity and should be used during the procedure.

IN-PLANE US TECHNIQUE

The in-plane US technique involves obtaining a sagittal, or long axis, view of the bladder while also aligning the needle in the sagittal plane of the patient's body (**Figure 174-10**). The entire shaft of the needle will be seen on the US monitor because it is parallel to the plane of the transducer (**Figure 174-11**). The



A

B

FIGURE 174-11. Longitudinal (sagittal) US appearance of the bladder with in-plane view of the needle. ***A.*** The anterior bladder wall is tenting as the needle tip is about to penetrate the wall. The remainder of the needle is not visible within the tissues. ***B.*** The needle is within the bladder. (Courtesy of Andrew Shedd, MD.)

benefit of this technique is that the entire needle, including its tip, is visualized ensuring proper placement within the bladder. The challenge with this technique is that it requires near-perfect alignment of the needle within the narrow plane of the US transducer (**Figure 174-11**).

First image the bladder to ensure there is sufficient urine to aspirate. Prepare the skin, apply sterile US gel to the patient's skin, and apply a sterile US transducer cover. Stand to the side of the patient so that your dominant hand is cephalad (**Figure 174-9**). Position the US monitor on the opposite side of the patient so that it is easy to see (**Figure 174-9**). Place the US transducer in the sagittal plane above the pubic symphysis. Move the transducer left and right on the patient as needed to center the bladder on the US monitor. An important tip to remember is that the transducer orientation indicator is on the traditional left side of the US monitor. Orient the indicator on the transducer to the left so that transducer movements on the patient correspond to the direction of motion on the US monitor.

Place the procedure needle with a 10 mL syringe in the midline of the transducer and angled caudally (**Figure 174-10**). The needle should be parallel to and directly under the US transducer. Insert the needle and slowly advance it. Visually ensure the needle is parallel to the transducer. Adjust the transducer side-to-side as needed to visualize the needle on the US monitor. **Avoid the rectus muscles and inferior epigastric vessels.** Aspirate as the needle is advanced. The tip of the needle will be seen tenting the anterior wall of the bladder as it penetrates the bladder (**Figure 174-11*A***). The entire shaft of the needle, including the point, will be visible on the US monitor as it enters the bladder (**Figure 174-11*B***). Aspiration should result in urine return when the needle enters the bladder. Remove the US transducer once urine is aspirated. The remainder of the procedure can proceed as previously described.

ASSESSMENT

If the first attempt at aspiration is unsuccessful, withdraw the needle to a subcutaneous position and redirect it at a different angle. **This should be done only if the bladder is clearly identified.** If the procedure is unsuccessful on the second attempt, the procedure should be delayed until the bladder is more distended. Consultation with a Urologist may be necessary.

AFTERCARE

No specific care is required after performing a suprapubic bladder aspiration. **Microscopic hematuria can occur following the procedure although gross hematuria is uncommon.** The patient may complain of mild pain or soreness in the suprapubic area. This can be relieved with acetaminophen or nonsteroidal anti-inflammatory drugs. The patient, if discharged, should be given specific instructions to return immediately if they develop gross hematuria, abdominal pain, fever, nausea, vomiting, or an infection at the puncture site.

COMPLICATIONS

Numerous complications can be associated with suprapubic bladder aspiration.[1-32] Fortunately, these are rare occurrences. Bowel perforation, intraabdominal visceral injury, uncontrolled hemorrhage, and needle misplacement are the major complications of suprapubic bladder aspiration. Infectious complications include abdominal wall cellulitis, abdominal wall abscess, sepsis, and peritonitis. Hematomas of the abdominal wall, bladder wall, and pelvis are usually self-limited and require no treatment.

In the unfortunate situation when bowel contents or continuous blood is aspirated, the appropriate surgical consultant should be contacted. Generally, simple penetration of the bowel is considered harmless and requires no specific treatment. Observe the patient for the development of signs and symptoms of peritonitis.

To avoid complications, it is key that the bladder be clearly identified prior to inserting the spinal needle. The use of ultrasonography may aid in substantial reduction of complications arising from needle misadventure. The use of strict aseptic technique should avoid most infectious complications. Delaying the procedure for infants who have urinated within the last hour and the correction of any bleeding diathesis before performing the procedure can help avoid complications.

Do not mistake ascites as urine on ultrasonography. Ascites will outline the bowel and appears in many places around the abdomen. Fluid in the bladder has rounded borders and is localized. It is easy to lose sight of the needle tip and misdirect the needle as it advances through the tissue. Always move the US transducer to keep the tip of the needle in sight. Sometimes the needle may not be visible. Look for tissue movement.

SUMMARY

In those clinical situations when transurethral urine collection is not desired or possible, suprapubic bladder aspiration is an alternative. A thorough understanding of the anatomy of the pelvic and abdominal regions, along with a complete history and physical examination, is necessary to assure patient safety and avoid complications. This procedure is a safe and effective means to obtain urine as long as the bladder can be properly identified by palpation, percussion, or ultrasonography. Suprapubic bladder aspiration provides urine that is free from urethral contamination.

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175 Suprapubic Bladder Catheterization (Percutaneous Cystotomy)

Richard Dean Robinson, Aaron Bull, and Andrew Shedd

INTRODUCTION

Complaints involving the lower genitourinary system are among the most common urologic problems encountered by Emergency Physicians. The collection and evaluation of urine play a critical role in the process of diagnosis and treatment. Volitional voiding and transurethral urinary catheterization (Chapter 173) are the preferred methods of bladder drainage and can be accomplished in most instances. There are situations when the transurethral route is contraindicated or technically not possible and alternate methods must be explored. A percutaneous approach to urinary bladder drainage and decompression becomes the solution, offering both therapeutic and diagnostic results.[1-11] Suprapubic bladder catheterization has been used for decades as an effective means of accessing the bladder.

Suprapubic bladder catheterization, or percutaneous cystostomy, has become the treatment of choice for patients with acute urinary retention regardless of the cause. It is commonly performed in the trauma patient with a known or suspected urethral injury. The catheters are well tolerated, easy to care for, and can easily be replaced and/or removed. The placement of a suprapubic catheter into the bladder is fast and may be performed under local anesthesia. It is a relatively safe procedure but does have potential complications that are significant.

ANATOMY AND PATHOPHYSIOLOGY

Residing in the retropubic space, approximately 5 cm above the superior margin of the symphysis pubis, the adult urinary bladder has both retroperitoneal and intraperitoneal attachments. A working knowledge of this anatomy makes percutaneous bladder manipulation both safe and possible. The rectum lies just inferior and posterior to the urinary bladder. This relationship must be kept in mind when attempting percutaneous access. The bladder dome has peritoneal attachments and access in this area carries a risk of bowel injury and intraperitoneal bladder perforation. Multiple vascular structures, including the common iliac and hypogastric vessels, reside in the bony pelvis alongside the bladder. These structures are lateral to the bladder and eccentric (i.e., off the midline) percutaneous access may result in iatrogenic injury and hemorrhage.

INDICATIONS

Suprapubic bladder catheterization is indicated in cases when the transurethral route of bladder drainage or decompression is technically not possible or contraindicated.[9,12,13] This includes patients with bladder neck injuries and lesions, enlarged prostates (e.g., benign hypertrophy or cancer), iatrogenic urethral injuries, intractable urinary incontinence, obstructing urethral lesions, neurologic disease, an obstructing phimosis, palliative care, postoperation, suspected or known traumatic urethral or prostatic disruption, a urethral foreign body, urethral scarring, and/or urethral strictures. Continuous bladder irrigation can be accomplished via a combined suprapubic and transurethral route. Long-term bladder drainage is the final indication for a suprapubic bladder catheterization.[14]

CONTRAINDICATIONS

Suprapubic catheterization is absolutely contraindicated in the absence of an easily palpable and distended or ultrasonographically localized and distended urinary bladder.[9] Under no circumstances should "blind" percutaneous access be attempted. The bladder must be distended to push the bowel away from the anterosuperior surface of the bladder to avoid perforating the bowel.

Patients with a coagulopathy are at an increased risk for significant hemorrhage from any percutaneous procedure including suprapubic bladder catheterization. Any coagulopathy, bleeding diathesis, platelet dysfunction, and/or thrombocytopenia should be corrected prior to performing this procedure.

Other absolute contraindications exist. The peritoneal cavity has been violated and the bowel may be displaced more caudally and to the level of the urinary bladder in individuals with prior lower abdominal surgery or traumatic injury. The bowel may be adhesed

to the anterior abdominal wall. This heightens the risk of inadvertent entry into the peritoneal cavity and bowel injury. It should not be placed in patients with abdominal wall infections, bladder cancer, or subcutaneous vascular grafts in the suprapubic region.

There are many relative contraindications to percutaneous bladder catheterization. These include patients who have ascites, patients with a history of pelvic cancer or pelvic radiation therapy, uncooperative patients, or patients who have urinary tract infections. A history of pelvic cancer or irradiation will increase the risk of adhesions, anatomic distortion, and scarring. Attempts at suprapubic cystostomy increase the risk of peritoneal and/or bowel perforation. The return of ascitic fluid may lead the Emergency Physician to a false sense of security when the catheter is actually intraperitoneal. Urine leakage in patients with a urinary tract infection may result in bacteremia, peritonitis, and/or sepsis. In these circumstances, consult a Urologist for an open suprapubic cystostomy or Interventional Radiology for a percutaneous cystostomy using fluoroscopic or ultrasonographic guidance. An uncooperative patient will require parenteral sedation or procedural sedation (Chapter 159) prior to performing this procedure. Any extremity contractures, physical alterations, spinal deformities, truncal obesity, or other conditions that would preclude the patient from lying supine and inhibit bladder palpation are also relative contraindications to performing a percutaneous cystostomy.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile gloves
- Percutaneous cystostomy catheter kit
- Foley catheter, 14 to 16 French
- 60 mL catheter-tipped syringe
- 10 mL syringes
- 24 to 25 gauge spinal needle, 3 inches long
- #11 surgical scalpel blade on a handle
- 3–0 nylon suture
- Needle driver
- Local anesthetic solution, with or without epinephrine
- 4×4 gauze squares
- 25 gauge needle, 1 inch long
- 18 gauge needle
- Urine meter or urine leg bag
- Sterile towels
- Sterile drapes
- Tincture of benzoin
- 2 inch tape
- Ultrasound machine (recommended)
- Sterile ultrasound gel
- Sterile ultrasound transducer cover
- Curvilinear abdominal or phased-array transducer for adults
- Linear transducer for children

The percutaneous cystostomy catheter kit is commercially available and prepackaged by several manufacturers.[15] One type contains a cystostomy tube, an obturator, and connector tubing (**Figures 175-1 and 175-2**). Another is a Seldinger-type kit with peel-away sheath.

A commonly used suprapubic catheter kit is the Rutner Percutaneous Suprapubic Balloon Catheter Set as seen in **Figure 175-2**

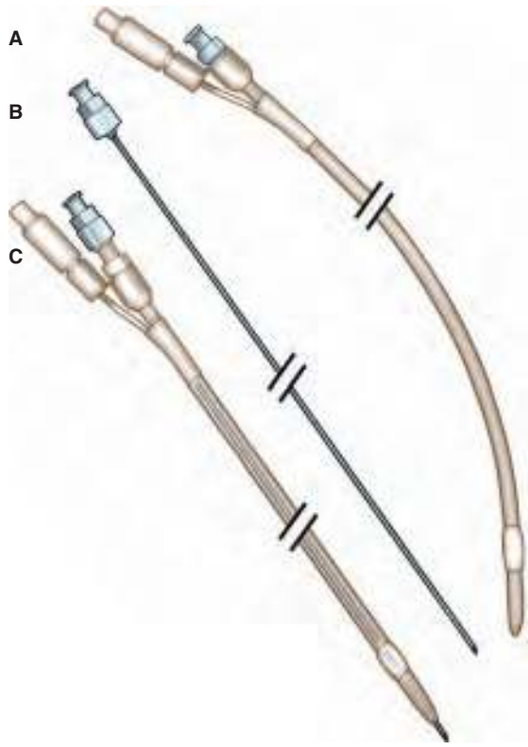


FIGURE 175-1. Schematic of a catheter and obturator system. ***A.*** The catheter. ***B.*** The obturator. ***C.*** The obturator within the catheter.

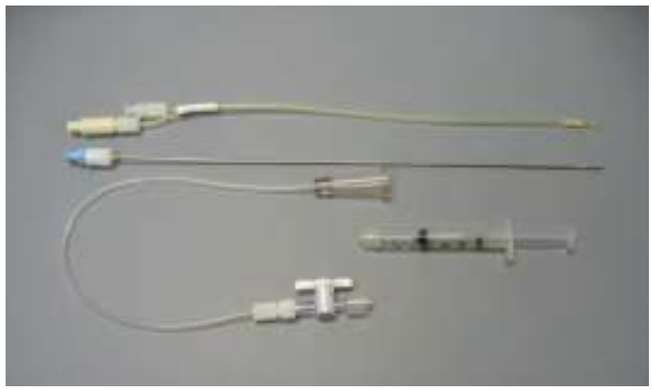
(Cook Urological Inc., Spencer, IN).[15] It consists of a 10 French catheter that is 22 cm long, a needle obturator, a connecting tube, and a 3 mL syringe (**Figures 175-1 and 175-2*A***). The proximal end of the catheter has two ports (**Figures 175-1 and 175-2*B***). One port is for the insertion of the obturator through the catheter. The obturator screws and locks into this port. The other port is an inflation port for the cuff of the catheter. A 3 to 5 mL syringe attaches to this port. The obturator is a hollow tube that tapers to a sharp point distally. When properly inserted and locked into the port, the obturator will project 2.5 mm from the distal end of the catheter (**Figures 175-1*C* and 175-2*C***). The cuff is donut-shaped and centered 1 cm from the tip of the catheter (**Figure 175-2*D***). The connector tube has a stopcock at its distal end that attaches to the catheter (**Figure 175-2*E***). The proximal end of the connector tube has a flared flange to attach to a urine drainage system.

PATIENT PREPARATION

As with all procedures, the risks and benefits of suprapubic bladder catheterization should be discussed with the patient and/or their representative. Obtain an informed consent and place it in the medical record.

Place the patient supine. Clean any dirt and debris from the abdominal wall. Shave the lower abdomen if the patient is hirsute. Identify the bladder by palpation, percussion, and/or ultrasonography. Apply povidone iodine or chlorhexidine solution to the lower abdomen, from the umbilicus to the pubis, and allow it to dry. Consider the administration of parenteral analgesics, sedatives, or procedural sedation (Chapter 159) as this is a painful procedure.

Anesthetize the abdominal wall. Fill a 10 mL syringe with local anesthetic solution. Apply a 24 to 25 gauge spinal needle onto the syringe. Identify the insertion site in the midline and 4 to 5 cm above the pubic symphysis. The use of ultrasound to verify the



A

B

C

D

E

FIGURE 175-2. The Rutner Percutaneous Suprapubic Balloon Catheter Set (Cook Urological Inc., Spencer, IN). ***A.*** The equipment. ***B.*** The proximal catheter ports. ***C.*** The distal end of the catheter with the obturator properly inserted. ***D.*** The distal end of the catheter with the cuff inflated. ***E.*** The proximal end of the connector tube contains a stopcock to attach to the catheter. The distal end has a flared flange (as seen in figure ***A***).

bladder location and to ensure that no loops of bowel are present between the abdominal wall and bladder is recommended.[9] Make a skin wheal with the local anesthetic solution at the insertion site. Insert the spinal needle at a 60° to 70° angle to the skin and aimed caudally (**Figure 175-3*A***). Advance the needle through the subcutaneous tissue, rectus sheath, and retropubic space while simultaneously aspirating and injecting 5 to 10 mL of local anesthetic solution. A loss of resistance will be felt as the spinal needle traverses the rectus sheath and enters the retropubic space. Continue to aspirate while advancing the spinal needle until the bladder is entered and urine fills the syringe. The bladder appears to tent as the needle pierces its anterior wall when ultrasonographic guidance is used.[8] **Note the needle direction and depth of insertion required to enter the bladder.**

TECHNIQUES

SELDINGER TECHNIQUE WITH A PEEL-AWAY SHEATH

This method is similar to that of placing a central venous catheter. The needle is used to locate the bladder. Place the needle on a 10 mL syringe. Insert the needle through the skin wheal of local anesthetic solution located in the midline and 4 to 5 cm above the pubic symphysis. Direct the needle caudally and at a 60° to 70° angle to the skin (**Figure 175-3*A***). **It is important to ensure that the needle enters and is advanced in the midline. This area is avascular. If the needle is paramedian it may traverse the rectus muscles and inferior epigastric vessels, resulting in significant hemorrhage.**

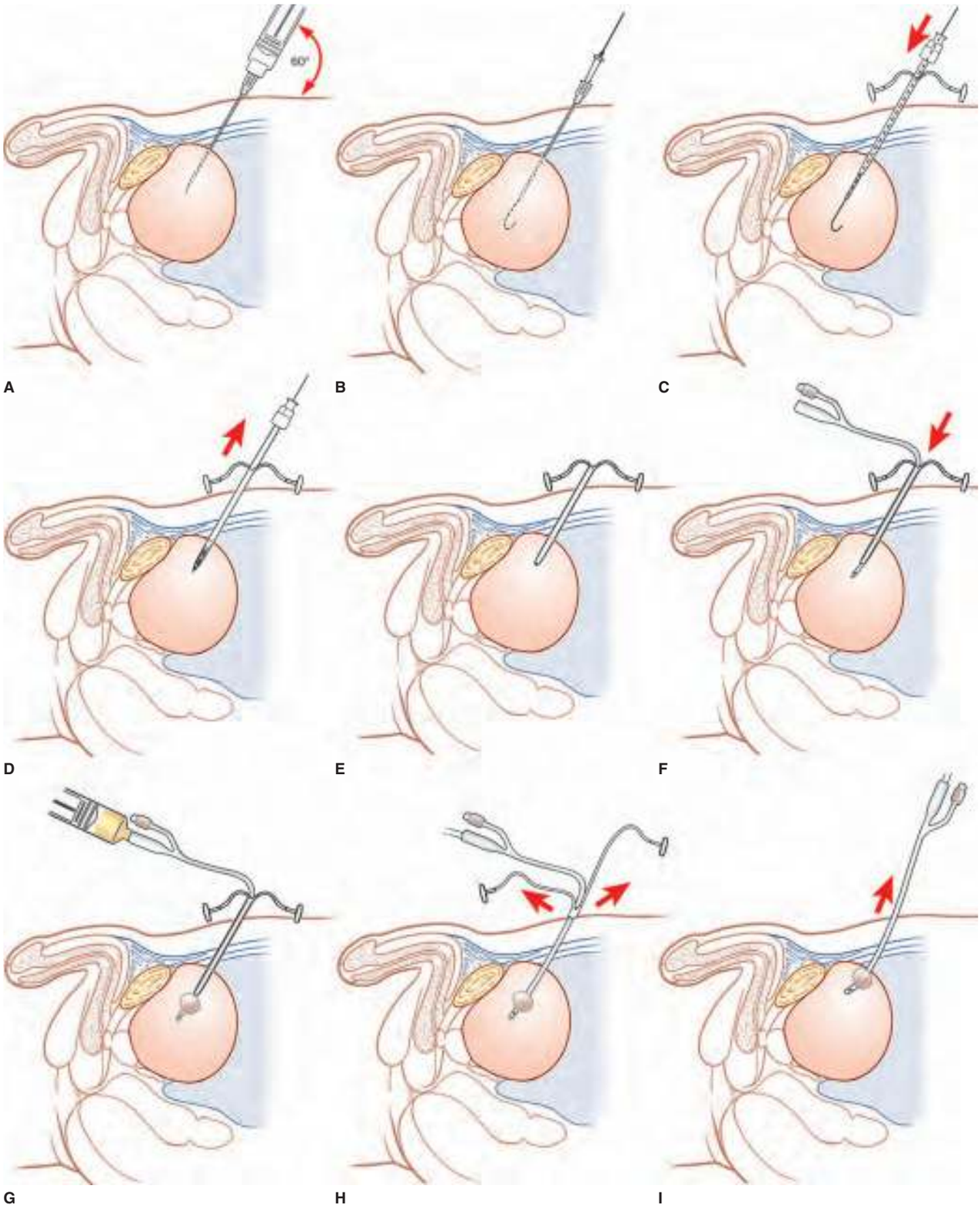


FIGURE 175-3. The Seldinger technique with a peel-away sheath. ***A.*** The finder needle is inserted 60° to 70° to the skin and advanced into the bladder. ***B.*** The syringe has been removed and the guidewire inserted through the needle. ***C.*** The needle has been removed. The dilator and peel-away sheath are inserted over the guidewire as a unit and into the bladder. ***D.*** The dilator and guidewire are removed. ***E.*** The peel-away sheath remains through the skin and into the bladder. ***F.*** A Foley catheter is inserted through the peel-away sheath and into the bladder. ***G.*** Urine is aspirated from the bladder. The cuff on the catheter has been inflated. ***H.*** The arms of the peel-away sheath are pulled upward and apart to remove the sheath. ***I.*** The cuff is lodged against the dome of the bladder.

Advance the needle caudally while applying negative pressure with the syringe. Stop advancing the needle when urine is aspirated into the syringe. Advance the needle an additional 2 to 3 cm into the bladder from the point at which urine is initially aspirated into the syringe. The aspiration of urine will confirm the proper position of the needle within the bladder.

Firmly hold the needle against the abdominal wall. Carefully remove the syringe from the needle. **Do not allow the needle to move as it may shear the bladder wall.** Advance the guidewire through the needle and into the bladder (**Figure 175-3*B***). Withdraw the needle over the guidewire while leaving the guidewire in place. Make a superficial stab incision with the #11 scalpel blade adjacent to the guidewire. This will facilitate passage of the dilator and sheath through the abdominal wall. Insert the dilator through the peel-away sheath. Advance the dilator and peel-away sheath as a unit over the guidewire and into the bladder (**Figure 175-3*C***). Remove the guidewire and dilator as a unit (**Figure 175-3*D***) leaving only the peel-away sheath (**Figure 175-3*E***).

Insert a Foley catheter through the peel-away sheath and completely into the bladder (**Figure 175-3*F***). The Foley catheter should be two sizes (i.e., 2 French) smaller than the size of the peel-away sheath. Urine should flow spontaneously from the Foley catheter. If not, insert a 60 mL catheter-tipped syringe into the Foley catheter and aspirate (**Figure 175-3*G***). The flow of urine will confirm that the catheter is properly positioned within the bladder. Inflate the cuff of the Foley catheter. Attach a urine collection system to the proximal end of the Foley catheter (**Figure 175-3*H***).

Remove the peel-away sheath. Grasp the free ends or arms of the peel-away sheath. Pull the free ends upward and opposite each other (**Figure 175-3*H***). The peel-away sheath will split in half as it is withdrawn over the Foley catheter and out of the abdominal wall. Gently withdraw the Foley catheter to ensure that the cuff is lodged against the bladder wall (**Figure 175-3*I***).

OBTURATOR TECHNIQUE

The trochar technique has been used for many years.[16] The obturator technique is a derivative of the trochar technique. Prepare the equipment. Insert the obturator through the catheter and lock it in position. Inflate the cuff with sterile saline and check its integrity. Deflate the cuff. Inflate and deflate the cuff two more times to soften the cuff. The spinal needle was previously used to infiltrate local anesthetic solution and locate the urinary bladder. This maneuver allows the operator to determine both the depth and angle needed for bladder entry.

Make a 3 to 4 mm stab incision in the midline and 4 to 5 cm above the pubic symphysis through the skin wheal of local anesthetic solution with a #11 scalpel blade. Place the tip of the obturator-catheter unit in the skin incision. **It is important to ensure that the unit enters and is advanced in the midline. This area is avascular. If the unit is paramedian, it may traverse the rectus muscles and inferior epigastric vessels resulting in significant hemorrhage.** Direct the unit caudally and at a 60° to 70° angle to the skin (**Figure 175-4*A***). Place the nondominant hand on the lower abdominal wall. Grasp the tip of the obturator-catheter unit with the thumb and index finger of the dominant hand. The hand and fingers will stabilize the unit and control the depth of insertion.

Advance the obturator-catheter unit with the dominant hand. Resistance may be felt as the trocar traverses the linea alba. Apply firm pressure to the unit to advance through the linea alba. **Do not plunge as the unit is advanced.** Continue to advance the unit through the retropubic space until resistance is felt. The tip of the trocar is now against the anterior bladder wall. Advance the unit with a short and rapid stabbing motion to enter the bladder (**Figure 175-4*A***). Urine should flow spontaneously from the proximal end of the obturator. If not, attach a syringe to the obturator and aspirate. The flow of urine will confirm that the tip of the obturator-catheter unit is properly positioned within the bladder. Advance the unit an additional 2 to 3 cm into the bladder to ensure the cuff is completely within the bladder.

Inflate the cuff of the catheter (**Figure 175-4*B***). Unscrew the obturator from the catheter. Securely hold the catheter, with the nondominant hand as it exits the abdominal wall. Remove the obturator from the catheter (**Figure 175-4*C***). Attach the stopcock at the distal end of the connector tube to the catheter (**Figure 175-4*D***). Gently withdraw the catheter to lodge the cuff against the bladder wall. Attach a urine collection system to the proximal end of the connector tube.

ULTRASOUND GUIDANCE

Bedside ultrasound (US) imaging can ensure there is sufficient urine to aspirate, confirm the anatomic location of the bladder, help guide needle placement, and decrease complications.[17-22] It is especially helpful in infants and toddlers who have smaller bladders. There are no contraindications to using US other than those for the procedure itself. For adults, a general-purpose curvilinear abdominal or phased-array transducer provides the best combination of penetration and field of view. A linear transducer could be used for children.

Either a static or dynamic (i.e., real-time) technique can be used. The static technique involves visualizing the bladder to confirm its size, bladder location, and needle insertion site. However, the actual needle insertion is then done blindly. The dynamic technique is preferred as it allows real-time visualization of the urinary bladder, needle, and surrounding structures while the needle is advanced. The dynamic technique is not only safer, but also increases the success rate for small bladders with minimal urine. The dynamic technique can be further divided into two approaches, out-of-plane and in-plane, depending on the orientation of the US transducer to the needle.

OUT-OF-PLANE ULTRASOUND TECHNIQUE

The out-of-plane US technique involves obtaining a transverse view of the bladder while advancing the needle in the sagittal plane of the patient's body (**Figure 175-5**). Because it is perpendicular to the transducer, the needle will be seen in its short axis on the US monitor and appear as a hyperechoic dot. The benefit of this technique is that the needle does not have to be perfectly aligned with the transducer and you are able to see structures adjacent to the bladder bilaterally. The downside to this technique is that it cannot always reliably determine where the tip of the needle is because the needle will appear as a "dot" on the US monitor regardless of which part of the needle is in view (**Figure 175-6**).

To perform the out-of-plane technique, first image the bladder to ensure there is sufficient urine to aspirate. The bladder will appear black or anechoic (**Figure 175-7**). Prepare the skin, apply sterile US gel to the patient's skin, and apply a sterile US transducer cover. Stand to the side of the patient so that your dominant hand is cephalad (**Figure 175-8**). Position the US monitor on the opposite side of the patient so that it is easy to see (**Figure 175-8**). Place the transducer in a transverse orientation just above the pubic symphysis. Angle the transducer caudally to view the largest anterior-posterior diameter of the bladder. Move the transducer left and right on the patient as needed to center the bladder on the US monitor.

Place the procedure needle with a 10 mL syringe in the midline of the transducer and angled caudally. The needle entry site may need to be slightly above the transducer depending on the body habitus of the patient and depth of soft tissues superficial to the bladder

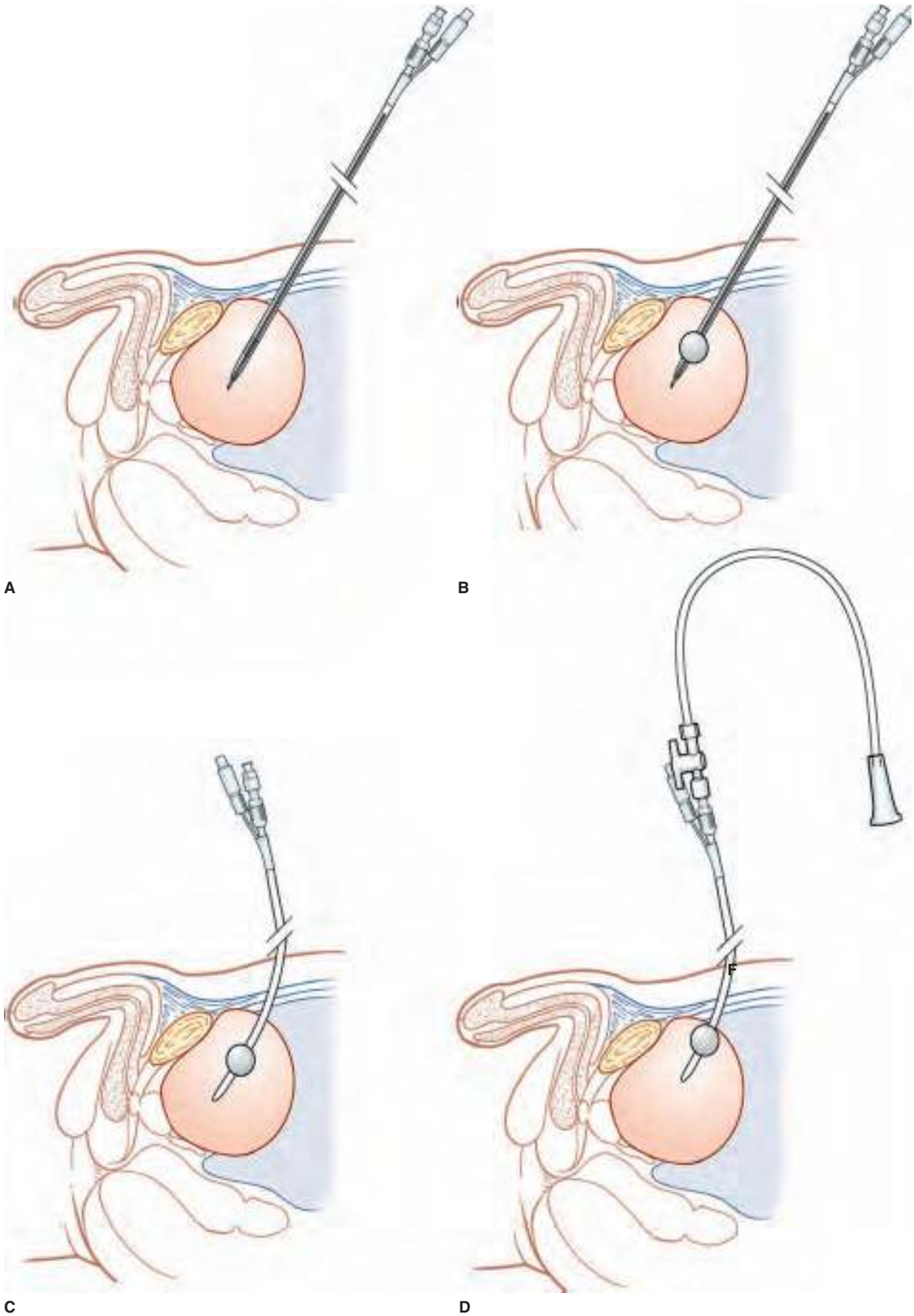


FIGURE 175-4. The obturator technique. ***A.*** The obturator is within the catheter. The system is inserted 60° to the skin and advanced into the bladder. ***B.*** The cuff is inflated. ***C.*** The obturator is removed while the catheter remains within the bladder. ***D.*** The catheter has been pulled upward and the collecting tube is attached to the catheter.

FIGURE 175-5. The spinal needle being inserted out-of-plane under the cephalic side of the transversely oriented US transducer and angled caudally. Note, the US transducer cover and sterile gloves have been removed for clarity and should be used during the procedure.

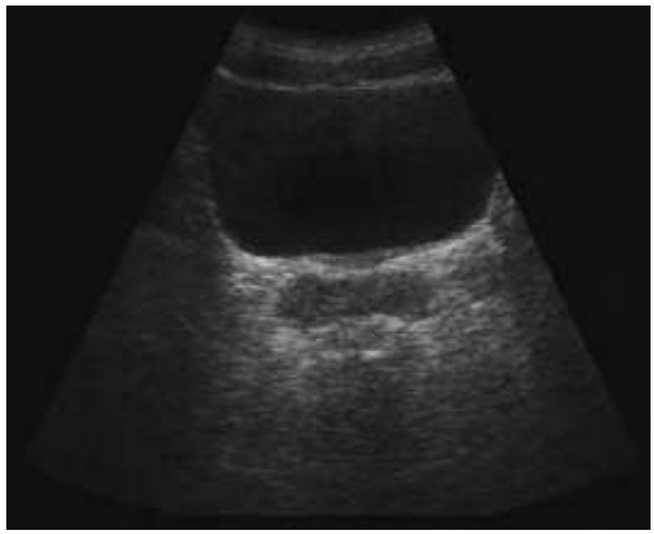


FIGURE 175-7. Transverse US view of the bladder.

so that as it enters the bladder as it traverses the plane of the US beam. Insert the needle slowly and advance it while monitoring its trajectory on the US monitor. If the needle is a small gauge and the US transducer is low frequency, the needle will not necessarily be visible in the superficial tissue planes. Look for movements in the tissue as the needle is advanced. Once the needle crosses the plane of the US beam, it will appear as an echogenic dot with a posterior repeating artifact or shadow (**Figure 175-6**). Aspirate as the needle is advanced. Aspiration should result in urine return when the needle enters the bladder. Remove the US transducer once urine is aspirated. The remainder of the procedure for the Seldinger technique can proceed as previously described. The US procedure is the same if using the obturator technique; simply substitute the catheter-obturator unit for the needle described above.

IN-PLANE ULTRASOUND TECHNIQUE

The in-plane US technique involves obtaining a sagittal, or long axis, view of the bladder while also aligning the needle in the sagittal plane of the patient's body (**Figure 175-9**). The entire shaft of the needle will be seen on the US monitor because it is parallel to the plane of the transducer (**Figure 175-10**). The benefit of this technique is that the entire needle, including its tip is visualized ensuring proper placement within the bladder. The challenge with this technique is that it requires near-perfect alignment of the needle within the narrow plane of the US transducer (**Figure 175-10**).

First image the bladder to ensure there is sufficient urine to aspirate. Prepare the skin, apply sterile US gel to the patient's skin, and apply a sterile US transducer cover. Stand to the side of the patient so that your dominant hand is cephalad (**Figure 175-8**). Position the US monitor on the opposite side of the patient so that it is easy to see (**Figure 175-8**). Place the US transducer in the sagittal plane above the pubic symphysis. Move the transducer left and right on the patient as needed to center the bladder on the US monitor.

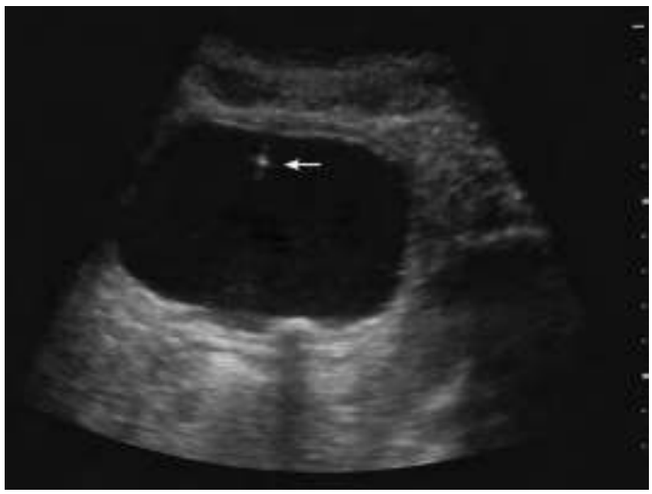


FIGURE 175-6. Transverse US appearance of the bladder and spinal needle as the spinal needle is oriented out-of-plane. The hyperechoic needle tip (arrow) is visible with a small ring-down (bright shadow) artifact immediately below it.

FIGURE 175-8. The US machine is positioned in the same sight line as the procedure. The physician's dominant (right) hand is cephalad and controls the needle. Note, the US transducer cover and sterile gloves have been removed for clarity and should be used during the procedure.

FIGURE 175-9. The spinal needle being inserted in-plane under the cephalic side of the longitudinally oriented US transducer and angled caudally. Note, the US transducer cover and sterile gloves have been removed for clarity and should be used during the procedure.

An important tip to remember is that the transducer orientation indicator is on the traditional left side of the US monitor. Orient the indicator on the transducer to the left so that transducer movements on the patient correspond to the direction of motion on the US monitor.

Place the procedure needle with a 10 mL syringe in the midline of the transducer and angled caudally (**Figure 175-9**). The needle should be parallel to and directly under the ultrasound transducer. Insert the needle and slowly advance it. Visually ensure the needle is parallel to the transducer. Adjust the transducer side-to-side as needed to visualize the needle on the US monitor. **Avoid the rectus muscles and inferior epigastric vessels.** Aspirate as the needle is advanced. The tip of the needle will be seen tenting the anterior wall of the bladder as it penetrates the bladder (**Figure 175-10*A***). The entire shaft of the needle, including the point, will be visible on the US monitor as it enters the bladder (**Figure 175-10*B***). Aspiration should result in urine return when the needle enters the bladder. Remove the US transducer once urine is aspirated. The remainder of the procedure for the Seldinger technique can proceed as previously described. The US procedure is the same if using the obturator technique; simply substitute the catheter-obturator unit for the needle described above.

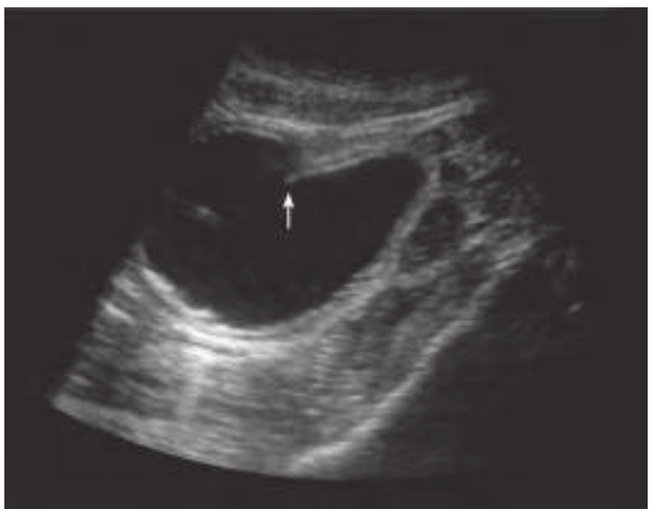
ASSESSMENT

The return of urine confirms the correct placement of the catheter into the bladder. If the catheter ceases to work after initially functioning properly, inspect the portion outside of the patient's body for kinks.[23] Flush the catheter with sterile saline to remove any potential clots. A catheter that flushes easily but cannot be subsequently aspirated suggests that the tip has been withdrawn into the perivesicular space of the pelvic cavity or that the catheter is kinked. The catheter is kinked if saline cannot be flushed through or aspirated. The catheter is properly positioned within the bladder if saline flushes and aspirates without difficulty. Consider the use of US to confirm catheter placement and the presence of urine within the bladder.

AFTERCARE

Secure the catheter to the abdominal wall (**Figure 175-11**). Place 4×4 gauze squares over the pubic symphysis to bolster the catheter as it exits the abdominal wall. Apply tincture of benzoin to the abdominal wall and allow it to dry. Tape over the catheter and gauze. The ends of the tape should be applied to the dried benzoin solution. Prophylactic antibiotics are not required unless a urinary tract infection is present.

Examine the puncture site twice a day for any signs of infection. Routine wound care should be performed at the puncture site. If removed within 7 days, the bladder wall and abdominal wall will heal without complications. A transurethral catheter should be inserted to ensure urine egress through the urethra and not the bladder wall while the suprapubic catheter tract heals. After 10 to 14 days, the tract of the catheter is epithelialized and mature. The catheter may be exchanged through the mature tract if necessary. Suprapubic tubes should not be left in place for more than 4 weeks.[9]



A

B

FIGURE 175-10. Longitudinal (sagittal) US appearance of the bladder with in-plane view of the needle. ***A.*** The anterior bladder wall is tenting as the needle tip is about to penetrate the wall. The remainder of the needle is not visible within the tissues. ***B.*** The needle is within the bladder. (Courtesy of Andrew Shedd, MD.)

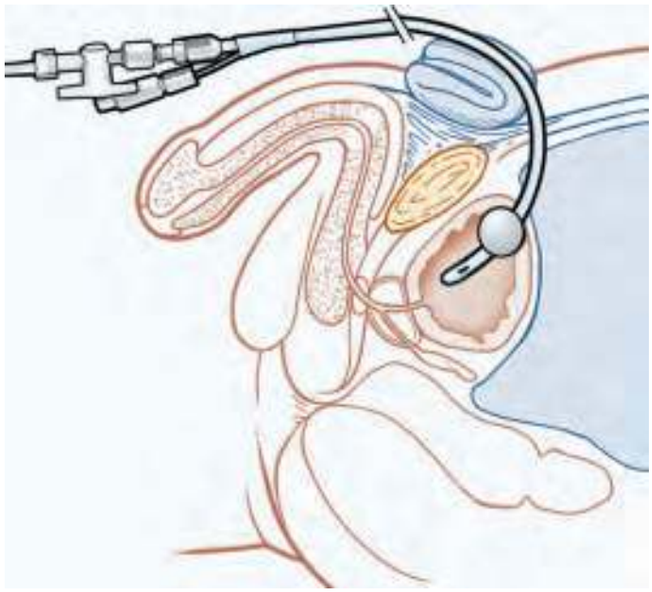


FIGURE 175-11. Securing the catheter.

COMPLICATIONS

Bowel perforation, catheter migration, catheter misplacement, intraabdominal visceral injury, peritonitis, uncontrolled hemorrhage and vascular injury are the major complications of a suprapubic cystostomy catheter placement.[1-9,24-26] Perforation of the bowel can be prevented by ensuring that the bladder is distended by palpation, percussion, or ultrasonography. Intraperitoneal perforation is more common in patients with ascites, a distended abdomen, or prior abdominal surgery. Gross hematuria is common and transient. Through-and-through perforation of the bladder can injure the rectum, vagina, and/or uterus. Other minor complications are associated with the length of indwelling catheter time and include bleeding, infections (e.g., cellulitis and abscesses), kinking, and stone formation.[23]

When catheter placement is in doubt or when a previously draining tube no longer continues to drain, simple flushing and irrigation will usually suffice. Bedside US is a quick means to confirm catheter placement. However, if concern persists, a gravity cystogram under fluoroscopy can be diagnostic. In the unfortunate situation when bowel contents or continuous blood is aspirated or flows from the catheter, the appropriate surgical consultations should be obtained.

SUMMARY

Suprapubic bladder access is the preferred alternative in those clinical situations when transurethral bladder decompression is no longer an option. Years of experience demonstrate suprapubic bladder catheterization to be an effective and relatively safe method of accessing the bladder. A prior understanding of the associated anatomy is paramount to avoidance of adverse results.

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176 Retrograde Urethrography and Cystography

Richard Dean Robinson and Sasha Michael Dib

INTRODUCTION

Urinary tract injuries may result from blunt trauma, penetrating trauma, urologic procedures, or may arise spontaneously.[1-14] Bladder injuries occur in up to 15% of pelvic fractures.[1-3] Associated urethral injuries occur in up to 11% of males and up to 6% of females.[1-3] **The role of retrograde urethrography and cystography in evaluation of the trauma patient is to rule out a partial urethral rupture,**

complete urethral rupture, and/or bladder rupture.[15-17] On initial presentation to the Emergency Department, there are clear indications for performing these procedures. The importance of proper training in these techniques must be stressed to avoid secondary injury.

The evaluation of trauma patients should include, if appropriate, an assessment of the bony pelvis and the genitourinary system. The identification of a pelvic fracture must be followed by an examination of the lower genitourinary tract to rule out an associated injury. Patients with disruption of the pubic symphysis or pubic rami or with a vertically unstable pelvic fracture have a high incidence of concomitant bladder trauma.[13] Patients with an isolated acetabulum, femur, or iliac crest fracture have a low incidence of bladder injury or rupture.[13] **The lack of a pelvic fracture does not eliminate the possibility of a bladder or urethral injury.** The most common signs seen with genitourinary tract injury are gross hematuria (82%) and abdominal tenderness (62%).[4] Other signs of genitourinary tract injury include blood at the urethral meatus, the inability to void, swelling or ecchymosis of the perineum or penis, a boggy prostate, and a high-riding prostate. An evaluation of the genitourinary tract is indicated in the presence of any of these signs. These assessments should be made early and interventions instituted immediately.

Traditional teaching suggests that urethral catheterization should be avoided if a potential injury to the bladder and/or urethra is suspected. This teaching requires performance of a retrograde urethrogram and cystogram to rule out any injuries prior to urethral catheterization. A preliminary study suggests that blind urethral catheterization, despite a potential injury, may be safe.[18] Additional large multicenter studies are required before this change in practice can be safely recommended. The guidelines do allow for a single attempt at catheter drainage if exceptional circumstances indicate a need for monitoring.[14]

ANATOMY AND PATHOPHYSIOLOGY

The lower urinary tract in males consists of the urethra and bladder (**Figure 176-1**). The urethra is divided into the fossa navicularis, the penile urethra, the bulbar urethra, the membranous urethra, and the prostatic urethra based on anatomic location. The bladder neck opens into the trigonal canal and funnels into the bladder. The male posterior urethra is 5.0 to 5.5 cm long, fixed to the urogenital diaphragm, and the area most susceptible to injury.[2,5] The female urethra is short, not rigidly fixed to the pubis or pelvic floor, mobile, and much less susceptible to injury.[3] The female urethra is equivalent to the membranous and prostatic (i.e., posterior) urethra in the male.[6]

The periurethral striated sphincter is composed of muscle from the urogenital diaphragm. This muscle layer unites with the distal smooth muscle at the intermuscular incisura. This is frequently seen on the voiding cystourethrogram and may be mistaken for a stricture or posterior urethral valves. The urogenital diaphragm surrounds the membranous urethra and may compress the urethra during voiding or on retrograde flow of contrast.[6]

The verumontanum and urethral crest protrude into the male prostatic urethral lumen and may extend into the membranous urethra. The prostatic gland ducts, prostatic utricle, ejaculatory ducts, and urethral gland ducts usually do not fill on voiding cystourethrogram; and when visible, filling usually denotes distal obstruction. These structures may also fill from aggressive injection of contrast during the retrograde urethrogram.

The anterior and posterior baseplates, of which the trigone is part, are seen as a diagonal plane sloping downward from posterior to anterior on the lateral view of the bladder. The bladder neck is visualized at the junction of the anterior one-third and the posterior two-thirds of the plane. Anteriorly the pubis abuts the baseplate. The fundus of the bladder becomes more dome-shaped as it fills with fluid. It may be compressed by the uterus or colon in the midline or on either side.[6]

INDICATIONS

The retrograde urethrogram should be employed in males presenting with blunt and penetrating trauma when there is any indication of a urethral injury. Indications include penetrating injury when involvement of the lower genitourinary tract is suspected, pelvic fractures, perineal or lower abdominal trauma with gross hematuria, vaginal lacerations, sacral spine fractures, blood at the urethral meatus, the inability to void, swelling of the perineum or penis, ecchymosis of the perineum or penis, hematoma of the perineum or penis, a high-riding prostate, or a boggy prostate.[7,8,13,19,20] Other indications include urethral strictures and obstructions, congenital abnormalities, periurethral or prostatic abscess, fistulae, and false passages. It may be indicated in females if urethral diverticula are suspected.

Retrograde cystography should follow retrograde urethrography, especially in patients who recall having a full bladder at the time of trauma and are later unable to void or have small amounts of bloody urine.[9] Retrograde cystograms are nearly 100% sensitive for

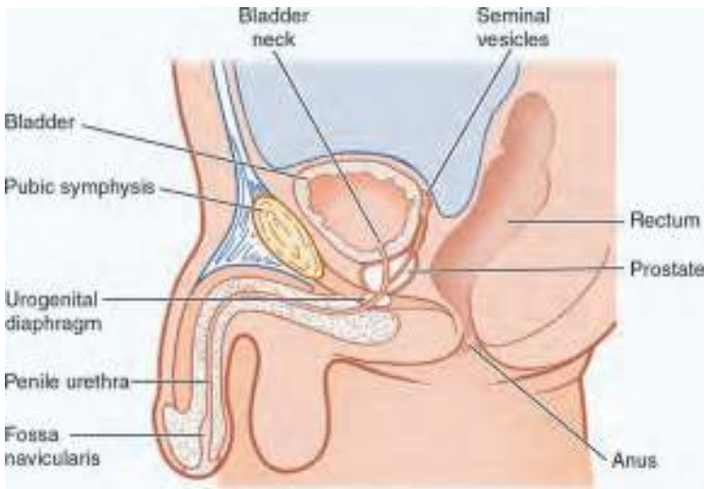


FIGURE 176-1. Anatomy of the male genitourinary tract.

detecting a bladder rupture provided that adequate distension is accomplished and that postvoid images are obtained.[13]

CONTRAINDICATIONS

There are few contraindications to retrograde urethrography and cystography. The patient's overall condition must be taken into consideration. Lifesaving procedures, such as securing an airway and stabilizing life- and limb-threatening injuries, must take precedence. Because septic shock and irreversible renal damage can occur, a relative contraindication in the setting of acute urethritis exists when the suspicion of genitourinary tract trauma is very low. **A urethral injury identified on the retrograde urethrogram is the only absolute contraindication to transurethral bladder catheterization and retrograde cystography**. Consult a Urologist if, in a patient with pelvic trauma, there is any difficulty in passing a urethral catheter into the bladder. **Do not try to advance a catheter against resistance as iatrogenic injury can result**. There is a small risk of allergic reactions to the contrast media. Patients with previous reactions should receive nonionic agents and be premedicated with corticosteroids and antihistamines.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Sterile gloves
- Sterile drapes
- Viscous lidocaine suspension
- Foley catheter, 5 to 18 French (**Table 176-1**)
- Brodney clamp
- Contrast material
- 60 mL catheter tip syringe
- Toomey syringe
- Christmas tree adaptor
- Surgical clamp
- Lead apron

A variety of contrast agents are available and may be used to perform retrograde urethrography and cystography. Agents used are specific to each institution. Most commonly used are full strength Hypaque (50% diatrizoate sodium), Renografin-60 (diatrizoate sodium), or Cystografin (18% or 30% diatrizoate meglumine). Alternatively, the same agents can be diluted with sterile saline in a 1:10 (i.e., contrast to saline) solution.

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. While a formal consent is usually not obtained since this is an emergent procedure, document in the medical record that the "risks and benefits were explained to the patient." If not contraindicated, administer parenteral sedation. Place the patient in a 45° oblique position with their left side on the bed. Flex the left leg at the knee and abduct the hip. Place a radiolucent wedge or rolled towels under the patient to maintain the oblique positioning. Completely extend the right leg. This is the ideal patient position. The degree of patient mobility and the medical condition at hand may dictate an alteration of this position. In patients with pelvic fractures, all radiographs should be taken with the patient in a supine position.

Prepare the penis and urethra. Clean any dirt and debris from around the penis. Retract the foreskin if the patient is uncircumcised. Apply povidone iodine or chlorhexidine solution to the penis and allow it to dry. Apply sterile drapes to delineate a sterile field. **The retrograde urethrogram must be performed under sterile conditions. Insert viscous lidocaine into the urethral meatus to anesthetize the urethra. Do not use lidocaine jelly as this may impede the flow of contrast material.** Allow the viscous lidocaine to remain in the urethra for 2 to 4 minutes prior to performing the procedure to provide adequate analgesia.

Obtain a flat plate or a KUB (kidney, ureters, and bladder) radiograph. This will be a baseline film for future reference. Carefully examine the radiograph for curvature of the spine, pelvic fractures, fractures of ribs 9 to 12, unilateral or bilateral loss of the normal psoas muscle shadow, and/or vertebral transverse process fractures. Any of these findings can signify a urinary tract injury. Observe and note any radiopaque material that may be present prior to the injection of contrast material.

The Emergency Physician should dress appropriately for the procedure. The Emergency Physician should wear a lead apron due to the proximity of the patient during the procedure and while radiographs are taken. Dress in a sterile gown and gloves. A cap and mask are not required.

TABLE 176-1 Estimated Foley Catheter Size Based on Patient Age

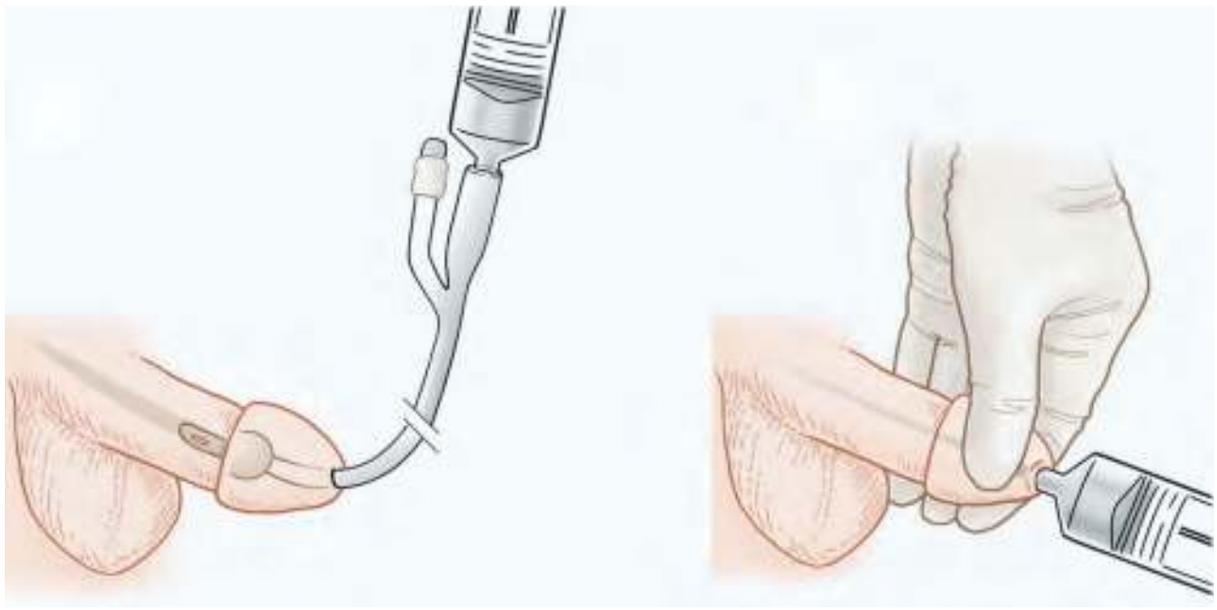
Patient age	Catheter size (French)
Premature or newborn	5
3–12 months	8
1–2 years	8–10
2–8 years	10
8–10 years	10–12
10–12 years	12
12–14 years	12–14
14+ years	16–18

RETROGRADE URETHROGRAPHY TECHNIQUES

FOLEY CATHETER TECHNIQUE

The Foley catheter technique is the preferred method. The Foley catheter causes little or no discomfort, is flexible, and the patient may be able to move if necessary. **Ensure there is no leakage of contrast from the urethra and onto the patient which can cause radiographic artifacts thereby making interpretation difficult.** An advantage to using the Foley catheter is that it can subsequently be advanced into the bladder to perform the cystogram. Newer types of catheters have two cuffs, one at the tip and the other near the balloon inflation hub of the catheter. The proximal cuff will expand when the pressure in the distal cuff exceeds the maximum safe pressure during cuff inflation. This allows excess pressure to be directed away from the distal cuff minimizing iatrogenic urethral injury. There are some disadvantages to this method. The inflated cuff may conceal an injury of the distal urethra at the fossa navicularis. Air bubbles are difficult to eliminate from the catheter. This may impair the flow of contrast material or produce artifacts due to air in the urethra. Air bubbles in the Foley catheter will alter the study integrity. Prime the tubing with contrast before inserting it into the urethra to eliminate any air. Place an x-ray plate under the patient's hips and pelvis.

Insert the catheter into the urethra. Advance the catheter until the cuff is within the fossa navicularis (**Figure 176-2*A***). Inflate the cuff with approximately 3 mL of sterile saline or to the point that it is snug and does not cause pain to the patient.[10] Gently straighten the urethra by directing the tip of the penis toward the dependent



A B

FIGURE 176-2. Retrograde urethrography. ***A.*** Foley catheter technique. ***B.*** Syringe technique.

shoulder or nipple and over the thigh. Avoid traction on the penis or the Foley catheter as this tends to narrow the urethra and/or dislodge the catheter. Attach a contrast-filled syringe to the proximal end of the Foley catheter (**Figure 176-2*A***). Gently inject 50 to 60 mL of contrast material over 5 to 10 seconds.

Obtain a plain radiograph at or near the end of the injection. Have the film developed and review the image. The entire urethra should be visible in a lateral projection. Repeat the procedure if this is not achieved. Allow the contrast to drain from the urethra through the Foley catheter and into a container. Deflate the cuff and remove the catheter from the urethra. Obtain an additional plain radiograph of the pelvis as a washout image. Have the film developed and review the image for any abnormalities in the urethra. Although not necessary, fluoroscopy may aid in obtaining an adequate study.[10]

Great care should be taken so as to not allow any contrast to leak out of the catheter or urethra. Any spill of contrast material will cause distortions on the radiographs. The contrast may also cause skin irritation. It should be wiped and rinsed off the skin immediately if this occurs.

SYRINGE TECHNIQUE

If a proximal urethral injury is suspected, the alternative method is to use a 60 mL catheter-tipped Toomey syringe or a 60 mL syringe with a Christmas tree adaptor. Because it is not flexible and cannot be fixed inside the penis, the syringe must be held in place at all times during the procedure. The penile shaft must also be held to secure the tip of the syringe inside the urethra. There is a significant chance of contrast leakage from the urethra using this technique.

Clean, prep, and drape the penis as described previously. Place an x-ray plate under the patient's hips and pelvis. Grasp and cradle the patient's penis with the nondominant hand. Insert the tip of the contrast-filled syringe into the urethra. Firmly squeeze the glans penis between the thumb and the index and long fingers (**Figure 176-2*B***). This will secure the catheter tip within the fossa navicularis. Do not squeeze the shaft of the penis with middle, ring, or little fingers as this can occlude the urethra. Gently straighten the urethra by directing the tip of the penis toward the dependent shoulder or nipple and over the left thigh. The remainder of the procedure is as described above in the Foley catheter section.

BRODNEY CLAMP TECHNIQUE

The Brodney clamp is a cage with rubber feet that clamp behind the corona of the glans (**Figure 176-3**).[10] In the center of the device, there is a blunt-tipped obturator that inserts into and occludes the urethral meatus. It may be used instead of a Foley catheter or 60 mL syringe for the procedure. The advantage of this device is that both the fossa navicularis and the distal urethra are visible on the radiograph. Air bubbles are easily eliminated from the obturator. The disadvantages of the clamp are that it is not flexible and it is heavy. It must be held during the procedure and fluoroscopy is impossible. If the patient moves, the clamp tends to dislodge. The clamp is difficult to use in older children, adolescents, and adults and in the presence of a phimosis.[10] Brodney clamps are usually not stocked in the Emergency Department and may be difficult to obtain in a timely manner.

FIGURE 176-3. The Brodney clamp.

Clean, prep, and drape the penis as mentioned previously. Insert the blunt-tipped obturator of the clamp into the urethra. Rotate the feet of the clamp so that they are grasping the coronal sulcus of the penis. These should not be so tight as to occlude the urethra ventrally. The remainder of the technique is as described in the Foley catheter section.

RETROGRADE CYSTOGRAPHY TECHNIQUE

Any abnormalities in the retrograde urethrogram, such as extravasation of contrast from the urethra or evidence of strictures, should prompt an urgent consult by a Urologist. If the study is normal, the retrograde cystogram should be performed. Gently advance and fully insert a Foley catheter into the bladder (**Figures 176-4*A* and 176-4*B***). Inflate the cuff at the tip of the Foley catheter. Gently pull on the Foley catheter to lodge the cuff at the bladder neck (**Figure 176-4*C***). Attach a 60 mL catheter-tipped syringe, without the plunger, to the Foley catheter. Pour contrast material into the syringe and let it drain by gravity to overdistend the bladder.

Continue to allow contrast to fill the bladder until 300 to 400 mL of contrast material is in the bladder of an adult or any child older than 11 years old. In younger children, the instilled volume is estimated in mL by the formulas weight (kg) × 10 for children < 1 year of age, (age in years + 2) × 30 for children > 1 year of age, or to the point of initiating a bladder contraction.[11] If a bladder contraction occurs, refill the bladder to the volume that initiated the contraction and clamp the Foley catheter. Volumes less than these can result in a false-negative study. Clamp the Foley catheter after the contrast is instilled with a hemostat.

Obtain two plain radiographs. These are the anteroposterior (AP) and the oblique, or lateral, views of the pelvis. Evaluate the radiographs for proper bladder filling with contrast and for extravasation of contrast. Release the hemostat and allow the contrast to drain out of the bladder through the Foley catheter and into the container. Obtain another AP radiograph of the pelvis as a washout film. This film is often helpful in picking up obscured areas of extravasation not visible on the initial film. Review the radiographs for intraperitoneal and extraperitoneal bladder rupture.

COMPUTED TOMOGRAPHY (CT) CYSTOURETHROGRAPHY

CT retrograde urethrography and cystography are variations on conventional plain film cystourethrography.[14,21] Using CT imaging may be an alternative in trauma patients requiring a CT scan of the pelvis and may reduce the time for patient evaluation. Plain film and CT imaging of the bladder are both considered appropriate methods in the evaluation of suspected bladder injury.[22,23] Sensitivity and specificity for bladder rupture have been demonstrated to be comparable between plain film and CT modalities.[14,24]

Performing a CT urethrogram is similar to the Foley catheter technique described previously. Place the Foley catheter into the distal urethra, inflate the balloon, and inject 10 mL of contrast material. Clamp the Foley catheter. Obtain a thin-cut CT scan through the pelvis, perineum, and penis. Instruct the CT technician to run a computerized three-dimensional reconstruction of the CT scan. Evaluate the CT reconstruction for contrast extravasation, urethral occlusion, and/or urethral narrowing.

To perform a CT cystogram, insert the Foley into the bladder, inflate the balloon, and instill 300 to 400 mL of contrast solution into the bladder. One approach is to use a gravity feed method by suspending contrast 3 to 4 feet above the patient just prior to and during imaging. An alternative is a CT of the abdomen and pelvis while obtaining complete views of the bladder.[24]

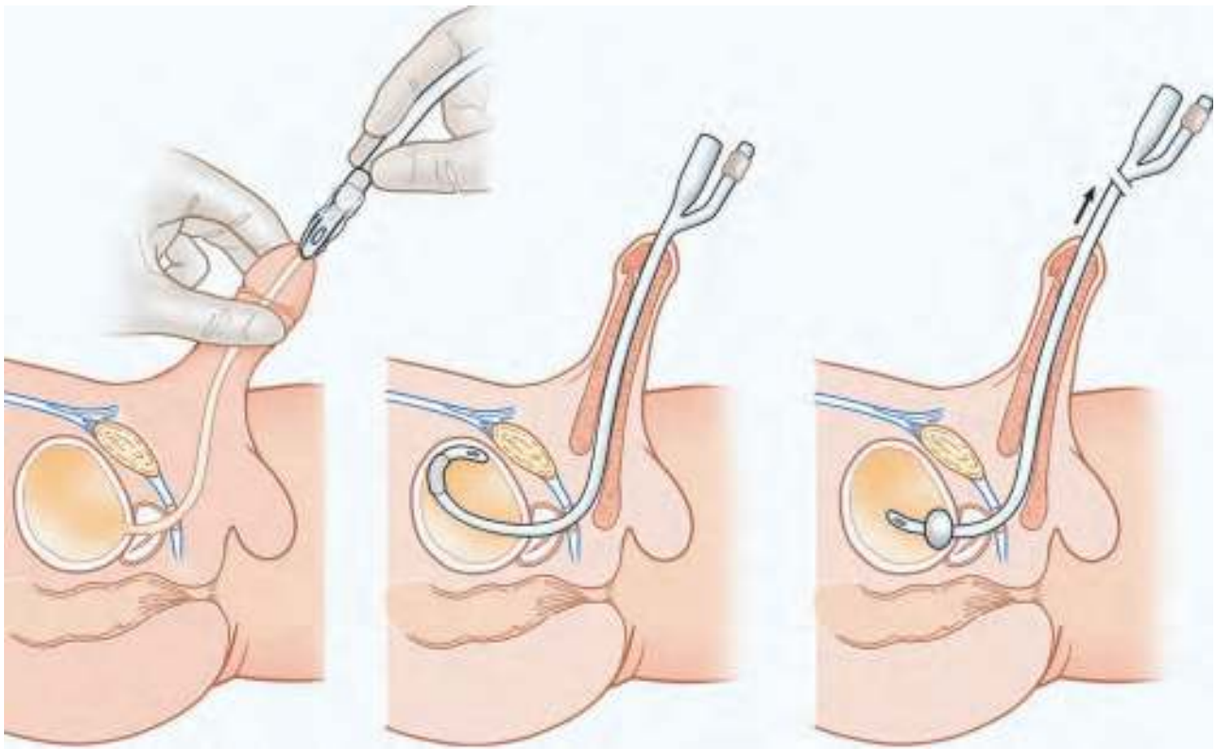


FIGURE 176-4. Retrograde cystography. ***A.*** The Foley catheter is inserted into the penis. ***B.*** The Foley catheter is advanced until it is inside the urinary bladder. ***C.*** The cuff is inflated. The catheter is then pulled until resistance is met to occlude the urethra with the cuff.

FIGURE 176-5. An extraperitoneal injury. Contrast extravasation from the region of the membranous urethra to the prostatic urethra and below the bladder neck. Note the femoral fracture, pelvic fractures, and the bladder filling representing a partial urethral disruption. (Courtesy of Eric F. Reichman, PhD, MD.)

FIGURE 176-6. The cystogram. The pear-shaped bladder is elevated from the pelvic floor by a hematoma seen on CT scans. Note the femoral fracture and the pelvic fractures. (Courtesy of Eric F. Reichman, PhD, MD.)

ASSESSMENT

In a normal retrograde urethrogram, the entire urethra should be visible. Some of the contrast material should also be seen within the bladder. Extravasation of contrast from the urethra will appear as a flame-like density outside the urethra (**Figure 176-5**). A partial urethral disruption will show extravasation of contrast as well as contrast within the bladder (**Figure 176-5**). An image where no contrast material is visible within the bladder is indicative of a complete urethral disruption. Contrast material may occasionally be seen in the venous plexus of the penis due to forceful injection of contrast into the urethra. **This is a common phenomenon and should not be mistaken for extravasation.** The venous flush will clear spontaneously on a postvoid film.

The evaluation of the retrograde cystogram can be performed after a plain film or CT scan (**Figure 176-6**). Intraperitoneal and extraperitoneal bladder injuries can be differentiated. **This differentiation is important because each requires a different treatment.**[25] Intraperitoneal bladder injuries usually result from a direct blow to the abdomen with a distended bladder.[26] Contrast material outlines the intraperitoneal organs with intraperitoneal bladder injuries. Extraperitoneal bladder injuries usually result from the shear forces from pelvic ring disruption due to pelvic fractures.[26] The extravasation of contrast material that appears as a flame-like projection confined to the pelvis constitutes an extraperitoneal injury. All intraperitoneal injuries are managed surgically whereas some extraperitoneal injuries can be managed with Foley catheter drainage (Chapter 173) or a suprapubic bladder catheter (Chapter 175).[7]

AFTERCARE

Patients may experience burning and dysuria due to the hypertonicity of contrast material and urethral stretching. Ensure that the patient is well hydrated after a normal study. The flow of urine will wash residual contrast out of the bladder and urethra.

COMPLICATIONS

Several complications can result from retrograde urethrography and cystography. Mechanical trauma from the catheter or balloon can result in bleeding, urethral injury, bladder perforation, and conversion of a partial urethral disruption into a complete urethral disruption. Adverse reactions to contrast agents can occur.[27-30] These may be due to local reactions or absorption of contrast into the blood. These reactions can range from itching and skin rashes to anaphylaxis and Stevens-Johnson syndrome. Treat any adverse reactions as any other allergic or hypersensitivity reaction.

SUMMARY

The retrograde urethrogram should be performed in any male with a pelvic fracture, lower abdominal trauma with gross hematuria, inability to void, hematoma of the perineum, or a high-riding or boggy prostate on physical examination. The cystogram should follow to evaluate potential bladder injury. The cystogram is used to evaluate and differentiate intraperitoneal versus extraperitoneal injuries.

Retrograde urethrography and cystography are diagnostic procedures that are easy to perform and have the potential to avoid major

complications related to urine leakage. Both of these procedures are safe and simple to perform. With basic equipment, invaluable information can be collected with minimal time investment. The early recognition of disruption of the lower genitourinary tract can prevent significant morbidity.

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177 Anesthesia of the Penis, Testicle, and Epididymis

Eric F. Reichman

INTRODUCTION

A wide range of urologic procedures are performed using local or regional anesthesia. This includes an orchiectomy, inspection of the painful testis, release of a paraphimosis, dorsal slit, circumcision, and even a hydrocelectomy or varicocelectomy done in the Operating Room. Emergency Physicians can use some of the same anesthetic techniques, namely the penile or spermatic cord blocks, to safely and painlessly perform many procedures in the Emergency Department. These techniques are easy to learn, simple to perform, and have a low risk of serious complications.

ANATOMY AND PATHOPHYSIOLOGY

Innervation of the penis arises from the pudendal nerve that is derived from sacral levels 2 to 4. The pudendal nerve divides into the perineal and the inferior rectal nerves. The perineal nerve further divides into the right and left dorsal nerves of the penis. The dorsal nerves of the penis pass under the pubic symphysis to penetrate the suspensory ligament of the penis.[1] They travel under Buck's fascia to supply sensory innervation to the entire penis (**Figure 177-1**).

The primary nerve supply of the testis and epididymis is from the ilioinguinal and genitofemoral nerves. The ilioinguinal nerve is derived from the first lumbar spinal nerve. It arises slightly inferior and medial to the anterior superior iliac spine and courses

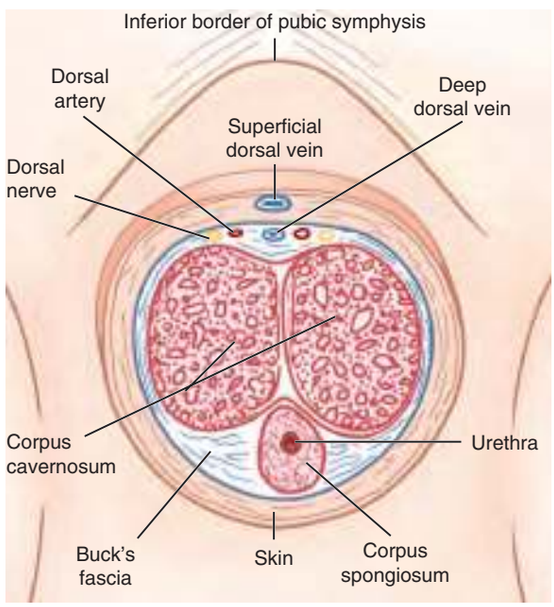

FIGURE 177-1. Transverse section through the base of the penis.

toward the pubic tubercle between the internal and external oblique muscles.[1,2] It enters the inguinal canal on the anterior surface of the spermatic cord. The ilioinguinal nerve provides sensory innervation to the skin of the upper thigh, base of the penis, and upper scrotum.[1] It also provides sensory innervation to the spermatic cord and testicle. The genitofemoral nerve is derived from the first two lumbar spinal nerves. It divides into the genital branch and the femoral branch. The genital branch enters the inguinal canal at the external inguinal ring and travels with the spermatic cord. It provides sensory innervation to the lower scrotum, cremaster muscle, spermatic cord, and scrotum. The femoral branch supplies the skin of the anteromedial thigh.[1]

INDICATIONS

Emergency Department procedures that are facilitated by local anesthesia of the penis include a dorsal slit of the foreskin, release of a phimosis or paraphimosis, repair of penile lacerations, and release of penile skin entrapped in zippers. Local anesthesia can also be used before performing a circumcision. However, this procedure is usually not performed in the Emergency Department.

The Emergency Department indications for a spermatic cord block include the relief of epididymal pain, the facilitation of a manual or ultrasound examination when differentiating between torsion and epididymitis, and to inspect the testis following trauma. Manual detorsion of a testis may be enabled by local anesthesia when a patient cannot tolerate the pain of palpation. **This should be performed only if the patient will be taken immediately to the Operating Room for confirmation of detorsion and an orchiopexy.** However, the risk of compromising the blood supply to the testis and the loss of patient assessment of pain in determining the success of detorsion are often cited as contraindications to spermatic cord blockade when testicular torsion is suspected.

CONTRAINDICATIONS

Local anesthesia is contraindicated in testicular torsion as there is a risk of compromising the testicular blood supply. The anesthetic effect will also eliminate the patient's assessment of pain that is needed to determine the success of manual detorsion. Agents containing epinephrine are not to be used in or around the penis.[3,4] The arteries of the penis are end arteries and vasoconstriction can result in tissue ischemia and sloughing. To minimize the spread of infection, local anesthetic should not be directly injected into an area of local infection.

EQUIPMENT

- 25 and 27 gauge needles
- Syringes (3 mL, 5 mL, 10 mL)
- Povidone iodine or chlorhexidine solution
- Alcohol pads
- 4×4 gauze squares
- Local anesthetic solution without epinephrine
- Ultrasound machine
- Ultrasound linear frequency transducer, 5 to 10 MHz
- Sterile ultrasound transducer cover
- Sterile ultrasound gel
- EMLA cream

Several local anesthetic agents are commonly used for genitourinary anesthesia. These include lidocaine, bupivacaine, mepivacaine, and chloroprocaine (**Table 177-1**). **The anesthetic agent, concentration, and technique must be chosen so that the maximum safe dose is not exceeded. This is especially important when performing anesthesia in children.**

TABLE 177-1 Local Anesthetic Agents Commonly Used for Genitourinary Anesthesia

Anesthetic agent	Strength (%)	Onset of action (minutes)	Duration of action (minutes)	Maximum dose (mg/kg)
Lidocaine	0.5, 1.0, 1.5, 2.0	5–15	45–90	4.5
Bupivacaine	0.25, 0.50	10–15	120–240	2.5
Mepivacaine	1.0, 1.5	10–15	120–240	4.0
Chloroprocaine	2.0, 3.0	10–15	20–40	11.0

The use of a local anesthetic agent containing epinephrine will prolong the block and give the patient a longer interval of pain-free time. However, the use of epinephrine is controversial. Epinephrine-containing local anesthetics can cause penile vasoconstriction and ischemia.[4,5] There are also reports of penile ischemia with local anesthetics not containing epinephrine.[6,7] Recently there was research stating epinephrine-containing local anesthetics in penile surgery are safe.[8] There is not enough information on the safety of epinephrine-containing local anesthetics on the penis. The safest recommendation is not to use epinephrine-containing local anesthetics on the penis as most Emergency Department procedures on the penis are short.

There are several alternative techniques that can be used. EMLA cream has been used in office procedures similar to the ones performed in the Emergency Department.[9] EMLA cream provided almost all participants excellent anesthesia. The only downside of using EMLA is the time it takes for the anesthesia to take effect. Acupuncture on bilateral forearm points prior to the penile block eased the pain when performing the block.[10] The use of acupuncture is outside the training of Emergency Medicine and not used in US Emergency Departments.

PATIENT PREPARATION

The patient and/or their representative must be informed of the procedure including its risks and benefits. The explanation must include the risks of local anesthetic injection and the possibility that the anesthetic may not effectively eliminate pain. The risks associated with the injection include pain, hematoma formation, and bleeding. Infection is a late risk that can be reduced by prepping the skin and using sterile technique. Obtain an informed consent for the anesthetic procedure in addition to the consent for the procedure that will be performed under anesthesia.

The patient's ability to cooperate with the planned procedures must be assessed. Consider oral or parenteral sedation, especially in anxious patients or children. Midazolam (0.02 to 0.04 mg/kg intravenously [IV]) or diazepam (2.5 to 5 mg IV) is often a good choice in adults. Children may benefit from midazolam parenterally (0.05 to 0.15 mg/kg IV) or orally (0.5 to 0.7 mg/kg). Ketamine (1.0 to 1.5 mg/ kg IV or 2 to 5 mg/kg intramuscularly) with or without atropine (0.01 mg/kg) to reduce respiratory secretions is an alternative, especially in children younger than 10.

Clean any dirt and debris from the skin. Identify the anatomic landmarks required to perform the anesthetic injection. Apply povidone iodine or chlorhexidine solution to the area where the anesthetic is to be injected. The povidone iodine or chlorhexidine may also be applied to the penis and scrotum if an invasive procedure is subsequently to be performed. Apply sterile drapes to delineate a surgical field. Allow the povidone iodine or chlorhexidine to dry.

Put on sterile gloves and reidentify the anatomic landmarks. Infiltrate with local anesthetic solution using sterile technique.

TECHNIQUES

LOCAL INFILTRATION

Local anesthetic infiltration of the skin of the penis is useful for the repair of superficial lacerations, dorsal slits of the foreskin, or freeing entrapped skin from a zipper. Local infiltration circumferentially around the penis will provide adequate anesthesia distal to the anesthetic injection site.[11] The circumferential subcutaneous injections can be performed directly on the penis or on the abdominal wall and scrotum surrounding the penis. While not contraindicated, some authors avoid direct infiltration of the foreskin as tissue sloughing may result.[2] **Local infiltration of anesthetic agents is often extremely painful for the patient. Consider premedicating the patient with parenteral benzodiazepines and/or narcotic agents**.

Local anesthetic agents can be injected subcutaneously into the penis to provide distal anesthesia. If performing a dorsal slit of the foreskin, raise a skin wheal of local anesthetic solution at the base of the foreskin in the 12 o'clock position (**Figure 177-2*A***). Insert the needle through the skin wheal aimed distally. Inject local anesthetic solution subcutaneously as the needle is advanced to the distal edge of the foreskin. Alternatively, local anesthetic solution can be circumferentially infiltrated around the penis (**Figure 177-2*B***). This block usually requires 6 to 10 mL of local anesthetic solution in the adolescent or adult and 2 to 3 mL in a child.

PENILE BLOCK

The objective of a penile block is to anesthetize the right and left dorsal nerves of the penis that provide sensation to the penis (**Figure 177-1**). **The dorsal nerves should be blocked as close to the base of the penis as possible. If the block is performed too distal to the pubic bone, the posterior branches of the dorsal nerves will not be anesthetized and the ventral surface of the penis will retain sensation**.[11-13] Multiple effective techniques will be described to perform a penile block. The technique chosen should depend on the specific procedure to be performed, the level of patient cooperation, and the preference of the Emergency Physician.

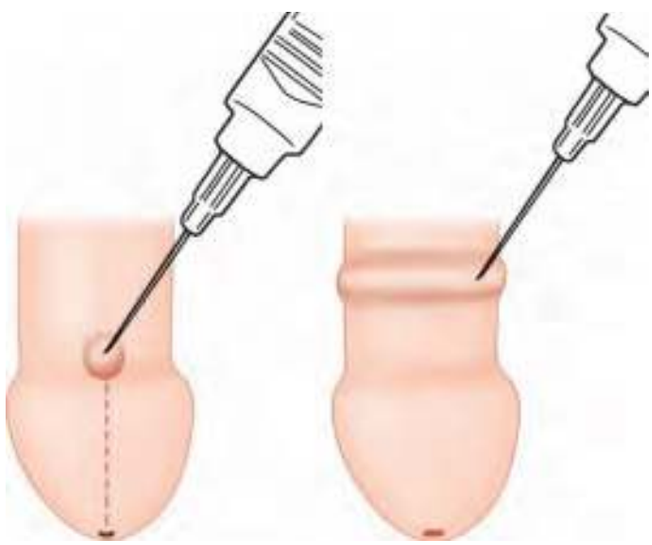

FIGURE 177-2. Local anesthetic infiltration into the penis. ***A.*** A skin wheal is raised and local anesthetic is injected distally (dotted line). ***B.*** Circumferential infiltration of local anesthetic solution.

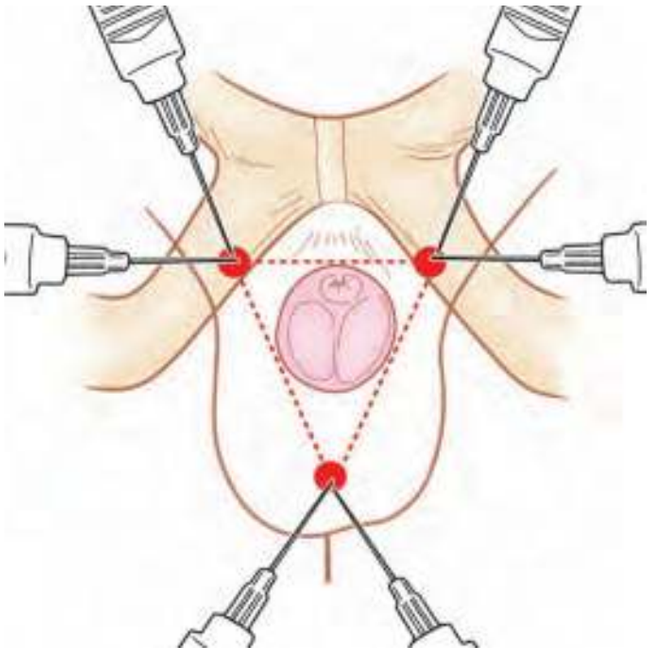

FIGURE 177-3. Local anesthetic infiltration around the base of the penis. The dots represent the locations of the skin wheals.

The penis may be anesthetized where it forms along the abdominal wall.[2] This block anesthetizes the nerves to the penis before they reach the penis. Place three skin wheals using local anesthetic solution on the abdominal and scrotal skin, 0.5 to 1.0 cm from the base of the penis, at the 2 o'clock, 6 o'clock, and 10 o'clock positions (**Figure 177-3**). Infiltrate subcutaneously with local anesthetic solution between the skin wheals to form a triangle of anesthetic that surrounds the base of the penis. This block usually requires 8 to 12 mL of local anesthetic solution in the adolescent or adult and 3 to 5 mL in a child.

The dorsal nerves of the penis can be anesthetized as they course onto the penis. Place a skin wheal of local anesthetic solution at the 2 o'clock and 10 o'clock positions.[14,15] Slowly insert a 27 gauge needle through the skin wheals until there is a slight loss of resistance indicating penetration of Buck's fascia (**Figure 177-4**). Aspirate to ensure that the needle is not within a blood vessel. Inject 2 mL of local anesthetic solution at each site in the adolescent or adult and 0.3 to 0.5 mL in a child.[1]

Another technique blocks the dorsal nerves as they pass through the triangular space bordered by the pubic symphysis, the corpora cavernosa, and Buck's fascia. Place a skin wheal of local anesthetic solution at the dorsal base of the penis. Insert the needle through the skin wheal and to the pubic symphysis. Withdraw the needle slightly and advance it caudally until a loss of resistance is felt indicating it has penetrated Buck's fascia. Aspirate to ensure that the needle is not within a blood vessel. Inject 10 mL of local anesthetic solution on each side of the suspensory ligament (midline) in the adolescent or adult and 2 to 3 mL in a child. Alternatively, the same block can be accomplished with a separate injection on each side of the midline.[1,16]

ULTRASOUND-GUIDED PENILE BLOCK

Ultrasound has been successfully used in the dorsal penile block.[17-20] Ultrasound allows real-time visualization of local anesthetic injection and spread, decreases inadvertent injection into the corpora

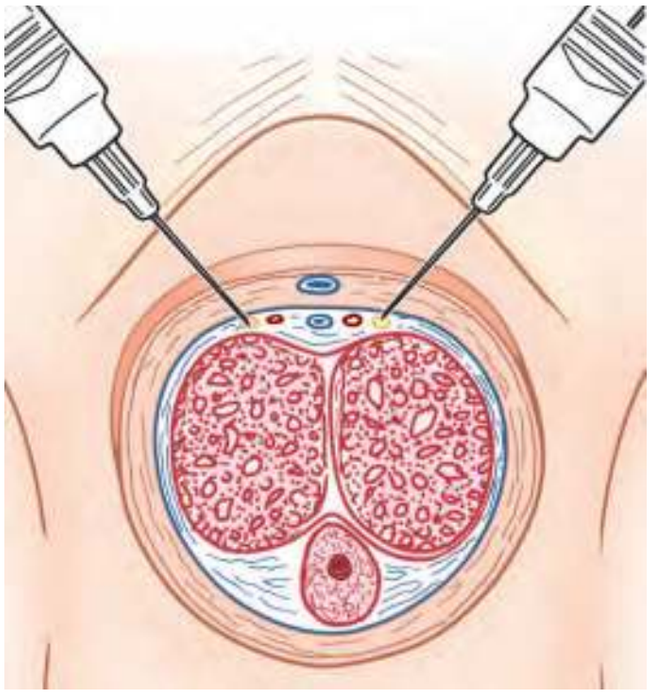

FIGURE 177-4. The penile block anesthetizes the left and right deep dorsal nerves of the penis.

cavernosa, decreases the risk of vascular injection, decreases volume of local anesthetic used, and increases success rates. The traditional blind or landmark technique does not always work due to injection of the local anesthetic above Buck's fascia, intracorporeally, or intravascularly. One study showed there was no difference in the ultrasound versus the landmark technique.[18]

Use a linear transducer in the 5 to 10 MHz range. Identify with ultrasound Buck's fascia, the dorsal penile nerves, and the dorsal penile vasculature (**Figure 177-5**). Clean and prep the penile skin.

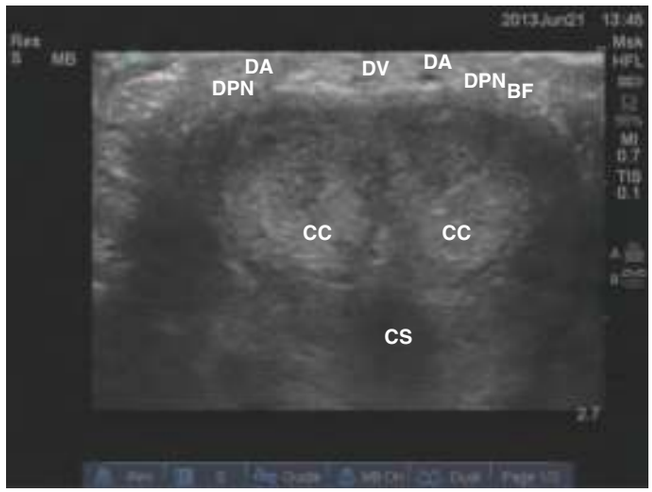

FIGURE 177-6. Ultrasound of the penis. DA, dorsal artery; DV, dorsal vein; DPN, dorsal penile nerve; BF, Buck's fascia; CC, corpus cavernosum; CS, corpus spongiosum. (Photo courtesy of M-I Suleman, from *Anesthesia News* 2015; 41(5):10-13.)

Prepare the transducer using sterile ultrasound gel and a sterile cover. Repeat the ultrasound, sterilely, to identify the structures (**Figure 177-6**). Use the in-plane approach. Inject the 2 to 4 mL of the local anesthetic agent at the 1 o'clock position (**Figure 177-7*A***). Watch the ultrasound machine screen to confirm the injection and spread of local anesthetic solution under Buck's fascia (**Figure 177-7*B***). This technique allows one injection to anesthetize the penis.

SPERMATIC CORD BLOCK

As the spermatic cord exits the external inguinal ring, it passes over the pubic tubercle and continues medially toward the scrotum. In this same location, the ilioinguinal nerve travels on the anterior surface of the spermatic cord and the genital branch of the genitofemoral

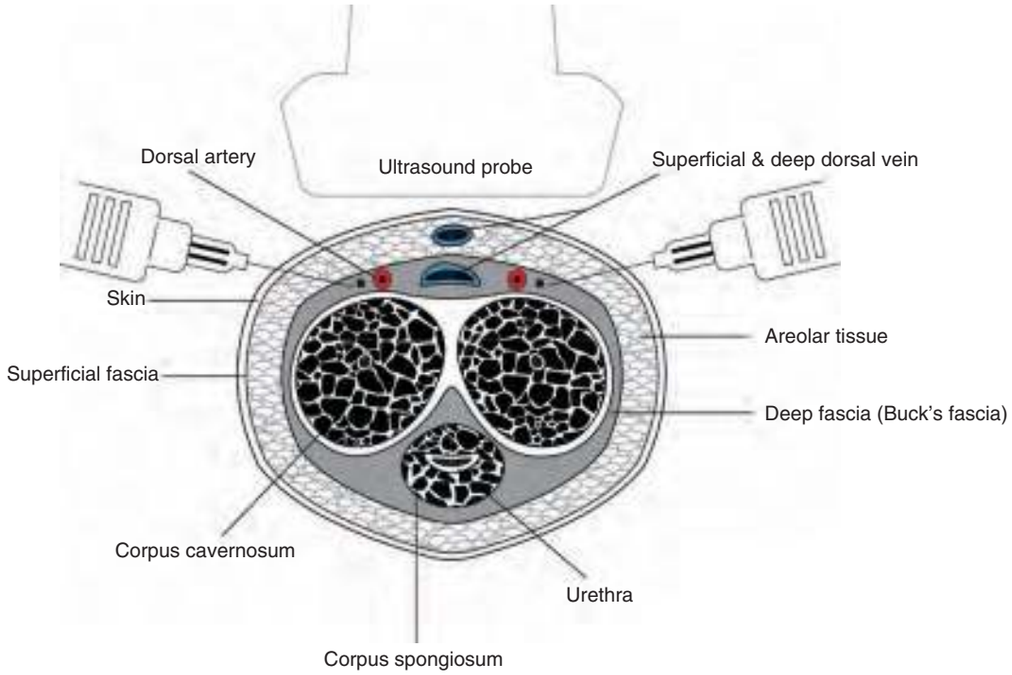

FIGURE 177-5. Artist illustration of the ultrasound-guided penile block. (Photo courtesy of M-I Suleman, from *Anesthesia News* 2015; 41(5):10-13.)

A

B

FIGURE 177-7. Injection of local anesthetic solution. ***A.*** Photo. ***B.*** Ultrasound. DA, dorsal artery; DV, dorsal vein; DPN, dorsal penile nerve; BF, Buck's fascia; CC, corpus cavernosum; CS, corpus spongiosum; LA, local anesthetic. (Photo courtesy of M-I Suleman, from *Anesthesia News* 2015; 41(5):10-13.)

nerve on the posterior surface. These two nerves supply sensation to the spermatic cord, epididymis, and testicle. Anesthesia of the spermatic cord in the region of the pubic tubercle will provide anesthesia to the testis and its covering, the epididymis, and the vas deferens.[13] **The spermatic cord block does not provide anesthesia to the skin of the scrotum.** Additional subcutaneous infiltration is necessary for an incision of the scrotal skin.

This first spermatic cord block technique is useful in thin patients with a palpable pubic tubercle. Identify the pubic tubercle by palpation. Inject local anesthetic solution to make a skin wheal just medial and 1 cm below the pubic tubercle (**Figure 177-8**). Gently advance a 25 gauge needle laterally through the skin wheal and spermatic cord until bone is contacted. Aspirate to ensure that the needle is not within a blood vessel. Inject 3 to 4 mL of local anesthetic solution as the needle is slowly withdrawn in the adolescent or adult and 1 to 2 mL in a child. Repeat the procedure two more times using the same skin puncture site, each time passing through the cord at a slightly different angle. This block requires a total of 10 to 12 mL of local anesthetic solution in the adolescent or adult and 3 to 5 mL in a child.[13]

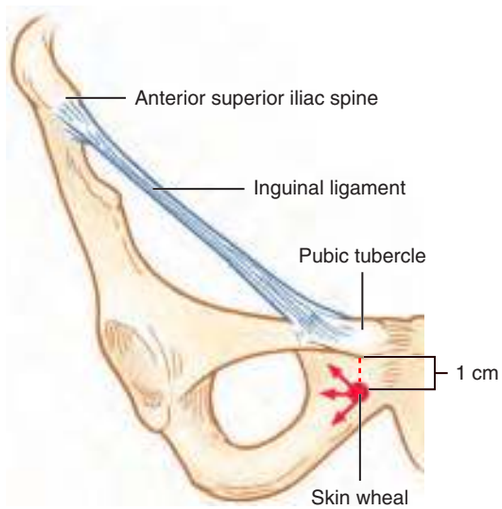

FIGURE 177-8. The spermatic cord can be anesthetized just below the pubic tubercle if the tubercle is palpable. The arrows represent the three different directions required to inject local anesthetic solution.

A modified technique is used for the patient in whom the pubic tubercle is difficult to palpate. Grasp the spermatic cord between the nondominant thumb and index finger as it enters the scrotum (**Figure 177-9**). Place a wheal of local anesthetic solution between the fingers and above the spermatic cord. Insert a 25 gauge needle through the skin wheal. Direct the needle anterior to the spermatic cord. Aspirate to ensure that the needle is not within a blood vessel. Inject 3 to 4 mL of local anesthetic solution in the adolescent or adult and 1 to 2 mL in a child. Repeat the process on the medial and lateral side of the spermatic cord.

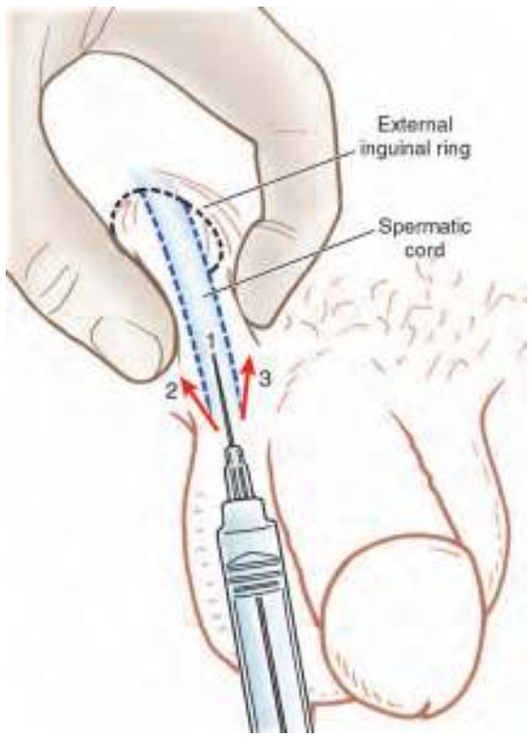

FIGURE 177-9. The spermatic cord block at the base of the scrotum. Local anesthetic solution is injected anteriorly (1), laterally (2), and medially (3) to the spermatic cord.

Alternatively, palpate the spermatic cord as it enters the scrotum.[21] Trace the spermatic cord superiorly to the pubic tubercle where it exits the external inguinal ring. Trap the spermatic cord between the second and third fingers of the nondominant hand and the pubic tubercle. Place a wheal of local anesthetic solution between the fingers and above the spermatic cord. Insert a 25 gauge needle through the skin wheal. Aspirate to ensure that the needle is not within a blood vessel. Inject local anesthetic solution anteriorly, medially, and laterally to the spermatic cord as described above. Injection of local anesthetic solution around the spermatic cord as it exits the inguinal canal may be less painful than injection as it enters the scrotum.

ASSESSMENT

Allow 10 to 15 minutes for the local anesthetic solution to take effect for a penile block. Test the level of anesthesia by pinching the skin with a forceps or by pinprick with a needle. The patient may feel a pressure sensation but should not feel pain. If the test stimulus is painful, repeat the block or use additional anesthetic techniques prior to performing the procedure.

Spermatic cord blocks require 10 to 15 minutes for maximal effectiveness. The patient should be warned that, despite an effective block, traction on the spermatic cord might cause nausea and a tugging sensation. These will resolve upon release of the traction.[13] Additional anesthesia must be applied to the scrotal skin for any incisions or procedures as a spermatic cord block does not anesthetize the scrotal skin.

AFTERCARE

Minimal aftercare is required for these local anesthetic techniques. Depending on the agent used, sensation may return in as little as 1 to 2 hours (e.g., lidocaine, chloroprocaine) or up to 4 hours (e.g., bupivacaine). The skin may reawaken with a pins-and-needles sensation. Patients should use caution when zipping their pants so as not to catch the penis, foreskin, or scrotum as they will not feel the injury. Patients should be warned that the site of a spermatic cord block might remain tender for as long as 10 days.[13] The application of cool compresses to the injection site and oral nonsteroidal anti-inflammatory drugs will provide adequate analgesia for the injection site pain. Patients should inspect the injection site and surrounding area three to four times a day for any signs of infection. They should return to the Emergency Department immediately if any problems or concerns arise.

COMPLICATIONS

The most procedure-specific complication of a penile block is sloughing of the penile skin. This is more common in the region of the glans. Performing the penile block at the base of the penis and using solutions without epinephrine minimize this risk.

Hematomas can be quite large in spermatic cord blocks because the venous plexus is usually pierced. The use of a smaller needle and careful application of pressure can help prevent hematomas. Blood loss from puncturing a vascular structure is easily controlled with direct pressure. A Urologist should be consulted urgently in the rare occasion that bleeding does not resolve with pressure. Most hematomas will resolve within 1 to 2 weeks. The application of warm compresses to the hematoma several times a day may result in quicker resorption.

Local infection at the injection site is possible. Patients should be warned about the signs of infection including fever, erythema, warmth, induration, increased pain, and purulent drainage. Patients should seek immediate medical attention for any of these symptoms. The injection of local anesthetic can cause inflammation in the short term.[22] It should not be confused with an infection.

Toxic levels of anesthetic agents can affect multiple organ systems, most notably the central nervous and cardiovascular systems. Anesthetic agents block the inhibitory neurons of the brain producing a state of neuro-excitation. Initial symptoms may include tinnitus, premolar numbness, disorientation, lightheadedness, or nystagmus. This may progress to seizures that can be accompanied by slow or absent breathing, acidosis, aspiration, and cardiovascular instability. Intravenous diazepam (2.5 to 5 mg) at the first sign of symptoms may stop the cascade.[2] Significantly higher doses of diazepam are required to treat local anesthetic-induced seizures. Very high levels of local anesthetics are cardiotoxic and may result in a heart block. Heart block from bupivacaine toxicity is associated with resistance to resuscitative maneuvers. The onset of all local anesthetic toxicities is faster with intravascular injection versus toxic tissue concentrations. Consider the use of intravenous lipid emulsion (Chapter 153) for any local anesthetic toxicity.

SUMMARY

Local anesthesia allows many painful genitourinary procedures to be performed in the Emergency Department. The techniques are simple and easy to perform with little to no experience. Local anesthetic solutions containing epinephrine should never be used to anesthetize the penis, scrotum, or spermatic cord.

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178 Priapism Management

Steven Go

INTRODUCTION

Priapism was first described in the English literature in an 1824 case report by Callaway.[1,2] **Priapism is defined as a prolonged engorgement or erection of the penis or clitoris that lasts over 4 hours, occurs beyond sexual stimulation or arousal, or occurs apart from sexual stimulation or arousal.**[3] The term "priapism" derives its origin from the name of a minor Greek god of fertility and growth named Priapus.[4] He was prenatally cursed by Hera with "out of proportion genitals, ugliness, lewdness, and impotence." A famous Pompeiian fresco graphically illustrates Priapus' plight as he weighs his massive phallus with a hanging scale.[4] His humiliated and demoralized demeanor in ancient mythology seems to parallel that of many present-day afflicted patients.[5]

Priapism affects an estimated 5.34 cases per 100,000 person-years in the general US male population.[6] It was present in 2.3% of Emergency Department visits by male sickle cell patients from 2006 to 2010.[7] There is a predilection for presentation during the summer months.[6] The most common form of priapism is often excruciatingly painful and is frequently embarrassing to the patient. This can result in delays seeking medical attention. Priapism can sometimes lead to permanent erectile dysfunction without swift and expert intervention.

Priapism is classified into the major subtypes of ischemic, nonischemic, and stuttering priapism (**Table 178-1**).[8-11] Stuttering priapism is often associated with sickle cell anemia. It is critical that the Emergency Physician determines which subtype the patient has because emergent management and prognosis differ considerably.

TABLE 178-1 The Clinical, Penile Blood Characteristics, and Color Doppler Flow Differences Between Ischemic and Nonischemic Priapism

	Ischemic priapism	Nonischemic priapism
Clinical characteristics	Pulsations	Less tense penis
	Rigid penis	Minimal pain
	Usually atraumatic	No pulsations
	Very painful	Often traumatic
Penile blood characteristics	Dark appearance	Bright red appearance
	$PaCO_2 > 60$ mmHg	$PaCO_2 < 60$ mmHg
	$PaO_2 < 30$ mmHg	$PaO_2 > 30$ mmHg
	pH < 7.25	pH > 7.25
	Thick consistency	Thin consistency
Color Doppler flow pattern	Low arterial inflow	High arterial inflow
	Low/no venous outflow	Venous outflow

Source: References 3, 8, 13.

TABLE 178-2 Some Etiologies of Ischemic Priapism

Drug-induced
α-Blockers
Androgens
Anticoagulants
Antidepressants
Antihypertensives
Antipsychotics
Anxiolytics
Cocaine
Crack cocaine
Erectile dysfunction agents
Ethanol
Gonadotropin-releasing hormone
Hormones
Impotence agents orally
Marijuana
PDE5 inhibitors (e.g., sildenafil)
Prostaglandins
Sympathomimetics
Testosterone

Hematologic disorders
Factor V Leiden
Fat emboli associated with total parenteral nutrition
Leukemia
Myeloma
Polycythemias
Sickle cell disease
Thalassemia

Iatrogenic
Intracavernous erectile therapy
Surgery

Idiopathic
Most common etiology
Occurs in 21%–33%

Malignancies
Metastatic cancer
Primary penile cancer

Miscellaneous
Amyloidosis
Black widow spider bites
Carbon monoxide
Dialysis
Fabry disease
G6PD deficiency
Genitourinary trauma
Gout
Herbal supplements
Hypertriglyceridemia
Malaria
Rabies
Scorpion stings
Total parenteral nutrition

Neurologic
Autonomic neuropathy
Cauda equina syndrome
Lumbar disk herniation
Medullary injury
Spinal anesthesia
Spinal cord lesions
Spinal stenosis
Stroke
Syphilis

Source: References 10, 23, 25-43.

The presenting subtype is ischemic (i.e., low flow or vaso-occlusive) in 95% of priapism cases and poses the greatest risk of permanent penile dysfunction. This type of priapism is of the greatest concern to the Emergency Physician. It is generally thought to result from an impediment to blood emptying from the penis regardless of the etiology (**Table 178-2**).

The patient presents with a very painful rigid penis with engorgement of both corpora cavernosa (**Table 178-1**). The corpus spongiosum and the glans are usually spared, although they can be involved in rare instances.[12] This situation forms a type of compartment syndrome if left untreated. It can result in fibrosis, loss of function, scarring, and penile gangrene in extreme cases. Priapism outcomes are often dismal. Reported rates of complete erectile dysfunction occur in > 35% of patients with treatment and to up to 90% without treatment.[13,14]

The second main subtype of priapism is nonischemic (i.e., high flow or arterial) and is far less common than ischemic priapism (**Table 178-1**). It is caused by excess penile arterial blood flow that is often secondary to perineal or penile trauma (e.g., including iatrogenic or postprocedure).[15,16] Patients present with a painless partially erect penis. Arterial blood is freely flowing through the penis and the outcomes of nonischemic priapism tend to be more favorable than those of ischemic priapism, although subsequent erectile dysfunction can occur.[17] The Emergency Department management is generally supportive and requires no procedural intervention in most cases.

Stuttering or intermittent ischemic priapism is a variant of ischemic priapism in which the patient suffers intermittent priapism episodes over time. This variant is most frequently seen in sickle cell disease patients. Stuttering priapism episodes may become prolonged (i.e., > 4 hours) and are treated in the same fashion as ischemic priapism.

ANATOMY AND PATHOPHYSIOLOGY

RELEVANT ANATOMY

The penis is primarily composed of the dual dorsal corpora cavernosa and a single ventral corpus spongiosum (**Figure 178-1**). The corpora cavernosa surrounds the cavernous and the helicine arteries. The corpora cavernosa are the structures engorged during most cases of priapism. The corpus spongiosum surrounds the urethra and forms the glans. A superficial and deep neurovascular bundle is located on the dorsal surface of the penis.

The corpora of the penis consist of spongy networks of collagen, endothelial tissue, nerves, smooth muscle, and vascular structures. The primary arterial supply to the penis arises from the internal pudendal artery which gives rise to the cavernous artery and supplies the lacunar spaces in the corpora cavernosa. Another two branches of the internal pudendal artery, the bulbourethral and dorsal arteries, supply blood to the corpus spongiosum and glans. The cavernous artery dilates during an erection and blood flows through the helicine arteries into the sinusoidal spaces of the corpora cavernosa causing them to expand. The corpora cavernosa swell against a strong fibrotic outer layer (i.e., tunica albuginea) which produces a rigid penis.[18] **An important functional anatomic feature is that blood flows freely between both corpora cavernosa through the incomplete septum between them.**

Venous outflow regulation plays an equally important role in maintaining an erection.[19] The venous drainage is complex with paired branches running with the arteries. An additional major drainage system enters the deep dorsal vein via the multiple emissary veins and circumflex veins. The extensive subtunical plexus of veins feeds into the emissary veins that perforate the tunica albuginea and drain into the circumflex veins. The increasing distention of the corpora cavernosa during an erection with arterial blood causes the compression of the subtunical venous plexus. Stretching of the tunica albuginea obstructs drainage through the emissary veins and restricts venous outflow further. There is no venous outflow obstruction to the glans and corpus spongiosum until the rigid-erection phase. The glans and corpus spongiosum do not typically develop the higher pressure of the corpora cavernosa.

NORMAL ERECTION PHYSIOLOGY

An erection begins when the parasympathetic nervous system activates the nitric oxide (NO)-cyclic guanosine monophosphate (cGMP) pathway.[20] This begins a cascade of neurotransmitter events involving adenosine.[21] It culminates in decreased intracellular calcium from smooth muscle cells. The erection is the result of relaxation of the smooth muscle in the arterial walls and the corpora cavernosa. The primary mechanism of detumescence is mediated through the cGMP-specific type 5 phosphodiesterase (PDE5) enzyme which helps bring about contraction of smooth muscle.[22] This reduces arterial inflow and reduces corpora cavernosa volume which serves to promote venous outflow resulting in penile flaccidity.

ISCHEMIC PRIAPISM PATHOPHYSIOLOGY

There have been many recent advances in the understanding of the molecular mechanisms of ischemic priapism.[20,23] Chief among

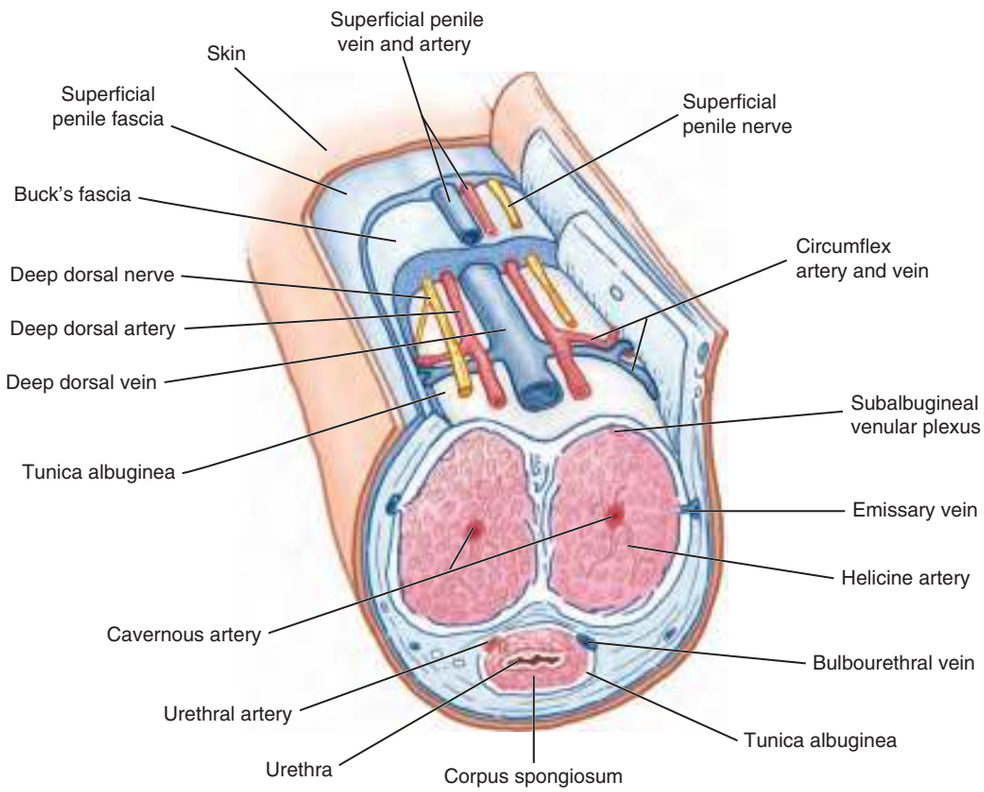


FIGURE 178-1. Cross-section of the penis demonstrating the anatomy.

these is the dysregulation of the NO-cGMP signaling pathway that results in an exaggerated erectile response in the smooth muscle.[22] The chronic decrease in NO causes reduced levels of PDE5 further hampering penile relaxation. Other defects in the RhoA/Rho kinase (ROCK) vasoconstrictor pathway, excessive adenosine signaling, increases in opiorphin levels, and oxidative-nitrosative stress have all been implicated in ischemic priapism.[22]

NONISCHEMIC PRIAPISM PATHOPHYSIOLOGY

Nonischemic priapism generally results from a trauma-induced arteriovenous communication between the cavernous artery and the sinusoidal spaces bypassing the high resistance helicine arteries. This results in unrestrained arterial flow. The penis is not at risk for ischemia because there is no increased venous constriction limiting outflow.

STUTTERING PRIAPISM PATHOPHYSIOLOGY

The mechanisms of stuttering priapism overlap with those of ischemic priapism. It includes additional mechanisms from the sickling erythrocytes in sickle cell disease patients (e.g., venous stasis and congestion).[24]

INDICATIONS

DETERMINING THE PRIAPISM SUBTYPE

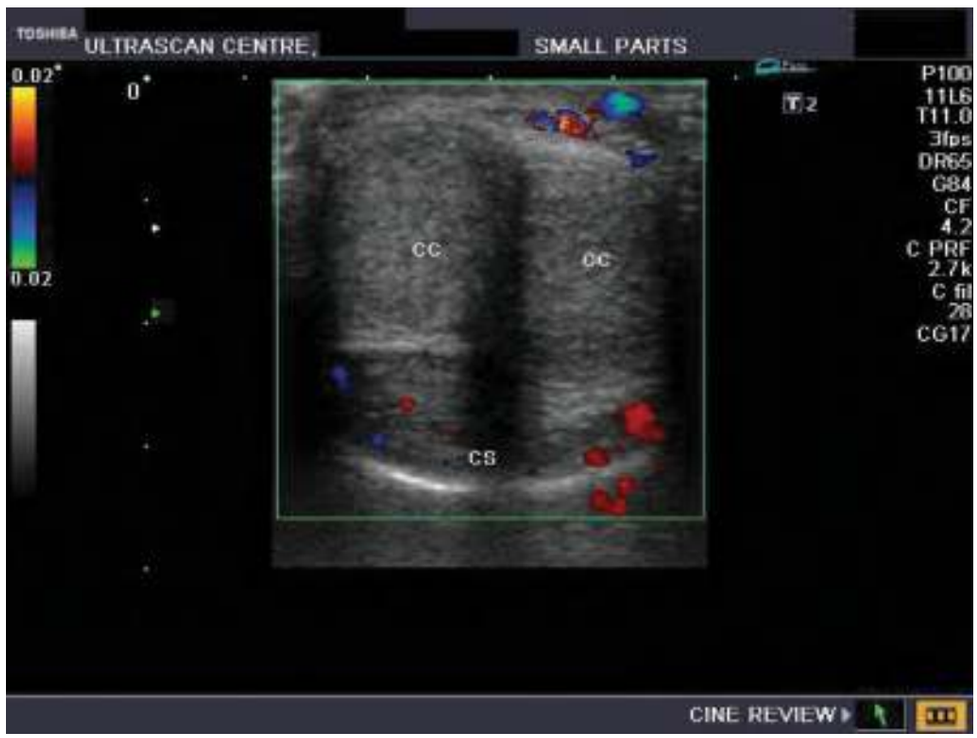
The initial evaluation by the Emergency Physician must be accurate, aggressive, and quick. The key diagnostic decision to be made in a patient presenting with priapism is to determine whether it is ischemic or nonischemic.[10] This distinction is important. Delays in emergent therapy increase the chance of poor outcomes in ischemic priapism whereas nonischemic priapism usually only requires supportive care and close follow-up with a Urologist. There are several differences in the presentation of ischemic and nonischemic priapism that allow the Emergency Physician to tell these subtypes apart (**Table 178-1**). Some of the etiologies of ischemic priapism are listed in **Table 178-2**.[10,23,25-43]

The clinical features that characterize ischemic priapism can generally be detected with a careful history and physical examination. **It is recommended to confirm clinical impression on every priapism patient by sampling the penile blood for arterial blood gas analysis or by a color Doppler ultrasound (Figures 178-2 and 178-3)**. Penile blood can be readily sampled at the same time therapy is initiated. The sampling is somewhat invasive and can be painful. Color Doppler ultrasound is less invasive but can delay definitive therapy if it is not readily available. The choice of confirmatory test must be driven by the clinical situation and setting.

Some have recommended additional diagnostic tests in patients who present with priapism.[3,13] This includes a complete blood count (CBC) with a manual differential to diagnose blood dyscrasias, a urinalysis as an infection screen, and various drug screens if indicated. **Do not delay seeking the definitive diagnosis and treatment of ischemic priapism to obtain ancillary studies**.

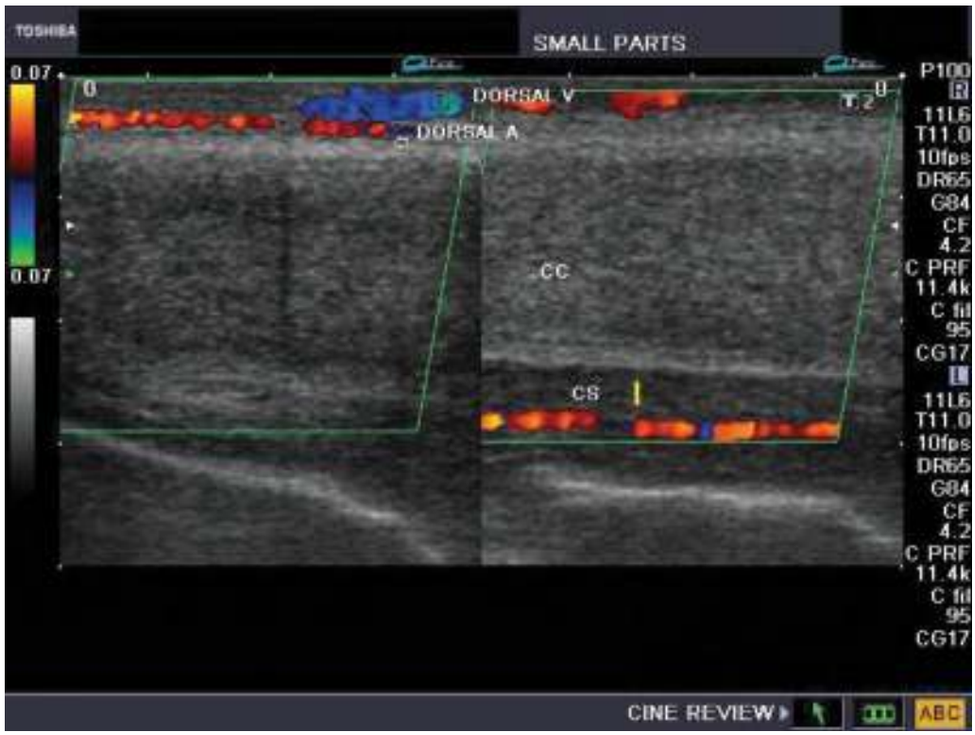
CONTRAINDICATIONS

The patient must be stabilized prior to dealing with the priapism if they have trauma-induced priapism and serious life-threatening injuries. Medical risks making priapism therapy inappropriate will occasionally occur and must be weighed against the fact that the natural course of priapism does not cause mortality. Do not administer



A

FIGURE 178-2. Transverse Doppler ultrasound image of the penile shaft in ischemic priapism. ***A.*** The corpora cavernosa (CC) have no flow, whereas the corpus spongiosum (CS) has some flow preserved. ***B.*** Close-up of the penile shaft showing flow to the tunica albuginea but not the corpora. (Images courtesy of Joe Anthony, MD, from his website: www.ultrasound-images.com.)



B

FIGURE 178-2. (*Continued*)

α-adrenergic agonists in patients with dysrhythmias, significant hypertension, unstable angina, and other high-risk cardiac conditions. They are also contraindicated if the patient is taking ergotamines (except ergoloid mesylates and nicergoline), linezolid, or monoamine oxidase inhibitors. Use α-adrenergic agonists with care in patients taking sympathomimetic drugs of abuse (e.g., cocaine). Penile aspiration, irrigation, and intracavernous injections are not efficacious in nonischemic priapism.

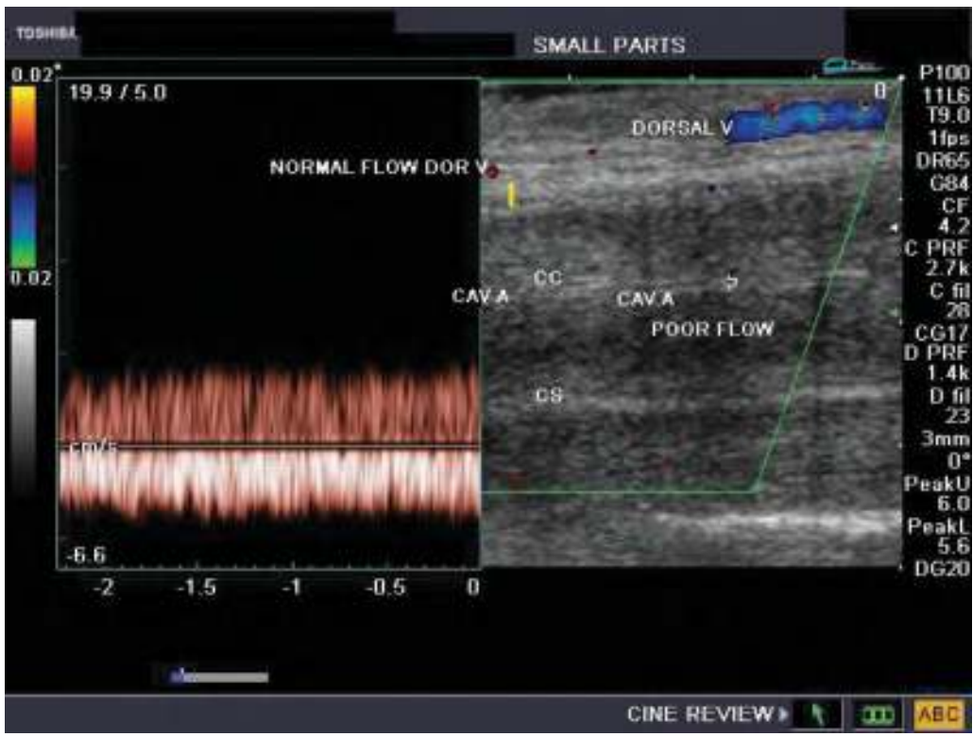


FIGURE 178-3. Transverse color spectral Doppler ultrasound image of the penile shaft in ischemic priapism. CAV A, cavernous artery; CC, corpus cavernosum; CS, corpus spongiosum; v, vein. (Image courtesy of Joe Anthony, MD, from his website: www.ultrasound-images.com.)

EQUIPMENT

- 19 gauge butterfly needle
- 21 gauge butterfly needle
- Arterial blood gas syringe
- Syringes, various sizes
- Povidone iodine or chlorhexidine solution
- 4×4 gauze squares
- Sterile drapes
- Sterile basin
- Three-way stopcock (optional)
- Phenylephrine, diluted as noted below[44-46]
- Local anesthetic without epinephrine
- Sterile normal saline, 1 L bottle
- Sterile normal saline, 10 mL vial
- Intravenous (IV) access supplies (Chapter 61)
- Blood pressure monitoring equipment
- Cardiac monitoring equipment
- Pulse oximeter
- Oxygen source and nasal tubing
- Sterile gloves
- Ultrasound machine, optional
- Ultrasound gel, optional
- Ultrasound transducer, optional

PATIENT PREPARATION

Obtain an emergency Urologist consult immediately when the presence of ischemic priapism is determined. Perform the procedure if a Urologist is not readily available or if the Emergency Physician is comfortable performing the procedure. Explain the risks, benefits, and alternative procedures to the patient and/or their representative. The discussion should include being transferred to a facility with a Urologist if one is not immediately available. **Informed consent is crucial in the treatment of ischemic priapism because the outcomes can be poor even with appropriate treatment.** Allow time for questions so that the patient can make an informed and voluntary decision. Document the patient's capacity to consent, benefits, risks, alternatives discussed, and the medical decision making in the chart and obtain a signed consent form.

Obtain IV access for pain control. Place the patient on the cardiac monitor, noninvasive blood pressure cuff, pulse oximetry, and supplemental oxygen to monitor them during and after the procedure as an added level of precaution. Some adverse events have been reported during the procedures described below. This is especially true in patients with a history of cardiac dysfunction, coronary artery disease, or hypertension. Administer IV hydration and oxygen to sickle cell disease patients.

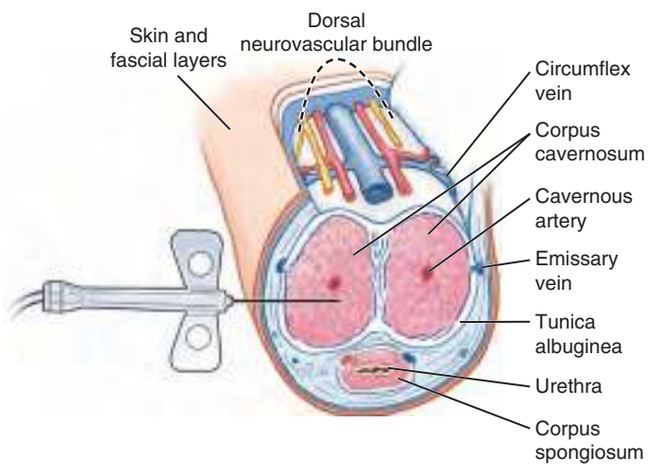
Perform ultrasonography of the penis if is available at the bedside in a reasonable amount of time. View the corpora cavernosa and vasculature of the penis. Use the normal gray scale and color Doppler (**Figures 178-2 and 178-3**). Power Doppler can be used to confirm the lack of flow to the corpora cavernosa.

Place the patient supine with their legs spread apart. Consider the administration of parenteral analgesics, sedatives, and/or conscious sedation (Chapter 159) as appropriate because these procedures can be very painful. Gently clean the penis, scrotum, and surrounding area of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the penis and surrounding area and allow it to dry. Apply sterile drapes to delineate a sterile field. Perform a dorsal penile nerve block or a circumferential penile block (Chapter 177).

TECHNIQUES

PENILE ASPIRATION

Attach a blood gas syringe to the tubing of a nonheparinized 19 gauge butterfly needle. While gently applying negative pressure via the syringe, insert the needle into the lateral aspect of one of the corpus cavernosa in the midshaft of the penis and perpendicular to the shaft (**Figure 178-4**). **Insert the needle at the 3 o'clock or 9 o'clock position to avoid the neurovascular structures between 10 and 2 o'clock.** Insert the needle just far enough to freely aspirate blood. Hold the needle still so it does not move. Remove the syringe. Send the aspirated blood for blood gas analysis. Attach an additional 10 or 20 mL syringe and aspirate enough blood to significantly soften the penis (usually about 5 mL) if color Doppler ultrasound has confirmed ischemic priapism or if the blood is dark. Both cavernosa



A

B

FIGURE 178-4. The technique of penile aspiration. ***A.*** Artist illustration. ***B.*** Clinical photo. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2016. Photo contributor: David Effron, MD.)

may be decompressed through the single drainage needle since the cavernosa communicate across the incomplete septum. Continue drainage until fresh red oxygenated blood is aspirated.[8] Penile blood aspiration alone is effective in inducing detumescence in approximately 30% of cases.[13]

PENILE IRRIGATION

Irrigation is appropriate if there is difficulty with the aspiration or if the priapism does not resolve with aspiration. Aspirate as described above. Carefully remove the syringe while leaving the needle in place. Insert a 19 or 21 gauge butterfly needle (i.e., the second needle) into the corpora in the proximal shaft area (**Figure 178-5**). **Insert the needle at the 3 o'clock or 9 o'clock position to avoid the neurovascular structures between 10 and 2 o'clock.** Begin gentle irrigation with normal saline through the second butterfly needle with outflow through the first butterfly needle (**Figure 178-5**). This will help to irrigate out the old, dark blood. **Use only normal saline to prevent damage to the endothelium if the solution is not isotonic**. Massaging the penis to "milk out" sludge can be helpful. Placement of additional 19 gauge butterfly needles may be used to improve drainage in difficult cases. **The fewer the injections administered, the less chance for forming a hematoma.**

INTRACAVERNOUS SYMPATHOMIMETIC INJECTION

Intracavernous injection is recommended if aspiration and irrigation fail to resolve the priapism. Phenylephrine is the agent of choice.[8,13,44-46] **Systemic sympathomimetic side effects may occur. Monitor the patient's vital signs and cardiac rhythm during the injection. Use great caution and lower dosages in vasculopathic patients and be prepared to deal with potential cardiovascular complications.** Many medications have been used to relieve

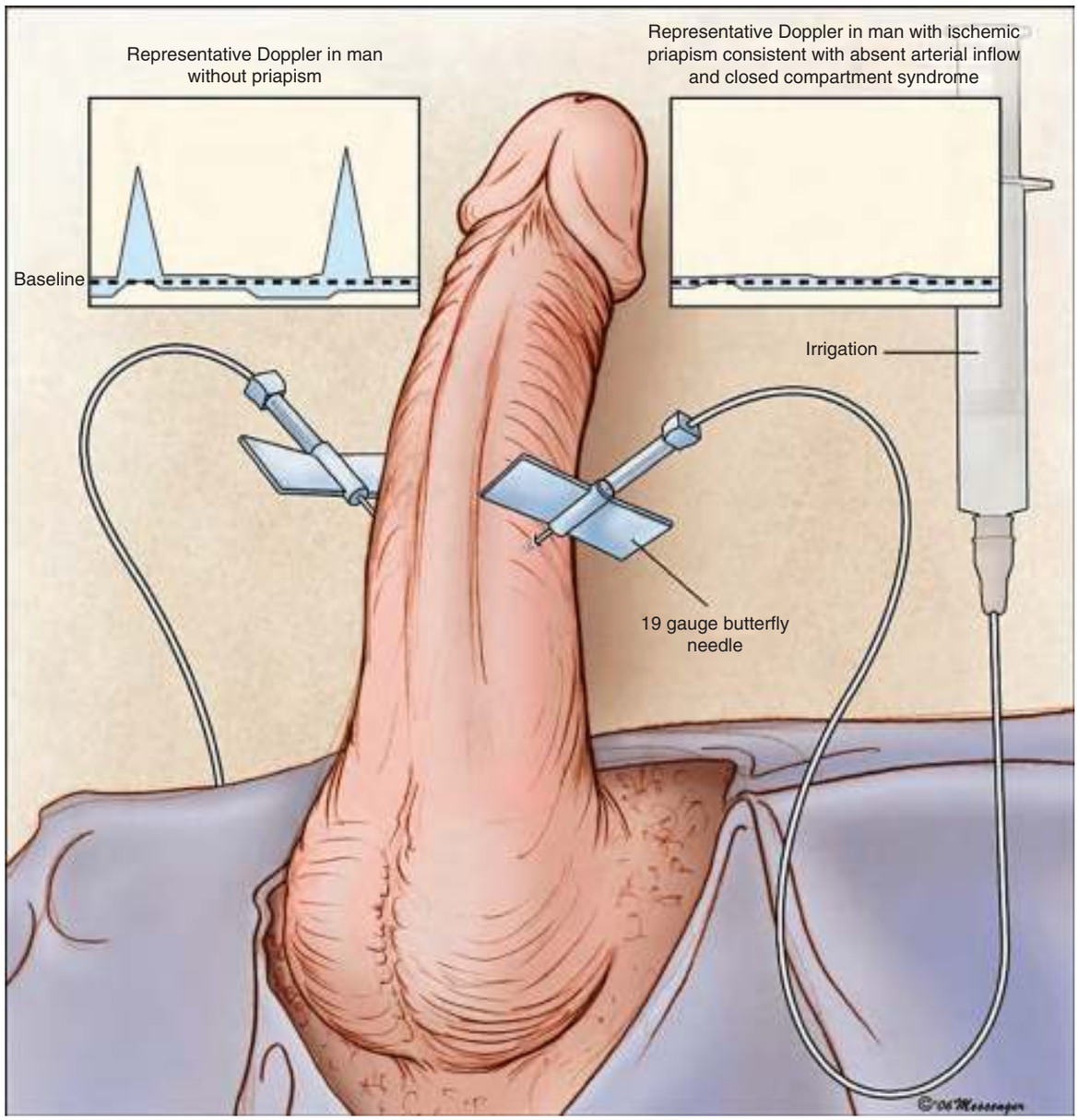


FIGURE 178-5. The irrigation of the corpora cavernosa. (Used with permission from reference 10.)

TABLE 178-3 The Medications Used to Treat Ischemic Priapism

Intracavernous injection
Epinephrine, 0.03–0.05 mg
Etylephrine, 2–20 mg
Methylene blue, 50 mg
Norepinephrine, 0.01–0.02 mg
Phenylephrine, 0.1–1.0 mg
Intramuscular injection
Ketamine, 1 mg/kg
Intravenous injection
Dopamine, 2–4 μg/kg
Ketamine, 1 mg/kg
Oral Administration
Pseudoephedrine, 0.5 mg/kg to maximum 30 mg does
Terbutaline sulfate, 5 mg

priapism (**Table 178-3**). The most experience in the Emergency Department is with phenylephrine. Deaths have been reported with agents other than phenylephrine. The use of phenylephrine rarely has been reported to give rise to serious complications and is the agent of choice.[44-46] Save the other medications for the Urologist.

Mix 10 mg of phenylephrine with 9 mL of sterile normal saline to produce a concentration of 1 mg/mL (i.e., 1000 μg/mL). Fill a tuberculosis syringe with the diluted solution through the butterfly catheter and into the penis. Observe the penis every 3 to 5 minutes and repeat the procedure if the erection returns or does not resolve. There is no definite limit to the cumulative amount of phenylephrine used for injections. Current recommendations are to not conclude that intracavernous injections have failed until the patient has been treated for 1 hour or with a total of 1 mg of phenylephrine.[8,13] **Discontinue the injection immediately if untoward cardiovascular side effects occur.[44-46]**

α_1-Adrenergic receptors are less able to cause smooth muscle contraction in longer episodes of priapism. Intracavernous injection therapy becomes less likely to succeed over time. Higher doses of up to 50,000 μg have been suggested in long-standing episodes of priapism.[47]

PEDIATRIC CONSIDERATIONS

The telling of others of the prolonged erection can be embarrassing. The treatment of ischemic priapism can emotionally and psychologically traumatize the pediatric patient. Use sensitivity toward the patient and their caregivers. Explain any treatment and allow questions. Aspiration and irrigation of the corpora cavernosa are the same as in an adult. Use 1 μg/mL of epinephrine for the intracavernosal injection if the patient is below the age of 11 years.

AFTERCARE

Remove the needle(s) and compress the puncture site for 30 to 60 seconds every time a needle is withdrawn from the penis to avoid a hematoma. The penis should resume a soft state after treatment. Reassess the penis with color Doppler ultrasound or repeat the penile blood gas assessment if it is not clear that treatment has been effective.[3] Absence of arterial blood in-flow on ultrasound or penile blood acidosis suggests treatment failure. Consider alkalization and exchange transfusion in sickle cell disease patients. An emergent surgical procedure by a Urologist (i.e., venous shunting) is indicated if aspiration, irrigation, and injection fail.[10]

Observe the patient for at least 30 minutes before assuming successful detumescence. **Do not apply a compression dressing to the penis.** There are no randomized controlled data to support the use of prophylactic antibiotics after aspiration, irrigation, and/or intracavernous therapy and they are not recommended in current guidelines.[8,13] Referral to an appropriate specialist depends on the cause of the priapism. Treat the offending mechanism or discontinue the medication if an underlying condition or medication is a suspected cause. Make the appropriate disposition and follow-up. **Follow-up with a Urologist within 24 hours is necessary in all patients. Document a detailed procedure note because of the legal risk involving priapism and its treatment. Give the patient written instructions and document these instructions in the medical record.**

COMPLICATIONS

The butterfly needle may lacerate the cavernous artery during aspiration which will be evidenced by the presence of pulsatile arterial blood. Emergent evaluation by a Urologist is indicated if such an injury is suspected. The long-term sequelae to unresolved priapism are fibrosis of the corporal tissue and loss of erectile function. Erectile dysfunction may still occur in patients despite successful detumescence. Varying degrees of fibrosis may be found on physical examination at a later date. Treatment by aspiration and irrigation can be associated with hematoma formation, infection, urethral injury, or vascular injury. The use of α-adrenergic agonists can have the systemic effects of flushing, headache, hypertension, reflex bradycardia, tachycardia, and other dysrhythmias. Deaths have been reported with agents other than phenylephrine. The use of phenylephrine rarely has been reported to give rise to serious complications and is the agent of choice.[44-46]

The patient needs to be fully informed of the long-term risk of sexual dysfunction even if detumescence is successful. Recurrence of the priapism is common. Warn the patient to seek immediate care if a recurrence occurs.

SUMMARY

Priapism is an uncommon but a vexing complaint in the Emergency Department. The two major subtypes of priapism are ischemic (i.e., low flow) and nonischemic (i.e., high flow). The key decision for the Emergency Physician is to determine what type of priapism is presenting. Ischemic priapism requires prompt treatment to reduce the possibility of permanent damage to the penile tissue and subsequent erectile dysfunction. It is imperative to confirm the diagnosis with either color Doppler flow ultrasound or penile aspiration. An emergent Urologist evaluation is indicated if ischemic priapism is strongly suspected or confirmed. Perform treatment in a stepwise fashion beginning with penile aspiration followed by irrigation.[23] Attempt intracavernous injection with phenylephrine if no contraindications exist. A Urologist should perform more invasive surgical procedures if these bedside strategies fail. It is imperative that the patient be fully informed and sign an informed consent given that the incidence of erectile dysfunction following low-flow priapism is high even with treatment.

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179 Paraphimosis Reduction

Matthew D. Schwartz and Ann P. Nguyen

INTRODUCTION

A paraphimosis is defined as the inability to reduce a proximally positioned foreskin over the glans penis and back to its normal anatomic position.[1] The most common cause for a paraphimosis is iatrogenic. Medical personnel may forget to reduce the foreskin over the glans following examination or instrumentation of the penis. This is particularly true in patients who are sedated, confused, demented, or delirious.

Elderly patients are particularly at risk as their predisposition to erectile dysfunction and decreased libido result in fewer erections to naturally dilate the foreskin. The elderly, demented, and cognitively delayed are at increased risk for poor genital hygiene and a paraphimosis.[2] Other patients may fail to reduce their foreskin after intercourse or urination.[3] Infants and toddlers have a normal physiologic phimosis that resolves by the age of 3 in over 90% of cases. This predisposes them to a paraphimosis when well-meaning caregivers forcibly retract the foreskin during cleaning. A paraphimosis may also occur when a narrowed or phimotic foreskin is retracted and unable to be reduced. Paraphimosis has been reported secondary to genital piercing, erotic dancing, and various cutaneous lesions (e.g., hemangioma and cutaneous lymphoproliferative disorders).[4-6]

A patient with a paraphimosis usually presents with severe penile pain. The process may have a more indolent presentation in persons with impaired pain sensation (e.g., the elderly or diabetics). Patients with altered mental status may simply present with agitation and are at risk for complications of a paraphimosis as they are often unable to complain of pain. This includes penile ulceration, infection, Fournier's gangrene, and partial penile autoamputation.[7,8] A careful and complete physical examination is mandatory in these patients. Penile edema secondary to a paraphimosis must be differentiated from edema due to infection, trauma, or allergic reactions.

FIGURE 179-1. The anatomy of a paraphimosis.

ANATOMY AND PATHOPHYSIOLOGY

The foreskin is composed of a double layer of epidermis overlying subcutaneous tissue. It is attached to the skin at the base of the glans penis. The foreskin covers the glans to a variable degree and can usually be completely pulled over the glans. The foreskin is retracted behind the glans and becomes edematous in a paraphimosis. The base of the foreskin is the location of the constricting or phimotic ring (**Figures 179-1 and 179-2**).

Arterial supply to the foreskin is derived from superficial branches of the external pudendal artery which originate from the femoral artery. These superficial arteries do not communicate with the deep arteries of the penis. Arterial supply to the glans penis is derived from the paired dorsal arteries of the penis which originate from the penile artery. Dorsal penile arteries run deep to Buck's fascia to enter the glans at the coronal sulcus. **Arterial supply to the glans penis is entirely separate from that of the foreskin.**

A retracted foreskin will block lymphatic drainage from the distal penis. Arterial inflow continues. Lack of lymphatic drainage will cause a progressive edema of the penis distal to the retracted foreskin. The phimotic ring becomes progressively tighter as the foreskin continues to swell. The phimotic ring will eventually obstruct venous outflow. The distal penis will become painful and hyperemic. The edema will progress to ultimately obstruct arterial inflow resulting in penile ischemia, necrosis, and gangrene. This series of events from retraction of the foreskin to arterial inflow obstruction can occur over a few hours to 2 days. The phimotic ring must be advanced (i.e., reduced) over the glans of the penis to relieve the obstruction.

A

B

FIGURE 179-2. A paraphimosis. ***A.*** Superior view. ***B.*** Inferior view.

INDICATIONS

All paraphimoses require reduction. The less edematous the tissues will be and the easier it will be to perform the reduction if it is reduced earlier in its time course. **Use nonsurgical techniques first. Surgical techniques are indicated if nonsurgical techniques are unsuccessful or the skin is compromised (i.e., infection, ulceration, or gangrene).**[6,9,10]

CONTRAINDICATIONS

There are no absolute contraindications to the reduction of a paraphimosis. Certain techniques are contraindicated in specific patient subgroups. Nonsurgical techniques are contraindicated in patients with ulcerated or necrotic foreskins as they may cause iatrogenic injury.[6,9] Some authors feel that surgical reductions in children should be reserved for Pediatric Urologists.[11] **Do not attempt a surgical reduction until noninvasive and lesser invasive techniques have been attempted.** The hyaluronidase technique is contraindicated in penile cancers or infections due to the possibility of spreading infected or malignant cells through the tissue planes.[12]

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Lidocaine jelly or EMLA cream
- Water-soluble lubricant
- Local anesthetic solution (1% lidocaine or 0.5% bupivacaine) without epinephrine
- 18 gauge needles
- 27 gauge needles
- 10 mL syringe
- 4×4 gauze squares
- Crushed ice
- Surgical gloves
- Babcock clamps, 6 to 8
- 2 inch wide roll of elastic bandage (e.g., Ace Wrap or Elastoplast)
- Sterile drapes
- Sterile surgical gloves
- Suture scissors
- 2 straight hemostats
- #15 surgical blade on a handle

- Needle driver
- 3–0 synthetic absorbable suture or chromic catgut suture
- Granulated (table) sugar
- Ampule of D50
- 20% mannitol
- Hyaluronidase, 150 units or 1 mL
- Tuberculin syringe
- Topical antibiotic ointment
- Petrolatum gauze

PATIENT PREPARATION

Reduction of a paraphimosis can be painful and distressing to patients. Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Explain that the progression from nonsurgical to surgical techniques may be required. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. Leave an indwelling urinary catheter in place during the reduction if the patient has one.

Penile analgesia can sometimes be achieved with topical lidocaine jelly or EMLA cream (i.e., 2.5% prilocaine and 2.5% lidocaine) applied liberally and held in place for 45 to 60 minutes by a biofilm dressing (Chapter 154). A technique especially effective in children involves cutting the finger from a surgical glove, filling it with EMLA cream, and securing this over the penis.[13] Topical anesthetics often do not provide adequate patient comfort. A ring block or dorsal penile nerve block performed with 0.5% bupivacaine and/or 1% lidocaine, potentially under ultrasound guidance, is becoming accepted as standard of care (Chapters 156 and 177).[14-16] Procedural sedation (Chapter 159) or general anesthesia may be required.[11]

TECHNIQUES

The techniques described below to reduce a paraphimosis begin with manual compression and progress to incision of the phimotic ring. They should be attempted in a stepwise manner from least invasive to most invasive in the order presented below.

MANUAL REDUCTION

This method has been extensively described.[6,11,14,17-21] Liberally apply an anesthetic jelly or water-soluble lubricant to the glans and foreskin. Do not coat the penile shaft or else it will be too slippery to facilitate an easy reduction.

Apply manual compression directly to the glans and edematous foreskin by grasping them with the palm of a gloved hand (**Figure 179-3**). Apply slow and steady pressure for 5 to 10 minutes. Many Emergency Physicians do not have the time or the strength to apply pressure to a patient's penis for 5 to 10 minutes. The parent of young children may be asked to provide the manual compression. Compression may be provided by circumferentially wrapping a bandage around the penis beginning at the glans and working toward the base of the penis.[22] Apply the bandage so that it places more pressure distally and less proximally. This will mobilize the edematous fluid from distal to proximal. The bandage can be an Ace Wrap, Elastoplast, or CoFlex gauze soaked in lidocaine jelly or gauze soaked in cold water.[10,14]

Remove the bandage or release manual compression after it has been applied for 5 to 10 minutes. Apply the index and middle fingers of both hands to surround the top of the penile shaft proximal to the phimotic ring and the ring fingers underneath the penile shaft (**Figure 179-4**). Place both thumbs adjacent to the urethral meatus (**Figure 179-4**). Push the glans proximally with the thumbs while the fingers simultaneously provide countertraction to pull the phimotic band over the glans. Apply continuous force until the phimotic band moves distal to the glans. This same technique can be applied if the patient has an indwelling urinary catheter (**Figure 179-5**).

FIGURE 179-3. Manual compression of the glans and foreskin to reduce the edema.

Alternatively, encircle the entire foreskin in one hand and pull distally while simultaneously pushing the glans proximally with the thumb of the opposite hand.[17] It is important to be very deliberate in the first attempt as the patient may become more anxious on subsequent attempts. This same technique can be applied if the patient has an indwelling urinary catheter.

ICED GLOVE TECHNIQUE

This technique combines cold to induce vasoconstriction and compression to reduce swelling.[9,17,21,23] It uses a glove to provide a circumferential ice pack to the foreskin to reduce edema. Half-fill a size 8 surgical glove with crushed ice and cold water. Squeeze the air from the glove and tie a knot in the wrist of the glove. Invaginate the thumb into the body of the glove. Liberally lubricate the glans and foreskin. Insert the penis into the invaginated thumb of the glove.

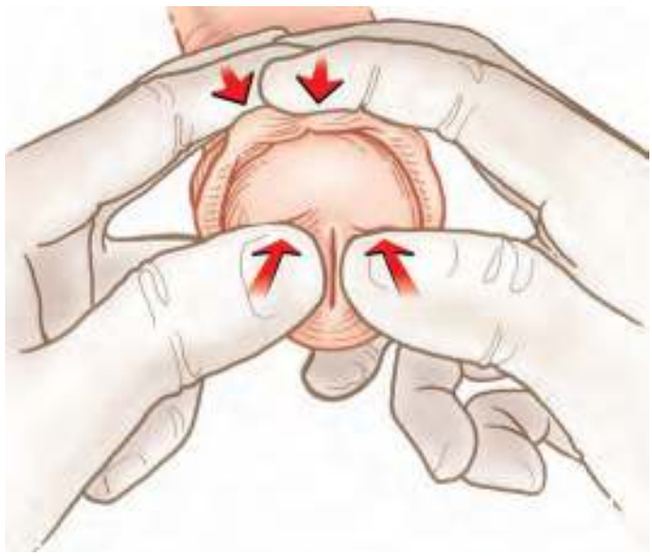

FIGURE 179-4. Manual reduction of a paraphimosis. The thumbs push the glans proximally while the fingers provide countertraction to slip the phimotic ring over the glans.

FIGURE 179-5. Manual reduction of a paraphimosis in the patient with an indwelling urinary catheter.

Apply manual pressure with a clenched fist or bandage for 5 to 10 minutes. Subsequent manual reduction was found to be successful after icing in 90% of cases.[9,11,17,23]

BABCOCK CLAMP TECHNIQUE

This technique can be used when manual reduction has been unsuccessful. It has a high rate of success in reducing a paraphimosis.[10,11,17] **This technique requires penile anesthesia (Chapters 156 and 177). Use only Babcock clamps. All other surgical clamps will crush and devitalize tissue.**

Apply six to eight Babcock clamps circumferentially around the phimotic ring (**Figure 179-6*A***). Place one edge of each clamp just proximal to the phimotic ring and the other edge just distal to the phimotic ring. **It is important to grasp enough tissue of the constricting ring to avoid tearing the skin once traction is applied.** Space the clamps evenly around the circumference of the penis. Grasp all the Babcock clamps in one hand. Simultaneously and slowly apply distal traction to the clamps to pull the phimotic ring over the glans (**Figure 179-6*B***). Remove the Babcock clamps after reduction of the phimotic ring. Examine the foreskin for any signs of traumatic injury from the clamps.

A variation of this technique uses two operators and two Adson forceps in place of Babcock clamps.[14,24] This method is briefly described for completeness but is not recommended even though it is described in the medical literature. Adson forceps are toothed forceps. Traction with an Adson forceps can result in the teeth pulling through the phimotic ring and tearing the foreskin. One operator applies an Adson forceps to the phimotic ring at the 3 o'clock position and at the 9 o'clock position. The first operator pulls the foreskin distally via the forceps while a second operator compresses the glans and pushes it proximally.

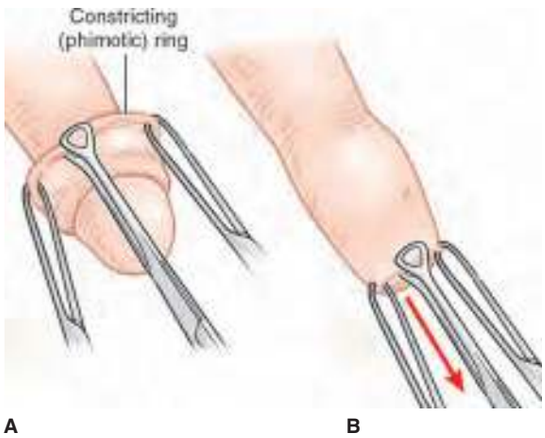

FIGURE 179-6. The Babcock clamp technique. ***A.*** Babcock clamps are placed along the phimotic ring. Note that only three Babcock clamps are seen in the illustration for the sake of clarity. ***B.*** Gentle traction is simultaneously placed on all the clamps to advance the phimotic ring over the glans.

NEEDLE DECOMPRESSION TECHNIQUE

This technique mechanically expresses fluid from the edematous foreskin via a series of puncture holes in the foreskin.[11,14,20,21,23,25,26] Penile anesthesia is required with this technique. Clean the penis of any dirt or debris. Drape and isolate a sterile field around the penis. Apply povidone iodine or chlorhexidine solution to the penis and allow it to dry. Perform a penile block (Chapters 156 and 177).

Insert a sterile, hollow-bore 18 gauge needle 3 to 5 mm deep into the edematous foreskin. The use of needles as large as 18 gauge to as small as 26 gauge have been described. An 18 gauge is preferred as it makes a hole large enough to allow blood and fluid to escape from the tissues and not close or clot closed. Continue to puncture holes circumferentially around the edematous foreskin (**Figure 179-7**). An average of 8 to 12 holes are required. Different recommendations exist for as few as 1 to as many as 20 punctures.[25,26] Wrap a gauze square around the foreskin and glans. Grasp the glans and foreskin in the palm of a gloved hand and apply manual compression. Edema fluid and blood will be expressed from the puncture holes allowing the foreskin to decompress. The gauze square will allow the Emergency Physician to maintain a grasp as the penis becomes slippery with blood and edema fluid. After the foreskin is decompressed, reduce it manually or with Babcock clamps.

DORSAL SLIT OF THE FORESKIN

The Emergency Physician must always be prepared to perform surgical techniques if nonsurgical reduction is unsuccessful. This technique involves the incision of the phimotic ring under strict sterile

FIGURE 179-7. The needle decompression technique.

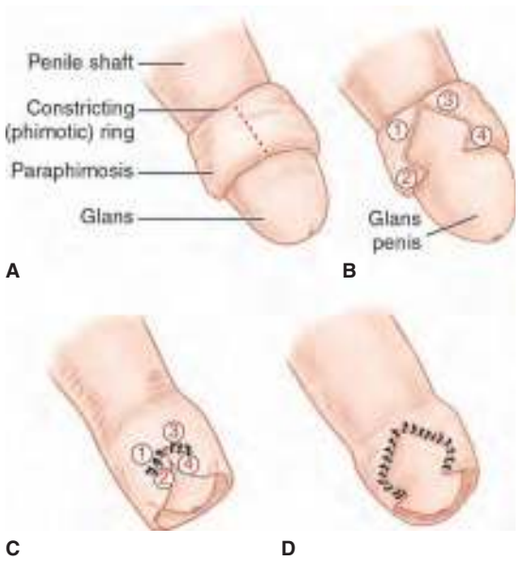

FIGURE 179-8. The dorsal slit of the foreskin. ***A.*** The dotted line represents the incision line. ***B.*** The foreskin opens after the incision is made. The numbers represent the edges of the incision. ***C.*** The foreskin has been reduced and the edges of the incision sewn shut. A small gap should remain in the midline that is free of sutures. ***D.*** Alternatively, sew the edges closed with a simple running stitch.

technique.[14,17-21] Place the patient supine and anesthetize the penis (Chapters 155 and 177) if it has not been done previously. Apply povidone iodine or chlorhexidine solution to the penis and let it dry. Apply sterile drapes to isolate a surgical field. Consider procedural sedation (Chapter 159) prior to performing this technique to alleviate the patient's pain and anxiety.

Clamp a straight-blade hemostat over the foreskin and phimotic ring at the 11 o'clock and 1 o'clock positions. Place one jaw of each hemostat beneath the phimotic ring and the other jaw on top of it. **Be careful not to clamp the skin on the shaft of the penis.** Pull the foreskin taut between the hemostats. Grasp and hold the hemostats with the nondominant hand or have an assistant hold them. Incise the foreskin and phimotic ring at the 12 o'clock position with a scissors or a #15 scalpel blade (**Figure 179-8*A***). **Be careful not to cut the skin on the shaft of the penis.** Remove the hemostats. The incision will open into a pentagonal shape (**Figure 179-8*B***). Reduce the foreskin over the glans. Cover the penis with sterile gauze and allow the edges of the incision to ooze for 10 to 15 minutes to decompress the foreskin.

This technique can be technically difficult for the non-Surgeon. As an alternative to the two-hemostat technique, a one-hemostat technique may be performed. Place one hemostat over the foreskin and phimotic ring at the 12 o'clock position (**Figure 179-8*A***). Remove the clamp after 1 to 2 minutes. Cut the crushed tissue with scissors through the phimotic ring. **Be careful not to cut the skin on the shaft of the penis.** The foreskin will open up (**Figure 179-8*B***).

Reduced the foreskin over the glans after it has been incised. Approximate the cut edges with 3–0 chromic suture using a simple interrupted stitch (**Figure 179-8*C***). Sew edge 1 and edge 2 on **Figure 179-8*C*** together. Sew edge 3 and edge 4 on **Figure 179-8*C*** together. A gap will remain at the 12 o'clock position that corresponds to the area of the initial incision. Alternatively, sew the cut edges with a simple running stitch (**Figure 179-8*D***). Loosely apply petrolatum gauze and gauze squares over the wound. Apply a piece of tape to hold the dressing on the penis. **Do not apply the tape circumferentially as this can result in penile ischemia.**

MODIFIED DORSAL SLIT OF THE FORESKIN

A modified dorsal slit technique has been described where only the phimotic ring is cut rather than the entire foreskin.[17] Clean, prep, anesthetize, and drape the penis as above. A penile block (Chapters 156 and 177) is the preferred method of anesthesia as injection into the distal penis is extremely painful. Infiltrate local anesthetic solution immediately under the phimotic ring at the 12 o'clock position using a 27 gauge needle as an alternative to a penile block. Be sure to raise a wheal both proximally and distally to the phimotic ring (along the dotted line in **Figure 179-9*A***).

Incise the phimotic ring with a #15 scalpel blade. **Incise only the phimotic ring. Do not extend the cut more than 3 mm proximally or distally to the phimotic ring.** The foreskin will spring open into a diamond-shaped defect (**Figure 179-9*B***). Reduce the foreskin. Cover the penis with gauze and allow the cut edges to ooze for 10 to 15 minutes to decompress the foreskin. Approximate the edges of the wound with 3–0 chromic suture in a running pattern (**Figure 179-9*C***). Apply a bandage of petrolatum gauze and gauze squares over the wound. Apply a piece of tape to hold the dressing on the penis. **The tape should not be applied circumferentially as this can cause ischemia to the penis.**

ALTERNATIVE TECHNIQUES

A variety of alternative techniques have been described in the literature. These techniques are variations on the basic principles previously discussed. We will briefly review the osmotic, hyaluronidase, and glans aspiration techniques.

OSMOTIC (SUGAR) TECHNIQUE

An alternative to surgical reduction is the osmotic technique.[10,14,20,21,23,27,28] It is an innovative and painless but time-intensive

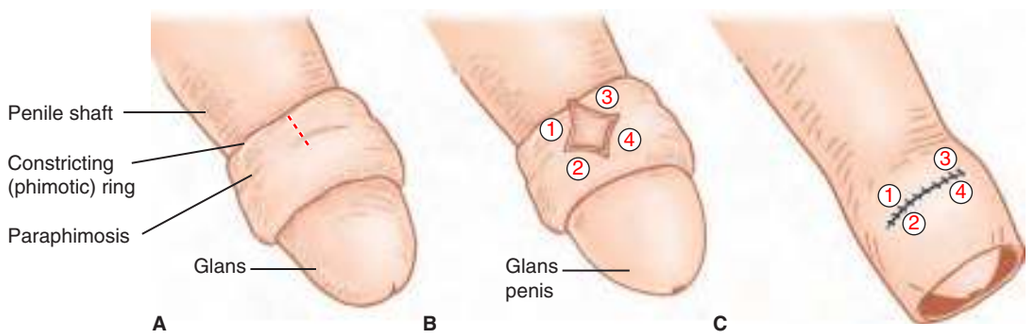

FIGURE 179-9. The modified dorsal slit of the foreskin. ***A.*** The dotted line represents the incision line. ***B.*** The foreskin opens after the incision is made. The numbers represent the edges of the incision. ***C.*** The foreskin has been reduced and the edges of the incision sewn shut.

method. The glans and foreskin are immersed or covered in an osmotic agent such as table sugar, 50% dextrose solution, or mannitol solution. An osmotic gradient is formed between the edematous foreskin and the sugar. The hypertonicity of the sugar draws the edema fluid out of the foreskin. The deflated foreskin can then be manually reduced over the glans. **This technique should not be performed if the foreskin or the glans is infected, gangrenous, or ulcerated.**

Place ordinary table sugar into the invaginated thumb of a large surgical glove. Insert the penis into the thumb of the glove as in the iced glove method. Alternatively, the penis may be wrapped distally to proximally in a gauze square saturated with a 50% dextrose solution or mannitol solution.[21,28] In all reports, the osmotic agent was left on the penis for at least 1 to 2 hours before manual reduction was successful. **Penile ischemia may be exacerbated by the prolonged time needed to reduce glans edema. The osmotic technique should not be considered a first-line procedure and should only be attempted after manual techniques have failed.**

HYALURONIDASE METHOD

Hyaluronidase (Amphadase, Amphastar Pharmaceuticals Inc., Rancho Cucamonga, CA; Hylenex, Halozyme Therapeutics, San Diego, CA; Vitrase, Ista Pharmaceuticals Inc., Irvine, CA) is a commercially available mammalian enzyme that causes hydrolysis of hyaluronic acid in the intercellular ground substance of connective tissue. It is widely used for subcutaneous hydration (Chapter 140), ophthalmologic surgery, and plastic surgery as a spreading agent. When injected into an edematous foreskin, hyaluronidase increases the tissue permeability, thus allowing the extracellular edema to disperse into the surrounding tissue of the penis and facilitate reduction of the paraphimosis.[10,12,14,20,21]

Clean, prep, drape, and anesthetize the penis. Inject hyaluronidase subcutaneously using a tuberculin syringe at various points around the circumference of the edematous foreskin. Use a total of 150 units (i.e., 1 mL) of hyaluronidase solution. An alternative is to inject the hyaluronidase into the puncture holes previously made from the needle decompression technique. The edema should resolve almost immediately. Manual compression may be applied to the foreskin for 1 to 2 minutes to help mobilize the fluid. Manually reduce the foreskin after the edema has decreased.

The advantage of this technique is the rapidity with which the edema resolves. Hyaluronidase is contraindicated in patients with penile infections or cancer. It may spread bacteria or malignant cells through the tissue planes. It is contraindicated if the foreskin or glans is gangrenous or ulcerated. Hyaluronidase may not be readily available in many Emergency Departments hindering the applicability of this technique.

GLANS ASPIRATION TECHNIQUE

This method is based on techniques used to treat priapism (Chapter 178).[29] The author reports using this technique on four patients with a 100% success rate and no complications. He recommended it for use when the foreskin is very friable and not amenable to vigorous manipulation. This technique is described here for the sake of completeness but is not recommended due to limited evidence.

Clean, prep, drape, and anesthetize the penis. Apply a sterile tourniquet to the penile shaft proximal to the phimotic ring. Use either umbilical tape or a Penrose drain. Insert a 20 gauge needle on a 10 mL syringe into the midline of the glans halfway between the meatus and the corona. Keep the needle and syringe parallel to the urethra. Advance the needle while aspirating with the syringe. Stop advancing the needle once blood is encountered. Continue to aspirate blood until the glans collapses completely. It is estimated that approximately 3 to 12 mL of blood will need to be aspirated depending on the size of the glans. Remove the needle. Grasp and firmly squeeze the glans with the nondominant hand. Maintain this pressure and release the tourniquet. Manually reduce the foreskin over the collapsed glans with the dominant hand.

ASSESSMENT

The successfully reduced paraphimosis should have the general appearance of a normal uncircumcised penis. The patient usually notes immediate pain relief. It is normal for residual edema to be present. Reassure the patient that the edema will spontaneously resolve over hours to days.[11,17] Observe the patient in the Emergency Department for 45 to 60 minutes to confirm hemostasis if invasive techniques were used. Observe the patient for full recovery from any sedation or procedural sedation techniques as per hospital policy.

AFTERCARE

Wound care following a surgical reduction is the same as that for any sutured laceration. Many Emergency Physicians do not dress the site at all. The site may be covered with antibiotic ointment or petrolatum gauze followed by a dry sterile gauze.[30] Instruct the patient on proper wound care. The patient should inspect the glans and foreskin three to four times a day if Babcock clamps, needle decompression, or surgical techniques were used to reduce the paraphimosis. They should return immediately to the Emergency Department if any signs of infection develop. Advise them to avoid intercourse or masturbation for 4 to 6 weeks.[30]

Infection after the reduction is an exceedingly rare complication due to the well-vascularized nature of the foreskin. Apply topical antibiotic ointment liberally and recommend it for aftercare. Oral antibiotics are generally not required if the foreskin is abraded by the reduction, torn by the reduction attempts, or torn by the invasive techniques (e.g., Babcock clamp technique, needle decompression, dorsal slit, modified dorsal slit, hyaluronidase technique, or glans aspiration).[31,32]

All patients should follow-up with a Urologist in 1 to 2 days. Instruct the patient not to retract the foreskin for 1 week after the reduction.[6] The definitive treatment to prevent recurrence is circumcision. This is typically delayed 7 to 10 days until any edema, inflammation, or ulceration has resolved.[20,21]

COMPLICATIONS

The most common complication is tearing of compromised penile skin during manual or Babcock clamp reduction. The treatment is to suture any tears that occur and apply antiseptic ointment.[6,17] Incomplete reduction and pain are possible complications of manual reduction. It is important to allow enough time for adequate dispersion of edema. Manual reduction may be associated with glans contusion and glans ischemia if manual pressure is too great. Cold injury can occur if the iced glove method is not properly monitored during use.[9]

Bleeding is a common complication in this very vascular tissue if surgical techniques are performed. Venous bleeding can be profuse. It is important to have good control of the tissue edges so that adequate hemostatic stitches can be placed. A compressive dressing may aid in hemostasis. Suture the penile shaft if it is lacerated during a surgical reduction. Infection may be a complication of the needle decompression, dorsal slit, modified dorsal slit, hyaluronidase, and glans aspiration techniques.

The glans aspiration method runs the risk of transecting the urethra if the needle is not advanced parallel to the urethra.

Complications of the hyaluronidase method range from minor ecchymoses at the injection site to anaphylaxis and shock if the hyaluronidase is inadvertently injected intravascularly.[12]

SUMMARY

A paraphimosis is a true urologic emergency and a problem that is best prevented. Replacement of the foreskin after urethral catheterization or glans cleaning is important. Early intervention is necessary and can prevent disastrous tissue loss.

There are no randomized controlled trials comparing the efficacy of the various reduction techniques. Our data primarily come from case series that do not demonstrate the superiority of any individual method.[23] Reduction techniques are still best attempted in a stepwise manner beginning with noninvasive methods and progressing to increasingly invasive techniques.

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180 Phimosis Reduction

Kevin O'Rourke

INTRODUCTION

A phimosis is a condition in which the foreskin cannot be retracted proximally to the glans penis.[1-18] The presence of a phimosis can interfere with cleaning under the foreskin, allows the accumulation of secretions and debris (i.e., smegma), and may predispose the patient to infections and possible malignancy.[18] It is classified into two subgroups as physiologic and pathologic. **Physiologic phimoses occur naturally in newborns.** In males younger than 4 years of age, it is normal for the foreskin not to be retractable and it should not be forcibly retracted.[19] In older boys and adults, the foreskin can usually be retracted without difficulty.[1] **A pathologic phimosis is the inability to retract the foreskin after it was previously retractable or after puberty, usually secondary to scarring of the foreskin (Figure 180-1).**[20,21] Surgical treatment for a phimosis has been known for hundreds of years.[2] A Byzantine surgeon by the name of Oribasius, in the fourth century AD, gave a seemingly well-acquainted description of a technique involving forced dilation

FIGURE 180-1. A phimosis in an adult. (Used from www.commons.wikimedia.org/wiki/Category:Phimosis#/media/File:Glande_intentando_salir.JPG.)

of the constrictive foreskin, scalloping out of its inner surface, then stretching it over a parchment-wrapped lead tube placed between the filleted skin and the glans.[2] Current techniques for the management of a phimosis in the Emergency Department are simple and remain an important intervention directed at relieving urinary obstruction.

ANATOMY AND PATHOPHYSIOLOGY

At birth there is a physiologic phimosis in the majority of male neonates. This is due to natural adhesions that exist between the foreskin and the glans of the penis. During the first 3 to 4 years of life, the penis grows and epithelial debris (i.e., smegma) accumulates under the foreskin. The debris gradually separates the foreskin from the glans. Intermittent penile erections aid in allowing the foreskin to eventually become retractable. The foreskin of most males will retract easily by the age of 4. **Forcible retraction should be categorically discouraged as this can result in scarring and constriction.**[1] Parents may report that the foreskin bulges during urination which is a normal finding with phimosis. For a nonobstructive phimosis in children, 4 to 6 weeks of topical steroids applied from the tip of the foreskin to the corona of the glans penis have shown excellent results in releasing the stubborn physiologic adhesions between the foreskin and the glans.[3,4,22,23]

A phimosis can be the cause or the effect of other medical conditions (**Figure 180-1**).[24] Recurrent infections (e.g., balanitis or balanoposthitis), repeated urinary catheterization, forceful foreskin retraction, and poor hygiene can lead to scarring of preputial orifices causing a pathologic phimosis. A pathologic phimosis may arise in diabetics due to the presence of glucose in their urine, giving rise to an infection of the foreskin. Once acquired, a phimosis can become a paraphimosis (Chapter 179) if the foreskin is retracted and not promptly reduced. Urinary tract infections can result from bacterial colonization of the phimosis or secondary to urinary obstruction from the phimosis. Males with a pathologic phimosis may report painful erections, hematuria, preputial pain, or a weakened urinary stream. Other complications of a phimosis include recurrent balanitis, local infections, urinary retention, carcinoma of the penis, the easy growth of venereal warts, and the easy acquisition of other sexually transmitted diseases. A phimosis may be the result of local trauma (e.g., zipper injuries, toilet seat trauma, or crush injuries) or the congenital lack of conversion to a mobile foreskin.[5] Penile carcinoma deserves special mention. Coexistent phimosis is seen in up to 52% of cases of penile carcinoma. **The need for timely follow-up should be impressed upon the patient regardless of the intervention required in the Emergency Department.**[6]

INDICATIONS

The sole indication for the surgical release of a phimosis is urinary obstruction that cannot be relieved by passing a urethral catheter (Chapter 173). A dorsal slit of the foreskin should be performed only after failure of noninvasive techniques. Most patients with a phimosis rarely require any emergency intervention and should be referred to a Urologist on an outpatient basis.

CONTRAINDICATIONS

The ability to pass a urinary catheter (i.e., a Foley or coudé) into the bladder eliminates the acute need for the reduction of a phimosis. Patients with bleeding disorders, gross infections of the foreskin, who are immunocompromised, or who have lesions of the foreskin should have a urinary catheter placed into the bladder (Chapter 173) rather than an incision of the phimotic foreskin. If a catheter cannot be inserted into the urethra in these patients, a Urologist should be consulted prior to any invasive procedures. Patients with a nonobstructing phimosis should be referred for an elective circumcision and not have a dorsal slit procedure.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Gauze squares
- Sterile drapes
- 1% lidocaine without epinephrine
- Lidocaine Uro-jet
- 27 gauge needle
- 5 mL syringes
- Hemostats, straight or straight Kelly clamps
- Urinary catheters (Foley, coudé)
- Sterile gloves
- Sterile drapes
- Scissors
- #15 scalpel blade on a handle
- 3–0 or 4–0 chromic catgut or Vicryl on a noncutting needle
- Needle driver
- Petrolatum gauze
- 1 to 2 inch bandage
- Topical antibacterial ointment

The use of tissue glue of the type normally found in Emergency Departments has been studied in children undergoing circumcision.[7] It was shown to increase infection, bleeding, and wound dehiscence when compared to suture. The use of suture must be recommended at this time to close the wound edges.

PATIENT PREPARATION

Explain the benefits, risks, potential complications, and aftercare of the procedure to the patient and/or their representative. An informed consent should be obtained and placed in the medical record.

Place the patient supine with their genitalia exposed. Prepare the penis for any intervention. Clean the penis of any dirt, debris, and discharge. Apply drapes to isolate the penis. Apply povidone iodine or chlorhexidine solution onto the penis. If possible, apply the solution under the foreskin using a cotton-tipped applicator. If the patient has pain in the area of the foreskin or if a procedure other than catheter insertion is to be performed, the penis should be anesthetized with a regional nerve block (Chapter 177).[25,26] Alternatively, infiltration of local anesthetic solution can be used to anesthetize the foreskin. Draw up 5 mL of local anesthetic solution without epinephrine into a syringe armed with a 27 gauge needle. Inject a subcutaneous wheal of local anesthetic solution 2 cm proximal to the distal end of the foreskin. Continue subcutaneous infiltration circumferentially around the penis with the local anesthetic solution. The patient may require intravenous sedation or procedural sedation (Chapter 159) prior to the injection of local anesthetic solution into the penis.

The use of topical anesthetics such as lidocaine jelly, prilocaine creams, or combinations (e.g., EMLA, LET, and LAT) is not recommended.[8] The application of these mixtures requires an occlusive dressing and 60 to 90 minutes for the anesthetic effect which may then be inadequate.

TECHNIQUES

TOPICAL CORTICOSTEROIDS

Up to 85% of pathologic phimoses that are mild to moderate will respond to the application of topical steroids to the preputial orifice.[22,23] It is reasonable for the Emergency Physician to prescribe 0.1% to 0.05% betamethasone dipropionate applied twice daily for 4 to 6 weeks with appropriate Urologic follow-up if there is no urinary obstruction or a urinary catheter can be placed.[11] The application of topical corticosteroids is the most cost-effective treatment if the circumstances permit.[19,23]

URETHRAL CATHETERIZATION

Attempt to pass a size 16 or 18 French Foley or coudé catheter into the bladder (Chapter 173). If this is too large, attempt to pass the largest possible catheter. Please refer to **Table 176-1** for an age-based list of proper catheter sizes. **Care should be taken to avoid forceful placement of the catheter between the foreskin and the glans.** Correct catheter placement will be confirmed by an outflow of urine.

FORESKIN DILATION

Most simply, the opening of the prepuce can be stretched. The patient's penis should be thoroughly anesthetized prior to performing this procedure (Chapter 177). Consideration should also be given to administering intravenous analgesics, intravenous sedation, or procedural sedation (Chapter 159).

Insert the jaws of a closed hemostat just inside the foreskin. Slowly open the arms of the hemostat slightly (i.e., 2 to 3 mm). **Palpate the foreskin to feel both jaws of the hemostat. If the jaws are not palpable, immediately remove the instrument as one of the jaws may be in the urethra.** If both jaws of the hemostat are palpable, open the arms to dilate the opening of the foreskin. Remove the hemostat. Clean the glans and undersurface of the foreskin with povidone iodine or chlorhexidine solution. Insert, using sterile technique, a urinary catheter if required (Chapter 173).

This approach is not the ideal technique as it carries significant risk of injury to the patient. Inadvertent placement of the hemostat jaw in the urethra can lacerate the urethra and glans of the penis. Dilating the foreskin can cause irregular tears. Injury may require operative intervention and circumcision to correct any iatrogenic trauma.

DORSAL SLIT OF THE FORESKIN

The preferred method to correct an obstructing phimosis in the Emergency Department is the dorsal slit procedure. The patient's penis should be thoroughly anesthetized prior to performing this procedure (Chapter 177). Consideration should also be given to administering intravenous analgesics, intravenous sedation, or procedural sedation (Chapter 159).

Insert the bottom jaw of a straight hemostat between the foreskin and glans at the 12 o'clock position (**Figure 180-2*A***). For those individual patients or cultural situations where dorsal incision of the foreskin, much less excision, is cosmetically unacceptable, a ventral approach may be substituted which will yield an apparently uncircumcised penis without obstructive symptoms.[9] Advance the hemostat until the tip of the jaw is at the coronal sulcus (**Figure 180-2*A***) where the foreskin attaches to the penis. Depending on the etiology of the phimosis, adhesions may be encountered. These should gently be broken as the hemostat is advanced. The skin of the prepuce is relatively thin and the jaw of the hemostat should be easily palpated. The tip of the jaw should be seen to tent the skin at the coronal sulcus when properly placed (**Figure 180-2*B***). **It cannot be overemphasized that the Emergency Physician must be confident that the instrument has not been inadvertently placed in the urethra. If the jaw of the hemostat cannot be felt and cannot be seen tenting the skin of the foreskin, remove the hemostat and reinsert it.**

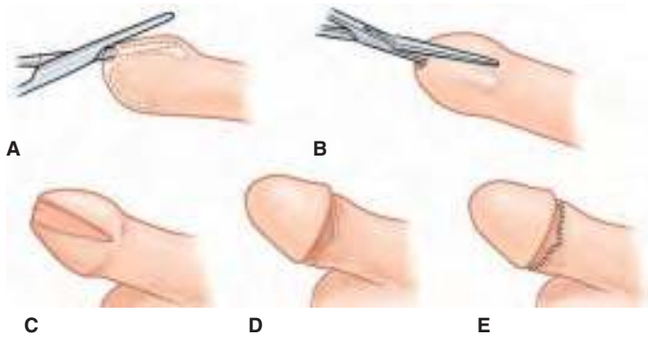

FIGURE 180-2. Dorsal slit of the foreskin. ***A.*** A hemostat is inserted under the foreskin and advanced to the coronal sulcus. ***B.*** The hemostat is elevated to tent the skin and confirm it is properly placed. ***C.*** The dorsal slit has been made. ***D.*** The foreskin is retracted. ***E.*** Interrupted sutures are placed along the cut edge of the foreskin.

Once properly placed, close the hemostat to crush the foreskin at the 12 o'clock position. Allow the hemostat to remain closed for 2 to 3 minutes to thoroughly crush the foreskin. Remove the hemostat. Insert a scissors and advance it with the same attention to position the tip at the coronal sulcus. Incise the crushed tissue to the level of the coronal sulcus (**Figure 180-2*C***). If straight scissors are not available, a #15 blade may be used to cut the crushed skin after another instrument is placed underneath the foreskin to protect the glans from injury. **This method of using a scalpel blade is dangerous and not recommended because control is reduced and the potential for error is unnecessarily increased.**

Retract the cut foreskin. This will leave an open wound edge on both sides of the midline (**Figure 180-2*D***). Suture the open wound edges using 3–0 or 4–0 chromic gut suture in an interrupted or running pattern. Begin suturing from the midline to the distal end of the incision (**Figure 180-2*E***).[5] Return the foreskin to its "resting" position to guard against a newly acquired iatrogenic paraphimosis. Generously apply a topical antibacterial ointment over the suture line and loosely cover it with a bandage of petrolatum gauze and gauze squares. Apply a piece of tape to hold the dressing on the penis. **The tape should not be applied circumferentially as this can cause ischemia to the penis.**

Should it be desired, a complete circumcision can be performed after the dorsal slit incision. Using two hemostats, make a series of crushing bites along the foreskin at the level of the coronal sulcus. **Use caution not to crush the skin on the shaft of the penis.** Using scissors, cut the foreskin along the crushed tissue. Approximate the wound edges using 3–0 or 4–0 chromic gut in an interrupted pattern. Carcinoma should always be considered as an etiology of a phimosis and all excised tissue should be sent to pathology for histologic evaluation.[18]

ALTERNATIVE TECHNIQUE

PREPUTIOPLASTY

The preputioplasty, although not typically performed in the Emergency Department, is an alternative surgical treatment to the more radical dorsal slit and circumcision. This tissue-sparing surgical technique can achieve full resolution of a phimosis. The preputioplasty is a conservative, less traumatic, and less invasive procedure.

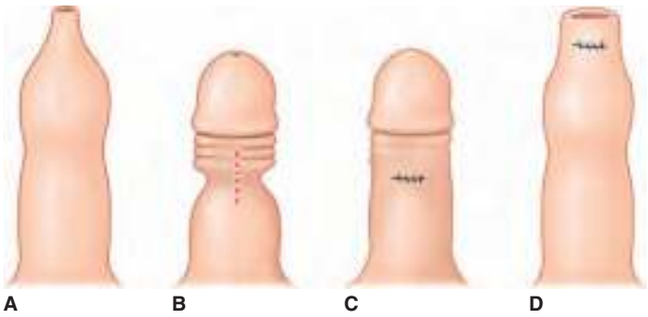

FIGURE 180-3. The preputioplasty. ***A.*** Penis with a tight phimotic ring making it difficult to retract the foreskin. ***B.*** Foreskin retracted under anesthesia with the phimotic ring or stenosis constricting the shaft of the penis and creating a "waist." The dotted line represents the vertical incision line. ***C.*** Incision closed laterally with interrupted sutures. ***D.*** Penis with the loosened foreskin replaced over the glans.

The procedure consists of one or more short longitudinal incisions that release the stenosis and are then closed transversely allowing a widening of the phimotic ring and loosening of foreskin (**Figure 180-3**).[13]

Clean, prep, anesthetize, and prepare the penis as described previously. Retract the foreskin until it is completely proximal to the glans (**Figure 180-3*B***). The phimotic ring will constrict the penile shaft (**Figure 180-3*B***). Carefully make a superficial incision in the phimotic ring with a #15 scalpel blade or fine scissors (**Figure 180-3*B***). The phimotic ring will open. Allow the wound to ooze for a few minutes. Close the wound using 3–0 or 4–0 chromic gut suture in an interrupted pattern (**Figure 180-3*C***). Return the loosened foreskin over the glans (**Figure 180-3*D***). Generously apply a topical antibacterial ointment over the suture line and loosely cover it with a bandage of petrolatum gauze and gauze squares. Apply a piece of tape to hold the dressing on the penis. **The tape should not be applied circumferentially as this can cause ischemia to the penis.**

ASSESSMENT

Regardless of the technique, observe the patient in the Emergency Department for 45 to 60 minutes to confirm hemostasis. Observe the patient for a full recovery from procedural sedation techniques, if performed, as per hospital policy.

AFTERCARE

The patient may be discharged with Urologic follow-up within 48 hours. Instruct the patient to return to the Emergency Department immediately if the wound is red, has any discharge, or is swollen, or if fever develops. The patient should be given oral analgesics for pain control. Patients should be discharged on oral antibiotics whose spectrum covers skin flora. Cephalexin, 500 mg orally four times a day, is usually adequate. Between discharge and examination by a Urologist, the patient should practice gentle daily washing of the penis with soap, avoid placing any powders or creams in the area, and check the wound three to four times daily for signs of infection. Sexual intercourse and masturbation should be avoided until the incisions have completely healed.

The dorsal slit procedure will result in a "beagle ear"-like deformity. While not problematic in most cases, it may increase the chance of getting the foreskin entrapped in a zipper. The Urologist will often perform an elective circumcision for cosmetic purposes.

COMPLICATIONS

Direct mechanical injury to the glans or urethra by inadvertent placement of instruments into the urethra could be devastating but is easily avoidable by following proper technique. Increased bleeding may result if the skin is not crushed by the hemostat before it is cut with the scissors.[5] It is also important to avoid advancing the clamp, and therefore the incision, beyond the comfortable limitation of the coronal sulcus. Otherwise, the skin on the penile shaft will be cut. This can result in a cosmetic defect and possibly skin sloughing requiring a skin graft. Lacerations to the penile shaft skin should be sutured with an absorbable suture. Wound infection and dehiscence are a possibility with any incision and greatly reduced in conscientious patients.

SUMMARY

From a variety of disease processes, the endpoint of a phimosis causing urinary obstruction lends itself to simple correction by the Emergency Physician. If there is not complete urinary obstruction, a trial of topical corticosteroids is simple, cost effective, and may result in the resolution of the phimosis. Careful performance of the described techniques will result in an excellent functional and aesthetic foreskin repair.

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181 Dorsal Slit of the Foreskin

Carlos J. Roldan

INTRODUCTION

Acute conditions affecting the foreskin that must be recognized in the Emergency Department include a phimosis and a paraphimosis. The Emergency Physician must be able to accurately identify and manage these conditions as well as recognize when an urgent Urology consultation is necessary. A dorsal slit of the prepuce or foreskin can be performed in the Emergency Department. This technique is used to relieve strangulation of the glans by a paraphimosis (**Figure 181-1**) or to aid in the visualization of the urethral meatus in patients with a phimosis (**Figure 181-2**).[1] The technique is easy to learn and simple to perform.

ANATOMY AND PATHOPHYSIOLOGY

The anatomy of the penis is simple (**Figure 181-3**). The prepuce, or foreskin, is the skin originating just proximal to the corona that encircles the glans and often extends beyond it.[2] It may be incomplete, primarily at the ventral midline or the frenulum. The frenulum is the fusion site of the preputial and urethral folds. The glans is composed of the corpus spongiosum that enlarges to cover the tips of the corpora cavernosa. It has less erectile tissue than the corpora cavernosa and contains the urethra.[2]

The blood supply to the foreskin and glans is provided by the superficial penile arteries. The arteries are derived from the inferior external pudendal arteries which are branches of the femoral arteries. The penile arteries travel in the superficial fascia of the penis and above Buck's fascia.[3] The left and right superficial penile arteries freely communicate over the midline. Superficial veins accompany the arteries and ultimately drain to the saphenous veins in the thighs. The lymphatics travel deep to Buck's fascia and ultimately empty into the inguinal chain of lymph nodes. The somatic nerves to the foreskin are derived from the pudendal nerves.[4]

FIGURE 181-1. A paraphimosis. (Photo courtesy of Eric F. Reichman, PhD, MD.)

FIGURE 181-2. A phimosis. (Used from Plisman at www.commons.wikimedia.org.)

A dorsal slit is performed to reduce a paraphimosis or phimosis when other less invasive techniques are unsuccessful. A paraphimosis is the inability to replace the retracted foreskin over the glans into its naturally occurring position (Chapter 179). It is considered an emergency since prolonged retraction of the foreskin creates a constricting ring that quickly compromises vascular and lymphatic circulation with eventual engorgement and necrosis of the glans and foreskin. A phimosis is the inability to retract the distal foreskin over the glans penis (Chapter 180).[5,6] A phimosis is no longer present once the foreskin can be retracted so that the glans penis partially appears.

INDICATIONS

Perform a dorsal slit of the foreskin to release a paraphimosis or a phimosis if noninvasive and lesser invasive techniques are unsuccessful. A paraphimosis is considered an emergency since prolonged retraction of the foreskin leads to swelling of the prepuce resulting in strangulation injury to the glans.[1] Release a phimosis if it causes urinary retention. The phimotic foreskin should not be forcibly retracted as this can result in a tearing of the foreskin.

CONTRAINDICATIONS

There are no absolute contraindications to the reduction of a paraphimosis. A dorsal slit in children should be performed by a Pediatric Urologist, a Urologist, or after consultation with a Urologist. Perform a dorsal slit in a child for the relief of a paraphimosis only after noninvasive and lesser invasive techniques have been unsuccessfully attempted (Chapter 179).[7-9]

The ability to pass a urinary catheter (i.e., Foley, coudé, filiforms, and followers) into the bladder eliminates the acute need for reduction of a phimosis (Chapter 173). Place a urinary catheter into the bladder rather than an incision of the phimotic foreskin if possible in patients with bleeding disorders or gross infections of the foreskin, patients who are immunocompromised, or patients who have lesions of the foreskin. Consult a Urologist prior to any invasive procedures if a catheter cannot be inserted into the urethra of

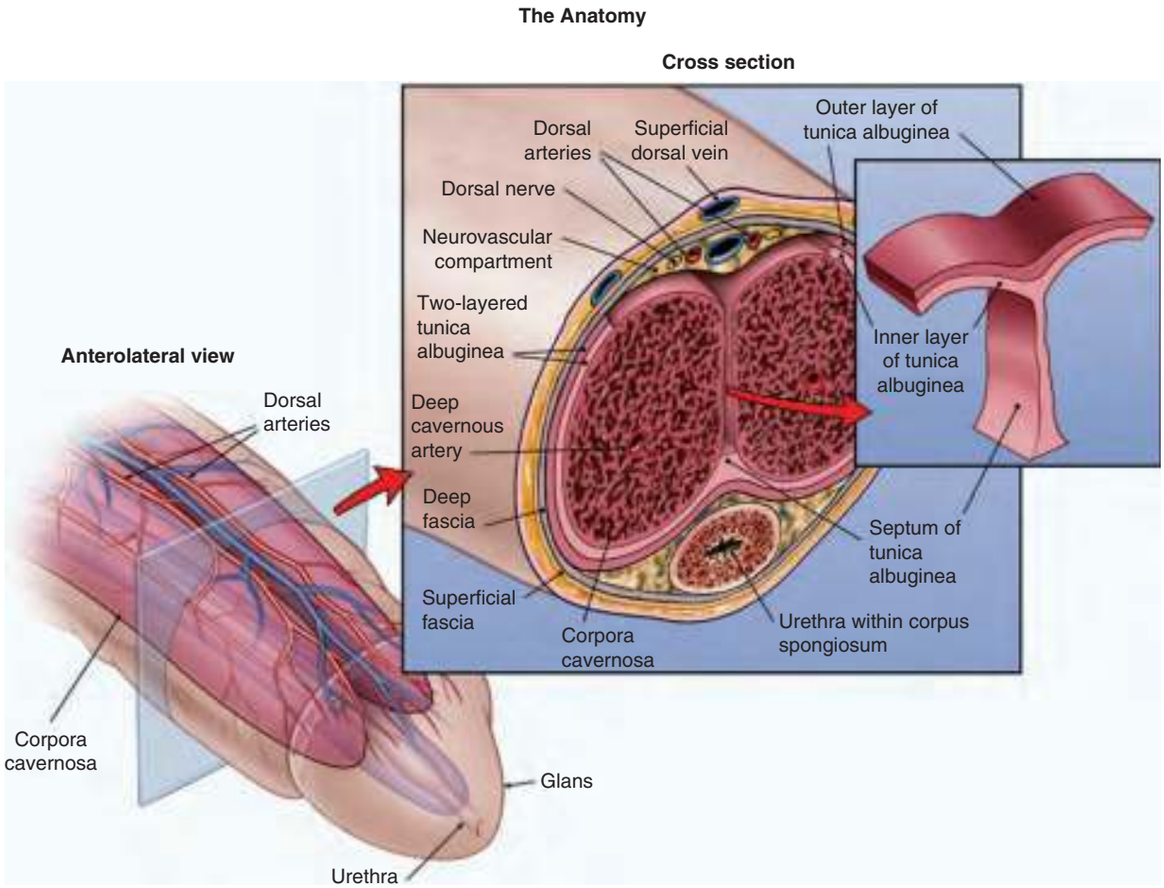

FIGURE 181-3. Anatomy of the penis. (Photo courtesy of www.peyronies.org.)

these patients. Treat patients with a nonobstructing phimosis with topical corticosteroids and referral for elective circumcision; these patients should not have a dorsal slit procedure in the Emergency Department.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Local anesthetic solution without epinephrine
- 5 mL syringe
- 27 gauge needle
- Hemostats, straight or straight Kelly
- #15 scalpel blade on a handle
- Needle driver
- 3–0 or 4–0 sutures (i.e., chromic catgut, Dexon, or Vicryl)
- Petrolatum gauze
- Metzenbaum scissors
- Suture scissors
- 4 × 4 gauze squares
- Sterile gloves
- Sterile drapes
- Topical antibacterial ointment

The use of tissue glue of the type normally found in Emergency Departments has been studied in children undergoing circumcision.[10] It was shown to increase infection, bleeding, and wound dehiscence when compared to suture. The use of suture is highly recommended to close the wound edges after the release of a paraphimosis or a phimosis.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Prepare the penis for any intervention. Clean the penis of any dirt, debris, and discharge. Apply drapes to isolate the penis. Apply povidone iodine or chlorhexidine solution onto the penis. Apply the solution under the foreskin using a cotton-tipped applicator if it is possible. Anesthetize the penis (Chapter 177) if the patient has pain of the foreskin or if a procedure other than catheter insertion is to be performed. An alternative is the infiltration of local anesthetic solution. Draw up 5 mL of local anesthetic solution without epinephrine into a syringe armed with a 27 gauge needle. Inject a subcutaneous wheal of local anesthetic solution 2 cm proximal to the distal end of the foreskin. Continue subcutaneous infiltration circumferentially around the penis with the local anesthetic solution. The patient may require intravenous sedation or procedural sedation (Chapter 159) prior to the injection of local anesthetic solution into the penis.

The use of topical anesthetics (Chapter 154) such as lidocaine jelly, prilocaine creams, or combinations (EMLA, LET, LAT) is not recommended.[11] The application of these mixtures requires an

occlusive dressing and 60 to 90 minutes for the anesthetic effect which may then be inadequate. Their use will delay the performance of the procedure.

TECHNIQUES

DORSAL SLIT OF THE PARAPHIMOTIC FORESKIN

Always be prepared to perform surgical techniques if nonsurgical reduction is unsuccessful. This technique involves the incision of the phimotic ring using strict sterile technique.[12-14] Place the patient supine and anesthetize the penis if it has not been done previously. Apply povidone iodine or chlorhexidine to the penis and let it dry. Apply sterile drapes to isolate a surgical field. Consider the administration of parenteral sedation prior to performing this procedure to alleviate the patient's anxiety.

Clamp a straight-blade hemostat over the foreskin and phimotic ring at the 11 o'clock and 1 o'clock positions. Place one jaw of each hemostat beneath the phimotic ring and the other jaw on top of it. **Be careful not to clamp the skin on the shaft of the penis.** Pull the foreskin taut between the hemostats. Grasp the hemostats with the nondominant hand or have an assistant hold them. Incise the foreskin and phimotic ring at the 12 o'clock position with a scissors or #15 scalpel blade (**Figure 181-4*A***). **Be careful not to cut the skin on the shaft of the penis.** Remove the hemostats. The incision will open into a pentagonal shape (**Figure 181-4*B***). Reduce the foreskin over the glans. Cover the penis with sterile gauze and allow the edges of the incision to ooze for 10 to 15 minutes.

This technique can be technically difficult to perform. A one-hemostat technique may be performed as an alternative to the two-hemostat technique. Place one hemostat over the foreskin and phimotic ring at the 12 o'clock position (**Figure 181-4*A***). Remove the clamp after 1 to 2 minutes. Cut the crushed tissue with scissors through the phimotic ring. **Be careful not to cut the skin on the shaft of the penis.** The foreskin will open as described (**Figure 181-4*B***). Bloody edematous fluid will ooze out and this can be sponged. Reduce the foreskin over the glans. **A total reduction of the paraphimosis must be accomplished to obtain satisfactory results.** Cover the penis with sterile gauze and allow the edges of the incision to ooze for 10 to 15 minutes.

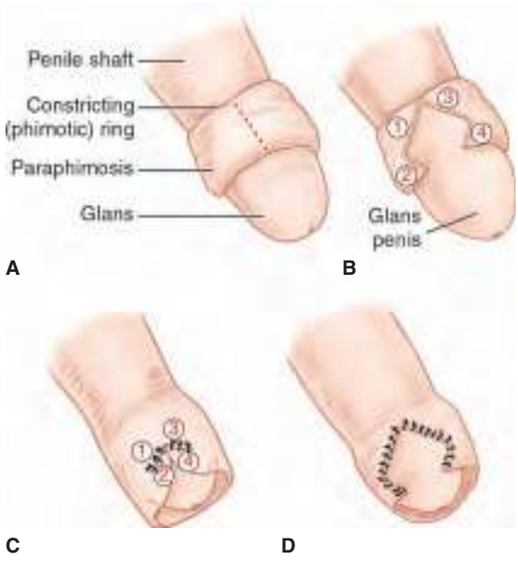

FIGURE 181-4. Dorsal slit of the paraphimotic foreskin. ***A.*** The dotted line represents the incision line. ***B.*** The foreskin opens after the incision is made. The numbers represent the edges of the incision. ***C.*** The foreskin has been reduced and the edges of the incision sewn shut. A small gap should remain in the midline that is free of sutures. ***D.*** Alternatively, sew the edges closed with a simple running stitch.

Approximate the cut edges with 3–0 chromic suture using a simple interrupted stitch after the foreskin has been incised and reduced over the glans (**Figure 181-4*C***). Sew edge 1 and edge 2 on figure 181-4C together. Sew edge 3 and edge 4 on figure 181-4C together. A gap will remain at the 12 o'clock position that corresponds to the area of the initial incision. Alternatively, sew the cut edges with a simple running stitch (**Figure 181-4*D***). Loosely apply petrolatum gauze and gauze squares over the wound. Apply a piece of tape to hold the dressing on the penis. **The tape should not be applied circumferentially as this can cause ischemia to the penis.**

MODIFIED DORSAL SLIT OF THE PARAPHIMOTIC FORESKIN

A modified dorsal slit technique has been described where only the phimotic ring is cut rather than the entire foreskin.[12,15] Clean, prep, anesthetize, and drape the penis as described previously. A penile block is the preferred method of anesthesia because injection onto the distal penis is extremely painful. An alternative to a penile block is to infiltrate under the phimotic ring at the 12 o'clock position with local anesthetic solution without epinephrine using a 27 gauge needle. Be sure to raise a wheal both proximally and distally to the phimotic ring (along the dotted line in **Figure 181-5*A***).

Incise the phimotic ring with a #15 scalpel blade. **Incise only the phimotic ring. Do not extend the cut more than 3 mm proximally or distally to the phimotic ring.** Once incised, the foreskin will spring open into a diamond-shaped defect (**Figure 181-5*B***). Reduce the foreskin. Cover the penis with gauze and allow the cut edges to ooze for 10 to 15 minutes to decompress the foreskin. Approximate the edges of the wound with 3–0 chromic suture in an interrupted or running pattern (**Figure 181-5*C***). Apply a bandage of petrolatum gauze and gauze squares over the wound. Apply a piece of tape to hold the dressing on the penis. **The tape should not be applied circumferentially as this can cause ischemia to the penis.**

DORSAL SLIT OF THE PHIMOTIC FORESKIN

The preferred method to correct an obstructing phimosis in the Emergency Department setting is the dorsal slit procedure. The patient's penis should be thoroughly anesthetized prior to performing this procedure. Consideration should also be given to administering intravenous analgesics, intravenous sedation, or procedural sedation.

Insert the bottom jaw of a straight hemostat between the foreskin and glans at the 12 o'clock position (**Figure 181-6*A***). A ventral approach may be substituted. This will yield an apparently uncircumcised penis without obstructive symptoms. For those individual patients or cultural situations where dorsal incision of the foreskin, much less excision, is cosmetically unacceptable.[12] Advance the hemostat until the tip of the jaw is at the coronal sulcus (**Figure 181-6*A***). The coronal sulcus is where the foreskin attaches to the penis. Adhesions may be encountered depending on the cause of the phimosis. Gently break the adhesions as the hemostat is advanced. The skin of the prepuce is relatively thin and the jaw of the hemostat is easily palpated. Visualize the tip of the jaw tenting the skin at the coronal sulcus when properly placed (**Figure 181-6*B***). **It cannot be overemphasized that the Emergency Physician must be confident that the instrument has not been inadvertently placed in the urethra. Remove the hemostat and reinsert it if the jaw of the hemostat cannot be felt and cannot be seen tenting the skin of the foreskin.**

Close the hemostat once properly placed. Allow the hemostat to remain closed for 2 to 3 minutes to crush the skin. Remove the

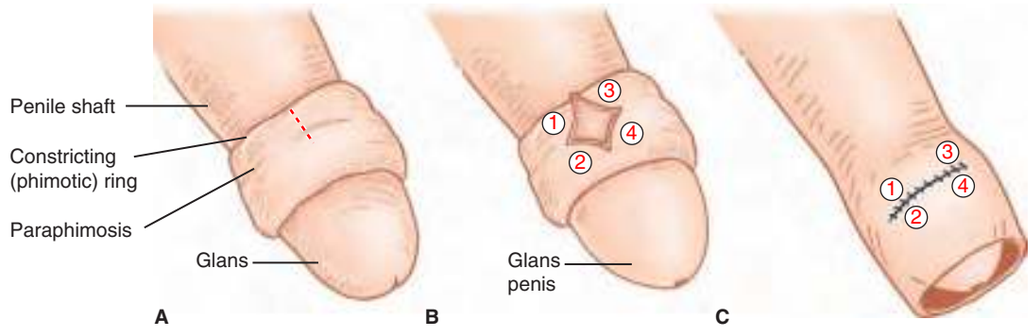

FIGURE 181-5. Modified dorsal slit of the paraphimotic foreskin. ***A.*** The dotted line represents the incision line. ***B.*** The foreskin opens after the incision is made. The numbers represent the edges of the incision. ***C.*** The foreskin has been reduced and the edges of the incision sewn shut.

hemostat. Insert a scissors and advance it with the same attention to position the tip at the coronal sulcus. Incise the crushed tissue to the level of the coronal sulcus (**Figure 181-6*C***). A #15 blade may be used to cut the crushed skin after another instrument is placed underneath the foreskin to protect the glans from injury. **This method of using a scalpel blade is dangerous and not recommended because control is reduced and the potential for error is unnecessarily increased.**

Retract the cut foreskin. This will leave an open wound edge on both sides of the midline (**Figure 181-6*D***). Cover the penis with gauze and allow the cut edges to ooze for 10 to 15 minutes. Approximate the open wound edges using 3–0 or 4–0 chromic gut suture in an interrupted or running pattern. Begin suturing from the midline to the distal end of the incision (**Figure 181-6*E***).[12] Return the foreskin to its "resting" position to guard against a newly acquired iatrogenic paraphimosis. Apply a bandage of petrolatum gauze and gauze squares over the wound. Apply a piece of tape to hold the dressing on the penis. **The tape should not be applied circumferentially as this can cause ischemia to the penis.**

ASSESSMENT

The foreskin is now be retractable to a sufficient degree to allow visualization of the meatus when performed for a phimosis or reduced over the glans to relieve pressure when performed for a paraphimosis. The successfully reduced paraphimosis should have the general appearance of a normal uncircumcised penis. It is normal for residual edema to be present. Reassure the patient that the edema will spontaneously resolve over hours to days. Observe the patient in the Emergency Department for 45 to 60 minutes to confirm hemostasis. Observe the patient for a full recovery from any sedation or procedural sedation techniques as per hospital policy.

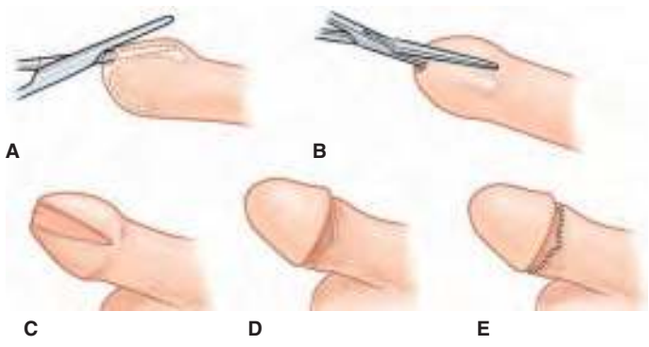

FIGURE 181-6. Dorsal slit of the phimotic foreskin. ***A.*** A hemostat is inserted under the foreskin and advanced to the coronal sulcus. ***B.*** The hemostat is elevated to tent the skin and confirm it is properly placed. ***C.*** The dorsal slit has been made. ***D.*** The foreskin is retracted. ***E.*** Interrupted sutures have been placed in the cut edges of the foreskin.

AFTERCARE

Wound care following a surgical reduction is the same as that for any sutured laceration. Many physicians do not dress the incision site. It may be covered with antibiotic ointment or petrolatum gauze and dry sterile gauze.[11] Council the patient on proper wound care. The patient should inspect the glans and foreskin three to four times a day. They should return immediately to the Emergency Department if any signs of infection develop. Advise the patient to avoid intercourse or masturbation for 4 to 6 weeks.[11]

Prescribe antibiotics that cover gram-positive organisms. An extended-spectrum penicillin or first-generation cephalosporin is most frequently prescribed. Cephalexin, 500 mg orally four times a day, is usually adequate.

All patients need to follow-up with a Urologist in 1 or 2 days. The definitive treatment is circumcision, which is typically delayed 7 to 10 days until any edema, inflammation, or ulceration has resolved.[13,14,16]

COMPLICATIONS

Direct mechanical injury to the glans or urethra by inadvertent placement of instruments into the urethra could be devastating but is easily avoidable by following proper technique. Increased bleeding may result if the skin is not crushed by the hemostat before it is cut with the scissors. It is also important to avoid advancing the clamp, and therefore the incision, beyond the comfortable limitation of the coronal sulcus. The skin on the penile shaft will otherwise be cut. This can result in a cosmetic defect and possibly skin sloughing requiring a skin graft. Wound infection and dehiscence are a possibility with any incision and greatly reduced in conscientious patients.

Bleeding is a common complication in this very vascular tissue. Venous bleeding can be profuse and it is important to have good control of the tissue edges so that adequate hemostatic stitches can be placed. A compressive dressing may aid in hemostasis. Suture the penile shaft if it is lacerated during a surgical reduction.

SUMMARY

A dorsal slit is an easy and rapid way to relieve strangulation pressure from a paraphimosis or to allow visualization of the urethral meatus from an obstructing phimosis. This procedure is performed primarily in emergency situations as the cosmetic result is usually suboptimal. Refer patients able to urinate with a phimosis to a Urologist before performing a dorsal slit. The dorsal slit procedure

should only be performed when a phimosis is obstructing and a urethral catheter cannot be placed. A paraphimosis is always an emergency. Often this will reduce with less invasive techniques that should be attempted first. Severe cases will require a dorsal slit.

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182 Manual Testicular Detorsion

Steven Go

INTRODUCTION

Testicular torsion was first described in the English literature in 1907.[1] Testicular torsion occurs when the testicle twists around its axis and causes vascular compromise of the testicle (**Figure 182-1**). **Testicular torsion is a clinically diagnosed and time-sensitive emergency.** The goal of the Emergency Physician is to suspect the diagnosis, make the diagnosis, and facilitate rapid operative detorsion by a Urologist. Manual detorsion can be attempted while awaiting surgical intervention.

The incidence of testicular torsion is approximately 3.5 to 4.5 in 100,000 males < 25 years of age.[2,3] It primarily affects young persons with a bimodal age distribution. The most common occurrence is in the neonatal period with the second peak at approximately age 13.[4] Most cases of testicular torsion occur in patients < 21 years of age.[5]

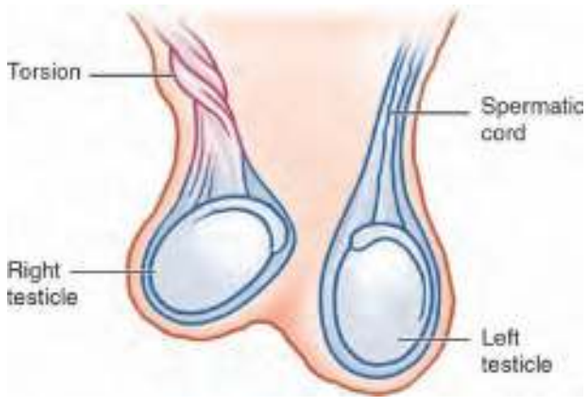

FIGURE 182-1. Torsion of the right testicle. The testicle lies horizontally and in a higher position than the normal testicle.

Rare cases of testicular torsion in men > 59 years of age have been reported.[6-9] The age range at risk for torsion is broader than commonly thought.

Testicular torsion cannot be ruled out by history and physical examination.[10,11] There are commonly associated history and physical examination features. Testicular torsion classically presents with acute onset of excruciating pain in the unilateral testicle or scrotum. Cases of bilateral testicular torsion can occur.[12] Testicular torsion may be associated with a recent history of genital trauma including self-mutilation in up to 8% of cases.[13,14] Associated symptoms may include a low-grade fever, nausea, and vomiting.[15,16] A history of a prior orchiopexy (i.e., surgical fastening of the testicle) does not exclude a testicular torsion.[17] Patients may present immediately upon the onset of pain or days later. Delayed presentation can sometimes occur in patients who have a history of chronic intermittent torsion.

Patients typically have testicular pain to palpation and a painful, edematous scrotum. The testicle can present with a high-riding horizontal lie with anterior rotation of the epididymis (**Figure 182-1**).[16,18,19] This testicle positioning is not universal.[10] A cremasteric reflex was typically absent in one trial.[20] The cremasteric reflex remains present in testicular torsion in 8% to 40% of cases.[19-24] Prehn's sign (i.e., relief of pain with elevation of the scrotum) is classically present in epididymitis and absent in testicular torsion. It too is an imperfect discriminator.[25-28] Color Doppler ultrasound (US) studies can be misleading because scrotal blood flow can be misinterpreted as testicular blood flow.[11,29] A lack of flow assumes testicular torsion is present with a sensitivity of 45% to 60%.[30]

Color Doppler US is the initial test of choice because of its greater availability, lower cost, lack of adverse effects, and no ionizing radiation. It has largely replaced testicular radionuclide scintigraphy for the confirmation of testicular torsion.[31] **Emergent consultation with a Urologist should not be delayed for the results of a radiologic study when testicular torsion is strongly suspected because outcomes worsen the longer detorsion is delayed.**[32-34] Color Doppler US has a reported sensitivity for testicular torsion of only 85.7% to 90%.[35-38] This sensitivity may be improved by examination of the spermatic cord to the level of the internal ring.[39] **Color Doppler US cannot completely exclude testicular torsion. The patient should be surgically explored by a Urologist if testicular torsion is strongly suspected and no other competing diagnosis can be definitively confirmed by color Doppler US.**[29]

Image the testes and spermatic cord to the abdomen if using US.[40-42] Look at the gray scale appearance, Doppler of arterial and venous flow, and the arterial Doppler waveform (**Figures 182-2**

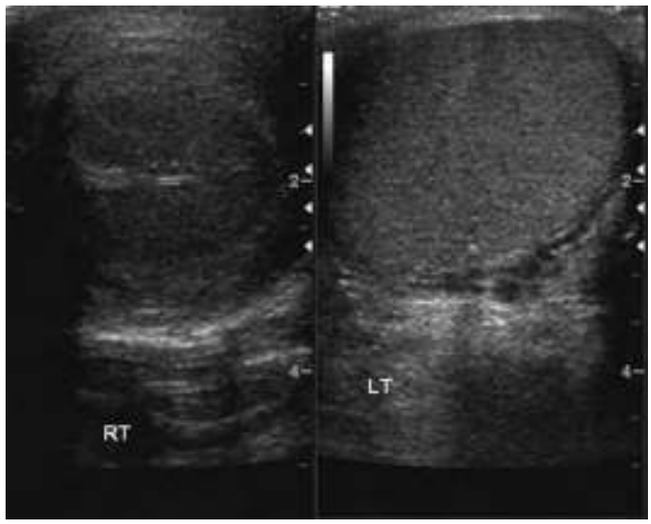

FIGURE 182-2. Gray scale US image showing abnormal hypoechogenicity in the right testicle (left side of picture) and a normal left testicle (right side of picture) for comparison. (Photo used with permission from www.iame.com.)

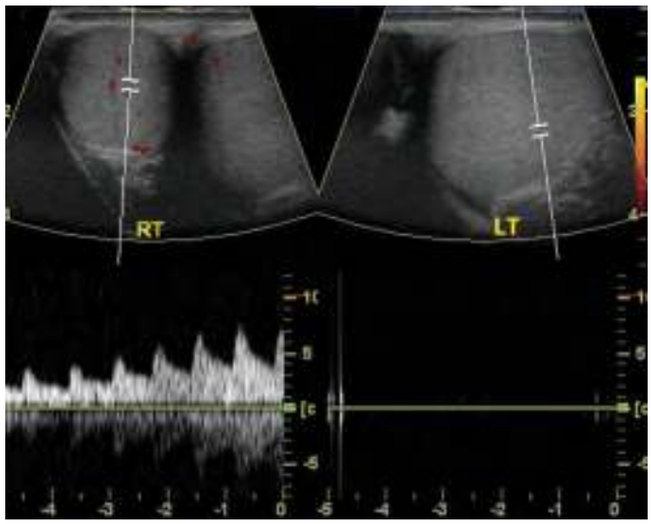

FIGURE 182-4. Color spectral Doppler US image showing the normal arterial flow in the right testicle (left side of picture) and no flow in the left testicle (right side of picture). (Photo used with permission from www.iame.com.)

through 182-4). Compare the normal testes to the torsed testes. Gray scale may show the torsed testicle to be enlarged, edematous, and heterogenous (**Figure 182-2**). Arterial and venous Doppler flow may be decreased or absent (**Figure 182-3**). Look for kinking or coiling of the spermatic cord. Blood flow to the normal testicle should persist throughout diastole. An increased flow during systole decreases gradually in diastole. Increased flow is seen in epididymoorchitis and torsion-detorsion. Hyperdynamic flow is seen after detorsion. Power Doppler waveforms show decreased or no flow in the arteries (**Figure 182-4**). US can identify other etiologies of testicular pain (e.g., hydrocele).

Scoring systems that involve clinical characteristics with or without US findings have been developed to detect testicular torsion and reduce the need for color Doppler US.[20,28,43] These studies were limited by small sample sizes and the use of non-Emergency Medicine providers to score patients. These scoring systems must await prospective, multicenter external validation before they can be recommended for widespread use.

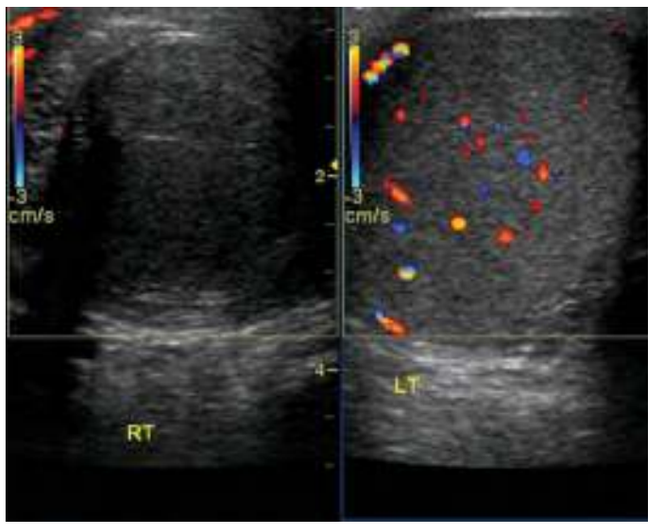

FIGURE 182-3. Color Doppler US image showing no flow in the right testicle (left side of picture) and normal flow in the left testicle (right side of picture). (Photo used with permission from www.iame.com.)

The differential diagnosis of the acute scrotum includes acute idiopathic scrotal edema, appendicitis, cellulitis, epididymitis, epididymoorchitis, Fournier's gangrene, Henoch-Schönlein purpura, hernias, hydroceles, infections, inguinal hernia, mumps, orchitis, testicle appendage torsion, trauma, tumor, varicoceles, and vasculitis (**Table 182-1**).[29,34,41,44] Testicular torsion is the most time-dependent of these conditions. **Consider testicular torsion first in the evaluation of the acute scrotum. Consult a Urologist immediately for a bedside evaluation and manual detorsion while awaiting more definitive intervention.**[45]

ANATOMY AND PATHOPHYSIOLOGY

The testicle is covered by the tunica albuginea. Underneath the tunica albuginea is the visceral layer of the tunica vaginalis. The parietal layer of the tunica vaginalis partially encloses the testicle and the epididymis. The testicular (i.e., spermatic) artery acts as the primary blood supply to the testicle and traverses the spermatic cord. Venous drainage is supplied by the pampiniform plexus within the spermatic cord. The above structures, as well as the vas deferens, are enveloped by the cremaster muscle and fascial layers.

There are two basic types of testicular torsion. They are intravaginal and extravaginal.[46] **An intravaginal torsion is the most common type of testicular torsion.** It occurs when the testicle twists within the tunica vaginalis. The classic anatomic abnormality that predisposes to intravaginal torsion is the "bell clapper" deformity which occurs when the tunica vaginalis inserts high on the spermatic cord (**Figure 182-5**).[41] This allows the testes to assume a horizontal lie and resembles the way a bell clapper sits in the housing of a bell. This abnormality allows the testicle to twist within the tunica vaginalis. **Extravaginal torsion occurs mostly in neonates and the testicle twists outside the tunica vaginalis.** The etiology and anatomic predisposing factors for extravaginal torsion are not currently known.[23,47]

The spermatic cord becomes twisted when the testicle twists. Venous flow is compromised first and results in edema. Arterial occlusion soon follows and testicular ischemia is the result. **The pathologic changes proceed along a spectrum from congestion to hemorrhagic infarction depending on the duration of the**

TABLE 182-1 The Characteristics of Common Presentations of Acute Testicular Pain

Characteristic	Epididymitis	Testicular appendage torsion	Testicular torsion
Dysuria	Possible but unlikely	Possible but unlikely	Possible but unlikely
Cremasteric reflex	Absent or present	Absent or present	Absent or present
Fever	Possible, especially in advanced disease	Possible but unlikely	Possible but unlikely
History of trauma	Possible	Possible	Possible
Swelling and tenderness	Epididymis, progressing to hemiscrotum	Epididymis or head of testicle	Testicle, progressing to hemiscrotum
Nausea and vomiting	Possible but unlikely	Possible but unlikely	Possible
Pain onset	Gradual	Gradual or sudden	Sudden
Patient age	Primarily adolescents and adults Can occur at any age	Prepuberty typically	Bimodal in neonates and adolescents Can occur at any age
Prior pain episodes	None	Sometimes	Sometimes with spontaneous detorsion
Pyuria	Possible but unlikely	Possible but unlikely	Possible but unlikely
Risk factor(s)	Genitourinary abnormalities Genitourinary instrumentation Promiscuity Sexual activity	Testicular appendages	Orchiopexy failure Rapid increase in testicle size in adolescent Undescended testicle
Testicle position	Normal Aligned vertically	Normal Aligned vertically	High-riding Aligned transversely

ischemia.[23] The time to permanent damage is not precisely known. Most authors believe that the testicle should be detorsed within 6 hours of onset for the best results.[11,48] The literature contains reports of good outcomes in testes that have been torsed for up to 48 hours.[10,11,34] Incomplete torsions exist and some blood flow to the testicle may be preserved. **Do not assume a testicle is not viable based on the timing of pain onset alone.**

It has been thought that there are negative effects of testicular torsion on spermatogenesis in the contralateral testicle. The published data are conflicted.[49-52] Studies demonstrating negative effects have relied on flawed surrogate markers of testicular function. More recent literature has demonstrated no residual effect on contralateral spermatogenesis.[52] It is not clear at present whether testicular torsion causes long-term functional damage to the contralateral testicle.

INDICATIONS

Preoperative manual detorsion is associated with improved testicular salvage.[48,53] **Attempt manual reduction of a suspected torsed testicle while awaiting the arrival of the Urologist**. This procedure was first described by Nash in 1893 and involves rotating the testicle in attempt to untwist the spermatic cord and restore blood flow.[54] There is minimal downside to attempting this procedure if it does not delay definitive surgical management.[45] Manual detorsion may be difficult due to edema.

CONTRAINDICATIONS

There are no absolute contraindications to attempting manual detorsion. The detorsion attempt may not be successful for various reasons (e.g., poor patient tolerance of the procedure or an already necrotic testicle fixed to the scrotal wall).[48] **An attempt at manual detorsion must not delay surgery**. Some patients may have contraindications to parenteral pain relief and sedation that may be required for the procedure.[55] **The immediate availability of a Urologist and operative management precludes attempts at manual detorsion**.

EQUIPMENT

No special equipment is required to manually attempt detorsion of a testicle. It is likely that parenteral analgesia and possibly procedural sedation may be necessary (Chapter 159). The use of color Doppler

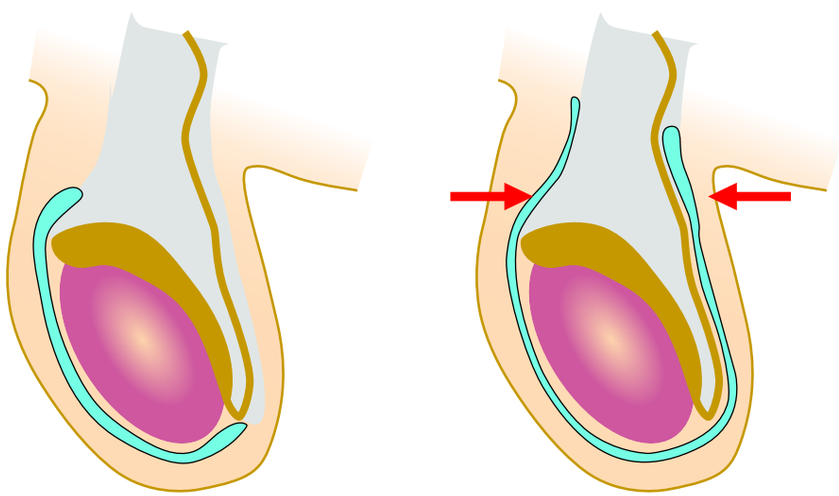

FIGURE 182-5. The normal testicle (left) and tunica vaginalis (purple) versus the bell clapper deformity (right). (Photo used with permission from A.K. Saxena, MD.)

US to assist with the determination of a successful detorsion is optimal but not required. The absence of an US machine should not preclude detorsion attempts if otherwise indicated.

PATIENT PREPARATION

Assess the patient's (or their representative's) capacity to consent for the manual detorsion. Carefully explain the procedure, potential benefits (i.e., restoration of blood flow with possible improved outcomes and relief of pain), risks (i.e., transient pain during the procedure or inability to resolve the torsion), and alternatives (i.e., wait for surgical detorsion). **Make the patient aware that a successful procedure does not guarantee a good outcome.** Explain that sedation is an option. It would be ideal for the patient to be able to communicate to ascertain the efficacy of the detorsion attempts. Ensure the patient and/or their representative understands that they will require a subsequent operation regardless of the outcome of the manual detorsion attempt to lessen the chances of a future torsion or to attempt to salvage the testicle if manual detorsion fails.[15] Inform the patient they will be kept nil per os (NPO; nothing by mouth). Document the informed consent in the medical record.

Anesthesia is not absolutely contraindicated. Insert an IV for vascular access. Place the patient on the cardiac monitor, noninvasive blood pressure cuff, pulse oximetry, and supplemental oxygen to monitor them during and after the procedure if sedation is to be administered. It is preferred not to perform a spermatic cord block or overly sedate the patient because the patient's pain response is used to help indicate if the testicle is being turned in the correct direction and when blood flow has been improved.[56] The injection of local anesthetic can further compromise testicular blood flow.[57] Some patients are in so much pain that they may require a block and/or procedural sedation to even make the attempt at manual reduction. **Pay careful attention in these cases to the restoration of normal anatomy to determine that detorsion has occurred.**[58] A color Doppler US is useful to confirm the clinical impression.

Place the patient supine with the bed set at a level comfortable for the Emergency Physician. The testicle can be manually reduced if standing is more comfortable for the patient. **The standing position is a contraindication to analgesia, parenteral sedation, or procedural sedation as the patient may fall.**

TECHNIQUE

The direction of most torsions has been described as inward or medial. The initial detorsion attempt should be in an outward or lateral direction (Figure 182-6). This motion is like "opening a book" with thumbs or rotation of the hand from pronation to supination.[56,59] The patient's left testicle is turned clockwise while the right testicle is turned counterclockwise (**Figure 182-6**).[56]

There is no evidence-based preferred technique or grip in rotating the testicle. It is simply a matter of placing the patient in a comfortable position and achieving a secure but gentle grip on the affected testicle. Stabilize the spermatic cord with the other hand to prevent it from moving. **Rotate the torsed testicle within the scrotum 90° at a time outward (Figure 182-6). There should be no resistance**. Reevaluate the patient's pain, testicle position, and arterial flow using US.

Resistance to manual detorsion or worsening of the pain indicates that the testicle should be turned in the opposite direction of the initial attempt.[48] Approximately 33% of testicular torsions are lateral instead of medial.[15,60] Some authors have maintained the need to rotate the torsed testicle in two planes (caudal to cranial and medial to lateral).[58] Others have reported success with single plane rotation.[46]

The degree of torsion ranges from 180° to 1080°, with a median torsion of 360° to 540°.[15] **Continue the manual detorsion until the pain is relieved and the normal anatomy is restored if no resistance or increased pain is encountered.**[46]

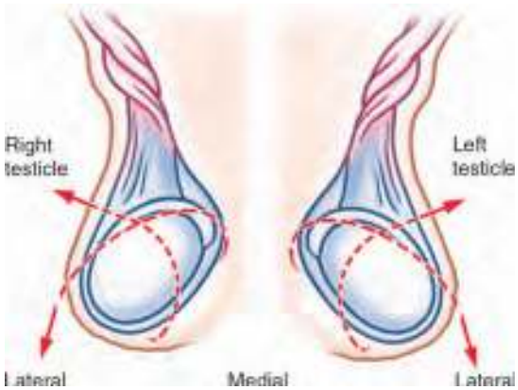

FIGURE 182-6. Manual detorsion of a testicle. The arrows represent the directions to initially attempt to rotate the testicle.

ASSESSMENT

Successful detorsion is indicated by immediate relief of pain and the restoration of the normal anatomy. Relief of pain alone is not adequate to ensure complete detorsion. Normal anatomy is indicated by a lengthening of the spermatic cord, a softening of the involved testicle, and a restoration of a normal vertical lie of the testicle.[58] Use color Doppler US if available to verify the restoration of blood flow after the detorsion attempt. Reperfusion may alter vascular flow patterns which may obscure the color Doppler US results.[23] Continue manual detorsion until the pain is relieved, the normal anatomy is restored, and flow is seen on the US.

AFTERCARE

Every testicular torsion patient should receive an emergent Urologist consult and be scheduled for surgery regardless of the results of the manual detorsion.[45] Surgery is required even with pain relief because the testicular torsion may not have been completely reduced. Bilateral orchiopexy is indicated to try to prevent future episodes since approximately 80% of patients with the "bell clapper" deformity have the deformity in the contralateral testicle.[23] The exact timing of the surgery will be influenced by the outcome of detorsion attempts and the Urologist.[61,62]

COMPLICATIONS

There are a few minor complications to manual detorsion but none that should deter the Emergency Physician from an attempt at detorsion should it be indicated. The procedure is painful. Detorsion in the wrong direction will initially increase the patient's discomfort. The injection of local anesthetic solution into the spermatic cord may further compromise testicular blood flow if a spermatic cord block is performed. There is the possibility that, despite proper technique, the Emergency Physician will be unable to detorse the testicle. The patient will have endured an uncomfortable procedure without gain if testicular torsion is not the correct diagnosis. Detorsion in the wrong direction may temporarily increase the degree of vascular compromise. Detorsion should cause no increase in ischemia if detorsed in the opposite direction. Complications may arise from a delay in notification of the Urologist.[56]

A more serious potential complication stems from the false sense of security obtained when the patient has pain relief. Manual detorsion may only partially restore blood flow and rapid surgical intervention may still be required. Delaying the trip to the Operating Room due to lack of pain may cause "castration by procrastination."[56]

Animal studies suggest that detorsion of an ischemic testicle can cause neurohormonal-mediated reperfusion damage.[63-65] A variety of substances are being investigated to see if this damage can be prevented.[65-70] These findings await confirmation in humans. Experimental data should not be used to justify inaction by the Emergency Physician.

SUMMARY

Emergency Physicians should suspect, recognize, and aggressively treat testicular torsion. Swift action is necessary to preserve function since "time is testicle." Consult a Urologist immediately when testicular torsion is strongly suspected. Attempt manual detorsion on all patients while awaiting definitive surgical management. The patient needs surgery to confirm the restoration of full blood flow even if manual detorsion is successful and for bilateral orchiopexy to reduce the chances of torsion in the future.

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183 Zipper Injury Management

Anthony W. Catalano and Ricky N. Amii

INTRODUCTION

Zipper injuries may involve any area of exposed skin.[1-21] Zipper injuries usually occur to the foreskin, the skin of the penis, and the scrotum. Zipper injuries result in skin and soft tissue entrapment when the zipper is opened or closed. It primarily occurs in uncircumcised young boys, intoxicated adults, the mentally handicapped, males not wearing underwear, and elderly men suffering from movement or cognitive disorders. The most common type of zipper entrapment compresses the skin between the sliding piece (fastener mechanism) and the teeth of the zipper. Another type of entrapment involves the skin between the teeth of the zipper after the sliding piece has moved beyond the area.[1,2] Multiple methods to extract the entrapped skin have been reported.[1-21] These methods range from manipulation to tooth-by-tooth extraction to circumcision. Significant swelling can obscure the zipper mechanism and make standard approaches impossible necessitating ingenuity to avoid secondary injury.[16] Treatment should be guided by the type of entrapment.[1] Removal of the zipper can be performed quickly using basic tools to extract the entrapped tissue and thus prevent or limit secondary injury.

ANATOMY AND PATHOPHYSIOLOGY

The zipper is a simple device that is used daily by millions of people (**Figure 183-1**). Although universally present on clothing and equipment, the simple zipper mechanism may be difficult to conceptualize. It consists of a sliding piece that moves in two directions. The sliding piece is composed of a front and back plate connected by the median bar (**Figures 183-1*B* and 183-1*C***).[5] Each plate has a small raised edge that helps to guide the two rows of teeth together (**Figures 183-1*B* and 183-1*C***). These edges do not connect to each other as the only portion of contact between the two plates is via the median bar. **Without this bar the mechanism falls apart**. The median bar is usually located at the top of the sliding piece. A finger grip is attached to the front plate of the sliding piece and functions as a handle to move the sliding piece. The teeth are two opposing sets of rectangular metal or plastic pieces attached to fabric to keep them aligned (**Figures 183-1*A* and 183-1*D***). Moving the sliding piece across an open zipper will interlock the teeth and close the zipper. Reversing the sliding piece direction will unlock the teeth and open the zipper. The importance of this interdigitating order is evident when this back-and-forth order is violated and the sliding piece cannot pass the defect without great force, if at all.

Although any area of skin can become entrapped in a zipper, it primarily occurs to the foreskin, penis, and scrotum. Entrapment often occurs in those who are in a rush to get dressed, not wearing underwear, intoxicated, or in a rush to zip up their pants. Zipper injuries can be extremely painful. Patients are often unable to undo the entrapment themselves and present to the Emergency Department for help. Because this is an embarrassing injury, patients often present after attempts at self-extraction and a few hours after the injury. Delays in seeking medical care may result in significant edema that can complicate the removal of the entrapped tissue.

INDICATIONS

Skin entrapped between the sliding piece and teeth or between the teeth of the zipper must be extricated. The skin should be released as soon as possible to minimize edema and prevent necrosis.

CONTRAINDICATIONS

There are no absolute contraindications to the removal of a zipper, the slider, or the teeth from an entrapped piece of skin. Consult a Urologist after releasing the entrapment in cases of significant edema, skin necrosis, urethral involvement, or infection.

EQUIPMENT

- 25 or 27 gauge needle
- Syringes (3, 5, and 10 mL)
- Scissors, bandage or Mayo
- Scissors, small or iris
- Topical anesthetic (e.g., EMLA cream or viscous lidocaine)

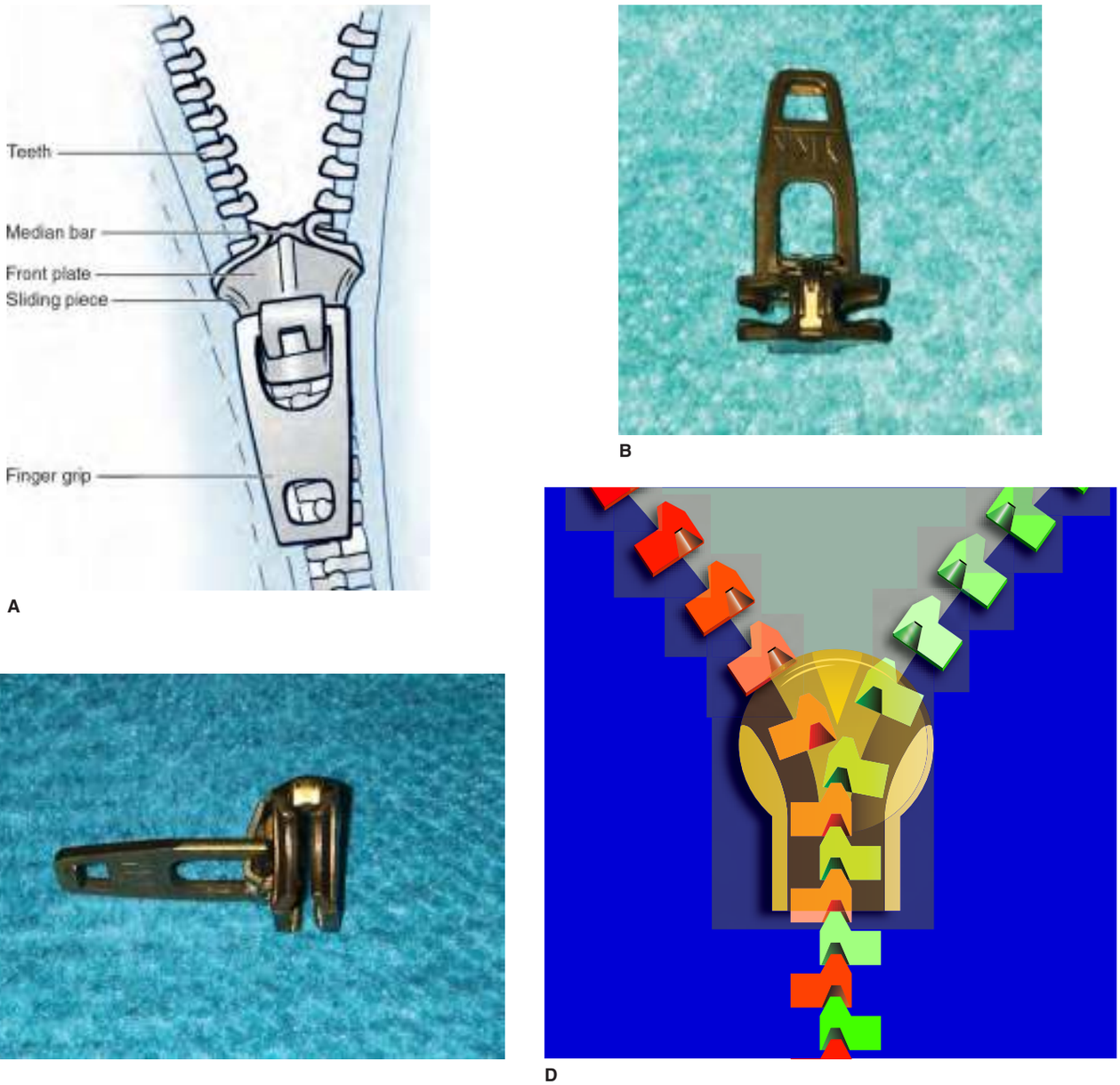


FIGURE 183-1. Anatomy of a zipper. ***A.*** Artist illustration of a zipper. ***B.*** Front view of the sliding mechanism. ***C.*** Side view of the sliding mechanism. ***D.*** Artist illustration of zipper close up. (Used from www.commons.wikimedia.org/wiki/File:Zipper_animated_(reversed).gif by Dominique Toussaint.)

- Local anesthetic solution without epinephrine
- Mineral oil or other lubricant (e.g., surgical lubricant)
- Antibiotic ointment
- Needle driver
- Dremel tool
- Nail lifter or flathead screwdriver
- Pliers
- Heavy-duty wire cutter, ring cutter, or bone cutter
- Miniature hacksaw
- Saline wash

Variations to the equipment list may be required depending on the location of entrapment and extrication approach.

PATIENT PREPARATION

Explain the procedure and aftercare required to the patient and/or their representative. Obtain appropriate consent to perform the procedure. This includes the type of anesthesia used. The discussion should include risk of neurovascular injury, skin necrosis, and infection, and the potential need for skin excision. Assess the patient's level of anxiety and pain as they must remain calm during the procedure to avoid secondary injury. The application of a topical anesthetic such as EMLA cream may be helpful but delays extraction of the entrapped skin. It has the added benefit of acting as a lubricant. If the patient has significant pain, local anesthetic solution can be subcutaneously infiltrated around the entrapped skin or a penile block (Chapter 156) can be performed. A penile block is preferred over local anesthetic injection into the entrapped skin. A local anesthetic injection will cause increased soft tissue edema

and make removal more challenging. The application of parenteral analgesia, sedation, or procedural sedation (Chapter 159) may be required in rare instances.

TECHNIQUES

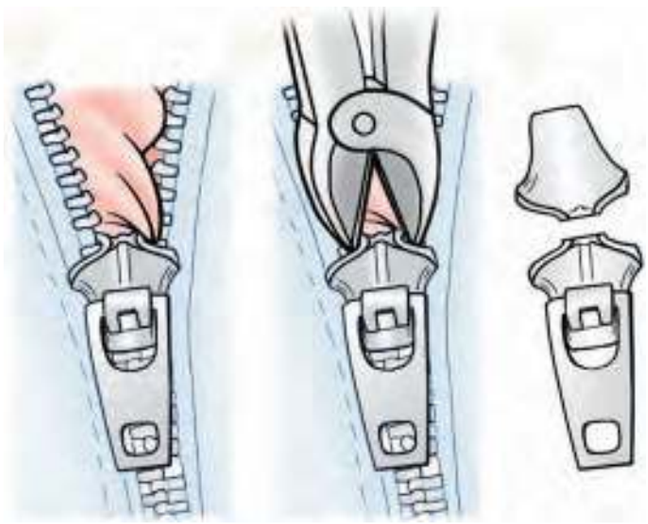
MANUAL REMOVAL

Apply mineral oil, surgical lubricant, or viscous lidocaine liberally to the zipper and entrapped skin. Allow the mineral oil to soak the skin and zipper for approximately 10 minutes.[9,15] Apply gentle but steady traction on the zipper away from the entrapped tissue with special care to avoid further injury.[9,15] Attempt to dislodge the zipper manually. **Do not forcefully try to unzip it. Excessive force is unnecessary and can result in avulsions and lacerations to the skin**. It may be easier to intentionally close the zipper on the entrapped skin completely then attempt to separate the zipper teeth. If this fails to release the tissue, perform one of the zipper disassembly techniques listed below.

CUTTING THE MEDIAN BAR

This is the method of removal recommended by the majority of authors.[6,7] Although simple in concept, it can be difficult in practice to cut the median bar as it is often obscured by the entrapped and edematous skin. The median bar is the only component of the mechanism that connects the upper and lower plates. Cutting the median bar should allow separation of the upper and lower plates and extrication of the entrapped skin. A wire cutter may not be readily available in the Emergency Department. It may be beneficial to obtain a wire cutter from the Maintenance Department.

The median bar can be cut with a heavy-duty wire cutter or bone cutter.[6,7] If the skin is entrapped between the sliding piece and the teeth (**Figure 183-2*A***), carefully cut the median bar (**Figure 183-2*B***). The front and back plates of the zipper will separate and release the entrapped skin (**Figure 183-2*C***). Occasionally, the skin is entrapped between the teeth of the zipper (**Figure 183-3**). Cut the median bar of the sliding piece to remove the zipper. Manually pull the two rows of teeth apart to release the entrapped skin.



A B C

FIGURE 183-2. Cutting the median bar of the zipper. ***A.*** Skin entrapped between the sliding piece and the teeth. ***B.*** The median bar is cut. ***C.*** The front and back plates separate after the median bar is cut and the skin is released.

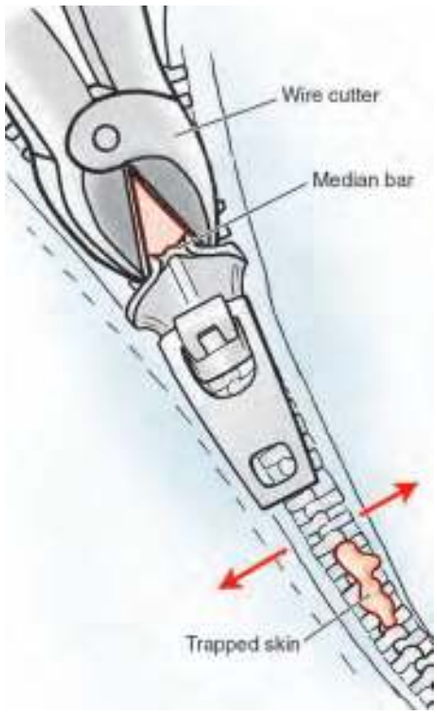


FIGURE 183-3. Skin entrapped in the zipper teeth. Cut the median bar to remove the zipper and manually separate (arrows) the two rows of teeth to release the entrapped skin.

An entrapment involving a heavy-duty zipper may not be amenable to using a wire cutter. Alternatives to the wire cutter exist. An attempt to cut the median bar may be made with a miniature hacksaw or Dremel tool.[10] **However, this carries a significant inherent risk of secondary injury to the skin or deeper structures.**

CUTTING OF THE CLOTH SURROUNDING ZIPPER TEETH

Occasionally, the skin is trapped between the teeth of the zipper and a wire cutter or bone cutter is not available.[8] Cut the cloth holding the zipper to the clothes with a bandage or Mayo scissors (**Figure 183-4**, long dashed lines). Using a small or iris scissors, cut the cloth between the individual teeth that are entrapping the skin (**Figure 183-4**, small dashed lines). Pull both halves of the zipper apart. Separate the teeth and free the entrapped skin. Extrication may be quicker with this method than cutting the median bar, although location is important.[12] Grasping the cloth and zipper with needle drivers may improve the success rate.[18]

This technique is less than ideal. Cutting the cloth between the teeth of the zipper may cause lacerations to the entrapped skin. It is very painful for the patient when the skin is cut. **Exercise great caution when cutting the cloth of the zipper.** Given this risk, some prefer cutting the median bar of the fastener mechanism as described above. It is worth the additional time to locate and borrow a heavy-duty wire cutter from the Maintenance Department to cut the median bar.

CUTTING THE ZIPPER TEETH

Cutting the median bar requires the proper equipment and significant strength. Skin trapped between the teeth of the zipper can be released by cutting across the zipper teeth.[8] Cut across the closed zipper teeth using a heavy-duty scissors or wire cutter. Pull both

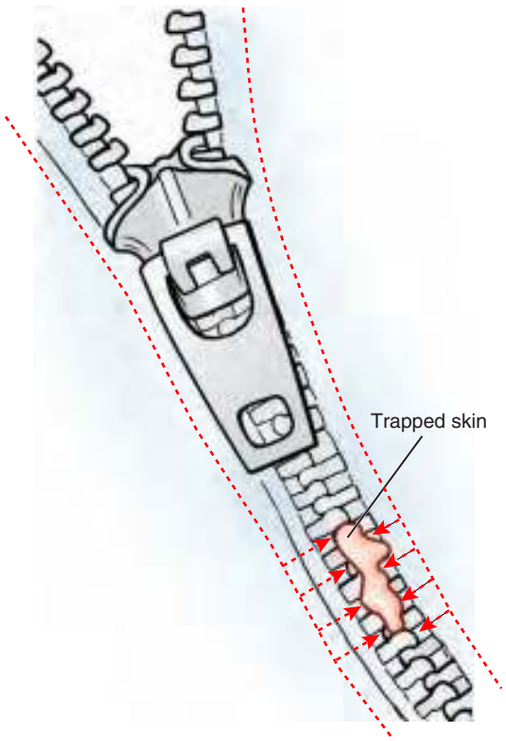


FIGURE 183-4. Skin entrapped in the zipper teeth. Cut the cloth holding the zipper to the clothes (long dashed lines), then cut the cloth between the teeth (short dashed lines).

halves of the zipper apart to release the entrapped skin. Grasping the cloth and zipper with needle drivers may improve the success rate.[18]

This method is faster and easier than cutting the median bar if the skin is trapped only in the zipper teeth.[12] There is the risk of secondary injury to the entrapped skin causing an additional laceration or damage to adjacent structures. It may be preferable to make a second attempt at pulling the two sides of the zipper apart prior to cutting the teeth at the level of the entrapment.

PLIERS

Case reports have discussed successful removal of entrapment with the use of pliers.[19] Cut the cloth attached to the zipper as close as possible to the zipper teeth intersection of the zipper. Grasp the proximal end of the mechanism with the pliers ensuring that both the upper and lower plates are grasped. Apply inward force with the pliers to deform the mechanism and release the entrapment. While successes have been reported, care should be taken to first attempt manual removal or cutting the median bar. Failure to achieve release with this technique may result in a deformed sliding mechanism and difficulty in removing the zipper by subsequent methods.

APPLYING LEVERAGE

If the location of the entrapment is firmly within the zipper mechanism and is not amenable to the above methods, a modified and more forcible attempt may be required. This is not ideal as it leaves the entrapped skin vulnerable to secondary laceration or avulsion but may be required for removal. The success of this method is dependent on the durability of the sliding mechanism as a heavy-duty zipper is unlikely to be easily manipulated.

Two techniques are available. A flathead screwdriver or nail lifter may be used to provide leverage to forcibly separate the upper and lower plates.[13] Insert the flat end in the distal portion of the sliding mechanism and twist it around its axis clockwise and counterclockwise. This technique uses one plate as a fulcrum to apply pressure. Alternatively, a needle driver may be used to grasp the upper plate while applying upward pressure. If space allows, grasp both the upper and lower plates.[20] This may sufficiently bend the median bar to separate the two plates. It is more likely to be used in conjunction with the manual removal technique. With the help of an assistant, one person can provide leverage while the other attempts to unzip the mechanism. **Extreme caution is required, as secondary injury to either provider may occur if leverage is lost or when the mechanism abruptly comes free**.

CUTTING THE FACEPLATE

This technique should only be used as a method of last resort in an attempt to prevent a larger skin excision. Close inspection of the upper plate shows that it is wider at its proximal end and narrows as it tapers to force the two opposing rows of zipper teeth together. If it is impossible to access the median bar and other methods of extraction have failed, an attempt to cut the upper or lower plate at a point where the two rows are not as tightly opposed can be considered with either a ring cutter or Dremel saw.[21]

It is important to note that the circular edge of either device will not cut the flat plate uniformly. Any attempt must be performed slowly and deliberately. The friction caused may heat the metal and puts the underlying skin at risk for secondary thermal injury. Periodic washing with saline to cool the metal zipper may be required. The use of a Dremel tool puts the underlying skin and deeper structures at risk for secondary injury. It is difficult to precisely tell when the circular edge of the Dremel tool passes completely through the plate. To avoid further injury, the entrapped skin should be lifted as far away from deeper structures as possible before cutting.

It may not be necessary to completely cut through the upper plate. Even cutting partially through the plate may decrease its structural integrity and make manipulation possible. Periodically attempt to grasp the upper plate with a needle driver to manipulate the upper plate or use a screwdriver or nail lifter to force the plates apart.

SKIN EXCISION

If all the previously described techniques have failed, a block skin excision may be the only viable removal option. This method should ideally be performed by a Urologist. However, in remote settings or when consultation is otherwise impossible, it may be attempted by the Emergency Physician. The techniques include an elliptical skin excision or circumcision.[6,15]

ASSESSMENT

Assess the tissue for any signs of injury after the zipper has been removed from the skin. Any open wounds should be cleaned. Apply an antibiotic ointment and gauze bandage over the wound. A Urologist should be consulted for injuries to deeper penile or scrotal structures, if the entrapped tissue is not viable, if there is violation of Buck's fascia of the penis or of the dartos fascia of the scrotum, if the tissue appears infected, or if the laceration is extensive.[11]

AFTERCARE

Patients with zipper injuries are usually discharged home from the Emergency Department. Their tetanus immune status should be checked and updated if necessary. The patient should be instructed on local wound care and signs of infection. They should return immediately to the Emergency Department if they develop redness, swelling, pus in the wound, or a fever. Otherwise, routine follow-up

within 48 hours to evaluate any wounds or injured tissue is satisfactory for most patients. Although no guidelines or evidence exists, some physicians often prescribe oral antibiotics if there are any abrasions or lacerations to the skin. Nonsteroidal anti-inflammatory drugs can be used to provide analgesia.

COMPLICATIONS

Very few complications are associated with the removal of a zipper. Lacerations to the skin and deeper structures may occur. Secondary soft tissue injury is the most common complication of zipper removal. This may occur due to zipper manipulation through edematous tissue or cutting the skin (with scissors, wire cutter, ring cutter, hacksaw blade, or Dremel tool). Superficial lacerations are commonly associated with cutting of the median bar. Deep lacerations are due to improper positioning of the wire cutter and can be prevented. Superficial lacerations will heal and require no special care. Large superficial lacerations and deep lacerations will require suturing. Hemorrhage is usually self-limiting and can be controlled with application of pressure. Significant or uncontrolled bleeding may indicate involvement of deeper vasculature and require urologic or surgical consultation. Thermal injury in the setting of the Dremel tool use is unlikely but any burns should be treated appropriately. Any secondary injury to deeper structures likely necessitates consultation with a surgical specialist for evaluation. Violation of the skin should be treated with topical and/or systemic antibiotics. Local infections may occur from zipper abrasions, abrasions and lacerations from removal of the zipper, or nonviable and crushed skin. Evidence of infection should always be treated with systemic antibiotics.

SUMMARY

Zipper injuries may occur when opening or closing a zipper. The foreskin, skin of the penis, and scrotum are the structures most commonly entrapped and injured by zippers. Injuries are most common in uncircumcised young boys but can occur in adults with some cognitive or physical impairment. After assessing the degree of injury and the type of entrapment, the zipper should be disengaged from the entrapped tissue. An Emergency Physician should be capable of multiple extrication approaches depending on the location and involvement within the zipper mechanism. Zipper removal is quick, simple, and very satisfying to the patient. Serious complications are unlikely.

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184 Genitourinary Foreign Body Removal

Dennis Hsieh, Claire Lyons, and Karen Lind

INTRODUCTION

The literature is replete with case reports of genitourinary foreign bodies. A 1932 case report of a pencil inserted into the penis describes genitourinary foreign bodies as "not so infrequent."[1] It is difficult to estimate the incident given the broad variety of anatomic locations and their frequent presence as ancillary complaints to a greater issue (e.g., an assault or psychiatric emergency). A tremendous variety of genitourinary foreign bodies present to the Emergency Department. Genitourinary foreign body insertion is typically iatrogenic, self-inflicted, or traumatic.[2-9] The location of foreign bodies can be categorized by location (e.g., external, subcutaneous, vaginal, and within the urethra or bladder). Foreign bodies that are easily visualized can often be handled in the Emergency Department. Those that are complicated or can only be appreciated by imaging are typically more suitable for removal in the Operating Room.

Polyembolokoilamania is the self-insertion of foreign bodies into various body orifices. It presents in many different populations with a variety of causes that can be grouped broadly.[10] The causes can be further distributed by patient age. These are usually the result of play, trauma, or abuse in children.[11] Iatrogenic foreign body retention has been reported.[11] Vaginal foreign bodies are common in prepubescent girls for a cause of vaginal bleeding.[12] Investigate all presentations of idiopathic atraumatic vaginal bleeding in prepubescent girls.[12] **Be vigilant for any signs or symptoms of sexual assault. Screen the patient and family as child victims of sexual assault may present with a vaginal foreign body.**[13] Most presentations in adults are self-insertions due to sexual pleasure, intoxication, or psychiatric conditions.[14,15] Other causes include penetrating trauma (e.g., bullet in the bladder), erosion or fistulization from adjacent structures (e.g., migrated intrauterine device), accidental (e.g., forgetting to remove a tampon or retained broken condom fragments), sexual assault, or a retained foreign body secondary to smuggling or storing items (e.g., drugs or money) inside the vagina.[16,17] Women who are victims of sexual assault or who are sex workers may experience vaginal penetration with a foreign body already inside the vagina. This can lead to the object becoming lodged more deeply within the vagina and increase the chance of soft tissue injury.

The presentation of a genitourinary foreign body is often obvious and the patient will openly admit to the issue. The patient may be reluctant to discuss this in triage and may only reveal their concern to the Emergency Physician during the private interview or as an aside at the end of the encounter. Genitourinary foreign bodies must be considered when complaints of dysuria, hematuria, pain (i.e., pelvic, genital, urethral, or lower abdominal), discharge (i.e., penile and vaginal), urinary retention, or urinary tract infection are evaluated as these foreign bodies may present in a delayed fashion or the history may be unreliable. Foreign bodies may present as secondary findings in patients with a presentation of multisystem trauma and assault.

ANATOMY AND PATHOPHYSIOLOGY

The external male genital structures include the penis and scrotum, with the penis serving as a genital and urinary organ (**Figure 184-1**). The external penis includes the penile epithelium, foreskin in uncircumcised males, glans penis, and urethral meatus. The male urethra is approximately 20 cm long. The anterior urethra courses through the urogenital diaphragm and the posterior urethra passes through the prostate en route to the bladder. Intraurethral foreign bodies may be found at any location within the urethra. Self-inserted foreign bodies are often distal unless the patient uses an instrument to insert the object more proximally. Intraurethral foreign bodies are much less likely to travel into the bladder in male patients due to the length of the urethra, although this has been reported.[18]

The vulva comprises the external female genitalia and is composed of the mons pubis, labia majora and minora, urethral meatus, vestibule of the vagina, and vestibular glands (**Figure 184-2**). The vagina is a muscular and cylindrical structure and approximately 9 cm long in the adult patient (**Figure 184-3**).

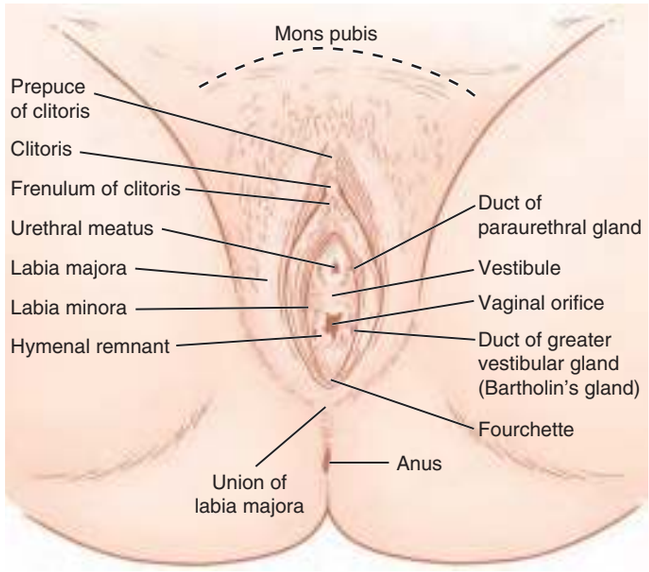

FIGURE 184-2. The anatomy of the female external genitalia.

The vaginal canal is immediately caudad to the urethra and terminates at the cervix. The uterus is approximately at a 90° angle to the vagina.[19] The female urethra is approximately 4 cm long, located inferior to the clitoris and within the labia minora superior to the vaginal introitus. The urethral meatus in women may be less visible externally in prepubertal and perimenopausal female patients.[19] The urethra may be located more posteriorly in patients with prolapse of the bladder or urethra into the vagina.[19] The route for a foreign body to enter the bladder is short and

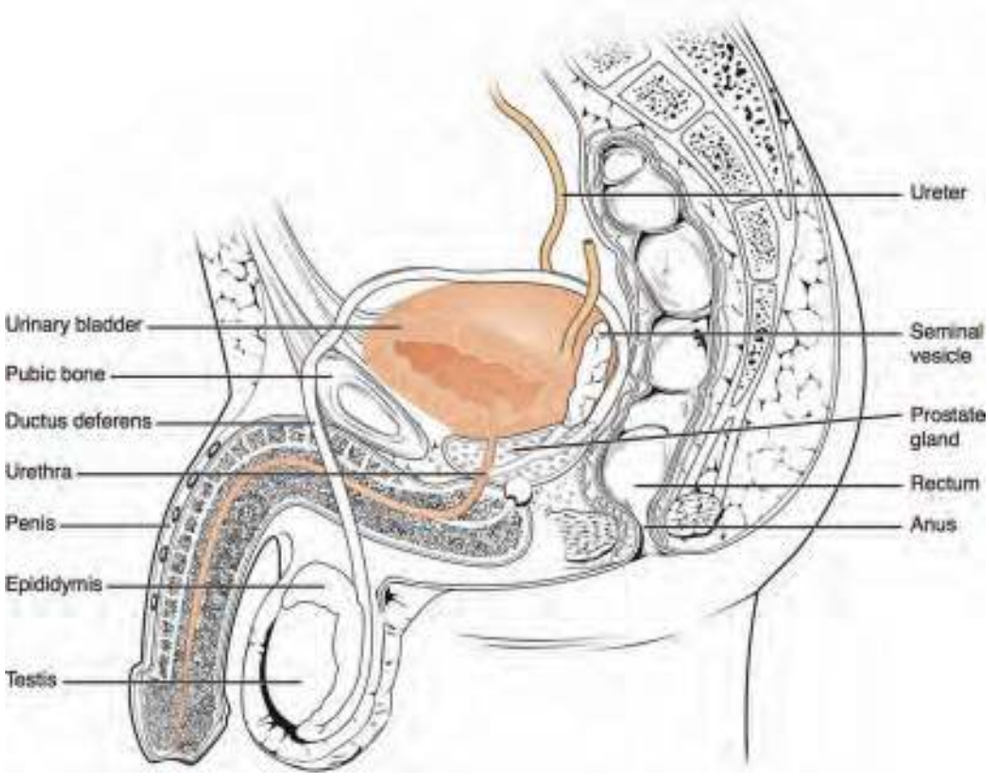

FIGURE 184-1. The male genitourinary tract. (Used with permission from www.openstax.org.)

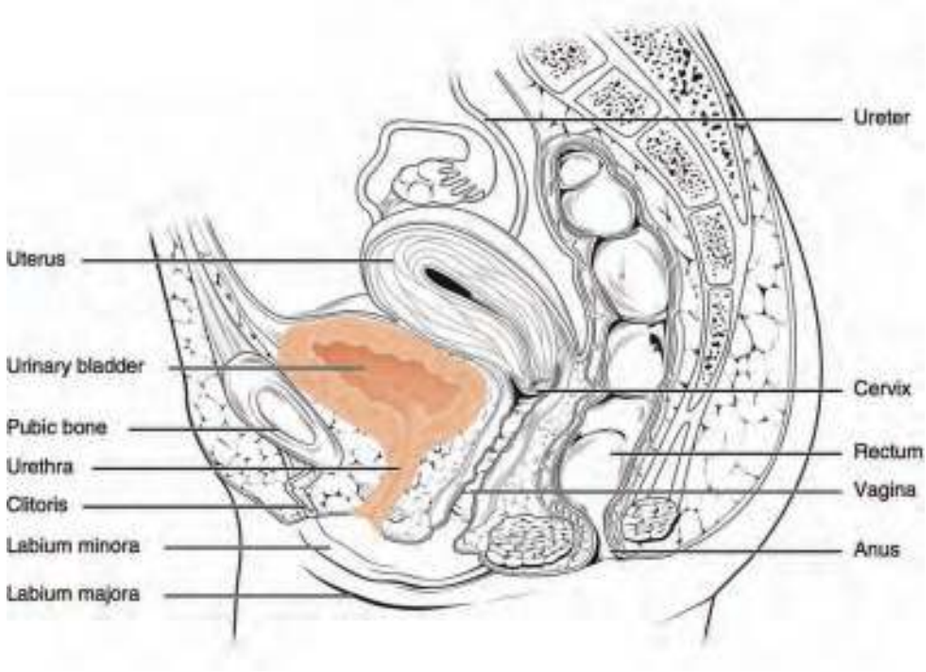

FIGURE 184-3. The female genitourinary tract. (Used with permission from www.openstax.org.)

more direct than in males with less risk of entering an adjacent structure (e.g., the prostate).

Common sites for male genital piercing include the glans penis, penile shaft, scrotum, frenulum, and perineum. Common sites for female genital piercing include the clitoris, labia majora, and labia minora. Subcutaneous genital beads (i.e., pearling) may be implanted by professional piercers or a patient may self-insert an object subcutaneously with or without epithelialization occurring. The Emergency Physician may be tasked with removing these piercings in the case of infection, superficial migration of the piercing (i.e., rejection by the body), or trauma (e.g., tearing or crushing) to the piercing area. These are usually within the purview of the Emergency Physician unless a deep structure is affected or the removal is complicated (e.g., a deformity of the piercing jewelry).

External penile or scrotal foreign bodies may lead to skin erosion, ulceration, and laceration. A very serious complication of external penile foreign bodies is penile incarceration. This may occur with circumferential foreign bodies or those that otherwise entrap distal tissue and place the patient at risk for tissue ischemia with compromised venous return.[20]

Most internal female genital foreign bodies able to be extracted in the Emergency Department reside within the vagina, cervix, and uterus. **Defer intraperitoneal and intrauterine foreign body management to Surgeons.** Intravaginal foreign bodies may cause trauma. Pressure on the vaginal wall from a retained foreign body may result in edema or erosion with ulceration. A sharp foreign body can cause vaginal or cervical perforation. Lacerations of the vaginal wall or cervix may lead to potentially life-threatening hemorrhage.[12] Visible vaginal wall trauma should prompt an investigation for trauma to adjacent structures (e.g., the bladder, uterus, and rectum). The comparative lack of estrogen in pediatric and postmenopausal patients may contribute to increased vaginal wall erosion from retained foreign bodies.

Foreign bodies retained within the urethra have been associated with urethral stenosis and urinary retention.[21] Self-inserted foreign bodies may cause posterior urethral injuries with urethral rupture. This type of injury is more commonly associated with trauma or iatrogenic instrumentation.[22] Retained intraurethral foreign bodies can mimic a urinary tract infection or nephritis. Urinary changes (e.g., hematuria, pyuria, and proteinuria) can be caused by urinary tract irritation secondary to a retained foreign body in the absence of infection.[23,24] Retained foreign bodies much less commonly may present as a nidus for infection in a critically ill patient (e.g., toxic shock syndrome linked to tampon use).[25] Prostatitis has been reported as the presenting symptom of a long-term retained foreign body in the prostatic urethra.[26] Bladder calculi may form as a reaction to a retained foreign body in the bladder.

INDICATIONS

Subcutaneous foreign bodies may be removed in the Emergency Department if deep structures are not involved. A subcutaneous foreign body purposely inserted for cosmetic or sexual pleasure (e.g., piercing or genital bead) should be removed if it is infected, if the area has been traumatized (e.g., tearing or displacement of the jewelry), or if it is beginning to migrate and cause inflammation. Patients may develop allergies or irritant dermatitis to subcutaneous foreign bodies which is an indication for removal. Delay in removal of problematic subcutaneous foreign bodies increases the likelihood of worsening infection, a poorer cosmetic outcome, or a tissue reaction and makes future removal more challenging.

Retained intraurethral foreign bodies with no medical purpose should be removed when possible in the Emergency Department. Consult a Urologist for more complicated cases. The risk of developing complications (e.g., infection, urethral stenosis, hematuria, pain, urinary retention, or other anatomic disruption) is unacceptably high to leave urethral foreign bodies in place without a plan for removal or observation and close follow-up.

Intravaginal foreign bodies should be removed to prevent them from becoming a nidus for infection. They should be removed if possible without risking significant trauma to the vagina that leads to hemorrhage or vaginal wall perforation.

CONTRAINDICATIONS

Some urethral foreign bodies will be beyond the capacity of the Emergency Physician to remove. A urethral foreign body may present in a location that cannot be reached easily. A urethral foreign body with sharp edges or other characteristics that would increase the likelihood of significant damage with removal should not be removed by the Emergency Physician. Those with evidence of significant surrounding tissue injury or reaction should not be removed by the Emergency Physician. Consult a Urologist in these situations.

Obtain a further workup or consultation prior to attempting removal if the patient presents with evidence of a secondary complication from the foreign body. Vaginal foreign bodies may extend into the peritoneum or into a surrounding organ or can lead to perforation from erosion.[27,28] These patients may present with peritoneal signs or prolapse of intraabdominal contents. Consult a Gynecologist immediately and further imaging versus immediate operative exploration will be decided in conjunction with the Gynecologist.

Consult a Urologist if the foreign body appears to have caused urinary obstruction, urethral rupture, or bladder perforation.[2] Obtain advanced imaging unless the consultant wishes to defer imaging and take the patient for surgical exploration. Tissue ischemia (e.g., from penile incarceration) is an indication for emergent consultation with a Urologist.

EQUIPMENT

- Local anesthetic solution
- 25 and 27 gauge needles
- 16 and 18 gauge needles
- 10 mL syringe
- Procedural sedation equipment and supplies (Chapter 159)
- Povidone iodine or chlorhexidine solution
- Personal protective equipment
- Sterile gloves
- #11 blade scalpel
- Hemostat
- Surgical pliers
- Ring forceps
- Tenaculum
- Foley catheter, various sizes
- Anoscope
- Vaginal speculum
- Nasal speculum
- Water-soluble lubricant
- Ring cutter
- Bolt cutter
- Pneumatic saw
- Topical antibiotic ointment
- Bandaging supplies
- Foley catheter and leg bag

PATIENT PREPARATION

The patient must undergo a thorough history and physical examination to determine the overall health of the patient, including a preoperative assessment if appropriate. **Attempt to identify immediately any hemorrhage, vaginal or urethral perforation, and new fistulas as these patients need to go to the Operating Room quickly.** Consider consulting a Psychiatrist for self-inflicted foreign bodies. **A high suspicion must be maintained for abuse. An appropriate consult (e.g., forensics, social work, or police) must be made when this is suspected or confirmed.**

Determine the type and number of foreign bodies through the work-up (e.g., history, physical examination, and directed imaging). **It is possible to remove the most proximal foreign body and miss others.[29] The patient may not be sure of this part of the history and imaging is usually necessary.** An intraurethral foreign body visible through the urethral meatus may have a misleading terminal end and be coiled or kinked in the bladder. This would not be discovered without imaging and attempted removal could be highly traumatic.[30] **Plain films are the initial imaging modality of choice. Computed tomography (CT) scans, cystograms, and urethrograms may be required to further characterize the foreign body and the extent of injury.[31]**

It is important to ascertain the technique of the insertion. **Patients with forcefully inserted objects should undergo a trauma-directed work-up. Screen all patients for sexual abuse or assault.** Inquire about the length of time of the insertion. The more edema and mucosal irritation will develop the longer the object is retained. This will make the extraction more difficult and more likely to require procedural sedation (Chapter 159) or removal in the Operating Room.

The parts of the physical examination that are most useful are the genitourinary, abdominal, and rectal examinations. The goal of the abdominal examination is to determine tenderness or signs of peritonitis. Examination of the external male and female genitalia consists of careful examination to determine if the foreign body is exposed, location, size, texture, mobility, and possible areas to grasp. Determining if the object is hard, soft, rubber, plastic, or glass will help determine the appropriate tool for grasping the object. The other consideration is whether the object is sharp or irregularly shaped. A sharp or irregular object can prevent removal or increase the risk of surrounding tissue damage from the removal process.

It is important to note than many patients will present with concern for a retained foreign body (e.g., a woman who is concerned about a retained tampon) and the foreign body may not be present. The vagina is a blind pouch and many women can adequately examine their own vagina for retained foreign bodies. Many women will already have noted they do not feel a tampon but present out of an abundance of concern. Not all women can examine their own vagina completely secondary to habitus, preexisting disease, or psychological reasons. **It is important to examine all patients for a retained foreign body if they report the possibility of one. Reassure the patient that an evaluation by a medical professional is appropriate if they have a concern for a retained object.** Sometimes broken condom fragments can lead to vaginitis that does not improve until the fragment is removed which can be impossible for the patient to visualize and grasp.

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Inform them that many foreign bodies may be extracted in the Emergency Department. Some will require general anesthesia or surgery. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. Keep all patients nil per os (NPO; nothing by mouth). Perform a standard preassessment for procedural sedation (Chapter 159) if that will be part of the procedure. Obtain an additional signed consent for anesthesia and any photographs taken.

Reassure all children prior to the physical examination that it is normal for a health care provider to examine their genitals.

Inform them to tell their parents or a teacher immediately if anyone else asks to see or touch their genitals.

Place the patient in the lithotomy position, particularly for intravaginal and subcutaneous female genital foreign body removal. Positioning is otherwise dictated by provider necessity and patient comfort. Many external penile foreign bodies are best removed with the patient supine. Patients undergoing procedural sedation should be supine or in the lithotomy position.

ANESTHESIA

Anesthesia may not be necessary for intravaginal foreign bodies that are not causing discomfort and with a plan for manual or forceps removal. Local anesthesia with regional infiltration of the surrounding soft tissue will likely be sufficient for subcutaneous or external foreign bodies in adults (e.g., piercings and genital beads). Avoid the use of epinephrine-containing agents for local anesthesia of the penis, particularly in patients with peripheral vascular disease. Refer to Chapter 177 for a detailed overview of regional anesthesia for the penis, testicles and epididymis. A pudendal nerve block (Chapter 162) may be used to achieve saddle anesthesia in men and women.

Viscous lidocaine may be used to anesthetize the urethra in the absence of a urethral injury. Avoid viscous lidocaine if there is concern for urethral rupture or further entrapping the foreign body within the urethra. Procedural sedation (Chapter 159) may be a better choice for complete relaxation and amnesia to the procedure in young children. Procedural sedation is an appropriate choice for adults requiring a complicated procedure or one with high risk for injury if the patient is not completely relaxed (e.g., pneumatic saw use to cut through external foreign bodies).

Consult a Gynecologist or Urologist for foreign bodies that fail Emergency Department management. These foreign bodies may require removal under general anesthesia.

TECHNIQUES

There are an infinite variety of objects inserted into the genitourinary system. It is impossible to give specific instructions on how to remove each of them. There are basic general principles that can guide the Emergency Physician. The first is to visualize the foreign body regardless of its location. It is best to abort the removal and proceed to more invasive methods if an intraurethral foreign body is externally visualized or protruding but cannot be removed easily with gentle continuous traction. **Further attempts at removal by traction may cause urethral or bladder damage.** Gentle suprapubic pressure may push the foreign body into the distal vagina. Sometimes a low-lying object may be grasped and removed with gloved fingers, an instrument, or a suction catheter using gentle continuous traction. **It is necessary to do this with direct visualization so that injury will not result.** Grasp metal or plastic objects visualized and low lying with a tenaculum or ring forceps**. Avoid inserting instruments other than a speculum unless the foreign body is visualized. Blind insertion of instruments may push the foreign body more proximally or cause injury.** Do not grasp a sharp foreign body or a foreign body against the vaginal wall blindly as this can cause injury to the vagina upon removal of the instrument. Vaginal foreign bodies may be removed by irrigation to minimize trauma.

PIERCINGS AND SUBCUTANEOUS FOREIGN BODIES

Use hydrogen peroxide or saline to remove any crusting at the site of insertion. The piercing jewelry often can be removed by opening the jewelry as normal if the patient presents with an intact piercing that is infected. Local or regional anesthesia is usually sufficient if the patient has significant pain. Most body jewelry will open by having one end unscrewed from the base or by opening the ring to allow the captive bead to fall out. A captive bead is held taut inside a jewelry ring to form a closed circle.

Rotate the jewelry until the opening site reached. Clamp a hemostat onto the end if the jewelry has a post with an end that unscrews. Attach the clamp to the shorter end to prevent it from becoming lost in the piercing site. Manually unscrew the other end. Clamp another hemostat on the other end of the jewelry and slowly rotate one hemostat opposite the other if unable to manually separate the ends. The hemostats may be clamped onto the jewelry at whatever angle the jewelry and patient presentation allow. Slow and gentle rotation with reclamping of the hemostats as the rotation continues will open many jewelry pieces and allow for removal.

The jewelry may be opened with surgical pliers if the jewelry is a ring with a captive bead. Open the pliers inside the ring to widen the ring and allow the captive bead to fall out. Alternatively, use one pliers to clamp the ring and a second pliers to manually force the ring open. Stabilize the hands and forearms with both techniques to prevent pulling on the jewelry. Apply firm and slow pressure to minimize traumatizing the site.

Other options do exist. Piercing sites that have become traumatized through ripping, crushing, or other another deformity may require the piercing site to be incised with a scalpel and the piercing removed directly. Attempt to use a ring cutter (Chapter 126) to cut through the jewelry and remove the separate ends.

Subcutaneous foreign bodies may be removed with similar techniques used on nongenital areas of the body if deeper structures are not involved. A genital bead or other item that was specifically designed to be implanted subcutaneously is less likely to have sharp or irregular edges than an object self-inserted by the patient. Removal of these objects has less chance of traumatizing local structures if they have not become displaced. Prepare the skin with chlorhexidine or povidone iodine and allow it to dry. Don sterile gloves.

Make the incision through the skin that has not completely closed over the jewelry with a #11 scalpel blade. Make the incision deeper and longer until the foreign body can be grasped manually, with forceps, or with a hemostat. Remove it with gentle and firm traction. **Stop the traction efforts if the foreign body becomes resistant to removal.** Proceed with further work-up or consult a specialist if there is concern for retained foreign bodies or debris that cannot be removed.

ANAL, SCROTAL, AND PERINEAL FOREIGN BODIES

The techniques for removing rectal foreign bodies are discussed in Chapter 90. Most scrotal and perineal foreign bodies are managed the same way as other subcutaneous or circumferential penile foreign bodies. They may involve entrapment or incarceration of the scrotum and/or testes. Foreign bodies that have involved the testis are much more likely to require a Urologist and potential operative removal with washout. Rectal foreign bodies can perforate the anterior wall and enter the posterior wall of the bladder.[27,32]

INTRAURETHRAL FOREIGN BODIES

The approach to urethral foreign bodies is determined by the location of the object at the time of presentation and the size of the object (**Figures 184-4, 184-5, and 184-6**). Attempt the extraction of small, mobile, palpable, and externally visualized foreign bodies if the patient has no urinary or abdominal complaints. Determine the

FIGURE 184-4. A radiograph of a fork in the urethra. (Used with permission from Naidu K, et al: An unusual urethral foreign body. *Int J Surg Case Rep* 2013; 4(11):1052-1054.)

extent of the foreign body within the urinary tract before proceeding with removal if it cannot be visualized or palpated, has migrated proximally, or is too friable to be pulled out with external traction. Proceed with imaging.

Visible intraurethral foreign bodies will more likely be amenable to removal from the shorter female urethra. Apply gentle traction manually or with forceps. Items that have migrated to the bladder will require consultation with a Urologist and the use of endoscopy or open cystotomy.[15] Longstanding foreign bodies in the bladder may require lithotripsy prior to removal.[33,34]

Urethral or bladder foreign bodies (**Figures 184-7 and 184-8**) can be removed several ways. The least traumatic technique is endoscopic removal within the genitourinary tract. This approach can be used for a large range of foreign body shapes and sizes. It is usually performed in the Operating Room under general anesthesia. It can occur in rare instances in the Emergency Department by a Urologist while the Emergency Physician administers procedural sedation (Chapter 159). The foreign body can be pushed retrograde into the bladder to facilitate removal with less trauma to the urethral tract.

More invasive methods when endoscopy fails include open techniques and adjuncts (e.g., open or external urethrotomy with reanastomosis over an indwelling catheter or stent placement). A cystotomy

FIGURE 184-5. A radiograph of an ink pen refill in the penile urethra. (Used with permission from www.medapparatus.com.)

A

B

FIGURE 184-6. A visible intraurethral foreign body. ***A.*** Forceps grasping the foreign body at the meatus. ***B.*** Removal of the foreign body. (Photos courtesy of Kevin Binder, PA.)

may be performed for objects that have migrated to the bladder or that were pushed retrograde into the bladder. These patients will be followed by a Urologist as outpatients and may have an increased risk of complications (e.g., urethral stenosis).[33,34]

FIGURE 184-7. A radiograph of a bobby pin in the bladder. (Used with permission from www.medapparatus.com.)

INTRAURETHRAL FORCEPS REMOVAL

Intraurethral foreign bodies may be removed with forceps if the distal end can be seen and grasped (**Figure 184-6*A***). Apply gentle and firm traction to the foreign body until it is removed (**Figure 184-6*B***). **Stop the traction efforts if the foreign body becomes resistant or is too friable to remove intact.** Downward traction on the penis may cause the urethra to kink near the penile suspensory ligament. Hold the penis taut when removing intraurethral foreign bodies.[19]

FIGURE 184-8. A radiograph of a wire in the bladder. (Used with permission from www.medapparatus.com.)

VAGINAL FOREIGN BODIES

Vaginal foreign bodies are a common concern for pediatric patients.[31,35] The patient is often brought in by parents who have noticed a purulent vaginal discharge. A vaginal foreign body in an adult may be the result of sexual practices, psychiatric disorders, or an attempt avoid law enforcement (i.e., concealment of weapons or drugs).[17] The first step is to visualize the foreign body using a vaginal speculum in the adult. Consider using a nasal speculum in the pediatric patient. **Proceed with imaging and consultation prior to traumatizing an intact hymen.** Determine the location and characteristics of the foreign body. Flush out friable or difficult-to-grab small foreign bodies (e.g., wads of toilet paper or pieces of a broken condom). Use warm water or saline in a syringe or feeding tube. Perform the repeat examination once the material has been flushed out to verify that all foreign bodies have been removed and to identify any mucosal injuries.

A common concern is a lost or forgotten tampon. This can be a nidus for infection and the source of a foul-smelling purulent vaginal discharge. Remove retained tampons with a ring forceps or a tenaculum. Pierce the jaws of the instrument through a finger of a latex glove before using it to remove the foreign body. The glove can be pulled over the removed malodorous foreign body to reduce the odor and be discarded. Irrigate the vagina if unable to visualize the walls easily. Empiric antibiotics may be prescribed or the patient can be reevaluated the following day to determine if the symptoms have resolved. Consider coverage for *Staphylococcus aureus*.[36] Antibiotics may not be necessary in the entirely asymptomatic patient who is reliable to return for any worsening conditions.

The female patient should empty her bladder and be placed in stirrups in the lithotomy position to allow visualization. Insert a Foley catheter into the vagina to break the suction between the foreign body and the vaginal mucosa. Grasp the foreign body digitally or with a ring forceps. Remove the foreign body with gentle steady traction. Gentle externally directed pressure (i.e., milking) the walls of the vagina or rectum can facilitate distal movement of the object and its removal prior to the use of forceps or other instrumentation.

Cervical foreign bodies are approached in the same way as vaginal foreign bodies. Removal is most often performed with ring forceps. Proceed with imaging before an attempted removal of the foreign body if there is concern for significant migration into the uterus or uterine perforation. Consult a Gynecologist or Surgeon for any complications.

Most vaginal foreign bodies are usually apparent on speculum examination. Foreign bodies that cannot be visualized in their entirety should be imaged with plain films or CT scans of the pelvis to better characterize the foreign body. Consider procedural sedation (Chapter 159) if the foreign body is easily visualized and is embedded, large, or sharp. General anesthesia may be required to perform a complete examination and facilitate removal under direct vision.

CIRCUMFERENTIAL PENILE FOREIGN BODIES

Penile foreign bodies are common (**Figures 184-9 through 184-13**).[8,37,38] A penile foreign body in an adult may be the iatrogenic, result from sexual practices, or be due to psychiatric disorders. Techniques first tried may be the same as ring removal (Chapter 126).[39] These techniques are often ineffective and more drastic techniques are required.

FIGURE 184-9. A ring causing constriction of venous return. (Photo courtesy of Alex Hoang, MD.)

FIGURE 184-10. An example of an external circumferential foreign body from a wrench. (Photo courtesy of Graham Brant-Zawadski, MD.)

FIGURE 184-11. The bolt cutter technique for removal of a hex nut. (Photo courtesy of Kevin Binder, PA.)

FIGURE 184-12. Pneumatic saw removal of a circumferential foreign body. (Photo courtesy of Kevin Binder, PA.)

RING OR BOLT CUTTER FOR CIRCUMFERENTIAL PENILE FOREIGN BODIES

A ring cutter or bolt cutter can be used to cut through external circumferential foreign bodies (e.g., a metallic ring) for removal (**Figure 184-11**). A penile nerve block (Chapter 177) supplemented with procedural sedation (Chapter 159) may be used to allow for maximum patient relaxation. **Use care not to damage the underlying tissue.** Cut the foreign body with the ring cutter or bolt cutter. Remove the foreign body manually or with two pliers. A second cut can be made to bisect the foreign body so it falls off the penis and is removed easier.

FIGURE 184-13. Saline cooling of a foreign body during pneumatic saw removal. (Photo courtesy of Graham Brant-Zawadski, MD.)

PNEUMATIC SAW FOR CIRCUMFERENTIAL PENILE FOREIGN BODIES

Circumferential foreign bodies that are too thick to be removed with a ring cutter or bolt cutter can be removed with a pneumatic saw (**Figure 184-12**).[37] This equipment is usually not stocked in an Emergency Department. Obtain it from hospital engineering or the fire department. Use procedural sedation (Chapter 159) to obtain maximum patient relaxation and minimize any movement that could lead to inadvertent trauma from the saw. Prepare a bag of normal saline with intravenous tubing attached. Instruct an assistant to control the fluid. The saw will heat the metal. Dripping saline onto the metal will cool it and prevent any smoldering from sparks in the oxygen-rich hospital environment. Have a fire extinguisher in the room for this procedure.

Place protective material (e.g., a section of plastic basin, another thick object, or a reflex hammer blade) above, below, and ideally in between the foreign body and the penis (**Figure 184-13**). This will reduce trauma from saw slippage and sparks. Use the pneumatic saw at a 90° angle to the object and as far away from the patient's skin as possible (**Figure 184-12**). Defer to the Urologist if unable to position the patient adequately to move the skin away and perform the procedure safely. Apply the running saw in 5 to 10 second bursts and check the object in between for temperature. It will likely need to be cooled with saline from the assistant (**Figure 184-13**). Reapply the saw once the object has cooled. Remove the foreign body manually or with two pliers. A second cut can be made to bisect the foreign body so it falls off the penis and is removed easier.

FOLEY CATHETER BALLOONS THAT ARE UNABLE TO BE DEFLATED

Self-retaining Foley balloon catheters are used frequently and typically work well. An indwelling catheter balloon will occasionally not deflate. Most often the problem is a malfunction of the flap-type valve of the catheter's balloon lumen. This valve normally allows fluid to fill the balloon and prevents it from passively leaking out. There are a few ways to deflate the balloon. Target the inflate-deflate channel. Cut off the port and the balloon will often deflate. Insert a needle on a syringe in the inflation channel and suck out the balloon fluid. There is a stepwise series of maneuvers if these are not successful. See Chapter 173 for the complete details of deflating a Foley balloon.

INTRAUTERINE DEVICES (IUDs)

An IUD that causes discomfort or bleeding can be removed in the Emergency Department.[40] Visualize the string. Firmly grasp the string and pull it gently. **Stop the traction efforts if the IUD is resistant to removal.** It may be embedded in the uterine wall. A Gynecologist must remove the IUD if the string is not visible or gentle traction does not remove the IUD.[41] Discuss the infected or migrated IUD with a Gynecologist before removal.[42] Obtain an ultrasound or plain film of the abdomen to locate a migrated IUD. Evidence of potential bowel or uterine perforation (e.g., systemic signs of infection or peritoneal signs) requires a CT scan and emergent consultation for potential surgery.[16]

A foreign body can enter the uterus (**Figure 184-14**). It is usually placed in the vagina as a result of sexual practices or psychiatric disorders or to avoid law enforcement (i.e., concealment of weapons or drugs). The foreign body starts in the vagina and then migrates into the uterine cavity. Iatrogenic foreign bodies may be left in the uterus after a procedure. Consult a Gynecologist for any foreign body in the uterus.

FIGURE 184-14. A radiograph of a bobby pin deep within the uterus. (Used with permission from www.medapparatus.com.)

BULLETS AND PENETRATING TRAUMA

Bladder perforation must be ruled out for penetrating trauma to the bladder from an object (e.g., a bullet). Delayed migration of a bullet into the urinary tract has been reported from tissue surrounding the bladder or from the renal parenchyma. The foreign body may be removed or the patient managed conservatively if bladder perforation is absent. The foreign body may sometimes exit spontaneously with voiding. Symptomatic foreign bodies (i.e., urinary retention, hematuria, infection, pain, or other symptoms) require endoscopic or cystoscopic removal. An open technique may be required if these approaches fail.[43,44]

SUPERGLUE OR SILICONE

Superglue or silicone application to the foreskin and distal urethra may be treated under regional anesthesia (Chapter 177). Options include soaking the penis in warm water, application of mineral oil or acetone to break down the glue, and gentle manipulation to avoid more invasive techniques. Viscous substances (e.g., superglue or silicone) inserted transurethrally can cause urethral obstruction from the substance solidifying. Avoid acetone applied inside the urethra as it may cause mucosal irritation.[45,46] Case reports discuss endoscopic versus external urethrotomy for the removal of the obstructing foreign body.

MAGNETS

One or more magnetic foreign bodies in the urethra or bladder can be removed like any other foreign body.[29,47,48] The exception to this

is when two magnets trap tissue between them (e.g., one magnet is in the urethra and the other is on the external penis or inside the vagina). Attempt to manually remove the magnets. This may require a penile block (Chapter 177) or procedural sedation (Chapter 159). Consult the appropriate Surgeon if the magnets cannot be separated. Leaving the magnets in place can lead to tissue necrosis and fistula formation.[49]

PEDIATRIC CONSIDERATIONS

Pediatric patients have several special considerations.[35,48,50] **Maintain a high degree of suspicion for abuse in any young child with a concern for vaginal discharge or foreign body insertion.** Difficulty in obtaining a history compounds this challenge. The Emergency Physician must often rely on symptoms or physical examination findings to diagnose the presence of a foreign body.[51] **Consult Child Protective Services if sexual abuse is suspected.** Provide careful explanations and anticipatory guidance to the child and parents so they understand what will take place.

Case studies have reported 4% to 5% of prepubertal girls with a genital complaint are found to have a vaginal foreign body.[31] A vaginal foreign body can be the cause of up to 50% of cases of vaginal bleeding and 18% of cases of vaginal discharge in prepubertal girls.[31] Test all prepubertal girls with vaginal foreign bodies for sexually transmitted infections to rule out sexual abuse. These symptoms will often present in a delayed fashion.[52]

Place pediatric patients in the knee-chest position (**Figure 88-5**). Rectal and abdominal examination may identify a foreign body. The foreign body may be expelled from the vagina by pushing with the examining finger in the rectum. Friable foreign bodies (e.g., wads of toilet paper) can be irrigated out using warm water and a standard syringe or infant feeding tube. Removal can also be performed using forceps or a cotton tip swab. A nasal or otoscope speculum can be used for smaller pediatric patients to directly visualize a foreign body and assess for signs of vaginal trauma.[13,51] Consider examination and removal under general anesthesia if there is a concern for a sharp object or if the above techniques fail.

Dysuria, urethral bleeding, and urethral discharge can be signs of male foreign bodies. As with female patients, these symptoms can be delayed for months or years.[53] A complete examination includes palpating the penis for masses and close examination of the urethral meatus. Endoscopic examination and/or surgical removal of foreign bodies may be necessary.

AFTERCARE

Update the patient's tetanus vaccination if there is a skin break secondary to the foreign body or the removal technique. Prescribe oral antistaphylococcal antibiotics if there is an infection or if removal has led to significant trauma to the urethra, deep structures, or adjacent organs (e.g., the cervix).

Inspect the skin of the penis for any erosions or lacerations after removal of circumferential penile foreign bodies (**Figure 184-15**). Any erosions or lacerations require wound care. The penis may remain quite engorged for some time. Carefully evaluate all areas that may be hidden within folds of tissue. Treat erosions and abrasions to the skin with antibacterial ointment. Instruct the patient to monitor the area for the signs associated with the development of an infection. Discharge patients requiring a skin incision to remove a subcutaneous foreign body with wound care to include topical antibiotic cream, bandaging supplies for the first 24 hours, a scheduled wound check, and return precautions. Antibiotics do not need to be prescribed for piercing or subcutaneous foreign body removal if there is no preexisting infection and deep structures are not involved.

FIGURE 184-15. Penile erosions from a foreign body. (Photo courtesy of Kevin Binder, PA.)

The patient should demonstrate the ability to urinate freely prior to discharge following removal of an intraurethral foreign body. Insert a Foley catheter after the removal of the foreign body to prevent urinary retention and stricture development if there is evidence of intraurethral trauma (e.g., blood, significant pain, or removal of tissue with the foreign body). An intravaginal or external foreign body with significant associated tissue swelling is an indication for a Foley catheter to prevent development of urinary retention. Apply a leg bag and teach the patient and/or their representative to empty the bag. Arrange follow-up with a Urologist for further management and Foley catheter removal.

Advise the patient to allow the site to heal without replacing the foreign body and to avoid repiercing the area if a purposely inserted subcutaneous foreign body (e.g., a piercing or genital bead) is removed. This is especially true if keloid development had complicated the initial piercing. Counsel the patient to avoid using that specific material elsewhere on their body if they have a dermatitis or other allergic reaction to the foreign body.

COMPLICATIONS

Trauma to the urethra may have similar consequences to trauma caused by the foreign body itself (e.g., infection, diverticula, false tracts, bladder perforation, fistula, and urethral tears) and lead to urethral stenosis.[28] **Document any associated complication from the foreign body prior to attempting its removal.** Any trauma can result in urinary retention or stenosis and necessitates the insertion of a Foley catheter.

FUTURE CONSIDERATIONS

The Emergency Physician may eventually use limited endoscopy to remove a urethral or bladder foreign body. This is analogous to the use of bronchoscopes. One system is the Isiris (Coloplast Group, Denmark).[54] This is a single-use cystoscope that connects to a portable monitor (**Figure 184-16*A***). The cystoscope has a lens on the tip of the catheter and a built-in grasper. The handle has simple fingertip controls for catheter tip deflection and the grasper. The monitor can record for teaching and the medical record. A wide variety of foreign bodies can be removed (**Figure 184-16*B***).

SUMMARY

Genitourinary foreign bodies are a common reason for presentation to the Emergency Department. Take a thorough history in patients who present with this as a chief complaint and those who present

A

B

FIGURE 184-16. The Isiris endoscopy system. ***A.*** The system. (Photo courtesy of Coloplast Group.) ***B.*** Foreign bodies removed with the Isiris. (Used with permission from reference 54.)

with symptoms that may be secondary to an underlying retained foreign body. Always consider abuse. Suspected foreign bodies or those that are not visible should be investigated with imaging (e.g., plain radiographs, ultrasound, and CT). There are a variety of removal tools and techniques in the Emergency Physician's arsenal. The indications for removal in the Operating Room include proximal location (e.g., uterus, bladder, or proximal urethra), significant associated illness or injury, or high risk for complications. Most patients not requiring surgical treatment may be discharged home from the Emergency Department. Observation is warranted in certain patients for the development of significant complications.

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SECTION 12

Ophthalmologic Procedures

185 Eye Examination

Shari Schabowski and Carmen Alcala

INTRODUCTION

Emergency Physicians often approach the eye examination with apprehension. Eye complaints compose up to 10% of Emergency Department (ED) visits. **A systematic approach to the eye examination can alleviate any discomfort and provide the basis for an accurate diagnosis and treatment.**[1-14] The most common ophthalmologic problems that present to an ED are infection, inflammation, injury, and visual disturbances.

A careful history will help guide the differential diagnosis and the physical examination. It must include a history of the presenting complaint, the mechanism of any injury, exposures to chemicals or infectious agents, baseline visual acuity, known ophthalmologic problems, baseline medical problems, current medications, and any known allergies. **The eye examination progresses from the outside and works inward beginning with the visual acuity to assess the function of the eye. It is important to routinely inspect all anatomic structures of the eye regardless of the presenting eye complaint. Secondary problems (e.g., corneal lesions associated with conjunctivitis) may be missed if a complete and systemic examination is not performed on all patients.**

EYE ANATOMY

The bony orbit is pyramidal in shape, surrounds the eye, and surrounds the associated neurovascular structures. The blood supply to the structures of the orbit originates from the ophthalmic artery. The anatomy of the eye and its surrounding soft tissue structures is demonstrated in **Figure 185-1**. A detailed discussion of the complex anatomy of the eye is beyond the scope of this chapter. The anatomy relevant to the eye examination will be discussed throughout this chapter.

VISUAL ACUITY

Visual acuity is referred to as the vital sign of the eyes. It provides a means for the functional assessment of this delicate sensory apparatus. **Documentation of the visual acuity is essential when approaching a patient with eye complaints.** Assess the patient's visual acuity as soon as possible, preferably as part of the triage assessment, but at least as the primary assessment during physical exam. There are few exceptions to this rule. Chemical exposures to the eye require immediate irrigation to avoid potentially irreversible visual loss. **Do not delay irrigation for visual acuity testing. Failure to document visual acuity is a common omission, may limit the differential diagnosis, and may have medicolegal ramifications.**

Always test each eye individually and then both simultaneously. Test and document the visual acuity with the respective annotation to the right eye (OD), the left eye (OS), and both eyes (OU). Test the problematic eye first. Completely cover the eye not being tested. Light shining into the opposite eye may adversely affect the results of visual acuity testing. A list of commonly used abbreviations in the measurement of visual acuity is provided in **Table 185-1**. Inquire as to the patient's baseline visual acuity and whether they wear corrective lenses for reference. Test the visual acuity using corrective lenses (CC) whenever possible (SC indicates without correction). Consider the use of a pinhole device to help correct any refractory errors when corrective lens are unavailable (**Figure 185-2**).

Use caution when allowing contact lenses to be used as a visual aid in patients with eye pain, an eye injury, or an eye discharge. Their application may worsen the ocular condition. **Remove all contact lenses before the slit lamp examination and fluorescein staining. Fluorescein will permanently stain soft contact lenses and may stain hard contact lenses.**

An accurate assessment of visual acuity is essential and this cannot be overemphasized. It is not uncommon to miss a secondary ophthalmologic diagnosis when there is no recognition of a change in visual acuity. **A patient with previously normal (20/20) vision with or without correction that has an acute deterioration to 20/50 or more suggests a serious ophthalmologic condition.** These cases require emergent consultation and prompt referral to an Ophthalmologist.

VISUAL ACUITY CHARTS

The Snellen eye chart is the most commonly used tool for assessing visual acuity (**Figure 185-3**). Place the Snellen chart on a flat wall in a well-lit room without obstructions. Place the patient standing 20 feet from the chart. Instruct the patient to read each line of the chart beginning at the top and proceeding to the bottom or until they are unable to correctly and consistently read the letters with one eye completely covered. **The visual acuity is the fraction corresponding to the last line for which the patient identifies at least half of the letters correctly (i.e., 20/40).** The numerator corresponds to what the patient can see at 20 feet. The denominator corresponds to the distance where a patient with normal vision would be able to read the same line accurately. For example, a patient with a visual acuity of 20/40 sees at 20 feet what a patient with normal vision would see at 40 feet. Document the fraction corresponding to the line minus the number of letters missed if the patient accurately identifies more than half of the letters on that line (e.g., 20/40 –3).

Move the patient closer and reevaluate the visual acuity when a patient is unable to read the Snellen chart at 20 feet. Test the patient 10 feet from the chart. The test is the same at 10 feet as it is at 20 feet and the documentation changes. Recall that the numerator corresponds to the patient's distance from the chart. Document 10/200 if starting at the 10 foot mark and the patient is able to read the letter at the line designated 20/200. The test can also be performed at 5 feet or closer with the appropriate change in the numerator.

The Snellen chart has historically been the most effective and readily available tool for documenting an accurate and reproducible visual acuity in the ED. The Early Treatment Diabetic Retinopathy Study (ETDRS) chart is more frequently used for optimal accuracy and research purposes (**Figure 185-4**). It is replacing the Snellen

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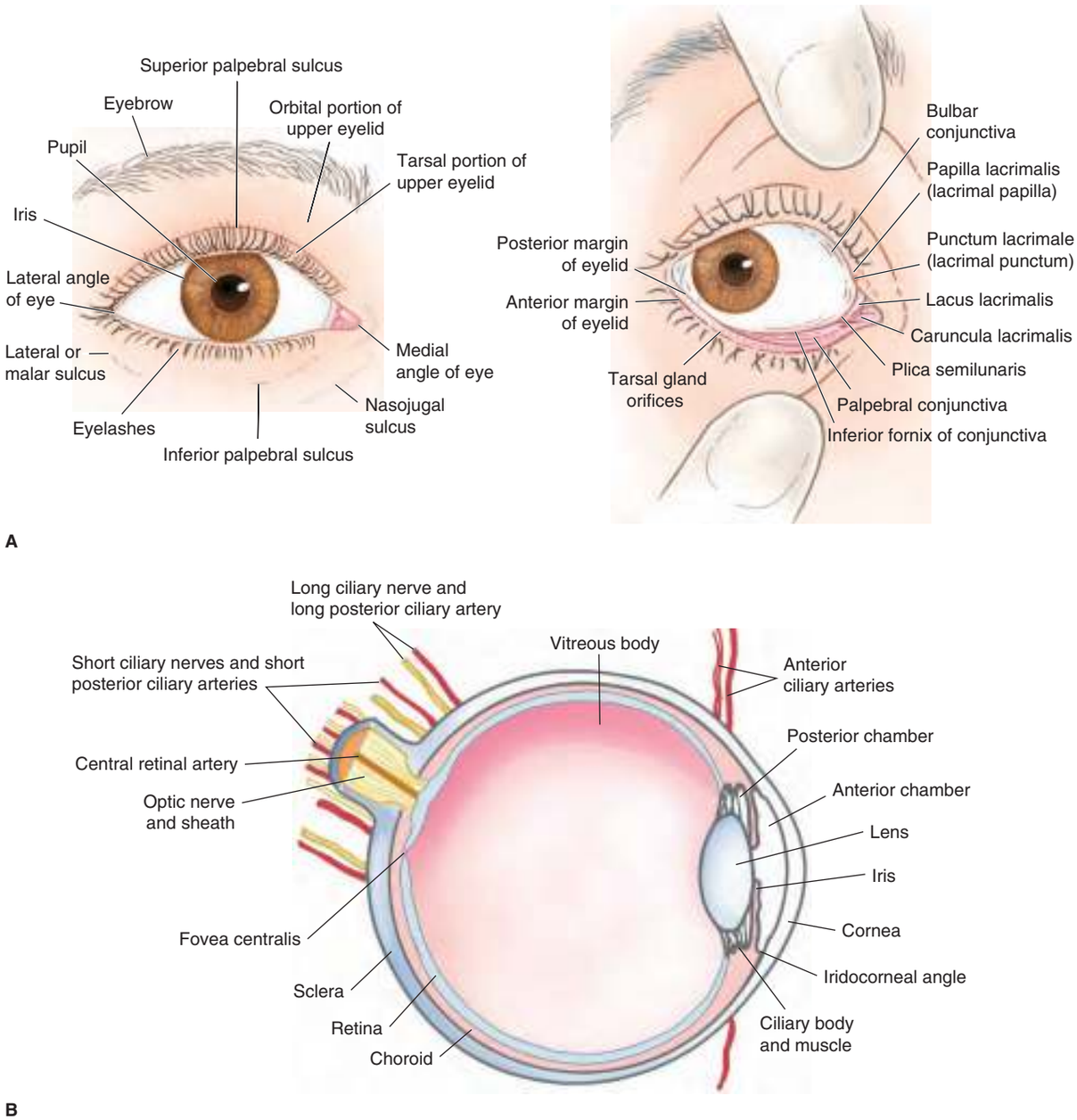


FIGURE 185-1. Anatomy of the eye and its surrounding soft tissues. ***A.*** Surface anatomy. ***B.*** Midsagittal section through the eye.

TABLE 185-1 Commonly Used Abbreviations for the Documentation of Visual Acuity

CC	with correction
CF	counts fingers
DD	disk diameters
HM	hand movement
LP	light perception
NLP	no light perception
OD	right eye
OS	left eye
OU	both eyes
PH	pinhole device
SC	without correction
+	positive
−	negative

charts. There are circumstances when these charts are less effective. These charts use the English alphabet and effective use requires that the patient can identify all letters. This is difficult, if not impossible, in patients who are illiterate or do not speak and/or read English. The illiterate E chart is an alternative (**Figure 185-5**). Instruct the patient to identify the direction that each "E" is facing. Another alternative is to use a pediatric visual acuity chart (**Figure 185-6**). Instruct the patient to identify the objects on each line. These visual acuity charts are assessed and documented like that of the Snellen chart.

There are a few circumstances where the standard eye chart may give falsely low and inaccurate readings. Patients with eye pain or photophobia may have difficulty reading the chart in bright light secondary to excessive blepharospasm, lacrimation, or pain. There are several options to provide a more optimal assessment of visual

A B

FIGURE 185-2. Pinhole devices. ***A.*** Black multi-pinhole occluder. ***B.*** Lorgnette 17 pinhole occluder.

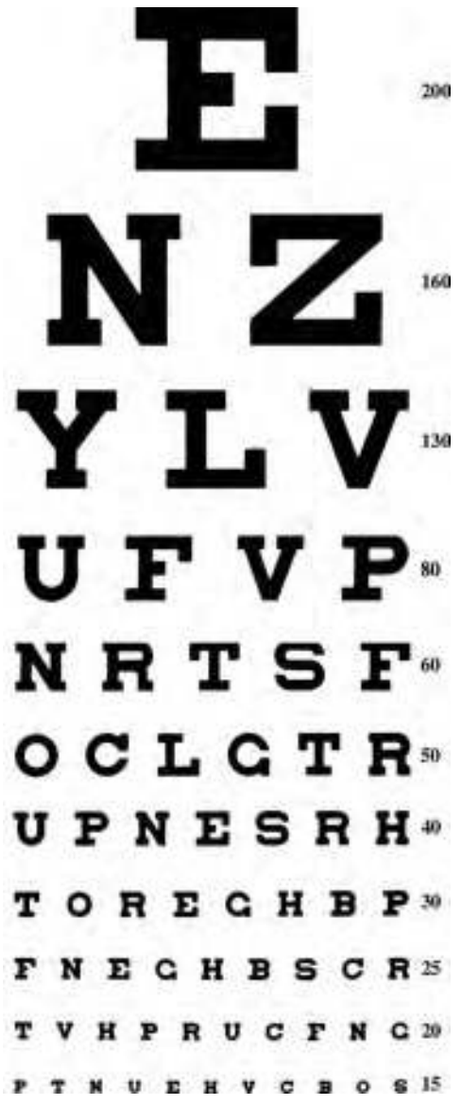


FIGURE 185-3. The Snellen eye chart.

acuity in this situation. A Rosenbaum card is the hand-held equivalent of the Snellen chart (**Figure 185-7**). It is viewed at 14 inches. Its advantages are that it can be used in the patient's room with less offensive lighting and can adjust for refractory errors in near-sighted patients who present without correction. A clever idea that some use is to attach a 14 inch string or suture to the Rosenbaum card so that it can be accurately tested at the exact distance each time. A near vision test for children may be used to assess visual acuity (**Figure 185-8**). Online versions are available for tablets and phones when standard charts are not available.

Photophobia or eye pain (e.g., from corneal injuries or lesions) may prevent the patient from complying with the visual examination. They may often have difficulty opening their eye. The instillation of a topical ophthalmic anesthetic agent may be remarkably helpful as an adjunct to the visual acuity evaluation and the remainder of the eye examination.

FIGURE 185-4. The Early Treatment Diabetic Retinopathy Study (ETDRS) chart.

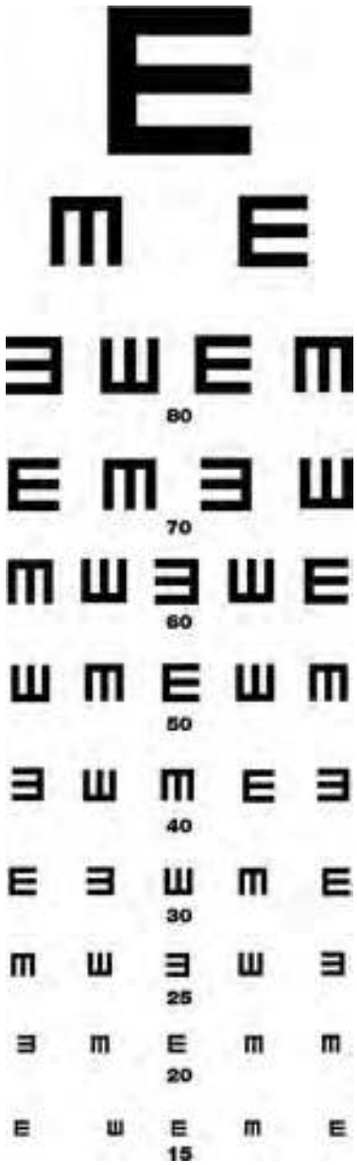


FIGURE 185-5. The illiterate E chart.

PINHOLE DEVICE

The pinhole device can filter out excessive light (**Figure 185-2**). It allows direct light rays to pass through the holes while blocking divergent light rays. This results in a sharper, but dimmer, image on the retina. The pinhole device may correct refractory errors to 20/30. An index card punctured multiple times with an 18 gauge needle can substitute if the pinhole device is not available. Use the pinhole device in a well-lit room because it results in a dimmer image on the retina. Instruct the patient to place the pinhole device over the eye being tested and close the other eye. Make a notation in the record that the pinhole device (PH) was used in the measurement of the visual acuity.

FINGER COUNTING

Another means of visual acuity assessment must be used if the patient is unable to identify the letter or object in the 20/200 position, the first and largest letter in the Snellen visual acuity chart. The

FIGURE 185-6. The pediatric visual acuity chart.

next level of visual acuity that is traditionally accepted is the ability to count fingers (CF). It is important for documentation that the record notes at what distance from the eye the patient consistently counted the examiner's fingers (e.g., OD CF at 12 inches). **The distance used for counting fingers is the distance where the patient is first able to accurately complete the test.** Test one eye at a time and then both eyes simultaneously.

Documentation of "CF at 2 feet" means that the patient can accurately count fingers starting at 2 feet. This implies that the patient cannot accurately count fingers if they were held at a distance greater than 2 feet. It is very important to determine the distance where the patient can accurately count fingers and measure this distance with a tape measure. **Do not estimate the distance.**

HAND MOVEMENTS

The next test of visual acuity to perform is hand movements if the patient cannot count fingers. Move or wave a hand back and forth in front of the eye being examined. Document hand movement positive (HM+) or hand movement negative (HM−). Note the distance from the eye that hand movement is first visible (i.e., how close to the eye must the hand be positioned) in the medical record (e.g., HM+ at 12 inches). Test one eye at a time and then both eyes simultaneously.

LIGHT PERCEPTION

The next determination of visual acuity is light perception if the patient is unable to perceive hand movement. Use a penlight or

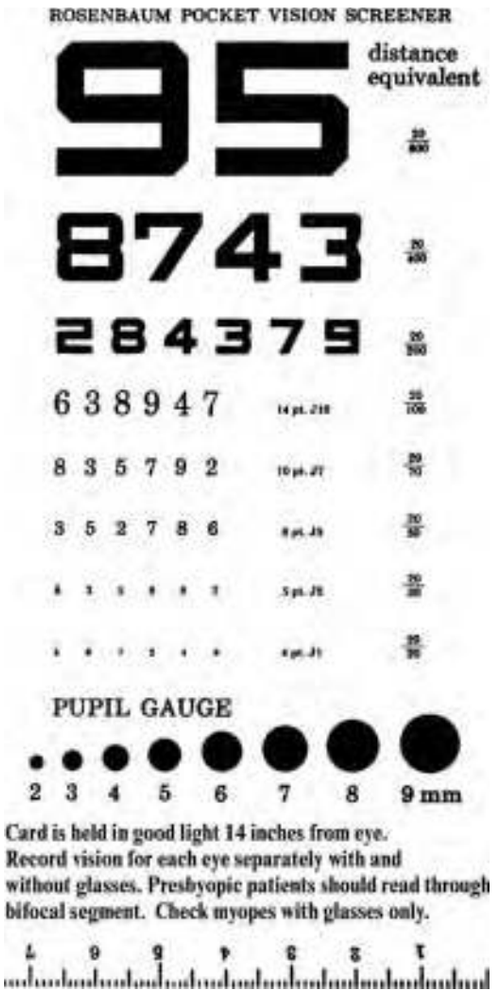


FIGURE 185-7. The Rosenbaum pocket vision screener.

ophthalmoscope to determine the presence or absence of light perception. Shine the light directly into one eye while the opposite eye is covered. Document the patient's ability to correctly identify when the light is on and off. This is noted as light perception positive (LP+) or light perception negative (LP–). Test one eye at a time and then both eyes simultaneously. **True blindness is present when the patient has no light perception.**

UNCOOPERATIVE OR UNRESPONSIVE PATIENTS

Unresponsive patients are a challenge for the evaluation of visual acuity. Shine a light into each of the patient's eyes to test for pupillary reactivity. Move the light slowly through the cardinal positions to identify if the patient tracks the light movement. Refer to the evaluation of extraocular movements section below. Perform the doll's eyes maneuver if the patient is unable to track the light (Chapter 151).

A difficult situation occurs in the patient whose visual acuity is "no light perception or LP–" but has normal pupillary responses and normal responses to the doll's eyes maneuver. This suggests that the visual loss is psychological and not anatomic or physiologic. Test the optokinetic reflex using a spinning device or a scintoscope to narrow the differential diagnosis. A patient who can see movement will not be able to resist the normal tracking response to the rotating cylinder. A positive response will appear as nystagmus upon examination.

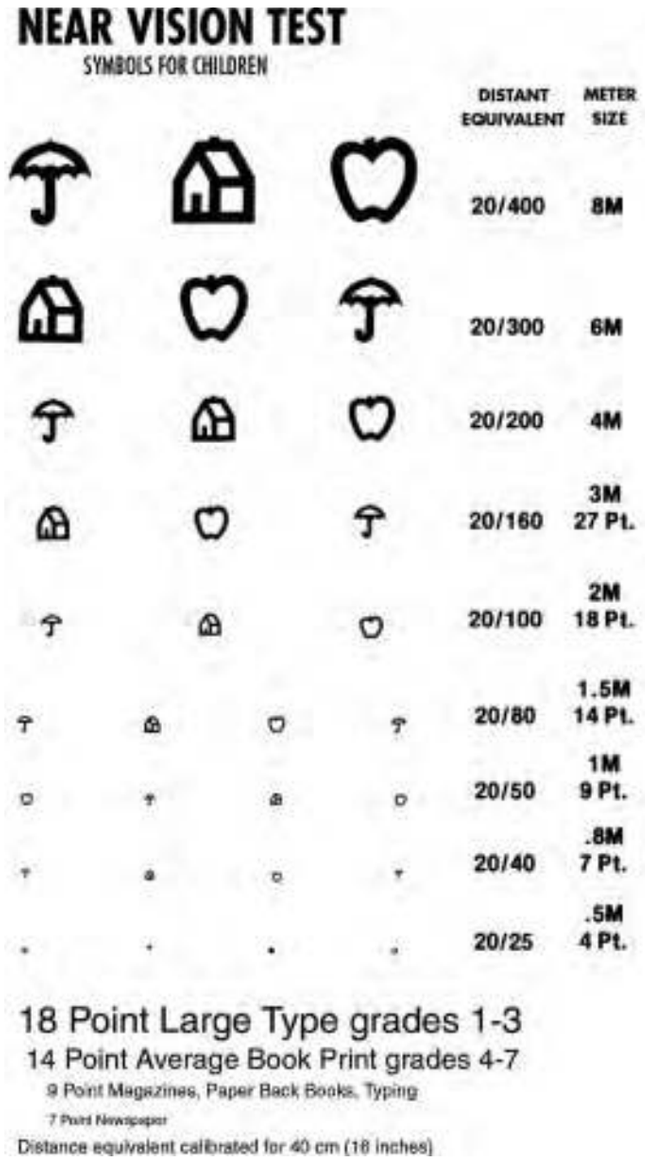


FIGURE 185-8. The near vision visual acuity test uses symbols for children.

PEDIATRIC VISUAL ACUITY TESTING

It is important to evaluate the vision of infants. **There is a critical time during which visual problems must be corrected to avoid permanent visual disturbances.** This is optimally before 4 months of age. The eyes continue to develop quickly up to 2 years of age. Refer any questionable visual disturbances to a Pediatric Ophthalmologist.

Infants from term delivery to 4 months of age are not able to consistently follow and track objects. An infant's eyes may not move in perfect alignment until the age of approximately 4 months. Infants begin to focus on faces and follow them at approximately 6 weeks of age. They should consistently focus on objects and follow objects at 4 months of age.

Testing an infant's vision can be quite difficult. Place the infant with their parent holding them in the feeding position. Cover one of the infant's eyes. Ask the parent to move their head from side to side. Note whether the infant's uncovered eye tracks the parent's face. Repeat this with the opposite eye covered. The inability to track objects suggests that the visual acuity is 20/200 or worse.

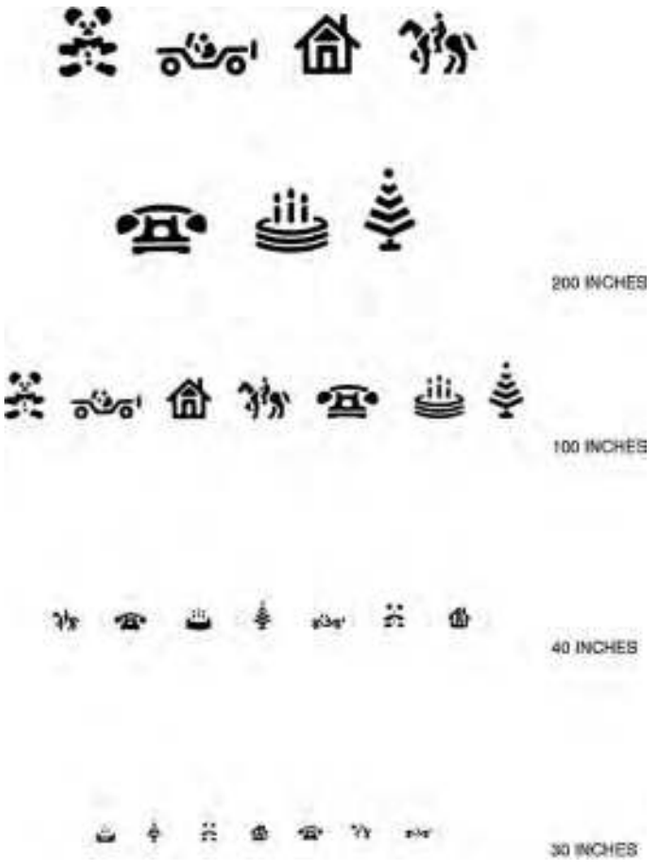


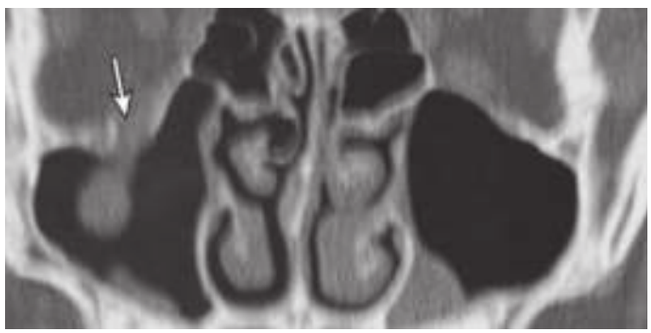
FIGURE 185-9. The Allen chart to measure pediatric visual acuity.

Children who cannot yet read or identify the letters of the alphabet are at a disadvantage when using the Snellen chart (**Figure 185-3**). Use the illiterate E chart (**Figure 185-5**), the pediatric visual acuity chart (**Figure 185-6**), the pediatric near vision test (**Figure 185-8**), or the Allen chart (**Figure 185-9**) to test their visual acuity. The illiterate E chart requires the patient to identify the direction that each variably rotated letter "E" faces (i.e., up, down, right, and left). Some clinicians find it helpful to describe the "E" as a table and ask which way the legs are facing. The pediatric charts use shapes of common objects in the place of letters and apply the same corresponding fractions for determining visual acuity. Have the child identify each of the pictures before beginning the examination to ensure that the child knows what the picture represents. Children may use unexpected words to identify the objects which may complicate the interpretation of the exam.

GENERAL INSPECTION OF THE EYE

The examination of the eye proceeds from the outside and works inward. Begin with inspecting the external structures. Note the presence of any enophthalmos or exophthalmos. This is best accomplished by viewing the eyes from above and behind the patient. The normal globe position is just within the orbital rim. Enophthalmos is a recession of the globe within the bony orbit. It is an important clue for the presence of a blow-out or orbital floor fracture (**Figure 185-10**).[15] Exophthalmos is a protrusion of the globe from the bony orbit. It may be an important clue to the presence of a retrobulbar hemorrhage (Chapter 194) or an orbital cellulitis.

A



B

FIGURE 185-10. Right orbital floor fracture or blow-out fracture. ***A.*** Entrapment of the right eye limits upward gaze. ***B.*** A computed tomography image of the fracture (arrow). (~~Photos~~ used with permission from reference 15.)

Esotropia is a form of strabismus that refers to an inward or nasal deviation of the globe (**Figure 185-11**). Exotropia refers to a form of strabismus that has an outward or temporal deviation of the globe (**Figure 185-11**).

Evaluate the eyes and eyelids for discoloration, lesions, eyelid position, obvious injuries, swelling, and/or symmetry. Ptosis suggests a Horner's syndrome or a third cranial nerve abnormality. **Any evidence of facial trauma or eye trauma raises the possibility of a ruptured globe. Avoid placing pressure directly on the globe if there is any possibility of a ruptured globe.** Inadvertent tactile pressure placed on a ruptured globe may result in extrusion of intraocular structures. This can result in an otherwise avoidable visual loss that is typically not repairable. Manipulate the eyelids by applying pressure over the bony orbit instead of directly on the globe (**Figure 185-12**).

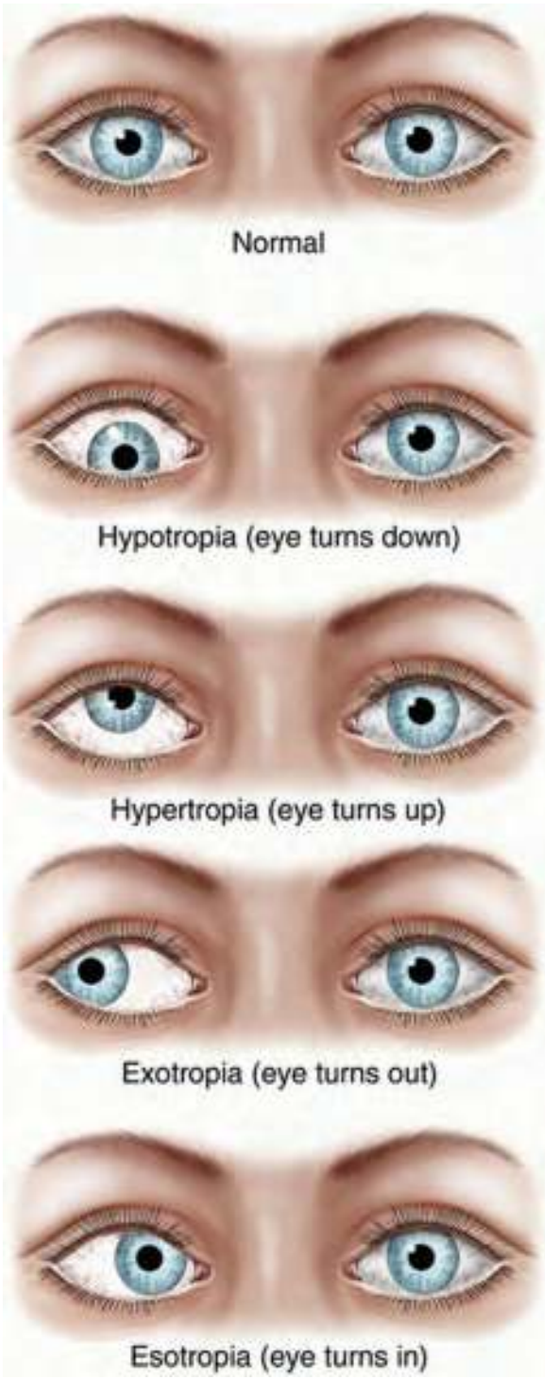


FIGURE 185-11. Types of strabismus.

EVALUATION OF EXTRAOCULAR MOVEMENTS

Extraocular movements are easily assessed in the cooperative patient. Instruct the patient to keep their head still and directed toward the examiner. Instruct the patient to follow the motions of a finger with only their eyes as it follows the pattern of an "H" (**Figure 185-13**). This is referred to as the six cardinal positions for testing extraocular muscle movements. It may be helpful to retract the patient's eyelids to better visualize the eye movements. Observe the eyes for symmetric or conjugate gaze as each position is reached.

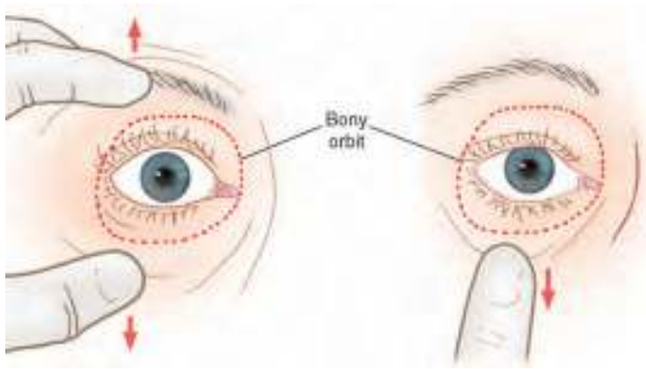


FIGURE 185-12. Open and close the eyelids by applying pressure over the bony orbit in suspected cases of a ruptured globe.

Note whether the patient sees one finger clearly at each position. Ask the patient to describe the orientation of the images (e.g., side to side) if more than one image is noted. Assess the far lateral positions by paying careful attention for evidence of nystagmus. A few beats of nystagmus that quickly extinguish are within normal limits. Keep in mind that the nose may obstruct the view at extreme points. Move the examining hand into a plane that is a few inches closer to the Emergency Physician if this occurs (i.e., a few inches away from the patient) and repeat the testing.

It is helpful to use a penlight directed toward the patient's eyes during the examination when a question of subtle disconjugate gaze is entertained, particularly in the position where the patient complains of diplopia. Look at the patient's pupils to see the reflection of the light (**Figure 185-14**). The reflection of the light is symmetrical and located in the same position in each pupil if the gaze is conjugate (**Figure 185-14*A***). Asymmetrical light reflection is noted in cases of disconjugate gaze (**Figure 185-14*B***).

Consider the innervation of the extraocular muscles to narrow the differential diagnosis when abnormal extraocular movements are identified. The lateral rectus muscle is innervated by the sixth cranial nerve (CN VI). The superior oblique muscle is innervated by the fourth cranial nerve (CN IV). The remaining extraocular muscles are innervated by the third cranial nerve (CN III). **An isolated sixth cranial nerve palsy, particularly in a child, strongly suggests an intracranial neoplasm. A complete third cranial nerve palsy suggests an intracranial aneurysm pressing on the oculomotor nerve, particularly when the pupil is involved.** A physical obstruction originating in the retrobulbar region manifests as restricted extraocular movements in one eye. Consider the presence of an orbital cellulitis or a retrobulbar hemorrhage in the appropriate clinical setting.

Consider the presence of an orbital floor or blow-out fracture if the patient is unable to elevate one of the eyes (**Figure 185-10**). The

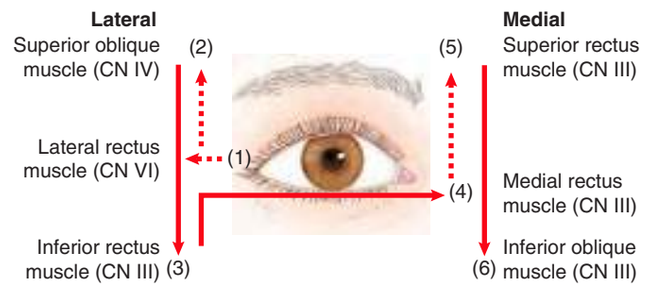


FIGURE 185-13. The six cardinal positions for testing extraocular muscle movements. Start with the eye facing forward and follow the order of the numbers following an H shape.

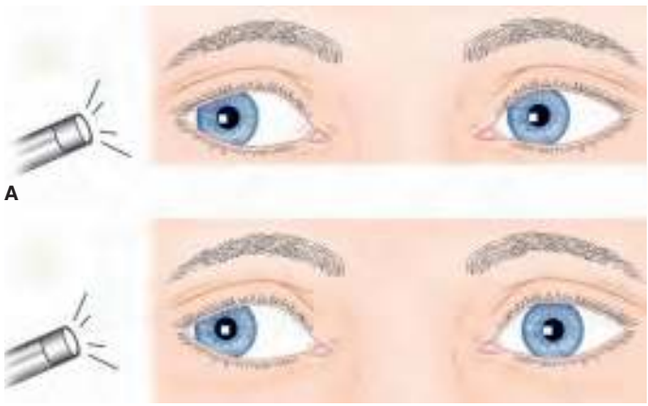


FIGURE 185-14. Shine a penlight into the eyes in the position that the patient complains of diplopia and observe the light reflection in the pupils. ***A.*** Conjugate gaze. ***B.*** Disconjugate gaze.

etiology of the restricted movement may be physical entrapment of the inferior rectus and/or inferior oblique muscles within the orbital floor fracture or a contusion of the nerve innervating the inferior oblique muscle. The two must be distinguished as the management, treatment, and follow-up are different. Place two drops of a topical ophthalmic anesthetic agent into the affected eye. **Gently grasp the bulbar conjunctiva with forceps and attempt to gently elevate the eye.** An entrapment is ruled out if the eye is elevated and a nerve contusion is more likely.

EXAMINATION OF THE PUPILS

Place the patient with their head looking directly forward while retracting the upper eyelid. Complete the pupillary examination and a cursory evaluation of the cornea and anterior chamber simultaneously. Clinicians commonly use the abbreviation "PERRL" as documentation for the pupillary examination. "PERRL" means the pupils are equal, round, and reactive to light. Disorders of accommodation rarely contribute to any ED diagnosis and it is not routinely tested. **Be sure that you assess accommodation if you use the acronym "PERRLA." The abbreviation "PERRL" is typically inadequate when a patient has emergent eye complaints.**

Document a detailed pupillary exam. Darken the room as much as possible. Instruct the patient to focus on a distant object. The normal response is symmetric dilation of the pupils. Note the size, in millimeters, of each pupil. Shine a focused beam of light into one eye to test reactivity or the direct pupillary response to light. The normal pupil will constrict immediately and briskly. Document the direct response to light in millimeters at the maximal constriction (e.g., OD = 4 mm → 2 mm). Note if the pupil is not briskly reactive (e.g., OS = 5 mm → 3 mm, sluggish) or if it dilates (e.g., OS = 3 mm → 6 mm). Repeat this test with the contralateral eye. There are handheld devices that can provide extremely accurate pupillary size and reactivity measurements but this is rarely necessary outside of the Intensive Care Unit.

The consensual pupillary response is the reaction of the contralateral eye to light shined into the opposite eye. The normal consensual pupillary response is constriction of the contralateral pupil when light is shined into the opposite eye. This occurs because some of the efferent optic nerve fibers cross the midbrain to the contralateral optic tract and result in constriction of the contralateral pupil. Be careful to document a normal consensual response if it is present.

The consensual pupillary response can be used as a diagnostic tool when assessing an inflamed eye. Close and cover the affected eye and shine the light in the opposite eye. Pain associated with the consensual pupillary response in the covered affected eye suggests deep inflammation or an uveitis.

A common example of an abnormal pupillary examination illustrates the importance of paying specific attention to the consensual pupillary response. The relative afferent pupillary defect (RAPD) results from the tested eye not perceiving the light. There is no perception of light (i.e., LP–) transmitted to the optic tracts. The tested pupil will not constrict to direct light. The pupil may inappropriately dilate in response to direct light. There will be no consensual response in the contralateral pupil. The contralateral pupil will constrict appropriately to direct light and the previously unresponsive pupil will demonstrate a normal consensual response. The "swinging flashlight test" is used to test for a RAPD. Shine the light into one eye. Note the direct and consensual responses. Swing the light over to test the opposite eye. Note the direct and consensual responses of each eye.

A common problem is the patient with unequal pupils. The primary consideration is typically a "blown pupil" suggesting an uncal brain herniation from a mass lesion. This would be very unusual in a patient who is awake, alert, and cooperating with the eye examination. A physiologic anisocoria is the most common cause. Patients with physiologic anisocoria will have a normal response to light and typically the difference in pupillary size will be no more than 2 to 3 mm. **Determine which of the two pupils is problematic if the response is abnormal.** The normal response to a darkened room is dilation. The pupil that does not dilate is abnormally miotic. The appropriate response to bright light is constriction. The pupil that does not constrict is abnormally mydriatic.

It is important to note and document the shape of each pupil as they should be round and regular. Irregularly shaped pupils may give important clues to undiagnosed injuries. A D-shaped pupil suggests a disruption of the ciliary muscles in the region adjacent to the flat part of the "D." A teardrop-shaped pupil suggests a ruptured globe with the point of the teardrop directed toward the point of penetration. A quivering and dilated pupil that does not react appropriately to light suggests a lens dislocation partially obstructing the visual axis. An irregularly shaped pupil may be an important diagnostic clue but do not be misled. The most common cause of an irregularly shaped pupil is a postoperative change. **Always inquire about previous eye surgery or old injuries, especially if an abnormality is noted.**

Perform a cursory evaluation of the cornea and anterior chamber simultaneously. Look at the shape and note any foreign bodies present. Look for the presence of a hyphema (**Figure 185-15**). This is blood in the anterior chamber. A small amount of blood

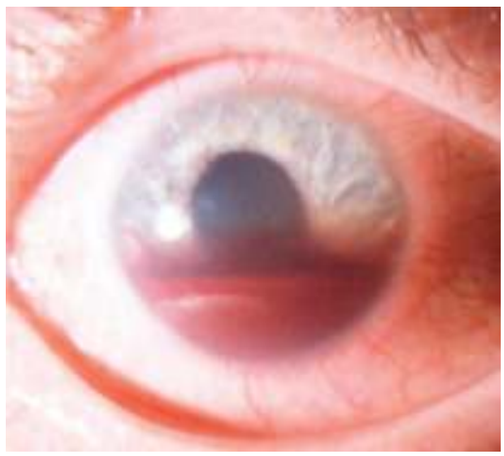


FIGURE 185-15. A hyphema. (Used with permission from reference 15.)

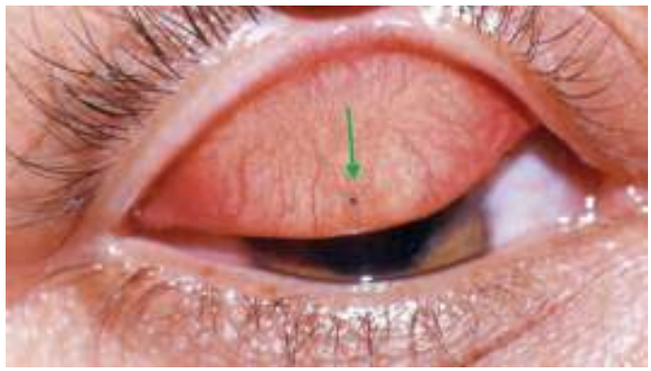


FIGURE 185-16. A foreign body embedded under the upper eyelid. (Used with permission from reference 16.)

may only be seen with the assistance of gravity when the patient is upright.

The NeurOptics NPi-100 pupillometer (NeurOptics, Irvine, CA) has been developed to accurately measure pupil size and reactivity. This hand-held device is easy to use, takes over 30 pictures a second, analyzes the pictures, and prints via infrared to a portable printer. It can display values over time numerically or graphically. It is not for routine daily use in the ED. It has a role in the evaluation of critically ill or injured patients.

EXAMINATION OF THE EXTERNAL STRUCTURES

The examination should proceed from the outside and work inward following an anatomic checklist. Examine the eyelids, lash line, and tarsal plates. Examine them for infection, inflammation, injury, normal position of the eyelashes, and symmetry. Examine the puncta of the lacrimal apparatus for signs of inflammation and obstruction. Examine the bulbar conjunctiva and vasculature for chemosis, ciliary flush, discharge, foreign bodies, and injection. Examine the cornea for clarity, evidence of injury, foreign bodies, or lesions. Examine the palpebral conjunctiva and cul-de-sac for discharge, foreign bodies, injection, and lymphatic (i.e., follicular) enlargement. Examine the anterior chamber for clarity, depth, and particulate matter.

Expose the structures of the inner aspect of the upper eyelid to look for a foreign body (**Figure 185-16**).[16] Evert the eyelids

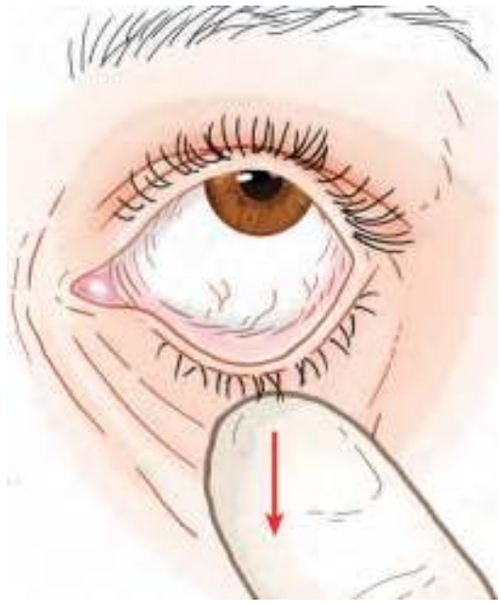


FIGURE 185-18. Eversion of the lower eyelid.

(**Figures 185-17 and 185-18**) or retract the eyelids (**Figures 185-19 and 185-20**). Place the patient facing forward with their eyes focused downward to evert the upper eyelid (**Figure 185-17**). **The patient's eyes must remain directed downward during the entirety of the examination as looking up or forward will cause the eyelids to return to their natural position.** This process requires the use of a cotton-tipped applicator. An assistant may be required to aim and focus a light source during the examination if it requires two hands to evert and hold the eyelids in position.

Grasp the midpoint of the upper eyelash line or the tarsal plate between the index finger and thumb (**Figure 185-17*A***). Place a cotton-tipped applicator 0.5 to 1.0 cm superior to the tarsal plate with the cotton tip in the midplane of the upper eyelid (**Figure 185-17*A***). Apply gentle pressure directed slightly downward against the upper eyelid with the cotton-tipped applicator. Use the other hand to pull the eyelid upward and evert it (**Figure 185-17*B***). The upper eyelid may not completely evert. Sweep the cotton-tipped applicator from left to right while still holding the lash line in one hand and simultaneously applying gentle downward pressure with the

FIGURE 185-17. Eversion of the upper eyelid. ***A.*** Grasp and pull down the upper eyelashes while simultaneously placing a cotton-tipped applicator at the base of the upper eyelid. ***B.*** Flip the upper eyelid over the cotton-tipped applicator. ***C.*** Hold the everted eyelid in place and gently remove the cotton-tipped applicator.

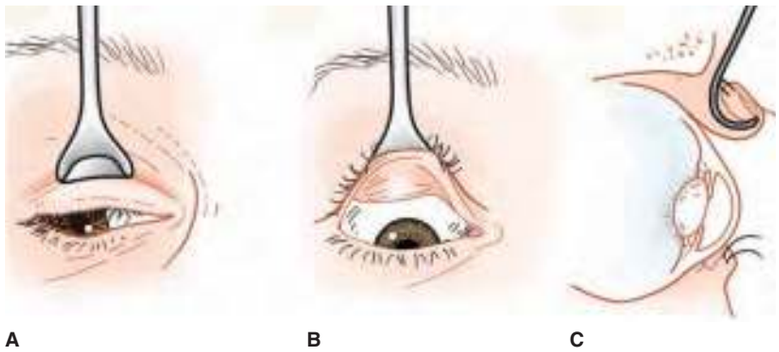


FIGURE 185-19. Eversion of the upper eyelid with a Desmarres retractor. ***A.*** Place the retractor 5 mm above the tarsal plate. ***B.*** Grasp the eyelashes and evert the upper eyelid over the retractor. ***C.*** Elevate the retractor to fully expose the under surface of the eyelid.

applicator within the false pocket to completely evert the upper eyelid (**Figure 185-17*C***). Gently and slowly remove the cotton-tipped applicator.

Eversion of the lower eyelid is much simpler (**Figure 185-18**). Instruct the patient to look upward. Place the index finger on the patient's lower eyelid. Apply downward traction to evert the lower eyelid.

The use of a Desmarres eyelid retractor is an alternative option to retract the eyelids. It is very difficult to manually evert the eyelids when they are swollen. Manual eyelid eversion can result in excessive pressure being placed on the potentially injured globe. An eyelid retractor can be used to avoid placing pressure on the globe (**Figure 185-19**). Place the patient facing forward with their eyes directed downward. Place the eyelid retractor approximately 5 mm above the tarsal plate on the outer surface of the eyelid (**Figure 185-19*A***). Grasp the midpoint of the upper eyelash line or the tarsal plate between the index finger and thumb. Slightly retract the upper eyelid away from the globe. Elevate and retract the tarsal plate upward to evert the eyelid onto the retractor (**Figure 185-19*B***). Lift the retractor upward to fully expose the undersurface of the eyelid (**Figure 185-19*C***).

This technique allows the eyelid to be retracted with one hand while directing the light with the other hand. It prevents any pressure from being placed on the globe if a rupture is suspected. It may be necessary to slide the retractor 0.5 cm to the left and to the right of midline to fully visualize the ocular structures. Use the same technique to retract the lower eyelids. Instruct the patient to direct their gaze and focus upward when retracting the lower eyelid.

The eyelids can be retracted but not everted with the Desmarres retractor (**Figure 185-20**). Place the eyelid retractor in front of the upper eyelid (**Figure 185-20*A***). Gently insert the retractor under the upper eyelid (**Figure 185-20*B***). Apply slight outward traction on the retractor to securely grasp the tarsal plate. Rotate the retractor upward to retract the upper eyelid (**Figure 185-20*B***). Elevate the retractor upward to open the eyelid (**Figure 185-20*C***). Use the same technique to retract the lower eyelid. **Never use this technique if a foreign body or ruptured globe is suspected.** It may cause secondary injury by embedding a foreign body, perforating the globe with the foreign body, or causing extrusion of the ocular contents if the globe is ruptured.

Lid retractors should be a part of an ED eye kit (**Table 185-2**). If a retractor is unavailable but it is necessary to retract a swollen lid, a paper clip can be configured into a retractor (**Figure 185-21**).[17] Take care to inspect the quality of the metal surface of the paper clip. It is possible that the metal could flake off and cause an eye injury. Plastic-coated paper clips might be a safer alternative to metal paper clips.

SLIT LAMP EXAMINATION

The slit lamp is essential for a thorough eye examination (**Figure 185-22**). It is an invaluable resource that can be used to identify and aid in the treatment of ophthalmologic problems that might otherwise go unrecognized. It provides an adjustable light source with variable magnification. The eyes remain in a fixed position while the light and microscope are independently adjusted. The result of these attributes is that it can focus precisely on the

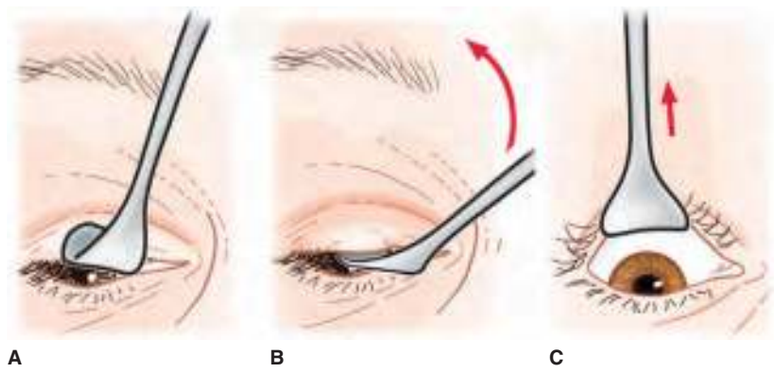


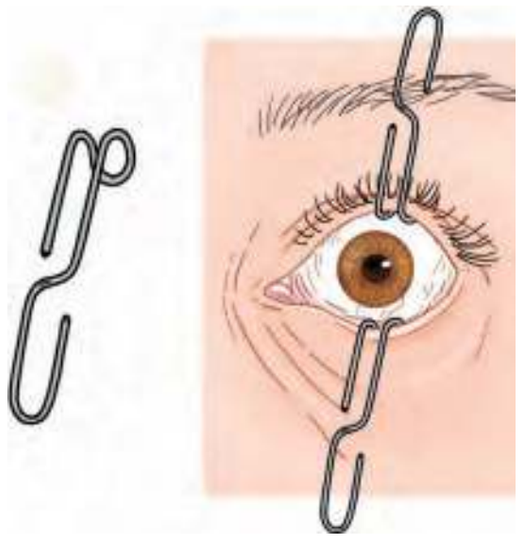
FIGURE 185-20. Retraction of the upper eyelid with a Desmarres retractor. ***A.*** Place the retractor in front of the eyelid. ***B.*** Insert the retractor and rotate it toward the forehead. ***C.*** Elevate the retractor to fully expose the globe.

TABLE 185-2 Recommendations for a Complete ED Eye Examination Kit

Alcohol swabs
"E" chart
Eyelid retractor
Fluorescein strips (individually packaged)
Ophthalmoscope
Pinhole device
Rosenbaum card
Slit lamp
Snellen chart
Topical anesthetic agents (e.g., proparacaine or tetracaine)
Topical cycloplegic agents (e.g., homatropine or cyclopentolate)
Topical mydriatic agents (e.g., phenylephrine)
Topical pupillary constrictors, (e.g., pilocarpine and timolol)

structures of the eye and provide a three-dimensional image. The slit lamp provides valuable information, particularly when examining the cornea and the anterior chamber. The three-dimensional microscopic capability is best demonstrated when examining the intricate topography of the iris.

It may be helpful to set up the slit lamp before the patient is in the room until one feels comfortable using the slit lamp. Place one hand against the middle of the forehead bar in the examination plane to help become familiar with the capabilities of the machine and the joystick. Practice focusing the slit lamp on the details of the skin on your hand. Adjust the focus and depth of vision by sliding the joystick to move the entire slit lamp apparatus forward and backward. Slide the joystick side to side to scan across and examine the entire width of the hand. Rotate the joystick clockwise and counterclockwise to move the line of vision up and down.



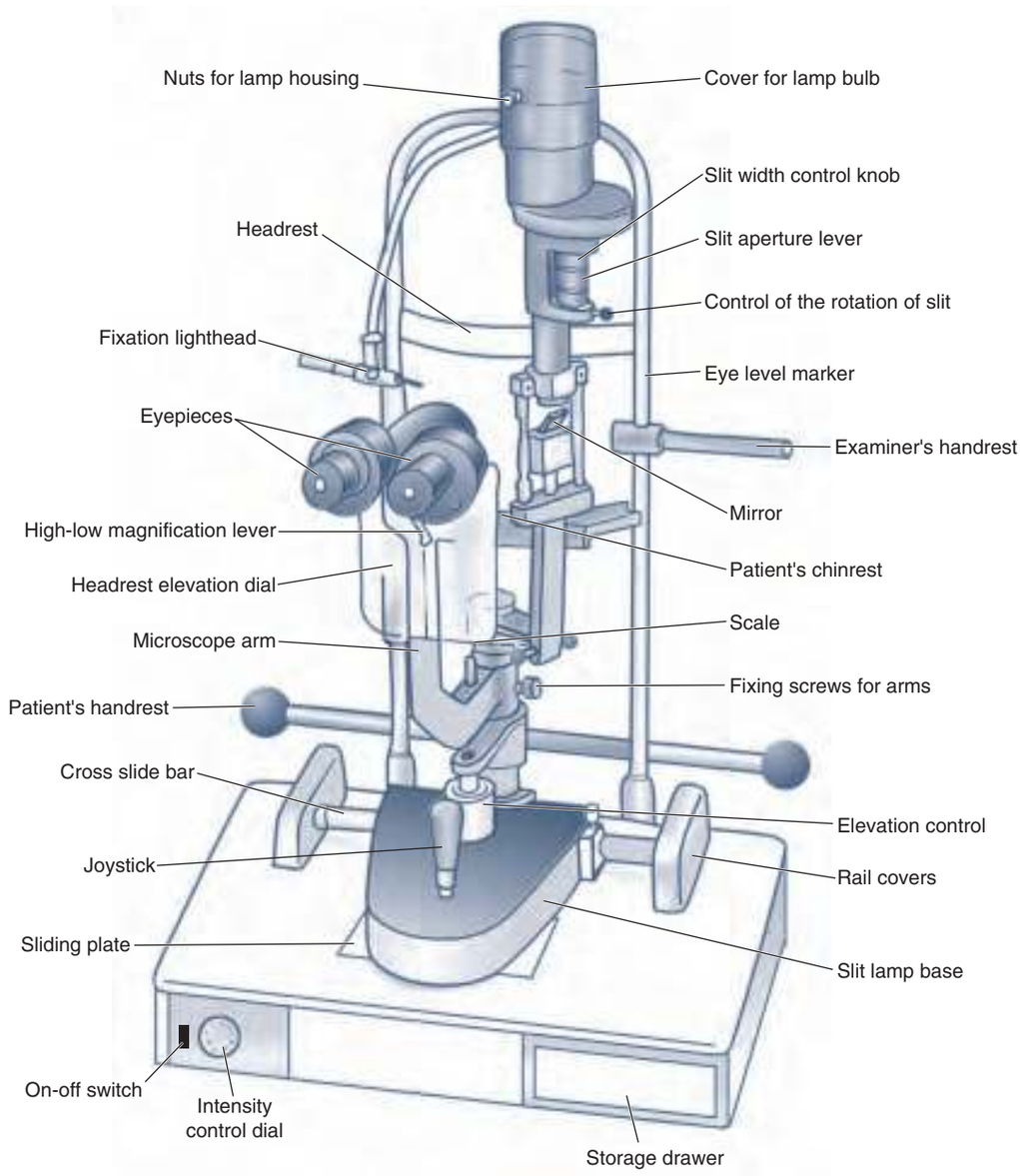
A B

FIGURE 185-21. An alternative to an eyelid retractor. ***A.*** Unfold a paper clip and bend it into shape with a hemostat. ***B.*** Paper clips used to retract the eyelids.

Place the slit lamp in the standard position. It takes very few adjustments of the slit lamp to complete the entire examination. The slit lamp can be focused and locked in position with the knob located on the table base for procedures that require the hands to be

A

FIGURE 185-22. The slit lamp. ***A.*** Photograph of an example. ***B.*** The parts of the slit lamp are labeled.



B

FIGURE 185-22. (*Continued*)

free (e.g., foreign bodies embedded within the cornea). This allows one hand to hold the eyelids open while using the appropriate tool in the other hand.

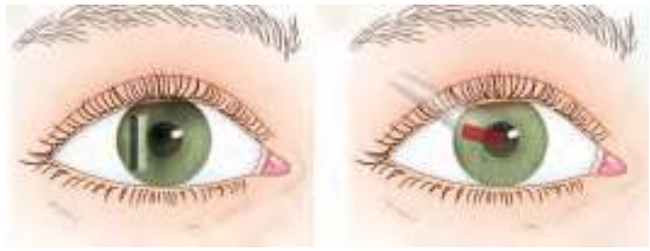
SETTING UP THE SLIT LAMP

The slit lamp has many parts, most of which are capable of movement. The slit lamp components are often moved into a state of complete disarray by previous users. It is important to start by readjusting the slit lamp to a "standard operating position."

The body of the slit lamp has three rotating arms. The neutral position is designated as 0° and the center of the chin rest is designated 180°. The lower arm rotates the binocular microscope. It is rarely, if ever, necessary to move the rotating binocular microscope from the 0° position for the purposes of an ED examination. The next two arms are moved as a unit. The lower arm rotates the light 90° in either direction. The light source remains directed toward the structure that is being focused upon. The angle of the light is changed when the arm is rotated up to 90° in either direction. The standard position for the light is to rotate it 45° to the Emergency Physician's left to examine the patient's right eye and 45° to the Emergency Physician's right to examine the patient's left eye. Rotation of the upper arm independently rotates the slit of light in the coronal plane. It is unnecessary to rotate the two upper arms independently during a routine examination.

The eyepieces of the binocular microscope can be adjusted and focused independently in accordance to the Emergency Physician's needs. The standard starting position is in the 0 and 0 position. The eyepieces can be adjusted to compensate for refractory errors and to correct for the interpupillary distance.

Two levels of magnification are typically available (e.g., 10× and 16×). The switch to change magnification is located just below the eyepieces. The standard beginning position is low magnification. The higher power may be helpful when examining the details of the



A B

FIGURE 185-23. The light of the slit lamp. ***A.*** A narrow slit to focus on the cornea. ***B.*** A focused beam of light directed from a 45° angle illuminates the anterior chamber.

cornea or looking for cellular or inflammatory material within the anterior chamber.

The focused light can be adjusted in color, horizontal width, intensity, and vertical width. The standard position is for the light to be at its maximum size with a circular beam of white light at a moderate intensity. This position is used for scanning the eye and examining the external structures. Three independent adjustment mechanisms interact to create the most appropriate illumination to meet examination needs.

The color of the light and the intensity of the light originate from the same mechanism. Most of the positions represent variations in intensity of a white light. There are two options for color. The cobalt blue light is used for emphasizing corneal lesions that pick up fluorescein stain. The green light or red filter is helpful for patients who cannot tolerate the white light secondary to photophobia. This green light will cause red structures to appear darker or black.

The horizontal width of the light can be adjusted with the knob located at the base of the middle arm. The light can be narrowed to 1 or 2 mm or a "slit" for evaluating the depth of corneal lesions (**Figure 185-23*A***). The vertical control is used in conjunction with the horizontal control. Narrow the horizontal width to 2 to 3 mm and the vertical width to 2 mm when examining the anterior chamber. This creates a small focused beam. Rotate the light 30° to 60° to illuminate the depth of the anterior chamber (**Figure 185-23*B***). This orientation is most beneficial when attempting to identify cellular or inflammatory material within the anterior chamber.

Several portable slit lamps are available (**Figure 185-24**).[18] The Kowa SL-17 (Kowa Company LTD, Torrance, CA) is a hand-held device (**Figure 185-24*A***). It uses AAA batteries and can be used for 140 minutes. It is less than 9 inches tall, has two magnifications, three slit widths, and a blue filter. The Arclight (Arclight Medical, Liverpool, United Kingdom) is a pocket ophthalmoscope and otoscope (**Figure 185-24*B***). It charges with a USB cable or sunlight. It is meant for use in low-income countries, for mission work, and in areas where it is impractical to carry and use a regular slit lamp.

PATIENT POSITIONING

Cleanse the chin rest and the forehead bar with an alcohol swab before the slit lamp is used to examine a patient. The patient must be able to tolerate a seated position, leaning slightly forward with their head placed in the chin rest and their forehead pressed against the bar to use the slit lamp (**Figure 185-25**). The slit lamp examination cannot be completed in a patient who cannot remain in a seated position. Patients will often need to be reminded to keep their head in the chin rest and their forehead against the bar. Subtle movements from this position will change the viewing plane and disrupt

A B

FIGURE 185-24. Portable slit lamps. ***A.*** The Kowa SL-17. (Photo courtesy of Kowa Co. Ltd, Torrance, CA.) ***B.*** The Arclight. (Photo courtesy of Arclight Medical, Liverpool, United Kingdom.)

the focus on the structures of the eye. Always check the patient's position if you are having difficulty focusing the slit lamp.

The slit lamp can be adjusted to match the height of a chair or stretcher. The table moves up and down by adjusting the lever underneath the front of the table. The level of the chin bar can be adjusted to account for the subtle differences in the length of individual patient faces. Rotate the knob located at the base of the chin

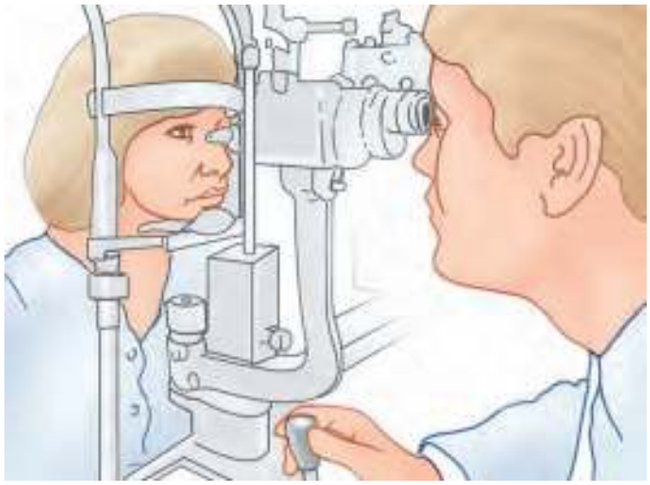


FIGURE 185-25. Patient positioning in the slit lamp.

rest apparatus to move it up and down. Raise or lower the chin bar so that the reference mark located below the forehead bar is at the patient's eye level.

Use the joystick to move the slit lamp and scan the patient's eye. The patient must keep their eye in a fixed position while the Emergency Physician uses the joystick to move the slit lamp and scan the eye. Instruct the patient to focus their eye on your shoulder or earlobe.

THE SLIT LAMP EXAMINATION

The slit lamp examination begins with an overall microscope-enhanced evaluation of the external structures of the eye including the lash line, the bulbar conjunctiva, the palpebral conjunctiva, and the lacrimal puncta. The cornea is best evaluated primarily with a wide beam and the fine details are evaluated with the slit. **Do not focus the light into the pupil for an extended time.** It is very uncomfortable for the patient and may result in injury. The normal cornea is perfectly clear and homogeneous. Note any deviation. Specific abnormalities are best documented in writing in addition to a basic schematic diagram.

The anterior chamber should be clear and without particulate matter. It may be difficult initially to identify cells floating in the anterior chamber. They appear like "dust in a sunbeam." The cells, if not clumped, are barely visible with the microscope at the lowest magnification. They often reflect the light slightly and this may not catch the examiner's eye. **Pay special attention to the lower one-fourth of the anterior chamber because cells and inflammatory products tend to settle and may form a meniscus in the upright position.** Flare is the noncellular inflammatory material in the anterior chamber that makes the aqueous humor appear hazy or gelatinous. Flare can obscure the details of the iris. Use the high-power magnification to identify particulate matter after scanning the anterior chamber with the low-power magnification.

ADMINISTRATION OF FLUORESCEIN

Repeat the corneal examination with the aid of fluorescein stain after the slit lamp examination is completed without stain. **Fluorescein is a hydrophilic substance that stains and illuminates any portion of the cornea where there is a breech in the epithelium.** The addition of fluorescein helps to illuminate corneal lesions that might otherwise go unrecognized (e.g., corneal abrasions or keratopathy associated with viral infections). Fluorescein is most

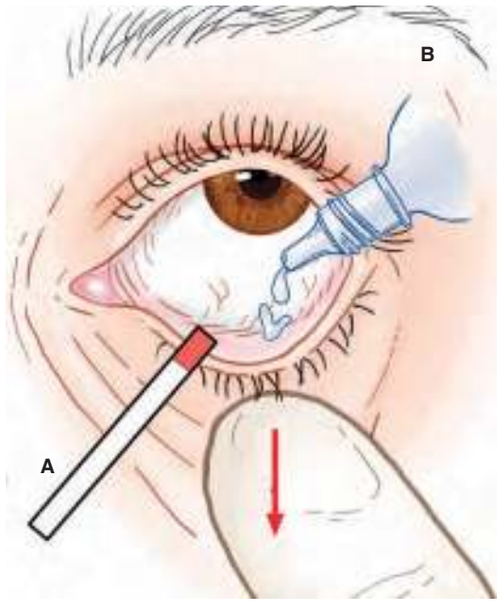


FIGURE 185-26. Instillation of fluorescein stain. ***A.*** Fluorescein-tipped strips. ***B.*** Aqueous fluorescein solution.

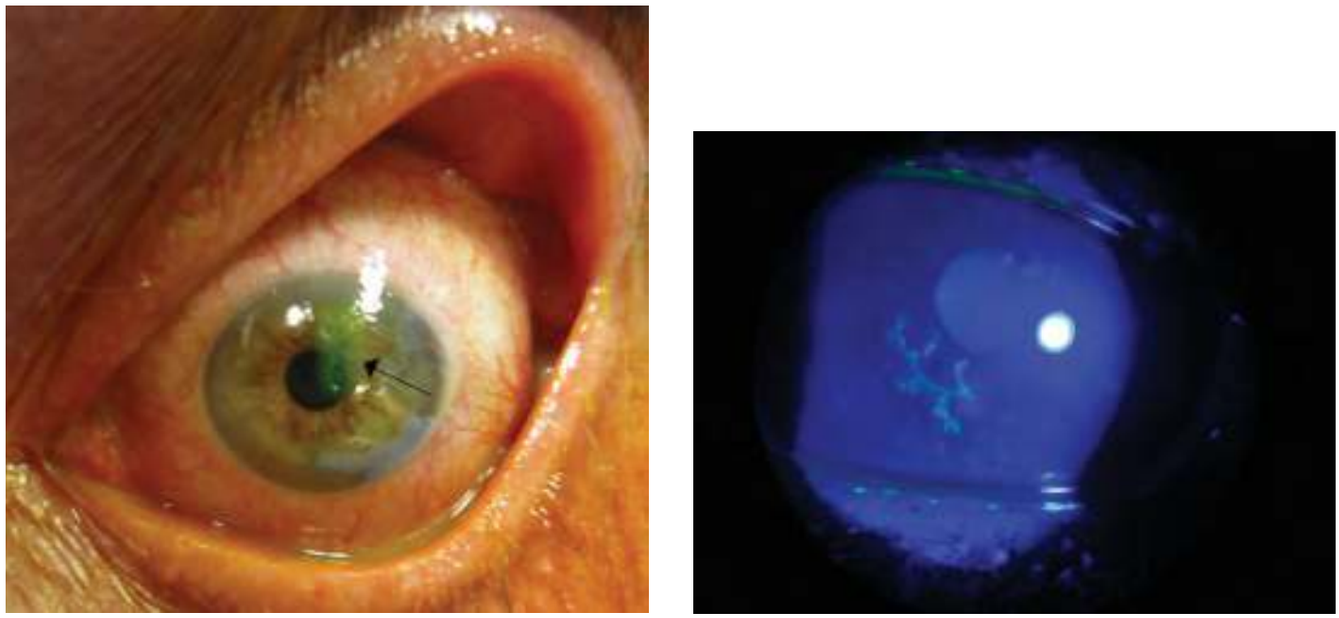
commonly packaged as individual single-use strips. There is the potential for infectious agents to be transmitted when one fluorescein strip is used for both eyes. Take care to avoid iatrogenically transmitted infections.

Remove contact lenses before the slit lamp examination and fluorescein staining. Place a small drop of saline or topical ophthalmic anesthetic solution onto the tip of the fluorescein strip. Retract the lower eyelid by placing pressure on the skin overlying the inferior orbital rim and distracting it downward (**Figure 185-26**). Ask the patient to look upward to facilitate the administration of the stain and to avoid inadvertent corneal injuries caused by the strip. Touch the fluorescein strip lightly against the palpebral conjunctiva of the inferior eyelid (**Figure 185-26*A***). Remove the fluorescein strip. Ask the patient to gently blink and distribute the fluorescein across the entire eye.

Aqueous fluorescein solution can be used as an alternative to the paper strips. Retract the lower eyelid and instill two drops of fluorescein into the cul-de-sac of the lower eyelid (**Figure 185-26*B***). The liquid solution or the tip of the applicator can become contaminated and transmit infection.

A small amount of fluorescein is required to adequately stain the cornea. A common mistake is to apply an excessive amount of fluorescein stain. This illuminates the entire tear film and makes it difficult to evaluate subtle lesions on the cornea. Ask the patient to blink several times to remove the excess fluorescein from the cornea. This maneuver will not remove the fluorescein adhering to any corneal defects. Place one or two drops of sterile saline or eyewash onto the eye if blinking does not remove the excess fluorescein. Avoid the use of excessive fluorescein as it is likely to obscure the exam and potentially stain the patient's skin.

The underlying cell layers of the cornea will hold the fluorescein stain if the corneal epithelium is injured. This allows corneal lesions to be visualized. Use the cobalt blue light, preferably in conjunction with the slit lamp, to identify and evaluate any corneal lesions that become apparent. The regions that stain with fluorescein and are visible with the cobalt blue light will appear bright yellow to yellow-green (**Figure 185-27**). **The green light on the ophthalmoscope and the slit lamp are not intended to be used**



A B

FIGURE 185-27. Corneal abnormalities seen on the fluorescein examination. ***A.*** Corneal abrasion. (Photo used from www.commons.wikimedia.org.) ***B.*** Herpetic dendrite.

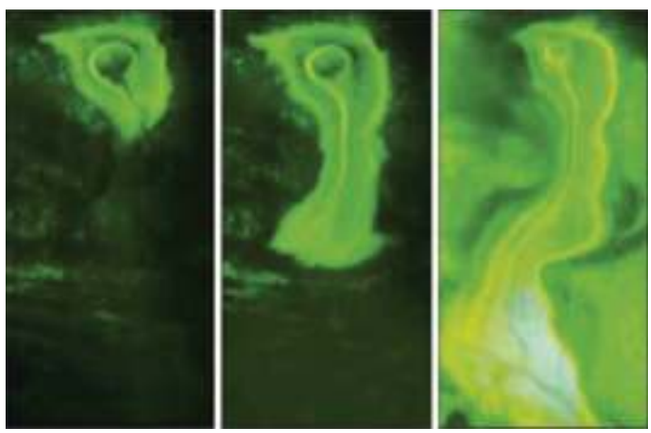
to illuminate the fluorescein stain. The green light will enhance the stain in some circumstances but is not as reliable as the cobalt blue light source.

The Seidel test uses fluorescein to assess the presence of anterior chamber leakage of a perforated eye (**Figure 185-28**).[15] It is named after the German Ophthalmologist Erich Seidel. The fluorescein "flows" from the site of an eye perforation with the aqueous humor.

EYEDROP ADMINISTRATION

TRADITIONAL METHODS

Topical ophthalmologic medications are used for examining and treating the eyes. Eyedrops and ophthalmologic ointments are most effectively and accurately instilled within the lower eyelid (**Figure 185-29**). Instruct the patient to direct their head toward the examiner with their eyes looking upward (**Figure 185-29*A***). Patients often close their eyes at this point. Retract the lower eyelid without placing pressure on the globe and expose the inferior cul-de-sac (**Figure 185-29*B***). Apply the drops or ointment in the cul-de-sac (**Figure 185-29*C***). **Close the eyelid and apply thumb pressure over the lacrimal duct (Figure 185-29*D*).** This prevents the liquid medicine from immediately draining through the nasolacrimal duct and into the nose. Instruct the patient to blink once or twice to quickly distribute the medication across the globe. Application of pressure on the lacrimal duct is unnecessary if instilling an ophthalmic ointment.



A B C

FIGURE 185-28. The Seidel test. ***A.*** The fluorescein attached to the injury. ***B*** and ***C.*** Progression of the aqueous humor flow. (Used with permission from reference 15.)

UNCOOPERATIVE PATIENTS

Some patients require but do not want medication in their eyes (e.g., infants, small children, and those with altered mental status). Prying the eyes of these patients open and applying the drops is difficult for both the Emergency Physician and the patient. Forcefully opening the eyelids is potentially dangerous because secondary injuries may occur. Most medication is typically applied to the Emergency Physician's hand or the stretcher as anyone who has tried this approach knows. An alternative approach in this situation is advisable.

The problem is the uncooperative patient squeezes their eyes closed tightly. Place the patient supine with an assistant holding the head in a fixed upright position with the eyes facing the ceiling to overcome this challenge (**Figure 185-30**). Note the small anatomic depression created over the medial canthus of each eye when they are closed tightly. Place the eyedrops in this anatomic depression to create a shallow pool (**Figure 185-30**). Firmly maintain the patient's head in this position until they spontaneously open their eyes and allow the medication to spread across the globe.

OPHTHALMIC ANESTHETIC AGENTS

Two types of eye medications are required in drop form for most routine ophthalmologic examinations (i.e., a topical ophthalmic anesthetic agent and a dilating agent). **Topical ophthalmic**

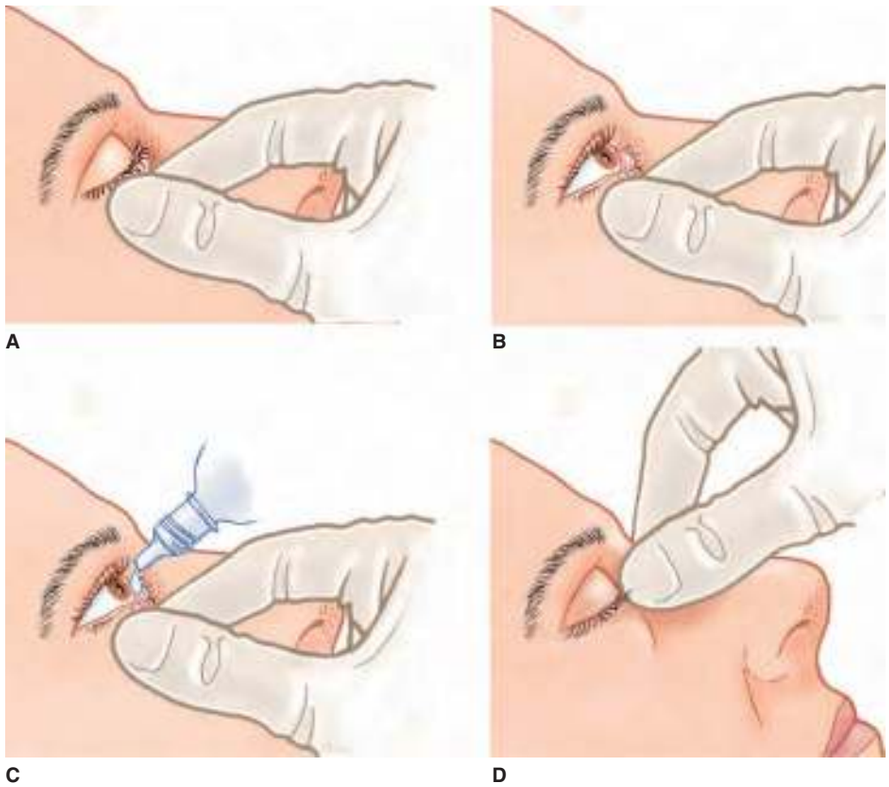


FIGURE 185-29. The instillation of eyedrops. ***A.*** Grasp the lower eyelid. ***B.*** Pull the lower eyelid downward and outward to expose the cul-de-sac. ***C.*** Place the drops into the cul-de-sac. ***D.*** Apply thumb pressure over the lacrimal duct to prevent drainage of the drops into the nose.

anesthetic agents provide relief from eye pain and photophobia. This may help the patient to cooperate with the necessary examination. Topical ophthalmic anesthetic agents may be used as adjuncts to fluorescein application and to facilitate the placement of eyelid retractors. They may at times be used as an adjunctive diagnostic tool. There are several possible causes of eye pain and photophobia that must be differentiated, some of which may coexist. Complete symptom relief with a topical ophthalmic anesthetic agent suggests that the etiology of the pain is from the conjunctiva or cornea.

FIGURE 185-30. The instillation of eyedrops in an uncooperative patient.

Tetracaine hydrochloride and proparacaine hydrochloride are the most commonly available agents. Their onset of action and duration of action are similar. The onset is within 1 minute and lasts approximately 10 to 20 minutes. Tetracaine hydrochloride (Pontocaine) instillation often results in a transient stinging sensation that lasts 3 to 10 seconds before dissipating. Proparacaine hydrochloride (Ophthetic or Ophthaine) does not result in the stinging sensation.

Topical ophthalmic anesthetic agents are toxic to corneal epithelial cells and can delay corneal healing.[19,20] They are currently used in the ED only to aid in the diagnosis and evaluation of an eye problem. As a rule, they are not prescribed for treatment outside the ED. These agents are currently being used for up to 4 days on an outpatient basis after photorefractive keratotomy surgery and some other conditions.[21] Several small studies suggest these agents are safe to use for a few days without adverse effects.[22-25] The ED practice of not prescribing topical ophthalmic anesthetic agents for outpatient use may change. A large study is needed to look at the safety of using these medications as an outpatient. The decision is up to the individual Emergency Physician. Do not consider using topical anesthetics for complicated corneal abrasions (e.g., 36 hours or longer after injury, both eyes injured, children, contact lens wearers, contaminated wounds, herpetic keratitis, tetracaine allergies, and unreliable patients). Use dilute 0.5% topical anesthetics if they are prescribed and for no more than 48 hours.

PUPILLARY DILATING AGENTS

Dilating agents are used as an adjunct to the funduscopic examination. **Always perform and document a complete pupillary examination before instilling dilating agents.** It is important to document the use of dilating agents and the time of administration.

Avoid using dilating agents in patients who require serial pupillary examinations to follow their neurologic status. Dilating agents are contraindicated in patients with a known or suspected narrow iridocorneal angle. Dilating the pupil may result in acute angle-closure glaucoma. Some intraocular lens implants may become dislodged if the pupils are pharmacologically dilated. Inquire and document any glaucoma, known narrow angles, and eye surgery prior to dilating any pupils. Consult an Ophthalmologist before dilating these patients' eyes.

Dilating agents are categorized as mydriatics and cycloplegics. All dilating agents are covered with a red cap. Mydriatic agents directly dilate the pupil whereas cycloplegics paralyze the ciliary muscles. Do not use cycloplegic agents for patients who require serial neurologic examinations. Choose a noncycloplegic mydriatic agent (e.g., phenylephrine) to dilate the pupils of a patient requiring serial examinations. The duration of action is significantly shorter than that of a cycloplegic agent. Phenylephrine is systemically absorbed and may be inappropriate for patients with hypertension or cardiac conditions. A combination of a mydriatic agent (e.g., phenylephrine) and a shorter-acting cycloplegic agent (e.g., cyclopentolate) is optimal when maximal dilation is necessary or when a single agent is ineffective.

Most EDs stock or have access to five dilating agents. The choice of agents depends upon the length of time required for dilation, the need for a cycloplegic agent, and the need for a mydriatic agent. Atropine (Isopto Atropine) produces cycloplegia lasting 5 to 10 days and mydriasis lasting 7 to 14 days. Cyclopentolate (Cyclogyl, AK-Pentolate, Pentolair) produces cycloplegia lasting 6 to 24 hours and mydriasis lasting 24 hours. Homatropine (Isopto Homatropine) produces cycloplegia and mydriasis lasting 1 to 3 days. Phenylephrine (Neo-Synephrine, Mydrin, Relief) produces mydriasis lasting 5 hours. Tropicamide (Mydriacyl) produces mydriasis lasting 4 to 6 hours.

THE FUNDOSCOPIC EXAMINATION

A fundoscopic examination is an essential part of every eye evaluation. Examine the fundus with an ophthalmoscope (**Figure 185-31**). It is particularly helpful when the patient complains of visual disturbances or visual loss. Examine the patient's right eye first. Hold the ophthalmoscope in the right hand and use the right eye to examine the patient's right eye. Remember "right eye to right eye and left eye to left eye" or the Emergency Physician may otherwise find they are in an awkward nose-to-nose position with the patient. Instruct the patient to focus the eye not being examined on a fixed object over the Emergency Physician's shoulder and approximately 6 to 8 feet away. It is important that the patient maintain their focus on the object throughout the complete examination of the eye. It is helpful to tell them to focus on the object with the eye not being examined. This will result in a stationary eye for examination.

Set the ophthalmoscope initially at 0. Adjust the dial as the examination begins to adjust for refractory errors. Moving the dial into the red numbers moves the point of focus forward toward the red retina. Moving the dial into the black numbers moves the point of focus closer to the Emergency Physician. Approach the eye from a slightly lateral position to quickly identify the medial position of the optic disk. Position the ophthalmoscope as close as possible to the patient's eye without making contact. Imagine looking at the fundus through a peephole. The closer you get, the wider the view on the other side. By the same analogy, the larger the peephole, the better the view on the other side. Dilate the patient's pupils if safe to do so to obtain the best results from the ophthalmoscope examination.

FIGURE 185-31. The ophthalmoscope. ***A.*** A standard version. ***B.*** The Panoptic.

Adjust the diameter of the light down to the size of the patient's pupil to avoid the bright reflection from the iris if dilation is not possible. This bright reflection is a common reason for inadequate visualization of the fundus. The patient's eyes are often moving if the structures seem to move in and out of focus. Remind the patient to focus on one point. Inspect the eye grounds for uniformity (**Figure 185-32**). The normal color of the fundus is creamy peach to pink but may be pigmented in darker skinned people. A pale fundus with a small cherry-red spot in the region of the macula suggests an infarction generally associated with a retinal artery occlusion. The optic disk is located on the nasal aspect of the fundus. If the

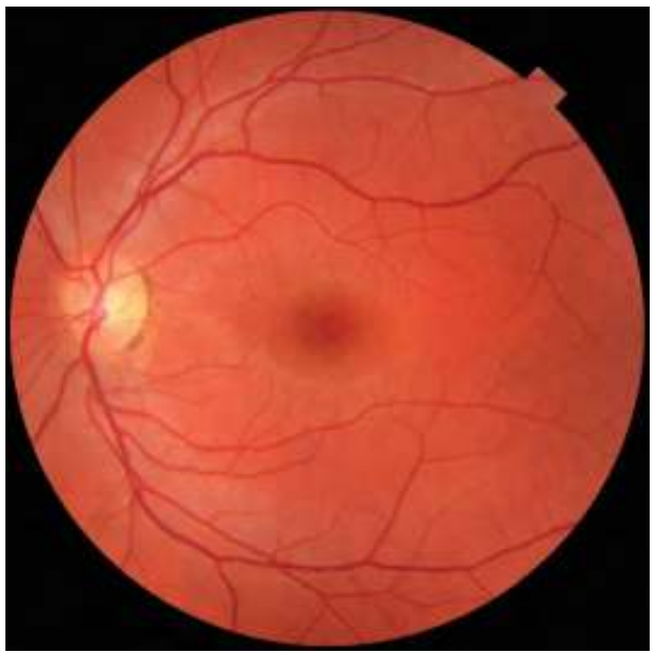


FIGURE 185-32. The normal fundus as seen through an ophthalmoscope. The macula and optic disk are clearly visible.

optic disk does not come into view, follow the blood vessels that extend from the disk to the periphery of the retina. The disk margin should be sharp except the nasal aspect that may appear slightly blurred in a normal eye (**Figure 185-32**). Papilledema appears as diffusely blurred disk margins (**Figure 185-33**). Papilledema may be the result of localized inflammation of the optic nerve or elevated intracranial pressure (**Figure 185-33**).

The veins will appear darker and wider than the arteries (**Figure 185-32**). Evaluate the blood vessels for evidence of hemorrhages and irregularities. Acute hemorrhages may be helpful in the diagnosis of a variety of problems (e.g., hypertensive emergency or the shaken baby syndrome). Carefully consider the clinical setting. A helpful finding in the patient suspected of having increased intracranial pressure is the presence or absence of venous pulsations. Venous pulsations are best seen as the veins cross into the optic cup. The presence of venous pulsations is a normal finding. The first sign as the intracranial pressure rises is a loss of venous pulsations followed later by papilledema. The green light or red filter helps to provide a sharper image when evaluating the retinal vessels in detail.

FIGURE 185-33. Papilledema as seen through the ophthalmoscope.

Examine the fovea and the surrounding macula (**Figure 185-32**). This area is located approximately 2 to 3 disk diameters (DD) lateral to the optic disk. Instruct the patient to look directly into the light to bring the fovea and macula into view. The target setting on the ophthalmoscope is helpful for this purpose. Look using the ophthalmoscope turned to the target. Instruct the patient to look directly into the center of the target. The fovea will be found at the center of the target in your view.

Examine the vitreous chamber for evidence of a vitreous hemorrhage when a retinal detachment is suspected or when the patient complains of floaters. The fluid within the vitreous chamber is gelatinous so blood within it does not dissipate quickly. Blood within this chamber appears as clouds, spots, or veils to the patient and the Emergency Physician. Their color is typically described as black or red. Set the ophthalmoscope to +10 to move the point of focus anteriorly toward the Emergency Physician to evaluate the vitreous. Dial the ophthalmoscope down as the examination proceeds. Each click results in focusing a little deeper within the vitreous chamber until the point of focus is upon the retina at the posterior aspect of the chamber.

INTRAOCULAR PRESSURE MEASUREMENT

Include the measurement of intraocular pressure in the eye examination (Chapter 188). It is particularly important in patients with eye pain and visual loss.

OCULAR ULTRASOUND

The application of bedside ultrasound (US) has become a widely used tool for the rapid diagnosis of ocular pathology in the ED. Its use by Ophthalmologists has been described as early as the 1950s. The use of US has been popularized by high-resolution machines allowing Emergency Physicians to detect subtle abnormalities (e.g., globe rupture, increased intraocular pressure, lens dislocation, retinal detachment, retinal hemorrhage, and vitreous hemorrhages). This section covers the basic anatomy, common ocular pathology, and techniques of ocular US.[26-28] An US examination can be performed quickly and without the patient leaving the ED; in addition, US machines are readily available and use no ionizing radiation.

ULTRASOUND TECHNIQUE

Perform ocular bedside US with a high-frequency, 7.5 to 10 MHz, linear transducer to scan the eye in two axes. Place the patient supine or in a 45° sitting position with their neck slightly extended. Apply a generous amount of US gel over the closed eye (**Figure 184-34*A***). An alternative to having the patient keep their eyelid closed is to place a clear dressing (e.g., Tegaderm) over the closed eyelid (**Figure 185-34*B***) and to place the gel over it. Brace the hand on the forehead to balance the transducer to offset any pressure over the eye. It is extremely important to avoid pressure, especially in the case of an undiagnosed globe rupture, in which US is contraindicated. Orient the transducer marker toward the patient's head in a longitudinal axis or to the right of the patient in a transverse axis while the patient is asked to look to the right and left to create a three-dimensional view of the eye (**Figure 185-35**).

A B

FIGURE 185-34. US of the eye through the closed eyelids. ***A.*** Without a clear dressing. (Used with permission from reference 33.) ***B.*** With a clear dressing. (Used with permission from www.aliem.com.)

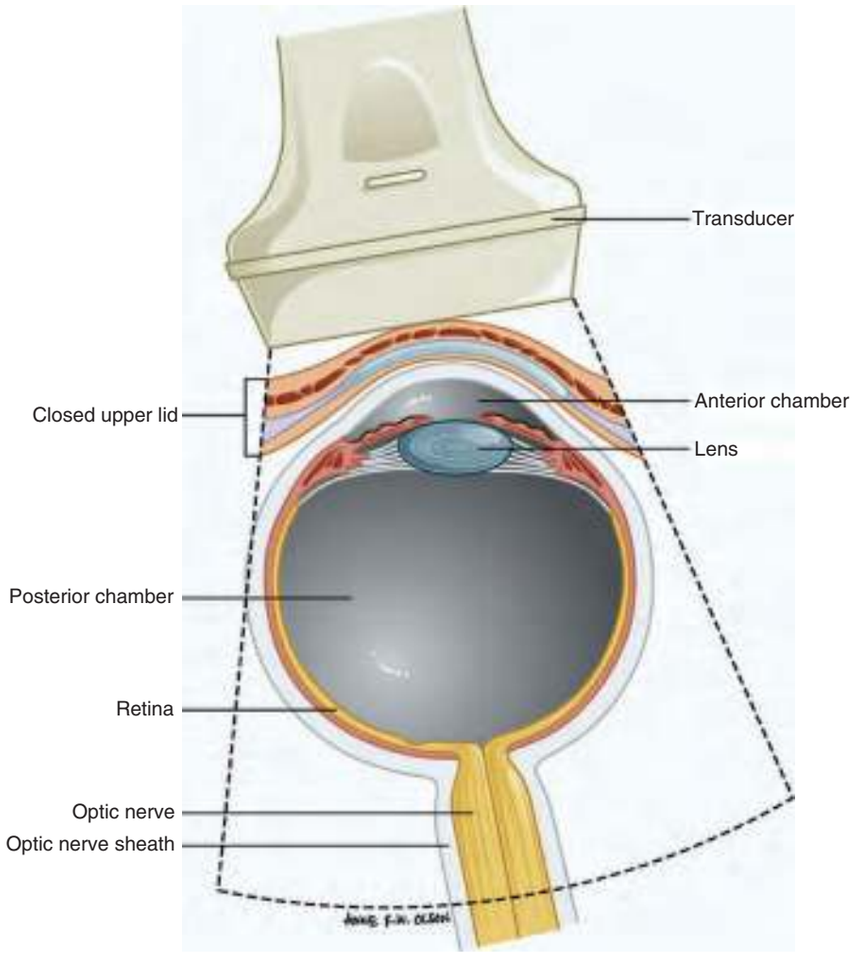


FIGURE 185-35. The US through the closed eyelids. (Used with permission from reference 33.)

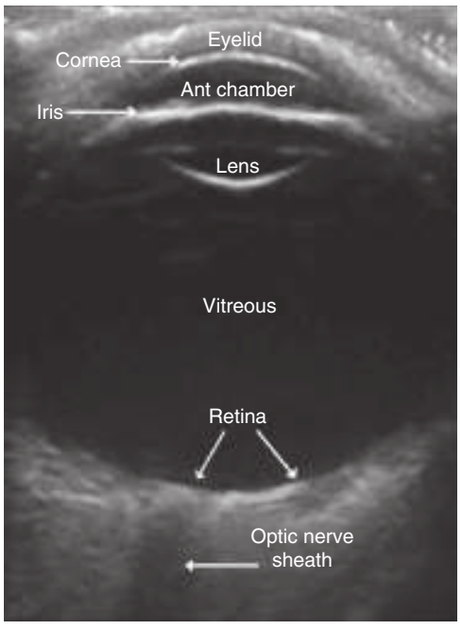


FIGURE 185-36. US image of eye anatomy. (Photo courtesy of Jennifer Cotton, MD, and www.sonomojoultrasound.org.)

OCULAR ULTRASOUND ANATOMY

A detailed overview of the eye anatomy is beyond the scope of this chapter. This section covers the relevant structures of the eye that can be visualized using US. The anatomy of the eye and surrounding structures can be seen in **Figures 185-1, 185-35, and 185-36**.

RETINAL DETACHMENT

Retinal detachments can be difficult to diagnose in the ED. The visualization of the posterior structures of the eye using US can make the diagnosis less challenging and may facilitate rapid treatment in the case of time-sensitive pathology (**Figure 185-37**).[26,29] Detection of retinal detachment has a sensitivity of up to 97% and specificity of 92%. Other small studies have shown a sensitivity of up to 100% and specificity of 83%. US findings of retinal detachments are significant for a string-like echogenic structure attached to the posterior globe.[26,29]

VITREOUS HEMORRHAGE

The diagnosis of a vitreous hemorrhage suspected by history and ophthalmic examination may be confirmed with US.[26,30] Hyperechoic globular and/or granular opacities will be present in the posterior globe (**Figure 185-38**). The appearance will be delineated

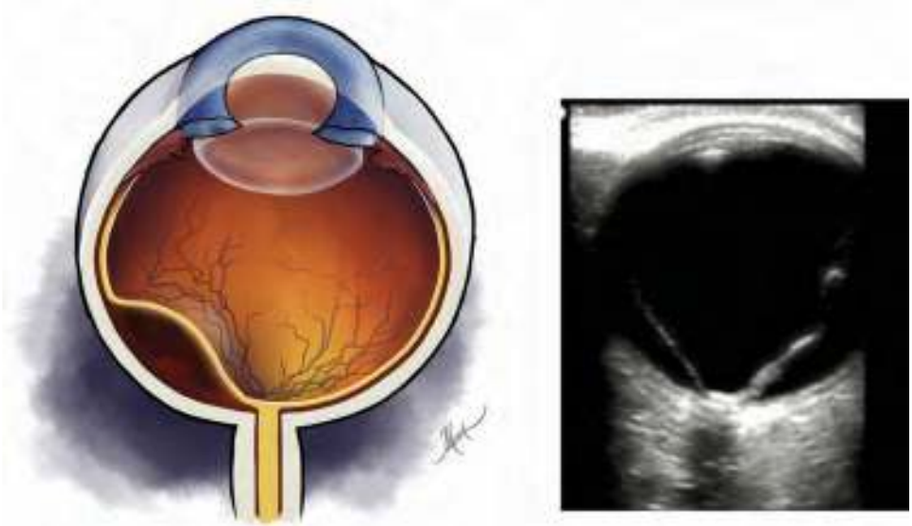


FIGURE 185-37. Retinal detachment. An artist illustration (left) and an US (right). The retina is completely detached in the US image. The retina is firmly attached to the optic nerve posteriorly and the ora serrata anteriorly to produce the V shape. (Used with permission from reference 35.)

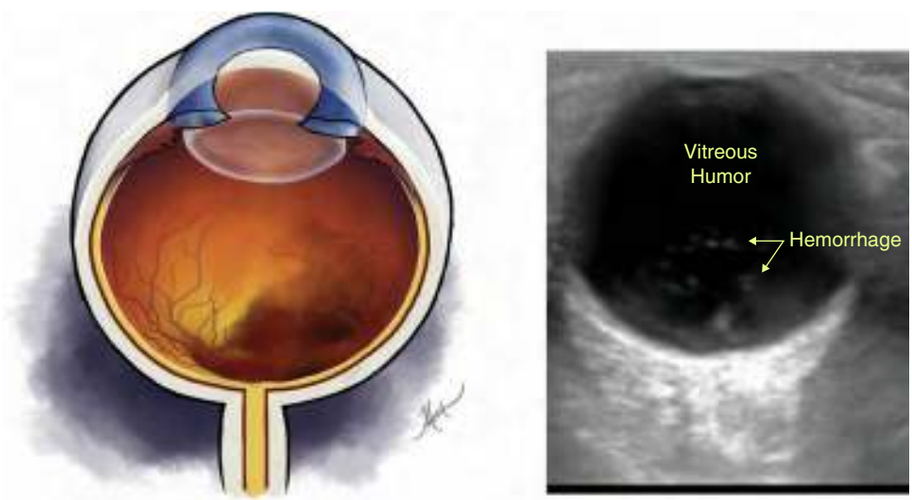


FIGURE 185-38. Vitreous hemorrhage. An artist illustration (left) and an US (right). (Used with permission from reference 35.)

FIGURE 185-39. Optic nerve sheath diameter (ONSD) measurement to assess for increased intracranial pressure. Distance 1 is the distance (i.e., 3 mm) behind the optic disk where the ONSD is measured. Distance 2 between the white arrows is the ONSD. (Used with permission from reference 35.)

when asking the patient to look to their left and right. These ocular movements create a swirling appearance in the posterior globe. This is a classic finding in vitreous hemorrhages known as the "washing machine" sign. Increasing the gain on the US machine may be necessary to better visualize posterior structures, especially with subtle abnormalities.[26,30]

INCREASED INTRACRANIAL PRESSURE

Intracranial pressure can be assessed with accuracy with the use of ocular US by measuring the optic nerve sheath diameter.[31-33] Several studies have compared its use with computed tomography for assessment of intracranial pressure. A meta-analysis shows a sensitivity of 95% and specificity of 95%. The optic nerve sheath appears as a hyperechoic vertical structure in the posterior globe (**Figure 185-39**). Measure the optic sheath diameter 3 mm posterior to the globe (**Figure 185-39**). An optic nerve sheath diameter more than 5 mm corresponds to an intracranial pressure above 20 mmHg. Measure the optic nerve sheath diameter bilaterally for comparison.[28,33,34]

SUMMARY

Approach all eye complaints with a detailed history that includes a chief complaint, the duration of symptoms, and the natural history of their evolution. It is important to inquire about exposures and trauma. The past medical history must include the patient's baseline visual acuity and any history of eye problems. Perform a complete examination on all patients with eye complaints. Always document an accurate visual acuity and carefully inspect the eye structures. A thorough approach in all patients with eye complaints improves diagnostic accuracy. The result is a decrease in morbidity and long-term complications in this vital sensory apparatus.

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186 Contact Lens Removal

Scott A. Heinrich and Dino P. Rumoro

INTRODUCTION

The Emergency Physician must be familiar with the proper technique of removing soft and hard contact lenses from patients who are unable to do so on their own. Patients with altered mental status are at risk of corneal damage if contact lenses remain in place. Healthy individuals who wear contact lenses overnight experience a 4- to 15-fold increase in the risk of corneal injury compared with those who remove their contact lenses daily.[1] The explanation for this increased risk of injury is based on the development of corneal hypoxia and an immune response to antigens present on the lens surface, both of which lead to an inflammatory response and susceptibility to infectious organisms.[1] This results in an increased incidence of ulcerative keratitis, *Pseudomonas aeruginosa* infection, and corneal neovascularization.[2,3]

ANATOMY AND PATHOPHYSIOLOGY

Contact lenses rest on a three-layer tear film (i.e., outer lipid, middle aqueous, and inner mucus layer) that covers the corneal and conjunctival epithelium. This tear layer provides oxygen and nutrients to the avascular cornea. The cornea receives nutrition from blood vessels at the limbus and the aqueous humor. It is believed that contact lenses increase tear evaporation and disrupt the three-layer tear film.[4] This leads to the lack of corneal oxygenation and the symptoms of dry eye.[4] A dry eye causes discomfort and corneal edema with resultant hazy vision. The normal blinking action initiates contact lens movement and a "fresh" flow of oxygenated tears over the cornea in an awake patient. Blinking is not present in the sleeping or comatose patient.

The normal resting position of the contact lens is over the cornea. It may occasionally drift from the center of the eye and relocate. It may relocate over the sclera, in various parts of the eye socket, or under the upper eyelid. **Thorough exploration of all aspects is essential when evaluating an individual for the presence of contact lenses prior to their removal.** Inspect under the lower eyelid margin and eversion of the upper eyelid. This requires double eversion of the eyelid for identification and retrieval (Chapter 185).[5] **Failure to adequately perform this examination can lead to the mistaken belief that a contact lens does not exist.** A contact lens that remains in place acts as a foreign body and can lead to chronic irritation, inflammation, and development of a mass. Mass development from a retained contact lens typically occurs in the upper fornix of the eyelid.[5]

INDICATIONS

Contact lenses must be removed from any patient who is unconscious or suffers an ocular injury. Remove contact lenses before fluorescein stain is used to examine the eye. Fluorescein can permanently stain the contact lens material. Give patients the opportunity to remove their own contact lenses if there are no contraindications (i.e., immobilization or ocular trauma). Patients are usually quite adept at removing their own contact lenses. Remove the contact lenses if the patient is unable to remove them or cannot remove them.

CONTRAINDICATIONS

The only absolute contraindication to removing a contact lens would be in the case of a ruptured globe. Leave the contact lens in place for the Ophthalmologist to remove at the time of their examination and/or surgical repair. Extreme caution must be exercised to avoid unnecessary pressure on the eye to not complicate the injury when severe ocular damage has occurred. Place an eye shield and not an eye patch to avoid pressure on the globe with resultant exacerbation of the injury (Chapter 193).

EQUIPMENT

- Normal saline
- Two cups, labeled "left" and "right"
- Hard lens remover suction cup device, optional
- Soft lens remover device, optional
- Cotton-tipped applicators

PATIENT PREPARATION

Explain to the patient, if they are awake, that their contact lenses must be removed. Explain the risks, benefits, and alternative procedures to the patient and/or their representative. A signed consent is not required to remove a contact lens. Place the patient sitting or supine, whichever is most appropriate for the current clinical situation. Place several drops of a saline solution onto the eye and wait 5 to 10 minutes to allow the saline to penetrate the lenses. This maximally moistens the contact lenses to the point where they can be seen to slide easily over the surface of the eye when the patient blinks.

All contact lenses should be centered over the cornea for ease of removal by gentle manipulation of the eyelids. The lenses may become displaced from the cornea. Care must be taken to search for the lens in other parts of the eye socket. Shine a penlight at an angle to the eye to aid in the search. A common location for a displaced contact lens to migrate is under the upper eyelid. **Evert the upper eyelid (Chapter 185) if a contact lens cannot be found elsewhere to complete the search before concluding that the lens fell out or the patient is not wearing contact lenses.** Instill fluorescein into the eye if the patient still insists that it is present. **The fluorescein will pool around the edges of the contact lens and make it easy to locate. Warn the patient that fluorescein may permanently stain their contact lens.**

Prepare the equipment. Locate the equipment required to remove the contact lenses. Label two cups with "left" and "right" in which to place the contact lenses after they are removed. Fill the cups with enough saline to cover the contact lenses. Wear powderless gloves to prevent the powder from irritating the eye. Wipe powdered gloves clean with a saline- or a water-moistened towel to remove the powder. Powdered gloves can be placed under running water to flush away the powder.

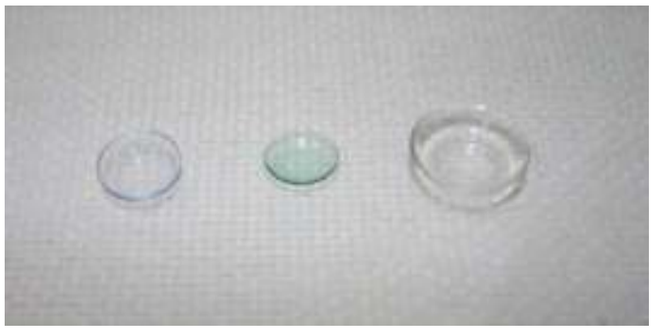

FIGURE 186-1. Examples of contact lenses. From left to right: soft lens, hard lens, and scleral lens.

HARD CONTACT LENS REMOVAL TECHNIQUES

A hard contact lens can be identified by its small size of 6 to 10 mm (**Figure 186-1**). It is smaller than the cornea. Center the hard contact lens on the cornea. Place the index finger (or thumb) of one hand at the base of the eyelashes of the upper eyelid and the index finger (or thumb) of the opposite hand at the base of the eyelashes of the lower eyelid (**Figure 186-2*A***). Gently, but firmly, approximate the eyelids by moving them toward the center of the cornea until the margins of the eyelids touch the edges of the hard contact lens (**Figure 186-2*B***). Press slightly harder on the lower eyelid until the bottom edge of the hard contact lens lifts off the cornea using the edge of the lower eyelid as a fulcrum (**Figures 186-2*C* and 186-2*D***). Continue to push the eyelids together until the hard contact lens is lifted completely off the cornea and can be easily grasped.[6,7]

The patient who is awake and alert may not like the Emergency Physician's fingers near their eyes. Pull the skin of the lateral margin of the eyelids laterally with an index finger (**Figure 186-3*A***). Alternatively, place one finger on the lateral edge of the upper eyelid and one finger on the lateral edge of the lower eyelid and pull laterally (**Figure 186-3*B***). Instruct the patient to look downward and inward toward their nose. The hard contact lens will pop off the cornea. Grasp and remove the contact lens. This technique does not put as much pressure on the globe as the previous technique which is an advantage if the patient has ocular trauma.

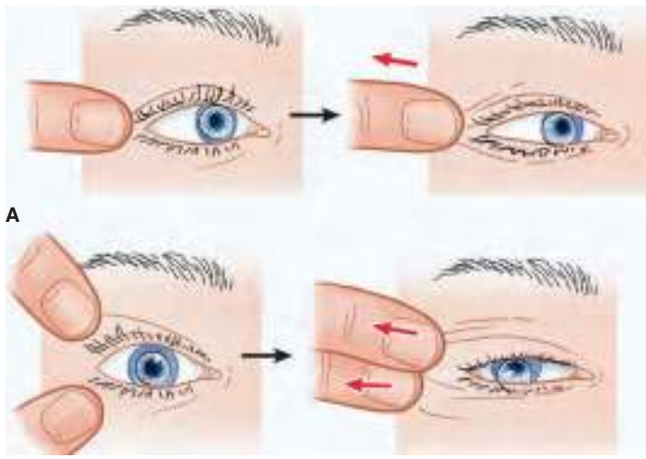

FIGURE 186-3. Alternative hard contact lens removal techniques. These methods do not apply pressure onto the globe. Apply laterally directed pressure to the skin lateral to the eyelids (***A***) or to the eyelids (***B***) to "catch" the contact lens and pop it off the eye.

A commercially produced suction cup-like rubber device, resembling a golf tee, can be used to remove hard contact lenses if available (**Figure 186-4**). Moisten the surface of the device with a drop of saline. Gently touch the suction cup to the center of the hard contact lens. Suction will form and result in the hard contact lens adhering to the device. Lift the device and the attached contact lens from the cornea. **Slide the hard contact lens sideways to remove it from the suction cup. Do not attempt to pull the hard contact lens off the suction cup as this may damage the contact lens.**

A final technique involves the use of a cotton-tipped applicator (**Figure 186-5**). Moisten the cotton with saline. Place the cotton-tipped applicator over the lower edge of the hard contact lens (**Figure 186-5*A***). Carefully and gently slide the hard contact lens off the cornea and onto the sclera with the moistened cotton-tipped applicator. Gently press the cotton-tipped applicator into the sclera and under the edge of the hard contact lens (**Figure 186-5*B***). Lift

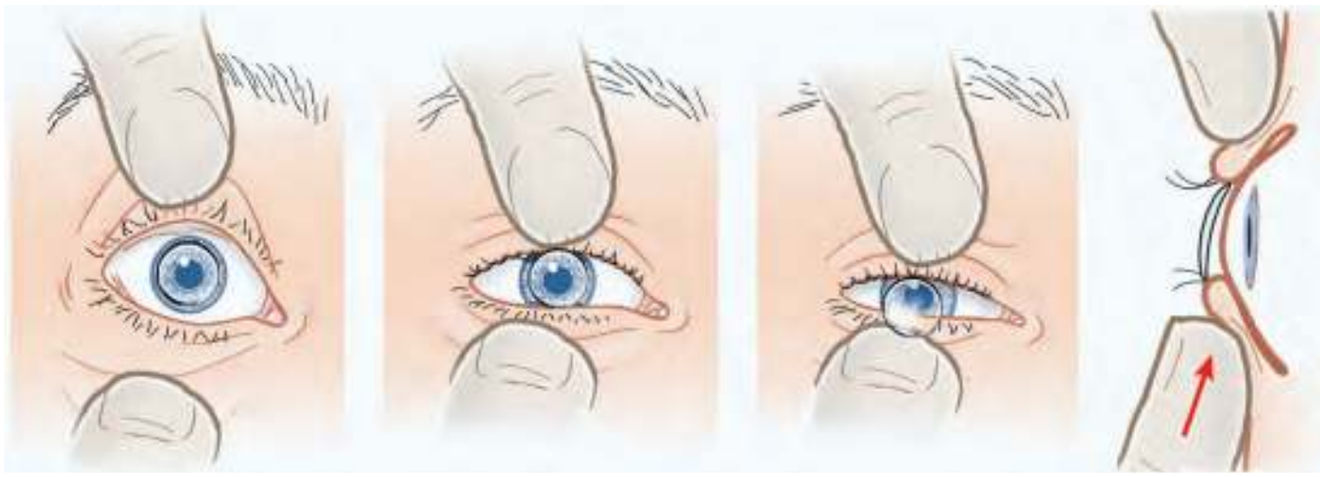

FIGURE 186-2. Hard contact lens removal. ***A.*** Use both thumbs to open the eyelids. ***B.*** Close the eyelids until the edges of the eyelids just contact the lens. ***C.*** Push the edge of the lower eyelid under the edge of the contact lens to pop it off the eye. ***D.*** Lateral view of the lower eyelid pushing the contact lens off the eye.

FIGURE 186-4. Suction cup removal of hard contact lenses. ***A.*** Suction cup on a plastic handle. ***B.*** Fingertip-held suction cup.

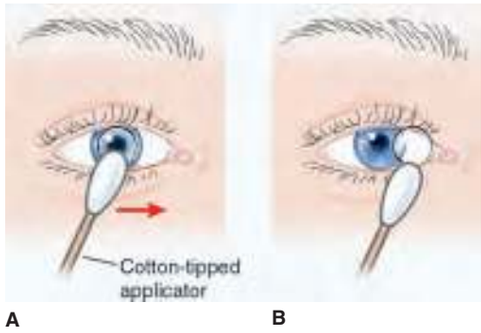

FIGURE 186-5. A cotton-tipped applicator to remove a hard contact lens. ***A.*** Place a moistened cotton-tipped applicator against the lower edge of the hard contact lens. Push the hard contact lens onto the sclera. ***B.*** Push the moistened cotton-tipped applicator under the edge of the contact lens to lift it off the eye.

the hard contact lens from the sclera. **Do not use the cotton-tipped applicator to elevate the hard contact lens from the cornea as this can result in a corneal abrasion.**[8]

SOFT CONTACT LENS REMOVAL TECHNIQUES

Soft contact lenses can be identified by their larger sizes of > 12 mm (**Figure 186-1**). They usually extend to, or just beyond, the corneal-scleral junction. There are numerous techniques to remove a soft contact lens (**Figures 186-6, 186-7, and 186-8**). The easiest and simplest method is to remove it manually (**Figure 186-6**). Retract the lower eyelid with the nondominant index finger (**Figure 186-6*A***). The soft contact lens will slide partially onto the conjunctival surface of the lower sclera. Gently grasp the soft contact lens between the thumb and index finger of the dominant hand. Pinch the fingers together and remove the soft contact lens.

A second technique to remove soft contact lenses is illustrated in **Figure 186-6*B***. Gently place the index finger and thumb of the nondominant hand on the upper and lower eyelids, respectively. Retract the eyelids. Gently grasp the soft contact lens between the thumb and index finger of the dominant hand. Slide the soft contact lens inferiorly. Gently pinch the fingers together to pull the soft contact lens from the eye.[6]

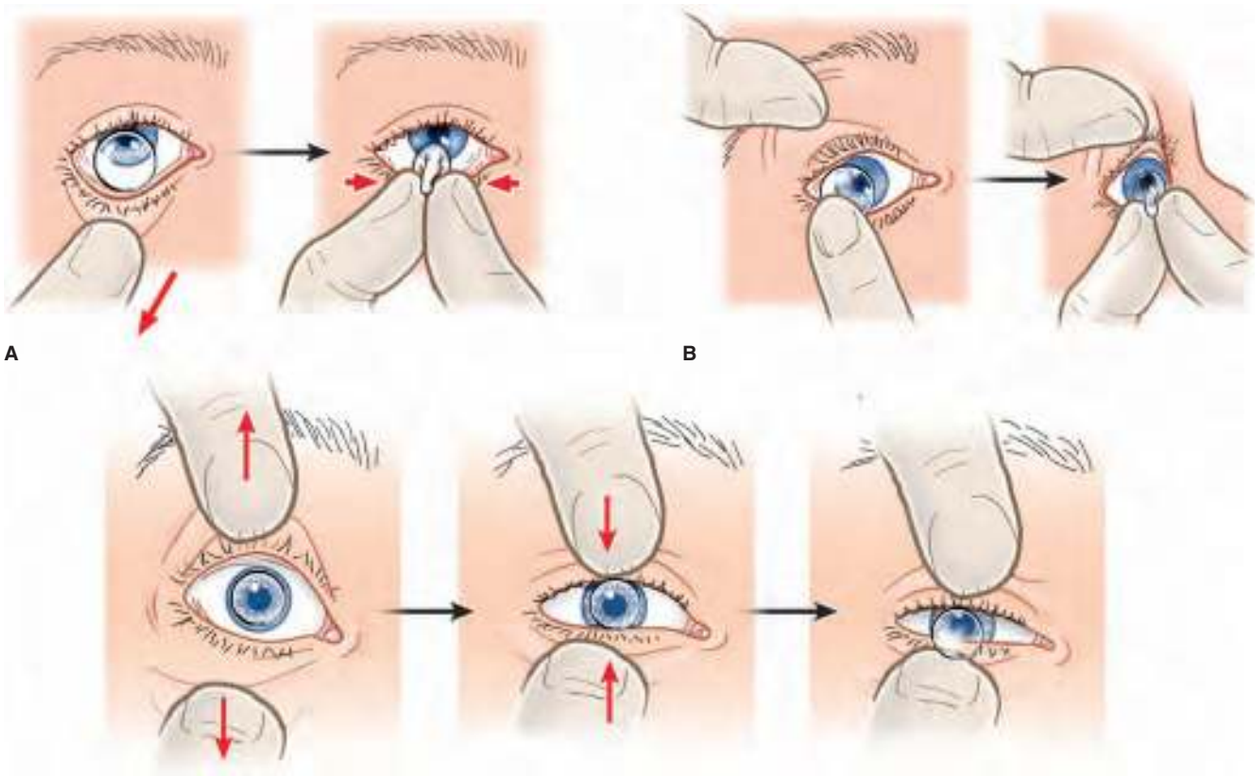

FIGURE 186-6. Manual soft contact lens removal techniques. ***A.*** Pull the lower eyelid downward and grasp the contact lens. ***B.*** Retract the eyelids. Slide the soft contact lens off the cornea and onto the sclera. Grasp and remove the contact lens. ***C.*** Use the patient's eyelids to pop the contact lens off the eye.

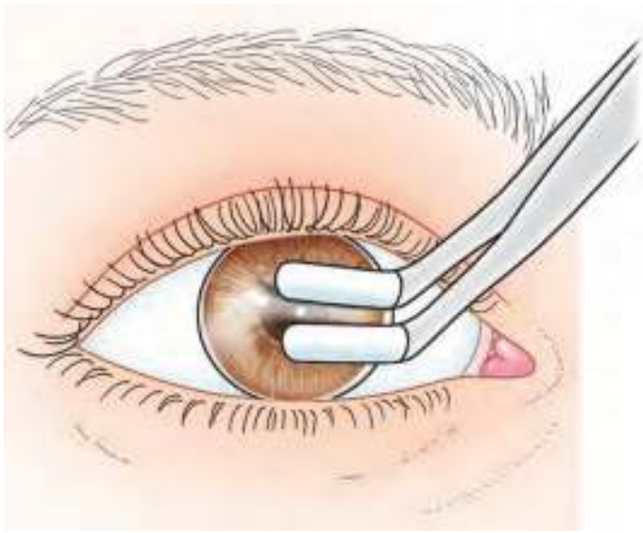

FIGURE 186-7. A tweezer-like device to grasp and remove the soft contact lens.

A third technique to manually remove a contact lens uses the patient's eyelids (**Figure 186-6*C***). Place the thumb of the nondominant hand and dominant hand on the upper and lower eyelid, respectively. Retract the eyelids until the edges of the contact lens are fully visible. Close both eyelids against the superior and inferior edges of the soft contact lens. Continue to close the eyelids until the contact lens pops off the eye. Grasp and remove the soft contact lens with the dominant hand.

A commercially available rubber tweezer-like device can be used to remove soft contact lenses. Place the rubber tips of the device onto the center of the soft contact lenses (**Figure 186-7**). Gently squeeze the tweezers closed using minimal pressure. The soft contact lens will fold and lift off the eye. Remove the soft contact lens from the eye.

A commercially available rubber disc on a stick can be used to remove soft contact lenses. Place the rubber disc over the center of the soft contact lens (**Figure 186-8**). Apply gentle pressure to slide the soft contact lens onto the sclera. Lift the stick to remove the attached soft contact lens. The rubber disc may occasionally not stick to the soft contact lens. Apply a drop of saline to the rubber disc and repeat the procedure. The drop of liquid will form a seal between the rubber disc and the soft contact lens.

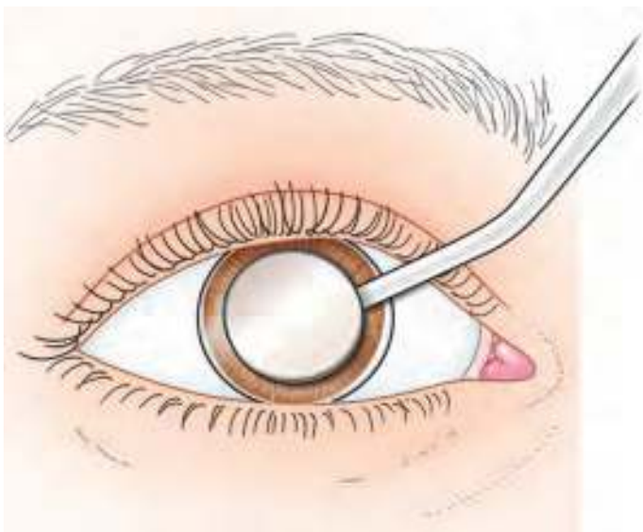

FIGURE 186-8. A rubber pad to remove a soft contact lens.

SCLERAL LENS REMOVAL TECHNIQUES

The scleral lens is essentially a very large, soft contact lens (**Figure 186-1**). It is approximately the size of a quarter and covers the cornea and sclera. Remove the scleral lens using the same techniques described to remove a soft contact lens.

ALTERNATIVE TECHNIQUE

An alternative technique was described to use moistened cotton-tipped applicators (**Figure 186-9**).[9] This technique uses two moistened cotton-tipped applicators like chopsticks to remove a contact lens. This technique may be used when the patient does not like the Emergency Physician's fingers near their eyes or as an alternative to using one applicator.

AFTERCARE

Place removed contact lenses in the appropriately marked container. Ensure that the contact lenses are covered completely with saline solution. Perform an eye examination using fluorescein eye drops

A

B

FIGURE 186-9. The chopsticks technique. ***A.*** Frontal view of an adult patient. ***B.*** Side view of a pediatric patient. (Used with permission from reference 9.)

if the patient complains of eye pain after contact lens removal. The procedure may have resulted in a corneal abrasion.

COMPLICATIONS

Any attempt to remove contact lenses with fingernails or other solid objects not approved for the removal of contact lenses can cause corneal abrasions. Their use must be avoided. Proper contact lens removal techniques can result in corneal abrasions. Do not patch corneal abrasions resulting from contact lens removal to prevent an infectious process.[8] The suction cup of the hard contact lens remover may occasionally be inadvertently placed on the cornea. **Do not pull it off the cornea.** Slide it to the lateral corner of the sclera and remove it with a twisting motion.

Never remove a contact lens if there is concern for a potential globe perforation. The techniques described in this chapter are gentle yet put pressure on the globe while removing a contact lens. This pressure on the globe may result in extrusion of the intraocular contents (i.e., lens or vitreous) and cause permanent blindness. Use a suction cup device on a hard contact lens or a rubber disc on a soft contact lens if the contact lens must be removed before the Ophthalmologist arrives.

Additional complications include the inability to remove the lens and damage to the contact lens itself.[8] Consult an Ophthalmologist or Optometrist if a contact lens cannot be removed from the cornea. Desiccated lenses that are not properly rehydrated prior to removal can result in the removal of the corneal epithelium and a corneal abrasion.[8] A properly hydrated lens will slide easily over the surface of the eye. Apply saline drops onto the eye to hydrate the contact lens prior to removal.

SUMMARY

Removal of contact lenses is a procedure that all Emergency Physicians must be able to perform on any patient who is unconscious, has ocular trauma, or cannot remove their own lenses. Failure to perform this simple procedure appropriately can result in serious ocular damage. The removal of a contact lens is easy, quick, simple, and straightforward. Never remove a contact lens if a globe perforation is suspected unless necessary.

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187 Ocular Burn Management and Eye Irrigation

Steven J. Socransky

INTRODUCTION

Ocular burns are true emergencies and represent a up to 10% of ocular trauma cases.[1,2] Chemical burns account for the large majority of ocular burns with thermal burns being the second most common cause.[3] Most victims are young males.[2,4,5] The industrial workplace is the most common setting, although a significant number of cases occur in the home.[4] Assaults are a significant cause of ocular burns in the lower socioeconomic groups of large cities.[4,6]

Caustic agents are primarily responsible for the most severe chemical ocular burns. Most reports indicate that alkali burns are more frequent than acid burns.[1,3,4,7] Examples of more common alkalis and acids are listed in **Table 187-1**.[4,8-15] Ammonia causes the most serious alkali burns.[4] Calcium hydroxide (i.e., lime) is the most common cause of alkali burns.[4] Hydrofluoric acid causes the most serious acid burns.[4] Sulfuric acid is the most common cause of acid burns.[4] Caustic agents account for only a minority of chemical ocular exposures.[1] Most chemical ocular exposures are due to relatively innocuous noncaustic substances (e.g., shampoos, hair sprays, and personal defense sprays) and therefore do not cause significant or lasting damage.[1,16-21]

Ocular irrigation is a simple procedure that is commonly employed in the Emergency Department.[22] It is potentially an eye-saving procedure in the setting of significant chemical ocular burns. Physicians, nurses, and emergency medical personnel who deal with ocular emergencies should be trained in ocular irrigation. First aid personnel in the workplace should be familiar with the use of ocular irrigation.[23] **Ocular irrigation must be employed rapidly.**[1,22,24,25] **Delays in irrigation can limit its effectiveness and increase morbidity.**[3] A treatment algorithm is shown in **Figure 187-1**.

ANATOMY AND PATHOPHYSIOLOGY

The anterior surface of the globe is the major target of toxins in ocular burns (**Figure 187-2**). The eyelids are the most important protective structure for the eye. The orbicularis oculi muscle is innervated by branches of the facial nerve and closes the eyelids in response to noxious stimuli. The cornea provides little in the way of protection from chemical agents. The cornea is composed of five convex and transparent tissue layers. The cornea's major function

TABLE 187-1 Common Caustic Agents and Their Sources

Substance	Class	pH	Source
Ammonium hydroxide	Alkali	11.6	Fertilizers, refrigerants, sparklers
Calcium hydroxide	Alkali	12.4	Mortar, plaster, cement
Magnesium hydroxide	Alkali	10.0	Sparklers, fireworks
Potassium hydroxide	Alkali	11.0	Oven and drain cleaners
Sodium hydroxide	Alkali	14.0	Lye soaps, airbags, EMLA cream, hair straightener
Sodium hypochlorite	Alkali	11.0	Bleaches, drain cleaners
Acetic acid	Acid	2.9	High vinegar concentrations
Chromic acid	Acid	3.0	Chrome plating
Hydrochloric acid	Acid	1.1	Household and pool cleaners
Hydrofluoric acid	Acid	2.1	Rust removers, glass, mineral, gasoline, silicone industries
Sulfuric acid	Acid	1.2	Industrial cleaners, battery acid
Sulfurous acid	Acid	1.5	Bleach, refrigerants

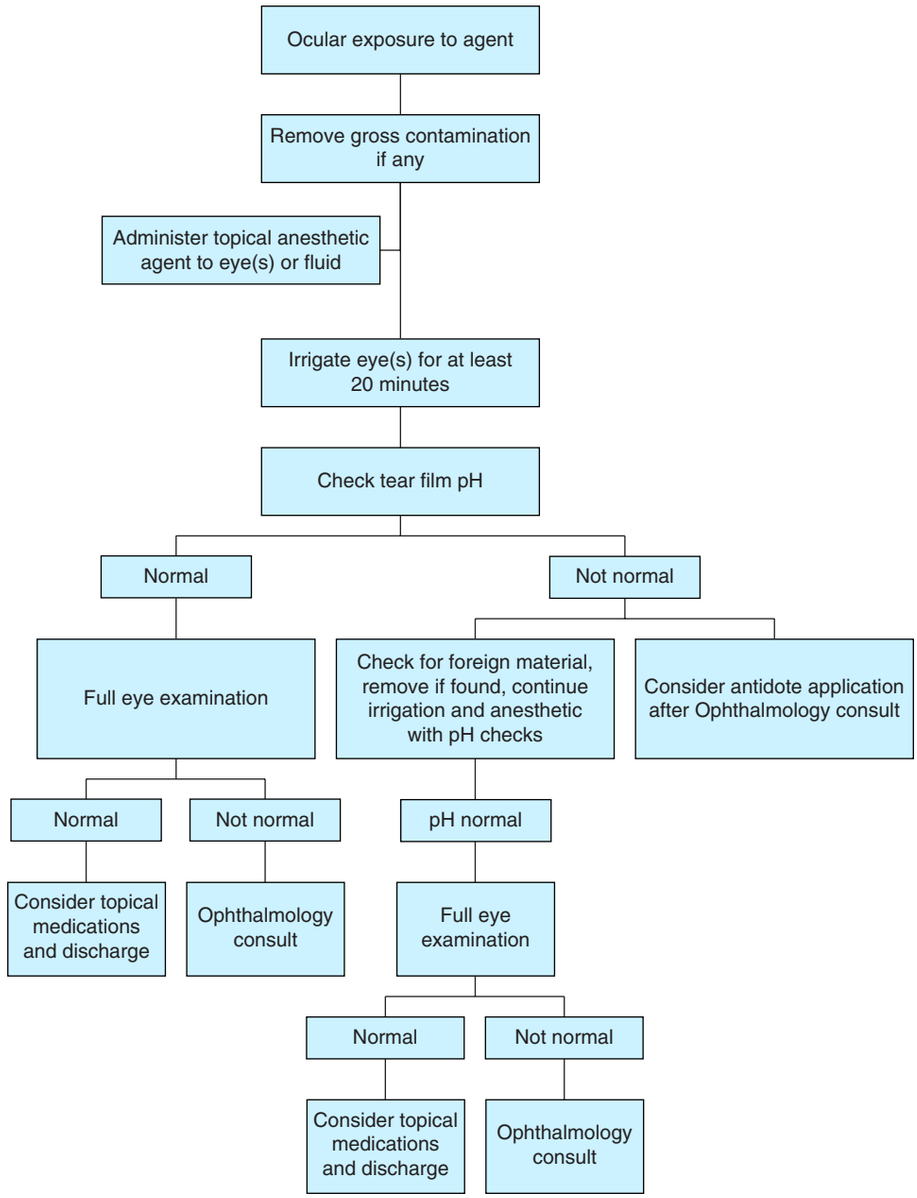


FIGURE 187-1. An algorithm for the Emergency Department management of ocular exposures.

is the refraction and transmittance of light. **The cornea is avascular and exquisitely sensitive to pain.** Extensive burns may be less painful due to destruction of corneal nerve endings and resultant anesthesia.[26] The cornea merges with the sclera to form the limbus at its outer margins. Stem cells at the level of the limbus are responsible for regeneration of the corneal epithelium. The corneal epithelium is unable to regenerate when the limbus is damaged. The fibrous sclera is tougher than the cornea and susceptible to chemical injury.

The sclera is covered by the bulbar conjunctiva which becomes the palpebral conjunctiva as it reflects onto the inner surface of the eyelids. These areas of reflection are referred to as the superior and inferior fornices of the upper and lower eyelids, respectively. The spaces between the eyelids and the globe are referred to as the superior and inferior conjunctival sacs (i.e., palpebral sulci). Posterior to the cornea lies the anterior chamber. It contains the aqueous humor. The anterior and posterior chambers are separated by the iris, ciliary body, trabecular meshwork, and lens.[27]

The damage produced to the eye by toxins depends on several factors: duration of contact; anion or cation concentration; and amount, pH, and inherent toxicity of the chemical.[24,28] **Alkalis generally produce the most damage.** Alkalis release hydroxyl ions that combine with tissue fatty acids and proteins causing liquefaction necrosis.[13] The resultant degradation of corneal tissue allows for easy passage of the chemical into the anterior chamber. This causes a rapid rise (e.g., within a few seconds to a few minutes of contact) in aqueous humor pH and consequent damage to the iris, lens, ciliary body, and other ocular structures.[4,26,29] Damage to these structures and the cornea results in decreased visual acuity, secondary glaucoma, and cataracts. This damage can continue as the anterior chamber pH may remain elevated for hours. **Measurement of tear film pH immediately after irrigation will often yield a normal pH.[9,10] This may be falsely reassuring.** It may either reflect the pH of the irrigant or a transient neutralization of the tear film pH.[26] Alkalis may diffuse from the anterior chamber to the anterior ocular surface several minutes

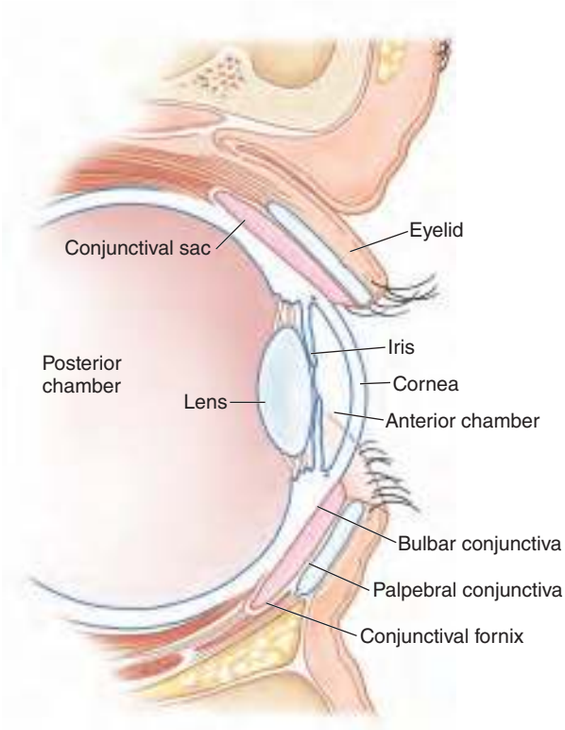


FIGURE 187-2. Cross-sectional anatomy of the eye and surrounding structures.

after irrigation has been discontinued. This causes the tear film pH to rise again.

Acids cause less damage than alkalis. Acids lead to coagulation necrosis and protein precipitation which usually prevents penetration beyond the cornea.[13] Exceptions include acids in higher concentration (e.g., sulfuric acid) and hydrofluoric acid which behave more like an alkali.[4,6,26] These acids can cause damage to deeper structures. Ocular burns caused by weaker acids can progress if treatment is delayed.[26]

Most noncaustic chemical eye exposures involve nontoxic agents that do not penetrate the cornea and cause only mild and self-limited irritation. Notable exceptions include surfactants and high-concentration lacrimators, both of which can cause damage to deeper structures.[12,26] Lacrimators in low concentration (e.g., tear gas) stimulate corneal nerve endings and cause pain and tearing. They do not cause deeper injury.[26] Surfactants can cause significant damage that is often minimally symptomatic.[12] Most solvents can cause sizable superficial corneal defects. Reepithelialization is the rule and deeper tissues are spared.[12,26] Corneal injuries due to capsicum (i.e., pepper spray) usually are self-limited injuries with occasional significant corneal defects requiring Ophthalmology follow-up.[30]

INDICATIONS

The treatment of chemical exposures to the eye is the primary indication for ocular irrigation (**Figure 187-1**). Irrigation should return the patient's vision to the preexposure level in most cases.[18,19,31] In this setting, irrigation has three principal objectives: immediate dilution of the offending agent, removal of the agent, and normalization of anterior chamber pH.[26] Nonembedded foreign bodies that are too small or too numerous to be removed adequately with forceps or a cotton-tipped applicator can usually be removed with irrigation. Foreign bodies that are suspected but cannot be visualized may be removed with irrigation. Certain ocular infections are treated with antibiotics delivered by eye irrigation, although this is not a usual indication in the Emergency Department.

CONTRAINDICATIONS

There are no true contraindications to ocular irrigation. **An irrigating lens (e.g., Morgan Lens) should not be used if a deep corneal injury or a foreign body is suspected.**[32] **This lens may cause the foreign body to further injure the cornea or penetrate the globe. It is preferable to carefully employ the traditional method of irrigation using intravenous tubing and eyelid retraction rather than commercial devices that contact the globe in the setting of an actual or potential globe penetration or rupture.**

EQUIPMENT

- Topical ophthalmic anesthetic agent, 0.5% proparacaine or 0.5% tetracaine
- Towels and a basin to collect fluid runoff
- Bags of crystalloid solution
- Intravenous tubing
- Gauze pads
- Cotton-tipped applicators
- Lid retractors, paper clip or Desmarres retractor
- Commercial irrigation device (e.g., a Morgan Lens)
- Protective eyewear, gloves, and gowns for healthcare personnel

PATIENT PREPARATION

Patients with ocular chemical burns may also have upper respiratory tract injuries, gastrointestinal tract injuries, and facial burns.[1] Nonchemical traumatic injuries are also possible. Airway, breathing, and circulatory problems should take priority. Significant ocular burns will need to be dealt with early and simultaneously with other problems. Irrigation of the periocular skin should be performed initially with ocular irrigation itself so that residual toxin does not enter the eye and cause further chemical injury.

Patients with isolated ocular injuries often have severe pain that requires parenteral narcotics. If this is the case, a monitored setting is appropriate. Intravenous sedatives and analgesics may facilitate ocular irrigation in a patient with severe pain and blepharospasm. Place the patient supine. Place towels and a basin under the patient's head to collect the runoff irrigant solution. Due to the speed with which ocular irrigation must be started, an explanation of the risks and benefits of the irrigation procedure should be offered to the patient while preparing for and initiating irrigation. Healthcare personnel and first-aid workers in the workplace are at risk for injury themselves. Protective equipment is essential and should be easily and rapidly accessible. Contaminated clothes should be removed and bagged in plastic until they can be cleaned or discarded.[17]

TECHNIQUES

A topical ophthalmic anesthetic agent, if immediately available, should first be instilled into the inferior conjunctival sac. Commonly available topical anesthetics include tetracaine and proparacaine. Proparacaine causes less irritation than tetracaine.[33] Each has a duration of action of approximately 10 minutes. Frequent readministration of the topical anesthetic may be necessary every 5 to 10 minutes to ease patient discomfort and facilitate irrigation. An alternative to readministration of local anesthetic would be to add 10 mL of 1% lidocaine to 1 L of crystalloid solution. The addition of lidocaine to the crystalloid solution was compared to an initial

instillation of two drops of tetracaine.[34] Lidocaine added to the irrigant decreased the discomfort of irrigation when the irrigation lasted more than 10 minutes.[34]

OCULAR IRRIGATION

Hang the bag of crystalloid solution at a height of 70 to 200 cm above the patient's head in order to obtain an adequate flow rate.[35,36] The traditional eye irrigation technique involves directing the flow of crystalloid solution over the globe at a wide-open rate (**Figure 187-3**). Hold the end of the intravenous tubing 3 to 5 cm above the patient's eye to avoid blunt injury to the ocular surface. An assistant is usually needed to hold the eyelids open. Dry gauze pads will facilitate one's ability to maintain a grip on the slippery eyelids and keep them open. Direct the flow of crystalloid solution at the entire surface of the globe including into the conjunctival sacs and down to the conjunctival fornices.[26,37] Having the patient look in a direction opposite to where the irrigant is directed helps in this regard.[38] **Although one can point the stream of irrigant directly at the conjunctiva, it is better to direct it across the cornea in order to reduce the potential for further corneal injury.**[32]

Manual retraction of the eyelids with gauze pads does not always allow for adequate irrigation of the conjunctival fornices. Eyelid retractors (e.g., Desmarres retractors or a bent paper clip) must be employed (**Figure 187-4**). **The eye must be well anesthetized when using eyelid retractors and care must be taken to avoid further ocular injury.** Desmarres retractors are preferred to paper clips.

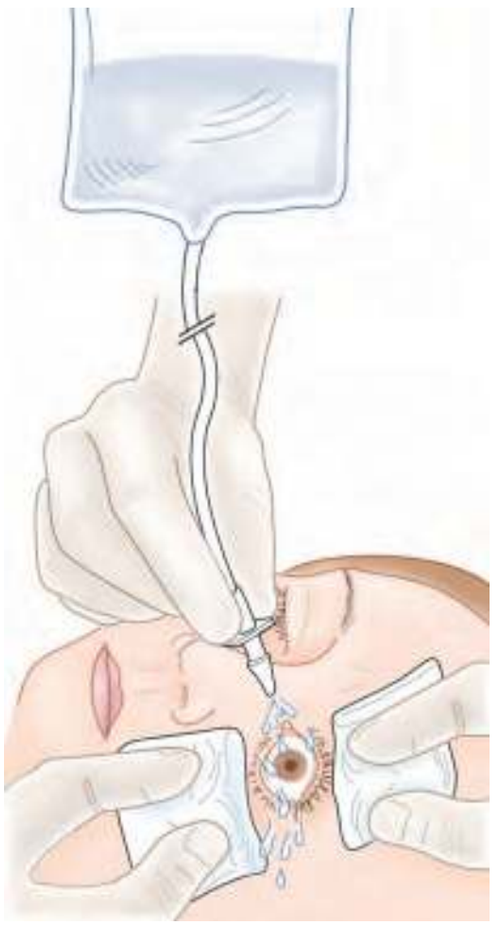
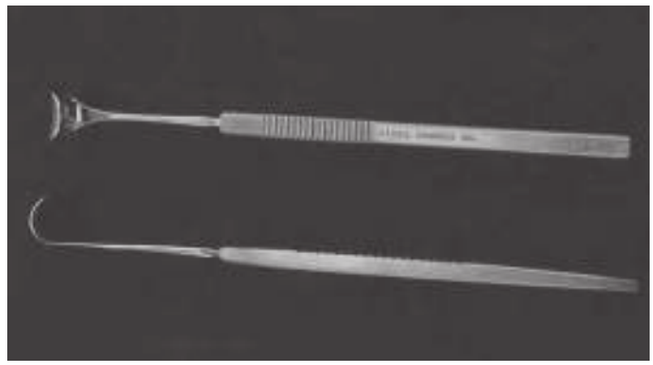


FIGURE 187-3. Standard ocular irrigation setup using intravenous tubing. An assistant retracts the eyelids using gauze pads or eyelid retractors. Complete irrigation of the conjunctival sacs is crucial.



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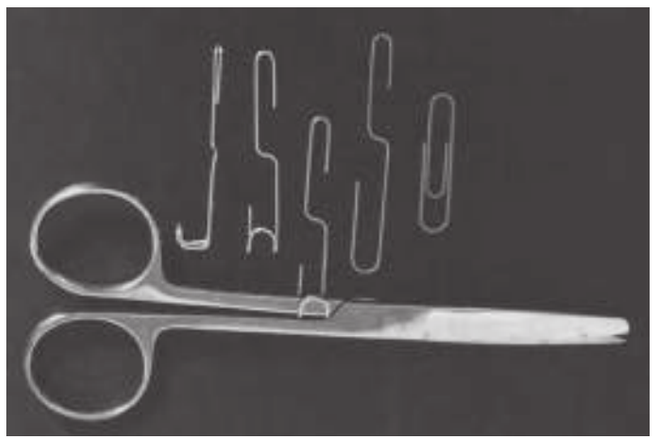


B

FIGURE 187-4. Eyelid retractors. ***A.*** The Desmarres eyelid retractor. ***B.*** An eyelid retractor fashioned from a paper clip.

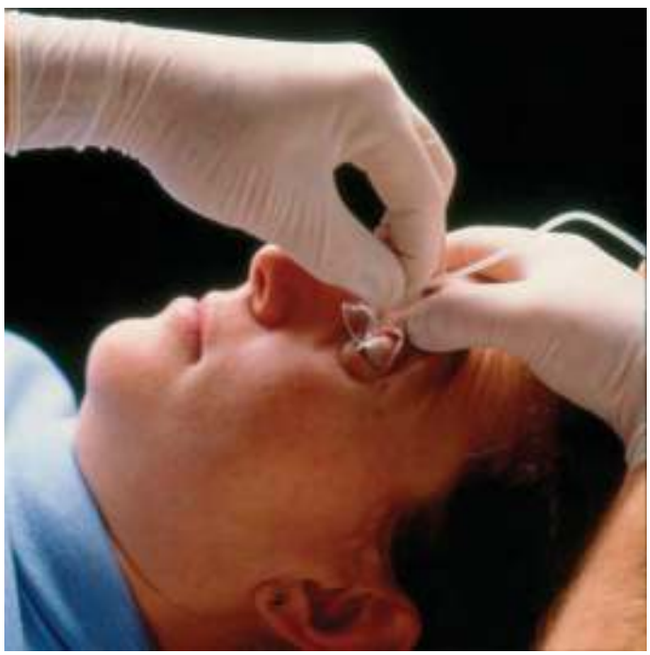
Many paper clips are coated with nickel or silver that can flake off and result in iatrogenic foreign bodies.[39] The use of a Water-Pik or handheld drench hose has been described but offers no definite advantages over intravenous tubing.[40,41]

The Morgan Lens (MorTan Inc., Missoula, MT) is a commercially available device that is less labor intensive and facilitates ocular irrigation. It is a scleral contact lens-type device that is designed to fit over the anterior ocular surface (**Figure 187-5*A***). Connect the proximal end of the device to intravenous tubing and start a minimal flow of irrigant solution through the Morgan Lens. Retract the upper eyelid and ask the patient to look down. Insert the lens under the upper eyelid (**Figure 187-5*B***). Retract the lower eyelid and ask the patient to look upward. Insert the lower part of the lens under the lower eyelid (**Figure 187-5*C***). Increase the flow of the irrigant solution through the lens. Remove the lens by reversing these steps (**Figure 187-5*D***). The company makes the Medi-Duct to collect the irrigant fluid after it flushes the eye (**Fluid 187-5*E***).

The EyeCap (Splash Medical Devices LLC, Atlanta, GA) is a simple device to irrigate an eye (**Figure 187-6**). The unit is quick to set up by just screwing it onto a bottle of sterile saline. The base of the unit has universal threads to attach to a bottle of sterile saline (**Figure 187-6**). The other end is wide and contoured to fit over the orbit. The device allows the saline to deflect off its sidewalls and pool over the eye. The patient can open their eye "under water" to gently allow high-volume eye irrigation. This device cannot be used for foreign bodies embedded in the cornea, for penetrating injuries, or if a ruptured globe is suspected.

Some Emergency Physicians use a nasal cannula for bilateral eye irrigation due to its convenience, low cost, and easy access when

A



B

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FIGURE 187-5. Eye irrigation with the Morgan Lens. ***A.*** The Morgan Lens. ***B.*** Placement of the lens under the upper eyelid. ***C.*** Placement of the lens under the lower eyelid. ***D.*** Removal of the lens. ***E.*** The Medi-Duct fluid collection system. (Photos courtesy of MorTan Inc., Missoula, MT.)

compared to the Morgan Lens. It is more tolerable for some patients than a Morgan Lens. Cut the nasal cannula tubing close to the branch point to eliminate any dead space. The long length of the tubing is not needed. Connect the proximal end (i.e., the spike) to the IV fluid bag. Put the cut end of the nasal cannula tubing into the distal end of the IV tubing. Apply tape to the connection to prevent leaks and to prevent the tubing from coming out of the IV tubing. An alternative is to use a nasogastric tube connector to connect the

FIGURE 187-6. The EyeCap device attached to a bottle of sterile saline. (Photo courtesy of Joseph Schultz, MD and BSN Medical Inc.)

two tubings. Place a basin to catch the used irrigation fluid. Cover the area with absorbent pads (i.e., chux) to keep the patient and bed dry. Place the prongs of the nasal cannula on the bridge of the patient's nose. Tape the nasal cannula in place. Put topical anesthesia in the eyes and IV fluid bag. Open the flow from the IV fluid bag wide open to irrigate the eyes.

SOLID PARTICLES

Ocular burns can be caused by chemicals that are primarily in the solid form (e.g., lime). **Attempt to remove as much solid as possible prior to irrigation using a moistened cotton-tipped applicator while everting and retracting the eyelids. Quickly proceed to irrigation once most of the solid material has been removed or if removal is limited by blepharospasm.** Copious irrigation is often successful in removing any residual solid material.[24] Proceed directly to irrigation when solid material is not suspected in significant quantity. Inspect the conjunctival sac for foreign material once the initial irrigation has been completed.

CONTACT LENSES

Concern that contact lenses may trap chemicals between them and the cornea appears to be unfounded. **Contact lenses may be protective and act as a shield between the toxin and the cornea.[42,43] Irrigation should not be delayed in order to remove a contact lens unless it appears that a contact lens can be removed easily and quickly (Chapter 186).** This is the case even when using commercial irrigation devices. Contact lenses can be removed once an initial period of irrigation has been completed. The contact lenses should be discarded as the toxin may be absorbed by the contact lens only to be released onto the surface of the cornea if reused.[17,42]

IRRIGATION FLUID

The choice of irrigation fluid is much less important than the speed with which irrigation is started.[26,29] Tap water is perfectly acceptable at the scene of the chemical exposure although there may be problems with patient discomfort.[24,25] Normal saline and Ringer's lactate solution are both acceptable during ambulance transport and in the Emergency Department. It has been thought that the more neutral pH of Ringer's lactate solution (pH 6.0 to 7.2) should cause less patient discomfort than normal saline (pH 4.5 to 6.0). Similarly, balanced salt solutions (e.g., BSS Plus, Alcon Laboratories, Fort Worth, TX) should theoretically cause less patient discomfort due to their enhanced buffering capacity, physiologic osmolarity, and physiologic pH. These theories have yet to be clearly substantiated.[16,44,45] **Use the irrigant fluid that is most readily available.[25]** Balanced salt solutions should be used only in patients who require prolonged irrigation and for whom other irrigants are unsuitable due to their expense and time-consuming preparation (e.g., reconstitution in glass bottles).[16,44]

The use of warmed irrigation fluid appears to increase patient comfort.[46] An optimal irrigant temperature is 32.2°C to 37.8°C (90°F to 100°F).[46] It was found that 120 seconds was required to heat 1 L bags of normal saline and Ringer's lactate solution to a temperature of 101°F in a microwave oven set at the highest cooking intensity.[47] **This cannot be routinely recommended as microwave ovens vary and overheated irrigant fluid will cause secondary injury. Irrigation should not be delayed while an irrigant fluid is being warmed despite the potential value of warmed fluid.** Commence irrigation with room temperature crystalloid solution and switch to a warmed crystalloid solution once prepared. Cooler liquids at the beginning of irrigation may help reduce the heat of the reaction and limit chemical injury.[29] The unproven theory that using a cold irrigant may provide cold anesthesia has been suggested.[48]

There is probably no chemical ocular burn for which crystalloid irrigants are contraindicated.[26] Metallic sodium, metallic potassium, and white phosphorus may react violently. Remove any visible solid particles with a cotton-tipped applicator prior to irrigation. Irrigation with copious amounts of crystalloid probably dissipates the heat of the initial reaction more than it initiates a thermochemical reaction.[26]

SPECIFIC ANTIDOTES

The mainstay of treatment for chemical ocular burns is early and copious irrigation. Specific antidotes usually play little role in the treatment of most toxic ocular exposures. There are some instances where antidotes can be helpful once the initial irrigation has been performed. **Consultation with a poison center or Toxicologist should be considered in cases of exposure to unusual toxins.[32]**

EDTA may be helpful in removing adherent calcium hydroxide corneal deposits from lime exposures that cannot be removed with a cotton-tipped applicator or toothless forceps.[1,12,32] A 0.05 M neutral solution of EDTA can be prepared by diluting 20 mL of Endrate disodium (150 mg/mL) with 180 mL of normal saline.[12] A cotton-tipped applicator soaked in the EDTA solution can be used to loosen such deposits. EDTA may also be useful in exposures to potassium permanganate and zinc chloride.[12]

Ascorbic acid may be useful in potassium permanganate exposures. A 5% solution can be prepared by dissolving a 500 mg tablet of ascorbic acid in 25 mL of normal saline. The resultant manganese oxide deposits can be dissolved by dripping the solution into the eyes from a moistened piece of gauze.[49]

Numerous other miscellaneous antidotes can be used for specific exposures. Copper sulfate in a 3% solution can be used to negate the effects of embedded white phosphorus.[12,32] Polyethylene glycol may be useful in phenol exposures.[50] Mineral oil has been suggested in the removal of cyanoacrylates.[32] Calcium gluconate solutions have no beneficial effect in ocular exposures to hydrofluoric acid.[51]

DURATION OF IRRIGATION

Irrigation should be continued for 20 minutes in the home or workplace prior to patient transport.[25] Emergency medical technicians should continue irrigation during ambulance transport until arrival at the Emergency Department.[52] The duration of any further irrigation in the Emergency Department depends on the severity of the exposure and the nature of the toxin.

Minor exposures with nontoxic substances need not undergo copious irrigation. Irrigation with crystalloid solution using a squeeze-type bottle may be sufficient. Irrigation that had been performed either in the home or in the workplace may be all that is needed. However, treatment should proceed as for caustic agents if the chemical nature of the substance is unknown. **Assume that any previous irrigation was inadequate and that further irrigation is necessary in the Emergency Department.[4]**

No definite standard duration for ocular irrigation is available in the literature. Most would agree that patients who are significantly symptomatic or who have received a caustic exposure should have their eyes irrigated with a minimum of 1 to 2 L of crystalloid solution over 20 to 30 minutes.[24,53] Exposures to noncaustic agents, milder acid burns, and very mild alkali burns will usually not need further irrigation. Moderate to severe acid burns and anything more than a mild alkali burn will likely require more prolonged irrigation.

The duration of irrigation for caustic exposures is in part governed by pH measurement. After the initial irrigation, measure the pH of the inferior conjunctival sac using wide-range pH paper (i.e., accurate in the pH range of at least 2 to 10) or litmus paper.[12] **Litmus paper with a narrower range and urine dipsticks may not be adequate. If the pH is abnormal, continue irrigation and recheck the pH at 10 to 15 minute intervals until it normalizes.[12,32]** If the pH has reached the near-normal range (i.e., 7 to 8), discontinue irrigation and recheck the pH in 10 to 30 minutes to ensure that it continues to remain normal.[26,32] **In clinically severe caustic burns, regardless of the ocular pH, irrigation should be continued until the patient is evaluated by an Ophthalmologist. Alkali burns are more likely to require prolonged irrigation than acid burns.** Several hours of irrigation may be required for severe alkali burns. The Ophthalmologist may opt to perform a regional nerve block to incapacitate the orbicularis oculi muscle to limit blepharospasm and improve patient comfort.[13]

ASSESSMENT

Ocular assessment should be limited to observation of gross injury, assessment of ocular pH, and quick verification of visual acuity until irrigation is complete. These assessments should not be prolonged and should not delay initial irrigation. A full ophthalmologic assessment is required once irrigation has been discontinued. This should include a recheck of visual acuity, measurement of intraocular pressures, and a slit lamp examination to evaluate for corneal injury, uveitis, and globe perforation.

AFTERCARE

The extent of aftercare required is dependent upon the severity of the injury.[7,19,22,54,55] Minor exposures to innocuous agents and very mild caustic exposures without corneal changes should be reevaluated in 24 hours if the patient is asymptomatic. Some exposures (e.g., gases, vapors, and fumes) can result in delayed evidence of corneal injury on slit lamp and fluorescein exams.[12] Patients with apparently minor exposures should be cautioned to return to the Emergency Department if their symptoms worsen or do not improve.

Patients with mild exposures that result in corneal defects should be treated with topical antibiotics that have antistaphylococcal and antipseudomonal activity.[12] Ascertain the patient's tetanus immune status and administer prophylaxis as indicated. Analgesics or an eye patch (Chapter 193) may be offered if patient discomfort is significant.[1,12] Cycloplegics should be prescribed in order to decrease the pain resulting from ciliary spasm and to decrease the risk of formation of posterior synechiae if anterior chamber involvement is suspected.[12,37] A patient chronically using phosphate buffer–containing eye drops (e.g., timolol or latanoprost) is at greater risk of corneal calcification after a chemical burn.[56] Telephone consultation with an Ophthalmologist is recommended. Ophthalmology follow-up should occur within 24 hours.

Moderate to severe ocular burns require admission and acute evaluation by an Ophthalmologist. Medical treatment of secondary glaucoma may be required. Anterior chamber paracentesis (Chapter 190) and lavage may be needed early in the course of severe alkali burns to decrease the anterior chamber pH and intraocular pressure.[1,12,32,50] Necrotic tissue will have to be debrided.[4] The goals of longer-term therapy include the prevention of corneal ulceration, prevention of scarring of the anterior ocular structures, prevention of globe perforation, and promotion of corneal reepithelialization.[32] Steroids, nonsteroidal anti-inflammatory agents, frequent lubrication, and soft contact lenses may help.[1,4] Ascorbic acid and collagenase inhibitors are experimental. More severe injuries require surgical intervention due to the loss of stem cells at the limbus and thus the loss of potential corneal reepithelialization.[4]

COMPLICATIONS

Pain and discomfort are common after ocular exposure to chemicals.[55] These can be minimized with topical ophthalmic anesthetic agents, parenteral sedatives, and parenteral narcotics in the Emergency Department. Corneal injury is usually the result of the primary chemical injury. Corneal injury may result from the irrigant or irrigating device, particularly if improper technique is used. Frequent use of topical ophthalmic anesthetic agents can lead to corneal injury. Extrusion of ocular contents is possible in the setting of a globe penetration. The diving reflex is a rare but possibly significant complication. This reflex is mediated by the ophthalmic branch of the trigeminal nerve and the vagus nerve. It is triggered by a cold water stimulus to the face and results in bradycardia without hypotension. The diving reflex is more common in infants and children. Its clinical importance is greatest in patients with significant comorbid disease. Continuous cardiac monitoring is wise in these patients. Irrigation with warm water may limit this reflex.[57,58]

SUMMARY

Ocular irrigation is an eye-saving procedure in the setting of significant ocular burns. It is an easy and safe procedure to perform. First-aid workers, emergency medical technicians, and Emergency Department personnel should be trained in its use. Ocular irrigation should be performed rapidly with the most available nontoxic irrigant as delays of even seconds can limit its effectiveness.

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188 Intraocular Pressure Measurement (Tonometry)

Rosalia Mbugua and Adam Jennings

INTRODUCTION

This chapter is designed to assist the Emergency Physician in the decision of when it is necessary to measure intraocular pressure (IOP) and will review several reliable methods of tonometry. There are multiple traumatic, pathologic, and postsurgical causes of changes in IOP. **The clinical signs and symptoms of elevated IOP are similar regardless of the etiology.** Digital palpation is the oldest and simplest form of tonometry and remains useful in select situations. Schiøtz indentation tonometry is discussed for historical purposes and is an accurate method to measure IOP. The nonportable Goldmann or applanation tonometer serves as the standard for measuring accurate IOP. It requires the use of a slit lamp and can be difficult to master. The hand-held Perkins and Kowa tonometers are based on the same principle as the Goldmann and require

experience to use effectively. The electronic Tono-Pen is best known to most Emergency Physicians and is discussed at length. The literature describes the measurement of anterior chamber depth with bedside ultrasound to measure IOP. This may be useful in patients with significant facial trauma and are unable to open their eyes. The Emergency Physician should be comfortable with one or more of these techniques. **The early detection of abnormal IOP can prevent irreversible vision loss.**

ANATOMY AND PATHOPHYSIOLOGY

Aqueous humor is produced by the ciliary body in the posterior chamber of the eye directly behind the iris (**Figure 188-1**). Most of the aqueous humor flows forward through the pupil and into the anterior chamber. It drains out of the eye through the trabecular meshwork located at the angle where the cornea and iris meet. This is the area referred to in open angle, narrow angle, and angle-closure glaucoma. Aqueous humor production is equivalent to outflow in healthy eyes. IOP reflects the pressure of the ocular contents and by convention is expressed in millimeters of mercury or mmHg.[1] **The mean IOP in the general population is 16 mmHg with a standard deviation of 3 mmHg.[2] Normal pressure ranges from 10 to 22 mmHg.**

Aqueous humor production and outflow can be dramatically affected by disease or injury of the eye. Even small changes in IOP over long time periods can be vision threatening. **Significant increases in the IOP can cause rapid and irreversible damage to vision in just a few hours.** Nontraumatic conditions that result in an elevation of IOP include primary angle-closure glaucoma and secondary angle-closure glaucoma. Traumatic conditions associated with elevated IOP include retrobulbar hemorrhage, hyphema, and traumatic iritis. Conditions associated with low IOP that threaten vision include penetrating trauma and postsurgical complications.

Patients with primary or secondary acute angle-closure glaucoma often present with ocular pain and decreased vision in one eye. They may describe a headache in the brow region, with or without associated nausea and vomiting. External examination frequently reveals that the conjunctiva is erythematous, the cornea appears milky or hazy, and the pupil is slightly dilated with a sluggish response to light (**Figure 188-2**).

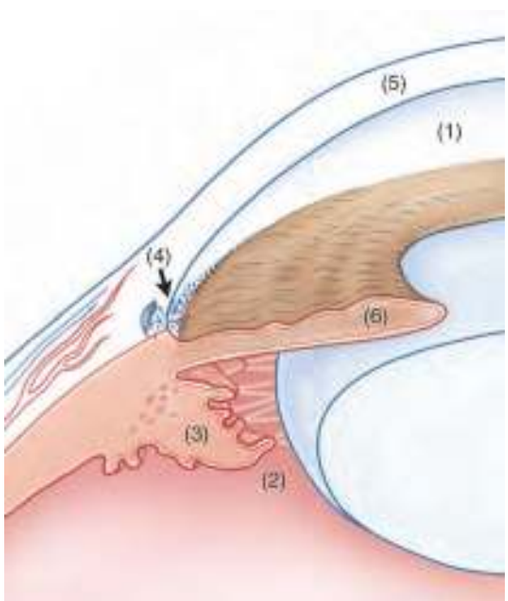


FIGURE 188-1. Anatomy of the anterior segment of the eye: (*1*) anterior segment; (*2*) posterior segment; (*3*) ciliary body; (*4*) trabecular meshwork; (*5*) cornea; (*6*) iris.

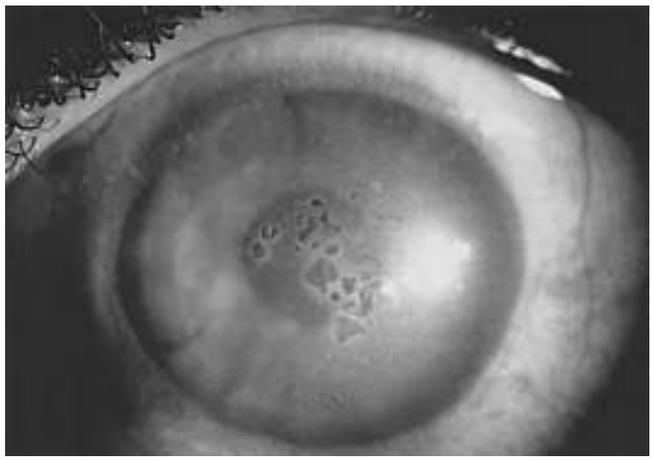


FIGURE 188-2. High magnification of an eye with angle-closure glaucoma and a markedly elevated IOP. Note the corneal edema and the mid-dilated pupil.

Traumatic retrobulbar hemorrhage (Chapter 194) can result in markedly elevated IOP. Patients will present with a painful proptosis and fullness of the periorbital tissues. There is usually restricted movement of the eye in one or more fields of gaze. Acute onset of retrobulbar inflammation will present the same way without the history of trauma. A spontaneous nontraumatic retrobulbar hemorrhage can occur in patients with a coagulopathy.

Traumatic iritis may result in an elevated or a reduced IOP. Traumatic iritis presents following blunt, nonpenetrating trauma to the eye. Inflammatory cells circulating in the anterior chamber may impede aqueous outflow resulting in high IOP. Iritis may affect the ciliary body causing a decrease in the production of aqueous humor and a lower IOP. These patients present with eye pain and photophobia. They usually describe their vision as slightly blurred and give a history of trauma within the past 48 hours. The history of recent trauma and light sensitivity is important given that the external examination of the eye may appear normal. The cornea will be clear. **A slit lamp examination is usually necessary to identify the inflammatory cells in the anterior chamber.**

A hyphema is visible blood that layers out in the anterior chamber between the iris and the cornea (**Figure 188-3**). A microhyphema is diffuse blood circulating in the anterior chamber. These patients present with decreased vision, photophobia, and blood in the anterior chamber. A common cause of blood in the anterior chamber is

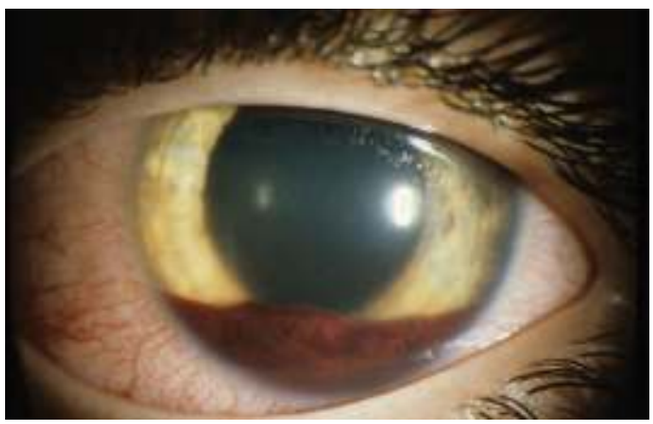


FIGURE 188-3. A traumatic hyphema with 10% to 15% layered blood in the anterior chamber.

blunt nonpenetrating trauma to the eye. A patient with blood in the anterior chamber and a history of sickle cell disease or trait is at a higher risk of elevated IOP.[3] Consult an Ophthalmologist if a sickle cell patient presents with blood in the anterior chamber.

INDICATIONS

Patients presenting with a painful eye, red eye, decreased vision, and no history of ocular trauma should have their IOP measured and documented. Do not measure IOP if there is a history of blunt trauma associated with these symptoms. Some of the common presentations that require documentation of the IOP are reviewed in the previous section. This includes confirming the clinical diagnosis of acute angle-closure glaucoma, determining IOP after blunt ocular trauma, and determining IOP in a patient with iritis.

CONTRAINDICATIONS

It is essential to rule out a ruptured globe before tonometry is performed when a patient relays a history of trauma. Tonometry should be strictly avoided if there is evidence of a ruptured globe or the suspicion of a ruptured globe. A ruptured globe from penetrating trauma to the anterior segment of the eye, including corneal or scleral lacerations, is usually obvious upon clinical examination. **Blunt trauma resulting in a ruptured globe may be very difficult to see on examination. Any blunt force (e.g., closed fist) causing an anteroposterior compression of the globe can result in a scleral rupture posterior to the attachment of the extraocular muscles. This is rarely obvious on external examination or a nondilated slit lamp examination.** The clue is that the patient has dramatically reduced vision and prominent swelling of the periorbital tissues.

Viral conjunctivitis is one of the most common ocular conditions seen in the Emergency Department. Conjunctivitis is so highly infectious that it is a relative contraindication to performing tonometry. These patients may present with ocular pain and decreased vision just like patients with angle-closure glaucoma. Conjunctivitis symptoms are usually binocular and the pain is not as severe. Acute angle-closure glaucoma almost always presents in one eye. It is reasonable to proceed with tonometry to rule out increased IOP as an etiology of a painful red eye if the diagnosis of conjunctivitis is uncertain. **Clean the instrumentation carefully to prevent spreading the infection to the contralateral eye.** The instrument of choice in cases of a suspected infection is a hand-held unit that uses disposable and single-use latex covers for the instrument.

EQUIPMENT

TOPICAL OPHTHALMIC ANESTHETIC AGENTS

- Proparacaine hydrochloride 0.5% (e.g., Alcaine, Ocu-Caine, Ophthaine, Ophthetic)
- Tetracaine hydrochloride 0.5% (e.g., AK-T-Caine, Altacaine, Opticaine, Protocaine)

TOPICAL STAIN FOR TEAR FILM

- Fluorescein sodium 1 mg strips (e.g., Fluorets, Fluor-I-Strip-AT, Ful-Glo)
- Fluorescein ophthalmic solution (e.g., Fluress, Fluorescein Sodium)

TONOMETERS

- Goldmann applanation tonometer (**Figure 188-4**)
- Hand-held applanation tonometer (**Figures 188-5, 188-6, and 188-7**)

FIGURE 188-4. The Goldmann applanation tonometer.

- Schiøtz indentation tonometer (**Figures 188-8 and 188-9**)

ANTISEPTIC

- 70% isopropyl alcohol solution and swabs
- 3% hydrogen peroxide solution

A B

FIGURE 188-5. The Icare hand-held tonometers. ***A.*** The TAO1i model. ***B.*** The il100 model. (Photos courtesy of Icare, Raleigh, NC.)

FIGURE 188-6. The Accutome hand-held tonometers. ***A.*** The AccuPen model. ***B.*** The AccuPen Silo model. (Photos courtesy of Accutome Inc., Malvern, PA.)

FIGURE 188-7. The "tonopen" hand-held tonometers. ***A.*** The Medtronic Tono-Pen XL model. (Photo courtesy of Medtronic, Minneapolis, MN.) ***B.*** The Reichert Tono-Pen model. (Photo courtesy of Reichert Technologies, Depew, NY.) ***C.*** The Reichert Tono-Pen Avia model. (Photo courtesy of Reichert Technologies, Depew, NY.)

PATIENT PREPARATION

Inform the patient of the need to measure the pressure in their eye. Explain that an instrument will contact their tear film directly over the cornea. Reassure the patient that tonometry is not painful. It is not necessary to have the patient sign a consent form as tonometry is part of a routine eye examination. One may choose to document the discussion with the patient for completeness.

Remove all contact lenses before the instillation of any topical ocular anesthetic agent or fluorescein. Measuring IOP through a contact lens is unreliable. The fluorescein dye will permanently stain contact lenses and clothing.

Instill one drop of a topical ophthalmic anesthetic agent in both of the patient's eyes. The contralateral eye is used as the control even if only one eye is of concern. Topical ophthalmic anesthetic agents begin working within 30 seconds and remain effective for 10 to 20 minutes. The drops may burn when applied and may cause a transient drying of the cornea. **Instruct the patient not to touch or rub their eyes until the anesthesia wears off.**

Instill fluorescein when using any prism applanation tip such as in Goldmann, Perkins, or Kowa tonometers. Fluorescein is not needed when using the Tono-Pen or the Schiøtz tonometer. Fluorescein is available on strips or in solution. Fluorescein strips are applied to the tear film on the inner layer of each lower eyelid. Dampen the

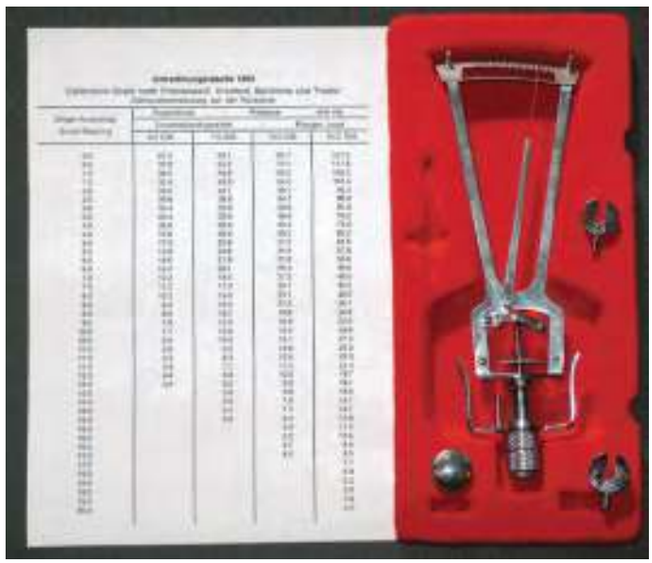


FIGURE 188-8. The contents of the Schiøtz tonometer case.

strip with a drop of topical anesthetic before application if the eye is extremely dry. The alternative is to instill a drop of fluorescein solution in the cul-de-sac of each lower eyelid.

All plastic prism applanation tonometer heads and the metal plunger of the Schiøtz need to be sterilized to prevent cross-contamination. Do not use sterilization solution on electronic tonometer tips. Use a prepackaged 70% alcohol swab or gauze soaked in 70% alcohol to sterilize the tip of a tonometer. Apply the alcohol to the tip of the prism for 10 seconds and allow it to air dry. Gauze soaked in 3% hydrogen peroxide may also be used. Rinse the tip with sterile saline to wash off the 3% hydrogen peroxide before application to prevent iatrogenic corneal abrasions. These described methods effectively disinfect for common bacteria and viruses, adenovirus, herpes simplex, and human immunodeficiency virus (HIV).[4,5] Replace the disposable cover over the tip of the tonometer after each use.

TECHNIQUES

DIGITAL PALPATION

Digital palpation is not as accurate as applanation tonometry. Digital palpation is an informal means of judging IOP. One can use the built-in comparison with the contralateral eye. Studies examining this technique have shown that inexperienced examiners can improve their accuracy with a short training session to within 5 mmHg of the actual IOP 88% of the time.[6] **Digital palpation should not be attempted if there is any potential for a ruptured globe.**

Place the patient supine or in a reclined position. Ask the patient to close their eyes and look straight ahead through the closed eyelids. Stand next to the patient's torso and face them. Place the right thumb over the patient's left eye and left thumb over the patient's right eye. Place the remaining fingers of both hands on the patient's temples for stability. Apply gentle alternating pressure to each globe. The Emergency Physician should be able to ascertain any difference in pressure between the two eyes. The Emergency Physician may also gently palpate their own globe as a normal control. This qualitative measurement is often sufficient to determine a significantly elevated IOP.

SCHIØTZ TONOMETRY

The Schiøtz tonometer estimates IOP by measuring the indentation of the globe caused by a known weight.[7] The Schiøtz tonometer is a sturdy, low-maintenance, and affordable instrument. It is gravity dependent and requires the patient to be supine or have their head fully extended to get an accurate reading. The Schiøtz tonometer case contains the Schiøtz tonometer, a calibration scale, weights, and a calibration platform (**Figure 188-8**).

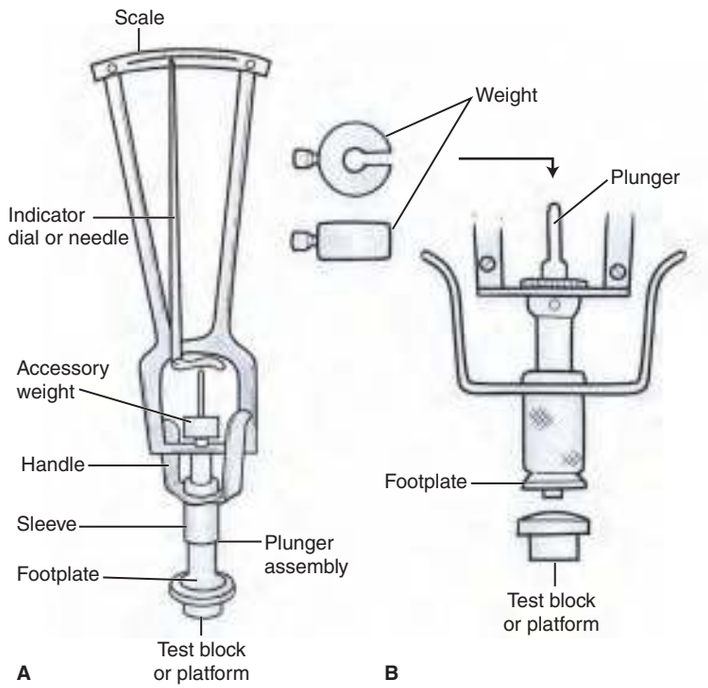


FIGURE 188-9. The Schiøtz tonometer. ***A.*** Schematic of the tonometer. ***B.*** Schematic of the plunger assembly.

The tonometer consists of a plunger and a hammer device connected to a needle (**Figure 188-9**). The needle is calibrated to a scale with each unit measuring 0.05 mm on the scale. The plunger, the hammer, and the needle weigh 5.5 gm. This can be increased to 7.5 gm, 10 gm, or 15 gm by adding known weights to the tonometer. The weighted plunger is heavy and has a large contact area that causes significant indentation of the cornea with each measurement. **The more weight it takes to move the needle on the scale or indent the cornea, the higher is the IOP.** The scale reading is converted to IOP in mmHg by a conversion chart supplied with each Schiøtz tonometer (**Figure 188-8 and Table 188-1**).

Place the patient supine or sitting with their head fully extended. Ensure that the patient is comfortable. A tight collar, flexed neck, breath-holding, squeezed eyelids, looking toward the nose, or accommodation results in a falsely elevated reading. Place two drops of a topical ophthalmic anesthetic agent onto each eye.

TABLE 188-1 The Schiøtz Tonometer Scale*

Tonometer reading	5.5 gm load	7.5 gm load	10 gm load	15 gm load
0.0	41.5	59.1	81.7	127.5
0.5	37.8	54.2	75.1	117.9
1.0	34.5	49.8	69.3	109.3
1.5	31.6	45.8	64.0	101.4
2.0	29.0	42.1	59.1	94.3
2.5	26.6	38.8	54.7	88.0
3.0	24.4	35.8	50.6	81.8
3.5	22.4	33.0	46.9	76.2
4.0	20.6	30.4	43.4	71.0
4.5	18.9	28.0	40.2	66.2
5.0	17.3	25.8	37.2	61.8
5.5	15.9	23.8	34.4	57.6
6.0	14.6	21.9	31.8	53.6
6.5	13.4	20.1	29.4	49.9
7.0	12.2	18.5	27.2	46.5
7.5	11.2	17.0	25.1	43.2
8.0	10.2	15.6	23.1	40.2
8.5	9.4	14.3	21.3	38.1
9.0	8.5	13.1	19.6	34.6
9.5	7.8	12.0	18.0	32.0
10.0	7.1	10.9	16.5	29.6
10.5	6.5	10.9	15.1	27.4
11.0	5.9	9.0	13.8	25.3
11.5	5.3	8.3	12.6	23.3
12.0	4.9	7.5	11.5	21.4
12.5	4.4	6.8	10.5	19.7
13.0	4.0	6.2	9.5	18.1
13.5		5.6	8.6	16.5
14.0		5.0	7.8	15.1
14.5		4.5	7.1	13.7
15.0		4.0	6.4	12.6
15.5			5.8	11.4
16.0			5.2	10.4
16.5			4.7	9.4
17.0			4.2	8.5
17.5				7.7
18.0				6.9
18.5				6.2
19.0				5.6
19.5				4.9
20.0				4.5

*Use the tonometer scale reading and the weight applied to the plunger (gram load) to determine the IOP reading in mmHg.

Calibrate the tonometer by placing the footplate of the plunger on the platform provided in the case (**Figures 188-8 and 188-9**). **The scale reading must be "0" while the footplate is on the platform. The instrument requires repair if it does not read zero while on the platform.**

Instruct the patient to look at a fixation target directly overhead and to open both eyes as wide as possible. Spread the eyelids with the nondominant index finger and thumb if necessary. Grasp the Schiøtz tonometer by its handle. **Ensure that the fingers holding the eyelids open are pushing on the orbital rim and not the globe falsely elevating the IOP.** Place the footplate of the Schiøtz tonometer directly over the pupil and gently lower it onto the tear film (**Figure 188-10**). Note the reading on the scale. Add more weight if the scale reading is less than 3 units. **Do not push down on the cornea with the Schiøtz tonometer. This will cause a false elevation in the IOP reading.** Repeat the measurement several times or until three readings are within 0.5 scale units. Convert the scale reading to IOP with the conversion chart (**Table 188-1**).[8]

Clean the tonometer immediately after each use.[9] Clean the barrel with two pipe cleaners, the first soaked in 70% isopropyl alcohol and the second one dry. Clean the plunger with an alcohol swab.

FIGURE 188-10. The Schiøtz tonometer positioned on the cornea.

Allow the instrument to air dry 1 to 2 minutes before being used in the contralateral eye so that the alcohol evaporates and is not transferred to the corneal surface.[10]

GOLDMANN (APPLANATION) TONOMETRY

The Goldmann tonometer is most used by Ophthalmologists and considered the clinical standard for measuring IOP (**Figure 188-4**).[11,12] Many use the "old fashioned" dial-type Goldmann tonometer. Some are using the newer digital readout Goldmann tonometer. This method of tonometry is based on the Imbert-Fick principle.[13] It states that the pressure inside an ideal dry, thin-walled sphere equals the force necessary to flatten its surface divided by the area of the flattening ($P = F/A$; where P = pressure, F = force, and A = area). IOP is proportional to the pressure applied to the eye and the thickness of its walls. The Goldmann tonometer determines the force necessary to flatten or applanate an area of the cornea 3.06 mm in diameter. The degree of flattening is determined while viewing the cornea through a split prism device in the tonometer head and represented by the two semicircles. Fluorescein is used to stain the tear film to distinguish the cornea from the tear film. The eye is viewed through the cobalt blue filter causing the fluorescein stained tear film to appear yellow-green. Inadequate fluorescein stain can affect the tonometer reading. The technique is described below.[5]

The Goldmann tonometer is not portable. It requires the patient to be cooperative, the patient to sit upright, and a working knowledge of the slit lamp. The Goldmann tonometer in combination with a slit lamp examination provides the clinician with the most detailed information about the patient's ocular condition.

Place two drops of a topical ophthalmic anesthetic agent onto each eye. Place fluorescein dye in the eye. Position the tonometer head and cobalt filter on the slit lamp. Set the tension knob to 10 mmHg. It is more accurate to measure IOP by increasing rather than decreasing the force of applanation. Seat the patient in front of the slit lamp. Support the patient's chin in the chin rest and place their forehead firmly against the strap. Instruct the patient to look straight ahead and open both eyes as wide as possible. Spread their eyelids with a thumb and forefinger if necessary. **Do not put pressure on the globe while holding the eyelids open.**

Move the tonometer head within one-half inch of the cornea (**Figure 188-11**). Use the control stick on the slit lamp to center the applanation head directly over the pupil. Look for two semicircles on the ocular surface as the tonometer head is advanced onto the corneal tear film (**Figure 188-12**). Ensure that the tension knob is set at 10 mmHg. The semicircles will not be touching if the patient's pressure is above 10 mmHg (**Figure 188-12*B***). Turn the tension knob clockwise to raise the reading and counterclockwise to lower the reading. Turn the tension knob up until the inside borders of the fluorescein rings are touching (**Figure 188-12*A***). Read the IOP on the tension knob. The semicircles will overlap if the pressure is below 10 mmHg (**Figure 188-12*C***). Turn the tension knob down until the inside borders of the fluorescein rings are touching (**Figure 188-12*A***). Read the IOP on the tension knob.

Clean the tonometer head with an alcohol swab immediately after use. Let it air dry 1 to 2 minutes so that the alcohol evaporates and is not transferred to the corneal surface. Repeat the procedure on the contralateral eye.

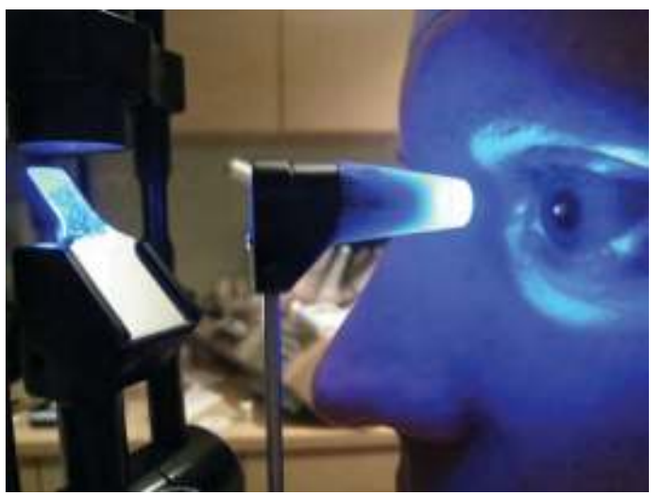


FIGURE 188-11. A patient positioned in the slit lamp with a Goldmann tonometer in position to be advanced onto the cornea.

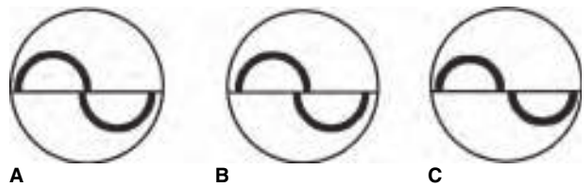


FIGURE 188-12. Semicircles as seen through the Goldmann tonometer. ***A.*** The inner edges of the semicircles are touching signifying the correct IOP reading. ***B.*** Semicircles are not touching because the pressure reading on the tension knob is too low. ***C.*** Semicircles are overlapping because the pressure reading is too high.

HAND-HELD TONOMETRY

The Tono-Pen XL and the Tono-Pen Avia (**Figures 188-7*B* and 188-7*C***) are lightweight, are simple to operate, and can record IOP with the patient in any position.[10] The small contact area is useful in patients with eyelid swelling and corneal surface irregularities. The Tono-Pen calculates IOP using a strain gauge that creates an electronic signal as the 1.5 mm footplate flattens the cornea.[10] A single microprocessor chip analyzes each application of the footplate and averages 4 to 10 valid measurements to form one reading. The instrument should be stored in its case when not in use. An Ocu-Film cover should remain over the tip when it is stored and changed with each new patient. **The Ocu-Film covers are made of latex. Ask the patient about latex allergies prior to using this device. The tip of the Tono-Pen is very sensitive, is easily damaged, and should never be touched.**

The Tono-Pen XL unit is internally calibrated. The instrument calibration should be checked before the first use each day or in the event of unanticipated readings. Calibration should also be performed whenever it is indicated by the LCD display, when batteries are replaced, or after unsuccessful calibration. **It needs to be calibrated if the word "bAd" appears in the LCD window.** The Tono-Pen is not used or calibrated daily in the Emergency Department. **It is necessary to check the calibration prior to each use.**

The display of "Good" in the LCD window indicates that the Tono-Pen is functioning properly. Press the operation button once. The Tono-Pen will display [8.8.8.8] in the LCD window, followed by a single row of dashes [----], and then a double row of dashes [= = = =]. This indicates that the Tono-Pen is ready to measure IOP.

To calibrate the Tono-Pen, aim the transducer end straight down. Depress the operation button twice within 1.5 seconds. The Tono-Pen will beep and display "CAL" in the LCD window. Wait for the Tono-Pen to beep and display "UP" in the LCD window. This can take up to 15 seconds. Immediately and quickly invert the Tono-Pen so that the transducer end is pointing straight up. The Tono-Pen

FIGURE 188-13. The Tono-Pen positioned in front of the cornea.

will beep and display "Good" in the LCD window if it is functioning properly. Repeat the calibration procedure if "bAd" is displayed in the LCD window.

Place the patient in a seated or supine position. Instruct the patient to look straight ahead. Place two drops of a topical ophthalmic anesthetic agent onto each eye. Fluorescein is not necessary when using the Tono-Pen. Grasp the Tono-Pen like a pencil and hold it perpendicular to the corneal surface (**Figure 188-13**). Gently contact the cornea directly over the pupil or central cornea. **Contact the cornea in a series of light taps. The Tono-Pen should not indent the cornea if used properly, reducing the risk if there is an unidentified ruptured globe or a hyphema.** The Tono-Pen chirps each time a valid IOP measurement is obtained. The microprocessor sounds a final beep after it receives four valid readings. The mean IOP will be displayed in the LCD window. A single row of dashes [----] indicates that an insufficient number of valid readings were collected. Obtain additional measurements after pressing the operation button.

REBOUND TONOMETRY

The unit(s) used in the Emergency Department are the Icare devices (**Figures 188-5*A* and 188-5*B***).[14,15] The Icare tonometers (Tiolat Oy, Helsinki, Finland) use a smaller zone to indent the cornea, do not require fluorescein, and do not require topical anesthesia.[12] There are numerous other advantages including no risk in using latex, no device calibration, preferred by patients, quick IOP measurement, and comparable to application tonometry. The Icare tonometer is useful for the Emergency Department. It uses disposable and single-use probes. The tip of the probe is rounded to minimize damage to the cornea. The lightweight probe is propelled toward the cornea, bounces back off the cornea and to the device, and the unit detects deceleration. The movement of the probe induces a voltage change that is converted to a magnetic field that is detected by the device. There is also a device for home use by the patient.[16,17] The home device records the IOP measurement, date taken, and time taken. The information can be uploaded to a computer and sent to the Ophthalmologist. The units are reliable, easy to use, and provide frequent IOP measurements during daily activities. The devices have been used in young children.[18]

ALTERNATIVE DEVICES

There are many additional hand-held tonometers of varying cost, ease of use, and availability. The Perkins MK-2 (Haag-Streit, Essex, United Kingdom) and the Kowa HA-2 (Kowa Optimed, Inc., Torrance, CA) are two hand-held tonometers using the Goldmann prism technique (i.e., aligning the semicircular images). They are portable but require a familiarity with Goldmann applanation. The Accupen (Accutome, Malvern, PA) is like the Tono-Pen with a few differences. These include ease of use and longer battery life. In addition, it does not require calibration before every use. The Diaton transpalpebral hand-held tonometer (Bicom Inc., Long Beach, NY) is designed to measure IOP through a patient's closed eyelids.[11] Another transpalpebral tonometer is the phosphene tonometer, the Proview Eye Pressure Monitor (Bausch & Lomb, New York, NY). Both transpalpebral tonometers can be used at home by patients. Unfortunately, both transpalpebral units do not correlate with applanation tonometry and have limited clinical use. The Pulsair IntilliPuff (Keeler, Brownhill, PA) is a the only portable, hand-held, noncontact tonometer (**Figure 188-14**). This unit may be valuable to measure the IOP if a globe rupture is suspected.[19]

FIGURE 188-14. The Pulsair IntilliPuff. (Photo courtesy of Keeler, Brownhill, PA.)

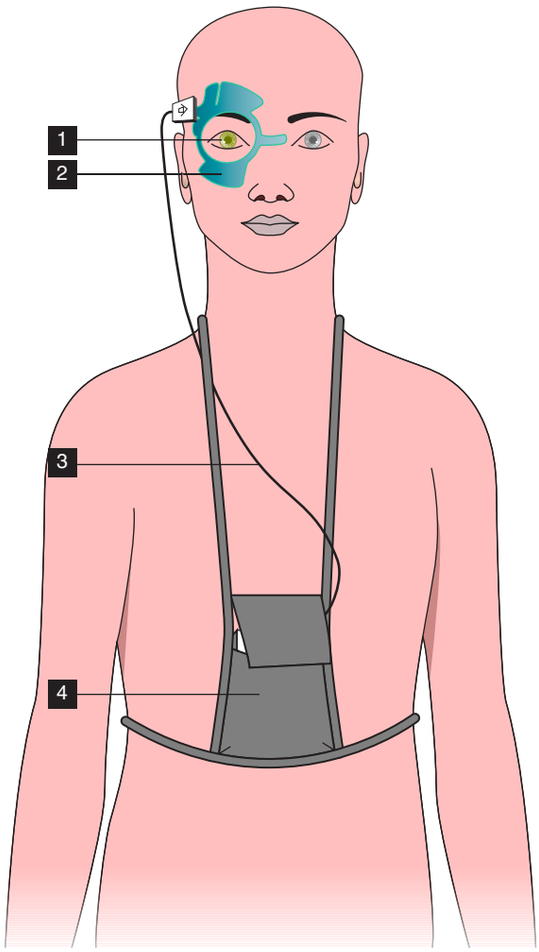
Measuring anterior chamber depth with bedside ultrasound is another alternative.[20] The patient's closed eye is covered with a Tegaderm and ultrasound gel is applied over the Tegaderm. Assess the anterior chamber using a high-frequency transducer. The anterior chamber depth is measured in the transverse and sagittal planes by measuring the widest depth between the anterior edge of the lens and the cornea.[20] This measurement is compared to the unaffected eye.

A final device is the Sensimed Triggerfish Contact Lens Sensor (Sensimed AG, Lausanne, Switzerland).[16,21-23] The Contact Lens Sensor is used by Ophthalmologists for outpatient monitoring of IOP. It consists of the lens and the monitoring system (**Figure 188-15**). The Contact Lens Sensor uses two titanium-platinum wire loop strain gauges to detect deformation of the cornea and sclera that corresponds to volume changes of the eye and thus IOP. The Contact Lens Sensor takes an IOP measurement every 5 minutes and provides 288 readings over 24 hours.

AFTERCARE

No aftercare is specific to the technique used to measure IOP. Instruct the patient, if using topical ocular anesthetic, to not rub their eyes until the anesthetic wears off. The fluorescein can stain

A



B

FIGURE 188-15. The Sensimed Triggerfish tonometry system hand-held tonometers. ***A.*** The contact lens. ***B.*** The system on a patient consists of the contact lens (*1*), the antenna around the eye (*2*), the thin flexible cable (*3*), and the recorder (*4*). (Photos courtesy of Sensimed AG, Switzerland.)

clothes and contact lenses. Do not replace contact lenses until the fluorescein wears off and instructed it is safe depending on the underlying eye condition. The device needs to be cleaned and sterilized after used on a patient.[9,24] Follow the hospital and device manufacturer's recommendations. The routine maintenance of the device is beyond the scope of this chapter.[24] Refer to the device manufacturer's recommendations.

ASSESSMENT

The normal range for IOP is 10 to 22 mmHg. A baseline IOP is specific to each patient and the patient's contralateral eye can serve as a control. We can make general assumptions about certain ranges of IOP to make rapid clinical assessments and facilitate patient care. Readings of 0 to 9 mmHg should be discussed with an Ophthalmologist, especially if there is a history of recent eye surgery or recent eye trauma. Readings of 10 to 21 mmHg are normal. Readings of 22 to 25 mmHg should be followed up with an Ophthalmologist within 2 to 3 days. IOP readings greater than 26 mmHg require an emergent consultation with an Ophthalmologist.

COMPLICATIONS

Infectious agents can be transferred via tonometer heads.[13] **It is essential to properly clean each instrument before use, before using it on the contralateral eye, and after use.** The use of a 70% isopropyl alcohol swab is an effective disinfectant for the Goldmann and Schiøtz tonometers. The Tono-Pen has single-use disposable latex covers.

Incorrect IOP measurements can occur for a variety of reasons. An improperly working plunger on the Schiøtz tonometer can result in falsely low measurements. IOP will be elevated if measured while the examiner's hand is holding the eyelids open and inadvertently pressing on the underlying globe. Measurement during eye movements, blinking, or eyelid movements will elevate IOP. Maneuvers that increase intracranial pressure (e.g., Valsalva or breath-holding) also increase IOP.

Corneal abrasions can occur while using contact tonometers. It is very important to have a cohesive tear film to avoid corneal abrasions. Instruct the patient to blink several times just prior to tonometry to spread the tear film. Apply artificial tears in patients with extremely dry eyes. The Schiøtz tonometer puts significant weight on the cornea and must be applied very gently. Warn the patient that their eye may be uncomfortable when the anesthetic wears off if a small abrasion is suspected after measuring the IOP. Instruct the patient to instill artificial tears every 4 to 6 hours. Treat larger abrasions with a topical ocular antibiotic of choice and schedule a follow-up visit.

FUTURE CONSIDERATIONS

The future for Ophthalmologists related to IOP monitoring shows several developments. Ambulatory IOP monitoring may be common.[25] The treatment of IOP may use drug-eluting ocular contact lenses.[26,27] These were approved by the U.S. Food and Drug Administration in early 2016. Patients using these devices will present to the Emergency Department when they experience problems or after office hours.

SUMMARY

The practice of tonometry is essential in guiding appropriate eye care. The Goldmann, Tono-Pen, Schiøtz, or Icare contact tonometers are readily available in most Emergency Departments. The literature frequently debates the comparative accuracy of each instrument. The Goldmann applanation tonometer is generally considered the clinical standard. All three instruments are useful for screening IOP in the Emergency Department. Factors such as the patient's ability to ambulate and the presence of periorbital swelling

will influence the choice of an instrument. The Emergency Physician should select a tonometer that feels comfortable and use it routinely. The Emergency Physician will be able to measure IOP rapidly and reliably with repeated use of the tonometer.

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189 Digital Globe Massage

Carlos Roldan and Eric F. Reichman

INTRODUCTION

Digital globe massage has been considered a heroic measure to salvage vision in cases of central retinal artery occlusion (CRAO), an ophthalmologic emergency.[1-11] CRAO is one of several diagnoses to consider in the patient with acute painless loss of vision or a visual field. The typical patient is older, between the ages of 50 and 80 years of age, and with significant systemic illnesses. CRAO is most likely an embolic event secondary to atherosclerotic disease. The vision loss is sudden, monocular, and painless. The outcome for CRAO is poor if treatment is delayed more than 2 hours. Spontaneous remission and recovery of visual function are rare. This condition requires an emergent consultation with an Ophthalmologist for medical management and the consideration of an anterior chamber paracentesis (Chapter 190). Digital globe massage can be used in an attempt to relieve the obstruction or break up the embolus so it moves distally to open some blood flow to the retina.

ANATOMY AND PATHOPHYSIOLOGY

The ophthalmic artery is a branch of the carotid artery (**Figure 189-1**). The first branch of the ophthalmic artery is the central retinal artery. This vessel runs along the optic nerve and enters the optic nerve. The central retinal artery is the main blood supply to the retina. The macula has an independent blood supply from other branches of the ophthalmic artery. An area between the macula and the optic nerve receives collateral circulation from the central retinal artery and the ciliary arteries in a small percentage of the population. This explains why a patient with a complete CRAO may have a normal-appearing macula and occasionally an area of perfusing retina adjacent to the optic nerve area.

The individual etiology remains unclear in many cases. Certain etiologies are more likely depending on the patient's age. The main cause of retinal arterial occlusions is an embolic event lodging in the central retinal artery where it narrows to pass through the lamina cribrosa or in a smaller distal branch arteriole. The embolism may be composed of aggregated fibrin and platelets arising from an ulcerated vessel wall thrombus, cholesterol from an ulcerated carotid artery plaque, material from cardiac valvular disease, or thrombus formation from giant cell arteritis in those over 70 years of age. The embolus may also result from an invasive procedure such as cardiac angioplasty, carotid angioplasty and stenting, or a carotid endarterectomy. Consider cardiac valvular disease in those under 40 years of age. Abnormal cardiac rhythms are considered an etiology for intracardiac blood clot formation in all age groups. These may embolize and lodge in the ophthalmic artery or distally in one of the branch arteries. Retrobulbar masses (e.g., a hematoma, neoplasm, or retrobulbar injection) may also lead to an optic nerve and central retinal artery compression.[1]

PHYSICAL EXAMINATION FINDINGS

The patient may display vision compromise ranging from a small visual defect to a decreased ability to finger count or perceive light to complete blindness. Grossly, the eye appears normal. However, an afferent pupillary defect may be evident with little or no reaction to direct light and a normal reaction to consensual light. The initial funduscopic examination may show a near-normal retinal appearance (**Figure 189-2*A***). This soon progresses to a pale retina,

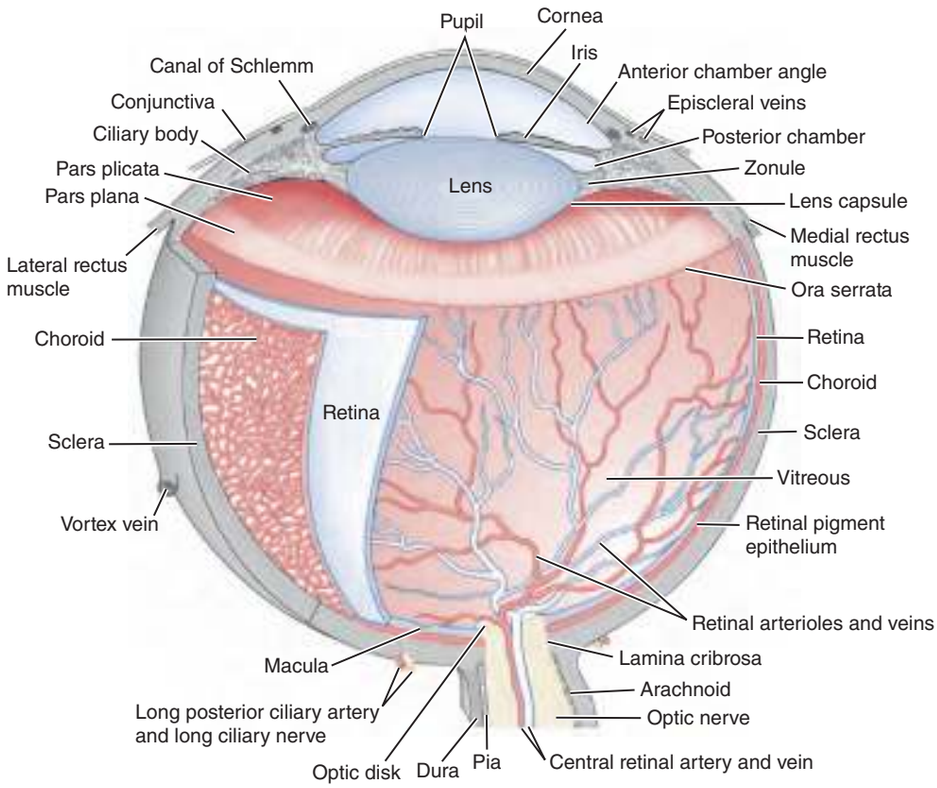
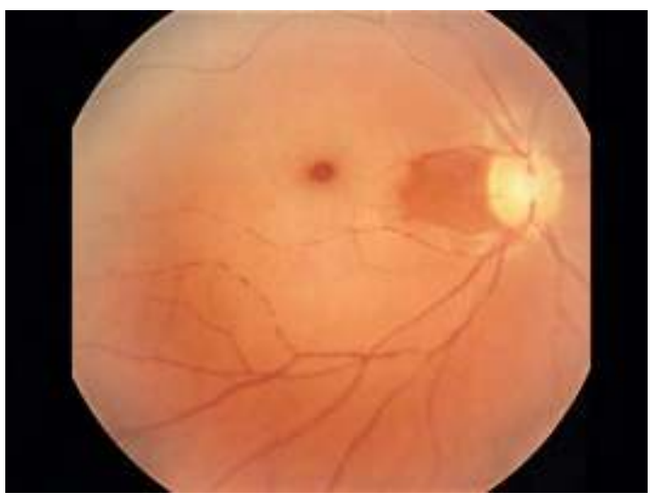


FIGURE 189-1. The anatomy of the eye. (Used with permission from Riordan-Eva P, Whitcher JP (eds): *Vaughan & Asbury's General Ophthalmology*, 17th ed. McGraw-Hill; 2008.)

bloodless or attenuated arterioles, and "boxcar" segmentation of the retinal veins (**Figure 189-2*B***). The funduscopic exam will then reveal a milky and edematous retina with a cherry-red macula if the entire central retinal artery is occluded. If the cilioretinal artery flow is not occluded, there will be an area of perfusion between the optic disk and the macula (**Figure 189-2*B***). An embolus may be visible in the vasculature of the optic disk in rare cases. If a branch retinal artery is occluded, an embolus may be visible in the vessel with ischemia and infarction distal to the occlusion.

It has been shown experimentally that the retinal damage is irreversible after 100 minutes of nonperfusion.[3] There is anecdotal evidence that heroic measures to salvage vision after a CRAO have sporadically resulted in the return of vision. A large study showed that the average final visual acuity in patients with a CRAO treated with heroic measures compared to those untreated was only a one-quarter line improvement in Snellen chart visual acuity. If the underlying cause of the CRAO is giant cell arteritis, up to 10% of the cases progress rapidly to bilateral vision loss if the arteritis is left untreated.

A



B

FIGURE 189-2. Funduscopic images of the eye. ***A.*** Normal examination. ***B.*** CRAO with cilioretinal vessel sparing. ~~Part B u~~sed with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine,* 3rd ed. New York: McGraw-Hill; 2010. Photo contributor: Thomas R. Hedges III, MD.

MEDICAL MANAGEMENT

Various methods have been employed to reduce intraocular pressure or to dilate the artery in an attempt to facilitate dislodgement of the embolus.[1-11] None of these have been proven to be of benefit in the management of a CRAO.[6] On the other hand, none of these have been proven to be harmful. Thus, they may prove to be useful in saving the patient's vision. The following interventions should be considered as co-adjuvants to digital globe massage. Oral nitrates (0.5 or 0.4 mg nitroglycerin pills or spray) may vasodilate the retinal artery. Instruct the patient to breathe into a paper bag to increase blood carbon dioxide levels and induce vasodilation. If available, administer carbogen (95% oxygen and 5% carbon dioxide) by face mask instead of using the paper bag.[11] Oral and intravenous carbonic anhydrase inhibitors (500 mg Diamox or acetazolamide, respectively) will reduce intraocular pressure and decrease ophthalmic arterial vascular resistance to forward flow.[1,11] Intravenous mannitol (1 gm/kg of a 20% solution) may cause intraocular fluid to exit the eye and decrease intraocular pressure.[1,11] Hyperbaric oxygenation treatment can compensate to prevent retinal hypoxemia and ischemia in some cases while awaiting the restoration of arterial flow.[3] Intra-arterial fibrinolysis with urokinase or recombinant tissue plasminogen activator (rTPA) can be successful in improving vision and shows promise.[1,5]

INDICATIONS

Digital globe massage can be performed in cases of CRAO where medical management is contraindicated or not successful. It can also be performed simultaneously with medical management. Treat the patient if they present to the Emergency Department within 24 hours of symptom onset.[7] It should be performed ideally within 90 minutes of the CRAO.

CONTRAINDICATIONS

Digital globe massage is contraindicated if the patient has had ocular surgery. Consult an Ophthalmologist in these cases before performing the procedure. Use caution if the patient is taking antiplatelet or anticoagulant drugs as they have a higher chance of intraocular bleeding with digital globe massage.[9] Consider discussing this with an Ophthalmologist first. Otherwise, there are no contraindications to digital globe massage in a nontraumatic CRAO. Digital globe massage is contraindicated if there is the possibility of a perforated globe.

EQUIPMENT

No special equipment is required to perform a digital globe massage.

PATIENT PREPARATION

Perform a funduscopic examination and visual acuity testing.[10] Perform intraocular pressure testing if no contraindications (Chapter 188) exist.[10] Document these results in the medical record. Explain the procedure, its risks, and benefits to the patient and/or their representative. This potentially vision-saving procedure should not be delayed. Verbal consent is adequate; document the conversation in the medical record. Place the patient supine or in a reclining position.

TECHNIQUE

Stand next to the patient's torso and facing them. Instruct the patient to tightly close the eyelids of the affected eye. Place the dominant thumb over the patient's eyelids. Apply firm and steady pressure with the thumb for approximately 5 seconds. Abruptly release pressure by quickly lifting the thumb off the eyelids. Repeat the procedure several more times as needed.[2]

ASSESSMENT

Immediately perform a repeat funduscopic examination, intraocular pressure measurement, and visual acuity testing. Document and compare these to the preprocedural evaluations. It should be noted that the funduscopic examination may show improvement while the visual acuity has changed very little or not at all. The intraocular pressure should be decreased if the arterial obstruction is relieved or broken up and moved to smaller (i.e., more distal) arterioles.[10] Depending on the ischemic time, visual acuity may never improve. Repeat the digital globe massage if there is little or no improvement in the funduscopic examination. If significant improvement is noted, the procedure may be repeated in the hope of further improvement.

AFTERCARE

All patients must be evaluated by an Ophthalmologist in the Emergency Department. If one is not available, consider transferring the patient to another facility to be evaluated by an Ophthalmologist. These patients require inpatient admission for further evaluation and management.

Patients with a CRAO usually have significant comorbidities such as hypertension, atherosclerosis, or diabetes. These patients are at risk for additional morbidity and require prompt medical referral to determine the etiology of the CRAO. Medical testing should include blood pressure evaluation, carotid ultrasonography, electrocardiography, echocardiography, blood glucose management, lipid and cholesterol testing, and hyperviscosity studies.[11] Any irregularities or abnormal studies will require further evaluation. Patients over the age of 60 need an immediate erythrocyte sedimentation rate (ESR) test in consideration of the possibility of giant cell arteritis.

COMPLICATIONS

If properly performed, digital globe massage has few immediate complications. Overvigorous digital globe massage can result in a lens dislocation or a ruptured globe.[4] Mechanical trauma can result in injury to the cornea, retinal detachments, and intraocular hemorrhage (e.g., hyphema and intravitreal hemorrhage).

SUMMARY

A CRAO is a true ophthalmologic emergency. The patient will present with sudden, painless, and unilateral loss of vision. Immediate management is required in an attempt to restore the patient's vision. This includes medical management, anterior chamber paracentesis, and digital globe massage. These techniques attempt to reduce intraocular pressure or break up the embolus in order to allow the embolus to move downstream and restore at least partial blood flow to the retina. Despite these interventions, the patient's vision may not recover.

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190 Anterior Chamber Paracentesis

Rene Pineda Carizey

INTRODUCTION

Anterior chamber paracentesis is the removal of fluid from the anterior chamber. The anterior chamber is the area just anterior to the iris and lens and immediately posterior to the cornea. Although not often formally taught or performed in the Emergency Department, an anterior chamber paracentesis is a quick, simple, and safe procedure with important diagnostic and therapeutic roles.[1-28] The long-term prognosis is directly related to the duration of symptoms for disease states that present with increased intraocular pressure (IOP) such as acute closure glaucoma and central retinal artery occlusion. In a sense, "time is eye." The Emergency Physician should become familiar with this technique. Its use can potentially prevent irreversible vision loss, especially when medical management is not sufficient in lowering IOP.

ANATOMY AND PATHOPHYSIOLOGY

The eye is a fluid-filled closed system with a one-way valve. Aqueous humor is produced by the ciliary body and flows from the posterior chamber to the anterior chamber. The aqueous humor from the anterior chamber travels through a fine trabecular meshwork at the anterior chamber angle (i.e., the one-way valve) and leaves via the canal of Schlemm.

IOP normally measures between 10 and 21 mmHg. This represents the balance between the production and outflow of aqueous humor.[3] Tonometry is used to measure IOP (Chapter 188). An increase in aqueous humor production, resistance to the outflow of aqueous humor, or additional fluid (e.g., pus or blood) in the eye can severely increase IOP and potentially cause permanent visual loss due to ischemia. Removing fluid via an anterior chamber paracentesis, in addition to medical therapies, will reduce IOP to help prevent further ischemia.

Central retinal artery occlusion (CRAO), usually from an atherosclerotic embolic event, is another potential cause of visual loss that may benefit from an anterior chamber paracentesis in combination with digital ocular massage (Chapter 189) and medical management.[4,21] Decreasing IOP increases retinal perfusion in attempts to propagate the embolus distally and minimize the amount of visual loss. Traumatic retrobulbar hemorrhages and other nonintraocular causes of elevated IOP do not benefit from an anterior chamber paracentesis.

INDICATIONS

An anterior chamber paracentesis will immediately reduce IOP but is not a treatment that resolves the underlying cause of the elevated IOP. Medical management is usually initiated first to lower IOP before attempting an anterior chamber paracentesis. An anterior chamber paracentesis is indicated whenever elevated IOP threatens visual loss and medical management is not successful in lowering IOP. Reducing the IOP acutely with an anterior chamber paracentesis in disease states such as acute angle-closure glaucoma, uveitis, hyphema, central retinal artery occlusion, and suppurative endophthalmitis may help prevent further irreversible vision loss if used in conjunction with other medical modalities.[5-7,23]

There are numerous nonemergent indications for an Ophthalmologist to perform an anterior chamber paracentesis. Diagnostically, an anterior chamber paracentesis can be used for aqueous humor sampling for a suspected infection, lymphoma, and intravitreal drug level injections and monitoring.[8,9,26,27] It is also performed for isotonic saline injection for flattened anterior chamber reformation and numerous ophthalmologic surgical procedures.

CONTRAINDICATIONS

There are no absolute contraindications to performing an anterior chamber paracentesis since the patient's vision is at risk. Consult an Ophthalmologist prior to the procedure if a ruptured globe is suspected. Relative contraindications include uncooperative patients, patients with allergies to topical ocular anesthetics, and if the Emergency Physician is not comfortable performing the procedure. The patient's airway, breathing, hemodynamic status, and other life-threatening events should always be addressed prior to the procedure.

EQUIPMENT

- Sterile povidone iodine solution
- Topical ophthalmic anesthetic drops, e.g., 0.5% proparacaine or tetracaine
- Broad-spectrum topical antibiotic eye drops, e.g., fluoroquinolone
- Sterile saline minim
- 15° super sharp microblade
- 1 mL tuberculin syringe
- 27 to 30 gauge, 5/8 inch needle[1,8,11,12]
- Slit lamp

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. Obtain a signed procedural consent and place it in the medical record. Reassure the patient that a "pressure sensation" might be felt during the procedure but they should not experience pain.

Remove all contact lenses prior to placing any topical ocular medications or using fluorescein. **Perform a visual acuity assessment (Chapter 185) and an IOP measurement (Chapter 188) prior to any intervention to document the patient's baseline status and for serial comparisons after the procedure.** Administer a tetanus booster intramuscularly if the patient's tetanus status is unknown or their last tetanus booster has been greater than 10 years. Administer antiemetic and pain medication if the patient is symptomatic.

Retching and vomiting increase IOP and complicate the procedure. Instill one to two drops each of the topical ophthalmic anesthetic and the broad-spectrum ophthalmic antibiotic onto the affected eye. Some Ophthalmologists instill two drops of sterile povidone iodine solution onto the cornea followed by the topical ophthalmic anesthetic drops to dilute it.[17,18,20,22] Place the patient comfortably in a chair. Position their chin on the slit lamp chin rest. Attempt to comfortably immobilize the patient's head. Use an assistant if needed. Check for proper anesthesia by softly brushing the cornea with a cotton-tipped swab or rechecking IOP.

TECHNIQUES

Numerous techniques have been described to perform an anterior chamber paracentesis. The quickest, simplest, and easiest to perform are the hypodermic needle and saline minim techniques. The surgical technique is often performed by an Ophthalmologist.

HYPODERMIC NEEDLE TECHNIQUE

Anesthetize the cornea and position the patient in the slit lamp. Arm a tuberculin syringe with a 27 to 30 gauge needle. Slightly withdraw the plunger to break the bead on the syringe. Position the tip of the needle at the limbus, somewhere between the 4 o'clock and 8 o'clock position (**Figure 190-1*A***). **Gently insert the needle through the cornea and angled anteriorly. This will ensure that the tip of the needle will enter the anterior chamber and not injure the ciliary body, iris, or lens (Figure 190-1*B*).** The anterior chamber holds approximately 0.3 mL of fluid. Slowly withdraw the plunger and aspirate 0.1 mL of fluid from the anterior chamber. Withdraw the needle. Apply topical ophthalmic antibiotic drops immediately after the procedure.

SALINE MINIM TECHNIQUE

A saline minim attached to a hypodermic needle is an alternative to using a syringe (**Figure 190-2**). This technique is simpler to perform when compared to using a hypodermic needle.[11,12,15] A saline minim is a single-use, soft-sided, disposable container containing 0.5 mL of sterile saline.

Anesthetize the cornea and position the patient in the slit lamp. Prepare the minim. Twist off and discard the tip of the minim. Firmly apply a 27 to 30 gauge needle to the open end of the minim. Grasp the needle-minim unit with the thumb and index finger of the dominant hand. Gently squeeze the minim until one drop of sterile saline is expressed from the tip of the needle. Do not release pressure on the sides of the minim. Position the tip of the needle at the limbus, somewhere between the 4 o'clock and 8 o'clock position (**Figure 190-2**). **Gently insert the needle through the cornea and angled anteriorly. This will ensure that the tip of the needle will enter the anterior chamber and not injure the ciliary body, iris, or lens.** Slowly open the thumb and index finger to release the compression on the minim. The aqueous humor will flow from the anterior chamber into the minim. **Do not withdraw enough fluid to dimple the cornea.** Withdraw the minim until the needle exits the eye. Apply topical ophthalmic antibiotic drops immediately after the procedure.

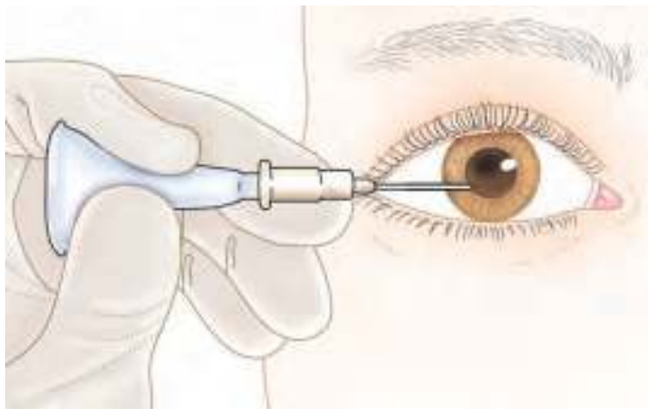
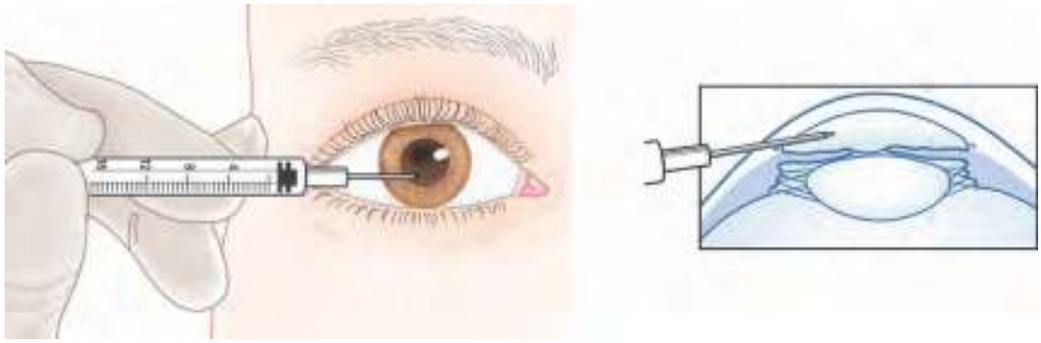


FIGURE 190-2. The saline minim technique.

SURGICAL TECHNIQUE

Anesthetize the cornea and position the patient in the slit lamp. Position the tip of the 15° super sharp microblade at the limbus, somewhere between the 4 o'clock and 8 o'clock position. **Gently insert the blade through the cornea and angled anteriorly. This will ensure that the tip of the blade will enter the anterior chamber and not injure the ciliary body, iris, or lens.** Withdraw the blade. Carefully and gently insert a 27 to 30 gauge needle on a tuberculin syringe through the incision. Aspirate 0.1 mL of fluid from the anterior chamber and withdraw the needle. As an alternative, a 27 to 30 gauge needle on a saline minim can be used. Apply topical ophthalmic antibiotic drops immediately after the procedure.



A B

FIGURE 190-1. The hypodermic needle technique. ***A.*** The needle is inserted obliquely at the limbus. ***B.*** Transverse view of the eye. The needle enters the cornea at the limbus and is directed anteriorly into the anterior chamber.

ASSESSMENT

Immediately perform an IOP measurement with the same device used before the procedure to measure IOP. The IOP and symptomatic complaints should significantly decrease after the anterior chamber paracentesis procedure. Perform serial IOP measurements every 30 minutes for 2 hours to assess for any recurrence of elevated IOP. Perform and document a postprocedure visual acuity. Readdress any symptomatic complaints of eye pain, nausea, and headache. Significant decreases in IOP have been shown immediately and up to 2 weeks after an anterior chamber paracentesis with near resolution of symptoms.[5,6,19]

AFTERCARE

An Ophthalmologist should evaluate the patient in the Emergency Department if an anterior chamber paracentesis was performed. Immediate surgical intervention may be indicated if there is no improvement in symptoms or the IOP is persistently elevated despite medical management and an anterior chamber paracentesis. Patients can usually be safely discharged home once symptoms resolve and the IOP has normalized. Arrange close follow-up with an Ophthalmologist within the next 24 to 48 hours. Patients are typically discharged with broad-spectrum topical ophthalmic antibiotics, oral pain medications, oral antiemetics, and other antiglaucoma medications (e.g., oral acetazolamide, topical ophthalmic beta-blockers, ophthalmic pilocarpine, and/or ophthalmic steroids).[10,15] The consulting Ophthalmologist will determine the proper medical management (i.e., the medications, their strength, and the frequency of administration). The patient should immediately return to the Emergency Department if they develop increased eye pain, severe nausea and/or vomiting, or any visual disturbances (e.g., decreased vision, photophobia, halos around lights).

COMPLICATIONS

Mechanical injury to ocular structures (e.g., ciliary body, corneal abrasions, iris, or lens), decompressive retinopathy, infection, and bleeding compose the most serious complications after an anterior chamber paracentesis.[13,14,25] Inadvertent injection of air into the anterior chamber occurs. This is typically a small amount and resolves spontaneously.[12] Allergic reactions to any of the medications used can also occur.

SUMMARY

Many types of disease pathologies can acutely elevate IOP and result in potential irreversible vision loss. Immediate reduction of the IOP by using medical treatments in conjunction with an anterior chamber paracentesis will reduce the amount of ischemic time and possible permanent visual impairment.

Multiple studies have repeatedly demonstrated the efficacy and safety of an anterior chamber paracentesis when used acutely to reduce IOP.[11,12] It is a procedure routinely performed in the office setting but is often met with apprehension by the Emergency Physician. Emergency Physicians should become familiar with an anterior chamber paracentesis because it is safe, simple, effective, relatively quick, and has the potential to save a patient's vision.

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191 Corneal Foreign Body Removal

Scott A. Heinrich and Dino P. Rumoro

INTRODUCTION

Corneal foreign bodies are a common complaint confronting Emergency Physicians and account for approximately 35% of all eye injuries seen.[1-3] Many objects have been implicated as a source of corneal foreign bodies (e.g., dirt, dust, glass, insects, metal, plant particles, and wood) (**Figure 191-1**).[1] Metal is the most frequently encountered ocular foreign body, often from hammering.[4] Most ocular foreign bodies require prompt removal. More than 75% of retained foreign bodies presenting on the eye surface are corneal in nature and result in a keratitis if left in place for more than 3 days.[5]

The prevailing symptom that forces patients to seek treatment is the sensation of an ocular foreign body or the pain associated with the foreign body. A variety of techniques exist for removal of ocular foreign bodies. A discussion of each of these techniques is necessary to determine the proper technique for a given situation.

A

B

FIGURE 191-1. Corneal foreign bodies. ***A.*** The foreign body is just outside the visual axis with a surrounding rust ring. ***B.*** Metallic foreign body over the center of the visual axis.

ANATOMY AND PATHOPHYSIOLOGY

Many foreign bodies are diverted from the surface of the eye by the rapid blinking action of the eyelids and the eyelashes. A foreign body may not lodge itself into the cornea or the surrounding scleral surface if it is able to get past the eyelids and eyelashes. It may be washed to the inner canthus by a combination of blinking and tear flow. The foreign body may occasionally be carried away via drainage through the lacrimal ducts and into the nose.[5] Objects may be found in the upper or lower fornices, the channels created by the fold of the inner surfaces of the eyelids in communication with the conjunctival surface of the eye. The foreign body in the upper fornix is typically found lodged in the subtarsal groove on the inner surface of the upper eyelid inferior to the tarsal plate.[5] Foreign bodies may travel deeper into the respected fornices where they may be difficult to find. Foreign bodies may be lodged into the surface of the conjunctiva overlying the sclera or the cornea. Foreign bodies in the cornea carry the most risk of serious injury or permanent scarring.

The cornea is only millimeters thick. It is composed of five layers (from outer to inner layer): epithelium, Bowman's membrane, stroma, Descemet's membrane, and the endothelial layer that lies directly over the anterior chamber.[6] The surface epithelium has five layers of squamous cells. Most superficial corneal foreign bodies become embedded in this layer and do not result in scarring. Bowman's membrane has no regenerative capacity, and if injured, scarring and permanent injury can result.[7] **Foreign bodies that violate Bowman's membrane are considered deep corneal injuries.** The stroma is composed of collagen and accounts for the largest portion of the cornea. Descemet's membrane is a basement membrane that can be regenerated if injured. The final component of the cornea is the endothelial layer which is composed of a single row of cuboidal cells that can regenerate if damaged.

Healthy cells adjacent to the injury slide over the damaged site and eventually replicate to the previous number of cells present when the corneal epithelium is injured.[4] The reepithelialization begins at the periphery and progresses toward the center of the corneal defect.[8] Conjunctival epithelium migrates over the cornea to aid in its repair. This is true even if the entire surface corneal epithelium is removed.

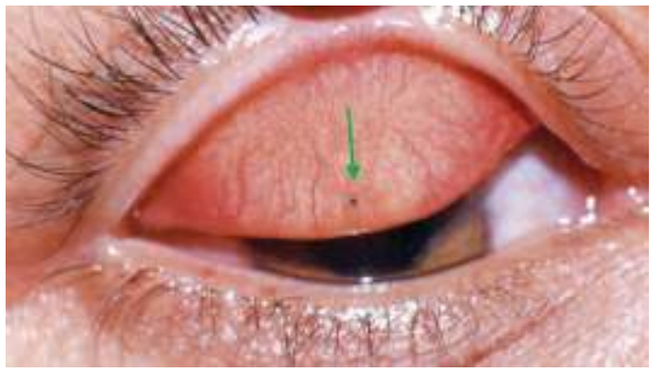
Corneal innervation is provided by sensory nerve fibers located in the surface epithelium. These are concentrated primarily in the center of the cornea and sparsely located in the periphery.[9,10] Injuries to the corneal epithelium produce foreign body sensations, pain, photophobia, and tearing.[10] More pain occurs when the central portion of the cornea is affected due to the larger distribution of sensory nerves.[10]

Patients are good at identifying the location of an embedded foreign body due to the corneal innervation. A study evaluated 50 patients with corneal foreign bodies and their accuracy in identifying the foreign body location.[11] Eighteen patients (36%) were unable to identify the foreign body location, 14 (28%) identified the exact location, and 18 (36%) were partially correct in identifying the location based upon vertical and horizontal components. **Always acknowledge the patient's sensation of an ocular foreign body and evaluate the area thoroughly.**

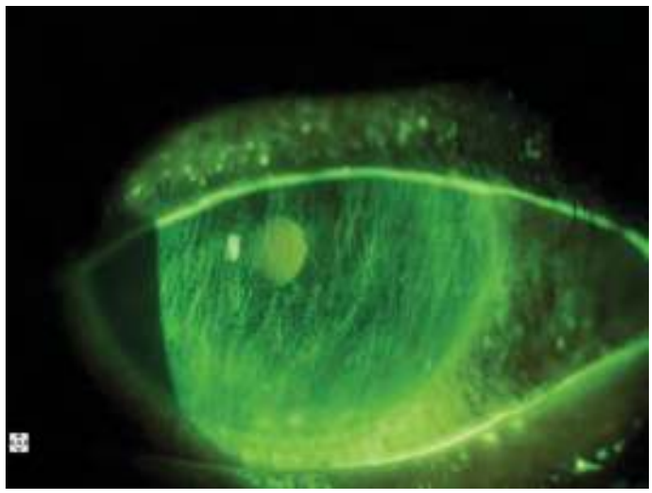
The presence of a metallic foreign body in the cornea can result in a rust ring (Chapter 192) (**Figure 191-1**). This is identified as a brownish ring surrounding the foreign body. **The rust ring and the metallic foreign body must be removed from the cornea.** The rust ring may be removed at the time of the foreign body removal or within 24 hours by an Ophthalmologist.

INDICATIONS

All foreign bodies involving the eye must be removed. They do not all need to be removed immediately in the Emergency Department. Foreign bodies that are superficial and located on



A



B

FIGURE 191-2. A foreign body under the upper eyelid. ***A.*** Clinical photo. (Reprinted with permission from Dr. Karin Lecuona, University of Cape Town.) ***B.*** Multiple vertical lines/abrasions seen using fluorescein. (Used with permission from Sonoran Desert Eye Center.)

the cornea, eyelid, lower fornix, sclera, or upper fornix can safely be removed by the Emergency Physician. Metallic foreign bodies require prompt removal to avoid the formation of a rust ring. The rust ring can be removed by the Emergency Physician with the foreign body or by an Ophthalmologist at the 24-hour follow-up visit. Vertical abrasions on the cornea during the fluorescein examination are indicative of a foreign body under the eyelid. Evert the upper eyelid to evaluate for the presence of a foreign body and remove it if one is found (**Figures 191-2**).[12]

CONTRAINDICATIONS

Corneal foreign bodies located in the direct axis of vision can cause permanent visual disturbances if improperly removed (Figure 191-1*B*).[2,7] Consult an Ophthalmologist before removing these foreign bodies as they often prefer to remove them. Deeply embedded objects or multiple foreign bodies that would require extensive debridement can result in significant scarring.[1] Consult an Ophthalmologist before attempting to remove these foreign bodies. **Avoid any manipulation of the eye if a perforated globe is suspected based upon either the direct examination of the eye or the mechanism of injury.** Foreign bodies embedded deeply within the cornea may be left in place if they are composed of an inert substance (e.g., glass).[1] This avoids the possibility of additional scarring from extensive debridement if the object is below the corneal surface and allows for healing of the epithelium over the object. **The decision to leave a corneal foreign body must be made in conjunction with an Ophthalmologist.** Consult an Ophthalmologist if an infection is associated with the foreign body (e.g., corneal clouding, corneal edema, or a purulent discharge). Refer injuries to an Ophthalmologist when they are old, the foreign body has been covered by corneal epithelium, or the foreign body is resistant to removal. Do not attempt to extract a corneal foreign body if the patient is confused or uncooperative as this can result in a perforated globe. Consider the use of intravenous sedation, procedural sedation (Chapter 159), or general anesthesia to extract the foreign body after consulting an Ophthalmologist.

EQUIPMENT

- Slit lamp
- Cotton-tipped applicator
- Corneal spud
- Ringer's lactate solution or normal saline
- Intravenous (IV) tubing
- 25 or 27 gauge needle
- Tuberculin syringe with needle
- Topical ocular anesthetic solution (e.g., proparacaine or tetracaine)
- Topical ophthalmic antibiotic
- Cycloplegic agents (e.g., cyclopentolate, homatropine, or tropicamide)
- Fluorescein strips or liquid
- Electric burr drill and burrs
- Eye patches
- Adhesive tape
- Benzoin solution
- Wood's lamp, if slit lamp is not available
- Eidolon Bluminator, if slit lamp is not available

Having a slit lamp available is preferred when removing a corneal foreign body (Chapter 185). There are alternatives if a slit lamp is not available or the Emergency Physician does not feel comfortable with using a slit lamp. The Wood's lamp can provide the appropriate blue light required for fluorescein staining and rust ring removal (**Figure 191-3*A***). This portable device has a built-in magnification lens. It has several disadvantages including having to be plugged into an outlet, an awkward shape, and a heaviness that must be balanced while using it. A device known as the Eidolon Bluminator (Eidolon Optical LLC, Natick, MA) is commercially available (**Figure 191-3*B***). It is a small, hand-held, self-contained, battery-powered device and includes a 7× magnification lens.

PATIENT PREPARATION

Perform a complete eye examination prior to removing a foreign body (Chapter 185). Measure visual acuities prior to any ocular procedure and following the procedure to document any changes. Note any irregularities in the contour of the eye, any loss of anterior chamber depth, prolapse of the iris through a corneal laceration, focal injection, a hyphema, or lens opacification. These signs may indicate a ruptured globe that requires an emergent Ophthalmology consultation.[7] Update the patient's tetanus immune status if needed.

Apply a topical ocular anesthetic agent into the affected eye if the patient has no allergies. Vary the beam of light from the slit lamp in its direction of illumination from direct exposure to indirect

A

B

FIGURE 191-3. Alternatives to the slit lamp. ***A.*** Wood's lamp. ***B.*** Eidolon Bluminator.

exposure and tangential exposure to highlight any surface defects.[13] Examine the anterior chamber for cells and flare that occur in older injuries as a result from a secondary iritis. The patient typically suffers from the discomfort of an anterior uveitis rather than the foreign body.[1]

Stain the eye with fluorescein dye (**Figure 185-26**). Fluorescein can permanently stain contact lenses and should not be used in their presence. Remove the contact lens before fluorescein staining (Chapter 186). Illuminate the eye with a cobalt blue light. Look for the green reflection of a corneal abrasion. Observe the site for the flow of fluorescein stain away from the site of a corneal puncture as anterior chamber fluid flows forward (i.e., Seidel sign, **Figure 185-28**).[7,13] **A positive Seidel sign indicates a ruptured globe.** Multiple vertical abrasions on the cornea during the fluorescein examination are indicative of a foreign body under the eyelid (**Figure 191-2*B***). Irrigate the fluorescein stain from the eye after the examination is complete to avoid any chemical-induced irritation.

Explain the risks, benefits, and alternative procedures to the patient and/or their representative for the corneal foreign body removal. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. Instill additional topical ophthalmic anesthetic solution as necessary.

TECHNIQUES

EYELID FOREIGN BODIES

Evert the upper eyelid (Chapter 185).[13] Instruct the patient to look downward. Use a cotton-tipped applicator to gently press on the upper eyelid over the tarsal plate. Grasp the eyelashes with a thumb and index finger. Pull the upper eyelid outward, downward, and upward to evert it over the applicator (**Figure 185-17**). Examine the undersurface of the eyelid for foreign bodies (**Figure 191-2**). Remove any foreign bodies by sweeping the area with a saline moistened cotton-tipped applicator (**Figure 191-4**).

Double evert the eyelid. Lift the inferior edge of the eyelid created by the initial eversion. This is accomplished by using a cotton-tipped applicator or an appropriate eyelid retractor (**Figures 185-19 and 185-20**). Examine the sclera and conjunctiva for a foreign body. Release the eyelid and instruct the patient to blink their eyes. This allows the upper eyelid to return to normal.

Evert the lower eyelid by pulling the lower eyelashes forward while the patient looks upward (**Figure 185-18**).[7] Examine the area. Sweep the scleral and palpebral (i.e., eyelid) conjunctival area with a saline-soaked cotton-tipped applicator if the patient experiences a foreign body sensation but no debris can be visualized.

IRRIGATION

Superficial foreign bodies on the conjunctiva or the cornea can sometimes be removed by using an irrigation technique (Chapter 187). A brief description is presented in this section. **Never use Morgan or Mediflow lenses (Figure 187-5) for eye irrigation in the face of a foreign body as it may lodge the object further into the cornea.**

The most appropriate solution to use is Ringer's lactate solution followed by normal saline. Tap water can be used in the prehospital setting. Ringer's lactate solution causes less irritation. The pH range of lactated Ringer's is 6.0 to 7.2 and is closer to the normal pH (i.e., 7.4) of the eye in comparison to the pH of 4.5 to 6.0 of normal saline.[7]

Place the patient supine with their head tilted toward the side being irrigated to aid in proper runoff of the irrigant. Hang the IV fluid bag above the patient's head. Insert the IV tubing spike into

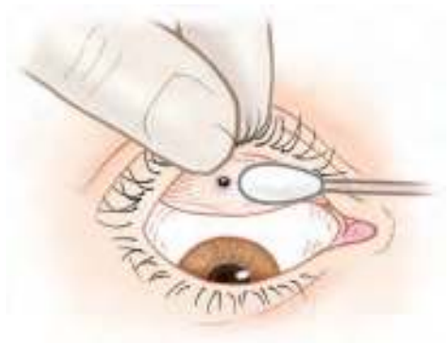


FIGURE 191-4. Evert the eyelids and remove the foreign body with a moistened cotton-tipped applicator.

the port on the IV fluid bag. Flush the patient's eye with sterile IV solution through the open end of the IV tubing while holding the patient's eyelids open (**Figure 187-3**). Flush from the scleral surface with the flow of solution directed over the cornea, thus washing the object out of the eye.[1,7,13] **Never direct the solution onto the object to avoid embedding it deeper into the soft underlying tissues. Never direct the flow directly onto the surface of the cornea to avoid secondary injury.** Use a forceps or cotton-tipped applicator to remove objects flushed onto the palpebral conjunctiva.[7] **Never use forceps on the eye.**

COTTON-TIPPED APPLICATOR

Use a cotton-tipped applicator premoistened with saline to remove a foreign body from the conjunctiva overlying the sclera or the eyelids (**Figure 191-4**).[13] Gently touch the premoistened tip of the cotton-tipped applicator to the conjunctival surface and lift off the foreign body. **Do not use the cotton-tipped applicator to remove a foreign body from the cornea.**[13] It can result in a large corneal abrasion by removing the surface epithelium.

MANUAL EXTRACTION

Extract the foreign body with a corneal spud or hypodermic needle if it is not removed via irrigation or if it is embedded superficially into the corneal surface.[14,15] A corneal spud is made of stainless steel that comes in a variety of shapes (**Figure 192-3**). It generally consists of a sharpened metal tip attached to a handle.[1] It is used to lift off or to carve out a corneal foreign body.[1] Many prefer to use a tuberculin syringe with a bent or straight needle. It allows better control of the needle by using the syringe portion as the grip. The authors always use a saline-filled tuberculin syringe. This allows the Emergency Physician to apply saline drops to moisten the eye as well as to flush away the foreign body after it is dislodged. An 18 gauge needle has been described for the removal of large foreign bodies because of its wide diameter.[1] The instrument used is physician dependent. **Proper explanation of the extraction procedure using a needle will often ease a nervous patient and ensure better compliance by limiting unexpected movements.**

Perform the procedure under direct visualization with the slit lamp. Place the patient seated at the slit lamp with their head firmly in place against the forehead rest to avoid any unexpected movement (**Figure 191-5*A***). The foreign body can be removed using the blue light of a Wood's lamp or Eidolon Bluminator if a slit lamp is not available. Hold the needle, or spud, between the thumb and index finger of the dominant hand as one would a pencil. Have the bevel facing the examiner. Stabilize the dominant hand on the patient or the slit lamp apparatus using the remaining fingers. Instruct the patient to focus their vision on a given point to avoid any eye movement.

Approximate the tip of the needle, or spud, to the foreign body with the naked eye before utilizing the slit lamp microscope to avoid inadvertent injury. Approach the foreign body from the periphery and not across the patient's field of vision (Figure 191-5*B*). Gently tease out the foreign body using the beveled edge of the needle, or spud, in a tangential direction in relation to the eye to avoid inadvertent deep puncture if the patient suddenly moves (Figures 191-5*C* and 191-6).[16] Use the tip to gently pry the foreign body loose if necessary but extreme care must be exercised (Figure 191-5*D*). Remove the loose foreign body with a moistened cotton-tipped applicator or with gentle irrigation.

ELECTRIC BURR DRILL EXTRACTION

An electric battery-powered drill equipped with various sized diamond dental burrs can be used for foreign body removal (**Figure 191-7**). Its use is typically associated with an increased tissue defect when compared to using a needle or a spud.[17] **Use caution as the burr drill can embed the foreign body deeper into the cornea.**

Choose a burr size that is slightly larger than the diameter of the foreign body. Load the burr onto the drill (**Figure 191-7**). Grasp the device like a pencil using the thumb and middle finger. Press the finger bar on the drill with the index finger to turn on the drill and

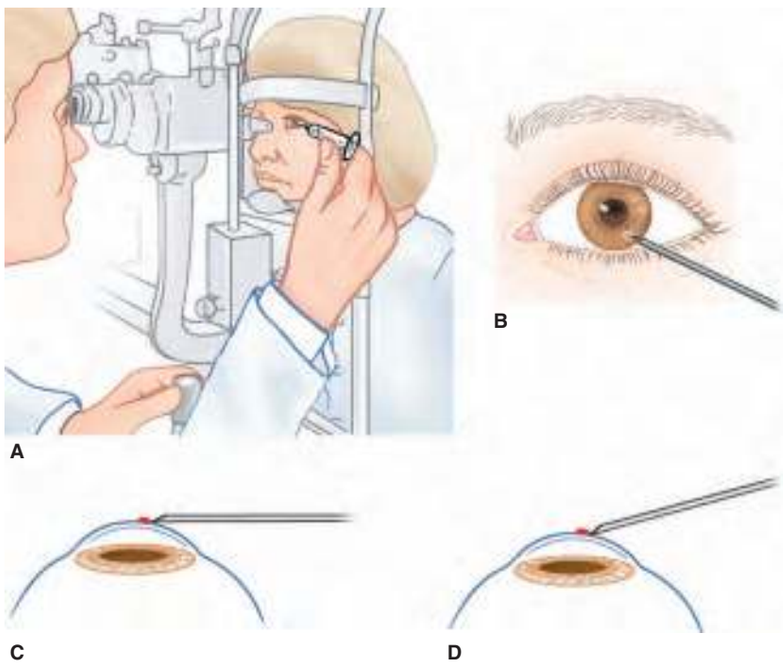


FIGURE 191-5. A hypodermic needle to remove a corneal foreign body. ***A.*** Position the patient in the slit lamp. Stabilize the hand holding the needle. ***B.*** Approach the foreign body from the periphery. ***C.*** Hold the needle tangential to the cornea to remove the foreign body. ***D.*** Slightly angle the needle to "pry" the foreign body off the cornea.

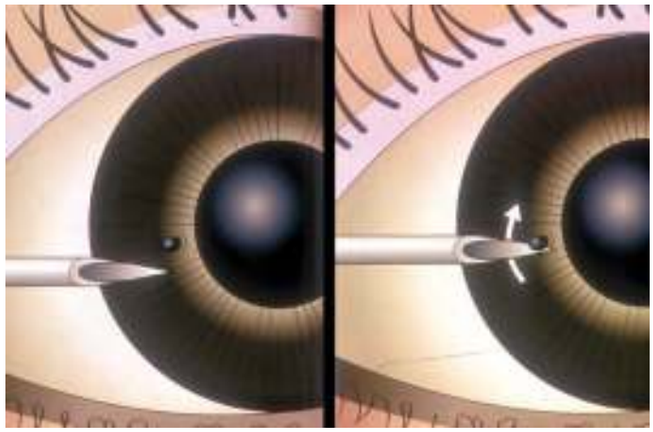


FIGURE 191-6. A needle to remove a corneal foreign body. (Used with permission from reference 16 and Cactus Design and Illustration Ltd.)

A

B

C

FIGURE 191-7. The burr drill used to remove a corneal foreign body. ***A.*** The drill. ***B.*** The burr bits. ***C.*** The burr drill with a burr bit inserted.

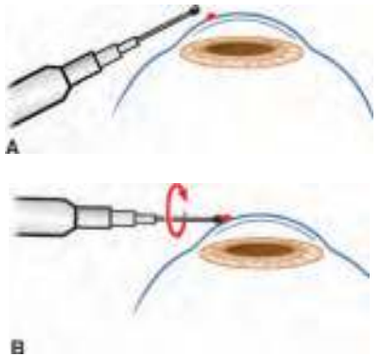


FIGURE 191-8. Removal of a corneal foreign body with a burr drill.

rotate the burr bit. Approach the foreign body tangentially to the eye (**Figure 191-8**). Gently place the rotating burr bit on the area to be debrided using short applications of one to two rotations of the burr. Lift the burr from the cornea after each application to examine the area and determine if the foreign body has been removed.

ASSESSMENT

Carefully examine the cornea under the slit lamp with and without the use of fluorescein dye. Ensure that the foreign body is completely removed. Consult an Ophthalmologist if the foreign body has broken off, is deeply embedded within the cornea, or is associated with a rust ring. Measure and record the patient's visual acuity after the extraction procedure. Compare this to the preextraction visual acuity. Consult an Ophthalmologist if there is a difference in the preextraction and postextraction visual acuity.

AFTERCARE

A corneal abrasion will be present upon removal of the foreign body. The defect is usually larger than the original foreign body and should be treated as a typical corneal abrasion. The average time to resolution of the epithelial defect is approximately 4.3 days.[8] Place a broad-spectrum ophthalmic antibiotic ointment or drops (e.g., a fluoroquinolone) on the eye.[18] Instruct the patient to instill topical broad-spectrum antibiotics every 4 hours if the eye is not patched.[1,7,9,13] Ophthalmic nonsteroidal anti-inflammatory drugs (NSAIDs) are safe to use and provide effective pain relief, especially in patients who can afford the medication and must return to work immediately.[19-22]

The use of an eye patch is controversial and usually not required (Chapter 193). A review showed that patching simple corneal abrasions may not improve healing or pain.[23] Consider the use of eye patches cautiously as they may be associated with additional discomfort, delayed healing, and the conversion of a corneal abrasion into a corneal ulcer.[24] Consider patching the eye closed if the defect is large and painful. Avoid pressure patching the eye in the case of small, superficial, or minimally painful abrasions. Avoid the use of eye patches in organic foreign bodies and patients who wear contact lenses as the bacterial milieu is favorable for the development of a local infection.[7] Consult an Ophthalmologist before patching an eye.

The proper method of applying an eye patch is to first administer all necessary medications. Instruct the patient to close both eyes. Apply a folded patch to the affected eye with the rounded edge pointed downward. Place an unfolded patch over the folded patch. Prep the skin with a benzoin solution to aid with tape adherence.

Apply strips of tape from the medial aspect of the forehead, over the patch, and to the lateral cheek (**Figure 193-2**). Apply the tape tightly enough to prevent eye opening but lightly enough to not cause discomfort. Apply strips of tape repeatedly over the patch until it is entirely covered. Apply the final pieces of tape over the center of the patch while holding the cheek soft tissue superiorly. This will ensure that the cheek, when released, will pull the bandage taut and avoid loosening.[7,13]

Cycloplegics (e.g., 1% cyclopentolate) can be used to alleviate pain or to limit pain with significant corneal defects or an anterior uveitis.[1] Topical steroids have no place in the treatment of traumatic corneal defects.[1] Oral analgesics are appropriate in cases of persistent discomfort but may not help. **Historically, topical ocular anesthetic agents were not prescribed due to their abusive potential and the direct deleterious effects of the anesthetic on the corneal epithelium.**[7] This toxicity was noted in animal studies with prolonged exposure or case reports of patient abuse.[25-27] Limited data suggest that dilute topical anesthetics for a short course may be effective.[28-33] Their safety for outpatient use is to be decided by each Emergency Physician.

Refer all patients to an Ophthalmologist for reexamination in 24 hours, especially if an eye patch has been applied. A rust ring can be removed by the Ophthalmologist at the follow-up visit. Instruct the patient not to operate a vehicle if the eye is patched. These instructions hold true if the eye is not patched but a procedure was performed to remove a foreign body.

Many eye injuries occur while at work due to failure to comply with wearing eye protection, resulting in a significant loss of working hours.[10,11,34-37] Take the time necessary to explain the importance of wearing safety goggles if the injury occurred while at work, gardening, or participating in hobbies (e.g., woodworking, drilling, auto repair).

COMPLICATIONS

A foreign body will occasionally not be able to be removed in the Emergency Department. Refer the patient to an Ophthalmologist for definitive treatment before further attempts at removal result in severe ocular injury and the potential for violation of the anterior chamber (i.e., perforation of the globe). **Notify an Ophthalmologist immediately if inadvertent puncture of the globe occurs from attempts at foreign body removal because a surgical emergency now exists.**

Improper placement of eye patches can cause a corneal ulcer or delayed corneal healing.[13] Caution the patient against driving while the eye is patched due to impaired depth perception.[13]

Continued discomfort at the 24-hour follow-up may indicate an anterior uveitis is present, a corneal rust ring from a metallic object has formed, the foreign body was not completely removed, or an infection or penetrating eye injury is present.[1] Contaminated corneal foreign bodies or those consisting of vegetative material can lead to corneal ulceration from the introduction of bacteria.[1]

Corneal defects will not always be completely healed at the 24-hour follow-up. The eye may require 1 additional day of patching if the defect is still a significant size. Treat the defect by applying a topical antibiotic for several more days if the defect is markedly smaller than the original injury.[1]

Care must be taken to avoid further damage to the eye when treating children or patients who are uncooperative. IV sedation and analgesia (Chapter 159) can be used in these situations. Consult an Ophthalmologist for treatment under general anesthesia if sedation is contraindicated or if not comfortable with removing the foreign body under procedural sedation.[7]

SUMMARY

Corneal foreign bodies are routinely encountered in the Emergency Department. They must be definitively treated to avoid long-lasting ocular disability. Documentation of a complete history and physical examination of the eye is mandatory before attempting to remove a foreign body. The most reliable method of corneal foreign body removal is the use of a hypodermic needle or corneal spud, taking care to avoid additional ocular trauma. Administer appropriate antibiotics and/or cycloplegics after removal of the foreign body. The application of an eye patch is not required after the foreign body is removed in most cases. The decision to apply an eye patch is best done in consultation with an Ophthalmologist. Refer the patient to an Ophthalmologist in 24 hours for evaluation of proper healing and the immediate treatment of any evolving complications.

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192 Corneal Rust Ring Removal

Adam Jennings, Marcus Holmes, and Devin Sandlin

INTRODUCTION

Corneal rust rings occur commonly when metallic foreign bodies become embedded in the cornea (**Figure 192-1**). **Removal of the rust ring is imperative to avoid permanent staining of the cornea, persistent inflammation, or disruption of corneal integrity (i.e., necrosis) with loss of stromal substance.**[1-3] Two techniques for the removal of rust rings are discussed. Topical deferoxamine as a chemical chelator should only be used by an Ophthalmologist and is mentioned only for the sake of completeness.

FIGURE 192-1. The corneal rust ring. (Used with permission from Tintinalli JE, et al: *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th ed. New York: McGraw-Hill; 2016.)

ANATOMY AND PATHOPHYSIOLOGY

The cornea is approximately 0.5 mm thick and is composed of five layers. From the outer to inner layer is the corneal epithelium, Bowman's membrane, stroma (largest layer), Descemet's membrane, an endothelial layer, lies directly over the anterior chamber. **Corneal rust rings are formed from the oxidation of iron present in metallic foreign bodies.**[4] As little as 3 hours of corneal contact are required to form the brown stain of a rust ring.[1]

INDICATIONS

All corneal metallic foreign bodies require prompt removal to avoid the possibility of rust ring formation. A rust ring requires complete removal in a timely fashion in order to avoid the damaging effects of rust on the cornea. While foreign bodies should be removed in the Emergency Department, the rust ring can be left for the Ophthalmologist to remove within 24 to 48 hours if the Emergency Physician does not feel comfortable removing the rust ring.

CONTRAINDICATIONS

Corneal foreign bodies and rust rings that are located in the direct axis of vision can cause permanent visual disturbances if improperly removed.[2] Consult an Ophthalmologist before removing these as they often prefer to remove them. Do not attempt to extract a rust ring if the patient is a young child, confused, or uncooperative as this can result in a perforated globe. These patients may require the use of intravenous sedation, procedural sedation (Chapter 159), or general anesthesia to extract the rust ring.

EQUIPMENT

- Slit lamp
- 25 or 27 gauge needle
- Tuberculin syringe with a needle
- Burr drill
- Burr bits
- Topical ocular anesthetic agent (e.g., proparacaine or tetracaine)
- Topical ophthalmic antibiotic
- Cycloplegic agents (e.g., cyclopentolate, homatropine, or tropicamide)
- Ringer's lactate solution or normal saline
- Fluorescein strips or liquid
- Wood's lamp, if a slit lamp is not available
- Eidolon Bluminator, if a slit lamp is not available
- Ophthalmic foreign body instruments, optional[5]

Having the availability of a slit lamp is preferred when removing a corneal rust ring.[6] There are alternatives if a slit lamp is not available or the Emergency Physician does not feel comfortable with using a slit lamp (**Figure 191-3**). The Wood's lamp can provide the appropriate blue light required for fluorescein staining and rust ring removal. This portable device has a built-in magnification lens. It has several disadvantages including having to be plugged into an outlet, an awkward shape, and a heaviness that must be balanced while using it. A newer device is the Eidolon Bluminator (Eidolon Optical LLC, Natick, MA). It is a small, hand-held, self-contained, battery-powered device and includes a 7× magnification lens.

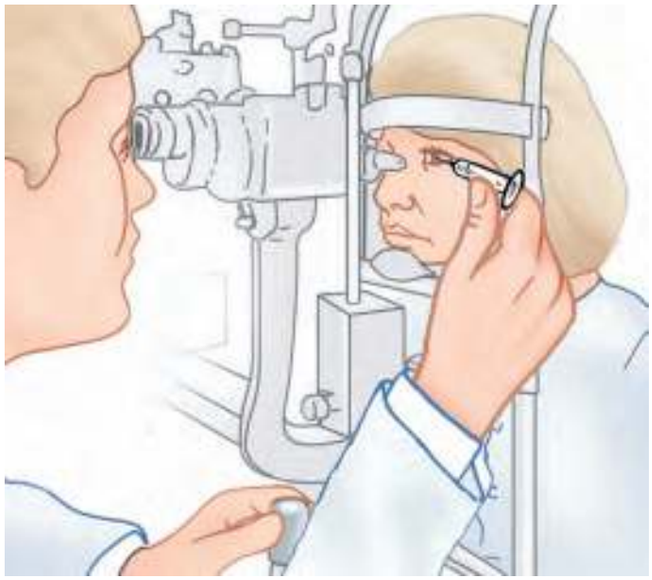

FIGURE 192-2. Positioning of the patient and the emergency physician's hand.

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. A calm and thorough explanation of what is going to occur and showing the tools to be used provides an adequate anxiolytic effect. Obtain a signed informed consent to perform this procedure. Apply a topical ocular anesthetic agent into the affected eye.[7,8] Determine the patient's visual acuity in the affected eye. Seat the patient at the slit lamp with their head firmly in place to avoid any unexpected movement (**Figure 192-2**). **Examine the eye via the slit lamp and rule out the possibility of an intraocular foreign body with a corneal perforation.** Perform a complete eye examination to rule out any other ocular problems. Remove the corneal foreign body (Chapter 191) if it is still present with the hypodermic needle, tuberculin syringe, or ophthalmologic instrument. The rust ring will often be removed simultaneously with the metallic foreign body. Make an attempt to remove the rust ring if it remains after removal of the foreign body.

TECHNIQUES

MANUAL EXTRACTION

Scrape out the rust ring in a similar fashion to the removal of a metallic foreign body (Chapter 191). **The entire area of rust-stained epithelium must be completely removed without any residual rust.**

Extract the rust ring with a corneal spud, another ophthalmologic instrument, or a hypodermic needle (**Figure 192-3**). A corneal spud is a stainless steel device that comes in a variety of shapes. It generally consists of a sharpened metal tip attached to a handle.[1] It is used to lift off or to carve out a corneal foreign body or rust ring.[1] Many Emergency Physicians prefer to use a tuberculin syringe. It allows for better control of the needle by using the syringe portion as the grip. The author always uses a saline-filled tuberculin syringe. Others use a needle that they bend attached to a syringe.[9] This allows the application of saline drops to moisten the eye as well as to flush away the rust ring after it is dislodged from the cornea. The instrument used is physician dependent. **Proper explanation of the extraction procedure using a needle will often ease a nervous patient and ensure better compliance by limiting unexpected movements.**

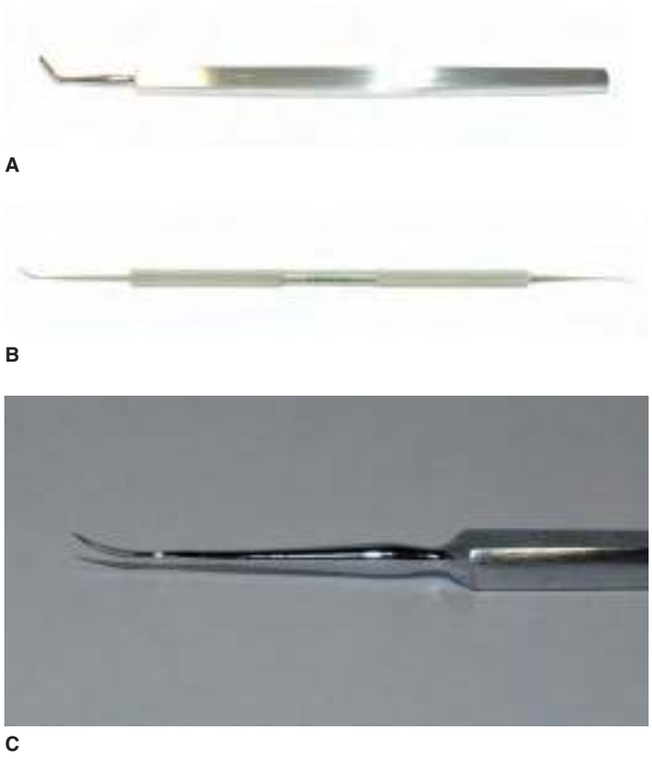

FIGURE 192-3. Instruments to manually remove a corneal rust ring. ***A.*** Corneal spud. ***B.*** Corneal dissector. ***C.*** Corneal foreign body needle.

Perform the procedure under direct visualization with the slit lamp. Place the patient seated at the slit lamp with their head firmly placed against the forehead rest to avoid any unexpected movement (**Figure 192-2**). The foreign body can be removed using the blue light of a Wood's lamp or Eidolon Bluminator if a slit lamp is not available. Hold the needle, or ophthalmologic instrument, between the thumb and index finger of the dominant hand with the bevel facing the examiner as one would hold a pencil. Stabilize the dominant hand on the patient or the slit lamp apparatus using the remaining fingers. Instruct the patient to focus their vision on a given point to avoid any eye movement.

Approximate the tip of the needle, or ophthalmologic instrument, to the rust ring with the naked eye before utilizing the slit lamp microscope in order to avoid inadvertent injury. Approach the rust ring from the periphery and not across the patient's field of vision (Figure 192-4*A*). Gently tease out the rust ring using the beveled edge of the needle, or ophthalmologic instrument, in a tangential direction in relation to the eye to avoid inadvertent deep puncture if the patient suddenly moves. Use the tip to gently pry the rust ring loose if absolutely necessary (Figure 192-4*B*).

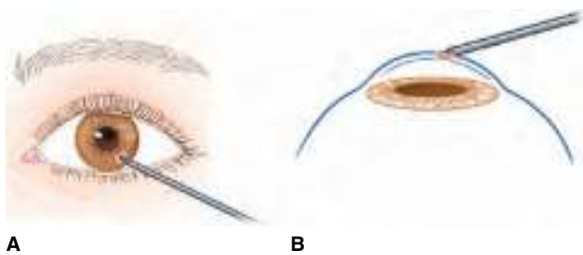

FIGURE 192-4. Manual extraction of the rust ring.

FIGURE 192-5. An example of a corneal burr drill with a drill bit.

Extreme care must be exercised. Remove the loose rust ring with a moistened cotton-tipped applicator or with gentle irrigation.

ELECTRIC BURR DRILL EXTRACTION

An electric battery-powered burr drill equipped with diamond burrs can be used for rust ring removal (**Figures 192-5 and 192-6**).[10] The burr bit cuts away corneal tissue very slowly. Corneal rust rings were induced in rabbits and comparisons were made between manual extraction versus electric burr drill extraction.[1] Both were equally effective for rust ring removal but the burr drill caused a deeper corneal defect. **Use caution when using an electric burr drill.** There was no difference in corneal scarring between the two techniques. The burr drill can also be used to extract rust located in the bulbar conjunctiva.[11]

Choose a burr size that is slightly larger than the diameter of the rust ring. Load the burr onto the drill. Grasp the device like a pencil using the thumb and middle finger. Press the finger bar on the drill with the index finger to turn on the drill and rotate the burr bit. Other burr drills turn on with a screw-type mechanism on the burr drill (**Figure 192-5**). Approach the rust ring tangentially to the eye (**Figure 192-6*A***). Gently place the rotating burr bit on the area to be debrided using short applications of one to two rotations of the burr (**Figure 192-6*B***). Lift the burr from the cornea after each application to examine the area and determine if the rust ring has been removed. **Thoroughly inspect the base of the crater to ensure that it is free of rust.**[11] Continue the process until the rust ring is removed. Use a slightly larger burr bit if rust still remains along the periphery of the crater.

DELAYED REMOVAL

The rust ring may be removed after it ages.[2,5,12] Allow the rust ring to remain for 24 to 48 hours. During this time, the iron deposits will kill the surrounding corneal epithelial cells. The rust ring will "soften" and be easily removed in one piece with a needle, ophthalmologic instrument, or burr drill. **Do not allow the rust ring to remain for longer than 24 to 48 hours as it can cause significant damage to the cornea.**

CHEMICAL CHELATION

Topical deferoxamine has been used experimentally for the nonsurgical removal of rust stains on the cornea.[13] It has been shown to remove rust, although perhaps not as reliably as the methods previously described. The potential exists for resultant eye irritation, corneal ulceration, or persistence of the rust stain.[13] The clinical use of deferoxamine should be done only in very select situations such as in children with multiple lesions or when the central axis of vision is involved.[14] This technique is reserved for the Ophthalmologist and is therefore not described in this text.

ASSESSMENT

Measure and document visual acuities before and after the procedure. Consult an Ophthalmologist if the postprocedural visual acuity is different from the preprocedural visual acuity.

AFTERCARE

A corneal defect resembling a corneal abrasion will be present after the rust ring is removed. The defect is usually larger than the original foreign body and should be treated as a typical corneal abrasion. Apply topical ocular antibiotic ointment in the eye. The use of an eye patch (Chapter 193) is controversial but considered by most as unnecessary. Contact lens wearers should switch to glasses until the eye is fully healed and evaluated by the Ophthalmologist. Instruct the patient not to operate a vehicle if possible. Administer cycloplegics to alleviate pain from an actual or potential anterior uveitis.[2] Oral analgesics are appropriate in cases of persistent discomfort. Consider prescribing topical nonsteroidal anti-inflammatory drugs (NSAIDs) for pain relief (e.g., bromfenac, diclofenac, or ketorolac).[15] Refer the patient to an Ophthalmologist for reexamination in 24 hours. The patient requires daily follow-up and corneal staining to ensure proper healing.[16]

Instruct the patient on the importance of proper eye protection.[17] Many eye injuries occur while at work due to failure to comply with wearing eye protection and result in a significant loss of working hours.[7,18,19] Take the time that is necessary to explain the importance of wearing safety goggles if the injury occurred while at work, gardening, or participating in hobbies (e.g., woodworking, drilling, and auto repair). The prevention of ocular foreign bodies and rust rings is preferable to treating them.[20]

Some consider sending the patient home with topical anesthetic eye drops.[7,21-24] Diluted drops are used for outpatients for the

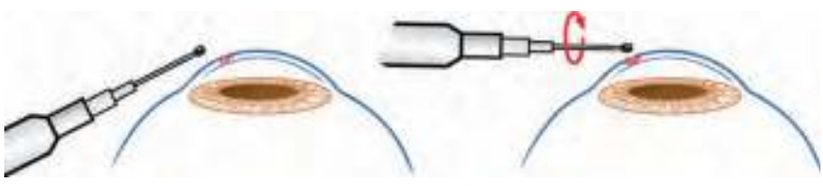

FIGURE 192-6. Burr drill extraction of the rust ring.

management of pain from a photorefractive keratotomy and other ophthalmic procedures. The use of these topical medications as an outpatient from the Emergency Department is controversial. This decision is left up to each Emergency Physician, their department protocols, and in consultation with local Ophthalmologists.

COMPLICATIONS

Abandon multiple attempts at removal of the rust ring to avoid severe ocular injury and the potential for violation of the anterior chamber if the depth of the rust cannot be determined or if repeated debridement is unable to completely remove the rust ring. The eye can be left unpatched for 24 hours and the patient referred to an Ophthalmologist for definitive removal the following day in these situations or in cases of hesitancy in removing a rust ring. Delayed removal is often easier than the initial attempts due to further oxidation and injury of the corneal epithelium resulting in "softening" of the rust. The "softened" rust ring is easily scraped out in 24 to 48 hours.[2] The rust ring can lead to chronic inflammation, corneal necrosis, corneal vascularization, or permanent scarring.[3]

SUMMARY

Removal of corneal rust rings is imperative in order to avoid permanent ocular injury. It is best accomplished with a small-gauge hypodermic needle or with an electric burr device. An alternative approach is to leave the rust ring for delayed removal by an Ophthalmologist in 24 hours when the rust has "softened." It is essential to completely remove the rust ring to prevent future ocular morbidity. The procedure is simple to perform with readily available equipment.

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193 Eye Patching and Eye Shields

Teresa D. Le and Adam R. Jennings

INTRODUCTION

Eye shields are used to protect the eye from further injury when the integrity of the globe is compromised or potentially compromised.[1] The best results are obtained when early repair of globe disruption occurs and before any globe contents leak out or change position.[2,3] Eye patches are intended to prevent movement of the eyelid over an injured but intact cornea.[1,4,5] Eye patching has historically been used to protect the eye from bright light, to facilitate healing of a corneal abrasion, or to protect the cornea from injury during sleep. There have been no substantial changes in the indications for eye shields and their method of application.[6-9] Eye patching has become increasingly controversial.[6-10]

EYE SHIELDS

INDICATIONS

Eye shields serve as temporary protection for patients in whom a penetrated or ruptured globe is suspected.[11-13] The purpose is to prevent further injury as extravasation of globe contents is associated with poor vision outcomes.[3,14] **A ruptured globe is a vision-threatening emergency.** The signs associated with a ruptured globe include bloody chemosis, increase or decrease in the depth of the anterior chamber, irregular or peaked iris, positive Seidel test, vitreous hemorrhage, low intraocular pressure, hyphema, and loss of visual acuity.[12,13,15-17] Globe rupture should also be suspected from penetrating corneal wounds from foreign bodies (e.g., tiny pieces of metal).[18,19] **Further eye examination or eye manipulation is contraindicated if a ruptured globe is suspected. Do not measure intraocular pressure. It can worsen or cause the extrusion of globe contents.**[20] An eye shield is applied immediately and to remain in place while other injuries are managed, tests are obtained (e.g., orbital computed tomography [CT] scans), and the consulting Ophthalmologist is contacted.[3,13]

CONTRAINDICATIONS

The only contraindication to the application of an eye shield arises when the surrounding face and orbits are so extensively damaged that the metal shield or its edges will directly injure the globe. The

FIGURE 193-1. A paper cup may be used if a commercial eye shield is not available. The top of the cup is cut in multiple places, and the resulting "flaps" are bent down to create a flat surface.

protective function of the eye shield relies on its edges being supported by the orbital rim, frontal bone, and maxillary bone.

EQUIPMENT

- Metal or plastic eye shield
- 1 inch tape

Most Emergency Departments stock commercially available eye shields. A clean, disposable paper drinking cup may be used if an eye shield is not available.[5,21] Use a scissors to make 3/4 to 1 inch deep cuts around the open end of the cup (**Figure 193-1**). Make approximately six to eight cuts around the circumference of the cup. Fold the flaps on the open end of the cup outward. **Styrofoam cups are not suitable because the flaps can easily break off and cause further injury to the globe.**

PATIENT PREPARATION

The first procedure in patients with a suspected globe disruption is the application of the eye shield.[13,16] There should be no preparations except to dry the area of skin that will receive the securing tape. Do not irrigate the eye, place any gauze or patch touching the eye, and/or apply ocular ointments or drops. Ocular tonometry is also contraindicated.[13] All the previously mentioned interventions are likely to cause further injury to the exposed globe.[3,22] Contact an Ophthalmologist immediately to expedite globe repair.[12,13,16] Obtain a CT scan of the orbits if a foreign body may have possibly penetrated the globe.[19,23]

It may be possible in the future to determine if a globe is ruptured without the radiation associated with a CT scan. A portable sensor is being developed that is inexpensive and can quickly determine if the globe has been penetrated.[24] The device measures vitamin C levels in the eye fluid. The interior of the globe has much higher levels than the normal tear film. Penetrating injury results in the aqueous fluid leaking out and causes the tear fluid to have a higher level of vitamin C.

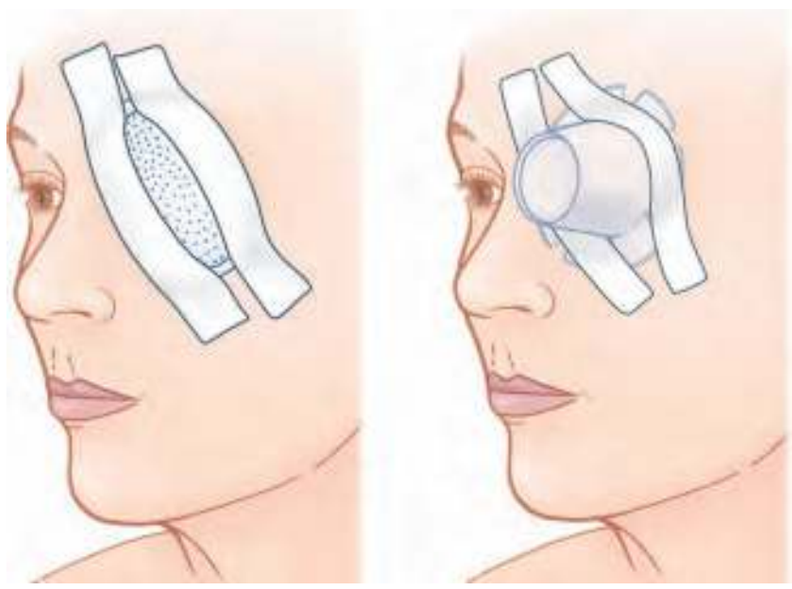
TECHNIQUE

Apply the commercial eye shield (**Figure 193-2*A***) or the one improvised from a paper cup (**Figure 193-2*B***) over the injured eye. **Ensure that its edges do not contact structures any closer to the eye than the orbital rim.** Tear off four to six strips of 1 inch tape, each 4 to 5 inches long. Apply the strips of tape diagonally from the center of the forehead to the cheek above the mandible to hold the shield in place (**Figures 193-2*A* and 193-2*B***).[21]

Care must be taken to avoid taping the nasolabial folds, lips, facial hair, and skin over the mandible.[4,5] Otherwise the mandibular movement of eating and talking may cause the eye shield to move and potentially injure the eye. The tape may not stick if the patient has facial hair. The patient may benefit from a quick shave in the Emergency Department. Some patients require the use of an oxygen mask. Trim the oxygen mask around the eye shield to prevent it from displacing the eye shield and causing further eye injury.

ALTERNATIVE TECHNIQUE

An alternative to the eye shield is a moisture chamber (Good-Lite Co., Elgin, IL). These foam-edge devices have a clear lens (**Figure 193-3**). These devices are not latex, fit over the eye, prevent air flow into the eye, and are comfortable to wear. They also avoid any issues related to taping. Apply the moisture chamber followed by gauze to prevent the patient from seeing and accommodating with the injured eye.



A B

FIGURE 193-2. Eye shields. ***A.*** A commercial eye shield is applied. ***B.*** An improvised eye shield is applied.

A

B

FIGURE 193-3. Moisture chambers. ***A.*** Single moisture chamber. ***B.*** Moisture chamber goggles. (Photos courtesy of Good-Lite Company.)

AFTERCARE

The eye shield position should frequently be checked for any movement or loosening of the tape due to blood, perspiration, or other fluids. **The shield must remain in place until tests demonstrate that the globe is intact or the consulting Ophthalmologist arrives.** Do not allow the patient to eat or drink. Additional therapy includes tetanus prophylaxis, prophylactic intravenous antibiotics, and parenteral antiemetics for nausea.[2,3,13,16,17,22] Vomiting will cause extrusion of the contents of the globe.[2,3,13,16,17,22] Position the patient with their head elevated if not contraindicated.

COMPLICATIONS

Poor positioning or shield movement can further injure the eye or cause extravasation of the globe contents. **The patient must be handled gently.** Wincing or squinting of the eye can increase intraocular pressure sufficiently to cause the extravasation of the contents of the globe.

EYE PATCHING

INDICATIONS

The indications for eye patches are steadily being reduced as a growing body of literature shows no benefit and potential harm.[10] Patches were indicated for corneal injuries due to abrasions, thermal burns, light burns, or chemical burns.[4] Patches were believed to promote healing and provide pain relief by decreasing movement of the eyelid across the recovering cornea.[4,5] Patches also block light in photophobic patients with ciliary spasm or reactive iritis due to corneal injuries.

Most of these assumptions are now questioned. Patching is believed to decrease corneal oxygenation, increase eye temperature, and increase corneal infection risk.[14,25,26] Patches can cause injury if the eye opens underneath it. Numerous trials and meta-analyses comparing patients with corneal abrasions receiving patching versus no patching have found no difference in pain scores or healing time for small abrasions less than 10 mm in diameter.[6-9,27] Decreased healing time was observed for patched patients with abrasions greater than 10 mm in diameter.[25,26] Patching is still recommended for patients with large defects but most authors also recommend further study in this group.[8]

Soft contact lens bandages have been used for patients who must maintain binocular vision.[28,29] Eye patches were also previously indicated during sleep to protect the cornea in patients with facial nerve palsy (e.g., Bell's palsy) and the inability to completely close their eyes.[30] Current recommendations include applying ophthalmologic lubricants with or without an eye shield in this group.[31]

CONTRAINDICATIONS

Absolute contraindications to eye patching are corneal abrasions due to the wearing of contact lenses. There is an increased incidence of infection and more pathogenic bacteria harbored by contact lens wearers.[25] **Do not place an eye patch on any patient considered at risk for penetration or rupture of their globe.**[13] Eye patching will increase intraocular pressure and may cause extravasation of the contents of the globe.[19] Patients must be carefully assessed for the presence of corneal ulcers masquerading as abrasions.[4] Eye patching of a corneal ulcer may place pressure on the friable tissue causing a deepening of the ulcer, subsequent globe perforation, and promotion of infection.

Relative contraindications are related to abrasion size and individual patient needs. Small abrasions heal well without patching. Patients who require binocular vision to function will benefit from not patching.[8,25,26] Patches that come loose and allow eyelid movement are more painful for patients than no patch. Contraindications to ophthalmoplegics used in conjunction with patches are patients with known glaucoma or narrow angles on physical examination.[4] The contraindication to contact lens bandages is an abrasion in a patient who was wearing contact lenses because of the risk of infection.[12]

EQUIPMENT

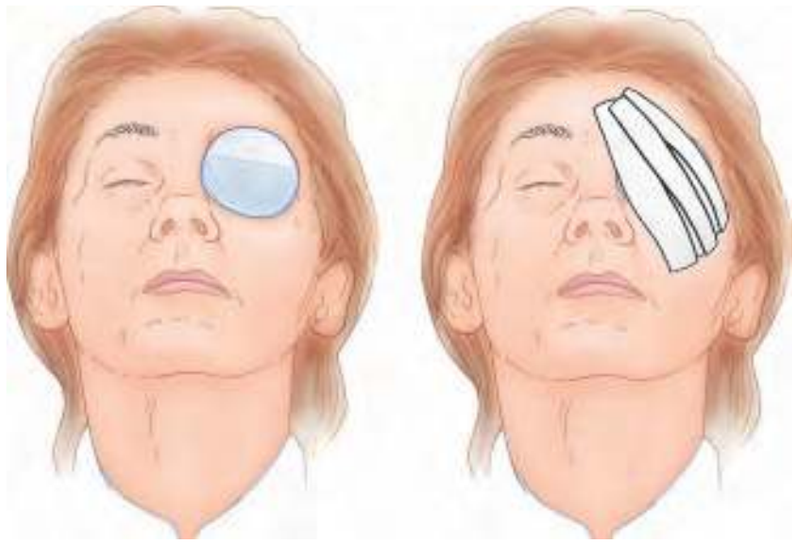
- Cycloplegic drops to reduce photophobia (e.g., 1% cyclopentolate or 5% homatropine)[5,14,25]
- Broad-spectrum antibiotic ointment or drops
- Cotton eye patches
- 1 inch tape
- Contact bandage soft lens with a power of minus 0.5 diopters[29]

PATIENT PREPARATION

Conduct a complete eye examination (Chapter 185) to ensure the integrity of the globe, the absence of an infection, and the absence of a foreign body. Document the visual acuity for both eyes.[21] Patients should receive tetanus prophylaxis if their immunizations are not up to date.[5] Apply cycloplegic drops and antibiotic ointment to the affected eye.[4,5] Obtain a CT scan of the orbits if a foreign body may have possibly penetrated the globe.[19,23]

TECHNIQUE

Instruct the patient to close both eyes and to keep them both shut for the remainder of the procedure. Tear off four to six strips of 1 inch tape, each 4 to 5 inches long. Obtain two cotton eye patches. Fold the first patch in half. Apply it to the affected eye while both eyes remain closed. Place the second unfolded patch over the first patch (**Figure 193-4*A***). Apply the pretorn strips of tape diagonally from the center of the forehead to the cheek just above the mandible (**Figure 193-4*B***).[4,5] Lift the lower cheek up slightly before securing



A B

FIGURE 193-4. Eye patching. ***A.*** A folded patch is applied over the eyelid then covered by an unfolded patch. ***B.*** The patch is secured with tape.

the first two strips of tape to ensure the correct amount of pressure.[4,5] The patch will hold the eye more securely if the nasal and temporal strips are placed in a slight arc concave toward the center of the eye.

Care must be taken to avoid taping the nasolabial folds, lips, facial hair, and the skin near the mandible.[4,5] Otherwise the mandibular movement of eating and speaking may cause the eye shield to move and potentially injure the eye. The tape may not stick if the patient has facial hair. The patient may benefit from a quick shave in the Emergency Department. Some patients require the use of an oxygen mask. Trim the oxygen mask around the eye patch to prevent it from displacing the eye patch and cause further eye injury.

SOFT CONTACT BANDAGE TECHNIQUE

Soft contact lenses can be applied as a corneal bandage for patients who require immediate use of both eyes.[28,29] Prepare the patient as described previously. **Apply ophthalmic antibiotic drops rather than ointment to the affected eye.** Remove the contact lens from the storage bottle. Rinse the storage solution from the contact lens. Place the contact lens on the index finger of the dominant hand. Use the nondominant hand to open the patient's eyelids. Instruct the patient to look straight ahead. Gently apply the contact lens directly to the cornea. The patient should remain in the Emergency Department for a recheck in 15 to 30 minutes to make sure that the contact lens fits properly and they can tolerate it.[29]

ALTERNATIVE TECHNIQUE

An alternative to the eye shield is a moisture chamber (Good-Lite Co., Elgin, IL). These foam-edge devices have a clear lens to allow the wearer to see through (**Figure 193-3**). These are not latex, fit over the eye, prevent air flow into the eye, and are comfortable to wear. They also avoid any issues related to taping. The plastic lens is scratch resistant.

ASSESSMENT

Refer to Chapter 185 on the examination of the eye for the complete details regarding the instillation of drops and ointment into the eyes. No further treatment is required for patients with small corneal abrasions. Make sure that the patient with an eye patch does not feel the lid of the injured eye moving when they blink the unaffected eye.[5] Ensure that the tape does not interfere with the patient's speech, chewing movements, or smiling.

AFTERCARE

Instruct the patient to remove the patch if it becomes loose. Eye patches should be changed or removed after 24 hours to reduce the risk of infection.[4,5] Most patients will need oral pain medications for the first 24 hours in addition to the patch.[5] Topical nonsteroidal anti-inflammatory ophthalmic drops have been useful alone or in conjunction with a contact lens bandage for pain relief.[14,29,32-34] The patient must be reexamined in 24 hours by an Ophthalmologist for assessment of the healing progress, to check for infection, and to check for other complications. The patient can expect complete resolution in 2 to 3 days. Instruct the patient not to climb, drive, or operate dangerous equipment due to the loss of binocular depth perception.[4,26] The elderly may need assistance with walking and stairs.[4] Discourage the patient from reading as this will cause involuntary movement of the patched eye.

COMPLICATIONS

Infection is rare but is most likely to occur in contact lens wearers or in patients who have used a patch for a prolonged period.[4,5,12] Patches applied too tightly can raise intraocular pressure high enough to cause retinal artery occlusion and lead to retinal ischemia, infarct, and blindness. Allergy to ophthalmic medications, the patch, or the tape materials can occur. Falls, auto accidents, and injuries due to lack of binocular depth perception can occur.[4,5] Healing times may be prolonged with loose patches that allow movement of the eyelid over the cornea.[4,5] The eyelashes may get caught between the eyelids and the cornea and further abrade the cornea.[4] Eye patching has become less commonly practiced due to the above complications. Contact lens bandages can cause infective ulcerative keratitis or corneal revascularization.[29]

SUMMARY

The eye is one of the most delicate and complex structures of the body. It is injured far more frequently than would be predicted based on its relative size.[35] Preservation of vision is essential to

maintaining quality of life. The cornea is one of the most sensitive organs of the body. Tiny injuries to the cornea can result in significant pain and loss of function. It is therefore essential that the Emergency Physician be skilled in vision-threatening eye injury management. The application of an eye patch or eye shield is a simple, rapid, and straightforward procedure. The improper application of these devices can result in significantly poor visual outcome.

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194 Acute Orbital Compartment Syndrome (Retrobulbar Hemorrhage) Management

Andrew F. Perin, Jamil D. Bayram, and Sami H. Uwaydat

INTRODUCTION

Acute orbital compartment syndrome is defined as an acute elevation of intraorbital pressure with resultant rapid ocular dysfunction. Patients typically present with ocular pain, proptosis, and blurry vision (**Figure 194-1**). Clinical signs of an acute orbital compartment syndrome include afferent pupillary defects, chemosis, decreased visual acuity, diminished retropulsion of the affected globe to direct manual pressure, elevated intraocular pressure (usually above 40 mmHg), exophthalmos or proptosis, mydriasis, ophthalmoplegia, and signs of retinal ischemia on funduscopic examinations (rare).[1-40]

Orbital compartment syndrome has been described in multiple clinical settings. The presentation that Emergency Physicians will most likely encounter is an acute posttraumatic retrobulbar hemorrhage leading to an orbital compartment syndrome with subsequent rapid loss of vision.[1,2] Orbital compartment syndrome has been documented following burns, aggressive fluid administration, blepharoplasty, retrobulbar anesthesia, orbital and sinus surgery, orbital fractures with intraorbital emphysema, spontaneous subperiosteal hemorrhages, and spontaneous retrobulbar hemorrhages.[3-9,36] Orbital compartment syndrome may also occur as the result of chronic and progressive disease processes (e.g., neoplasms, infections, inflammations).[10]

Acute orbital compartment syndrome demands prompt recognition because irreversible loss of vision (even permanent blindness) occurs without rapid treatment.[11] Once the diagnosis of an acute orbital compartment syndrome is made, emergent surgical intervention is indicated. An immediate lateral canthotomy and cantholysis are indicated within 1 hour of injury and ocular dysfunction.[32,40] Medical interventions aimed at reducing intraocular pressure (e.g., mannitol, acetazolamide, topical beta-blockers) should be considered adjunctive therapy and not a substitute for surgical intervention.

A

B

FIGURE 194-1. A traumatic retrobulbar hematoma with exophthalmos of the right eye. ***A.*** Clinical photo (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine,* 4th ed. New York: McGraw-Hill; 2015. Photo contributor: David Effron, MD.) ***B.*** Computed tomography (CT) scan. (Used with permission from Li Y, et al: Intraorbital traumatic ophthalmic artery aneurysm: case report. *Neurol India* 2012; 60(6):657.)

ANATOMY AND PATHOPHYSIOLOGY

The orbit is a closed space posterior to the orbital septum and contained within the bony orbit. The lateral wall of the orbit is formed by the zygomatic bone. The posterior wall is formed by the sphenoid bone. The medial wall is formed by the ethmoid bone. The roof is formed by the frontal bone. The floor is formed by the maxillary bone. The globe is enclosed in a fascial envelope within the bony orbit.

The medial and lateral canthal tendons provide structural fixation of the eyelids to the orbital rim. The lateral canthal tendon (LCT) is located posterior and inferior to the lateral canthal fold (**Figure 194-2**). The LCT originates from the superior and inferior lateral tarsal plates (**Figure 194-2**) and attaches to the lateral orbital tubercle of the zygoma (**Figures 194-2 and 194-3**). The LCT is Y-shaped and consists of a superior crus from the superior tarsal plate and an inferior crus from the inferior tarsal plate. The LCT measures 10.6 mm (standard deviation [SD] ± 0.9 mm) in length from its attachment site to the lateral canthal angle. It is 10.2 mm

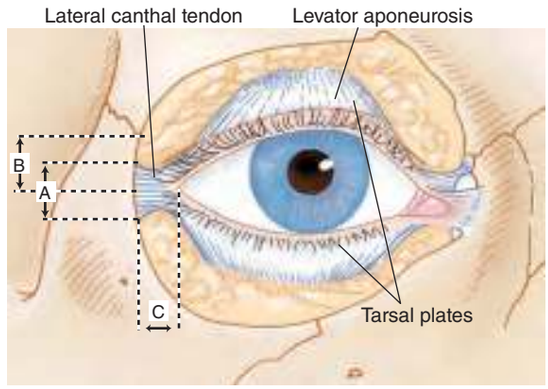

FIGURE 194-2. The lateral canthal tendon or LCT (*A* = the vertical height of the LCT, *B* = the distance from the frontozygomatic suture to the midpoint of the LCT origin, *C* = the length of the LCT as measured from the canthal margin to its origin).

(SD ± 0.8 mm) in width at its origin at the lateral ends of the tarsal plates. It attaches 1.5 mm (SD ± 0.3 mm) behind the orbital margin and approximately 9.7 mm (SD ± 0.8 mm) below the frontozygomatic suture at the lateral orbital tubercle.[12] Immediately anterior to the LCT is Eisler's pocket, a collection of adipose tissue. Posterior to the LCT, at its attachment site to the lateral orbital tubercle, is the check ligament of the lateral rectus muscle (**Figure 194-3**). The levator aponeurosis of the upper eyelid and Lockwood's suspensory ligament of the lower eyelid also attach at the lateral orbital tubercle.

Any increase in intraorbital contents (e.g., retrobulbar hematoma, intraorbital emphysema, retrobulbar abscess) will result in an elevation of intraorbital pressure because the orbit is a closed space. Intraocular pressure elevation correlates with the degree of intraorbital pressure elevation.[13] The globe itself may partially accommodate some of the elevation in intraorbital pressure by prolapsing forward (**Figure 194-4**). This will result in ocular pain and proptosis. The intraorbital pressure rises dramatically as the orbit approaches maximal distention. This rise in intraorbital pressure leads to chemosis, elevated intraocular pressure, and compression of the intraorbital cranial nerves. If the compression is severe enough, the patient develops an ophthalmoplegia and an afferent pupillary defect.

The exact mechanisms by which orbital compartment syndromes result in blindness remain speculative.[14-25] A common theory is that as the intraorbital pressure increases, orbital venous outflow is impeded and leads to diminished retinal and optic nerve arterial perfusion pressures. This results in an afferent pupillary defect, diminished visual acuity, and rarely funduscopic signs of retinal ischemia. Over time, the elevated intraorbital pressure leads to irreversible optic nerve and/or retinal ischemia. Experimental studies have demonstrated that irreversible ischemic injury to the retina may occur within 90 minutes of vascular insufficiency.[19-22,31,32,40] Additional theories suggest that direct mechanical compression or longitudinal traction on the optic nerve may contribute to the loss of vision in orbital compartment syndromes.[23,25] Elevated intraorbital pressure may also occur from large-volume fluid resuscitation in patients without ocular trauma.[30]

A history of progressive loss of vision following orbital trauma suggests a reversible disease process (e.g., retrobulbar hemorrhage). If loss of vision is immediate and complete following orbital trauma, the chance of recovery of vision is poor.[26] This is because the immediate loss of vision after trauma is due to direct optic nerve, retinal, or vascular injury rather than an orbital compartment syndrome.

Ultrasound (US) can be used to diagnose a retrobulbar hemorrhage after trauma.[37,38] US allows quick evaluation of the orbit for a

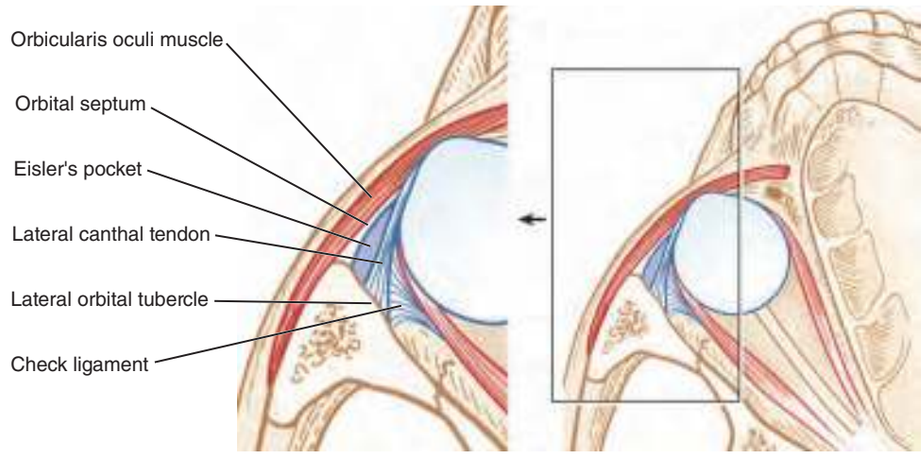

FIGURE 194-3. An axial view of the orbital contents. Identification of the LCT will require dissection of the conjunctiva and fascial tissues with a hemostat and iris scissors. A pocket of adipose tissue (Eisler's pocket) will be encountered beneath the superficial tissue layers. The LCT lies just posterior to this adipose tissue collection.

retrobulbar hemorrhage. The patient's closed eye should be covered with Tegaderm to prevent irritation from the US gel. Use a large amount of gel so the transducer is not against the globe. Use a linear array transducer at a frequency of 7.5 to 10 MHz. The hemorrhage is anechoic or hypoechoic behind the globe. The hemorrhage is confirmed by computed tomography scan if there is time before the procedure. **An US should not be performed if one suspects or there is an actual ruptured globe.**

INDICATIONS

An acute orbital compartment syndrome is an indication for immediate orbital decompression.[24] Multiple case series have documented the efficacy of immediate orbital decompression in restoring visual acuity to affected patients.[25-29]

CONTRAINDICATIONS

Except for a ruptured globe, there are no definite contraindications to performing this procedure, as permanent loss of vision may result from untreated acute orbital compartment syndromes. The patient's airway, breathing, circulation, and any life-threatening injuries must be addressed prior to performing this procedure. If the patient is very young, confused, or uncooperative, sedation and restraint or procedural sedation (Chapter 159) will be required to prevent iatrogenic injury to the globe.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Topical ocular anesthetic solution, proparacaine or tetracaine
- Lidocaine 2% with epinephrine
- 30 gauge 0.5 inch needle on a 3 mL Luer-lock syringe
- Ocular tonometer (e.g., applanation, Schiøtz, or Tono-Pen)
- Sterile saline or sterile water
- Straight mosquito hemostat
- Iris scissors
- Tissue forceps
- 2×2 gauze squares

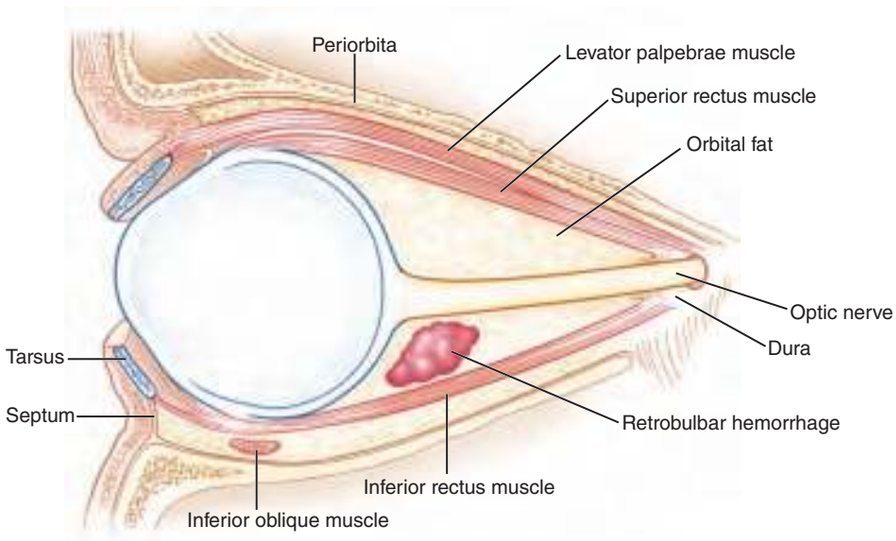

FIGURE 194-4. A sagittal view of the orbital contents. Note the location of the retrobulbar hemorrhage.

- Topical ocular antibiotic ointment (e.g., bacitracin, ciprofloxacin, erythromycin, gentamicin, Neosporin, Polysporin, or sulfacetamide)
- #10 disposable scalpel (optional)
- Disposable hot tip cautery pen (optional)
- Desmarres retractor (optional)
- US machine (optional)
- US gel (optional)
- US transducer, linear array of 7.5 to 10 MHz (optional)
- Tegaderm (optional)

PATIENT PREPARATION

Explain the procedure to the patient and/or their representative including the risks, benefits, and outcome if it is not performed. This is a painful procedure for the awake and alert patient. Lucid patients will require parenteral medications for analgesia and sedation in addition to local anesthetics. Consider the use of procedural sedation (Chapter 159) if not contraindicated. Measure and record the visual acuity. Perform a brief ophthalmologic examination. Apply topical ocular anesthetic solution to the conjunctiva of the affected eye. Measure and record the intraocular pressure.

TECHNIQUE

Place the patient supine. Clean the eyelids and surrounding skin of any blood, dirt, and debris. Apply povidone iodine solution to the eyelids and allow it to dry. If a patient reports iodine allergy, chlorhexidine solution can be used with care to ensure it does not contact the ocular surface. **Do not let these solutions drip into the eye.** Irrigate the lateral canthal fold region with sterile saline or sterile water. Using sterile technique, identify the lateral canthal fold (**Figure 194-5**). Inject 1 mL of local anesthetic solution with epinephrine subcutaneously using a 30 gauge, 0.5 inch long needle on a 3 mL syringe along the lateral canthal fold. **The goal is to anesthetize the tissue extending laterally from the canthal fold to the orbital rim. Caution must be exercised to avoid inadvertent needle puncture of the globe.**

Insert a straight mosquito hemostat at the lateral canthal fold. Place one jaw of the hemostat anterior and one jaw posterior to the lateral canthal fold. Advance the tips of the hemostat laterally until the orbital rim is encountered. Clamp and compress the intervening tissue for approximately 1 minute. This will minimize any bleeding precipitated by the lateral canthotomy. Remove the hemostat. Cut all the tissue layers along the lateral canthal fold up to the orbital rim (lateral canthotomy) with an iris scissors (**Figure 194-5**). **All tissue layers, from the skin to conjunctiva, must be incised down to the orbital rim.** Use a #10 scalpel blade to cut the tissues in cases of severe distortion of the anatomy due to edema or if iris scissors are not readily available. A disposable hot cautery pen, if available, can be used to achieve hemostasis. In the setting of significant periorbital and eyelid edema, an assistant may use a Desmarres retractor to elevate the upper eyelid or separate the lids to improve exposure of the lateral canthus.

Gently grasp the cut end of the lower eyelid with a hemostat or forceps and retract it outward away from the globe. Identify the lateral canthal tendon located just posterior and inferior to the lateral canthal fold (**Figures 194-2 and 194-3**). The LCT can be easily identified by strumming it with the end of a closed scissors. Dissect the conjunctiva and fascial tissues with a hemostat or iris scissors. A pocket of adipose tissue (Eisler's pocket) will be encountered beneath the superficial fascial planes (**Figure 194-3**). The lateral canthal tendon lies just posterior to this adipose tissue collection (**Figure 194-3**). Completely divide the lateral canthal tendon vertically at its midportion with an iris scissors or #10 scalpel (lateral cantholysis) to transect the inferior crus of the tendon. If outward tension is kept on the lower lid with forceps during division of the LCT, there should be a notable release with successful completion of the cantholysis and the lid should separate from the globe. If unable to feel release in tension of the lid, then it is likely that only a partial cantholysis was achieved.

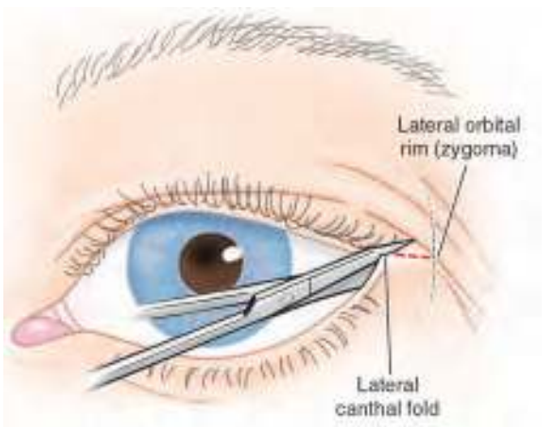

FIGURE 194-5. Illustration of lateral canthotomy. Iris scissors are used to cut all tissue layers along the lateral canthal fold up to the orbital rim.

ASSESSMENT

A successful lateral canthotomy and cantholysis will cause an immediate decrease in intraocular pressure to less than 40 mmHg. Recheck the intraocular pressure. **If the intraocular pressure remains elevated, reexplore the lateral canthal tendon region to make sure that the inferior crus has been completely transected. If transected, cut the superior crus of the tendon to transect it.**[34,35] Occasionally, the intraorbital and intraocular pressures will remain elevated despite successful cantholysis. These refractory cases necessitate emergent decompression of the deep orbital wall. Such decompressions call for operative techniques that are to be performed by Ophthalmologists and Otolaryngologists.

The resolution of proptosis and afferent pupillary defects and restoration of visual acuity will usually not occur immediately.[39] Patients who respond to surgical intervention will gradually regain their visual acuity over a period of hours to days.

AFTERCARE

Apply a topical antibiotic ointment along the canthotomy site. In orbital compartment syndromes, elevated intraocular pressures merely reflect elevated intraorbital pressures. Therefore, any attempts to decrease intraocular pressures will not reliably reduce retrobulbar optic nerve compression. Medical interventions decreasing intraocular pressure may be used following, or in conjunction with, a lateral canthotomy and cantholysis. These interventions are similar to those employed for the management of elevated intraocular pressures in patients with acute angle-closure glaucoma. This includes the use of topical beta-blockers, central acting alpha agonists, intravenous mannitol, and carbonic anhydrase inhibitors**. These medications are not a substitute for surgical orbital decompression.**

Many patients with an orbital compartment syndrome have other injuries requiring hospital admission following their

Emergency Department evaluation and treatment. Ensure a timely Ophthalmologic consultation for a complete eye exam and possible repair of the canthotomy and cantholysis sites once the orbital compartment syndrome resolves. Repair of the lateral canthal fold and lateral canthal tendon is controversial. Many Ophthalmologists do not advocate suture repair as a majority of wounds heal without complication by secondary intention. Patients with no other acute medical or surgical conditions, restored vision, a normal ophthalmologic examination, and normal postprocedural intraocular pressures may be discharged after evaluation by an Ophthalmologist.

COMPLICATIONS

Time constraints, abnormal anatomy as a result from traumatized tissue, and lack of familiarity with the lateral canthotomy and cantholysis techniques can make this a challenging procedure. Hemorrhage is often minimal and can be controlled with direct pressure. The use of a disposable cautery pen can be helpful. Mechanical injuries can include globe perforation, injury of the lateral rectus muscle, scleral lacerations, and secondary ectropion. Most of these complications can be prevented by knowing and identifying the anatomic landmarks, reviewing the procedure before it is performed, and using extreme care in performing the canthotomy and cantholysis. Despite the use of sterile technique, infections at the canthotomy and cantholysis sites can occur. They should be treated with parenteral antibiotics that cover typical skin flora.

SUMMARY

Emergency Physicians must be able to promptly recognize and be prepared to manage an acute orbital compartment syndrome defined by an acute elevation of intraorbital pressure with resultant ocular dysfunction. Patients will present with ocular pain, proptosis, an afferent pupillary defect, and diminished visual acuity. An acute orbital compartment syndrome requires emergent treatment to preserve vision. An immediate lateral canthotomy and cantholysis are indicated, preferably within 1 hour of injury and ocular dysfunction. Medical management should be considered an adjunctive therapy. Many patients will regain their visual function if the procedure is performed soon after the injury and before permanent ischemic changes occur.

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195 Globe Luxation Reduction

Briana D. Mazur, Adam Jennings, and Heidi Knowles

INTRODUCTION

Luxation of the globe is a rare event whereby the eyelids slip behind the midcoronal plane of the eye in an extremely proptotic eye (**Figures 195-1 and 195-2**). Subsequent spasm of the orbicularis oculi muscle maintains the luxation of the globe. Extraocular eye movements become severely limited. The optic nerve and retinal vasculature are subjected to an abnormal amount of traction which results in possible damage to these structures or the retina.[1] **This can result in partial or full blindness of the affected eye if it is not reduced before onset of irreversible ischemic changes.**

ANATOMY AND PATHOPHYSIOLOGY

There are three major causes of globe luxation: spontaneous, voluntary, and traumatic. Spontaneous luxation tends to occur in individuals with shallow orbits (**Figure 195-1*A***).[2] Structural abnormalities

A

B

C

FIGURE 195-1. Globe luxations. ***A.*** Spontaneous luxation. Note the eyelids behind the midcoronal plane of the eye and the lacrimal gland in front of the eyelids. (Used with permission from www.clinicalgate.com.) ***B.*** Voluntary luxation. ***C.*** Luxation associated with forceps delivery. (Used with permission from reference 16.)

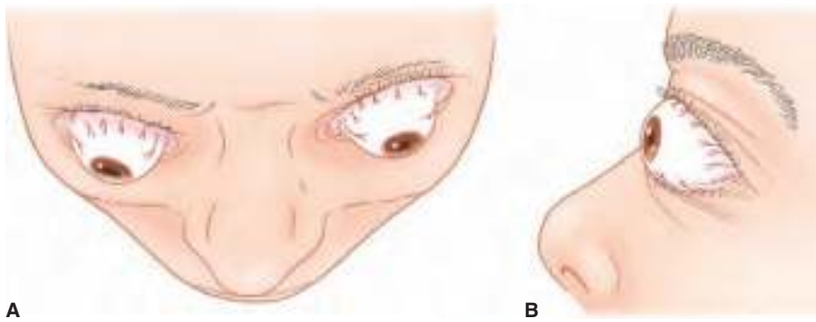

FIGURE 195-2. The luxated globe. ***A.*** Superior view. ***B.*** Lateral view.

(e.g., anomalous extraocular muscles, laxity of the supporting fascia, and laxity of the supporting muscles) can predispose to spontaneous luxation.[2-4] Pathologic processes that can cause proptosis can predispose to luxation. The literature documents cases of luxation associated with cerebral gummas, chronic obstructive pulmonary disease, craniofacial dysostosis, Cushing's syndrome, floppy eyelid syndrome, Graves' disease, histiocytosis X, and orbital tumors.[1,5-9] Voluntary luxation occurs in individuals who learn to cause globe propulsion by using a digit or their extraocular muscles (**Figure 195-1*B***). Some patients use a Valsalva maneuver to luxate their globe(s) voluntarily.

Traumatic luxation results from injury to the globe or the surrounding bony orbit.[10,11] It can occur from motor vehicle accidents or relatively minor trauma to the face.[12,13] Traumatic luxation can be associated with complete avulsion of the optic nerve. Traumatic luxation can occur from intentional eye gouging or during the forceps-assisted delivery of a neonate (**Figure 195-1*C***).[14-16]

The normal anatomic relationship of the globe to the surrounding structures is seen in **Figure 195-3**. The midcoronal plane of the globe is through the widest portion of the eye and divides it into anterior and posterior halves. The eyelids get behind the midcoronal plane of subluxed globes, the orbicularis oculi muscle is pulled taut, and the orbicularis oculi muscle begins to go into spasm. This spasm prevents spontaneous reduction of the eye.

INDICATIONS

Globe reduction is indicated to relieve traction on the optic nerve and retinal vessels. The visual acuity may be compromised without prompt reduction. Sustained globe luxation is physically and psychologically uncomfortable for the patient, may result in permanent loss of vision, and is often difficult to reduce without general anesthesia. **Attempt to reduce all globe subluxations if an Ophthalmologist is not immediately available.**[17]

CONTRAINDICATIONS

Rupture of the globe and extensive orbital fractures that require immediate surgical intervention are relative contraindications to globe reduction. Edema and retrobulbar hemorrhage can make reduction outside the Operating Room difficult or impossible.[1,9] Consult an Ophthalmologist for globe subluxations that cannot be reduced, when procedural sedation is contraindicated, or when the patient is uncooperative.

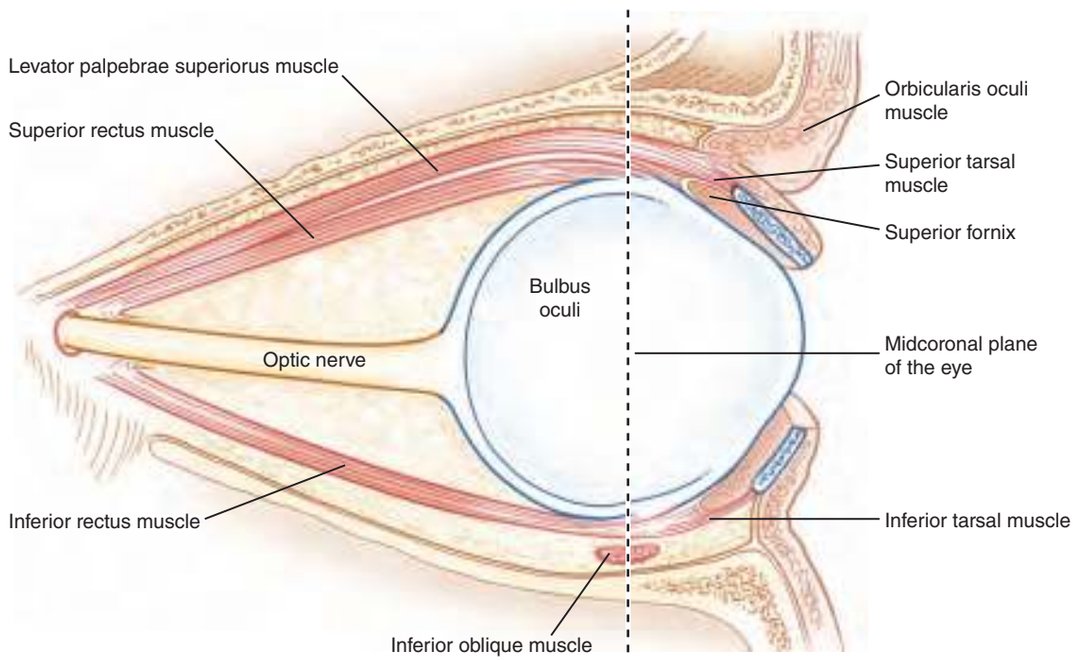

FIGURE 195-3. Anatomy of the eye and orbit.

EQUIPMENT

- Topical ocular anesthetic agent (e.g., 0.5% proparacaine or tetracaine)
- Sterile gauze and gloves
- Sutures or eyelid retractors
- Local anesthetic solution if eyelid retaining sutures need to be placed
- 3 mL syringe
- 27 gauge needle

PATIENT PREPARATION

Perform a directed eye examination (Chapter 185) addressing the integrity of the globe, visual acuity, pupillary reactivity, and range of ocular motion prior to any attempt at reduction. Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Describe the procedure and answer any questions. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Relaxation of the patient and the orbicularis muscles is essential for procedural success. The use of a parenteral analgesic, parenteral anxiolytic, or procedural sedation (Chapter 159) is recommended if not contraindicated.[18] Place the patient supine. Instill a topical ocular anesthetic agent onto the affected eye. Allow 1 to 2 minutes for the anesthetic to take full effect.

TECHNIQUE

Reduction of a globe luxation ideally requires two people (**Figure 195-4**).[18,19] Instruct an assistant to apply steady upward and outward traction on the upper eyelid and downward and outward traction on the lower eyelid by grasping and pulling on the eyelashes. Instruct the patient to maintain a constant downward gaze. The downward gaze posture negates the retracting action of the levator palpebrae superioris muscle and simultaneously relaxes the superior rectus muscle.[19] Apply steady and gentle pressure with a gloved thumb to the exposed superior sclera of the globe in a simultaneously downward and posterior direction while traction is being applied to the eyelids. Physical contact over the sclera minimizes discomfort and avoids possible corneal abrasions.[19] Continue to apply constant and gentle pressure until the globe is manipulated back into the orbit.[1,9]

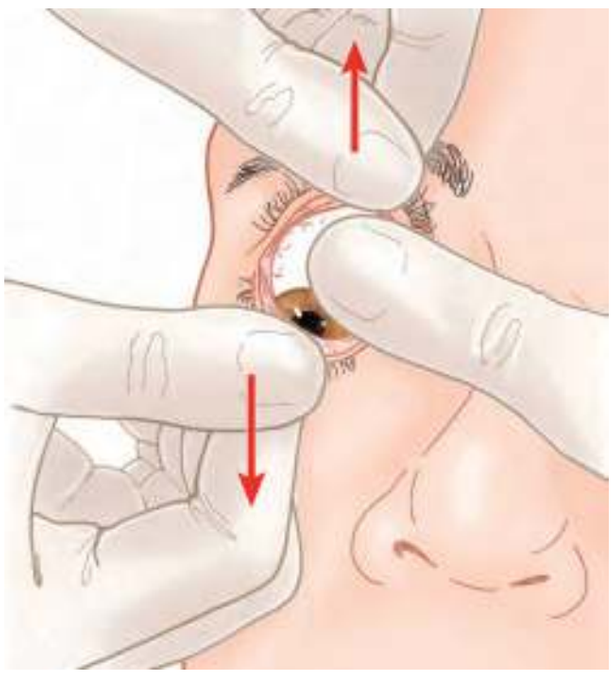

FIGURE 195-4. Reduction of a globe luxation.

ALTERNATIVE TECHNIQUE

The patient's eyelashes are occasionally small or not accessible, or an assistant is not available. Place an eyelid retractor behind the eyelids to provide countertraction. Other authors have recommended the placement of a suture through the anesthetized skin of the eyelids to help retract them.[1,4,9] **Extreme care must be taken to prevent penetration of the globe by the anesthetic needle or the suture needle if retaining stitches are placed in the eyelids.** The globe is then reduced in the same manner as described previously.

ASSESSMENT

A repeat and complete eye examination must be performed after the procedure and documented in the medical record. Pay special attention to visual acuity, pupillary reflexes, and range of extraocular muscle movement.[1,9,15] Full visual acuity may not return to baseline for several days or longer.[4] Initiate a search for the possible causes of a nontraumatic luxation in the Emergency Department.[1,5,8,9] This includes but is not limited to thyroid function testing and orbital imaging to rule out a tumor. Perform this evaluation after consultation with an Ophthalmologist. Conduct a search for associated injuries (e.g., bony periorbital fracture, globe rupture, intracranial injury, or a retro-orbital hematoma) in traumatic luxations.

AFTERCARE

Discharge patients with spontaneous luxations that reduce without difficulty and who are without visual impairment after consultation with an Ophthalmologist. These patients require follow-up with the Ophthalmologist within 24 hours.[1,9] Instruct the patient to avoid Valsalva maneuvers and any triggering actions (e.g., aggressive eyelid manipulation while inserting contact lens).[1] All patients with traumatic luxation require emergent Ophthalmologic consultation and imaging of the orbits.[1,9] Treat any corneal abrasions.

COMPLICATIONS

It is common for eyelashes to be retained in the conjunctival fornices after the reduction.[15] Perform a thorough evaluation and remove any free eyelashes to prevent corneal abrasions or eye injury.[1,9,15] Instill saline drops to the eye and apply an eye shield (Chapter 193) to prevent further injury while an emergent Ophthalmologic consult is obtained if one or two attempts at reduction are not successful.[1,9] Reducing an open globe (i.e., when known or unknown) can expel the eye contents and make vision loss permanent. **Always perform an eye examination before reducing any luxation. Place an eye shield and consult an Ophthalmologist if there is any question of a globe perforation.**

SUMMARY

Globe luxation is a rare entity that can be effectively dealt with in the Emergency Department. Prompt intervention by an Emergency Physician can result in the preservation of visual acuity. Perform a brief initial eye examination. Uncomplicated cases can be reduced

by the Emergency Physician. Complicating factors (e.g., the presence of trauma, an open globe injury, or the inability to reduce the globe) require an emergent Ophthalmologic consultation. Investigate any predisposing factors for a spontaneous luxation.

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196 Hordeolum (Stye) Incision and Drainage

Sami H. Uwaydat, Andrew F. Perin, and Jamil D. Bayram

INTRODUCTION

A hordeolum is a suppurative infection of one of the eyelid glands.[1-15] The nomenclature that describes infectious and inflammatory conditions of the eyelid glands is at times confusing. A brief description of the eyelid margin anatomy may help resolve some of the confusion. The patient usually presents with an acutely painful, erythematous, localized, and tender mass on either the upper or lower eyelid. A hordeolum may be associated with a blepharitis.

ANATOMY AND PATHOPHYSIOLOGY

The eyelid is composed of numerous structures (**Figure 196-1**). The eyelid skin is thin and vascular. The orbicularis oculi muscle encircles the eyelids and controls their movements. The hair follicles that form the eyelashes are fine and delicate in comparison to hair on other body areas. Numerous glands are contained within the eyelids. The glands of Zeis are modified sebaceous glands associated with the hair follicles. The glands of Moll are modified sweat glands that open into the base of the hair follicles. The tarsus is a rigid fibrous plate containing the sebaceous Meibomian glands. The orifice of the elongated Meibomian glands can be seen posterior to the eyelashes at the eyelid margin. The thin and delicate conjunctiva lines the inner aspect of the eyelids.

A hordeolum can be located internally or externally in relation to the eyelid and tarsal plate. It is essentially an abscess of the eyelid. An internal hordeolum is a bacterial infection of the Meibomian gland and usually points to the inside or conjunctiva (**Figure 196-2**). An internal hordeolum is usually larger than an external hordeolum. If the infection blocks the neck of the Meibomian gland, the infection points toward the conjunctival surface of the eyelid. If the neck of the Meibomian gland is not blocked, the infection often points to the eyelid margin. An external hordeolum, also known as a stye, is a bacterial infection of the glands of Zeis or Moll. These tend to be small, superficial, and point to the eyelid skin or, more commonly, the eyelid margin. The most common causative agent in both internal and external hordeola is *Staphylococcus aureus*.[1] Other infectious agents and conditions can be the etiology.[15]

Patients with a hordeolum almost always present with localized pain, redness, and swelling of the eyelid margin. It is usually a benign process but can progress to a preseptal or septal cellulitis. It may rarely result in a corneal epithelial defect,[2] periorbital necrotizing fasciitis,[3] and bacteremia.[4]

A chalazion is a chronic (lasting > 2 weeks) granulomatous inflammation of a sebaceous Meibomian gland. Acute chalazion lesions, those less than 2 weeks old, are often difficult to distinguish from an acute hordeolum. Clinically, the management of an acute chalazion and an acute hordeolum are the same and it is not necessary to differentiate the two processes. A chalazion may persist for many months and slowly enlarge over time. A chronic chalazion is characterized by a painless localized swelling of the eyelid margin with no inflammatory signs. It feels rubbery upon palpation. A chronic chalazion does not require an urgent intervention and should be managed by an Ophthalmologist. A chalazion that is acute, large, or causes local irritation may require incision and drainage. Recurrent chalazia require an evaluation by an Ophthalmologist to rule out a malignancy.

MANAGEMENT

Hordeola are initially managed conservatively.[13] Warm compresses should be applied over the eyelid for 10 to 15 minutes at a time three to six times per day. An alternative and portable solution to warm compresses would be to place 8 to 10 ounces of dry rice in a sock and heat it in a microwave (typically for 30 seconds). The rice tends to maintain the heat for a longer period compared to the warm compresses. Apply diluted baby shampoo to the eyelid margins with a washcloth or cotton-tipped applicator for daily eyelid scrubs. Topical antibiotic ointments (e.g., erythromycin) can be placed in the conjunctiva or on the eyelid margin three times per day. The purpose of topical antibiotics is to prevent the infection from spreading to adjacent hair follicles. Oral antibiotics are indicated only if the signs and symptoms of a cellulitis develop. Most hordeola will spontaneously drain and resolve within 5 to 7 days with conservative management. Treat any accompanying blepharitis to prevent the formation of additional hordeola.

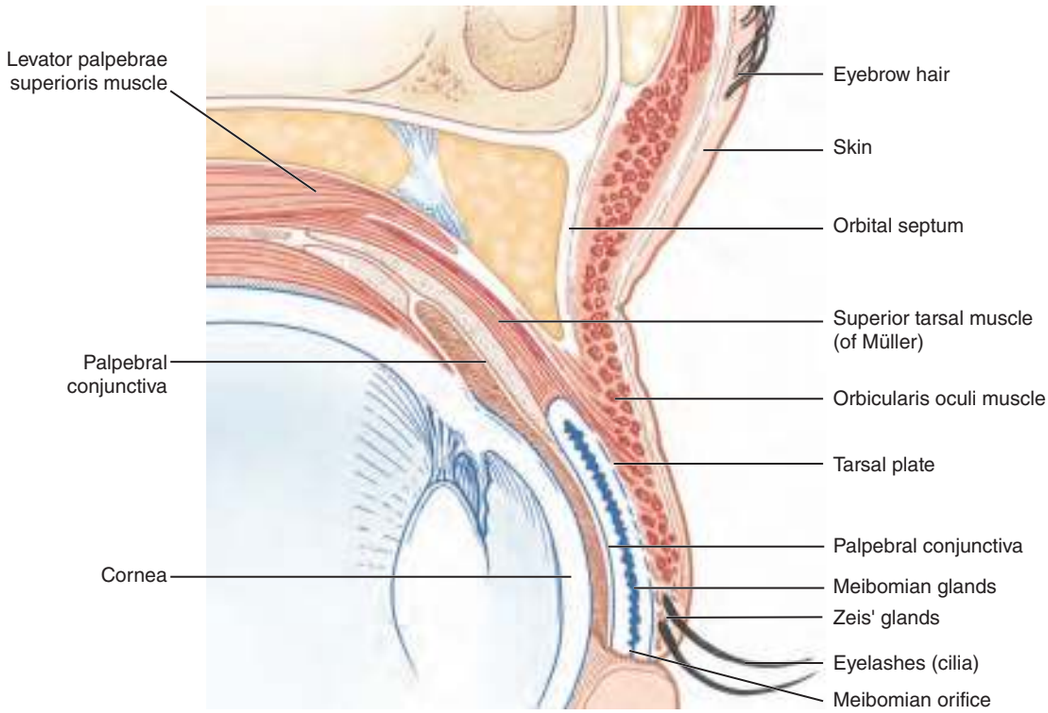

FIGURE 196-1. Eyelid margin anatomy. The type and location of the infected gland determines whether it is an internal hordeolum or an external hordeolum (stye).

INDICATION

Surgical excision is warranted if the hordeolum does not resolve with conservative management and the patient has significant discomfort. A hordeolum can be excised if it causes a cosmetic deformity, blocks the visual axis, or is of a significant size.

CONTRAINDICATIONS

Not all hordeola should be incised and drained in the Emergency Department. Hordeola close to the lacrimal puncti or the medial canthus are best managed conservatively until surgery can be coordinated with an Oculoplastic surgeon. Drainage in patients on blood thinners and in those who are unable to follow commands is contraindicated. Sedation is required for children, the confused patient, and the uncooperative patient. Consult an Ophthalmologist prior to sedating a patient to incise and drain a hordeolum. A recurrent hordeolum should be referred to an Ophthalmologist for biopsy and evaluation of the lesion for a malignancy.[14] Consult an Ophthalmologist prior to the procedure if the hordeolum affects visual acuity or ocular movements.

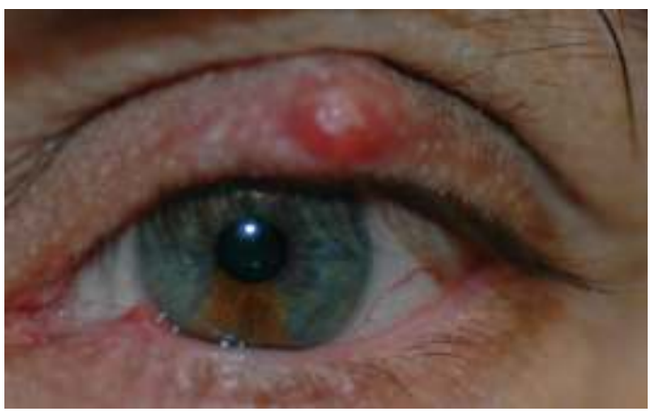

FIGURE 196-2. An internal hordeolum with redness and swelling.

EQUIPMENT

- Buffered local anesthetic solution (e.g., lidocaine with epinephrine and $NaHCO_3$)
- 30 gauge, 0.5 inch needle on a 3 mL syringe
- 0.5% proparacaine drops
- Povidone iodine or chlorhexidine swabs
- Small drape with central opening
- Sterile 4×4 gauze squares
- Sterile cotton-tipped applicators
- Sterile marking pen
- Corneal Eye Shield or Morgan lens (Chapter 187, Mortan Inc., Missoula, MT)
- #11 or #15 scalpel blade on a handle
- Westcott scissors
- Desmarres chalazion clamps, multiple sizes
- Meyerhoefer chalazion curettes, multiple sizes
- Castro-Viejo forceps
- Low-temperature disposable cautery unit
- Topical ophthalmic antibiotic ointment without steroids (e.g., erythromycin)
- Eye pad
- Culture swabs
- 1-inch tape

A chalazion clamp is a device specifically designed to aid in the incision and drainage of a chalazion or hordeolum (**Figure 196-3**).

FIGURE 196-3. The chalazion clamp.

It is similar to a forceps except the distal ends are expanded and it has a clamp mechanism. One of the distal ends is an open circle used to surround the hordeolum. The other distal end is flat, solid, and acts to prevent injury to underlying structures. Just proximal to the distal expanded ends is a screw mechanism that allows the distal ends to be closed and clamped securely in place.

PATIENT PREPARATION

Perform a brief ophthalmic examination including visual acuity, motility, and an anterior segment assessment. Document the size and location of the hordeolum. Explain the procedure, its risks, and benefits to the patient and/or their representative. Place a signed informed consent in the medical record. Many patients are apprehensive when a surgical procedure is performed close to their eye. It is imperative that the patient remains still during this procedure given that sharp instruments will be exchanged over the globe. A thorough explanation of the surgical steps should alleviate some of the patient's fears. Consider the use of parenteral sedation or procedural sedation (Chapter 159).

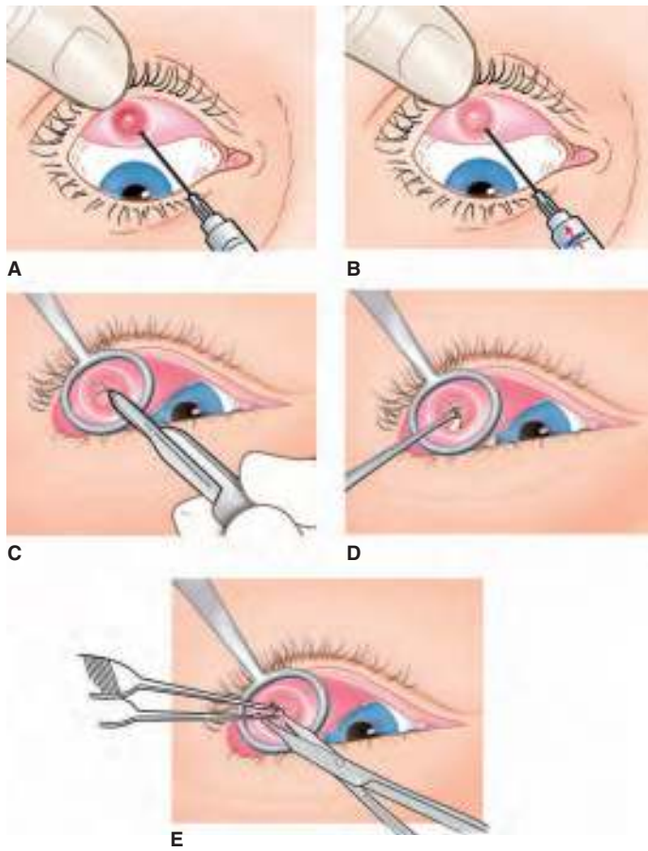

FIGURE 196-4. Incision and drainage of a hordeolum. *A.* The hordeolum is identified and a circle is drawn around its margins. A needle is inserted to inject local anesthetic solution. *B.* Local anesthetic solution is injected into and around the hordeolum. *C.* A chalazion clamp is applied and an incision is made over the hordeolum. *D.* Purulent material is expressed from the incision using a chalazion curette. *E.* Inflammatory tissue is removed.

Place the patient supine. Support their head with a headrest or a foam doughnut. Place one drop of proparacaine in the cul-de-sac of the affected eye. Mark the circumference of the hordeolum with a sterile skin marker (**Figure 196-4*A***). This is important as the injection of local anesthetic solution will obscure the margins of the hordeolum. Gently wipe the eyelid skin with an alcohol swab. **Do not allow the alcohol to get onto the eye.** Clean the skin of the eyelid with povidone iodine or chlorhexidine solution. **Do not allow these solutions to get onto the eye.** If any solution does get onto the eye, irrigate the eye copiously with normal saline. Arm a 30 gauge needle onto a 3 mL syringe. Draw up 1 mL of buffered local anesthetic solution with epinephrine. Inject the solution around and into the hordeolum (**Figures 196-4*A* and 196-4*B***). **Direct the syringe tangential to the skin (for an external hordeolum) or conjunctiva (for an internal hordeolum) so as to avoid inadvertent penetration of the globe.** Place a corneal shield or Morgan lens (Chapter 187), if available, over the cornea.

TECHNIQUES

INTERNAL HORDEOLUM

Cleanse and anesthetize the area as described above. Apply a chalazion clamp to the eyelid. Place the loop of the clamp on the

conjunctival surface of the eyelid and encompassing the marked edges of the hordeolum (**Figure 196-4*C***). Place the plate of the clamp on the skin surface of the eyelid. Gently close the clamp by turning the screw mechanism. Gently flip the clamp to expose the hordeolum. Make a superficial cruciate incision in the hordeolum with a #11 or #15 scalpel blade (**Figures 196-4*C* and 196-4*D***). Gently insert the chalazion curette to scoop out the purulent material (**Figure 196-4*D***). Alternatively, use a sterile cotton-tipped applicator. Grasp the edge of the inflammatory tissue with the Castro-Viejo forceps. Carefully use Westcott scissors to dissect the planes between the hordeolum and the tarsus (**Figure 196-4*E***). Release the chalazion clamp and gently remove it from the eyelid. Bleeding may occur as the clamp is released. Careful application of low-temperature cautery can be used to achieve hemostasis. Alternatively, apply gentle pressure with a gauze square to the incision to control any bleeding. **Be careful to not touch the eye with the gauze and cause a corneal abrasion.** Culture the expressed purulence. Place ophthalmic erythromycin ointment in the cul-de-sac. Apply a pressure patch over the eye (Chapter 193).

EXTERNAL HORDEOLUM

Cleanse and anesthetize the area as described above. Make an incision over the hordeolum with a #11 or #15 scalpel blade. **Do not cut the eyelid margin or the underlying tarsus.** Gently roll a sterile cotton-tipped applicator over the eyelid skin and toward the incision to express the purulent material. Apply gentle pressure with a gauze square to the incision to control any bleeding. **Be careful to not touch the eye with the gauze and cause a corneal abrasion.** Culture the expressed purulence. Place ophthalmic erythromycin ointment on the eyelid skin. Apply a pressure patch over the eye (Chapter 193).

ASSESSMENT

Palpate the eyelid at the end of the procedure. If a residual pus collection is detected, attempt to express the pus with a cotton-tipped applicator. If this fails, a second incision parallel to the first one is required to completely drain the hordeolum. **Do not undermine the delicate eyelid tissues to express the purulent material.** While not required, some physicians may perform a repeat fluorescein examination to ensure that they did not cause a corneal abrasion during the procedure.

AFTERCARE

The follow-up instructions are simple and straightforward. Instruct the patient to remove the pressure patch in 1 to 2 hours. It was placed to minimize any edema and bleeding from the procedure. If significant bleeding is noted when the patch is removed, the patient should immediately return to the Emergency Department. They should apply a warm compress (or warm rice in a sock) to the eyelid every 3 to 4 hours for the first 24 hours. An eyelid skin incision should remain clean and dry for 24 hours. Prescribe topical ophthalmic antibiotic ointment (e.g., erythromycin if no contraindications exist) to be applied on the skin incision or in the cul-de-sac three times a day for 3 to 4 days. Someone must follow-up on the cultures results in 12 to 24 hours. If methicillin-resistant *S. aureus* (MRSA) is detected, the patient must be notified and they require follow-up with an Ophthalmologist within 24 hours or return to the Emergency Department for the consideration of oral antibiotics. Treat any associated blepharitis to prevent the formation of a new hordeolum.

The patient should follow up with an Ophthalmologist within 5 to 7 days for an evaluation of the eyelid. They need to be evaluated sooner if the cultures are positive for MRSA. They should immediately be seen by an Ophthalmologist or return to the Emergency Department if any of the following develop: uncontrollable bleeding, increase in eyelid pain and/or swelling, the appearance of yellow or green pus in the eye or on the eyelid, and fever.

Oculoplasty referral is warranted to evaluate recurrent hordeolum and chalazion to rule out malignancy.[5,14] Recurrent bilateral hordeola may indicate an underlying immunodeficiency (e.g., immunoglobulin M).[6]

COMPLICATIONS

Numerous complications are possible both during and as a result of the incision and drainage procedure. Inadvertent injury to the globe (e.g., corneal abrasion or corneoscleral perforation) or eyelid structures (e.g., lacrimal duct or punctum) may occur.[7] Early complications include infections such as a cellulitis or conjunctivitis. Most bleeding can be controlled with gentle pressure or tying off of the offending vessel with fine absorbable sutures. Careful and gentle portable cautery can be applied to control bleeding but extensive cautery can lead to excessive eyelid scarring. Uncontrolled bleeding is rare but can occur and will require an Ophthalmologist to control. Late complications occur from excessive scarring at the incision site. If an external incision was made, the contracting scar can lead to an ectropion, eyelid retraction, uneven eyelid margins, and trichiasis. Maintaining a vertical incision perpendicular to the eyelid margin mitigates the risk of future eyelid malposition. If an internal incision was made, conjunctival scarring can lead to an entropion and chronic conjunctivitis.

SUMMARY

Patients with an acute hordeolum often present to the Emergency Department with a painful, localized, red, and swollen eyelid margin. They should be initially managed conservatively. Subsequent incision and drainage may be required to alleviate the patient's discomfort. The incision and drainage procedure is simple to perform and can be done by the Emergency Physician.

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SECTION 13

Otolaryngologic Procedures

197 External Auditory Canal Foreign Body Removal

G. Carolyn Clayton, Katarzyna M. Gore, and Melissa M. Rice

INTRODUCTION

Foreign bodies are commonly found in the external auditory canal (EAC) of children and sometimes in adults.[1] Children commonly place small objects such as food (e.g., beans, peas, corn, and seeds) or small round objects (e.g., beads, rocks, and toys) in the EAC.[2-6] Adults are more likely to suffer from items used to clean or scratch the ear (e.g., cotton swab, paper, paper clips, and pencil lead) and insects that crawl into the ear.[4,6] The EAC and tympanic membrane (TM) are exquisitely sensitive and delicate.[3,7] Foreign bodies in the EAC are extremely irritating to patients, especially live insects that scratch the TM trying to escape. **Injuries can occur unless proper care is taken in the removal of EAC foreign bodies.**

ANATOMY AND PATHOPHYSIOLOGY

The EAC is an S-shaped structure that extends from the auricle to the TM and is approximately 2.5 cm long in adults.[8,9] The lateral or distal third is cartilaginous with thick skin. It has more hair follicles, glands, and subcutaneous tissue than the medial or proximal two-thirds of the EAC. The medial EAC is bony with a thinner and more fragile layer of skin.[8-11] The narrowed isthmus of the EAC is located between the cartilaginous and bony portions.[2,8] The EAC ends medially at the TM. The TM is angled obliquely to increase the surface area for carrying sound energy to the middle ear.[10] The anteroinferior EAC is 0.6 mm longer than the posterosuperior portion.[8] Auriculotemporal branches of cranial nerves V, VII, IX, and X and the greater auricular nerve of the cervical plexus supply sensation to the EAC.[8] The EAC and TM have separate innervation. Indications for anesthetizing these areas are distinct from those for performing an auricular block (Chapter 156). The posterior auricular, superficial temporal, and deep auricular arteries provide the blood supply to the external ear. The posterior auricular and superficial temporal veins drain the EAC. The posterior auricular vein can connect to the sigmoid sinus and provide a route for spread of infection between the ear and the intracranial cavity.[9]

INDICATIONS

All EAC foreign bodies must be removed. The questions are how quickly this must be done, who should do it, and which is the safest technique. The method used is individualized to the patient, type of foreign body, Emergency Physician preference and experience, and the availability of an Otolaryngologist. **The most urgent indication for immediate removal is an alkaline button battery because of the extensive and severe damage it may cause in a very short time.[1,12]**

Some foreign bodies are very easily and safely removed with the equipment available in any Emergency Department. Other foreign bodies may require removal under general anesthesia by an Otolaryngologist. Remove foreign bodies that are in the lateral half of the EAC, graspable, light or small, and have space around the foreign body for inserting an instrument. Things that make removal difficult are caustic foreign bodies, EAC blood, EAC edema, foreign bodies in the EAC for >24 hours, foreign bodies that touch the TM, and proinflammatory foreign bodies. Other foreign bodies (e.g., those that are impacted, involved with the TM or middle ear structures, large in size, or in young or mentally disabled patients, or have sharp edges) will require removal under general anesthesia or via a surgical approach.[2,7,13,14]

CONTRAINDICATIONS

These can be thought of as indications for referral to an Otolaryngologist for removal of the foreign body rather than contraindications to removal. The major contraindication to removal in the Emergency Department is probable injury with direct removal (e.g., foreign bodies located in the middle ear or those that have perforated the TM). Removal may cause further damage to the TM as well as potential disruption of the middle ear ossicles and hearing loss. These foreign bodies require removal under general anesthesia with the aid of an operating microscope.[2,3,5,13] Another contraindication is a large object that has impaled itself in the wall of the EAC. Direct removal will require anesthesia and possibly a surgical approach from outside the EAC to avoid denuding the skin of the EAC.[3] Refer patients with foreign bodies that are difficult to remove or patients who cannot hold still for the procedure (e.g., mentally disabled patients or young children) to an Otolaryngologist.[2,5,13,15] These cases are likely to result in injury if the foreign body is removed in the Emergency Department.

Irrigation is almost always safe to attempt with some critical exceptions.[16] One such contraindication is button battery removal.[1,12] There are two mechanisms for the rapid destruction of surrounding tissues by the batteries. The moisture and cerumen in the EAC have a high conductivity. This causes conduction of electric current from the battery and results in localized electrical burns. Local inflammation from burns will cause fluid exudation into the EAC. This increases the electrical conduction injury and causes the battery to begin leaking alkaline electrolyte solution with resultant liquefaction necrosis.[3,17] The degree of damage may be dependent on the remaining voltage in the battery, its chemical composition, its location, and the duration of mucosal exposure.[3,17,18] **Irrigation with water or saline is contraindicated in button battery removal and another technique must be used.[1]**

Another important contraindication for irrigating the EAC is an acute or chronically ruptured TM.[13,16,19-21] Water forced into the middle ear can lead to disruption of the ossicles, labyrinthitis, loss of balance, loss of hearing, mastoiditis, and otitis media. Some authors recommend alternate methods for any patient who has never had an ear exam to document the integrity of the TM.[13,16,19] Completely impacted foreign bodies that leave no space for the irrigant fluid to

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flow behind it will be driven deeper into the EAC making subsequent removal more difficult.[1,22]

A relative contraindication for irrigation with water is an organic object (e.g., dry bean, seed, or rice) or toy expanding beads.[22-26] Organic foreign bodies will absorb water and swell making removal more difficult. Irrigation can be attempted if the foreign body is very small and irrigation is expected to rapidly succeed in removal before swelling occurs. Irrigate the foreign body with alcohol, remove it with instruments, or extract it with suction.

Directly grasping the foreign body with forceps is contraindicated for large or spherical objects that will not allow clear passage of the forceps jaws along its sides. Attempts to grab this type of foreign body will drive it deeper into the EAC.[22,27]

EQUIPMENT

ANESTHESIA

- 1%, 2%, or 4% lidocaine solution or gel
- 1 mL syringe
- 5 mL syringe
- 27 or 30 gauge needle
- EAC speculum

IRRIGATION

- 10 or 20 mL syringe
- Butterfly catheter with the needle and most of tubing cut off
- Plastic portion of 18 gauge angiocatheter
- Kidney basin
- Chux or other water barrier
- Tap water or saline at or slightly above body temperature[7,10,16,20,21]

ALTERNATE IRRIGATION EQUIPMENT[10]

- Plastic portion of 18 gauge angiocatheter
- Dental jet irrigation device (e.g., Water-Pik)
- DeVilbiss irrigator and a compressed air source
- Metal ear syringe

INSTRUMENTS TO PASS BEHIND FOREIGN BODY AND PULL IT OUT (**FIGURES 197-1** TO **197-4**)

- Ear curettes or cerumen spoon (e.g., disposable plastic or metal)
- Wire loop
- Blunt or ball right-angle hook

INSTRUMENTS USED TO GRASP FOREIGN BODY DIRECTLY (**FIGURES 197-1** AND **197-3**)

- Alligator forceps
- Hartman forceps
- Lighted device (Bionix Medical Technologies, Toledo, OH)

SUCTION

- Frazier suction tips (**Figure 197-1*E***)
- Schuknecht catheter
- Intravenous tubing with flange created at tip using a heat source
- Vacuum or suction source
- Connection tubing
- Hemostat

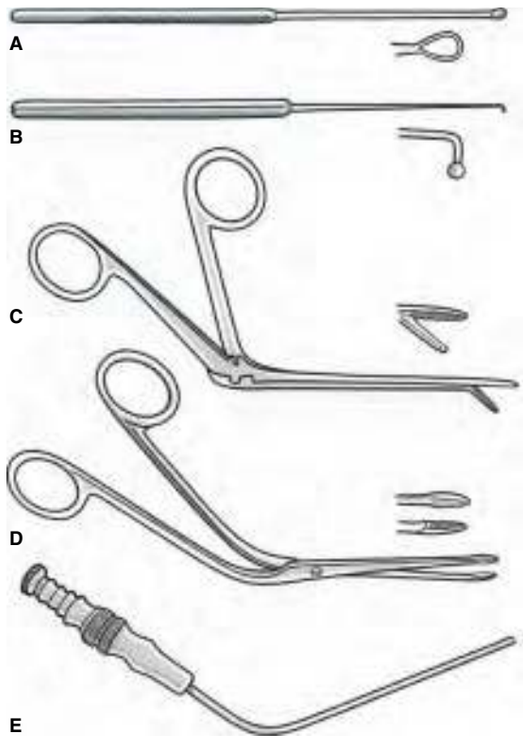


FIGURE 197-1. Instruments used for removal of foreign bodies from the external auditory canal. ***A.*** Cerumen loop. ***B.*** Right-angle ball hook. ***C.*** Alligator forceps. ***D.*** Hartman forceps. ***E.*** Frazier suction catheter.

CYANOACRYLATE GLUE[28]

- Ear speculum
- Cyanoacrylate tissue adhesive
- Cotton-tipped applicator
- Cyanoacrylate removal equipment

CYANOACRYLATE TISSUE ADHESIVE REMOVAL[29]

- Acetone to debond glue from skin
- Cotton balls
- Cotton-tipped applicators
- Irrigation or instruments for final removal

MAGNET REMOVAL

- Pacemaker magnet
- Metallic medical instrument
- Household magnet

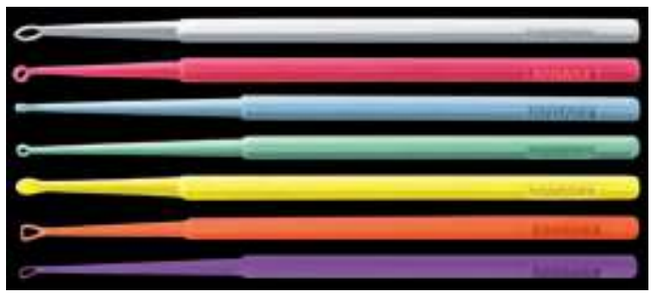
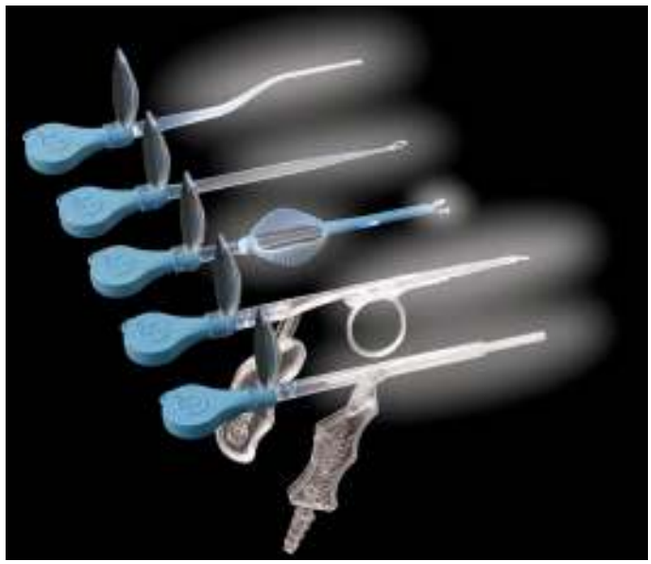


FIGURE 197-2. Examples of plastic disposable curettes. Note the variety of head shapes. (Photo courtesy of Bionix Medical Technologies, Toledo, OH.)



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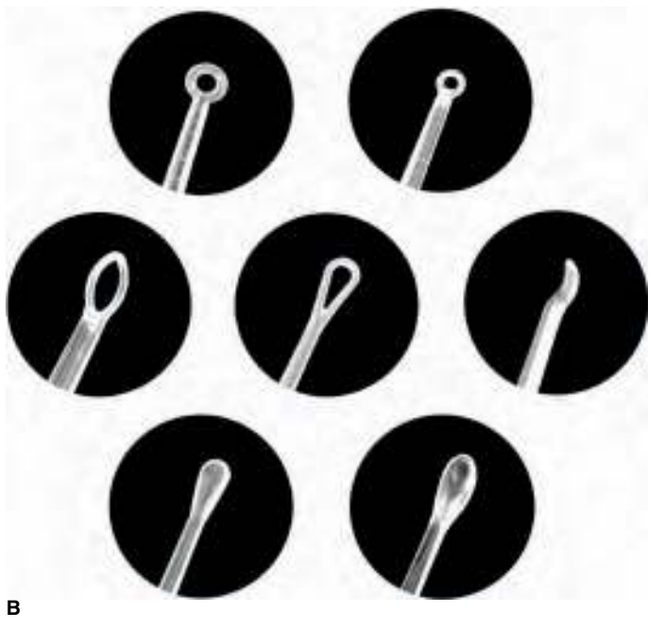

B

FIGURE 197-3. Lighted, disposable, and single patient use devices to aid in cerumen removal. ***A.*** From top to bottom: lighted placement tool, lighted ear curette, lighted forceps for foreign body removal, articulating ear curette, and the lighted suction removal device. ***B.*** Tips of the lighted curettes. (Photos courtesy of Bionix Medical Technologies, Toledo, OH.)

FIGURE 197-4. The EasiEar metal curette (Splash Medical Devices LLC, Atlanta, GA.)

COMMERCIALLY AVAILABLE DEVICES

- Hognose otoscope tip
- Gatornose otoscope tip
- Katz Extractor
- OtoClear Ear Irrigation Tip

PEDIATRIC IMMOBILIZATION

- Sheets
- Commercial immobilization device (e.g., Papoose board)

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. The discussion should include discomfort, dizziness, minor bleeding, postprocedural otitis externa, and TM perforation. **Discuss the importance of remaining still during the procedure.**[27] Warn the patient that they may experience an occasional loud noise, especially if suction is used. Obtain an informed consent for the procedure and place this in the medical record.

The most convenient position for adult patients is to remain seated with the affected ear facing the Emergency Physician. Children can sit on the lap of a parent or attendant with the affected ear facing the Emergency Physician. The parent or attendant should wrap one arm around the child's arms and body while stabilizing the head with their other arm (**Figure 197-5**).[3] Smaller children can be swaddled in a

FIGURE 197-5. A parent holding a child.

papoose made from a sheet and tape or a commercial immobilization device (Chapter 232).[3] Place the patient supine with their face toward the ceiling. **Do not place the patient with the affected ear facing up as this may cause the foreign body to move farther into the EAC.**

All removal techniques require the EAC to be straightened before inserting any device. This is accomplished in the older child, adolescent, and adult by pulling the pinna up and back while simultaneously pulling it straight out from the head.[10] Pull the pinna down, back, and slightly out from the head in the small child.[7,10]

The patient may require anesthesia of the EAC. Fill the EAC with 5 to 10 mL of 1% lidocaine using the syringe tip, an angiocatheter without the needle, or a butterfly catheter with the needle cut off. This will result in substantial but short-lived topical anesthesia for the procedure.[22] The topical application of lidocaine can be repeated as needed or longer-acting agents may be used if the effect wears off before the procedure is complete.

Injection of local anesthetic solution is usually not necessary. It may be required in some patients. Fill a 1 mL syringe with local anesthetic solution and apply a 2 inch long, 27 or 30 gauge needle. Position a plastic or metal ear speculum into the EAC. Insert the needle through the speculum. Inject 0.25 mL subcutaneously in the superior and inferior quadrants of the EAC distal to the isthmus. All four quadrants can be injected if needed.[22,23] Refer to Chapter 156 for a complete discussion of EAC anesthesia. A final option is to use procedural sedation and analgesia (Chapter 159) to control the patient and remove the foreign body.[30]

TECHNIQUES

PRECAUTIONS FOR USING INSTRUMENTS IN THE EAC

There are two major precautions to avoid pushing the foreign body further into the EAC, causing damage to the EAC, or causing damage to the TM. **The procedures must be performed under direct vision. The hand holding the instruments must remain firmly in contact with the patient's head to stabilize the hand to avoid abrading the EAC, lacerating the EAC, or damaging the TM if the patient abruptly moves.**[2,10] A very cooperative patient may move due to an involuntary reflex cough.[7]

IRRIGATION

Irrigation is the safest method of foreign body removal.[2,10,13,31] It is most successful for nonimpacted and smaller foreign bodies. **Be sure to use only body temperature or slightly warmer fluid to avoid complications from caloric stimulation.**[10,16] Tuck a water barrier into the patient's gown or collar. Place a kidney basin under the ear and against the cheek to catch the irrigation solution and the foreign body. Instruct the patient or an assistant to hold the basin in place. Attach a butterfly needle to a 10 or 20 mL syringe. Cut the needle and most of the tubing from the butterfly leaving only 1 to 2 cm of the tubing (**Figure 197-6**). The remaining tubing will usually be curved which is optimal for precisely directing the irrigation stream. An alternative is to use a 14 or 16 gauge plastic angiocatheter.[32] **Wider diameter instruments deliver fewer pounds per square inch of pressure with a reduced chance of injury.**[32]

Draw up body temperature or slightly warmer tap water or saline into the syringe. Straighten the EAC by manipulating the pinna. Insert the butterfly tubing or angiocatheter 1 cm into the EAC with the tip aimed upward and away from the foreign body (**Figure 197-6**).[20] Rapidly inject the irrigating solution. This will cause the fluid to shoot past the foreign body, bounce off the TM, and carry the foreign body out of the EAC along with the irrigating solution.[2,13]

Care must be taken to make sure that the irrigation stream and foreign body can easily exit the EAC to prevent an increase in hydrostatic pressure which could damage the TM.[19] Sometimes irrigation will not completely remove the foreign body but move it more laterally where an instrument can be safely used to grasp and remove it. Remove all water or saline from the EAC when done to prevent otitis externa.[10,16]

Many physicians have used mechanical dental irrigation devices (e.g., Water-Pik) to irrigate the EAC and remove either cerumen or a foreign body. These devices shoot a stream of fluid from its tip. The direct fluid stream can rupture a TM.[16,32,33]

The OtoClear Ear Irrigation Tip (Bionix Medical Technologies, Toledo, OH) is an improvement for EAC irrigation (**Figure 197-7*A***). This device is designed as disposable and single patient use. It attaches to the Luer hub of a syringe. The OtoClear tip can be attached to a spray bottle or dental irrigating device or bought from Bionix (**Figure 197-7*B***). This allows these devices to be safely used. The flared base of the OtoClear fits snugly and prevents it from being inserted too far into the EAC. Holes in the base of the OtoClear allow for the egress of irrigation fluid into the basin held against the skin. The OtoClear tip directs fluid toward the walls of the EAC and prevents damaging the TM (**Figure 197-7*B*, inset**). The OtoClear may not provide enough flow or pressure in some cases to remove a foreign body.

SLIDING A FOREIGN BODY OUT WITH AN INSTRUMENT FROM BEHIND

This technique requires either a cerumen spoon or wire loop for small objects or a right-angle hook for larger ones (**Figures 197-1**

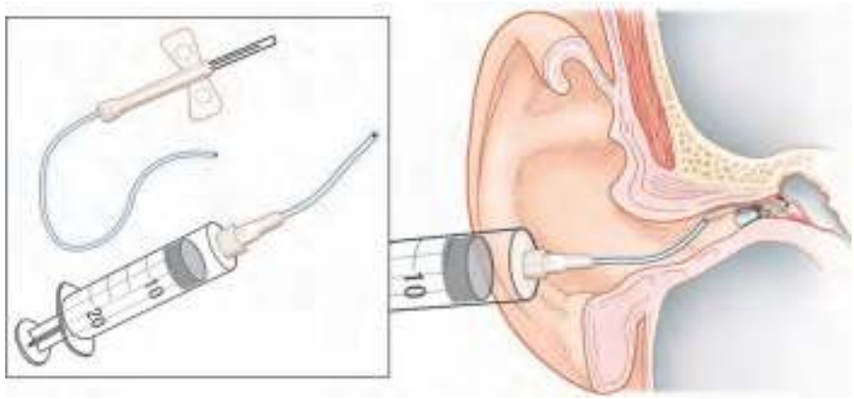

FIGURE 197-6. Irrigation with butterfly catheter tubing attached to a syringe. The stream of fluid is aimed toward the top of the external auditory canal and above the foreign body.

FIGURE 197-7. The Bionix OtoClear. ***A.*** The device. ***B.*** The device on a spray bottle and an irrigation device. The reusable basin on the left slips on the base of the spray bottle or irrigation device for storage and can be used to catch the irrigating solution. The inset shows the irrigation solution coming out the tip of the OtoClear. (Photos courtesy of Bionix Medical Technologies.)

through 197-4). Straighten the EAC by manipulating the pinna. Pass the tip of the instrument over and behind the foreign body with the spoon, loop, or hook in the same plane as the EAC wall. Rotate the instrument 90° to bring the loop or hook behind and directly in

FIGURE 197-8. The Bionix lighted forceps for foreign body removal with a magnification lens. (Photo courtesy of Bionix Medical Technologies.)

contact with the back (medial) side of the foreign body. Gently pull the instrument out of the EAC and pull the foreign body out with it.[2,10] **Take extra care so that the EAC and TM are not injured during the instrument insertion or removal.**

The EasiEar Disposable Comfort Curette (Splash Medical Devices LLC, Atlanta, GA) is an improvement to the standard disposable plastic curette (**Figure 197-4**). It is a stainless steel, single patient use, and disposable curette. The rounded wire head is smooth. It lacks the jagged and sharp plastic edges that are often found on molded plastic curettes. The EasiEar has no abrasive edges, seams, or surfaces to potentially abrade the EAC. This design may prevent EAC abrasions, bleeding, and lacerations. The spring wire shaft provides flexibility and enhanced maneuverability when compared to molded plastic curettes. The angled head and flexible shaft allow it to be manipulated within the EAC to remove a foreign body.

GRASPING FOREIGN BODIES WITH FORCEPS

There must be sufficient space between the EAC wall and the foreign body or a projecting edge that can be grasped to avoid pushing it further into the EAC.[2,4,11,13] This technique is contraindicated if the object is spherical and located against the TM. The TM can be injured during the procedure.[4,27] The most commonly used instruments include the alligator forceps (**Figure 197-1*C***) and the Hartman forceps (**Figure 197-1*D***). Lighted forceps (Bionix Medical Technologies, Toledo, OH) may be used (**Figures 197-3 and 197-8**).

Straighten the EAC by manipulating the pinna. Insert the forceps into the EAC and grasp the foreign body (**Figure 197-9**). Gently withdraw the instrument and the foreign body. Take care not to abrade the EAC wall. Irrigation may be able to move the foreign body laterally for subsequent grasping if the foreign body is located too far medially or instrumentation would cause pain or damage to the TM.

FIGURE 197-9. Forceps removal of a foreign body. ***A.*** Hartman forceps. ***B.*** Alligator forceps.

SUCTION REMOVAL

Frazier suction catheters (**Figure 197-1*E***) are most successful with small foreign bodies. Attach the Frazier suction catheter to the suction tubing. Turn on the suction source. Straighten the EAC by manipulating the pinna. Gently insert the catheter into the EAC. Place a thumb over the hole in the catheter handle to direct the suction through the tip of the catheter. Gently advance the suction catheter until its tip is in contact with the foreign body. Withdraw the Frazier suction catheter with the attached foreign body from the EAC. The Schuknecht catheter functions similarly to a Frazier suction catheter. It has a small, central, retractable stylus that can be helpful in grasping foreign bodies with a central opening.[11]

Suction with plastic intravenous tubing can be used for impacted smooth spherical objects.[1,34] Cut a short length of plastic intravenous tubing and attach one end to the suction source. Fashion the other end into a small flange using a heat source and any metal object with a rounded end (e.g., the tip of a hemostat or larger clamp). Heat the jaws of the hemostat and insert them into the plastic tubing just enough to create a flange. Turn on the suction source. Place a hemostat onto the tubing to temporarily clamp the suction tubing. Straighten the EAC by manipulating the pinna. Gently advance the flange tip into the EAC until it contacts the foreign body taking care not to push it inward (**Figure 197-10**). Have an assistant remove the hemostat from the tubing to activate the suction. Gently but quickly remove the tubing and attached foreign body from the EAC.

The Bionix Suction Removal Device (**Figure 197-11*A***) is used to remove foreign bodies. It is a plastic, disposable, single-use device that contains a magnification lens. The device attaches to portable suction or low wall suction (**Figure 197-11*B***). Removal of the foreign body is described above.

CYANOACRYLATE TISSUE ADHESIVE ASSISTED REMOVAL

Cyanoacrylate tissue adhesive can be used to extract impacted spherical objects that allow no space for irrigation or instrument removal.[1,28] This technique can be fraught with complication.[29] **Always use an ear speculum to decrease the chance of gluing the foreign body to the EAC or TM and to prevent creating a glue foreign body.** Insert an ear speculum into the EAC with the tip near the foreign body. **Do not touch the foreign body with the speculum to prevent it from being impacted.** The ear speculum will prevent the Emergency Physician from touching the EAC with cyanoacrylate tissue adhesive.

FIGURE 197-10. Suction removal of a foreign body.

A

B

FIGURE 197-11. The Bionix lighted suction removal device. ***A.*** The device. ***B.*** The device attached to a portable suction unit. (Photos courtesy of Bionix Medical Technologies.)

Obtain a cotton-tipped applicator. Moisten the tip of the applicator stick with a very tiny amount of cyanoacrylate tissue adhesive. A larger amount can drop off into the EAC. Insert the ear speculum. Straighten the EAC by manipulating the pinna. Quickly insert the applicator stick through the ear speculum before the cyanoacrylate tissue adhesive dries. Advance the stick until it just touches the foreign body. Maintain this position for 30 to 60 seconds to allow bonding of the cyanoacrylate tissue adhesive to the foreign body. Remove the applicator stick with the foreign body attached and the speculum together.

REMOVAL OF CYANOACRYLATE TISSUE ADHESIVE

Debonding glue from the patient's tissues with acetone must precede the foreign body removal for iatrogenic or patient-introduced cyanoacrylate adhesive.[1,29,35] Failure to do this will result in tearing of the skin or TM when the dried cyanoacrylate is removed. **Do not apply acetone if the TM cannot be visualized or is not intact.**[16] Infuse acetone into the EAC using cotton balls or swabs. Allow it to

remain in the EAC for 5 minutes. Several applications may be necessary since acetone evaporates rapidly. The glue mass can be removed by irrigation, instruments, or suction once it is free.

MAGNET REMOVAL

Attempt removal using magnets when the foreign body is magnetic.[34,36-38] One method includes magnetizing a medical instrument (e.g., forceps or hemostat) by placing it in series with a pacemaker magnet. Place the instrument in the EAC to attract the metallic foreign body. Another method is to use a household magnet at the entrance of the EAC in attempts to attract the foreign body. The strength of the magnet and the distance to the foreign body may limit this method.[24,36]

HOGNOSE OTOSCOPE TIP

The Hognose (IQDr. Incorporated, Manitou Springs, CO) is a disposable, latex free, and single-use device that attaches to a standard otoscope (**Figure 197-12**). It comes in three sizes (i.e., 3, 4, and 5 mm) with a color-coded tip. The size represents the cup size at the tip of the device. The tip is soft, self-molding, and looks like the nose of a hog. It has an insufflation port and suction tubing attached to its side. The adapter on the suction tubing attaches to standard wall suction tubing.

Attach the Hognose to the otoscope as if attaching a disposable speculum to an otoscope. Turn on the otoscope light source. Attach the hognose tubing to suction tubing and a suction source. Turn the suction source to low or medium. Grasp the otoscope with the dominant hand. Straighten the EAC by manipulating the pinna. Insert the Hognose into the EAC while visualizing the foreign body through the otoscope head. Place an index finger over the insufflation port to engage the suction when the tip of the Hognose is just next to the foreign body. Gently advance the otoscope until the tip of the Hognose is against and attached to the foreign body. The soft tip has collapsed on itself if black is visible through the otoscope. Remove the finger over the insufflation port and reapproach the foreign body. Withdraw the Hognose with the foreign body attached while maintaining suction.

FIGURE 197-12. The Hognose otoscope tip attached to an otoscope. (Photo courtesy of IQDr. Incorporated, Manitou Springs, CO.)

GATORNOSE OTOSCOPE TIP

The Gatornose (IQDr. Incorporated, Manitou Springs, CO) is a disposable, latex-free, and single-use device that attaches to a standard otoscope (**Figure 197-13**). It twists onto an otoscope like a speculum. It comes with three different jaw types that attach to the body of the device. These jaws are small flat jaws, large flat jaws, and open loop jaws. A trigger on the body of the device controls jaw opening and closing.

Attach the Gatornose to the otoscope as if attaching a disposable speculum to an otoscope. Select the appropriate jaws and attach these to the Gatornose base attached to the otoscope. Turn on the otoscope light source. Grasp the otoscope with the dominant hand. Insert the ring finger into the trigger. Pull the trigger to close the Gatornose jaws. Straighten the EAC by manipulating the pinna. Gently insert the Gatornose jaws just into the EAC. Push the trigger to open the Gatornose jaws and view through the otoscope. Gently advance the otoscope while visualizing the foreign body through the otoscope head. Position the jaws above and below or anterior and posterior to the foreign body. Pull the trigger to close the jaws onto the foreign body. Withdraw the otoscope with the foreign body in the jaws of the Gatornose.

KATZ EXTRACTOR

The Katz Extractor Oto-Rhino Foreign Body Remover (InHealth Technologies, Carpinteria, CA) is a device designed to extract foreign bodies from the nasal and auditory passages (**Figure 197-14**). It is a disposable single-use device consisting of a balloon-tipped catheter attached to a syringe. Always test the device before using it. Push the plunger to inflate the balloon and inspect it for any air leaks. Release the plunger to deflate the balloon.

Grasp the device with the dominant hand (**Figure 197-15**). Gently insert the catheter along the wall of the EAC until the balloon is just past the foreign body (**Figure 197-15*A***). Inflate the balloon by depressing the plunger on the syringe (**Figure 197-15*B***). Withdraw

FIGURE 197-13. The Gatornose otoscope tip attached to an otoscope. Note the three types of jaws that are available to snap onto the base of the device. (Photo courtesy of IQDr. Incorporated, Manitou Springs, CO.)

FIGURE 197-14. The Katz Extractor. The balloon at the tip inflates and deflates by pushing and releasing the plunger, respectively. (Photo courtesy of InHealth Technologies, Carpinteria, CA.)

the catheter and foreign body from the EAC while maintaining the balloon in the inflated state (**Figure 197-15*C***). Insert the catheter through the hole rather than behind it if the foreign body has a central hole (e.g., candy or bead).

LIVE INSECT REMOVAL

A live insect in the EAC is one of the most painful and upsetting foreign bodies. The patient suffers as the insect moves, vibrates, tries to flap its wings, or pushes against the sensitive TM to escape.[3] The Emergency Physician may attempt removal by turning off the main room lights and using a small source light near the EAC to lure the insect out.[14] Many substances have been tried to kill live insects in the EAC.[3,4,9,22,39]

The preferred technique is to immobilize the insect with an infusion of 1% lidocaine into the EAC.[40] A topical anesthetic composed of benzocaine and antipyrine (e.g., Auralgan) may be used. The local anesthetic results in the insect becoming inert much quicker than with mineral oil. The EAC and TM are anesthetized for patient comfort and the immobilized insect is more likely to be removed in a single piece than with mineral oil.

The insect can now be easily removed by irrigation or instrument removal. Sometimes both techniques are used. Irrigation can be used to move the dead insect more distally in the EAC followed by instrument removal. Irrigate and inspect the EAC after the insect is removed to make sure that no tiny parts of the insect remain in the EAC which can cause otitis externa.[3]

Mineral oil was infused in the past to smother and kill the insect prior to its removal.[4,22,40] This method has several disadvantages. A significant time passes while the patient is still suffering and the insect dies. The insect is more difficult to remove from the viscous oil and frequently breaks into multiple pieces. It is impossible to subsequently anesthetize the EAC with topical lidocaine which is repelled by the mineral oil.

ASSESSMENT

Reexamine the patient to confirm that the EAC, TM, and hearing are all normal and have not been damaged after the foreign body has been removed.[3,4,31] It is prudent to examine the other ear, the nostrils, and any other orifice that may be harboring an unsuspected foreign body in children, patients with mental disabilities, or patients with psychiatric problems.[3-5] Remove any remaining irrigation fluid in the EAC to prevent otitis externa.[10]

AFTERCARE

Consider prescribing several days of topical otic drops to prevent or treat subclinical otitis externa which is often precipitated by an abrasion of the EAC or inflammation due to foreign body

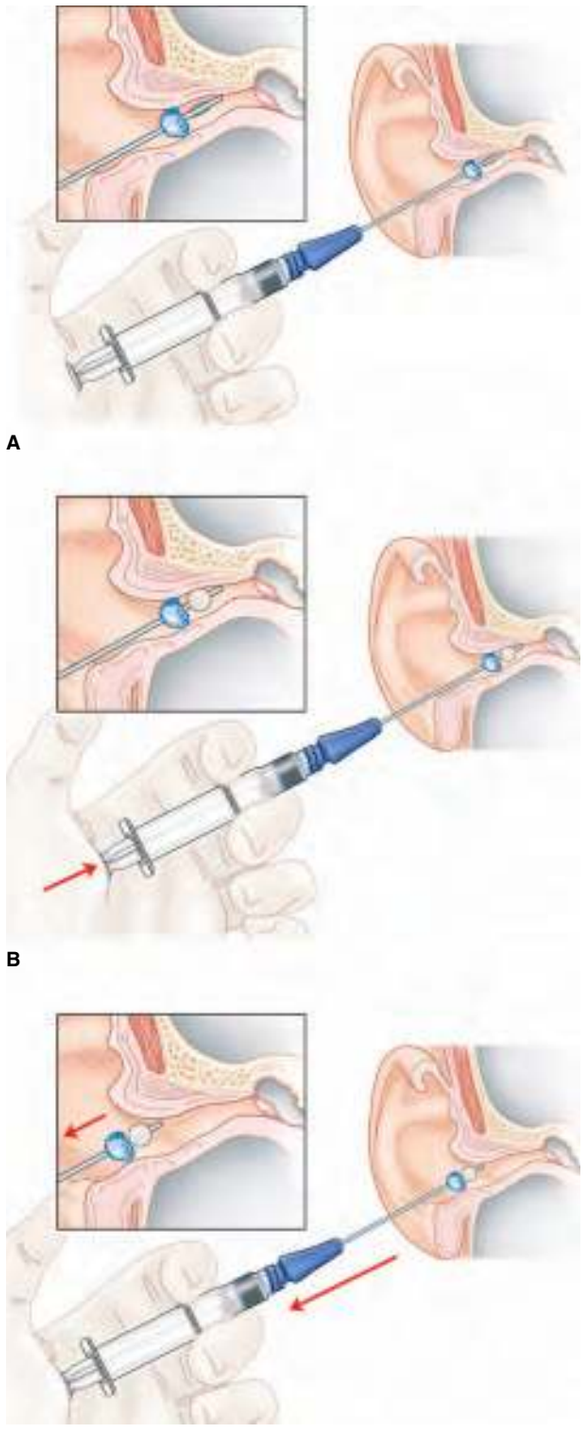

FIGURE 197-15. The Katz Extractor removing an EAC foreign body. ***A.*** The device is inserted until the balloon is just past the foreign body. ***B.*** The balloon is inflated. ***C.*** The device is removed with the balloon inflated and the foreign body is removed.

impaction.[3] Often recommended are topical combinations of antibiotics and steroids (e.g., Cortisporin otic or Ciprodex). Consult an Otolaryngologist for patients with injuries, hearing deficits, severe otitis externa, or in whom foreign body removal was unsuccessful.[41] The patient can follow-up with their Primary Care Physician in 48 to 72 hours after removal of the foreign body. Instruct the patient or their caregiver in the proper application of ear drops. The patient

should return to the Emergency Department if they develop ear pain, ear discharge, fever, decreased hearing, headache, stiff neck, or vertigo.

COMPLICATIONS

Numerous complications can result from the removal of a foreign body from the EAC.[3-5,10,16,19,21,22,31] The complication rate for irrigation is reported as 1 per 1000 cases.[21,31] It is higher for all other techniques.[10,16] Irrigation can push a foreign body further into the EAC. Caloric stimulation of cold irrigating solution can result in bradycardia, syncope, vertigo, and vomiting.[1,16] Middle ear debris can be forced through a preexisting or iatrogenic TM defect resulting in central nervous system infections, damage to the ossicles, labyrinthitis, loss of hearing and balance, mastoiditis, or otitis media.[13,16,19-21] Otitis externa can result from abrasions to the EAC or water left behind after irrigating. Butterfly tubing is less likely than an angiocatheter to damage the EAC or TM because of its more pliable nature, larger diameter, and curved tip.

Mechanical dental irrigation devices can rupture the TM.[16,32,33] This can occur at low pressures. These devices are not recommended for the removal of foreign bodies from the EAC.

Use instrumentation and suction with caution to prevent secondary injury. This includes EAC abrasions, EAC lacerations, disruption of the ossicles, infection, pushing of foreign bodies further into the EAC, and TM rupture.[27] Abrasions or lacerations to the EAC can result in mastoiditis, meningitis, or otitis externa. A foreign body in the EAC can lead to these infectious complications.

Cyanoacrylate tissue adhesive can cause complications and should be used with caution. The Emergency Physician may glue their fingers together or to the instruments. The foreign body may be glued to the pinna, EAC, or TM. Removal of the cyanoacrylate can cause abrasions, irritate the skin, and irritate the TM.

SUMMARY

Foreign bodies in the EAC are common. Most foreign bodies can be successfully removed in the Emergency Department from the EAC with adequate anesthesia, careful planning, and gentle handling.[43] The removal techniques require equipment that is readily available in the Emergency Department. The removal techniques are easy to perform, quick, and simple to learn. An impacted button battery is a true emergency and requires immediate removal. An emergent consultation with an Otolaryngologist is required if the button battery cannot be removed or if any evidence of injury is present after the button battery has been removed.

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198 Cerumen Impaction Removal

Dayle Davenport

INTRODUCTION

Removal of impacted cerumen (**Figure 198-1**) is one of the most common otolaryngologic procedures performed by physicians who are not Otolaryngologists.[1] It is also a common cause of iatrogenic otolaryngologic complications with medical malpractice suits that frequently result in payment.[2] Approximately 150,000 ears are irrigated weekly and 8 million annually in the United States to remove cerumen.[3,4]

ANATOMY AND PATHOPHYSIOLOGY

The S-shaped external auditory canal (EAC) is 2.5 cm long in adults.[5] The lateral or distal third is cartilaginous, with thicker skin, more hair follicles, glands, and more subcutaneous tissue than the medial or proximal two-thirds which is bony and has a thinner, more fragile layer of skin.[5,6] The narrowed isthmus is located between the cartilaginous and bony portions.[5] The canal ends medially at the tympanic membrane (TM) which is situated obliquely to increase the surface area for carrying sound energy to the middle ear.[5] The anteroinferior EAC is 0.6 mm longer than the posterosuperior portion.[5] Auriculotemporal branches of cranial nerves V, VII, IX, and X and the greater auricular nerve of the cervical plexus supply sensation to the EAC.[5]

FIGURE 198-1. Cerumen from the external auditory canal on a cotton-tipped applicator. (Used from www.commons.wikimedia.org/wiki/File:Earwax_on_swab.jpg.)

Cerumen (**Figure 198-1**) is a mixture comprising secretions of the ceruminous glands of the lateral two-thirds of the EAC, the pilosebaceous glands located at the roots of EAC hairs, and sloughed squamous epithelial cells.[5,7] Cerumen forms a barrier against infection, has antimicrobial activity, and protects the skin of the EAC as it is water repellant. It is expelled naturally by migration and assisted by chewing movements.[7,8]

There are many reasons for cerumen to become impacted.[4,7] The most common is self-cleaning with cotton-tipped applicator swabs that can push cerumen further into the EAC (**Figure 198-1**). The abundant hairs in the EAC, more common in males than females, can obstruct cerumen migration. A small (e.g., especially in children), tortuous, or scarred EAC will obstruct cerumen migration. Some people produce large quantities of cerumen. Diseases such as Parkinson's can alter the consistency of the cerumen and inhibit its migration. Hearing aids, earbud headphones, stethoscope earpieces, or any other object in the EAC may compact the cerumen. Deficits in the substances that cause sloughed squamous epithelial cells to separate will inhibit the movement of cerumen. Nonimpacted cerumen exposed to water can swell and obstruct the EAC.

INDICATIONS

The term "impaction" is defined as cerumen that causes patient symptoms or prevents a necessary examination of the EAC or TM, even without complete obstruction of the canal.[4] The primary indication for removal of cerumen is the symptomology of impaction.[4] The most common complaint is hearing loss which is often abrupt and expressed as a "blocked ear." Hearing remains normal or nearly so as long as there is a small space in the EAC through which sound can pass and cause vibration of the TM. The hearing loss becomes subjectively significant when the canal is completely obstructed or when the TM is compressed by cerumen and cannot freely move.[4,8] Other typical symptoms of cerumen impaction include fullness, itching, odor, pain, tinnitus, vertigo, unsteady gait, or reflex cough due to vagus nerve stimulation.[4,7] Other indications for removing cerumen include the need to examine the ear canal, to examine the TM, or to test the patient's hearing.[4,8,9] Cerumen can cause feedback sound loops in those with hearing aids or damage the appliance.[4]

CONTRAINDICATIONS

There are several general contraindications to cerumen removal. An uncooperative patient or young patient who cannot follow instructions or be safely restrained can suffer an iatrogenic injury. Previous ear surgery or radiation therapy to the ear with scarring of the EAC or TM risks iatrogenic injury. A known or suspected cholesteatoma is a contraindication to cerumen removal. Do not attempt removal if the anatomy of the EAC or TM cannot be clearly defined or is distorted.

The remaining contraindications to cerumen removal are specific to each removal technique. Nearly all cerumen can be safely removed by using one of the techniques listed. Often, two or more techniques can be used together with increased success.[9]

The most important contraindication for EAC irrigation is an acute or chronically ruptured TM.[4,7-12] Fluid forced into the middle ear can lead to otitis media, labyrinthitis, mastoiditis, disruption of the ossicles, and loss of balance or hearing.[4,10,12] Some authors

recommend alternate methods for any patient who has never had an ear examination to document the integrity of the TM.[9,10] Most patients can provide the information of having a history of a ruptured TM. A relative contraindication is moderate to severe otitis externa.[4,7,8]

The major contraindication to instrument removal of cerumen is a patient, usually pediatric, who is so uncooperative that injury to the EAC or TM is likely to occur with movement.[12,13] The second contraindication is cerumen pushed directly against the TM. In these circumstances, the TM can be abraded or perforated during cerumen removal with instruments.

The major contraindication to suction removal is a single, hard, irregular, and impacted cerumen plug as suction will be unsuccessful. Suction works best if there are multiple tiny cerumen fragments or very soft cerumen.[9]

Cerumen softening or cerumenolytic agents should not be used if the patient has a ruptured TM. Other contraindications to softening agents are an allergy to the agent or otitis externa.[4,7,14,15]

EQUIPMENT

IRRIGATION

- 10 to 20 mL syringe
- Butterfly catheter with any size needle
- Kidney basins
- Chux (or other water barrier)
- Tap water or saline at or slightly above body temperature[5]

ALTERNATE IRRIGATION EQUIPMENT[4]

- Plastic portion of an 18 gauge angiocatheter
- Oral jet irrigation (i.e., Water-Pik) no longer recommended (see below)[3]
- DeVilbiss irrigator and a compressed air source
- Metal ear syringe

INSTRUMENTS TO SEPARATE AND LOOSEN CERUMEN (FIGURES 198-2 TO 198-5)

- Ear curettes or cerumen spoon, metal or disposable plastic
- Wire loop
- Blunt or ball right-angle hook

INSTRUMENTS TO GRASP CERUMEN DIRECTLY (FIGURES 198-2 AND 198-4)

- Alligator forceps
- Hartman forceps
- Lighted forceps (Bionix Medical Technologies, Toledo, OH)

SUCTION

- Frazier suction catheters
- Suction source
- Connection tubing
- Hemostat

CERUMEN-SOFTENING AGENTS

- Warm tap water
- Saline solution
- Hydrogen peroxide
- Olive, mineral, vegetable, or almond oil

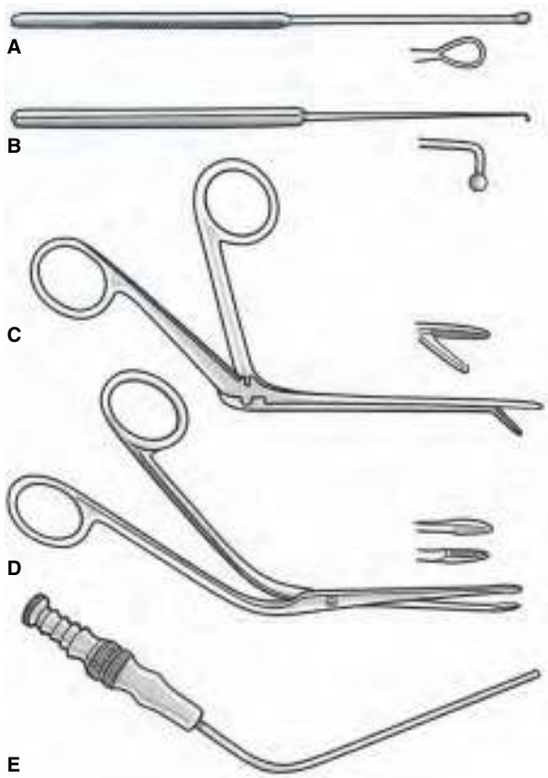

FIGURE 198-2. Instruments used for removal of cerumen from the external auditory canal. ***A.*** Cerumen loop. ***B.*** Right-angle ball hook. ***C.*** Alligator forceps. ***D.*** Hartman forceps. ***E.*** Frazier suction catheter.

- 5%, 10%, or 15% sodium bicarbonate solution
- Commercial agents (e.g., Cerumenex, Cerumol, Auralgan, Waxsol)
- 10% triethanolamine polypeptide oleate condensate
- Carbamide peroxide (i.e., Debrox)
- Colace
- Glycerin
- Alcohol
- Propylene glycol

PEDIATRIC IMMOBILIZATION

- Sheets
- Commercial immobilization device (e.g., Papoose board)

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. **The discussion should include discomfort, dizziness, minor bleeding, postprocedural otitis externa, and TM perforation. Discuss the importance of remaining still during the procedure.** Warn the patient that they may experience an occasional loud noise, especially if suction is used. Obtain a signed consent for the procedure. Some Emergency Physicians omit the signed consent and place the following statement in the procedure note: "The risks, benefits, and complications were described and discussed with the patient. They understood this and gave verbal consent for the procedure." This decision must be based on physician preference, hospital guidelines, and state guidelines for documentation requirements.

The most convenient position for adult patients is to remain seated with the ear facing the Emergency Physician. Children can sit on the

A

B

FIGURE 198-3. Examples of plastic disposable curettes. ***A.*** The curettes. ***B.*** Note the variety of head shapes. (Photo courtesy of Bionix Medical Technologies, Toledo, OH.)

lap of a parent or attendant with the affected ear facing the Emergency Physician. The parent or attendant should wrap one arm around the child's arms and body while stabilizing the head with their other arm (**Figure 197-5**). Smaller children can be swaddled in a papoose made from sheets and tape or a commercial immobilization device.[16]

Most patients do not require topical anesthesia. Occasionally, a patient will require it. Turn the affected ear toward the ceiling and fill the EAC with 5 to 10 mL of local anesthetic solution or suspension using the syringe tip, an angiocatheter, or a butterfly catheter with the needle cut off. This will result in substantial but short-lived topical anesthesia for the procedure. The topical application of local anesthetic can be repeated as needed or longer acting agents may be used if the effect wears off before the procedure is complete.

All cerumen removal techniques require the EAC to be straightened. This is accomplished by grasping the pinna and pulling it slightly up and back while simultaneously pulling it straight out from the head in the older child, adolescent, and adult.[6] Pull the pinna down, back, and slightly out from the head in the young child.[6,17]

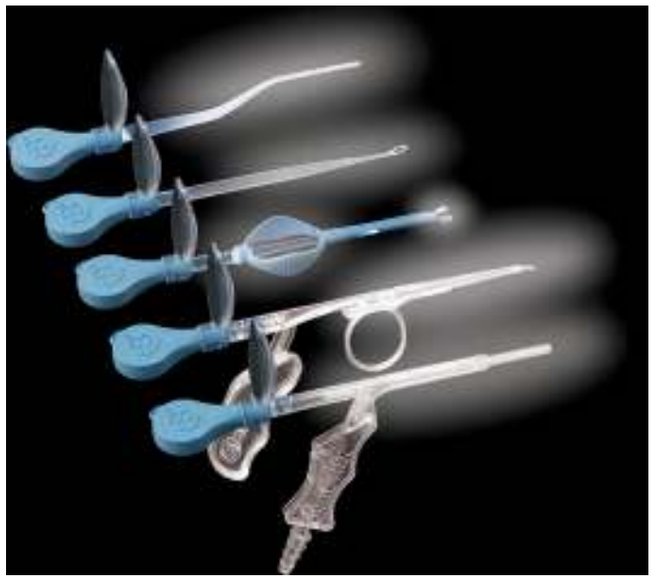

FIGURE 198-4. Lighted, disposable, and single patient use devices to aid in cerumen removal. From left to right: lighted placement tool, lighted ear curette, lighted forceps for foreign body removal, articulating curette, and lighted suction removal device. (Photo courtesy of Bionix Medical Technologies, Toledo, OH.)

TECHNIQUES

PRECAUTIONS FOR USING INSTRUMENTS IN THE EAC

There are two major precautions the Emergency Physician must take to avoid pushing cerumen further into the EAC or causing damage to the EAC and TM. **First, the procedures must be performed under direct vision.[6] Second, the hand holding the instrument must remain firmly in contact with the patient's head at all times to stabilize the hand and avoid scrapping the canal wall or puncturing the TM should the patient move.[6,15]** Even a very cooperative patient may move due to an involuntary reflex cough.[17]

IRRIGATION

Irrigation is the safest and easiest method of removing cerumen and is usually successful.[2,4,6,10,15] Removal is facilitated by the use of water or a cerumenolytic for 15 to 30 minutes prior to irrigation.[18] It is less likely to lacerate or damage the EAC or TM than other techniques. It is also the technique most commonly used by physicians who are not Otolaryngologists.

Place a kidney basin under the affected ear and against the patient's cheek to catch the exiting irrigation solution and cerumen. Instruct

FIGURE 198-5. The EasiEar metal curette (Splash Medical Devices LLC, Atlanta, GA.)

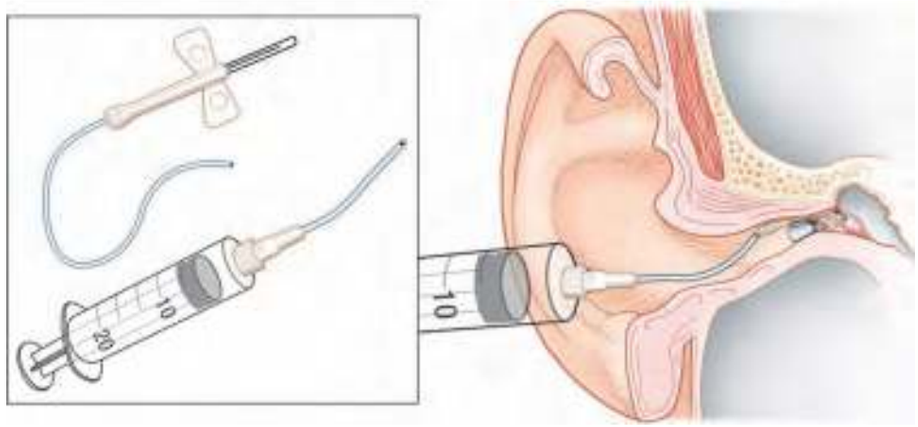

FIGURE 198-6. Irrigation with butterfly catheter tubing attached to a syringe. The stream of fluid is aimed toward the top of the external auditory canal and above the cerumen.

the patient or an assistant to hold the basin in place. Cut a butterfly needle to remove the wings and needle and allow only 1 to 2 cm of tubing to remain to improvise an irrigation system (**Figure 198-6**).[13] This remaining tubing will usually be curved which is optimal for precisely directing the irrigation stream. Alternatively, a 14 or 16 gauge plastic angiocatheter can be used.[12] It is important to remember that wider diameter instruments deliver fewer pounds per square inch of pressure with a reduced chance of injury.[12] Attach the irrigation tubing or angiocatheter onto a 10 or 20 mL syringe (**Figure 198-6**). Draw up body temperature or slightly warmer normal saline or tap water into the syringe to prevent a caloric-reflex response.[4] Insert the butterfly tubing or angiocatheter 1 cm into the EAC with the tip aimed in a direction opposite to the location of the cerumen (**Figure 198-6**).[8] This will cause the water to shoot past the cerumen, bounce off the TM, and force the cerumen out of the EAC along with the irrigating solution.[10] Direct the stream superiorly or slightly anterior and superiorly if there is no obvious break in the cerumen.[4,10] **Instill the stream gently and check the EAC intermittently for clearance of cerumen. Stop immediately if the patient experiences pain, bleeding, or vertigo.[19]**

Care must be taken to make sure that the irrigation stream can easily exit the EAC to prevent an increase in hydrostatic pressure which could damage the TM.[9] Irrigation will often have to be repeated numerous times but persistence is usually rewarded with success.[6] Irrigation will sometimes have to be combined with cerumen-softening agents, manual separation of cerumen from the EAC wall, or direct grasping of a partially dislodged cerumen plug.[4,6]

Many physicians have used mechanical dental irrigation devices (e.g., Water-Pik) to irrigate the EAC and remove cerumen. These devices shoot a stream of fluid from its tip. While they will often remove the cerumen, the direct fluid stream can rupture a TM.[3,4,12,20,21]

The OtoClear Ear Irrigation Tip (Bionix Medical Technologies, Toledo, OH) is another method for EAC irrigation (**Figure 198-7**). This device is designed as disposable and single patient use. It attaches to the Luer hub of a syringe. The OtoClear tip can also be attached to a spray bottle or dental irrigating device, allowing these devices to be safely used. When inserted into the EAC, its flared base fits snugly and prevents it from being inserted too far. Holes in the base allow for the egress of irrigation fluid and cerumen into the kidney basin held against the skin. The OtoClear tip directs fluid toward the walls of the EAC (**Figure 198-7**) and away from the TM, theoretically reducing damage to the TM.[22]

Commercial devices are available for the irrigation of cerumen from the EAC. Two of the devices are the Aural Irrigation System and the Ear Wash System (Welch Allyn, Skaneateles Falls, NY).[23] They attach to a standard sink faucet to provide a regulated flow of continuous water. The water flows from the sink through the unit to a hand-held piece held in the EAC. These types of devices are rarely available in the Emergency Department.

INSTRUMENT REMOVAL: SEPARATION OF CERUMEN FROM THE EAC WALL

This technique is a useful adjunct to irrigation and/or instrument removal.[15] Sometimes the cerumen is firmly pressed against the EAC wall in all visible directions. Irrigation will not work or may even worsen the situation by pushing the cerumen further into the EAC. Use either cerumen curettes (**Figures 198-3 to 198-5**), loops (**Figure 198-2*A***), right-angle hooks (**Figure 198-2*B***), or wires to gently separate the cerumen from the EAC wall and compress it into the center of the lumen.[9]

The EasiEar Disposable Comfort Curette (Splash Medical Devices LLC, Atlanta, GA) is an improvement to the standard disposable plastic curette (**Figure 198-5**). It is a stainless steel, single patient use, and disposable curette. The rounded wire head is smooth and lacks the jagged and sharp plastic edges that are often found on molded plastic curettes. The EasiEar has no abrasive edges, seams, or surfaces to potentially abrade the EAC. This design may

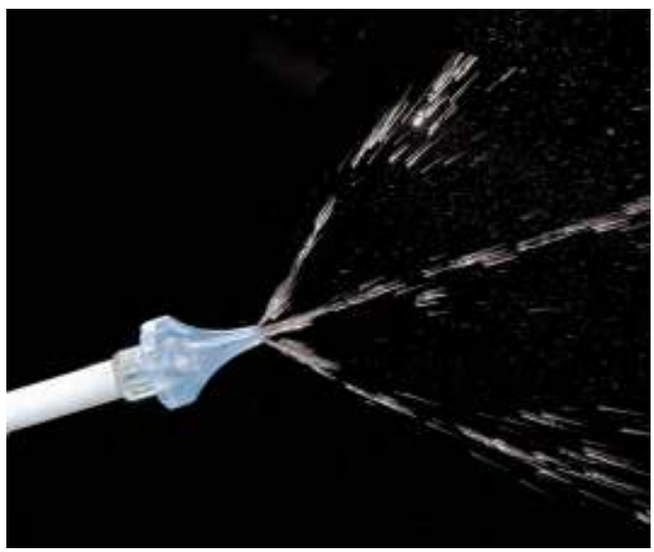

FIGURE 198-7. The OtoClear Ear Irrigation Tip. (Photo courtesy of Bionix Medical Technologies, Toledo, OH.)

prevent painful cerumen removal, EAC abrasions and lacerations, and procedure-related bleeding. The spring wire shaft provides some flexibility and enhanced maneuverability when compared to molded plastic curettes making the cerumen removal process easier.

The best place to start is superiorly as the superior portion of the cerumen plug tends to be thinner.[24] **The cerumen should not be pulled out with the same movement as this can abrade the EAC.**[15] Once there is a visible passage to the TM, irrigation as described above is highly successful and safe. Alternatively, separation of the cerumen from the EAC wall can be performed circumferentially all the way around the cerumen plug. This results in a cerumen plug freely suspended in the EAC that can easily be removed by irrigation, pulled out with a right-angle hook, or grasped with forceps.[15]

INSTRUMENT REMOVAL: SLIDING CERUMEN OUT WITH INSTRUMENTS FROM BEHIND

This technique requires either a cerumen spoon (**Figure 198-2*A***), a wire loop for small cerumen particles, a right-angle hook (**Figure 198-2*B***) for larger quantities, or an EasiEar curette (**Figure 198-5**). Separate the cerumen from the EAC wall as described above. Position the instrument with the loop or hook in the same plane as the EAC wall. Insert the tip of the instrument into the EAC above and just beyond the cerumen to be removed. Rotate the instrument 90° to bring the loop or hook behind and directly in contact with the back (i.e., medial) side of the cerumen. Gently pull the instrument out of the EAC, pulling the cerumen out with it.[24] **Take care not to abrade the EAC wall with the instrument.**

INSTRUMENT REMOVAL: GRASPING CERUMEN WITH FORCEPS FOR REMOVAL

There must be sufficient space for the jaws of the forceps on both sides between the EAC wall and the cerumen or a lateral leading edge that can be grasped. Separate the cerumen from the EAC wall as described above. Insert the forceps into the EAC with the blades closed and taking care not to push the plug further back in the ear. When the obstruction is encountered, grasp the cerumen with the jaws of the Hartman (**Figure 198-8*A***) or alligator forceps (**Figure 198-8*B***).[24] Gently withdraw the forceps taking care not to abrade the EAC wall. If the cerumen is located too far medially or instrumentation would cause pain or damage to the TM, irrigation can move the cerumen plug laterally for subsequent grasping with forceps.

The Emergency Physician may need to alternate irrigation and instrument removal to remove all of the cerumen plug safely and completely.[4,6] For example, irrigation may gently soften or loosen the plug. A cerumen loop or curette can then be used to separate it from the EAC wall. Irrigation can be used again to move it more laterally. Finally, a forceps or hook can be used to remove the cerumen plug from the EAC.

FIGURE 198-9. Suction removal of cerumen.

SUCTION REMOVAL

This technique requires soft cerumen or multiple small flakes.[9] Use the same preparations and precautions described above. Attach the Frazier suction catheter or suction tubing to the suction source. Turn on the suction source. Insert the suction catheter into the EAC. Gently advance the suction catheter until the tip is in contact with the cerumen (**Figure 198-9**). If using a Frazier suction catheter, place the thumb over the hole on the catheter shaft to direct the suction through the tip of the Frazier catheter. Gently advance the Frazier catheter until its tip is in contact with the cerumen. Withdraw the suction catheter or Frazier catheter and cerumen from the EAC. Usually the tip must be withdrawn and cleaned continuously as most cerumen will plug the suction tip. For additional safety, insert the suction catheter through a plastic otoscope speculum and withdraw both together.

A relatively new device is available from Bionix (**Figure 198-10**). The Lighted Suction device (Bionix Medical Technologies, Toledo, OH)

A B

FIGURE 198-8. Forceps removal of cerumen. ***A.*** Hartman forceps. ***B.*** Alligator forceps.

FIGURE 198-10. A disposable, lighted, single-use suction curette containing a magnification lens. (Photo courtesy of Bionix Medical Technologies, Toledo, OH.)

is an economical, plastic, single-use, and disposable device. It has the advantage of providing its own light. It attaches to wall suction or portable suction through an adapter built in to the handle. The built-in magnifying lens helps the Emergency Physician see the walls of the EAC and TM very easily.

CERUMEN-SOFTENING AGENTS

Apply a "cerumenolytic" or cerumen-softening agent if the cerumen is so impacted and hard that the above techniques are likely to cause pain, are likely to cause injury, or if the cerumen cannot be removed.[18] After the cerumen has softened, it can be removed using the techniques described above. Many studies have compared different agents and most are surprisingly comparable in their effectiveness.[1,4,7,14,18,25-27] In fact, even water or saline is effective for cerumen softening and disintegration.[1,4,7,14,25-27] This may explain why persistent irrigation is so frequently successful. The more commonly available agents in the Emergency Department are listed in the "Equipment" section.

Fill the EAC with one of the cerumen-softening agents. Insert a cotton ball into the opening of the EAC to prevent the agent from leaking out. Apply the agent to the contralateral EAC if indicated. An alternative is to place the patient lying on their side with the affected ear facing upward. Insert the agent into the EAC. The agent will not leak out if the patient remains in this position. The disadvantage of placing the patient on their side is that only one ear can be worked on at a time.

Most authors recommend waiting 15 to 30 minutes after the application of the cerumen-softening agent before attempting to remove the cerumen.[8,18] Some recommend using the agents at home for up to a week before further removal attempts.[8,9] Prolonged exposure to these agents can precipitate otitis externa, allergic reactions, or a contact dermatitis in the EAC. These agents must be removed and the canal thoroughly dried after their use.[4]

ASSESSMENT

It is critical to reexamine the ear to confirm that the EAC, TM, and hearing are all normal after the cerumen has been removed.[2,4] Remove all remaining water and softening agents to prevent otitis externa.[4]

AFTERCARE

Many authors recommend prescribing several days of topical otic drops to prevent or treat subclinical otitis externa which is frequently present in these patients. Often recommended are topical combinations of antibiotics and steroids in solution or suspensions. Commonly prescribed agents include otic quinolones, Corticosporin Otic, Otic Domeboro, Otobiotic, Pediotic suspension, and VoSol, to name a few. Consult an Otolaryngologist for patients with injuries, hearing deficits, severe otitis externa, or in whom cerumen removal was unsuccessful. Otherwise, the patient can receive follow-up with their Primary Care Physician in 48 to 72 hours. Instruct the patient in the proper application of ear drops. They should return to the Emergency Department if they develop ear pain, ear discharge, fever, decreased hearing, vertigo, headache, or a stiff neck. They should also be cautioned against future use of cotton swabs or other instruments in the EAC.

COMPLICATIONS

Numerous complications can result from the removal of cerumen from the EAC. These include but are not limited to perforation of the TM, otitis externa, damage to the external canal, deafness, vertigo, and tinnitus.[2-4,7,9,12,13,20,26,27] The complication rate for irrigation is 1 per 1000 cases.[2,4,7] Irrigation can push cerumen further into the EAC. Irrigation fluid must be body temperature or slightly warmer. Cold fluid can cause caloric stimulation resulting in vertigo, vomiting, bradycardia, or syncope. Middle ear debris can be forced through a preexisting or iatrogenic TM defect resulting in an otitis media, ossicle damage, labyrinthitis, mastoiditis, loss of hearing and balance, or a central nervous system infection. Otitis externa can result from abrasions to the EAC or retained fluids. Butterfly tubing is less likely than an angiocatheter to damage the EAC or TM because of its more pliable nature, larger diameter, and curved tip.

It is known that mechanical dental irrigation devices, even at low pressures, can rupture the TM. For this reason, they are not recommended for cerumen removal.[3,4,12,21]

Instrumentation and suction should be used with caution to prevent secondary injury. This includes lacerations or abrasions of the EAC, rupture of the TM, pushing foreign bodies further into the EAC, and disruption or removal of the ossicles. Abrasions or lacerations to the EAC can result in otitis externa.

Cerumen-softening agents can cause a contact reaction in the EAC and may lead to otitis externa. If the TM is not intact, these agents may cause permanent middle ear damage. Do not use these agents if the TM is ruptured acutely or the history suggests a potential defect.

SUMMARY

Successful cerumen removal will often require using multiple techniques in sequence and adapted to the individual patient's situation. Removal of cerumen requires adequate anesthesia, careful planning of the procedural sequence, and gentle handling. Those who have been relieved of a cerumen impaction will be among your most grateful patients.

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199 Tympanocentesis

Paul J. Jones

INTRODUCTION

Tympanocentesis, first described in 1768, is a diagnostic and therapeutic procedure in which a needle is inserted through the tympanic membrane to aspirate fluid from the middle ear (**Figure 199-1**). The procedure is considered diagnostic when the material obtained is sent for laboratory and/or microbiological analysis. It is considered therapeutic in most instances because it relieves pressure, reducing pain, and often shortens the course of acute otitis media (AOM).

The procedure is quick, simple, and not as frequently performed as it should be. General practitioners and Pediatricians would frequently perform the procedure for the relief of pain in the preantibiotic era. Tympanocentesis is making a resurgence. It should be considered when a patient presents to the Emergency Department seeking treatment for a painful AOM.[1] The American Academy of Family Physicians, American Academy of Pediatrics, and the Centers for Disease Control and Prevention all include tympanocentesis in their practice guidelines for AOM. Many authors are calling for culture-directed antibiotic therapy for otitis media to reduce the need for broad-spectrum antibiotics and prevent the emergence of multiresistant organisms.[2-7]

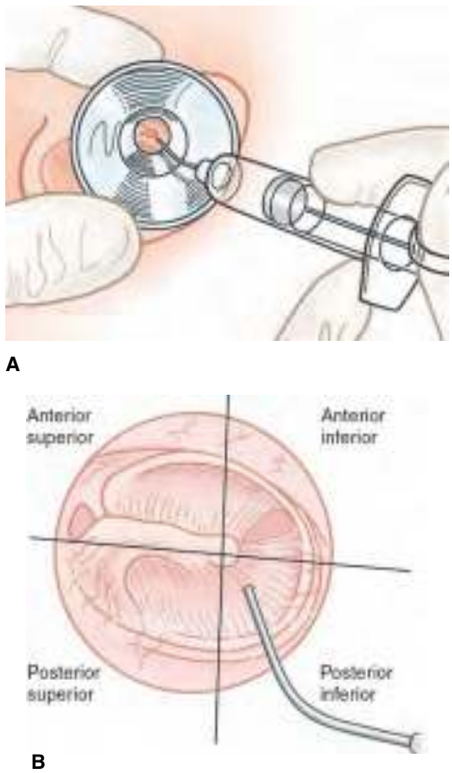


FIGURE 199-1. Tympanocentesis of the right ear. The patient is lying supine with their head directed to the left. ***A.*** An ear speculum is inserted into the external auditory canal. A needle is inserted through the posterior inferior quadrant of the tympanic membrane to aspirate middle ear fluid. ***B.*** Magnified view of the tympanic membrane.

ANATOMY AND PATHOPHYSIOLOGY

The ear is divided into the external, middle, and inner parts. The external ear is comprised of the auricle, the external auditory canal, and the external auditory meatus. The middle ear contains an air space and mastoid cells ventilated by the eustachian tube, the tympanic membrane, and the three ossicles. The inner ear is comprised of the cochlea, semicircular canals, fluids, and cranial verve VIII. The facial nerve courses through the middle ear space and mastoid process. It can be affected by a severe infection in these areas. Facial asymmetry during an acute ear infection is an indication of an unusually severe infection.

Inspection of the tympanic membrane will usually show it to be bulging during an acute infection with loss of mobility on pneumatic otoscopy. Conditions that are more chronic may show color changes of the tympanic membrane. There may be associated scarring and distortion.

INDICATIONS

Tympanocentesis is performed to obtain fluid for microbiological culture and antibiotic sensitivity testing to determine the infectious cause of a middle ear effusion.[1] Tympanocentesis is warranted for patients with AOM that is severe or unresponsive to conventional antimicrobial therapy for 48 to 72 hours or in a child less than 8 weeks of age to rule out gram-negative organisms. Tympanocentesis is warranted for

patients with an AOM and an immunodeficiency as they will often require directed therapy. Patients who develop AOM while taking appropriate antimicrobial therapy should undergo tympanocentesis to be evaluated for the organism responsible and its sensitivity to antibiotics. Tympanocentesis is performed when the patient has an AOM associated with unusually severe pain, signs of toxicity, or bullous myringitis. Tympanocentesis will provide immediate relief of pain, pressure, and/or hearing loss associated with AOM or a middle ear effusion. It may be performed in a patient with AOM and multiple antibiotic allergies to determine appropriate antibiotic selection. A tympanocentesis can be performed in the patient with AOM prior to the 48-hour "watchful waiting" period to allow accurate antibiotic selection if the patient is still symptomatic after 48 hours.

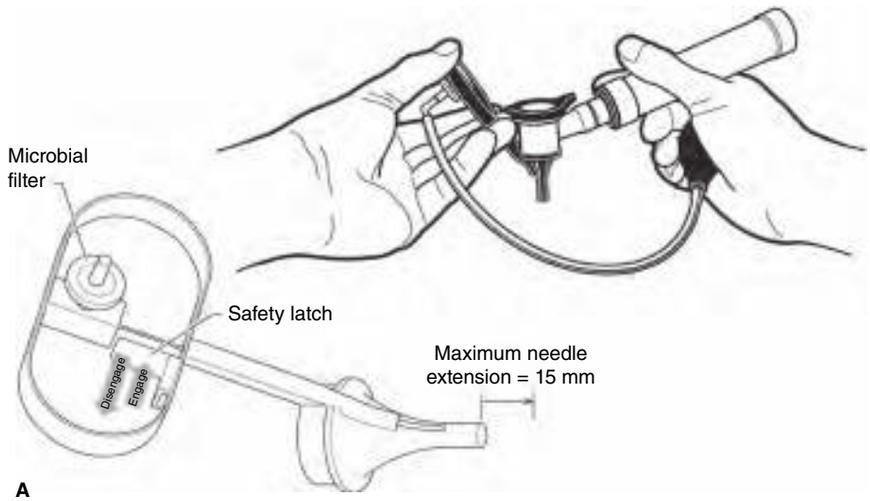
CONTRAINDICATIONS

There are no absolute contraindications to tympanocentesis. It should not be performed in a patient who is uncooperative and cannot be restrained and/or sedated as secondary injury may result. Uncooperative patients will require sedation to perform this procedure. **Tympanocentesis should be performed by an Otolaryngologist if the landmarks on the tympanic membrane are unable to be absolutely identified or are obscured.** Consult an Otolaryngologist prior to performing a tympanocentesis if an AOM is associated with anticoagulation, blood dyscrasias, a brain abscess, cochlear or other implant, dural sinus thrombosis, encephalitis, facial nerve palsy, intratympanic tumors, mastoiditis, meningitis, or stenosis of the external auditory canal. Do not perform a tympanocentesis if the patient has otitis externa.

EQUIPMENT

- Povidone iodine solution
- Topical otic anesthetic solution
- 21 gauge spinal needle, 2.5 or 3 inches long
- 3 mL aspirating syringe
- Ear speculum
- Ear wax curette
- Culture swabs and media
- Laboratory tubes for fluid cell count and differential
- Intravenous extension tubing
- Otoscope
- Headlamp or overhead surgical light source
- Frazier suction catheters
- Suction tubing
- Suction source
- CDT Speculum, optional

The Channel Directed Tympanocentesis or CDT Speculum (Walls Precision Instruments LLC, Baker City, OR) is a single patient use, disposable, easy to use, and sterile device with built-in safety features. It was designed for use by Primary Care Physicians in the outpatient setting. The CDT Speculum attaches to most commonly available otoscopes (**Figures 199-2 and 199-3**). The device allows tympanocentesis and the aspiration of middle ear effusions.



A

B

FIGURE 199-2. The CDT Speculum attached to an otoscope. ***A.*** Artist illustration. ***B.*** Photograph. (Photos courtesy of Walls Precision Instruments LLC.)

FIGURE 199-3. Proper grasping of the CDT Speculum attached to an otoscope. Note that the dominant thumb is placed on the actuator. (Photo courtesy of Walls Precision Instruments LLC.)

There are several advantages to the CDT Speculum over the traditional spinal needle on a syringe. The ensheathed needle protects the Emergency Physician as well as the patient from accidental needlesticks. The needle is incorporated into a speculum to prevent it from contacting the external auditory canal. The needle is automatically retracted by a spring into a protected position when not being used. A safety latch prevents accidental needle extension. Needle extension is limited and prevents it from being inserted too far into the middle ear cavity.

Some form of light is needed. One device is the Bionix Lighted Placement Tool (Bionix, Toledo, OH). The device is single use, disposable, and cost effective (**Figure 199-4**). Many Emergency Departments may have this device for cerumen removal. Other options include a headlight or overhead light. The use of an overhead light is the least favored option. It needs to be constantly moved, the Emergency Physician's head gets in the way, and it can cast shadows of items in the path of the beam.

A

B

FIGURE 199-4. The Bionix Lighted Placement Tool. ***A.*** The tool. ***B.*** The tool in use. (Photos courtesy of Bionix.)

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. The postprocedural care should also be discussed. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents.

Clean and prepare the ear and external auditory canal. Place the patient supine on a locked gurney. View the tympanic membrane with an otoscope. Remove any cerumen from the external auditory canal using a curette (Chapter 198). The cerumen may also be flushed from the external auditory canal. The patient may need analgesia before cerumen removal if they are significantly tender. View the tympanic membrane again with the otoscope. Make sure the external auditory canal is clear. Apply povidone iodine solution to the external auditory canal for 2 to 3 minutes while it dries. Copiously irrigate the external auditory canal with sterile normal saline to remove all the povidone iodine.[8]

Administer analgesic medicine in advance of the procedure. Topical anesthetic solutions include benzocaine-antipyrine (e.g., Auralgan), viscous 4% lidocaine, 8% tetracaine, and cocaine. The administration of topical or local anesthesia is often not helpful in the presence of an acute infection. Topical anesthetics can potentially affect culture results due to their antimicrobial activity. If culture results are not a concern, apply a topical anesthetic solution into the external auditory canal followed by a cotton ball. Allow the solution to remain for 5 to 10 minutes. Use a wick to absorb the topical anesthetic solution and dry the external auditory canal.[8] Refer to Chapter 159 for performing a regional block of the ear and external auditory canal as an alternative to topical anesthesia.

Children and uncooperative patients must be restrained and/or sedated so that their head is immobile. A papoose board is effective for young children. Some practitioners use intravenous sedation. Only rarely is procedural sedation (Chapter 159) or general anesthesia required.

TECHNIQUES

TRADITIONAL NEEDLE-BASED TECHNIQUE

Bend a 21 gauge spinal needle at the hub to approximately 60°.[8] Attach the spinal needle to a 3 mL aspirating syringe. Insert the ear speculum into the external auditory canal (**Figure 199-1*A***). View the tympanic membrane through the ear speculum using a headlight or an overhead surgical light source for illumination.

Insert the spinal needle through the speculum. Advance the needle and penetrate just into the inferior half of the tympanic membrane (**Figures 199-1*A* and 199-1*B***). **Avoid inserting the needle through the posterior superior quadrant of the tympanic membrane. This location is near the ossicles. Any movement of the**

needle near the ossicles could result in disarticulation of the ossicles requiring surgical repair. Aspirate the middle ear fluid into the syringe (**Figure 199-1*A***). Simultaneously withdraw the syringe and ear speculum.

CHANNEL DIRECTED TYMPANOCENTESIS (CDT) SPECULUM

Remove the sterile CDT Speculum from the package. Attach the aspirator bulb and tubing to the CDT Speculum. Attach the CDT Speculum to the otoscope (**Figures 199-2 and 199-3**). Align the arm of the CDT Speculum with the insufflator port of the side of the otoscope head. Firmly attach the CDT Speculum onto the otoscope. Rotate the CDT Speculum 90° clockwise so that the arm is aligned with the seam on the top of the otoscope head. Disengage the safety latch.

Grasp the CDT Speculum with the dominant hand while simultaneously holding the otoscope and aspiration bulb with the nondominant hand (**Figure 199-3**). Compress the aspirator bulb. Insert the CDT Speculum into the external auditory canal. Visualize the tympanic membrane through the CDT Speculum. Look through the CDT Speculum. Press the actuator with the dominant thumb to extend the needle toward the inferior portion of the tympanic membrane (**Figure 199-3**). Continue to press the actuator to keep advancing the needle through the tympanic membrane and no more than 1 to 2 mm into the middle ear cavity. Release the compression on the aspirator bulb to aspirate the middle ear fluid. Release the pressure on the actuator to retract the needle. Withdraw the CDT Speculum from the external auditory canal.

Place the tip of the CDT Speculum over the culture swab or medium. Compress the aspirator bulb to expel the middle ear fluid sample. Discard the CDT Speculum.

ALTERNATIVE TECHNIQUE

An alternative method involves attaching intravenous extension tubing between the spinal needle and the syringe. The Emergency Physician can observe the tympanic membrane, insert the spinal needle, and leave a hand available for manipulation of the ear speculum or a suction catheter. An assistant is required to hold the syringe and aspirate the middle ear fluid. The remainder of the technique is as described previously.

ASSESSMENT

Tympanocentesis will decompress the middle ear pressure and provide the patient significant symptom relief. Transfer the fluid into appropriate laboratory medium and containers as quickly as possible. Label the containers and have them transported to the laboratory for a Gram's stain, culture and sensitivities, cell count, and differential of the cells present.

AFTERCARE

Most of the distress associated with the procedure in small children is due to immobilization. Immediately release the child from the papoose or restraining device. Most patients will have immediate improvement in their pain and their hearing. Some Otolaryngologists will rinse the external auditory canal after a tympanocentesis with a 3% peroxide solution and then absorb the solution with a wick. This is optional and at the discretion of the treating Emergency Physician. The small opening in the tympanic membrane may drain (i.e., blood and middle ear fluid) for 48 to 72 hours. Instruct the patient and/or their caregivers to keep the ear dry for 2 to 3 days. This is especially true during bathing or hair washing. A cotton earplug coated with a thin film of petroleum jelly (e.g., Vaseline) works well. The tympanic membrane usually spontaneously heals within 48 to 72 hours but can take up to 10 days. Follow-up for laboratory results and documentation of antimicrobial change or appropriateness is necessary in 48 to 72 hours.

COMPLICATIONS

Complications are uncommon. The most frequently cited complications include laceration of the ear canal, persistent TM perforation with or without otorrhea, development of a scar on the tympanic membrane, and otitis externa. Pain and bleeding are usually minimal and self-limited. One of the most significant and rare complication is the disruption of the ossicles of the middle ear. **Disruption of the ossicles can be avoided by inserting the needles into the inferior half of the tympanic membrane and preventing patient movement during the procedure.** Injury to the chorda tympani, facial nerve, or internal carotid artery is theoretically possible but virtually unheard of with this procedure.

SUMMARY

Acute otitis media is a common infection of childhood but is also seen in adults. Most episodes respond quickly, with or without antimicrobial therapy. Tympanocentesis can be used to direct antimicrobial therapy in patients when a clinical response is delayed, host immunosuppression exists, or unusual organisms are suspected. Tympanocentesis can be used to provide immediate pain relief from a severe middle earache. The procedure is quick and simple to perform in the Emergency Department.

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200 Auricular Hematoma Evacuation

Eric F. Reichman

INTRODUCTION

Blunt trauma to the auricle can cause abrasions, ecchymosis, hematoma formation, and lacerations. Abrasions and ecchymosis of the auricle require no therapy other than oral analgesics and observation for infection.[1] Some authors recommend the application of topical antibiotics to all abrasions as prophylaxis for infection.[2]

Lacerations to the auricle are addressed in Chapter 119. This chapter addresses the management of an auricular hematoma.

Injuries to the auricle are common due to its exposed position and lack of protection from surrounding structures.[3] The most common cause for an auricular hematoma formation is blunt trauma while participating in the contact sports of wrestling or boxing.[1,2,4-6] Such trauma may occur in other situations including assaults, falls, fights, and motor vehicle crashes. Auricular trauma and hematomas are common in children due to the high incidence of head injuries during playtime.[2,4] Blood dyscrasias may also cause an auricular hematoma.

An auricular hematoma presents as a firm and painful swelling that obscures the normal convolutions on the lateral aspect of the auricle. It can develop within minutes to hours of the blunt trauma. **An auricular hematoma must be evacuated to prevent the cosmetic disfigurement known as cauliflower ear.[7] The sooner it is evacuated, the less chance of permanent disfigurement**.[4,5] After evacuation, the patient requires a pressure dressing to the auricle, oral antibiotics, and close follow-up to prevent complications.[2,5,8,9]

ANATOMY AND PATHOPHYSIOLOGY

The auricle is that portion of the external ear that projects from the side of the head. It functions to augment sound delivery to the tympanic membrane and assist in sound localization. It is fixed in position by both ligaments and muscles.[10] It has an underlying cartilaginous framework that is 0.5 to 1.0 mm thick and provides the auricle with its unique shape. The cartilage is a single, thin sheet of flexible yellow elastic cartilage with many convolutions on the lateral surface.[11] The only portion of the auricle without cartilage is the lobule in which fibrofatty tissue replaces the cartilage.[10] **The cartilage is avascular and derives its blood supply and nutrients from the adjacent perichondrium.[12]**

The skin covering the auricle is similar to that elsewhere on the body.[13] It contains sebaceous glands and a varying number of hair follicles. The skin on the lateral surface of the auricle is tightly adherent to the perichondrium and lacks a subcutaneous layer (**Figure 200-1**). The skin on the medial surface of the auricle is loosely attached and has a layer of subcuticular tissue between the skin and perichondrium.

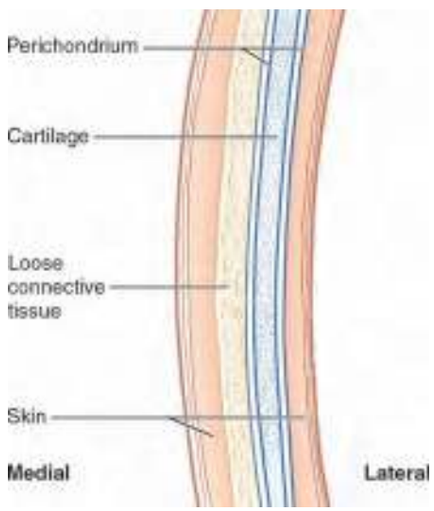


FIGURE 200-1. Cross-section of the auricle. The skin on the medial surface has a layer of loose connective tissue that is lacking on the lateral surface.

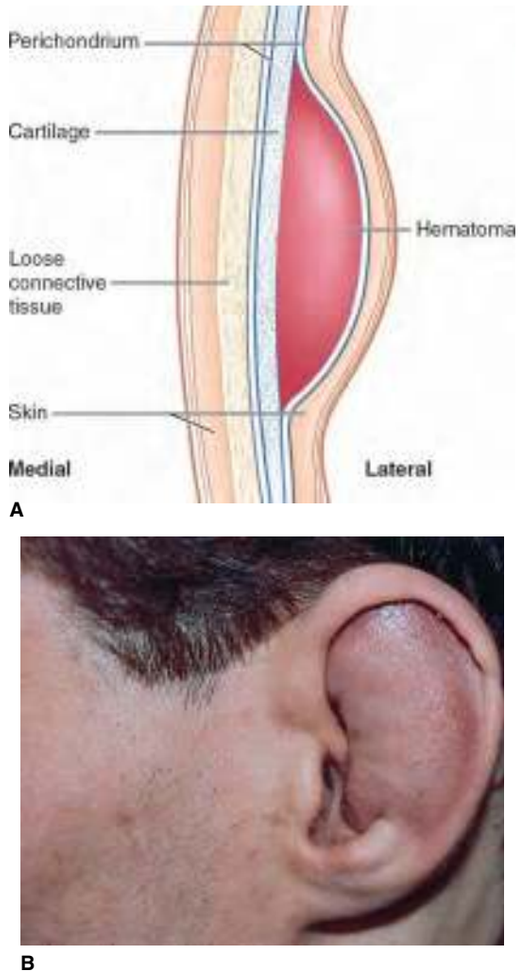


FIGURE 200-2. An auricular hematoma. ***A.*** The blood collects between the perichondrium and the cartilage. ***B.*** Photograph of a patient. (Used with permission from Knoop KJ Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 3rd ed. New York: McGraw-Hill; 2010. Photo contributor: C. Bruce MacDonald, MD.)

Trauma may cause the perichondrium to be torn off the underlying cartilage due to the tight attachment of the skin to the perichondrium on the lateral surface of the auricle. This traumatic avulsion of the perichondrium causes hemorrhage into the space between it and the cartilage allowing a hematoma to form (**Figure 200-2**). Bleeding into this potential subperichondral space causes dissection of the perichondrium from the underlying cartilage. Auricular hematomas can be painful due to the rich sensory innervation to the area and the accumulation of blood in this relatively closed space.[12] **Failure to adequately evacuate a hematoma may lead to cartilage necrosis and a deformed ear (Figure 200-3)**.[7,14,15] The necrosis is due to a combination of separating the cartilage from its blood supply and direct pressure effects from the hematoma.

The cauliflower ear is a purely cosmetic deformity that results from an auricular hematoma not being evacuated and allowing the auricle to spontaneously heal. The hematoma is invaded by fibroblasts and slowly replaced by fibrous tissue. This organization of the hematoma causes irregular thickening of the auricle. The perichondrium, elevated from the cartilage by the hematoma, senses the lack of adjacent cartilage and activates chondroblasts. New cartilage is deposited on the surface of the hematoma causing further thickening and deformity of the auricle.

FIGURE 200-3. An undrained auricular hematoma results in a cauliflower ear. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine,* 3rd ed. New York: McGraw-Hill; 2010. Photo contributor: C. Bruce MacDonald, MD.)

INNERVATION OF THE AURICLE

A brief review of the innervation of the auricle will later aid in understanding the technique of regional anesthesia. Three nerves contribute to the sensory enervation of the auricle (**Figure 200-4**).[9,12,13,16-18] The auriculotemporal branch of the mandibular division of the trigeminal nerve supplies sensation to the upper, lateral surface of the auricle. This nerve emerges subcutaneously just anterior to the auricle at the level of the external auditory canal. Two branches of the cervical plexus become subcutaneous at the posterior border of the midportion of the sternocleidomastoid muscle and ascend to the auricle. The lesser occipital nerve provides sensory innervation to the upper medial surface of the auricle and middle of the lateral auricular surface. The great auricular nerve provides sensory innervation to most of the medial surface and the lower half of the lateral auricular surface.

INDICATIONS

An auricular hematoma must be evacuated. The indication for evacuation is to prevent the cosmetic deformity known as the cauliflower ear (**Figure 200-3**).[8] It is preferable to evacuate the hematoma within 12 to 24 hours after its occurrence. Although there is no urgency to immediately evacuate the hematoma, the longer it remains, the higher the chance of clot organization and new cartilage deposition.[8,19]

CONTRAINDICATIONS

There are no absolute contraindications to the evacuation of an auricular hematoma. If the skin overlying the hematoma is cellulitic or if purulent material is drained from the hematoma, the patient will require hospital admission and intravenous antibiotics.[1,9] An Otolaryngologist or Plastic Surgeon should be immediately consulted on these patients. The auricular hematoma should be evacuated by a consultant if it has been present for more than 5 to 7 days. These hematomas have already begun organization and new cartilage has developed requiring curettage associated with the evacuation.[8,19-21] An uncooperative patient (e.g., a young child or due to altered mental status) may require evacuation under procedural sedation or in the Operating Room.

EQUIPMENT

AURICULAR ANESTHESIA

- Povidone iodine or chlorhexidine solution
- 1 mL syringe
- 10 mL syringe
- 25 to 30 gauge needles, 2 inches long
- 10 to 20 mL of local anesthetic solution without epinephrine

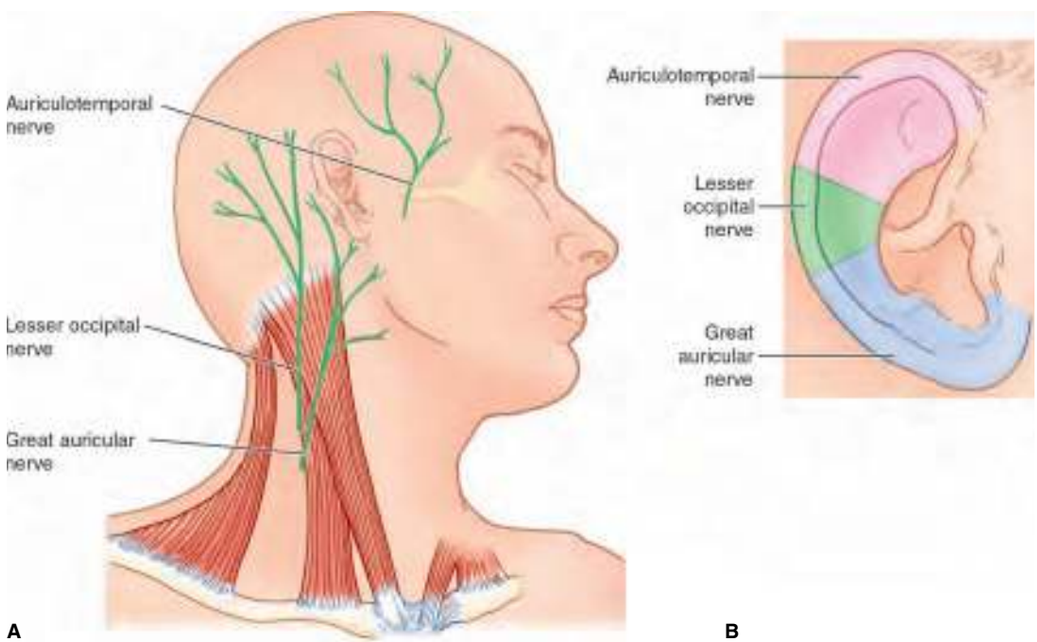


FIGURE 200-4. The sensory innervation of the auricle. ***A.*** The distribution of cutaneous nerves surrounding the auricle. ***B.*** An enlarged view of the auricle demonstrating the cutaneous innervation.

AURICULAR HEMATOMA ASPIRATION

- Povidone iodine or chlorhexidine solution
- Tuberculin or insulin syringe
- 0.25 mL of local anesthetic solution without epinephrine
- 10 mL syringe
- 18 gauge needle
- Topical antibiotic ointment

AURICULAR HEMATOMA INCISION AND DRAINAGE

- Auricular anesthesia as above
- #15 surgical scalpel blade on a handle
- Curved hemostat
- Sterile drain (optional)
- 10 mL syringe
- 18 gauge angiocatheter without the needle
- Sterile saline
- Topical antibiotic ointment
- Forceps

MASTOID PRESSURE DRESSING

- Petrolatum gauze
- Cotton balls soaked in sterile saline
- Dry cotton balls
- 4×4 gauze squares
- 4 inch elastic bandage
- Scissors

SURGICAL PRESSURE DRESSING

- Cotton bolsters or dental rolls
- Needle driver
- 4–0 monofilament nylon suture

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. The postprocedural care should also be discussed. Obtain a signed consent for the procedure. Some Emergency Physicians omit the signed consent and place the following statement in the procedure note: "The risks, benefits, and complications were described and discussed with the patient. They understood this and gave verbal consent for the procedure." This decision must be based on physician preference, hospital guidelines, and state guidelines for documentation requirements.

Remove any dirt and debris from the auricle and surrounding skin. Apply povidone iodine or chlorhexidine solution to the same areas. Follow aseptic technique for the remainder of the procedure.[4] In patients who are anxious and without other associated injuries, the administration of intramuscular, intravenous, or oral lorazepam (Ativan) or diazepam (Valium) may be beneficial.[18] Most patients do not need procedural sedation and analgesia.

AURICULAR ANESTHESIA

The local anesthetic solution used for auricular anesthesia should contain no epinephrine.[2,16,17,22] Epinephrine is not used for fear of intense vasoconstriction of end arterioles resulting in decreased perfusion with possible ischemia and necrosis of the auricle. Some authors recommend the use of 1/100,000 epinephrine mixed with the local anesthetic solution.[1,18,19] The epinephrine may decrease bleeding by its vasoconstrictive action. It may also prevent reaccumulation of the hematoma after it has been evacuated. Authors who advocate using epinephrine state that the auricle has a rich blood supply and that, based on anecdotal evidence, there is no danger of ischemia or necrosis from the use of epinephrine in healthy patients without evidence of traumatized vascularity. **Although many physicians will use epinephrine, it has not been proven safe to use or proven to prevent reaccumulation of the hematoma.** It may be wiser to be conservative and not use epinephrine than to use it and have to deal with the complications to the patient and potential litigation.

The choice of which local anesthetic to use is physician-dependent. Lidocaine (1%) is the most commonly used local anesthetic. Long-acting local anesthetic solutions, such as bupivacaine (Marcaine) or etidocaine (Duranest), may be used to provide analgesia for several hours after the procedure is completed.[17]

The methods of anesthesia for evacuation of an auricular hematoma range from none to a superficial skin wheal to a regional block. Some authors advocate using no anesthesia if needle aspiration of a small and fresh hematoma is performed.[2] This is not generally recommended as the pain from an 18 gauge needle aspiration is more uncomfortable than local anesthesia infiltration.

If using the aspiration technique to evacuate the hematoma, local anesthetic solution can be infiltrated directly over the hematoma. Apply a 25 to 30 gauge needle on a 1 mL syringe. Place a skin wheal using 0.25 mL of local anesthetic solution without epinephrine over the hematoma in the area of maximum fluctuance. **When placing the skin wheal be careful not to inject the local anesthetic solution into the hematoma.** This will cause expansion of the hematoma and increase the separation of the perichondrium from the underlying cartilage.[22] It may also cause new bleeding, which can increase the possibility of hematoma reaccumulation.

A regional auricular block is the preferred method to obtain anesthesia.[16] It prevents distortion of the auricle from direct injection and further separation of the perichondrium from the underlying auricular cartilage.[22] Subcutaneous infiltration of the surrounding skin is less painful than injection directly into the sensitive auricular skin.[18] The landmarks for regional anesthesia are simple to locate, consistent, and predictable. The greatest reason for failure of an auricular block is incorrect needle placement.[17] A regional auricular block can be done prior to using the aspiration technique or the incision and drainage technique to evacuate the hematoma. If the aspiration technique fails (i.e., hematoma reaccumulates), then there is no need to reprep and perform an auricular block prior to performing the incision and drainage.

There are three methods to perform a regional auricular block (**Figures 200-5 and 200-6**). Each method blocks the lesser occipital, great auricular, and auriculotemporal nerves. Some Emergency Physicians prefer to subcutaneously inject local anesthetic solution circumferentially around the attachment of the auricle to the head (**Figure 200-5**).[2,12,16,22] An alternative method is based on blocking the sensory supply to the ear in a more anatomic distribution (**Figure 200-6**).[9,17] This latter method uses half the anesthetic and half the number of subcutaneous injections than the former method. For these reasons, this author prefers the second method which is described in the following paragraph.

To perform a regional auricular block, first cleanse the auricle and surrounding skin of any dirt and debris. Apply povidone iodine or chlorhexidine solution. Place a skin wheal of local anesthetic solution 0.5 cm below the pinna of the auricle (**Figure 200-6*A***). Insert

FIGURE 200-5. Regional anesthesia of the auricle. The technique of circumferential application of local anesthetic solution. Shaded areas represent subcutaneous infiltration of local anesthetic solution.

a 2 inch, 25 or 27 gauge needle through the skin wheal aimed just posterior to the attachment of the auricle to the head. Infiltrate subcutaneously, in a superior direction, always remaining 0.5 to 1.0 cm posterior to the auricular attachment to the head. Stop infiltrating at the level of the superior attachment of the auricle to the head. This infiltration requires 4 to 7 mL of local anesthetic solution. Withdraw the needle almost completely. Redirect the needle through the skin wheal and aimed just anterior to the attachment of the auricle to the head. Infiltrate subcutaneously, in a superior direction, always remaining 0.5 to 1.0 cm anterior to the auricular attachment to the head. Stop infiltrating at the level of the superior attachment of the auricle to the head. This infiltration also requires 4 to 7 mL of local anesthetic solution. **Care must be taken not to inject too deeply anterior to the auricle as it can cause temporary paralysis of the facial nerve.** An alternative to the anterior infiltration is the injection of 3 to 4 mL of local anesthetic solution just superior and anterior to the tragus (**Figure 200-6*B***).[1,23] This injection blocks the auriculotemporal nerve at its origin. **Allow 10 to 15 minutes for the full anesthetic effect prior to beginning the procedure**.[22]

TECHNIQUES

The methods for treating and managing an auricular hematoma require the evacuation of the hematoma and replacing the perichondrium onto the underlying cartilage. The techniques include aspiration, incision and drainage, and closed suction drainage.

FIGURE 200-7. The aspiration of an auricular hematoma. The hematoma is expressed with the thumb and index finger as negative pressure is applied to the syringe.

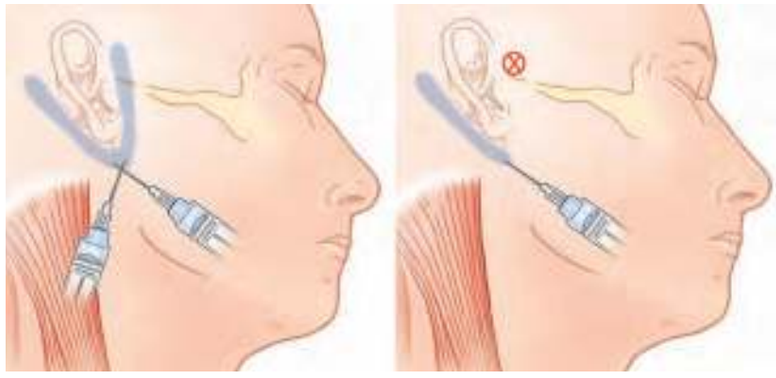
The first two techniques will be described in detail. The closed suction technique requires inpatient admission and is not a procedure to be performed in the Emergency Department. It will therefore not be described here.

ASPIRATION

Some consider the aspiration technique to be the primary method to evacuate an auricular hematoma.[2,3,5,6,24] They reserve the incision and drainage technique for incomplete aspiration or recurrence of the hematoma. Unfortunately, the hematoma frequently recurs after using the aspiration technique and the patient requires a second procedure to evacuate the hematoma.[1,3,6,25] Because of the high rate of recurrence, this author and others prefer to use the incision and drainage technique as the primary procedure.[19]

Clean and prep the skin. Anesthesia is achieved by performing a regional auricular block or by placing a skin wheal of local anesthetic over the hematoma. Attach an 18 gauge needle onto a 10 mL syringe. Insert the needle into the area of maximum fluctuance (**Figure 200-7**). Apply negative pressure to the syringe by withdrawing the plunger to evacuate the hematoma. Express or "milk" the hematoma between the thumb and index finger of the nondominant hand while applying negative pressure with the syringe to ensure complete evacuation of the hematoma (**Figure 200-7**).

Remove the needle and apply manual pressure to the area of the former hematoma for 3 to 5 minutes. If the hematoma recurs or if complete aspiration is not possible, perform the incision and drainage technique. If the hematoma is completely evacuated and does not recur, apply topical antibiotic ointment and a pressure dressing.



A B

FIGURE 200-6. Regional anesthesia of the auricle. ***A.*** A more anatomic auricular block. ***B.*** An alternative regional block that anesthetizes the auriculotemporal nerve at its origin. Local anesthetic solution is injected at the ⊗ symbol. Shaded areas represent subcutaneous infiltration of local anesthetic solution.

There are several disadvantages to this technique. First, the hematoma frequently recurs.[1,3,6,25] This means that the patient often needs a second drainage procedure. Even if the hematoma is adequately evacuated, the elimination of the dead space is problematic.[25] The dead space may fill with blood or serous fluid requiring a second procedure. A surgically applied pressure dressing may alleviate the dead space and make the aspiration technique more successful. Finally, the aspiration technique may not remove all of the hematoma.[1] Again, the patient will need a second procedure. For these above stated reasons, the aspiration technique is not the preferred method to drain an auricular hematoma.

A modification of this technique aspirates the hematoma with an angiocatheter.[26] Aspirate the hematoma with a 27 gauge angiocatheter. Leave the catheter in place while removing the needle. Carefully cut off the hub from the catheter. The catheter is left in place like a drain. Apply a dressing. This was a small study using only 53 patients. In six patients (11%), the catheter fell out within 72 hours allowing the hematoma to reaccumulate in three of the patients (6%). They repeated the procedure in these three patients. The auricle healed without complications in all the patients.

INCISION AND DRAINAGE

This is the preferred technique to evacuate an auricular hematoma.[19] It requires a regional auricular block for anesthesia. This technique takes more time to perform than the aspiration technique.

As noted previously, the skin should be cleaned and prepped in the usual manner. Perform a regional auricular block to provide adequate analgesia. Allow the block 10 to 15 minutes to achieve maximal effect.[22] Incise the auricular skin with a #15 surgical scalpel blade at the edge of the hematoma (**Figure 200-8*A***). **When making the incision, it should follow the curvature of the pinna and be no longer than 1 cm.** Gently peel this skin and attached perichondrium off the hematoma using forceps. Express or "milk" the hematoma with the thumb and index finger of the nondominant hand. Insert a curved hemostat and gently loosen any remaining blood clot (**Figure 200-8*B***). Fill a 10 mL syringe with sterile saline and attach a plastic 18 gauge angiocatheter. Gently flush out the area of the hematoma (**Figure 200-8*C***).

Reapproximate the skin and perichondrium on the cartilage. Compress the tissue to eliminate any fluid and dead space. Some authors apply a small rubber drain through the incision to prevent accumulation of blood or serous fluid.[4,9,27] The use of a drain is optional. A drain can be made by cutting a small strip from a sterile Penrose drain.

Apply manual pressure to the area of the former hematoma for 3 to 5 minutes. If the hematoma or serous fluid does not reaccumulate, apply topical antibiotic ointment and a pressure dressing as described below. If the hematoma or serous fluid does reaccumulate, consider inserting a rubber drain or applying a surgical pressure dressing.

PRESSURE DRESSINGS

A pressure dressing must be applied to the auricle after the successful drainage of an auricular hematoma. It prevents reaccumulation of the hematoma or serous fluid and supports the auricle while the perichondrium reattaches to the cartilage.[3,6,11] The pressure dressing must be applied for at least 48 hours.[4] The pressure dressing can be the traditional mastoid dressing or a surgically applied dressing.[19,22,25,28] **These pressure dressings apply even pressure over the entire auricle without compromising the blood flow while simultaneously eliminating the dead space within the wound.**[22]

MASTOID PRESSURE DRESSING

The most commonly applied dressing is the mastoid pressure dressing (**Figure 200-9**).[29] Although simple to place, it has many disadvantages when compared to the surgically applied pressure dressing. It is bulky and hard to keep in place. It is very conspicuous. Patients must keep it dry and remain relatively inactive to prevent it from coming off.

Place a piece of sterile dry cotton in the external auditory canal and level with the base of the auricular cartilage. Mold a sterile material that conforms easily onto all the convolutions of the auricle until it is level with the lateral helical rim (**Figure 200-9*A***). The choices of material include cotton balls soaked in mineral oil, cotton balls soaked in saline, or petrolatum gauze. The material chosen is left to Emergency Physician preference and what is available in the Emergency Department. This author prefers to use petrolatum gauze. The petrolatum gauze allows the dressing to be removed with minimal trauma to the ear. Pack the auricle with saline-soaked cotton balls. **All the convolutions of the auricle must be thoroughly packed. The packing of the auricular convolutions assures even application of pressure to all portions of the auricle.**

Cut out and discard a semicircle or a V-shaped section from a pile of gauze squares. Place the remaining C-shaped gauze pads behind the auricle (**Figure 200-9*B***). The gauze pads should be built up

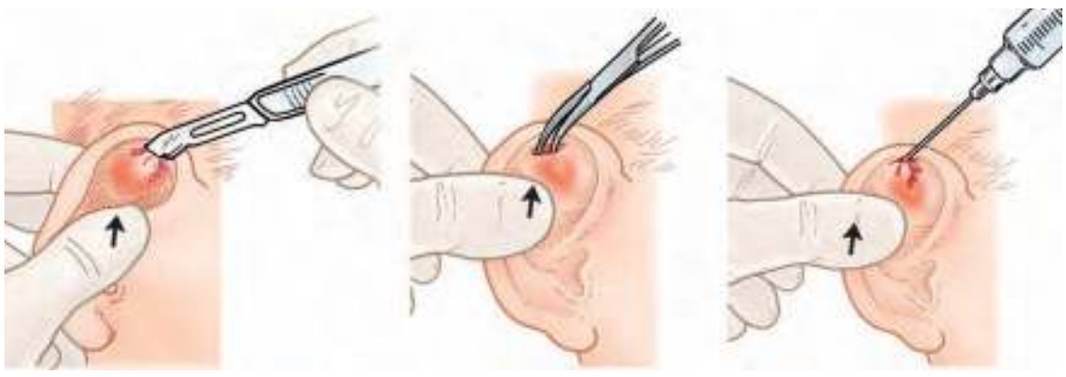


FIGURE 200-8. Incision and drainage of an auricular hematoma. ***A.*** An incision is made along the helical rim. ***B.*** The hematoma is evacuated with the aid of a hemostat. The thumb and index finger express the hematoma from the subperichondral space. ***C.*** The subperichondral pocket is flushed with normal saline to remove any residual blood and clot.

A B C D

FIGURE 200-9. The traditional mastoid dressing. The convolutions are covered with a layer of petroleum gauze. ***A.*** Saline-soaked cotton balls are packed over the convolutions, level with the helical rim. ***B.*** Trimmed gauze squares are placed between the auricle and the head. ***C.*** Fluffed gauze is placed over the auricle. ***D.*** The circumferential application of an elastic gauze bandage to the head. The bandage should cover the injured auricle and not the contralateral auricle.

until they completely fill the area between the auricle and the head. This padding is used to support the auricle as well as prevent undue contortion or uneven pressure on the auricle from the compression dressing.[2]

Unfold and fluff several gauze squares or open a roll of gauze. Place the fluffed gauze over the lateral surface of the auricle (**Figure 200-9*C***). Wrap an elastic bandage snugly over the auricle and around the head to hold the dressing in place (**Figure 200-9*D***). **The circumferential head dressing should not encompass the opposite auricle.** The elastic bandage is used to compress the auricle between the two layers of gauze padding.

SURGICAL PRESSURE DRESSING

A surgically applied pressure dressing may be used instead of the traditional mastoid dressing (**Figure 200-10**).[1,8,19,25] This dressing applies even pressure over the former hematoma site to prevent reaccumulation. It takes more skill to apply than the traditional mastoid dressing. Aseptic technique is mandatory to prevent infection and perichondritis. This dressing requires a skin prep of a much wider area to include the entire medial and lateral surface of the ear and the surrounding skin.

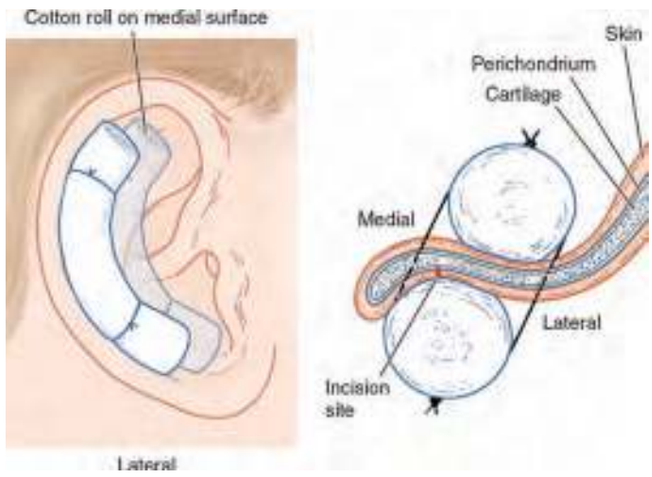


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FIGURE 200-10. A surgically applied pressure bandage. ***A.*** A cotton roll is applied to the lateral surface of the auricle over the site of the evacuated hematoma. A second cotton roll is applied on the medial surface of the auricle directly beneath the first cotton roll. The cotton rolls are secured with 4–0 monofilament nylon suture. ***B.*** Cross-sectional illustration of the surgical pressure dressing. The position of the cotton rolls and suture is seen in relation to the incision site. Note that the perichondrium is apposed to the cartilage and the dead space is eliminated.

The surgically applied pressure dressing has many advantages over the traditional mastoid dressing. It produces pressure exactly where it is needed. The wound is not obscured with a bulky dressing. This allows the patient to easily observe and monitor the site for reaccumulation of fluid and possible infection. This dressing is comfortable and well tolerated by the patient. It is unlike the bulky and conspicuous mastoid dressing that is difficult to keep in place, especially when sleeping. The dressing can remain in place while monitoring the auricle unlike the mastoid dressing that must be removed and replaced daily. The patient can remain active, including taking a shower, without fear of the dressing coming undone. The surgical pressure dressing is adaptable to any location or a variety of hematomas.

Trim a cotton dental bolster or cotton roll to fit the convolution of the auricle over the site of the drained hematoma. Place the cotton roll over the site of the former hematoma (**Figure 200-10*A***). Place a second dental roll on the medial surface of the auricle opposite the first dental roll (**Figures 200-10*A* and 200-10*B***). Place the needle of a 4–0 monofilament nylon suture immediately adjacent to the first cotton bolster. Pass the suture through the entire thickness of the auricle and out the medial surface. Pass the needle over the second cotton bolster and back through the auricle. Snugly tie the suture over the anterior cotton bolster (**Figure 200-10*B***). The suture should be snug enough to allow the cotton bolsters to firmly hold the perichondrium to the cartilage without causing vascular compromise. Apply additional sutures using the same technique until the cotton bolsters are firmly attached and any dead space is eliminated. Depending upon the location of the hematoma, it may require a minimum of two to a maximum of four bolsters to reapproximate the skin and perichondrium and to eliminate all of the dead space.

Monofilament nylon is the preferred suture material for this procedure.[8] It causes less tissue reaction than silk, cotton, gut, or absorbable sutures. It has less of a tendency to cut the ear tissues when tied over the cotton bolsters. Monofilament nylon is less likely to wick bacteria into the auricle and cause an infection than multifilament nylon. The suture may be safely left in place for up to 3 weeks without any complications.

ALTERNATIVE TECHNIQUES

An alternative to packing the pinna with petrolatum gauze is to use either ENT silicone putty or dental impression material.[30,31] Evacuate the hematoma as described previously. Mix the chosen material

to form a uniform composite and activate it. Mold it to fill the pinna, wrapping over the pinna and filling the space between the medial surface of the ear and the head. The putty or impression material will solidify in a few minutes after mixing it and the application. Apply an elastic bandage as described previously. Unfortunately, ENT silicone putty and dental impression material are not often available in the Emergency Department.

The placement of cotton bolsters is often cumbersome and difficult if the Emergency Physician has little experience with this technique. An alternative is to use thermoplastic splinting material.[32] Trim a sheet of thermoplastic splinting material to form a lateral and medial splint of an appropriate size to conform to the lateral and medial surfaces of the ear, respectively. Place the splints in warm water to soften them. Apply the splints to the surfaces of the ear, mold them to the contours of the ear, and apply pressure until they cool and become rigid again. Suture the rigid splints through the ear as described previously.

A second surgical alternative to the use of bolsters is to apply running mattress sutures through the ear to reapproximate the skin and perichondrium.[33-38] Begin posteriorly and weave 4–0 Vicryl or chromic gut suture through-and-through the ear in a running pattern to reapproximate the soft tissues and repair the ear contours. The main advantages of this technique include eliminating bolsters, eliminating the bulky dressing, and the comparable success rates while minimizing the reaccumulation of blood or fluid.

A promising technique involves the injection of OK-432 into the hematoma.[39] This agent was first developed as an immunotherapy for cancer. The advantages of OK-432 injection are that it is simple, is easy to accomplish, and avoids an incision and drainage. There is not enough information to recommend this technique at this time.

Another small study injected fibrin glue after the incision and drainage procedure.[40] This was followed by suture-secured dental bolsters. This seemed to work, allowing healing with no complications. There is not enough information to recommend this technique at this time.

AFTERCARE

The postprocedural care of the auricle is as important as the initial hematoma evacuation. Proper follow-up and care can minimize or prevent any cosmetic deformities. All patients should be seen within 12 to 24 hours to reevaluate the auricle for infection or reaccumulation of the hematoma or serous fluid. Analgesia can be provided by the use of nonsteroidal anti-inflammatory agents. Determine the patient's tetanus immune status and provide prophylaxis as required.

Provide all patients with oral and written instructions regarding the signs and symptoms of cellulitis and perichondritis. This includes the immediate return to the Emergency Department for increasing pain, progressive swelling, redness, tenderness, and warmth. Prophylaxis with oral antibiotics is indicated with any laceration, incision, or puncture of the auricular skin.[4] Prescribe antibiotics for 5 days.[4,5,19] Recommendations include a range of first-generation cephalosporins, antistaphylococcal antibiotics, or antipseudomonal antibiotics.[4,5,19]

Apply the pressure dressing for a minimum of 48 hours.[4] A pressure dressing is recommended for a total of 4 to 5 days. A mastoid dressing must be removed daily to evaluate the auricle and then reapplied. A surgically applied dressing will avoid daily visits to the physician.

Drain any residual clot or serous fluid at the follow-up visit. This can easily be accomplished by needle aspiration and replacement of the pressure dressing.[6,28] If necessary, a sterile drain can be inserted.[9] All drains must be removed within 48 hours of placement to decrease the risk of infection.

COMPLICATIONS

The complications are primarily related to incomplete evacuation of the hematoma or reaccumulation of the hematoma. Incomplete evacuation of the hematoma, if by the aspiration technique, requires an incision and drainage of the hematoma. If necessary, gentle curettage of the cartilage with a hemostat should dislodge the clot. Reaccumulation of the hematoma or serous fluid must be evacuated. Consider placing a sterile drain into the incision.[9] Another option is to place a surgical pressure dressing.[8] A hematoma located within the cartilage will result in a treatment failure and require operative management.[41]

Infection may complicate any surgical procedure. This may be due to reaccumulation or incomplete evacuation of the hematoma which then becomes infected. Strict adherence to aseptic technique will usually prevent infection. Since patients are prophylactically placed on antibiotics, any cellulitis of the auricle at the follow-up visits requires hospital admission and intravenous antibiotics.

Perichondritis is the most feared complication. It is an aggressive and rapidly progressive infection. The auricle will become red, hot, and exquisitely tender if perichondritis develops. This is followed by diffuse swelling and abscess formation. This infection can lead to significant cartilage necrosis and a deformed ear if not promptly treated. *Pseudomonas aeruginosa* is isolated in 95% of patients with perichondritis.[1] Over 50% of cases are polymicrobial with *Staphylococcus aureus* associated with *Pseudomonas aeruginosa*. These patients require Otolaryngology or Plastic Surgery consults for surgical debridement, broad-spectrum intravenous antibiotics, and hospital admission.[1,9]

SUMMARY

An auricular hematoma forms in minutes to hours after blunt trauma to the ear. It presents as a firm and painful swelling that obscures the normal convolutions on the lateral aspect of the auricle. It requires drainage to restore the normal convolutions of the auricle and prevent future deformity. Complete evacuation can be accomplished by needle aspiration or by incision and drainage. After the procedure, prescribe oral antibiotics prophylactically to prevent infection. Follow-up is required within 12 to 24 hours of the procedure to evaluate the patient for reaccumulation of the hematoma and infection. Patients should be educated about the signs and symptoms of perichondritis. Frequent wound evaluation is required until the auricle is healed.

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201 Nasal Medication Administration

Crystal Ives Tallman and Joel Tallman

INTRODUCTION

The rapid administration of lifesaving medications in situations of overdose or epilepsy is of indisputable importance in the prehospital and Emergency Department (ED) settings. Decreasing the time to analgesia for children and adults with painful conditions is a clear priority and the intranasal administration of medications is ideal. It is an important tool for the Emergency Physician to have in their armamentarium. The intranasal administration of medications for anxiolysis in children and adults may limit the need for painful intravenous (IV) line insertion or intramuscular (IM) administration. The old method of dripping the medication from a syringe was not effective. Most of the medication ran into the nasopharynx, was swallowed, and resulted in erratic absorption. Atomizers are now inexpensive, single use, and available in many EDs.

ANATOMY AND PATHOPHYSIOLOGY

The normal physiologic function of the nasal cavities is to warm, humidify, and partially filter outside air. The qualities that make the nose effective physiologically also make it an ideal site for drug administration. The nose has a large surface area of approximately 160 cm^2 with a thin permeable barrier that is 2 to 4 mm thick and an extensive capillary network.[1] The blood flow to nasal mucosa per cubic centimeter of tissue is greater than brain, liver, or muscle tissue.[2] The large mucosal surface with its rich vascular bed provides for excellent absorption and drug bioavailability. Relatively lipid-soluble drugs are rapidly and reliably absorbed without the first-pass metabolism of taking medications orally.[2]

Atomized medication delivery improves bioavailability by increasing mucosal coverage and decreasing the amount of medication lost to swallowing. The maximum absorption is reported for doses of 0.15 mL per nostril.[2] The recommended maximum volume is less than 1 mL per nostril for adults and 0.3 mL per nostril for pediatric patients. Medication may need to be divided into multiple smaller doses to maximize efficacy and minimize patient discomfort. Using a more concentrated formulation of a medication may decrease the number of doses required. Using both nostrils improves absorption.[3]

INDICATIONS

The intranasal administration of medications is indicated for certain medications when rapid delivery is desired. It is also indicated for patients with limited or difficult vascular access. An additional benefit is the avoidance of painful IV line insertion attempts or IM injections. The advantages and disadvantages of intranasal medication are listed in **Table 201-1**. It can be used prior to many ED procedures (**Table 201-2**). The indications and dosing for medications commonly given by the intranasal route are listed in **Table 201-3**.[1-54] The specific characteristics of each medication is noted in Chapter 12.

ANALGESIA

Intranasal fentanyl provides rapid onset of pain relief, results in minimal side effects, and decreases the time to analgesia.[4-13]

TABLE 201-1 The Advantages and Disadvantages of Intranasal Medication Administration

Advantages	Disadvantages
Avoids liver and gastrointestinal first-pass metabolism	Absorption inhibited by blood
Avoids needle anxiety	Absorption inhibited by secretions
Can administer with nausea and vomiting	Atomizer device not available
Fast onset	Chronic nasal conditions affect absorption
Inexpensive atomizers	Drip from a syringe not very effective
Minimal cooperation needed	Limited medications to be administered
Noninvasive	Nasal mucosa burning
No palatability issues	Nasal mucosa irritation
No possible needlesticks	
Plasma profile similar to IV route	
Premedicate special needs children	
Premedicate young children	

Intranasal fentanyl provides effective analgesia for adults and provides equivalent analgesia to IV morphine.[5,14,15] Intranasal ketamine provides effective analgesia in children and adults but is associated with more minor side effects (e.g., dizziness and drowsiness).[18-20] A diamorphine spray is available for nasal pain relief.[21]

ANXIOLYSIS

Intranasal midazolam is effective for anxiolysis and sedation in pediatric patients prior to painful procedures.[22,23] Intranasal midazolam is frequently used in adults for preprocedural anxiolysis with good effects and reduces the need for IV lines. No cases of paradoxical reactions are documented in the literature from the use of intranasal midazolam. Intranasal dexmedetomidine is an effective agent for anxiolysis and sedation prior to procedures or imaging in children.[24-27] It may be more effective than intranasal midazolam.[24,25] Intranasal fentanyl can provide analgesia and sedation for children undergoing painful procedures.[28,29]

OVERDOSE REVERSAL

The intranasal administration of reversal agents can be lifesaving when IV access is not available. Naloxone has been long established as an effective reversal agent for opiate overdose.[30] Intranasal naloxone administration before hospital arrival decreases opiate-related deaths.[31] Intranasal administration is effective and offers a needleless administration alternative.[32] Flumazenil may be administered intranasally for benzodiazepine reversal.[3,33,34]

TABLE 201-2 Some of uses for the Administration of Nasal Medications

Abscess incision and drainage
Bite wound cleaning and repair
Burn wound care
Casting and splinting
Foreign body removal
Joint dislocation reduction
Laceration repair
Lumbar puncture
Nail bed repair
Paracentesis
Patients with nothing by mouth (NPO) status
Premedication prior to procedures
Procedures for special needs children
Procedures in young children
Radiologic imaging (e.g., computed tomography, magnetic resonance imaging, ultrasound)
Seizures
Sexual assault examinations
Status epilepticus
Thoracentesis

TABLE 201-3 Commonly used Intranasal Medications in Children and Adults

Medication	Indication and dosing	Comments
Fentanyl	Pain control: 1–2 µg/kg Sedation: 2–3 µg/kg	Time of onset: 5–10 minutes Duration of action: 30–120 minutes Concentration: 50–1000 µg/mL
Midazolam	Sedation: 0.3–0.5 mg/kg Seizure: 0.2–0.3 mg/kg	Time of onset: average of 5–10 minutes Duration of action: average of 40 minutes; ranges from 30–120 minutes Concentration: 5 mg/mL
Ketamine	Pain control: 0.5–1 mg/kg	Time of onset: 5–15 minutes Duration of action: 30–60 minutes Concentration: 50 mg/mL
Dexmedetomidine	Sedation: 2.5–3.5 µg/kg	Time of onset: average of 12 minutes Duration of action: average of 90 minutes; ranges from 30–120 minutes Concentration: 100 µg/mL
Naloxone	Opioid overdose: 0.4–2 mg Pediatrics: 0.1 mg/kg	Time of onset: 2–3 minutes Half-life: 125 minutes Concentration: 0.2–2.0 mg/mL
Flumazenil	Benzodiazepine reversal: 0.025 µg/kg, up to 400 µg/dose	Time of onset: 1–3 minutes Duration of action: 122 minutes Concentration: 0.1–1.0 mg/mL
Oxymetazoline and lidocaine mixture	Oxymetazoline 1.0–1.5 mL 4% lidocaine 0.5 mL	Can be used in combination or just lidocaine can be used for epistaxis, nasogastric intubation, and nasogastric tubes

Source: Compiled from references 1-54.

SEIZURES

Intranasal midazolam is effective for seizure termination in the home, prehospital setting, and ED.[35-39] Intranasal administration is better tolerated by patients and families than rectal administration.[35-38] Intranasal midazolam is frequently used in the author's ED for seizure termination in pediatric and adult patients when IV access is delayed.

OTHER USES

Intranasal medications have been used for many other diseases. Lidocaine has been used to treat migraine headaches.[40] Anesthesia of the nasal cavity is used prior to epistaxis management, nasogastric intubation, and nasotracheal intubation.[41] Other medications are used for intranasal treatment of migraines (e.g., dihydroergotamine, hydroxocobalamin, rizatriptan, sumatriptan, and zolmitriptan).[42] Ketorolac has been used successfully to treat pain.[43] Intranasal midazolam has broken cyanosis associated with tetralogy of Fallot spells.[44] Ketamine is used on the violent or agitated patient.[45] Vaccines, steroids, and desmopressin for diabetes insipidus have all been successfully used intranasally.[46] Intranasal glucagon has been used for insulin-associated hypoglycemia.[47,48] These uses of intranasal medication administration may eventually be used routinely

in the prehospital setting and in the ED. More uses of intranasal medications can be found at www.intranasal.net.

CONTRAINDICATIONS

A large amount of blood or mucus in the nasal passages limits nasal absorption. Intranasal medication administration is not recommended with significant maxillofacial trauma.

EQUIPMENT

- Medication to be administered in appropriate dose and concentration
- Syringe
- Mucosal atomizer device (**Table 201-4 and Figure 201-1**)

Atomizer devices are often available in the ED. Many devices or available commercially. These are disposable devices that are single patient use (**Figure 201-1 and Table 201-4**). Popular devices are the LMA MAD line of mucosal atomizer devices (Teleflex Medical, Morrisville, NC). There are multiple devices for all topical needs (**Figure 201-1**). The devices make the particle size approximately 30 to 100 μm to enhance absorption through the nasal mucosa.

TABLE 201-4 Some of the Nasal Atomizers Commercially Available*

Name	Manufacturer	Comments
DeVilbiss Model 15 Medical Atomizer	DeVilbiss Healthcare	Disposable and reusable versions Adjustable tip
Enk Fiberoptic Atomizer Kit	Cook Medical	Pressure-resistant oxygen tubing Atomizes through fiberoptic bronchoscope working channel
EZ-Spray	Alcove Medical	Disposable Trigger valve system
LMA MADdy Pediatric Mucosal Atomizer Device	Teleflex Medical	Disposable single use Comes in various colors and shapes Child friendly Attaches to standard syringe 11.4 cm long
LMA Bottle MADomizer Atomizer	Teleflex Medical	Disposable single-use tips Reusable bottle 10 cm long
LMA MADgic Laryngo-Tracheal Atomizer	Teleflex Medical	Disposable single use Attaches to standard syringe Flexible tube 21.6 cm long
LMA MAD Nasal-Intranasal Mucosal Atomizer Device	Teleflex Medical	Disposable single use Attaches to standard syringe

*Some of these devices are not used in the Emergency Department due to the requirement of sterilization between patients.

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. Advise adult patients of the planned route of medication administration and provide a description of the expected procedure to maximize cooperation. Some consider obtaining an informed consent for the administration of nasal medications. This is considered because the patient and nursing staff are not as accustomed to intranasal administration of medication. Pediatric patients may need an appropriate distraction or physical restraint (Chapter 232).

A

B

C

D

FIGURE 201-1. Some common nasal atomizers for topical nasal anesthesia. ***A.*** The LMA MAD nasal. ***B.*** The LMA MADgic laryngeotracheal. ***C.*** The child's LMA MADdy. ***D.*** The MADomizer.

FIGURE 201-2. The administration of intranasal medication using the LMA MAD. (Photo courtesy of www.intranasal.net.)

Apply cardiac monitoring, a noninvasive blood pressure cuff, and pulse oximeter if indicated. Parents use nasal midazolam at home in high concentration without any monitoring.[35] **Sufentanil is the exception and the patient must be monitored.** Sufentanil is more potent than fentanyl, rapidly absorbed through the nasal mucosa, and nasal administration can result in respiratory depression, sedation, and apnea (verbal discussion with T. Wolfe, MD, from www.intranasal.net). Place the patient in a position of comfort (e.g., sitting upright, semirecumbent, or supine). Examine both nares and suction out any blood and/or mucus. **Avoid the nasal cavity if any anatomic abnormalities are noticed.** Draw up the medication to be administered. Use the highest concentration of the medicine to limit the volume administered.

TECHNIQUE

The technique of nasal medication administration is easy, quick, and simple. Position the patient so there is easy access to the chosen nasal cavity. Gently insert the nasal mucosal atomizer device into the patient's nostril (**Figure 201-2**). Apply the medication briskly using the syringe attached to the atomizer or other atomizer device. The brisk application increases atomization, limits dripping of the medication into the nasopharynx, improves efficacy, and minimizes choking. Remove the device from the patient's nostril and allow the medication to take effect. Wipe any drips from the nostril.

ASSESSMENT

Give the medication several minutes to work. The time allowed depends on the medication given and the time for it to work. **Repeat the medication dose if there is no effect, if the patient shows no adverse side effects, and if below the maximum dose. Always remember that all the medication administered intranasally contributes to the maximum dose whether or not the patient shows the desired effect.**

AFTERCARE

Close observation of the patient is recommended after intranasal medication administration. Discourage the patient from blowing their nose or sniffing for several minutes. This ensures the proper time for the medication to be absorbed through the nasal mucosa.

COMPLICATIONS

The most commonly reported complications of intranasal medication administration are bad taste and vomiting.[4] Patients will often experience a burning sensation upon administration of nasal medication. This usually dissipates within several minutes. No cases of respiratory depression or oversedation were reported with the administration of appropriately dosed intranasal fentanyl in children.[4] **This does not imply that these complications never happen.** Intranasal medication administration is generally considered to be as safe as other routes of medication administration.

SUMMARY

Intranasal administration of certain medications for analgesia, anxiolysis, seizure termination, and opiate reversal is safe and effective in children and adults. Intranasal administration may decrease time to medication delivery and decreases the need for IV access. Use of an atomization device is recommended. Careful attention to the volume and dose of medication delivered to each nasal cavity is necessary for maximum efficacy.

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202 Nasal Foreign Body Removal

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INTRODUCTION

Nasal foreign bodies are commonly seen in children, particularly those between 1 and 4 years of age. Young children are naturally curious and spend a great deal of time investigating themselves and the world around them. This involves handling, tasting, and smelling whatever they get their hands on. The Emergency Physician is faced with a foreign body in the nose when these investigations go too far. Adult patients with mental disabilities or psychiatric illness can present with a nasal foreign body. The most common foreign bodies found are beads, food (e.g., corn, nuts, peas, and popcorn), paper, rocks, and toy parts.[1,2] Nasal foreign bodies result from attempts to clean the nasal cavity or to control bleeding with cotton swabs, paper, or sponge material.[3]

Children may present with a known nasal foreign body. Other presentations may be subtle. This includes an odor, halitosis, persistent unilateral nasal discharge, or recurrent epistaxis. Foreign bodies can be found incidentally during a routine examination or on radiographs in the asymptomatic child.[1,4-6]

The task of the Emergency Physician is fourfold.[7,8] Suspect the presence of a nasal foreign body. Perform a thorough physical examination, including a search for the foreign body. Visualize the foreign body. Remove the foreign body efficiently and with minimal trauma.

ANATOMY AND PATHOPHYSIOLOGY

The nasal cavity consists of two passages on either side of the nasal septum. The superior, middle, and inferior bony turbinates project medially into each passage and are covered by a mucous membrane overlying a venous plexus. Foreign bodies can be located anywhere in the nose. Most foreign bodies are found on the floor under the inferior turbinate or anterior to the middle turbinate.[9] The cartilaginous septum is covered by a thin mucosa and receives its blood supply from the overlying mucoperichondrium. Sensory nerves of the nasal cavity are branches of the greater palatine nerve and sphenopalatine ganglion.[10] These nerves are easily numbed with topical anesthetics. The nasal cavity is separated from the orbit by the thin lamina papyracea and from the anterior cranial fossa by the cribriform plate of the ethmoid bone. See Chapter 203 on nasal fracture reduction and Chapter 205 on epistaxis for a more detailed review of nasal cavity anatomy.

A foreign body in the nasal cavity sets off an inflammatory response and the venous plexus becomes congested. Swelling may eventually obscure the foreign body from view. The longer the foreign body remains in the nasal cavity, the more likely the patient is to develop pressure necrosis, granulation tissue, infection, and a purulent discharge. The foreign body can erode into the surrounding tissues over time if it is not removed. A unilateral malodorous discharge and/or epistaxis from a child's nose is a sign of a foreign body.

INDICATIONS

All nasal foreign bodies must be removed to prevent complications. The direct observation of a foreign body in one or both nostrils is an indication for its removal. The presence of signs or symptoms such as unilateral persistent nasal discharge, recurrent unilateral epistaxis, halitosis, or an unusual body odor should prompt a search for a nasal foreign body. Imaging studies are not routine but may be useful in a symptomatic patient where direct visualization is not possible or inconclusive.[11] More than 90% of nasal foreign bodies can be removed by the Emergency Physician with readily available equipment.[1,4,8]

The presence of a disk or button battery in the nasal cavity requires urgent removal.[12-15] These batteries contain a strong alkali, usually potassium hydroxide or sodium hydroxide. The moisture in the nasal cavity may cause corrosion of the battery, leakage of the battery contents, and a low-voltage direct current between the anode and cathode. This may cause liquefaction necrosis, tissue electrolysis, and tissue destruction (i.e., mucosa, cartilage, and bone) within hours.[10,16] These patients need to be seen and followed by an Otolaryngologist after the battery is removed. An electrical thermal burn may cause damage to the nasal tissues that is more extensive than is visible initially.[17,18] Patients may develop a delayed septal perforation and alar collapse from extensive tissue damage.[19]

Small magnets can produce pressure necrosis in the tissues. This is particularly true if they span both sides of the cartilaginous septum or a turbinate. Magnets are commonly used as beads, as clasps for necklaces and bracelets, and in faux piercings.[20,21]

CONTRAINDICATIONS

There are a few contraindications to the removal of a nasal foreign body in the Emergency Department. One contraindication is if the airway is in danger. **Do not attempt to retrieve a nasal foreign body if the patient is in distress or unstable.** This might be due to a posteriorly placed foreign body or an uncooperative patient. **Management of the airway in the Emergency Department or in the Operating Room must take priority to foreign body removal.**

Consult an Otolaryngologist in patients with nasal foreign bodies that are impacted, cause excessive bleeding, or have resulted in nasal perforation or penetration. Foreign bodies located posterior and superior to the middle turbinate pose a risk of being pushed back during retrieval and may perforate the cribriform plate. Larger foreign bodies entering the nose traumatically should be removed by an Otolaryngologist as they may have penetrated the cranial cavity, the orbit, or a sinus cavity.[22,23] Other indications to consult an Otolaryngologist include failure of the Emergency Physician to remove the foreign body after repeated attempts, failure to properly sedate the patient, or the inability to sedate the patient. These contraindications may require foreign body removal in the Operating Room under more controlled circumstances and with equipment not available in the Emergency Department.

EQUIPMENT

NASAL ANESTHESIA AND VASOCONSTRICTION

- 1% or 2% lidocaine solution
- 4% lidocaine solution with 0.25% phenylephrine
- 4% cocaine
- 0.25% phenylephrine (e.g., Neosynephrine)
- 5 mL syringe
- Cotton pledgets
- Atomizer device, if available

MANUAL REMOVAL OF FOREIGN BODY (FIGURE 202-1)

- Head light or head mirror, surgical lamp, or overhead light
- Nasal speculum
- Alligator forceps
- Hartman forceps
- Bayonet forceps
- Lighted forceps (Bionix Medical Technology, Toledo, OH)
- Frazier suction catheter

FIGURE 202-1. Instruments used for the manual extraction of a nasal foreign body. Top row (from left to right): disposable medicine cup, pledgets, nasal decongestant spray. Bottom row (from left to right): 0° telescope, bayonet forceps, nasal speculum, 90° blunt-tipped ear pick or mastoid probe, small alligator (ear) forceps, and large alligator or Blakesley forceps.

- Ear curette
- Blunt mastoid hook
- Wire loop

CATHETER REMOVAL OF A FOREIGN BODY

- Fogarty vascular catheter, size #4 or #5
- Foley catheter, 5 to 6 French
- 5 mL syringe
- Katz Oto-Rhino Extractor (InHealth Technologies, Carpinteria, CA)

POSITIVE PRESSURE

- Cooperative parent
- Bag-valve device
- Face mask, various sizes
- Male-male tube adapter

CYANOACRYLATE TISSUE ADHESIVE

- Cyanoacrylate tissue adhesive
- Cotton-tipped applicator
- Cyanoacrylate removal equipment (Chapter 197)

MAGNET REMOVAL

- Pacemaker magnet
- Metallic medical instrument
- Household magnet

NASAL WASH

- Bulb syringe
- Normal saline

PEDIATRIC IMMOBILIZATION

- Sheets
- Commercial immobilization device (e.g., Papoose board) (Chapter 232)

PATIENT PREPARATION

Discuss the risks, benefits, complications, and different techniques available with the patient and/or their representative. Include the possibility of using procedural sedation initially or if initial attempts at removal are unsuccessful. Obtain a signed informed consent for the removal procedure and the procedural sedation.

The type, shape, size, and location of the foreign body are important factors to consider in choosing the most appropriate technique. The patient's age and ability to cooperate during the examination and removal and the experience and skill of the Emergency Physician can influence the choice of techniques.

Observe universal precautions, especially eye protection, while working in proximity to the mucous secretions of the airway. It is recommended to wear a gown, gloves, and a face mask with an eye shield or goggles. **Carefully inspect both nasal cavities for foreign bodies before and after the mucosa is decongested.** A child will sometimes insert foreign bodies up both nostrils. A good light source is indispensable for examining the nasal cavity and removing the foreign body.

Preparation is important to ensure that the first attempt at retrieval is successful. Have a variety of equipment readily available at the bedside if additional attempts and techniques are required. Have a Frazier suction catheter to clear the nasal passages of blood and/or mucus. **A good hands-free light source (i.e., overhead light or a head lamp) is important in directly visualizing the foreign body and facilitating its removal.**

Anesthesia of the nasal mucosa is obtained by the topical application of lidocaine to a maximum of 4 mg/kg or cocaine to a maximum of 3 mg/kg. Cocaine has the added benefit of vasoconstriction and decongestion of the nasal mucosa. Add a vasoconstrictive agent (i.e., epinephrine or phenylephrine) to lidocaine if no contraindications exist. A syringe can be used as a dropper to apply the medication intranasally (Chapter 201). This is best done by administering several drops at a time and then reassessing visibility before adding more. **The patient is more apt to blow the medication out their nose before it can take effect if the entire dose is added at one time.** An atomizer may be used. Some of these devices attach to a syringe or a nasal decongestant container. Atomizers are disposable devices that are single patient use (**Figure 201-1 and Table 202-1**). A popular device is the MAD line of mucosal atomizer devices (LMA North America, San Diego, CA). They make multiple devices for all topical needs. Allow 5 to 10 minutes for the medication to take effect.

It would be appropriate to have the patient attempt to blow the foreign body out of their nostril if the patient is cooperative. Have the patient sit up and lean forward. Instruct the patient to blow forcefully through their nose while covering the uninvolved nostril with a finger. Even if this has failed at home, it may work with the nasal mucosa swelling alleviated by the decongestants.[19]

Most children will need to be restrained while the foreign body is being removed. The child who appears cooperative and is adequately anesthetized may move suddenly while instruments are in the nasal cavity. One method is for the child to sit on their parent's lap (**Figure 202-2**). The adult should cross their legs over the child's legs, using one arm to restrain the child's arms and trunk and the other arm to hold the child's forehead (**Figure 202-2**). Alternatively, instruct an assistant to hold the patient's head while the parent controls the body and limbs. Another alternative is to wrap the child in

TABLE 202-1 Some of the Commercially Available Atomizers

Name	Manufacturer	Comments
DeVilbiss Model 15 Medical Atomizer	DeVilbiss Healthcare	Adjustable tip Disposable and reusable versions
Enk Fiberoptic Atomizer Kit	Cook Medical	Atomizes through fiberoptic bronchoscope working channel Pressure-resistant oxygen tubing
EZ-Spray	Alcove Medical	Disposable Trigger valve system
LMA MADdy Pediatric Mucosal Atomizer Device	Teleflex	11.4 cm long Attaches to standard syringe Child friendly Comes in various colors and shapes Disposable single use
LMA Bottle MADomizer Atomizer	Teleflex	10 cm long Disposable single-use tips Reusable bottle
LMA MADgic Laryngo-Tracheal Atomizer	Teleflex	21.6 cm long Attaches to standard syringe Disposable single use Flexible tube
LMA MAD Nasal-Intranasal Mucosal Atomizer Device	Teleflex	Attaches to standard syringe Disposable single use

FIGURE 202-2. Method to properly restrain a child.

FIGURE 202-3. The Lighted Forceps for Foreign Body Removal. (Photo courtesy of Bionix Medical Technologies, Toledo, OH.)

a sheet or use a commercial restraint device (e.g., Papoose board) (Chapter 232).[19]

Uncooperative patients may require anxiolysis, procedural sedation (Chapter 159), or general anesthesia prior to removal of the foreign body. Procedural sedation may be used to facilitate foreign body removal in the uncooperative or fearful patient. Approximately 21% of pediatric patients with nasal foreign bodies required procedural sedation.[4] The nasal foreign body was successfully removed in 95% of these cases. Ketamine was the most commonly used agent.

TECHNIQUES

DIRECT INSTRUMENTATION OR MANUAL REMOVAL

The most straightforward and common method is to remove the anteriorly located nasal foreign body under direct vision. Do not attempt instrument removal if the foreign body is located posteriorly. The instruments most often used include alligator forceps, bayonet forceps, straight forceps, or mosquito forceps. Hooked probes (e.g., right angle or curved hooks, ear curettes, wire loops, or mastoid hooks) can be utilized. A novel device is the Lighted Forceps for Foreign Body Removal (Bionix Medical Technologies, Toledo, OH). It is a single patient use and disposable device that contains a light source and acts like forceps (**Figure 202-3**).

The EasiEar Disposable Comfort Curette (Splash Medical Devices LLC, Atlanta, GA) is an improvement to the standard disposable plastic curette (**Figure 202-4**). It is a stainless steel, single patient use, and disposable curette. The rounded wire head is smooth. It lacks the jagged and sharp plastic edges that are often found on molded plastic curettes. The EasiEar has no abrasive edges, seams, or surfaces to potentially abrade the nasal mucosa. This design may prevent abrasions, bleeding, and lacerations. The spring wire shaft provides flexibility and enhanced maneuverability when compared to molded plastic curettes. The angled head and flexible shaft allow it to be manipulated within the nasal cavity to remove a foreign body.

The above instruments enable the Emergency Physician to grasp the foreign body directly or pull it out from behind. Anteriorly located foreign bodies are often easily removed with instrumentation. Forceps are better suited for soft and irregularly shaped foreign

FIGURE 202-4. The EasiEar Metal Curette (Splash Medical Devices LLC, Atlanta, GA).

FIGURE 202-5. Using forceps to remove a thumb tack lodged in the nostril.

bodies (**Figure 202-5**).[9,24] Curettes and hooks are more effective for hard and spherical foreign bodies.[9,24]

Insert the nasal speculum to hold the nostril open. Adjust the headlight or head mirror to illuminate the nasal cavity. **It cannot be overemphasized how crucial adequate light and visibility are to successfully remove the foreign body.** Remove any mucus or blood with the Frazier suction catheter. Grasp an irregularly shaped foreign body with forceps (**Figure 202-5**). Forceps may cause a round or smooth foreign body to slip farther posteriorly when the jaws close. Pass a curette, wire loop, or mastoid hook behind a round or smooth foreign body and pull it out (**Figure 202-6**).[19,25] The jaws of a small alligator forceps can be passed through the opening of a bead if it is facing outward (**Figure 202-7**). Open the jaws when they are beyond the lumen of the bead and pull the bead from the nasal cavity.

Relative contraindications to instrumentation include posteriorly located foreign bodies, friable foreign bodies, round foreign bodies, and smooth foreign bodies. Potential complications include posterior displacement of the foreign body leading to aspiration, epistaxis, lacerations, mucosal abrasions, and nasal obstruction.

BALLOON CATHETER EXTRACTION

Some Emergency Physicians prefer to use a catheter with a balloon to pull foreign bodies from the nasal cavity. This technique has a reported success rate of 90% and is used most successfully for foreign bodies that are round, smooth, and cannot be grasped readily.[26] **A balloon catheter does not work if the foreign body fully obstructs the nasal passage.** Authors describe using a variety of catheters

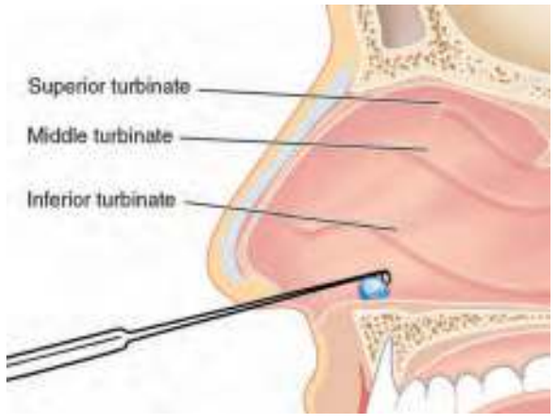

FIGURE 202-6. Removing a round, smooth foreign body from the nasal cavity. The curette, mastoid hook, or wire loop is passed through the nares and behind the foreign body. The hook is used to pull the foreign body out of the nasal cavity.

(e.g., a #4 to #8 Fogarty vascular catheter, a #6 Fogarty biliary catheter, and a 5 to 6 French Foley balloon catheter).[4,19,27,28]

Test the balloon on the catheter device. Inflate the balloon to ensure it has no leaks. Deflate the balloon. Lubricate the catheter. Insert the catheter until the balloon is beyond the foreign body. The catheter may be placed above, below, or to the side of the foreign body.[19] Inflate the balloon with 2 to 3 mL of air. Gently pull the catheter. The balloon will push the foreign body out of the nostril. A balloon catheter can be used to stabilize a foreign body from behind while it is removed with forceps.

The disadvantages of this method are that it is more traumatic and epistaxis is more common.[2] The Emergency Physician risks pushing the foreign body posteriorly and impacting it, obstructing the nasal passage, or dislodging it into the airway if the catheter is not passed under direct visualization or if it is too large to pass around the foreign body.

KATZ EXTRACTOR

The Katz Extractor Oto-Rhino Foreign Body Remover (InHealth Technologies, Carpinteria, CA) is a device designed to extract foreign bodies from the nasal and auditory passages (**Figure 202-8**). It is a disposable, single-use device consisting of a balloon-tipped catheter attached to a syringe. Always test the device before using it. Push the plunger to inflate the balloon and inspect it for any air leaks. Release the plunger to deflate the balloon.

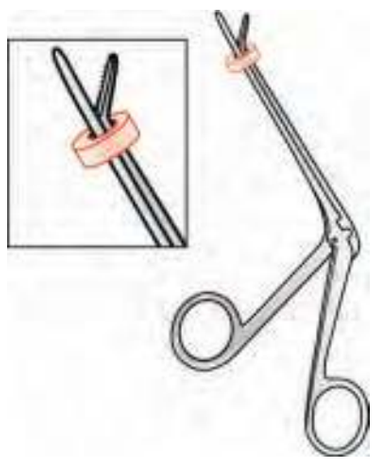

FIGURE 202-7. An alligator forceps is used to remove a bead or other foreign body with a hollow center.

FIGURE 202-8. The Katz Extractor. The balloon at the tip inflates and deflates by pushing and releasing the plunger, respectively. (Photo courtesy of InHealth Technologies, Carpinteria, CA.)

Grasp the device with the dominant hand (**Figure 202-9**). Gently insert the catheter along the wall of the nasal cavity until the balloon is just past the foreign body (**Figure 202-9*A***). Inflate the balloon by depressing the plunger on the syringe (**Figure 202-9*B***). Withdraw the catheter and foreign body from the nasal cavity while maintaining the balloon in the inflated state (**Figure 202-9*C***). Insert the catheter through the hole, rather than behind it, and inflate the balloon if the foreign body has a central hole (e.g., candy or bead).

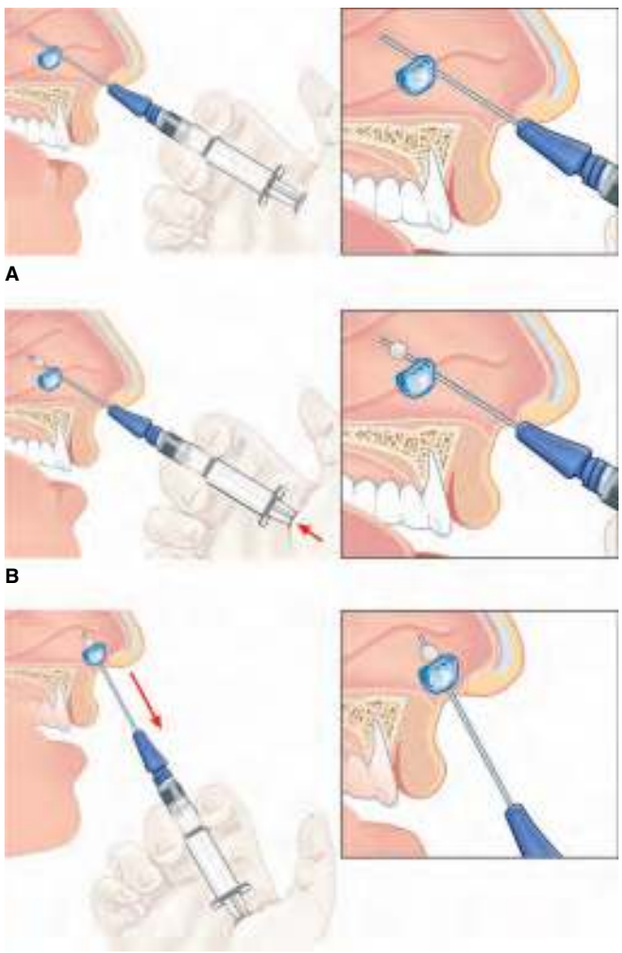

FIGURE 202-9. The Katz Extractor removing a nasal foreign body. ***A.*** The device is inserted until the balloon is just past the foreign body. ***B.*** The balloon is inflated. ***C.*** The Katz extractor is removed with the balloon inflated and the foreign body is removed.

FIGURE 202-10. The Hognose otoscope tip attached to an otoscope. (Photo courtesy of IQDr. Incorporated, Manitou Springs, CO.)

HOGNOSE OTOSCOPE TIP

The Hognose (IQDr. Incorporated, Manitou Springs, CO) is a disposable, latex-free, single-use device that attaches to a standard otoscope (**Figure 202-10**). It comes in three sizes (i.e., 3, 4, and 5 mm) with a color-coded tip. The size represents the cup size at the tip of the device. The tip is soft, self-molding, and looks like the nose of a hog. It has an insufflation port and suction tubing attached to its side. The adapter on the suction tubing attaches to standard wall suction tubing.

Attach the Hognose to the otoscope as if attaching a disposable speculum to an otoscope. Turn on the otoscope light source. Attach the hognose tubing to suction tubing and a suction source. Turn the suction source to low or medium. Grasp the otoscope with the dominant hand. Insert the Hognose into the nasal cavity while visualizing the foreign body through the otoscope head. Place an index finger over the insufflation port to engage the suction at the device tip when it is next to the foreign body. Gently advance the otoscope until the tip of the Hognose is against and attached to the foreign body. The soft tip has collapsed on itself if you suddenly see black through the otoscope. Remove the finger over the insufflation port and reapproach the object. Withdraw the Hognose with the foreign body attached while maintaining suction.

GATORNOSE OTOSCOPE TIP

The Gatornose (IQDr. Incorporated, Manitou Springs, CO) is a disposable, latex free, single-use device that attaches to a standard otoscope (**Figure 202-11**). It twists onto an otoscope like a speculum. It comes with three different jaw types that attach to the body of the

FIGURE 202-11. The Gatornose base attached to an otoscope. Note the three types of jaws that are available to snap onto the base of the device. (Photo courtesy of IQDr. Incorporated, Manitou Springs, CO.)

device. These jaws are small flat jaws, large flat jaws, and open loop jaws. A trigger on the body of the device controls jaw opening and closing.

Attach the Gatornose base to the otoscope as if attaching a disposable speculum to an otoscope. Apply the appropriate arms onto the Gatornose base. Turn on the otoscope light source. Grasp the otoscope with the dominant hand. Insert the ring finger into the trigger. Pull the trigger to close the Gatornose jaws. Gently insert the Gatornose jaws just into the nasal cavity. Push the trigger to open the Gatornose jaws and view the nasal cavity through the otoscope. Gently advance the otoscope while visualizing the foreign body through the otoscope head. Position the jaws above and below or anterior and posterior to the foreign body. Pull the trigger to close the jaws onto the foreign body. Withdraw the otoscope with the foreign body in the jaws of the Gatornose.

SUCTION TECHNIQUE

Frazier suction catheters are most useful with small or round foreign bodies where suction can be maintained between the device and the foreign object. Suction is relatively contraindicated if the foreign body is shaped so that the suction device cannot form a seal or if the foreign body is friable. This technique will be otherwise unsuccessful or will push the object farther into the nasal cavity. This technique is best reserved for large, round foreign bodies where suction can be maintained between the device and the foreign body. Complications include trauma to the surrounding tissues and epistaxis.[29]

Attach the Frazier suction catheter to the suction tubing. Turn on the suction source to at least 100 mmHg. Gently insert the catheter into the nasal cavity. Place a thumb over the hole in the catheter handle to direct the suction through the tip of the catheter. Gently advance the suction catheter until the tip is in contact with the foreign body. Withdraw the Frazier suction catheter and foreign body from the nasal cavity.

Use suction with plastic intravenous tubing for impacted smooth, spherical objects.[30] Cut a short length of plastic intravenous tubing and attach one end to the suction source. Fashion the other end into a small flange shape using a heat source and any metal object with a rounded end (e.g., the tip of a hemostat or larger clamp). Heat the jaws of the hemostat and insert them into the plastic tubing just enough to create a flange. Turn on the suction source. Place a hemostat onto the tubing to temporarily clamp the suction tubing. Gently advance the flange tip into the nasal cavity until it contacts the foreign body taking care not to push it inward. Have an assistant remove the hemostat from the tubing to activate the suction. Gently but quickly remove the tubing and attached foreign body from the nasal cavity.

POSITIVE PRESSURE

This technique is best utilized for foreign bodies that are large, posteriorly located, or occlude the nasal passage.[31-33] It involves the generation of positive pressure in the nasopharynx behind the foreign body to force it out of the nostril. There are several ways to generate positive pressure within the nasal cavity. One is to simply ask the patient to occlude the unaffected nostril, take a deep breath through their mouth, close their mouth, and then forcefully exhale the air out through the nostril with the foreign body while keeping their mouth closed. This technique is most successful in adolescents and adults who are cooperative and can coordinate the movements. The advantage to this technique, if it is successful, is that no instruments are placed into the nose. The disadvantage is that the foreign body can be forcefully expelled from the nasal cavity. Eye protection is advised.

Other variants of this technique have been developed. The "big kiss" is also known as the "mother's kiss," "parent's kiss," or "mouth-to-mouth" technique (**Figure 202-12**).[34-39] It involves asking the parent to blow into the child's mouth. This is like rescue breaths during cardiopulmonary resuscitation (CPR). Explain the procedure to the child and the parent. Instruct the parent to open the child's mouth with one hand and stabilize the chin while occluding the unaffected nostril with a finger from their other hand. Instruct the parent to open their mouth, take a big breath, and then place their mouth over the child's open mouth. The child's mouth must be completely covered by their parent's mouth and a good seal formed. Instruct the parent to deliver a sudden and forceful breath into the child's mouth.

FIGURE 202-12. The "mother's kiss." (Photo used with permission from reference 34.)

This entire sequence of events should only take 3 to 4 seconds to complete.

This maneuver causes the child's glottis to close. The foreign body will be expelled from the affected nostril if enough pressure is generated. This technique may be less traumatic to the child and involves no instrumentation or restraint.[34-39] High success rates have been reported.[34-39] This technique may be difficult for some parents to perform.

A modified version of this technique involves using a drinking straw or small tube between the child's mouth and parent's mouth.[31] The parent gives a big puff of air through the straw while the child tries to make a tight seal between their mouth and the straw. The Emergency Physician can perform this technique without placing their mouth on the child's mouth.[31]

A bag-valve-mask device (i.e., the Ambu-bag) can be used to blow the foreign body out of the nostril.[40] Choose a face mask that is small enough so that it only covers the patient's open mouth. Cover the patient's mouth tightly with the mask. Close the unobstructed nostril with a finger (**Figure 202-13**). Squeeze the bag to force air into the mouth and lungs and out the nose.[40] A variant of this method uses an anesthesia bag connected to high-flow oxygen at 10 to 15 L/min. Cover the mouth with the mask, close the thumb hole, and allow the bag to expand and gradually increase the airway pressure. If this does not expel the foreign body, compress the bag.[4]

The "Beamsley Blaster" technique makes use of the positive pressure generated from the application of high-flow oxygen at 10 to 15 L/min from tubing attached to an oxygen wall outlet.[41,42] The other end of the tubing is attached to a male-male adapter that is inserted into the unaffected nostril. The set-up generates enough pressure in the posterior nasopharynx to dislodge the foreign body from the nostril. The patient's mouth must be closed during the procedure to create a seal. There were no complications noted in the initial study. A subsequent case of barotrauma (e.g., subcutaneous orbital emphysema) has been reported.[43] An alternative is to use a 60 mL syringe with air.

Positive-pressure methods are contraindicated if there are foreign bodies in both nasal passages. It is important to be sure that air is forced in the open nasal cavity or the foreign body could be blown into the trachea or esophagus. Blow short puffs of air during the child's cries. The soft palate will close when the child is vocalizing and direct both the air and the foreign body out of the other nostril.[2]

FIGURE 202-13. Bag-valve-mask method of removal. The child is restrained and the contralateral nostril is occluded. Bag-valve-mask is applied on the mouth to create a seal and a few breaths are delivered until the foreign body is expelled.

CYANOACRYLATE TISSUE ADHESIVE ASSISTED REMOVAL

Cyanoacrylate tissue adhesive can be used to extract spherical and other solid foreign bodies that are visible.[44] This technique can be fraught with complications. There is a chance of gluing the foreign body to the nasal mucosa as well as creating a glue foreign body. The foreign body can be further impacted if pushed posteriorly or fall into the oropharynx and aspirated.

Obtain a cotton-tipped applicator. Moisten the tip of the applicator stick with a very tiny amount of cyanoacrylate glue. A larger amount can drop off into the nasal cavity. Insert the applicator stick before the glue dries and until it just touches the foreign body. Maintain this position for 30 to 60 seconds to allow bonding of the glue to the foreign body. Do not allow the drop of glue to fall from the stick and bond to the nasal mucosa. Do not touch the tip of the wooden applicator stick to the nasal mucosa as it will bond to the mucosa. Remove the applicator stick with the foreign body attached. The main disadvantages of this technique are potential bonding of the nasal mucosa and the time it takes for the glue to bond to the foreign body.

ALTERNATIVE TECHNIQUES

NASAL WASH

The nasal wash technique involves the introduction of high-pressure normal saline through a bulb syringe into the unaffected nasal cavity. It is best used with friable and nonorganic materials that are posterior in location.[31] Place approximately 7 mL of normal saline into a bulb syringe. Insert the bulb syringe into the unaffected nostril. Advance the bulb syringe into the nostril until a seal is created. Forcefully squeeze the bulb syringe. The pressure generated is believed to expel the saline and the foreign body from the other nostril.

The use of this technique carries a high risk of aspiration of saline and the foreign body. The saline may reflux into the sinuses or eustachian tubes. Its use should be restricted to older patients who can cooperate and protect their airway.[11,45] **Do not use this technique in patients with a button battery or organic matter in their nasal passages.** The increased moisture will hasten corrosion of the battery and cause swelling of the organic matter making additional attempts at removal more difficult. Do not use this technique in young infants, those with airway abnormalities, or those with neurologic abnormalities.

MAGNET REMOVAL

An occasional patient will present with a mini-magnet adhered to the nasal mucosa. The source of the magnets can be from their insertion in young children or nasal jewelry.[20] Adherence of magnets across the nasal septum creates the potential for septal necrosis and perforation.[21,46,47] They may be difficult to remove because of the attractive force created across the nasal septum.

Numerous methods can be used to facilitate magnet removal. They may be grasped with a variety of forceps or hemostats. A hook can be made from a nonferromagnetic blunt probe.[20] A magnet can be used to facilitate nasal magnet removal.[21,48,49] A pacemaker

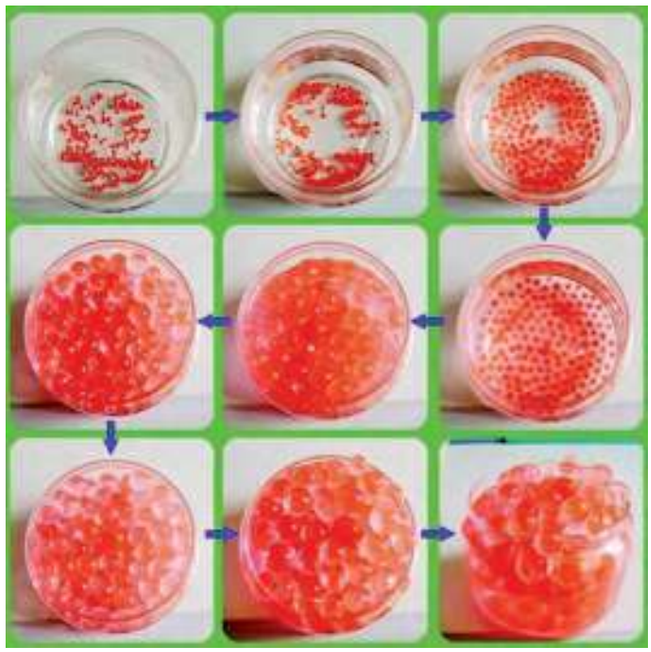

FIGURE 202-14. Hydration of expanding beads.

magnet is readily available in most Emergency Departments. A small and strong household magnet may be available from the hospital maintenance department. Metallic objects that are ferromagnetic may be removed with the aid of other magnets. One method includes magnetizing a medical instrument (e.g., forceps or a hemostat) by placing it in series with a pacemaker magnet. The instrument can then be placed in the nasal cavity to attract the metallic foreign body. Another method is to use a household magnet at the entrance of the nasal cavity in attempts to attract the foreign body. The strength of the magnet and the distance of the object may limit this method.[48,50]

EXPANDING BEADS

These beads are small, brightly colored, and resemble candy. The beads swell when in contact with liquids (e.g., nasal secretions or water) (**Figure 202-14**).[51,52] These are commonly used as decorations, to preserve flowers, and as toys. Children can smell the beads to determine if they are flavored and accidentally inhale them into their nose. They can be difficult to remove when swollen. Do not use fluid irrigation techniques in removing these foreign bodies.

ASSESSMENT

Inspect the nasal cavity for any remaining fragments of the foreign body, abrasions, bleeding, lacerations, a second foreign body, or ulcerations. A gray precipitate may be noted if a disk battery has been removed. Irrigate the nasal cavity with saline after a battery is removed to eliminate any electrolyte solution or precipitate to prevent further damage.[22] Bleeding can be controlled with topical phenylephrine or epinephrine, pressure, or any of the methods described to control epistaxis (Chapter 205).

AFTERCARE

Discharge the patient home with nasal saline spray to the affected nostril until secretions are clear of blood or pus. Oral antibiotics may be prescribed if the patient has developed sinusitis from a retained foreign body. Patients with ulcerations on opposing sides of the nasal cavity must be followed-up to prevent obstructive synechiae or adhesions from forming. Patients with button batteries removed, mini-magnets removed, injuries, or complications should follow-up with an Otolaryngologist. Educate the caretakers to remove any small objects from the child's reach, supervise children when they have access to small objects, and childproof the house.

COMPLICATIONS

Complications can arise from the nasal foreign body or the removal technique.[15,16] Foreign bodies can cause aspiration, epistaxis, infection, nasal obstruction, perforation, pressure on surrounding tissues, or ulceration.[9] The risk of complications increases the longer the foreign body is present. Epiglottis, facial cellulitis, meningitis, orbital cellulitis, otitis media, and sinusitis have been associated with retained nasal foreign bodies.[9,18]

The most significant complication would be to dislodge the foreign body into the airway. This is most likely to occur in an uncontrolled situation. Placing the patient supine and in the Trendelenburg position may prevent accidental aspiration of the foreign body.[53] **Consider the use of sedation, procedural sedation (Chapter 159), or general anesthesia if the patient is uncooperative**.

Potential complications associated with each technique were described with each specific technique.[54] Aspiration from posterior dislodgment of a nasal foreign body remains a serious potential complication for all of the techniques. Positive-pressure techniques can result in barotrauma. Periorbital emphysema has been described using the "Beamsley Blaster" method.[43] Secondary infection may result from the procedure. Antibiotics are generally not indicated in most cases once the foreign body has been removed. Complications related to manipulation within the nasal cavity include bleeding or subsequent infection. An anterior nasal pack may be required in rare instances to tamponade bleeding. Toxic doses of cocaine and/or lidocaine may result in cardiac arrhythmias and seizures.

SUMMARY

Most nasal foreign bodies can be safely removed by the Emergency Physician with instruments that are readily available in the Emergency Department. There are multiple techniques for removing foreign bodies from the nasal cavity. Each has its own advantages and disadvantages. These methods include the use of balloon catheters, forceps, and positive pressure. Choose the method according to the shape and location of the foreign body, the tools available, the cooperativeness of the patient, and experience with the different removal techniques.

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203 Nasal Fracture Reduction

Eric F. Reichman

INTRODUCTION

Nasal fractures due to blunt trauma are a common occurrence (**Figure 203-1**). Fights, auto accidents, and sports accidents account for most fractures in an urban setting.[1-4] Work, farm, sports, or leisure activity accidents account for most of these injuries in rural

FIGURE 203-1. A photograph of a patient with a nasal fracture. (Used with permission from Shah BR, et al: *The Atlas of Pediatric Emergency Medicine*, 2nd ed. New York: McGraw-Hill; 2013.)

areas.[5] The majority of nasal fractures occur in males aged 15 to 25 years old, with fights being the major etiology.[6-9] Nasal fractures are often missed on initial evaluation, especially when there are many more urgent trauma concerns. It is best to perform closed or open reduction of a nasal fracture within the first 2 weeks when it is easiest to avoid more elaborate operations later to correct the disfigurement and nasal airway obstruction. Perform the reduction in children within 3 to 7 days as fracture fixation occurs faster than in adults.[10]

Suspect a nasal fracture in the blunt trauma patient with a history of epistaxis, new-onset nasal blockage, or a change in nasal appearance. Determine whether the patient has a prior history of a nasal bone fracture as repeat nasal bone fractures will be more difficult to reduce. An old photograph of the patient may aid this determination. One study revealed that 30% of injured noses had a preexisting nasal deformity.[11] At least 48% of the general population has a deviated nasal septum.[12] Physical examination of the blunt trauma patient may demonstrate skin lacerations, nasal tenderness and mobility, internal mucoperichondrial tears, ecchymosis, or a septal hematoma. A septal hematoma must be drained to avoid cartilage necrosis and the subsequent saddle nose deformity. Refer to Chapter 204 regarding the complete details of managing a nasal septal hematoma.

ANATOMY AND PATHOPHYSIOLOGY

The mechanism of injury, force of impact, direction of impact, and history of any prior nasal deformity must be ascertained from the patient in order to understand the potential magnitude of the fracture. If possible, obtain photographic documentation before any attempts at nasal manipulation. The Waters and lateral nasal radiographs will often support the physical findings of a nasal fracture (**Figure 203-2**). Many Surgeons recommend radiographs as part of the medical legal documentation, although many do not agree. Plain radiographs can have a high false-negative rate due to the lack of fine resolution or a high false-positive rate from the misinterpretation of the normal bony sutures.[13,14] A computed tomography (CT) scan is more sensitive and specific to identify a nasal bone fracture. Unfortunately, its cost and the radiation exposure are significant. A nasal fracture can often be diagnosed based upon the history and the physical examination. Reserve the CT scan for those patients with suspected nasal fractures who may have additional injuries requiring a CT scan.

A bedside ultrasound can be used to evaluate the patient for a nasal fracture (**Figures 203-3 and 203-4**).[15-21] Use a high-frequency transducer with the smallest footprint available. A clear container containing water or saline can be placed over the patient's nose to get a clearer view using the ultrasound transducer.[22] Ultrasound was superior to plain films to assess the lateral nasal walls whereas plain films were superior to assess the nasal dorsum.[15] Ultrasound may become the imaging modality of choice as it has better accuracy than plain films and CT scans.[16,17]

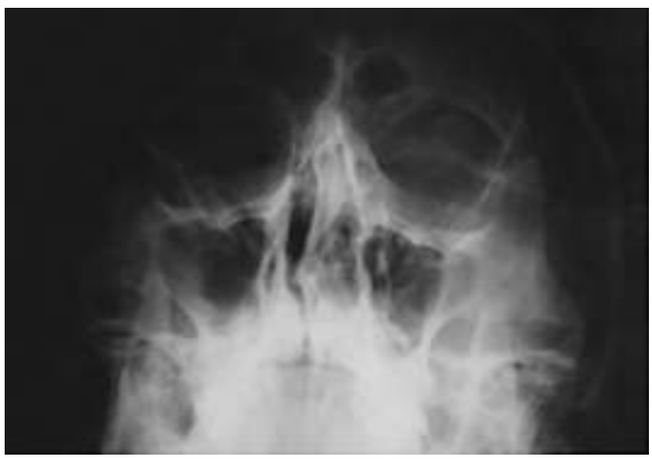
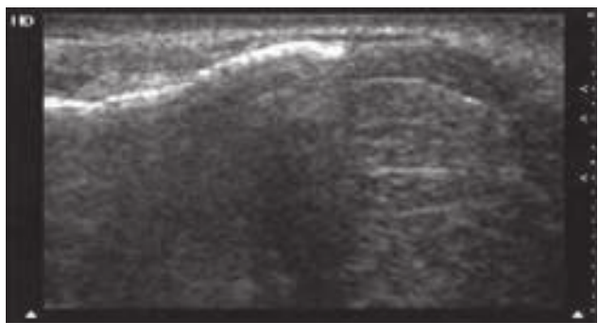


FIGURE 203-2. Radiograph demonstrating a Waters view of a deviated nasal septum and right nasal bone fracture.

A



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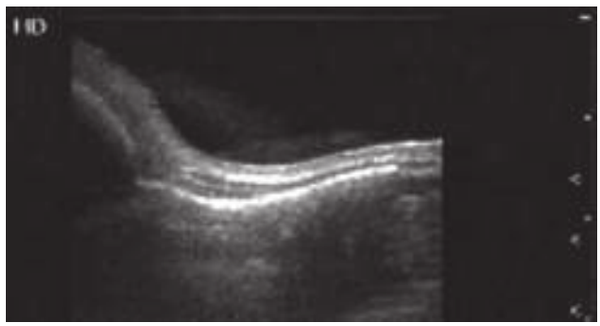


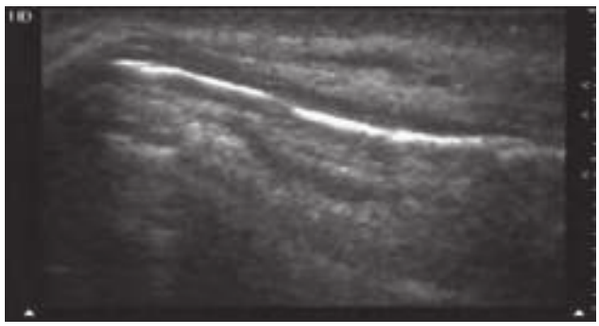
C

FIGURE 203-3. Ultrasonography to assess the nasal pyramid. ***A.*** Transducer placement. ***B.*** The normal nasal bone. ***C.*** The normal nasal bone and part of the frontal bone. (Used with permission from Ma OJ, et al: *Ma & Mateer's Emergency Ultrasound*, 3rd ed. New York: McGraw-Hill; 2014.)

The direction of the force to fracture the nasal bone is variable. There are several lateral and frontal force injury classifications but no consensus exists.[9,23,24] One study demonstrated that 66% of nasal fractures were due to lateral forces, 13% were from frontal forces, and 21% were from mixed forces.[7] This predominance of lateral force fractures is due to several factors.[25] It takes more than twice the force, in cadavers, to cause a frontal impact fracture than to cause a lateral impact fracture.

A

B



C

FIGURE 203-4. Ultrasonography to assess the nasal pyramid. ***A.*** Assessment of the patient's right side. ***B.*** Assessment of the patient's left side. ***C.*** The normal nasal bone, the slightly hypoechoic nasomaxillary suture, and the frontal process of the maxilla. (Used with permission from Ma OJ, et al: *Ma & Mateer's Emergency Ultrasound*, 3rd ed. New York: McGraw-Hill; 2014.)

Always rule out any associated maxillofacial injuries, especially when the patient presents with ocular hypertelorism and lid lateral-pull laxity.[26] Thoroughly evaluate the patient for the presence of other head or neck injuries if they have a nasal fracture. Associated head and neck injuries take priority for management and evaluation as they can result in significant morbidity. Subcutaneous emphysema may be found in nasal trauma cases as well as with simple lamina papyracea fractures and nose blowing. Scleral chemosis, subconjunctival hemorrhage, eyelid edema, periorbital ecchymosis, or subconjunctival hemorrhage may all be associated with a complex facial fracture or an isolated nasal fracture. A history of anosmia indicates a possible cribriform plate fracture. Evaluate the patient for cerebrospinal fluid rhinorrhea and the presence of beta$_2$-transferrin.[5] Consider obtaining axial and coronal CT scans of the paranasal sinuses with possible cisternography.

The nose consists of the external nose, the bilateral nasal cavities, and the nasal septum. The external nose consists of the bony upper third, the cartilaginous lower third, and the surrounding soft tissue and skin (**Figure 203-5**). The bony part consists of the paired nasal bones, the frontal processes of the maxillary bones, and the nasal processes of the frontal bones. The inferior nasal bones are thinner than the superior nasal bones. Thus, the lower bony portion is the most common area of the external nose to be fractured.

The cartilaginous components consist of the arched paired upper lateral cartilages and the horseshoe-shaped paired lower lateral cartilages. The former articulates with the cartilaginous septum and nasal bones, while the latter has ligamentous attachments to each other in front of the cartilaginous septum. The lower and upper lateral cartilages articulate with each other and form a crucial part of the nasal valve, the critical control point of nasal resistance and obstruction. Any of these relationships can become dislocated from a traumatic impact and will require repair for an optimal functional and aesthetic outcome. The more lateral sesamoid cartilages are relatively unimportant in these regards.[27]

The bony nasal septum consists of the perpendicular plate of the ethmoid bone, the vomer, the anterior nasal spine, the maxillary crest, and the palatine bone (**Figure 203-6**). The perpendicular plate of the ethmoid bone is thin except at its articulation with the vomer and cartilaginous septum. **The perpendicular plate of the ethmoid bone is the most common location for a septal fracture.** The quadrangular cartilage of the septum is anteriorly located on the maxillary crest. It can be dislocated into either nostril with a significant force, especially from an inferior direction, and result in nasal airway obstruction.[9]

The inner walls of the nose contain an extensive network of nerves and blood vessels (**Figures 203-7 and 203-8**). These networks form an anastomosis within the walls of the nasal cavity. This is the reason injuries to the nose hurt and bleed so much.

The bony nasal septal fracture is often similar regardless of whether the etiology is a frontal or lateral impact (**Figure 203-9**). It usually begins just posterior to the nasal spine at or just above the maxillary crest. The fracture can progress through this area and straight into the vomer where it curves like a "C" vertically upward and into the perpendicular plate of the ethmoid bone.[28]

Nasal fractures increase in severity from a unilateral depression without a septal fracture, to a nasal twist or deviation, to a significantly comminuted nasal fracture as the frontal or lateral force of impact increases (**Figure 203-10**). Between these extremes are the moderately severe bilateral fractures with the contralateral side being driven outward and more significantly impacted traumas, such as the previously described C-shaped septal fracture with overriding and interlocked fragments. This latter condition often appears as the classically shortened and twisted nose with tip ptosis from columellar retraction. There is often significant nasal airway obstruction as the quadrangular cartilage telescopes over the perpendicular plate

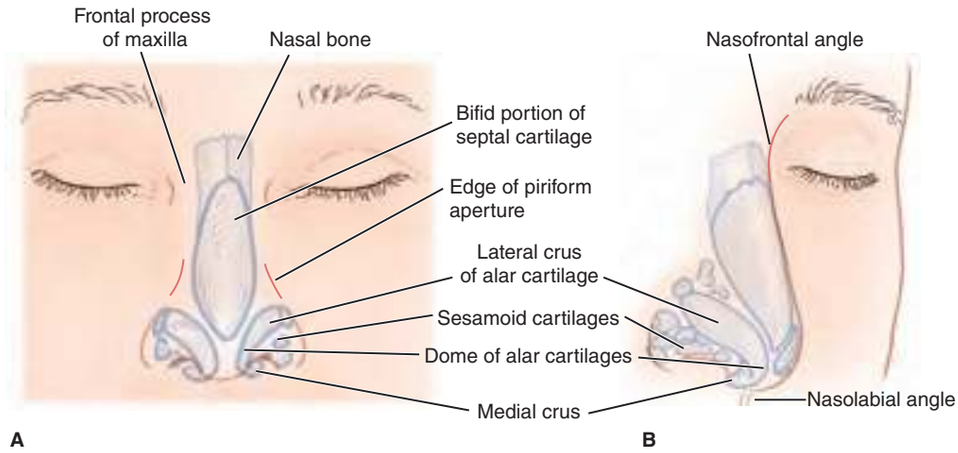


FIGURE 203-5. Anatomy of the external nasal cartilages. ***A.*** Frontal view. ***B.*** Lateral view.

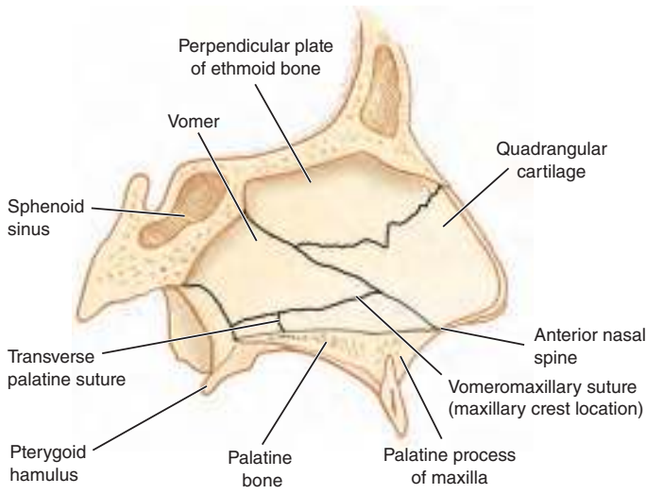


FIGURE 203-6. Anatomy of the nasal septum.

of the ethmoid causing thickening at this point and the appearance of a bilaterally deviated nasal septum.[29]

The more common lateral force shifts the bony pyramid laterally (**Figure 203-10**) while a frontal impact broadens the nose. "Open book fractures" may occur in pediatric patients due to the open midline suture of the nasal bones. This results in each of the nasal bones being shifted laterally (**Figure 203-11**). Children have fewer nasal fractures because of their smaller and more cartilaginous (i.e., more elastic) noses.[30,31] Greenstick fractures are more likely in children because of their resilient noses and can be present when it is not apparent externally.[32,33]

INDICATIONS

Attempt to reduce nasal fractures within the first 3 to 6 hours after the trauma before significant edema develops. Otherwise, perform the reduction 3 to 10 days after the injury when the edema subsides.[34] The indications for a closed nasal reduction include unilateral or bilateral nasal bone fractures, with or without a nasal septal

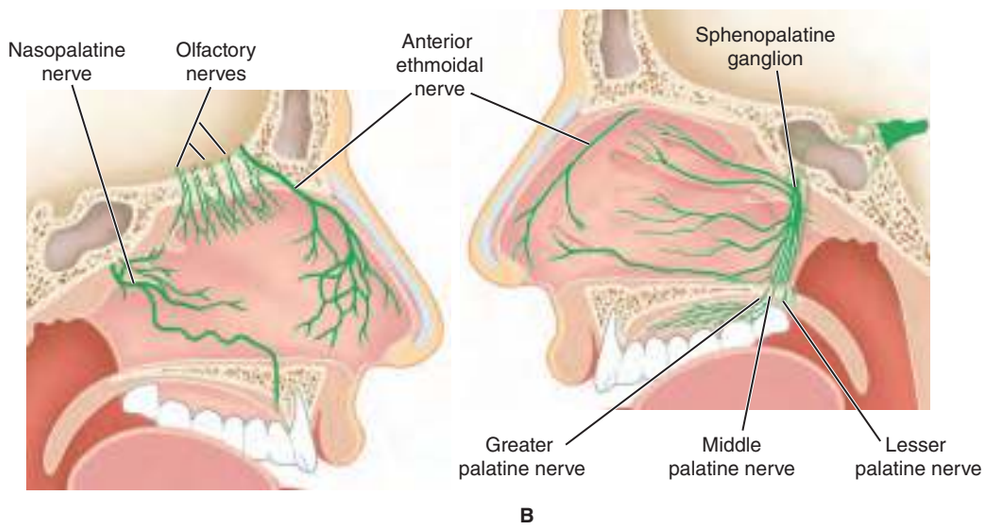


FIGURE 203-7. Nerve supply of the nasal mucosa. ***A.*** The nasal septum. ***B.*** The lateral nasal wall.

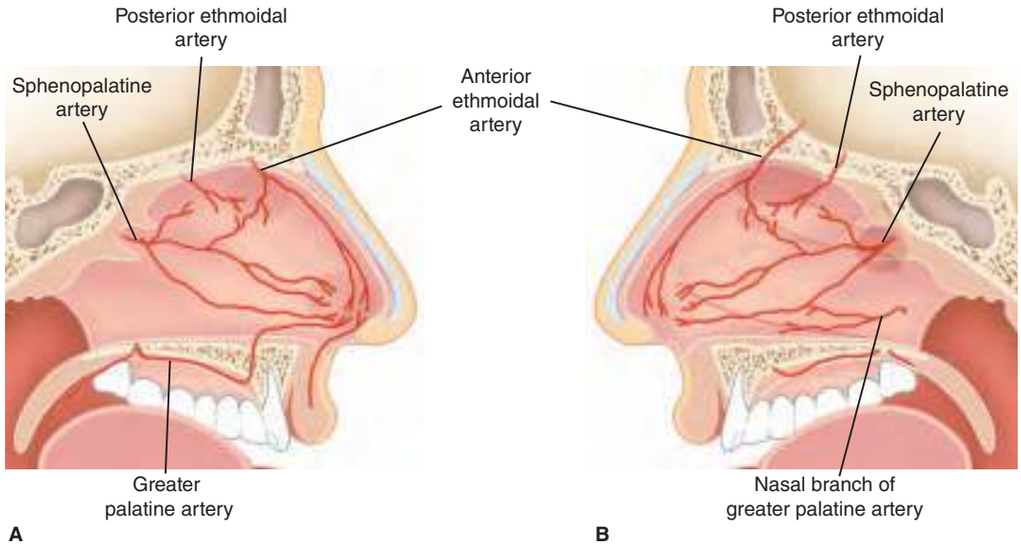


FIGURE 203-8. Blood supply of the nasal mucosa. ***A.*** The nasal septum. ***B.*** The lateral nasal wall.

fracture. Interestingly, one study found that 30% of nasal fractures that underwent closed nasal reduction were still malaligned postoperatively.[29] Strongly consider reduction under procedural sedation or general anesthesia.[35]

CONTRAINDICATIONS

There are no absolute contraindications to nasal fracture reduction as long as the timing, the patient's health status, and associated injuries are considered. The proper approach varies by the extensiveness of the fracture. Open nasal reduction is required for more severe nasal fractures, especially those with C-shaped septal fractures and cartilage telescoping over the perpendicular plate of the ethmoid bone.[7] Some authors have defined the indications for this more aggressive approach as being a nasal pyramid deviation exceeding one-half of the width of the nasal bridge.[29] Consider using an open approach, either during the same setting or within a few days, if deformities persist after closed reduction. Relative indications for the open approach include fracture-dislocations of the caudal septum, open septal fractures, septal hematomas, displaced fractures of the anterior nasal spine, associated alar cartilage deformities, or recent intranasal surgery. An open approach may be required if an extensive septal deformity exists.[6] Otherwise, the nasal deformity will be difficult to reduce against the interlocked forces.[6]

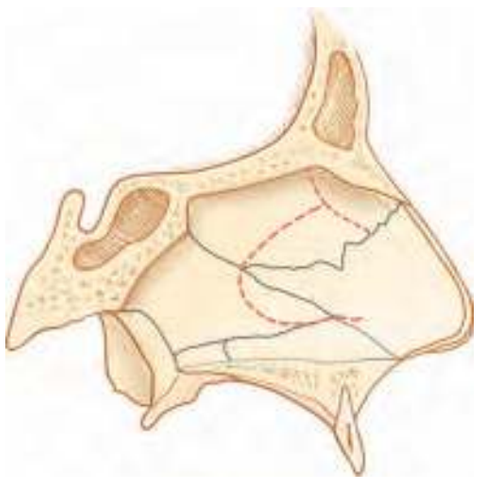


FIGURE 203-9. Schematic illustration of a common nasal septal fracture pattern. The fracture proceeds from the area of the maxillary crest, through the vomer, and up into the perpendicular plate of the ethmoid like a backward "C" going up into a "T" as represented by the dashed line.

EQUIPMENT

- Headlight with light source, or overhead light
- 5 mL syringe
- 27 gauge needle, 1.5 inches long
- 25 gauge spinal needle
- Local anesthetic solution containing 1:100,000 epinephrine
- Oxymetazoline nasal spray
- 4% cocaine, 4 mL
- Other nasal decongestants and anesthetics (Chapter 205 and **Table 205-1**)
- Atomizer (**Table 203-1**) (optional)
- Surgical cotton paddies, 0.5 × 2 inches
- Bayonet forceps
- Walsham forceps (**Figure 203-12**)
- Asch forceps (**Figure 203-12**)
- Rubber tubing
- Boies nasal fracture elevator (**Figure 203-12**)
- Killian nasal speculum
- Silastic nasal splints or Goldsmith splints
- Antibiotic-impregnated strip gauze or nasal tampon
- Plaster of Paris splinting/casting material
- 5–0 plain gut suture

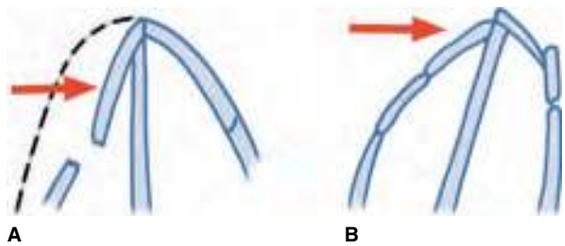


FIGURE 203-10. Nasal fractures resulting from lateral forces. ***A.*** Unilateral nasal fracture. ***B.*** Bilateral nasal fracture. The arrows depict the direction of the force of impact.

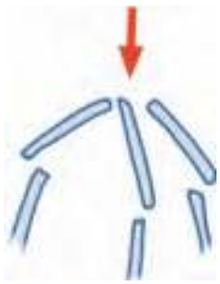


FIGURE 203-11. An "open book" nasal fracture resulting from a frontal impact.

- Paper tape, 1 inch wide
- Benzoin solution
- Ultrasound machine
- Ultrasound gel
- High-frequency (10 to 20 MHz) transducer with a small footprint

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Significant complications include patient dissatisfaction, the possible need for open reduction within 2 weeks, and formal nasal reconstruction months later. Other potential complications of much lower probability include adverse reactions to the local anesthetics, excessive bleeding, infection, saddle nose deformity, septal perforation, cerebrospinal fluid rhinorrhea, and/or visual disturbances. Almost all of these complications can result from the reduction procedure, but also from the nasal fracture without the reduction attempts. Obtain an informed consent for the procedure.

Place the patient sitting upright in a multiposition procedure chair, or on a stretcher, with the head elevated to decrease blood flow and bleeding. Obtain an objective measure of the degree of lateral displacement. Draw a perpendicular line from the midpoint of the interpupillary line and extending downward to the base of the nasal columella. A point one-third of the way down from the cephalic end is the reference point for any deviation.[36] Consider the administration of parenteral analgesics, sedatives, or procedural sedation. Refer to Chapter 159 regarding the details of procedural sedation and analgesia. Obtain a prereduction photograph of the patient's face and nose, if possible.

TABLE 203-1 Atomizers Currently Available

Name	Manufacturer	Comments
DeVilbiss Model 15 Medical Atomizer	DeVilbiss Healthcare	Adjustable tip Disposable and reusable versions
Enk Fiberoptic Atomizer Kit	Cook Medical	Atomizes through fiberoptic bronchoscope working channel Pressure-resistant oxygen tubing
EZ-Spray	Alcove Medical	Disposable Trigger valve system
LMA MADdy Pediatric Mucosal Atomizer Device	Teleflex	11.4 cm long Attaches to standard syringe Child friendly Comes in various colors and shapes Disposable single use
LMA Bottle MADomizer Atomizer	Teleflex	10 cm long Disposable single-use tips Reusable bottle
LMA MADgic Laryngo-Tracheal Atomizer	Teleflex	21.6 cm long Attaches to standard syringe Disposable single use Flexible tube
LMA MAD Nasal-Intranasal Mucosal Atomizer Device	Teleflex	Attaches to standard syringe Disposable single use

FIGURE 203-12. Nasal fracture reduction instruments. From left to right: an elevator, a Walsham forceps, and an Asch forceps (with rubber tubing on one tong).

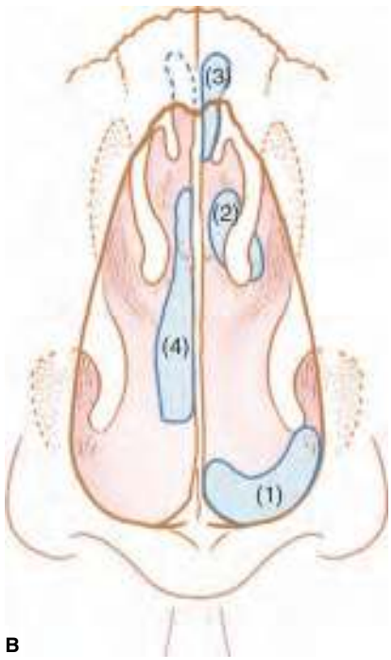
NASAL ANESTHESIA

Anesthetize the nasal mucosa. Insert a nasal speculum. Apply oxymetazoline nasal spray bilaterally to decongest the nasal mucosa. Use an atomizer (**Table 203-1**) if available to increase the placement of the spray. Place 4 mL of 4% cocaine onto cotton pledgets. Cocaine is a vasoconstrictor, a decongestant, and an anesthetic. Insert the nasal speculum and pack the cocaine-soaked pledgets into the nose using a bayonet forceps (**Figures 203-13**). Ideally, place four pledgets in each nostril at the strategic points of the neurovascular supply (**Figure 203-13**). These areas include the posterior edge of the middle turbinate (to anesthetize and shrink the sphenopalatine ganglion and artery), the anterior to middle turbinate and opposing septum (to anesthetize and shrink the anterior ethmoid nerve and artery), the nasal floor (to anesthetize and shrink the branches of both nerves and arteries), and the mid-septum (to anesthetize and shrink the branches of both nerves and arteries).[10,27] Allow the cocaine pledgets to remain in the nasal cavity for 10 minutes. Other anesthetics, decongestants, and anesthetic-decongestant combinations can be used depending upon availability. Refer to Chapter 205 for a more complete discussion of topical nasal anesthesia and decongestion.

Inject local anesthetic solution to anesthetize the remainder of the nose. The most commonly used agent is 1% or 2% lidocaine containing 1:100,000 epinephrine.[37] Acidosis from the injured tissues can make local anesthesia less effective. The addition of sodium bicarbonate at a 1:10 dilution will counteract the acidosis and lessen the discomfort from the injection.[38]

Perform the local anesthetic injections intranasally to avoid the added pain of puncturing through the skin (**Figures 203-14 and 203-15**). Infiltrate subcutaneously to anesthetize the external, nasal, infratrochlear, and infraorbital branches of the trigeminal nerve.[7,8] Insert the needle into the nasal cavity. Infiltrate along the nasal floor to anesthetize the superior alveolar nerve and ganglion. Infiltrate posterior to the inferior and middle turbinates to block the sphenopalatine nerve and ganglion. Allow 10 to 15 minutes for the local anesthetic agent to take effect. Assess the adequacy of the anesthesia

A



B

FIGURE 203-13. Topical anesthesia of the nasal mucosa. ***A.*** Surgical cotton pledgets soaked with cocaine placed in the nares. ***B.*** Typical sites for placement of the pledgets: (*1*) nasal floor, (*2*) posterior aspect of middle and inferior turbinate, (*3*) intranasal dorsum, and (*4*) along the nasal septum.

FIGURE 203-14. Intranasal injection with speculum exposure and a fine needle.

using pinprick of the nasal mucosa. If intranasal injections are performed properly, in a patient who is under procedural sedation and analgesia, the rare but potentially serious complications of using topical cocaine may be avoided. One paper proposed using EMLA (eutectic mixture of local anesthetics) cream externally and cocaine intranasally.[39] This has yet to become a broadly accepted technique. The Emergency Physician may not feel comfortable with the technique of intranasal injection and the nerves may be anesthetized percutaneously as described in Chapter 156.[40]

TECHNIQUES

NASAL BONE REDUCTION

Several studies have demonstrated that closed nasal reduction is 80% to 95% successful.[12,41-46] Measure the distance from the nostril rim to the nasofrontal angle in order to avoid putting the blade in too far (**Figure 203-16*A***). Insert one blade of the Asch forceps, one blade of the Walsham forceps, or a Boies nasal fracture elevator into the nostril to the measured distance. Place the instrument against the depressed nasal bone. Apply a gentle upward and outward force while simultaneously applying digital counterpressure to guide the bone into reduction (**Figure 203-16*B***). Alternatively, cover the other blade of the Asch or Walsham forceps with rubber tubing so that it can provide counterpressure to the intranasal blade during outward reduction (**Figure 203-17**).[10] Repeat the procedure on the contralateral side if there is a bilateral nasal bone fracture.

NASAL SEPTAL REDUCTION

A nasal septal fracture is often present if bilateral pyramidal fractures exist. **Always reduce the nasal bone fracture(s) before manipulating the septum.** Insert a nasal speculum and determine whether the reduction of the nasal bones results in a straightening of the septum. If not, withdraw the nasal speculum and reduce the septum with the Asch or Walsham forceps (**Figure 203-18**). Insert the forceps with one blade in each nostril. Gently close the blades of the forceps to grasp the septum. Elevate the septum upward and anteriorly to disimpact any interlocked fragments. Reinsert the nasal speculum and visualize the nasal septum. Insert a Boies or Freer elevator under direct visualization and straighten the septum more precisely.

ULTRASOUND-GUIDED REDUCTION

Ultrasound can be used in the diagnosis of a nasal fracture.[15-21] Use the highest frequency transducer, ideally 20 MHz, as possible.[18] Ultrasound was also found to be useful to assess the positioning of the nasal bones.[19] Ultrasound should be used before the reduction to plan, during the reduction to evaluate, and after the reduction to confirm the reduction. Otherwise, the reduction technique is the same as described above. Interestingly, one study found visual inspection and palpation were just as reliable as ultrasound in confirming the reduction.[47]

ASSESSMENT

Thoroughly evaluate the nasal cavity and nose. Determine the adequacy of the reduction procedure. Determine visually whether the nasal bones and the nasal septum are reduced. Consult an Otolaryngologist if the manipulations fail to provide a satisfactory reduction, if the fracture appears too comminuted, or if the nose or septum is too deviated for a closed approach. The patient may require open reduction 2 weeks from the day of the trauma. **Perform a thorough examination to rule out an associated septal hematoma. This must be evacuated and managed to prevent complications.** Refer to

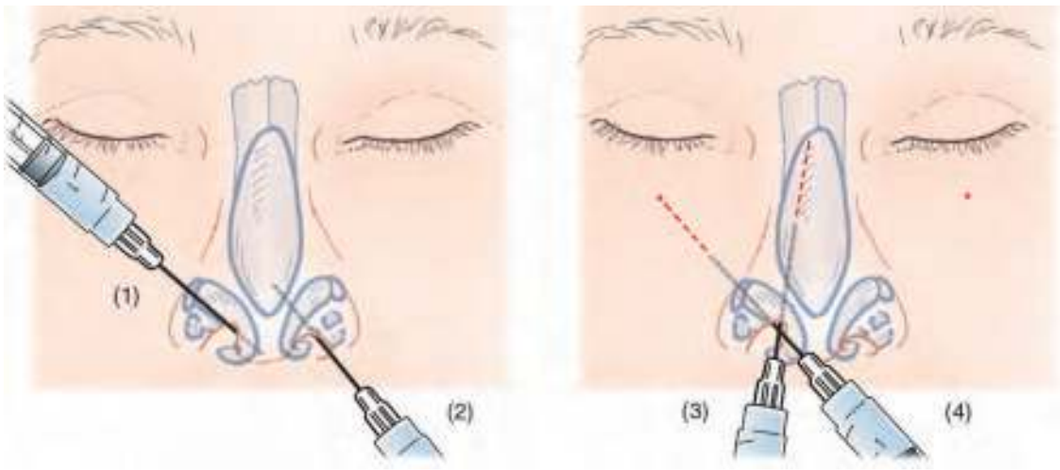


FIGURE 203-15. Intranasal injection of local anesthetic solution: (*1*) the nasal spine, (*2*) the nasal tip, (*3*) the nasal dorsum along the outside of the nasal bones, and (*4*) the infraorbital nerve.

Chapter 204 regarding the details of managing a nasal septal hematoma. Attempt to suture any nasal septal mucosal tears that exist from the fracture or the reduction procedure using 5–0 plain gut suture. Warn the patient of the potential for a septal perforation that can lead to chronic crusting, bleeding, or an audible whistling. Obtain postreduction photographs of the nose, if possible. Place the prereduction and postreduction photographs in the patient's medical record.

AFTERCARE

Brace the septum for stability with bilateral Silastic splints for 5 to 10 days, although 1 day may be as effective.[48] This also helps to prevent the formation of a nasal septal hematoma. Doyle or Goldsmith septal splints have lumens that allow the patient to nasally breathe in the postoperative period. These splints must be kept open by the patient applying six drops of hydrogen peroxide to each nostril three times a day while avoiding swallowing this solution. A simpler and more convenient alternative is bilateral anterior nasal packing. The equipment is readily available in every Emergency Department. This is especially useful in cases of epistaxis. Apply bilateral petrolatum-impregnated gauze ribbon, iodinated gauze ribbon, nasal sponges/tampons, or balloon catheters. **Avoid overpacking the nasal cavity as this can splay out the fractured nasal bones.**[10]

All patients with nasal packing must be placed on oral antibiotics with gram-positive and gram-negative coverage, such as cefazolin (i.e., Keflex), for the duration of the packing. This will aid in the prevention of a sinusitis or toxic shock syndrome. Leave the nasal packing in place for 1 to 7 days depending upon the amount of manipulation, the amount of bleeding, and Emergency Physician preference. Avoid nasal splinting and packing in patients with nasal fractures requiring a minor degree of manipulation, especially when patients are assessed to have a low probability of follow-up and compliance.

Consider applying an external splint in addition to the internal splint or nasal packing. Some authors do not advocate external splinting and its use is based on Emergency Physician preference. The splint serves to support the healing fracture and to remind the patient to keep their nose protected. Cleanse the skin of the nose and the surrounding area with alcohol. Apply benzoin solution to the nose and surrounding area. Allow the benzoin to dry and become tacky. Apply 1 inch wide paper tape in horizontal strips layered downward from the nasofrontal angle to the tip of the nose. Apply benzoin solution over the tape. Allow the benzoin to dry and become tacky. Apply three to five layers of 2 inch wide orthopedic plaster of Paris over the tape. Alternatively, apply an appropriately sized Denver aluminum, polyvinyl siloxane, or Thermoplast splint

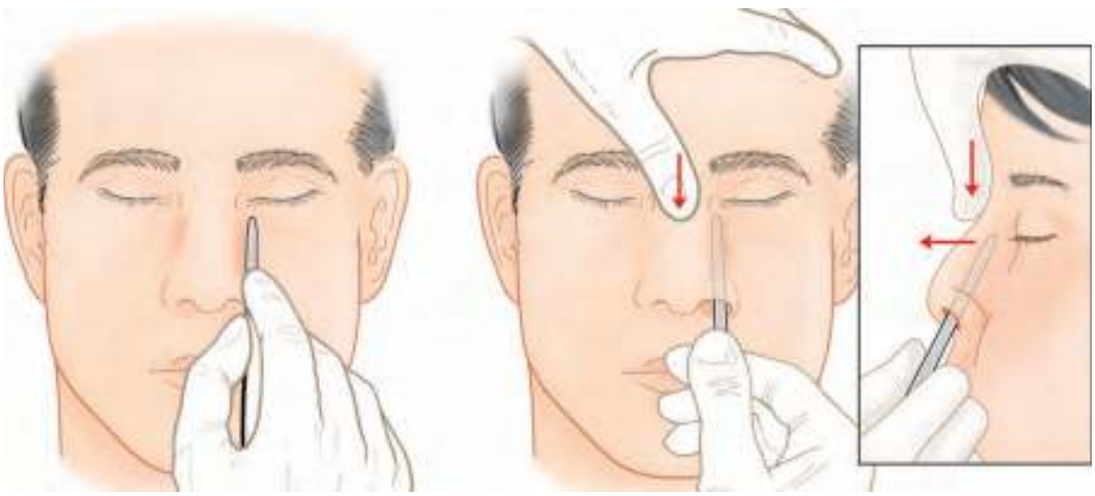


FIGURE 203-16. Closed reduction of a nasal fracture. ***A.*** Measure the nostril to nasofrontal angle (N-NFA) distance with the Boies elevator or another instrument. ***B.*** Place the elevator or forceps intranasally just less than the N-NFA distance and elevate the depressed bone. Simultaneously reduce the contralateral nasal bone downward.

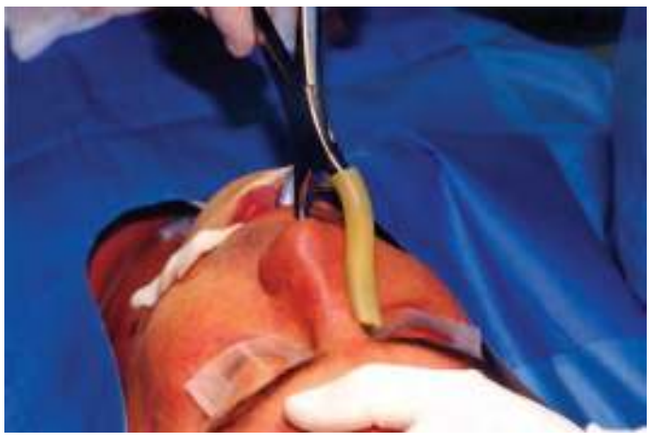


FIGURE 203-17. Placement of nasal reduction forceps. Note the rubber tubing over the outer tong to protect the skin.

FIGURE 203-19. Postreduction Denver splint applied after appropriate taping and midline vertical foam rubber placement.

over the tape (**Figure 203-19**).[49] Ensure that the foam rubber strut is placed vertically over the midline length of the nose to prevent any skin necrosis. Place tape over the splint to secure it.

Instruct the patient to return to the Emergency Department for excessive bleeding, increased pain, a purulent or foul nasal discharge, or a fever. Prescribe acetaminophen supplemented with narcotic analgesics as needed. Instruct the patient to avoid aspirin-containing products and nonsteroidal anti-inflammatory drugs as they can increase the risk of bleeding. Arrange follow-up with an Otolaryngologist or Plastic Surgeon within 24 to 48 hours to assess the reduction and the need for further management. The use of ice packs several times a day may decrease pain and swelling.

COMPLICATIONS

Pack (or repack) the nasal cavity, after providing adequate local anesthesia, if bleeding occurs. A septal hematoma is likely if a patient complains of persistent or excessive pain, has noticeable nasal widening, or has an ecchymotic septum. This must be evacuated as described in Chapter 204. Infection, cartilage necrosis, and disfiguring nasal dorsal saddling may occur if the septal hematoma is not evacuated. A nasal dorsal hematoma can occur as well and must be recognized and evacuated. Cerebrospinal fluid rhinorrhea and visual impairment are rare complications of the nasal manipulation but more likely complications of the original trauma. Cerebrospinal fluid rhinorrhea can be delayed from the initial trauma until the edema has subsided. The reduction may be inadequate and require further closed or open reduction.

The patient may not be satisfied with the results of the reduction, especially if the nasal fracture results from an assault.[43] This is more likely to occur in Single Immature Males that are Overtly expectant and Narcissistic, also referred to by the acronym SIMONs, despite an adequate reduction.[43] Other studies have shown that overall patient satisfaction did not differ whether reduced under local anesthesia versus in the Operating Room under general anesthesia.[44,50]

FIGURE 203-18. The Asch forceps to elevate a frontal/inferior force fracture and straighten the associated deviated nasal septum.

SUMMARY

Nasal fractures may be reduced and repaired in the Emergency Department using a closed technique. Perform the reduction ideally within 3 hours of the injury before significant edema occurs. It is otherwise best to wait until the swelling subsides in approximately 1 week. Repair any nasal septal fractures at the same time as the nasal reduction. Severe fractures sometimes require an open reduction. Photographic and radiographic documentation can be important medicolegally as part of the preoperative evaluation. Drain any septal or nasal dorsal hematomas. Internal and external splinting are useful to help ensure good postoperative healing. Antibiotics are essential if nasal packing is applied.

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204 Nasal Septal Hematoma Evacuation

Michael Friedman and Jessica Tang

INTRODUCTION

Soft tissue and bony injuries of the nose are common because the nose is centrally located and the most anteriorly protruding structure of the face.[1,2] Suspect a nasal septal hematoma, although an uncommon complication of nasal trauma, in any individual who has sustained a nasal injury.[3,4] All individuals who have sustained nasal trauma must undergo a careful examination of the septum and nasal passages regardless of the mechanism of injury or the findings on external examination.[3,5,6] A nasal septal hematoma is blood or a clot accumulated between the cartilaginous nasal septum and the overlying nasal mucosa.[7]

Blunt trauma, either intentional or unintentional, is the most common cause of a nasal septal hematoma. Consider a bleeding diathesis if the hematoma develops after a seemingly trivial injury.[3,8-10] Other etiologies include sports injuries and child abuse.[8,9] Iatrogenic nasal septal hematomas following nasal septal surgery are probably more common than reported in the literature. Evaluate patients who have had recent nasal surgery and present with complaints of pain and nasal obstruction for a possible nasal septal hematoma.

Nasal septal hematomas are characterized by severe localized nasal pain, tenderness, instability on palpation of the nasal tip, and a cherry-like swelling or bluish discoloration of the nasal mucosa emanating from the septum that obstructs all or a portion of the nasal passage (**Figure 204-1**).[1,3,4,11-13] **Septal hematomas can also present bilaterally if a fracture of the nasal cartilage is involved and allows blood to dissect through the fracture. Thus, it is critical that both nares be examined scrupulously.** Evacuation must be performed to prevent complications.[1,3,8,10,14,15] **Infections can develop as soon as 3 days after injury. A simple incision to allow the clot to drain followed by anterior packing is usually adequate as an initial management.**

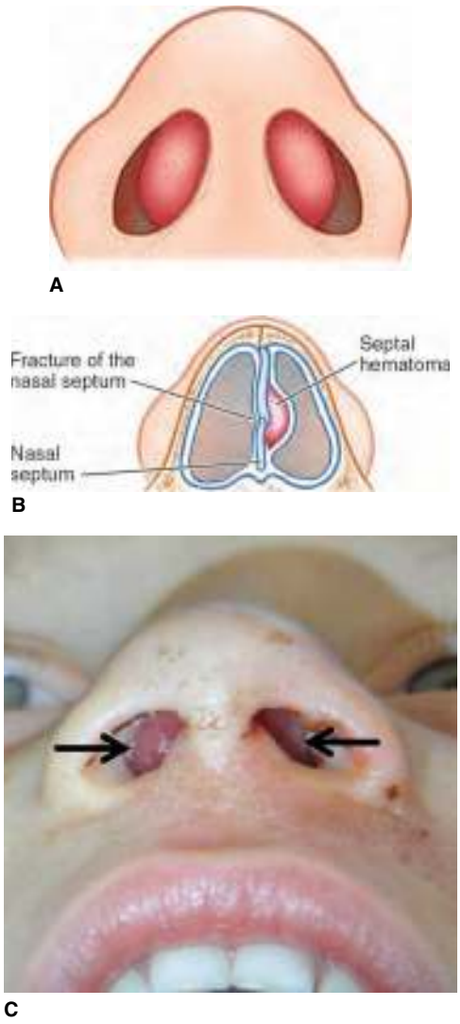


FIGURE 204-1. The nasal septal hematoma. ***A.*** Bilateral nasal septal hematomas creating a partial obstruction. ***B.*** Anatomy of a nasal septal hematoma. ***C.*** Bilateral nasal septal hematomas (arrows). (Photo used with permission from reference 14.)

The most common complication of a nasal septal hematoma or an abscess is cartilage necrosis that results in nasal structural collapse and a saddle nose deformity (**Figure 204-2**).[2]

ANATOMY AND PATHOPHYSIOLOGY

The nose is a sensory organ and a respiratory organ. It performs an important function for the entire body by providing physical and immunologic protection from the environment.[16] The nose aids in the formation of basic speech sounds.[16] The supporting structure of the nose consists of bone, cartilage, and connective tissue. The nose, on frontal view, is in the shape of a pyramid, of which approximately the upper two-fifths comprise the bony vault and the lower three-fifths comprise the cartilaginous vault. The upper narrow end joins the forehead at the glabella and is referred to as the root of the nose. The two nares are separated from each other by a skin-cartilage septum known as the columella.[16,17] The cartilaginous framework of the nose provides its structure and function. The septal cartilage is avascular and receives its blood supply from the adherent mucoperichondrium.

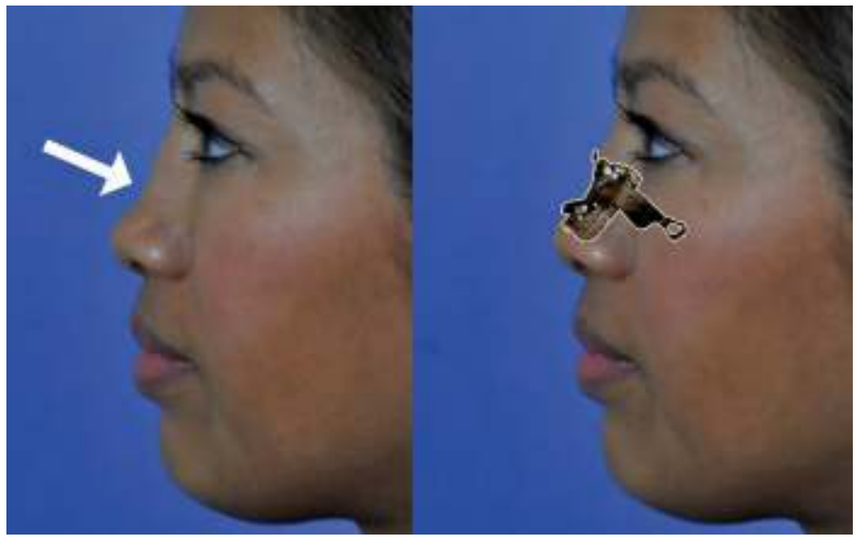
The skin covering the external nose is thin and contains an areolar type of subcutaneous tissue. The skin is loosely attached over the upper half. The skin over the lower half of the nose is intimately bound to the lower lateral cartilage and may sometimes be thick, fatty, and contain sebaceous glands.[9,10]

FIGURE 204-2. Artist illustration of the saddle nose deformity.

The precise mechanism for a nasal septal hematoma formation following nasal trauma is not known. Nasal septal hematomas are thought to occur when a mechanical force to the nasal cartilage results in rupture or leakage from the perichondrial blood vessels of the nasal septum. In instances where the nasal cartilage is fractured, blood may dissect through the fracture line and form bilateral septal hematomas. Most nasal septal hematomas are secondary to a fracture or surgery. **They almost always distend the mucoperichondrium on both sides of the nose.** Accumulation of the extravasated blood strips the perichondrium from the cartilage forming a closed space in which the blood accumulates (**Figure 204-1**). The nasal septal hematoma, when not recognized initially and evacuated promptly, can expand and mechanically obstruct the blood vessels that supply the nasal cartilage.[18] Pressure-induced avascular necrosis of the nasal septal cartilage can develop rapidly because there is no alternative blood supply to the cartilage.[1,3,4,13] The accumulated blood and necrotic tissue can form a nidus for infection from bacteria that colonize the nasal mucosa.[1,3,4,13] **Prolonged ischemia can cause septal perforation and irreversible damage if untreated for 3 to 4 days.**

Cartilage necrosis subsequently leads to the saddle nose deformity (Figures 204-2 and 204-3). The term "saddle nose deformity" is a nonspecific description of a nose with a depression over its dorsal surface.[16] The saddle nose deformity is a result of the nasal septal cartilage becoming ischemic from loss of its blood supply, subsequent nasal septal cartilage resorption and collapse, and the lack of support for the nasal bridge. The deformity is the ultimate result of a nasal septal hematoma or abscess not being evacuated. Necrosis with subsequent fibrosis may develop causing a permanent thickening or absorption of the nasal septum with partial obstruction of the nasal airway.[13,16,19] **Other facial deformities including displacement of the maxilla, retraction of the anterior nasal septum (i.e., columella), widening of the nasal base, and diminished nasal cavity size can result when necrotic tissue is replaced by fibrous tissue with subsequent retraction of scar tissue.**

The sphenopalatine branches of the internal maxillary artery and the ethmoidal arteries from the ophthalmic artery supply the internal nose. The veins terminate in the anterior facial and ophthalmic



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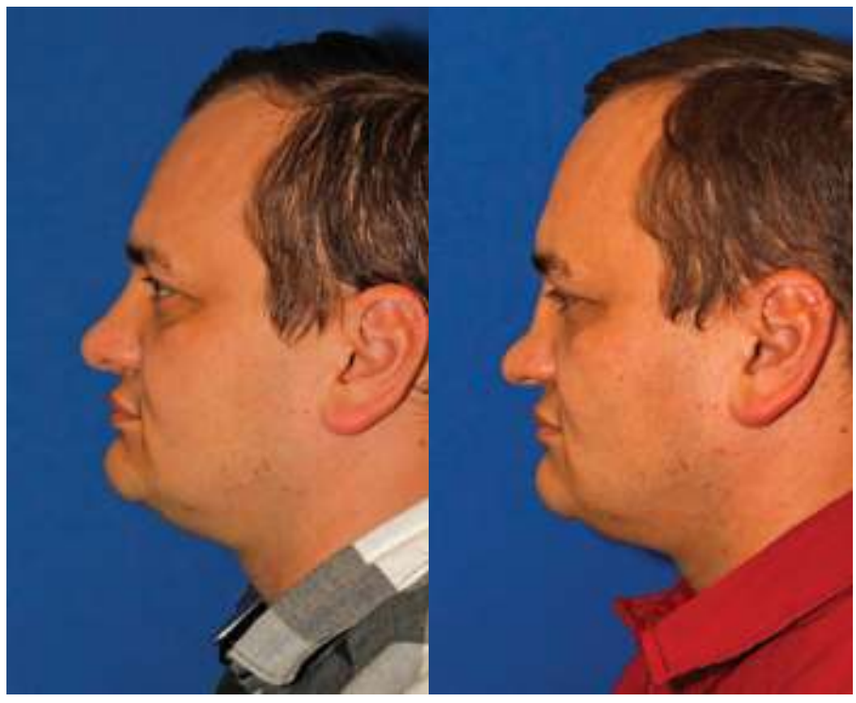

B

FIGURE 204-3. The saddle nose deformity. ***A.*** A young woman. (Photo courtesy of J. Hilinski, MD.) ***B.*** A middle-aged man. (Photos courtesy of T. Lampertini, MD, at www.drlampertini.com.)

veins.[17] Venous drainage is clinically important in understanding the complications of a septal abscess. Branches of the trigeminal nerve provide sensory innervation to the nose.[16,17]

INDICATIONS

Any significant nasal septal hematoma requires evacuation. A nasal septal hematoma may progress to form an abscess with associated complications of avascular necrosis of the nasal septum, meningitis, or cavernous sinus thrombosis in as little as 3 to 4 days.[13,19] It is not urgent to evacuate a simple nasal septal hematoma in the Emergency Department as complications occur over a period of days. It is essential, however, to identify a nasal septal hematoma so that a treatment plan is initiated and to rule out a septal abscess that does require immediate evacuation.[8]

CONTRAINDICATIONS

There are no absolute contraindications to the evacuation of a nasal septal hematoma. Address any life-threatening or serious injuries prior to drainage of the nasal septal hematoma. **This procedure requires a cooperative patient to prevent secondary iatrogenic injury.** Any patient who is uncooperative, unable to follow instructions, very young, or who has an altered mental status may require evacuation under procedural sedation (Chapter 159) or general anesthesia.

EQUIPMENT

- Headlight
- Nasal speculum
- Nasal vasoconstrictor (e.g., 2% ephedrine, 0.25% oxymetazoline or phenylephrine)
- Nasal anesthetic (e.g., 4% cocaine, 2% Pontocaine, 4% lidocaine)
- Nasal atomizer device, optional
- Alcohol swabs
- Povidone iodine or chlorhexidine solution
- Cotton-tipped applicators
- 1 and 10 mL syringes
- 25 or 27 gauge needle, 1½ inches long
- Local anesthetic solution containing epinephrine, lidocaine, or bupivacaine
- #15 surgical blade on a handle
- Frazier suction catheter
- Suction source and tubing
- Nasal speculum
- Bayonet forceps
- Nasal tampon or sponge
- Iodoform gauze

PATIENT PREPARATION

Explain the risks, benefits, complications, and aftercare of the procedure to the patient and/or their representative. Obtain a signed consent for the procedure. **Aseptic technique should be followed and maintained throughout the procedure.** The procedure is considered clean, but not sterile, as the nasal mucosa can never be sterilized. **It is crucial that the instruments used are sterile.**

Administer anesthesia for the evacuation of a nasal septal hematoma by one of two routes: topical application with supplemental infiltration or a regional block. Some authors recommend using both techniques. Most nasal septal hematomas can be adequately evacuated with topical anesthesia supplemented by local infiltration.[13] Dampen the swabs of four cotton-tipped applicators with either a solution of 2% Pontocaine with ephedrine or cocaine. Insert a nasal speculum to expose one nostril. Gently insert the applicator beneath the roof of the nose so that it reaches the branches of the anterior ethmoidal nerve (**Figure 204-4**). Pause until some vasoconstriction and anesthesia takes place if it meets resistance on insertion and then advance the applicator further (**Figure 204-4**). Pass an applicator into the nasal fossa and move it from the floor of the nasal vestibule, across the midportion of the inferior turbinate, and reaching the posterior aspect of the middle turbinate in the region of the sphenopalatine foramen.[20] Infiltrate a maximum of 0.5 mL of local anesthetic solution containing epinephrine, if additional anesthesia is desired, through the mucoperichondrium and circumferentially around the hematoma.

An atomizer or nasal spray bottle can provide an alternative and commonly used method for nasal septal anesthesia (Chapter 205 and **Table 205-1**). Although an actual atomizer is not often available in the Emergency Department, its use is less painful to the patient than the application of cotton-tipped applicators into the nasal cavity. Alternatives to an atomizer are devices that attach to a syringe to atomize the contents or use of a commercially available nasal spray container after emptying its contents. Mix equal amounts of 2% Pontocaine or 4% lidocaine with ephedrine and place this in the atomizer or nasal spray bottle. Insert the nasal speculum to gain access to the entire nasal cavity. Open the nasal speculum vertically to expose as much of the nasal septum as possible. Insert the atomizer or nasal spray bottle into the nostril. Instill two puffs of solution into the nostril. Allow 5 to 10 minutes for the anesthetic to take effect. Infiltrate a maximum of 0.5 mL of local anesthetic solution containing epinephrine, if additional anesthesia is desired, through the mucoperichondrium and circumferentially around the hematoma.

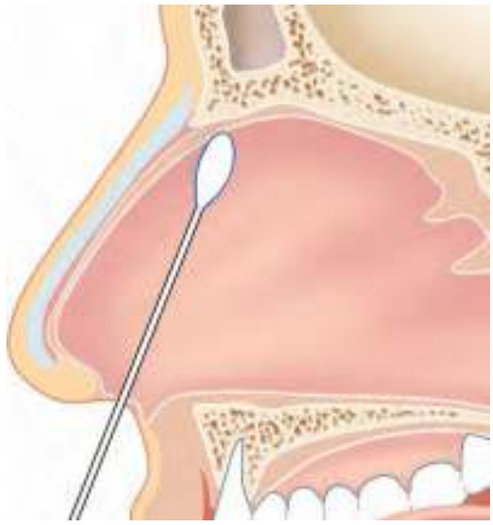

FIGURE 204-4. Cotton swab technique for nasal septal anesthesia.

Reconfirm the presence of a septal hematoma.[5] Compress the area with a cotton-tipped applicator. The bulge from the hematoma is compressible with the applicator. **A nasal septal hematoma will not shrink with the application of a topical vasoconstrictor.**[3,9,21]

TECHNIQUES

ASPIRATION

Some feel that simple aspiration with an 18 gauge needle may be adequate for a small and early hematoma. Most patients require a more extensive evacuation as clotted blood cannot be removed with aspiration. Needle aspiration is generally not effective since blood clots form within minutes and remain in clotted form for days. Simple aspiration may, however, be used to distinguish a septal hematoma from an abscess.[9,13,19]

Insert the nasal speculum. Open the nasal speculum vertically so that the nasal septum is maximally visible. Instruct an assistant to hold the nasal speculum in position and open. This will allow the Emergency Physician to have both hands free to perform the procedure. Insert an 18 gauge needle attached to a 10 mL syringe into the nasal septal hematoma. Aspirate the contents of the hematoma. No additional procedure is necessary if the hematoma can be completely evacuated by aspiration. Simple clinical assessment of the septum is the guide to determine if the hematoma has been evacuated. Apply a nasal pack as described below.

INCISION AND DRAINAGE

The incision and drainage technique is the primary procedure to drain a nasal septal hematoma.[11,21-23] Cleanse the skin surrounding the nares of any dirt and debris. Apply an alcohol swab, povidone iodine, or chlorhexidine to the area and allow it to dry. Insert the nasal speculum. Open the nasal speculum vertically so that the nasal septum is maximally visible. Instruct an assistant to hold the nasal speculum

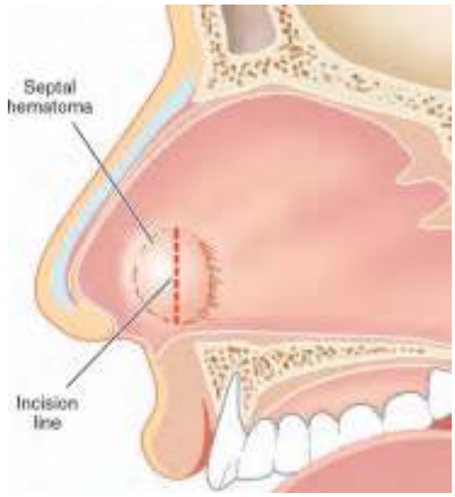

FIGURE 204-5. Nasal septal hematoma with markings for a vertical incision through the mucoperichondrium.

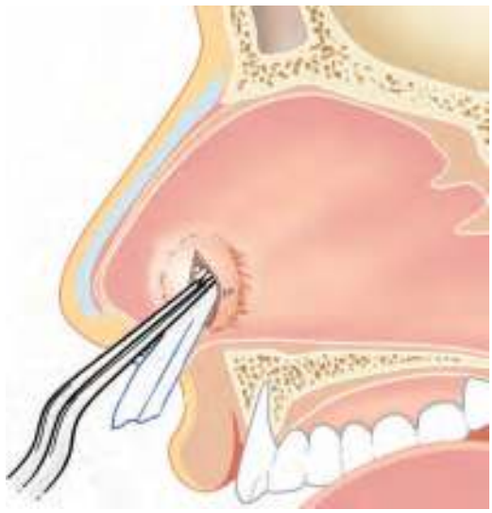

FIGURE 204-6. Iodoform gauze is placed between the separated mucoperichondrium and the cartilaginous septum.

in position and open. This will allow the Emergency Physician to have both hands free to perform the procedure. Make a 1.0 to 2.0 cm long vertical incision with a #15 scalpel blade in the septal mucoperichondrium overlying the hematoma (**Figure 204-5**). **Be cautious to not cut into the cartilaginous septum.** The length of the incision depends upon the size of the nasal septal hematoma. Some authors prefer to use an L-shaped incision in the mucoperichondrium.[24-28] These authors feel that this incision allows the mucoperichondrium to reposition flat against the cartilaginous septum. The choice of incision type is left to the preference of the Emergency Physician.

Use the Frazier suction catheter to gently evacuate any clot or necrotic debris from the hematoma cavity. Clots are difficult to evaluate by suction and may require mechanical removal. Always send a sample of the fluid or hematoma to the laboratory for Gram's stain, anaerobic cultures, and aerobic cultures.[3,13,19] Apply topical antibiotic ointment over the incision, a wick, and nasal packing as described below.

A bilateral septal hematoma can almost always be evacuated from one side. Apply gentle pressure to the contralateral hematoma to express it out of the incision. Make a second vertical incision contralaterally and anterior or posterior to the first incision. **The staggering of the incisions prevents septal perforation if complete evacuation is not achieved unilaterally.**

WICK INSERTION

Insert a wick of 1/8 inch iodoform gauze through the incision (**Figure 204-6**). Allow 1 inch of the wick to extend into the nasal cavity for easy removal. **Be careful to ensure that the wick is flat between the mucoperichondrium and the cartilaginous septum. Do not pack the cavity with the wick.** Some authors recommend inserting a Penrose drain, or a piece of one, instead of the iodoform gauze.[9] This has not been found to be beneficial when compared to iodoform gauze.[9] The Penrose drain can be a substitute if iodoform gauze is not available or the patient is allergic to iodine.

NASAL PACKING

Apply bilateral nasal packs following the successful drainage of a septal hematoma (Chapter 205). Packing inhibits the reaccumulation of the hematoma or serous fluid and prevents the severe complications associated with a septal hematoma. The use of commercially available nasal packing devices (e.g., nasal tampons or sponges) provides adequate protection against reaccumulation.[21] Patients report these to be significantly more comfortable than traditional gauze packing. In addition, their ease of insertion when compared to gauze packing has made these commercial nasal packing devices very popular.

Place the patient's head in the sniffing position. Coat a nasal tampon with a water-soluble antibiotic ointment. Insert the nasal speculum to obtain adequate visualization of the nasal septum. Grasp the nasal tampon with a bayonet forceps. Introduce the forceps with the nasal tampon in a horizontal position through the speculum. **Be careful to not tear the incision in the mucoperichondrium open or to dislodge the wick.** Place the nasal tampon on the floor of the nasal cavity and against the nasal septum.[19] Withdraw the bayonet forceps and the nasal speculum. Repeat the nasal packing procedure on the contralateral side. **Both nasal cavities must be packed to maintain the septum in the midline, prevent bowing of the septum into the contralateral nasal cavity, and prevent the reaccumulation of the hematoma.**

PEDIATRIC CONSIDERATIONS

Nasal septal hematomas can occur at any age.[29] The diagnosis and treatment are the same for adult and pediatric patients.[5,29] Perform any treatment for children under procedural sedation in the Emergency Department or general anesthesia in the Operating Room.[14] **It is crucial to sufficiently inspect the intranasal cavity of children with good lighting because septal injury may still occur in the absence of external physical exam findings. The injury may further evolve over 24 to 72 hours following the initial injury. Periodic examinations for at least 12 to 18 months after injury should be performed to monitor for any cosmetic deformities.**

AFTERCARE

Proper follow-up is vital to prevent any infectious process or cosmetic deformity. All patients must be reevaluated within 24 hours with an Otolaryngologist to prevent complications following the evacuation of the hematoma.[3,10,19] The patient should follow up again in 48 hours for removal of the nasal packs. The drain can be removed at this follow-up if there is no further blood draining or hematoma formation.[11] Prescribe acetaminophen and narcotic analgesics for pain control. Prescribe broad-spectrum antibiotics, specifically those with staphylococcal coverage, as prophylaxis against infection and the development of a septal abscess.[9] Draining a septal hematoma can convert a closed fracture into an open fracture. Instruct the patient to avoid

nonsteroidal anti-inflammatory drugs (NSAIDs) as they increase the risk of bleeding. Provide all patients with proper discharge instructions. The patient should return to the Emergency Department immediately if they experience increased pain, bleeding, or a fever.

COMPLICATIONS

The most common complication of a nasal septal abscess is cartilage necrosis that results in nasal structural collapse and a saddle nose deformity (**Figure 204-2**).[2] The most common acute complication of a nasal septal hematoma is an abscess or cosmetic deformity. Complications are primarily related to incomplete evacuation or reaccumulation of the hematoma. This may be avoided by adequate removal of the hematoma with suction, placement of an iodoform gauze wick, and bilateral nasal packing.

Distinguishing an uncomplicated nasal septal hematoma from one that has become infected is difficult. This is particularly true if there has been several days of delay in seeking medical attention following the injury.[3] Nasal septal abscesses are a rare complication of nasal septal hematomas that occur following nasal trauma. **Septal abscesses can be distinguished from an otherwise uncomplicated hematoma in that they are generally larger, more painful, and inflamed, and inflammatory exudates may be present on the overlying mucosa.[3] It is important to recognize septal abscesses as they may result in ascending infections including meningitis and cavernous sinus thrombosis.[18,19]** *Staphylococcus aureus* is the primary pathogen isolated from most reported cases regardless of patient age. Prescribe appropriate prophylactic antibiotics.[3,8,9]

Many patients cannot tolerate complete hematoma evacuation under local anesthesia. Consult an Otolaryngologist and schedule the patient for evacuation under anesthesia within 24 hours when complete evacuation is not performed. Early and/or appropriate treatment may not prevent complications if the nasal septal cartilage is already ischemic at the time of the procedure.

Other complications of nasal septal hematoma drainage are rare and include nasal bleeding, inadequate evacuation, and septal perforation. Nasal bleeding can usually be controlled with the prescribed packing. Incomplete removal of the hematoma is not a serious problem if identified. A nasal septal hematoma may reoccur if the incision is made on opposite sides of the septum. **Limit the incision to one side if possible. The second incision, if required, on the contralateral side must not oppose the first incision.**

SUMMARY

A nasal septal hematoma is a rare but potentially serious complication of nasal trauma. Proper management consists of early recognition, prompt evacuation, wick insertion, and bilateral nasal cavity packing. Administration of antimicrobial therapy is necessary to prevent or treat a secondary nasal abscess. Follow-up with an Otolaryngologist is required within 24 hours of the procedure to evaluate for the reaccumulation of the hematoma and/or an abscess. These patients require continual follow-up until the nasal septum is healed.

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205 Epistaxis Management

Steven Lai and Matthew Waxman

INTRODUCTION

Epistaxis is an extremely common condition in the United States with an incidence estimated at 10 per 10,000 people per year and a lifetime incidence of approximately 60%.[1] It is a common reason for patient visits to the Emergency Department. Epistaxis has a bimodal age distribution with an early peak in those less than 10 years of age.[2] The frequency of epistaxis decreases in the teens

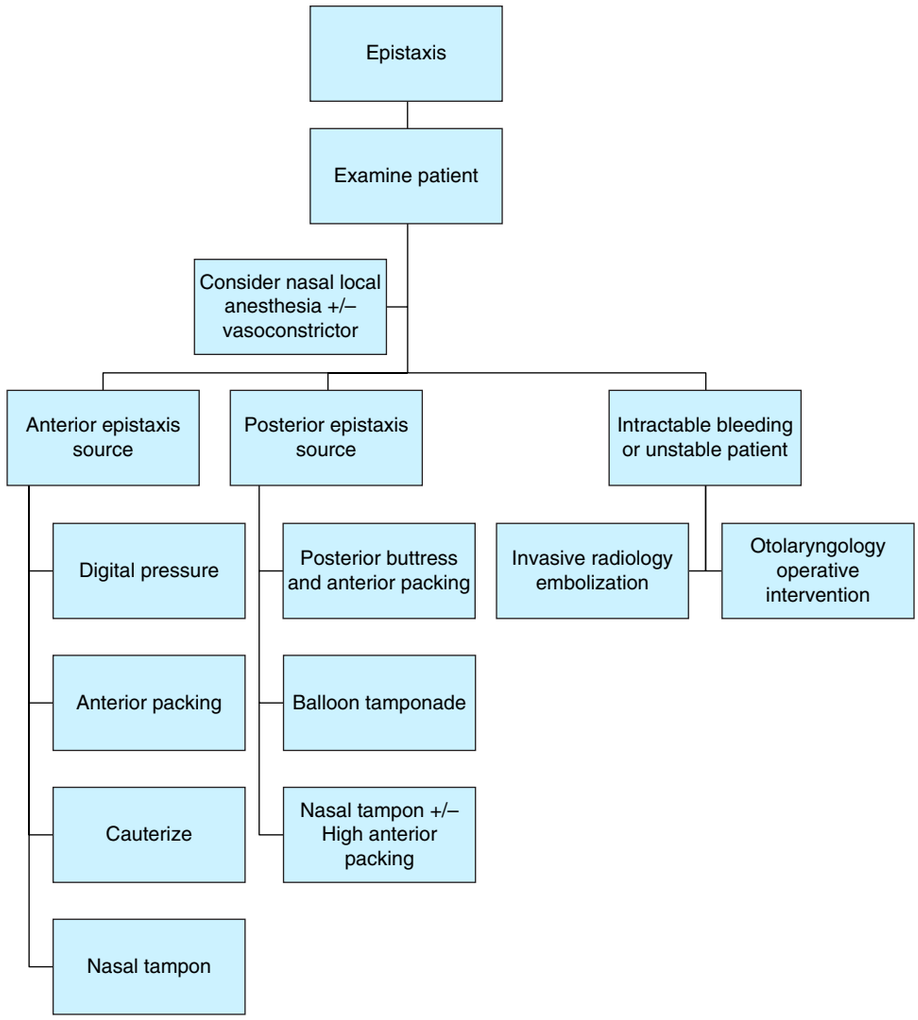

FIGURE 205-1. Methods to control epistaxis.

followed by a progressive increase after 20 years of age with the highest frequency in the elderly.[2] Epistaxis is usually the result of intranasal trauma. It may be the initial sign of a more serious underlying systemic illness.[3] Epistaxis is often self-limited and can be managed conservatively.[4] Epistaxis can also manifest as a profuse spontaneous hemorrhage that is extremely difficult to control and can result in aspiration, hypotension, cardiovascular collapse, syncope, and airway compromise.

The proper management of epistaxis and the prevention of adverse consequences depend on a timely and thorough evaluation of the patient, appropriate intervention, and expeditious intervention. The Emergency Physician must be familiar with a variety of techniques to control intranasal hemorrhage (**Figure 205-1**).[5-8]

ANATOMY AND PATHOPHYSIOLOGY

An understanding of the vascular anatomy supplying the nasal cavity is essential to the efficient and timely control of epistaxis. The blood supply to the sinonasal cavity arises from branches of the internal and external carotid arteries (**Figure 205-2**). **The sphenopalatine artery is the primary blood supply to the sinonasal cavities.** The sphenopalatine artery arises from the terminal branches of the internal maxillary artery which is a branch of the external carotid system. The anterior and posterior ethmoid arteries, the terminal branches of the internal carotid system, supply blood to the superior straits of the nose. The superior labial branch of the facial artery supplies the anterior nasal cavity and anastomoses with branches from the anterior ethmoid artery and the sphenopalatine artery in Little's area of the anterior and inferior nasal septum known as Kiesselbach's plexus (**Figure 205-3**). **It has been estimated that 90% of all nasal bleeding occurs anteriorly in Kiesselbach's plexus.**[9] This is particularly true for children and young adults. Older adults have a higher incidence of bleeding from the posterior nasal cavity which receives its blood supply from branches of the sphenopalatine and posterior ethmoidal arteries. It is in Woodruff's area located at the posterior end of the inferior turbinate. Atherosclerosis and use of anticoagulants are often causes of epistaxis in the elderly.[9]

Epistaxis may result from a combination of local and systemic factors that damage the delicate mucosal lining of the nasal cavity and expose the underlying vasculature.[10] The most common cause of epistaxis is accidental or self-inflicted trauma from digital manipulation of the nasal mucosa (i.e., nose picking). This eventually heals and crusts over but is subject to repeated irritation and bleeding when the patient sneezes or blows their nose. The anterior source of

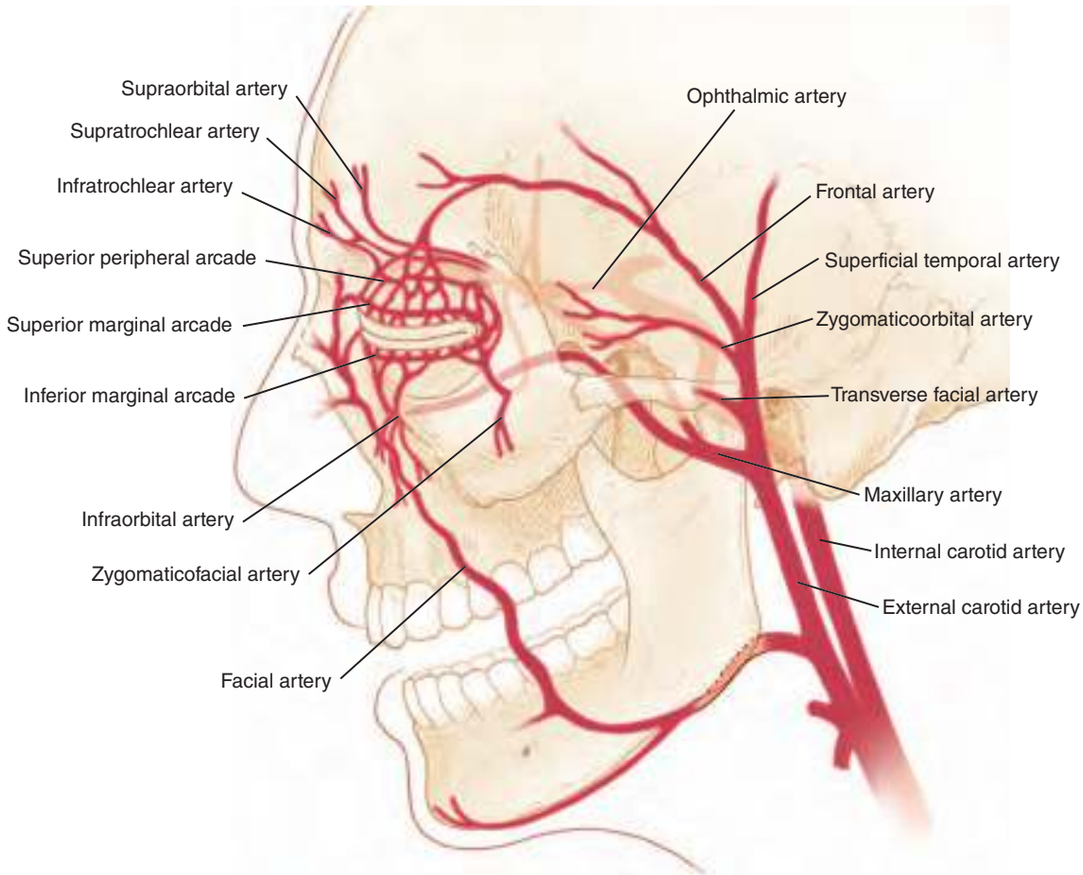

FIGURE 205-2. The blood supply of the nasal cavity arises from the internal and external carotid artery systems.

this bleeding makes it very easy to treat. High-velocity trauma to the midface and skull base may manifest as a severe or life-threatening hemorrhage that may be extremely difficult to control.

Local inflammatory reactions due to acute upper respiratory infections, allergic rhinitis, and chronic sinusitis may cause epistaxis.[11]

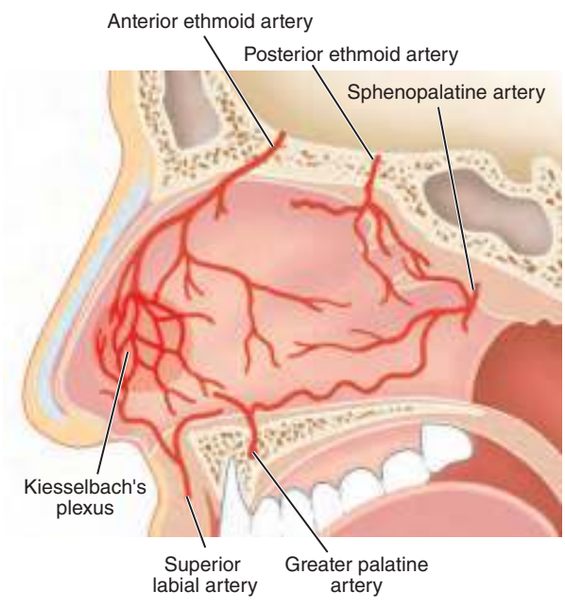

FIGURE 205-3. Arterial supply to the nasal septum and Kiesselbach's plexus.

The nasal mucosa becomes inflamed, hypervascular, and is easily disrupted with forceful nose blowing. The presence of an intranasal foreign body may irritate the nasal mucosa and form granulation tissue that is prone to bleeding. This should be expected if the bleeding occurs unilaterally and is associated with nasal obstruction and foul rhinorrhea. Illicit intranasal drug insufflation with cocaine or methamphetamine can result in epistaxis due to local trauma, inflammation, or vasoactive effects from these substances.[12]

Epistaxis may be attributed to a nasal septal deformity.[13] The deflected nasal septum creates turbulent airflow that desiccates the mucosa and leads to crusting and bleeding. Epistaxis attributed to nasal septal deviation presents just posterior to the deflection and may be difficult to control. Nasal septal perforations bleed frequently and easily from mucosal irritation and granulation tissue.

Postoperative bleeding may be encountered from sinonasal surgery (e.g., septoplasty, rhinoplasty, turbinectomy, and endoscopic sinus surgery). Blood-tinged nasal secretions are to be expected for the first 2 weeks after surgery. Severe epistaxis may occur during the first postoperative week with an estimated incidence ranging from 0.9% to 8.9%.[14] The bleeding is usually posterior and brisk and may be difficult to control. Consult the Otolaryngologist who performed the procedure in all cases of postoperative bleeding.

Nasal bleeding may be the first sign of a sinonasal neoplasm. The bleeding is usually intermittent and often accompanied by nasal obstruction and pain. **Severe bleeding is unusual.** An exception occurs with juvenile nasal angiofibromas which have a higher incidence in adolescent males.

Patients without identifiable local causes for epistaxis most likely suffer from an underlying systemic process.[3] Congestive heart failure

raises the venous pressure in nasal venules and makes them more prone to bleeding.[3] Consider the possibility of a defect in the coagulation cascade. Hypertension is often cited as a risk factor for epistaxis. Studies have been unable to demonstrate a significant difference in the prevalence of epistaxis in patients with hypertension versus patients without hypertension.[15,16] Nonsteroidal anti-inflammatory drugs and aspirin-containing products have been associated with epistaxis.[17,18] Osler-Weber-Rendu disease or hereditary hemorrhagic telangiectasia is an autosomal dominant inherited condition in which the blood vessel walls lack contractile elements.[19] Prolonged heavy bleeding occurs from relatively minor insults as the vessels are unable to contract and allow clotting to take place. The patient's coagulation profile is normal and the diagnosis is made by the family history. Other systemic etiologies for epistaxis include alcoholism, blood dyscrasias, liver disease, malnutrition, and pregnancy. Routine laboratory studies are not needed unless the patient is symptomatic for blood loss or has indications of an underlying bleeding disorder.[20]

INDICATIONS

A patient with epistaxis must be evaluated immediately. All patients with epistaxis require a thorough examination to identify the source and control the bleeding (Figure 205-1). Epistaxis that has resolved still requires evaluation and potential management to prevent rebleeding.

CONTRAINDICATIONS

There are no absolute contraindications to the management of epistaxis. Disrupted anatomy, significant facial injury, or nasal hematomas may be relative contraindications to nasal packing. **Manage any unstable vital signs, impending airway compromise, life-threatening or limb-threatening injuries, or any complications related to blood loss (e.g., hypotension, chest pain, syncope) before managing the epistaxis.** Apply a nose clip (**Figure 205-4**), nasal sponge, or nasal tampon to control the bleeding in the interim. A thorough examination and more definitive means of control can be performed after the patient has been stabilized.

EQUIPMENT

- Nose clip
- Headlight or overhead light source
- Yankauer suction catheter
- Frazier suction catheters, #5 and #7
- Nasal speculum, short and medium lengths
- Bayonet forceps
- Kidney basin
- Tongue depressor
- Gown, gloves, face mask with an eye shield or goggles
- Cotton balls
- Topical anesthetics and vasoconstrictors
- Silver nitrate applicator sticks
- Petroleum (e.g., Vaseline) impregnated gauze ribbon, 0.5 inches wide
- Nasal tampons, various lengths
- Nasal balloons (e.g., anterior, posterior, anterior and posterior)
- 14 French Foley catheter with a 30 mL balloon
- Umbilical clamp

A

B

FIGURE 205-4. The nasal clip to stop epistaxis. ***A.*** The clip. ***B.*** The clip applied to a patient. (Courtesy of www.intranasal.net.)

- Gelfoam
- 4×4 gauze squares
- Surgicel
- ENTaxis nasal packing

TABLE 205-1 Atomizer Devices Available

Name	Manufacturer	Comments
DeVilbiss Model 15 Medical Atomizer	DeVilbiss Healthcare	Disposable and reusable versions Adjustable tip
Enk Fiberoptic Atomizer Kit	Cook Medical	Pressure-resistant oxygen tubing Atomizes through fiberoptic bronchoscope working channel
EZ-Spray	Alcove Medical	Disposable Trigger valve system
LMA MADdy Pediatric Mucosal Atomizer Device	Teleflex	Disposable single use Comes in various colors and shapes Child friendly Attaches to standard syringe 11.4 cm long
LMA Bottle MADomizer Atomizer	Teleflex	Disposable single-use tips Reusable bottle 10 cm long
LMA MADgic Laryngo-Tracheal Atomizer	Teleflex	Disposable single use Attaches to standard syringe Flexible tube 21.6 cm long
LMA MAD Nasal-Intranasal Mucosal Atomizer Device	Teleflex	Disposable single use Attaches to standard syringe

- 3 inch long dental rolls or tonsil packs or 3×36 inch Vaseline gauze
- Umbilical tape or 0 silk suture
- Red rubber catheters
- Lubricant

The list of equipment required to manage a patient with epistaxis is quite long. Most Emergency Departments keep all the required equipment in a rolling cart or tackle box except the medications. This system allows for more efficient stocking and restocking. It is more efficient to have all the required equipment readily available in one convenient and portable location. Single patient use and disposable nosebleed trays are commercially available (**Figure 205-5**). They contain some of the most used instruments that are disposable but lack the medications and other supplies. Become familiar with your institutions kit in advance of its need.

Atomizer devices are usually not available in the Emergency Department. Many devices are available commercially, are disposable, and are single patient use (**Figure 205-6 and Table 205-1**). A popular device is the MAD line of mucosal atomizer devices (LMA North America, San Diego, CA). They make multiple devices for all topical needs.

PATIENT PREPARATION

Treatment of any patient with epistaxis should start with ensuring a secure airway and hemodynamic stability. Resuscitative efforts should take initial priority over stopping epistaxis if the patient is unstable. Severe epistaxis may obstruct visualization of the airway and make direct laryngoscopy difficult. **Fiberoptic airway devices should be available to assist with intubation in anticipation of a difficult airway.**

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. Some Emergency Physicians just note a verbal consent in the medical record. Ensure that the patient has a thorough understanding of the postprocedural care instructions and follow-up requirements after the epistaxis has resolved.

Position the patient sitting in an upright multipositional procedure chair. Alternatively, place the patient sitting upright on a gurney with the back elevated. Prepare the wall suction unit to make sure

A

B

C

FIGURE 205-5. Examples of disposable nosebleed trays.

A

B

C

FIGURE 205-6. Some common nasal atomizers for topical nasal anesthesia. ***A.*** The LMA MAD nasal. ***B.*** The LMA MADgic laryngeo-tracheal. ***C.*** The child's LMA MADdy.

that it is working. Apply suction tubing and a Frazier suction catheter to the suction source. Don protective equipment (e.g., gowns, gloves, and protective eyewear) to prevent body fluid exposure.

The importance of good lighting cannot be overemphasized. Don a headlamp if one is available. An alternative is an overhead adjustable light source. Position the light so that it is aimed in the patient's mouth. A headlamp is preferred as the overhead light often hits the examiner in the head, casts shadows, and is too bright for the patient's eyes. It is also difficult to position properly as both hands must be used for the procedure.

Instruct the patient to blow their nose firmly one nostril at a time to evacuate the nasal cavities before the examination. This allows the anterior nasal septum, Kiesselbach's plexus, the nasal vestibule, the inferior turbinate, and the floor of the nasal cavity to be examined. Inspect the posterior oropharynx for active bleeding using a tongue depressor. Insert a short nasal speculum (**Figure 205-7**) to evaluate each side of the anterior nasal cavity. Open the speculum vertically. Suction out any blood and blood clots with the Frazier suction catheter. **Consider a posterior source of epistaxis if unable to identify a**

A

B

FIGURE 205-7. The Bionix disposable nasal speculum. ***A.*** The speculum. ***B.*** Using the nasal speculum. (Courtesy of Bionix, Toledo, OH.)

source of bleeding. It is sometimes difficult to determine from which side the bleeding is originating. A patient will occasionally present with bilateral epistaxis. Ask the patient which side started bleeding first. This is usually the side where the source of bleeding can be found.

Vasoconstrict and anesthetize the nasal mucosa (**Table 205-2**).[21] Decongest the nasal mucosa with an aerosolized agent (e.g., topical oxymetazoline or Afrin). Use an atomizer (**Figure 205-6**) if available. Put oxymetazoline diluted in saline in a syringe. Attach an atomizer. Give the patient a tissue to control any drips from their

TABLE 205-2 Anesthetics and Vasoconstrictors of the Nasal Mucosa

Anesthetics
4% cocaine
4% lidocaine
2% pontocaine
Topical anesthetic spray (e.g., Cetacaine)
Vasoconstrictors
4% cocaine
3% ephedrine
1:1000 epinephrine
0.5% oxymetazoline
0.5%–1.0% phenylephrine
0.05%–0.10% xylometazoline
Anesthetic and vasoconstrictor combinations
4% cocaine
0.25 mL of 1:1000 epinephrine and 20 mL lidocaine mixture
4% lidocaine and 0.05% oxymetazoline, 50:50 mixture
4% lidocaine and 0.5%–1.0% phenylephrine, 50:50 mixture

FIGURE 205-8. The use of the LMA MAD nasal to apply anesthetic and vasoconstrictor to the nasal cavity. (Courtesy of www.intranasal.net.)

nose. Instruct the patient to sniff in deeply after the spray is applied. Put the atomizer tip in the patient's nostril and briskly depress the syringe plunger (**Figure 205-8**). Allow 3 to 5 minutes for the vasoconstriction to occur. The use of oxymetazoline to vasoconstrict the nasal mucosa may stop the epistaxis and avoid nasal packing.[22] A small retrospective study demonstrated that 65% of Emergency Department patients with epistaxis were successfully treated with oxymetazoline and continuous pressure.[23] Next apply a topical anesthetic spray to the nasal passageway. Put 4 mL of 4% lidocaine in a syringe. Attach an atomizer. Give the patient a tissue to control any drips from their nose. Instruct the patient to sniff in deeply after the spray is applied. Put the atomizer tip in the patient's nostril and briskly depress the syringe plunger. Consider spraying the back of their oropharynx followed by the patient spitting it out. Spraying achieves excellent vasoconstriction and anesthesia as the agents diffuse through the entire nasal cavity and pharynx.

Alternatively, it is possible to anesthetize and vasoconstrict the nasal mucosa in one step by using cocaine or a combination of an anesthetic and vasoconstrictor agent (**Table 205-2**). Place cotton pledgets soaked with cocaine hydrochloride (i.e., 4% or 10% solutions) or LET (i.e., lidocaine 4%, epinephrine 0.1%, and tetracaine 0.4%) into the nasal cavity for 5 to 10 minutes to achieve anesthesia and vasoconstriction (**Figure 203-13**). Direct the pledgets along the floor of the nose, against the nasal septum, and toward the superior straits of the nose. Monitor the patient's vital signs when vasoconstrictors are applied.

There are numerous techniques to manage epistaxis (**Figure 205-1**). These include the use of absorbable packs, electrocautery, Foley catheters, gauze rolls, intranasal balloon catheters, petrolatum-impregnated ribbon gauze, nasal tampons or sponges, and silver nitrate. The technique and material of choice depend upon the location of the bleeding (i.e., anterior versus posterior) and Emergency Physician preference. Different techniques and equipment are required to control anterior versus posterior bleeding.

RADIOLOGIC STUDIES

Routine imaging is not indicated for the evaluation and management of epistaxis in the absence of trauma. A study looking at the utility of computed tomography (CT) scanning in a pediatric population with epistaxis found that 79% of the studies demonstrated some abnormality but none of the patients were found to have a neoplasm.[24] Obtain CT imaging of the paranasal sinuses and facial bones when there is a history of facial trauma or when there is a high index of suspicion for a neoplasm.

ANTERIOR EPISTAXIS MANAGEMENT TECHNIQUES

Anterior nasal packing is required when local measures fail to control epistaxis. This may be due in part to anterior or structural problems in which the bleeding source cannot be identified. It may be due to heavy or profuse bleeding. **Nasal packing is effective by providing mechanical pressure and tamponades the bleeding site.[25] Nasal packing is an uncomfortable procedure. The previously described steps for applying topical anesthesia should be undertaken.**

ABSORBABLE PACKING

Diffuse bleeding is frequently encountered in patients with coagulopathies and blood dyscrasias. The trauma of inserting the nasal packing (e.g., a tampon or petrolatum gauze) can lead to more serious bleeding. A Gelfoam sponge or oxidized cellulose (i.e., Surgicel) is often effective in stopping epistaxis. These substances provide adequate pressure and hemostasis without extreme trauma to the nasal mucosa. This packing does not need to be removed and will slowly dissolve with the use of a topical saline spray which may be started within 24 hours of the packing being placed.

The two most commonly used absorbable dressings are Gelfoam and Surgicel. Gelfoam is an absorbable gelatin sponge that is readily available and inexpensive. It forms a scaffold for the formation of a blood clot. Surgicel is composed of oxidized and regenerated cellulose. It promotes coagulation better than Gelfoam. Surgicel may result in delayed healing and its use should be reserved for persistent bleeding or when Gelfoam is not available.

Absorbable packs may be used for primary and secondary hemostasis. Apply a piece of Gelfoam or Surgicel directly over the site of discrete bleeding or diffuse oozing. The material may be used for secondary hemostasis via placement over an area that has clotted and stopped bleeding. This can serve as a "Band-Aid" to help prevent premature clot dislodgement and rebleeding. These packs can be placed over areas that have been chemically or electrically cauterized. An absorbable pack can be placed prior to packing the nasal cavity with a sponge/tampon or gauze. The absorbable pack will prevent the clot from becoming dislodged when the sponge/tampon or gauze is removed.

Insert the nasal speculum and identify the scabbed or bleeding site. Apply a piece of Gelfoam or Surgicel over the site. Allow a clot to form onto the absorbable packing. The nasal cavity may then be packed with a sponge/tampon, petrolatum gauze, or a balloon catheter if the Emergency Physician chooses to do so in the clinical setting. Packing with absorbable materials tends to be easy to do and comfortable for the patient as the material conforms to the patient's nasal contours.

Two additional absorbable dressings are topical thrombin and collagen. They are expensive, are not usually available in the Emergency Department, and should be limited to circumstances where other hemostatic methods have failed. Topical thrombin is derived from bovine thrombin. Place a piece of Gelfoam saturated with thrombin over the bleeding site. Thrombin converts fibrinogen to fibrin and bypasses the coagulation cascade to form a clot. Collagen is available in multiple forms from a variety of sources. It promotes platelet aggregation and forms a scaffold for the formation of a clot. Cover the bleeding site with collagen followed by a piece of Gelfoam.

CHEMICAL CAUTERIZATION

The most common location of bleeding is within the anterior nasal cavity and on the anterior nasal septum. Sometimes no active bleeding is found at the time of evaluation. Suctioning of the nasal cavity will remove clots and may allow the site to bleed and be visualized. A scabbed excoriation or an exposed blood vessel may be found along the nasal septum. Chemical cauterization of these areas can be achieved using silver nitrate applicators.[26]

Insert the nasal speculum and identify the scabbed site or the exposed vessel. Apply silver nitrate under direct visualization by rolling the applicator on the area immediately surrounding the scab or blood vessel (**Figure 205-9**). Apply the silver nitrate for 3 to 10 seconds. **Do not apply the applicator in any one spot for more than 10 seconds. This may cause damage and mucosal necrosis to the underlying cartilaginous septum. Do not apply the silver nitrate excessively or in the same spot on both sides of the septum. This may result in a septal perforation.** Apply a topical antibiotic ointment to the area and consider placing a piece of Gelfoam or Surgicel over the site to help stabilize the clot.

A relatively dry field is required to use the silver nitrate applicator. Moderate to severe bleeding results in the silver nitrate coagulating the blood. This prevents contact with the mucosal tissue and bleeding will continue. Attempt to simultaneously suction the blood while using the silver nitrate applicator. Unfortunately, the suction often pulls off the coagulum and the bleeding continues. A final technique is to apply the silver nitrate centripetally around the bleeding site. This will cauterize the feeder vessels and may stop the bleeding. **Do not cauterize an area over 0.75 cm in diameter. This can result in damage to the underlying cartilaginous septum.** Pack the nasal cavity with a sponge/tampon, petrolatum gauze, or a balloon catheter if these techniques fail.

ELECTRICAL CAUTERIZATION

Electrocautery can effectively control bleeding if the site is identified. **This technique is reserved for the experienced Otolaryngologist.** An electrocautery device should be available for use by the Otolaryngologist. It can cause significant damage to the mucosa and cartilage in inexperienced hands. The technique of electrocautery is not discussed for these reasons.

RIBBON GAUZE PACKING

The traditional technique of anterior nasal packing consists of using 0.5 inch wide petrolatum-impregnated gauze ribbon (**Figure 205-10**). This technique is extremely effective but not often used as it is cumbersome, time-consuming, and simpler methods now exist (e.g., silver nitrate cautery, sponges, tampons, and balloon catheters).

Insert a nasal speculum. Grasp one end of the petrolatum gauze with a bayonet forceps. Insert the petrolatum gauze into the nasal cavity and along the nasal floor (**Figure 205-10*A***). Tightly pack the nasal cavity in a layered fashion from bottom to top, extending as far back as possible toward the choanal arch (**Figure 205-10*B***).

FIGURE 205-9. Using silver nitrate for chemical coagulation of epistaxis. (Courtesy of www.intranasal.net.)

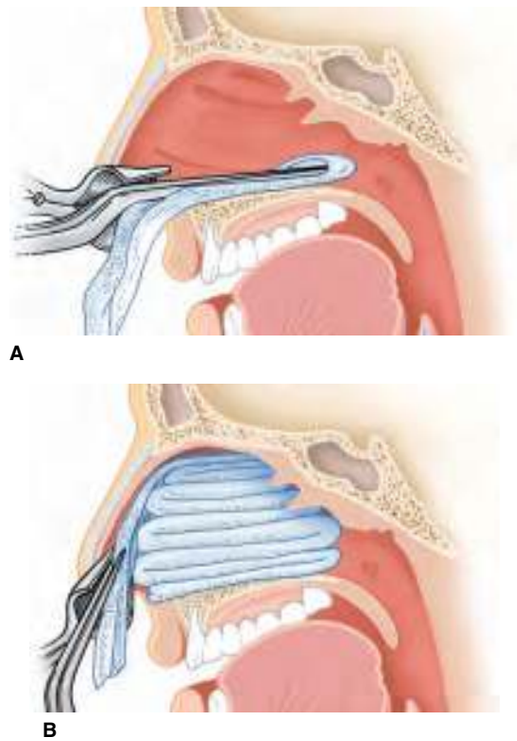

FIGURE 205-10. Anterior nasal packing using petrolatum (e.g., Vaseline) impregnated gauze ribbon. ***A.*** Insert a nasal speculum and begin packing inferiorly to superiorly. ***B.*** The gauze-packed anterior nasal cavity.

Be careful to avoid injuring the mucosa overlying the septum and the turbinates. Cut the petrolatum gauze so that it protrudes approximately 2 cm from the nostril. Tape this loose end to the patient's cheek so that it does not accidentally pull the packing out. The packing is later removed by gently pulling on this free end of gauze ribbon protruding from the nostril.

The pressure of one-sided anterior nasal packing can sometimes bow the septum contralaterally allowing the packing to "loosen" and the bleeding to restart. Consider packing the contralateral anterior nasal cavity to maintain the septum in the midline and exert pressure on the bleeding site.

EXPANDABLE NASAL SPONGES/TAMPONS

One of the easiest, quickest, and most effective techniques to control anterior epistaxis is to insert an expandable sponge or tampon (e.g., Rhino Rocket or Merocel packing). These packs are particularly useful when the bleeding is diffuse, a specific site cannot be clearly identified, or the bleeding is heavy. The packs come in various sizes (e.g., 4.5, 6, 8, and 10 cm), shapes, and styles (**Figure 205-11**). Initially quite rigid, they soften and expand with the absorption of saline or surrounding blood. A 4.5 or 6 cm sponge is generally adequate for anterior epistaxis.

Prepare the sponge/tampon. Cut the string from the sponge/tampon as it is not necessary to remove the packing. The sponge/tampon is barely visible when properly inserted. The string hanging from the nostril can be irritating to the patient and is not cosmetically appealing. Lightly coat two-thirds of the sponge/tampon with a non–water-soluble lubricant (e.g., Vaseline) or antibiotic ointment (e.g., Neosporin). This will prevent premature expansion of the tampon from a water-soluble lubricant or antibiotic ointment, nasal secretions, or blood.

Insert and open the nasal speculum within the affected nasal cavity. Grasp the unlubricated end of the sponge/tampon with a bayonet forceps or the dominant thumb and index finger. Insert and advance the sponge/tampon just lateral to the nasal septum, in a vertical position, with the length of the pack directed along the floor of the nose (**Figure 205-12*A***). Advance the sponge/tampon until it is completely within the nasal cavity. The sponge/tampon will expand from the blood and secretions within the nasal cavity (**Figure 205-12*B***). Slowly drip 1 to 3 mL of tap water or saline onto the unlubricated tip of the sponge/tampon to help it expand more rapidly.

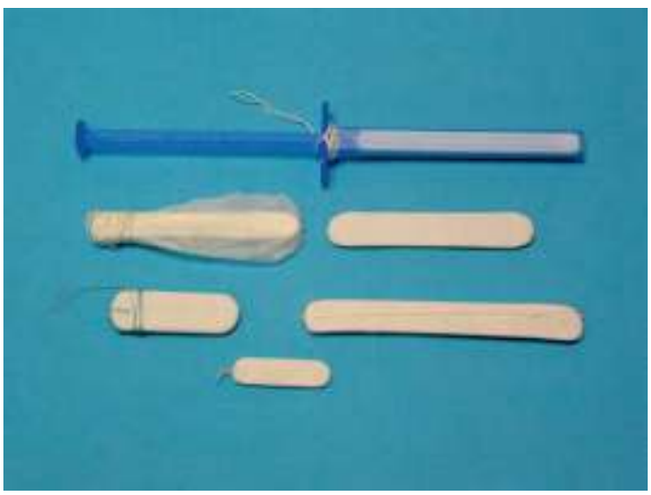

FIGURE 205-11. Examples of some of the various sizes, shapes, and styles of nasal sponges/tampons.

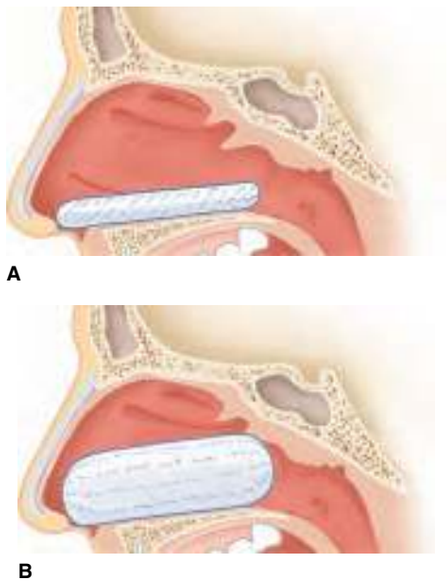

FIGURE 205-12. The expandable nasal sponge/tampon. ***A.*** Insertion along the floor of the nasal cavity. ***B.*** The expanded state.

Inspect the oropharynx for bleeding. Persistent bleeding may be due to the septum bowing contralaterally. Pack the contralateral anterior nasal cavity with a sponge/tampon of equal length and size. Observe the patient for continued bleeding anteriorly or posteriorly. Continued bleeding requires removal of the sponge/tampon from the bleeding nasal cavity and insertion of a larger one, two small ones, or Vaseline gauze packing.

Keep several helpful hints in mind when using the nasal sponges/tampons. Trim long sponges/tampons to 5 to 6 cm in length. The extra length is not required for anterior epistaxis, is uncomfortable for the patient, and is more difficult to remove. The Rhino Rocket is a sponge/tampon within a plastic syringe-like device. Consider removing the sponge/tampon from the syringe-like device before inserting it. The device can generate significant force and result in mucosal tears, septal injuries, and turbinate injuries. Insert and advance the sponge/tampon rapidly to prevent premature expansion. There is no advantage to using a non–water-soluble antibiotic ointment versus a lubricant. The antibiotic ointment is more expensive and its antibacterial activity lasts only 2 to 4 hours. Insert two sponges/tampons side by side if the patient has a large nasal cavity. It is important to instruct the patient to keep the packing moist with saline nasal drops when leaving any nasal packing in place.

Removal of the sponge/tampon is simple and quick. Apply 1 to 2 mL of tap water, saline, or a diluted vasoconstrictor in a dropwise fashion to the tip of the sponge/tampon in the nostril. This will thoroughly hydrate the packing and ensure that it can be withdrawn without causing further trauma. Allow 5 to 10 minutes to ensure that the packing is completely hydrated. Grasp the end of the sponge/tampon with a hemostat. Place a kidney basin under the patient's nose. Pull quickly and parallel to the floor of the nasal cavity to remove the packing. Epistaxis after removal is often due to dislodgement of the clot. Use an absorbable pack or silver nitrate to stop the bleeding. Look at the sponge/tampon to identify the blood spot and the location of the bleeding.

INFLATABLE NASAL BALLOON CATHETERS

The development of plastic inflatable balloon catheters has greatly simplified the management of epistaxis as they are easy to use and quick to place. Nasal balloon catheters are available in a variety of

FIGURE 205-13. Examples of some inflatable nasal balloon catheters.

sizes and shapes with anterior balloons, posterior balloons, or dual balloons (**Figure 205-13**). The anterior balloon, often covered with a carboxycellulose outer layer to promote platelet aggregation, fills the nasal cavity and acts as an anterior pack. The posterior balloon occludes the nasopharynx and acts as a posterior pack. The inflatable balloons are more expensive than other methods used to manage epistaxis. The increased cost is offset by decreased Emergency Physician contact time as compared with that required for petrolatum gauze packing. The balloons have a maximal inflatable volume that is manufacturer-specific, on the packaging, and on the balloon's inflation hub.

Prepare the equipment for an anterior epistaxis. Elect an anterior balloon catheter. A dual-balloon catheter may be used if an anterior balloon catheter is not available. Inflate the balloon with air to just below its maximum capacity. Observe and palpate the balloon for leaks. It may be inflated in a cup of water to look for leaks. Completely deflate the balloon and apply a lubricant over the catheter and balloon.

Insert the nasal speculum and identify the bleeding location. **Insert the catheter with the distal bevel toward the nasal septum.** This prevents the distal end of the catheter from getting caught on the turbinates, damaging the mucosa overlying the turbinates, and causing a second source of epistaxis. Advance the catheter along the floor of the nasal cavity until just the inflation hub is protruding from the nostril (**Figure 205-14**). Inflate the balloon with 10 mL less air than the maximum volume of the balloon (**Figure 205-14**). **Do not use saline or water to inflate the balloon.** Rupture of the balloon can result in aspiration if it is filled with liquid. **Do not inflate the balloon with more than the manufacturer's recommended volume.** The balloon may have been inflated larger than the nasal cavity if the patient complains of pain. Slowly deflate the balloon in 2 mL increments until the pain subsides.

Observe the patient for continued bleeding. Increase the volume of the balloon to the maximum volume if the patient does not complain of pain. The balloon may cause a bowing of the septum to the contralateral side. Pack the contralateral anterior nasal cavity to keep the septum in the midline if the bleeding continues. Observe the patient for further bleeding from the nostril or into the nasopharynx. Continued bleeding suggests that the source is high in the nasal cavity or posterior. **The inflatable balloons do not always fill the upper portion of the nasal cavity.** Deflate the balloon, pack the high anterior nasal cavity with Vaseline gauze, and reinflate the balloon. Observe the patient for continued bleeding that would require a posterior pack as described in the following sections.

RAPID RHINO

The Rapid Rhino Nasal Pac catheter (Smith & Nephew, Inc., Austin, TX) is a form of balloon catheter. The balloon is covered with a hydrocolloid fabric that is self-lubricating, is easy to insert, and promotes platelet aggregation. It is available in different lengths (i.e., 4.5, 5.5, and 7.5 cm) to accommodate children, anterior epistaxis, and posterior epistaxis. It is available in a unilateral balloon design and a bilateral balloon design with a single inflation port. The bilateral model allows for bilateral nasal packing with a single inflation port and maintains equal pressure on both sides of the nasal septum. Dip the fabric-covered balloon in sterile water for approximately 30 seconds to prelubricate it prior to insertion. Insert the balloon and inflate it as described previously. The advantage to this product is the ease of insertion, minimal bleeding upon removal, and patient comfort.[27-29]

ENTaxis NASAL PACKING

An anterior nasal packing is the ENTaxis (**Figure 205-15**). It is composed of a natural polymer derived from seaweed and contains calcium alginate.[30] The packing provides hemostasis, has healing properties, and is atraumatically inserted and removed. Hydration with normal saline activates the calcium alginate and makes

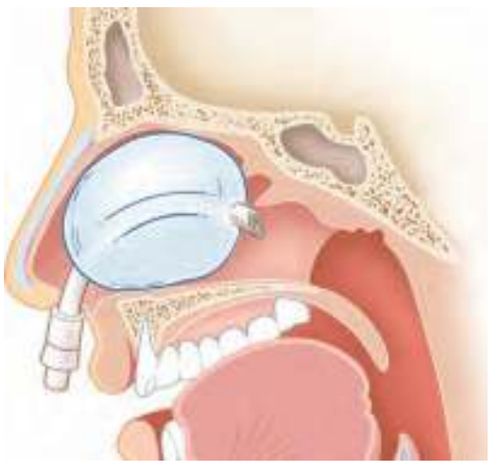

FIGURE 205-14. Inflation of an anterior balloon catheter to control anterior epistaxis.

FIGURE 205-15. The ENTaxis nasal packing. (Boston Medical Products, Westborough, MA.)

it pliable. The packing expands 300% and conforms to the anterior nasal cavity. It activates platelet aggregation to provide hemostasis, keeps the nasal mucosa moist, and gels into a smooth surface that allows easy removal.

Prepare the packing. The ENTaxis is packaged in a plastic tray with a tear-off paper lid. Peel the lid completely off the tray. Fill the tray with normal saline to saturate the packing. Remove the hydrated packing from the tray. Squeeze out the excess saline from the packing.

Insert a nasal speculum. Grasp the packing on the end opposite the string with a bayonet forceps. Insert the packing along the floor of the nasal cavity. Continue to insert the packing in an accordion-like fashion until the entire packing is within the nasal cavity. The packing will expand and gel to fill the nasal cavity. Secure the string to the patient's cheek or nose with a piece of tape.

Removal of the packing is simple and quick. The packing remains hydrated and in a gelled state while within the nasal cavity. Place a kidney basin under the patient's nose and remove the tape from the string on the patient's face. Grasp the packing with a bayonet forceps and gently withdraw the packing from the nasal cavity.

TOPICAL THROMBIN

Topical bovine thrombin is approved for the management of epistaxis. It works on sites of minor bleeding and oozing. The thrombin causes fibrinogen in the blood to clot without the requirement of platelet activation. The thrombin comes in many forms (e.g., dry powder, liquid in syringes, and liquid spray).

Insert a nasal speculum and find the bleeding source. Hydrate and mix the topical thrombin according to manufacturer directions. Attach the nasal delivery device. Insert the nasal delivery device into the nares and spray the thrombin solution onto the nasal mucosa. Remove the device and allow the solution to form a clot. Apply additional thrombin solution if the bleeding continues. Consider applying a piece of Gelfoam to the newly formed clot to support it.

THROMBIN-GELATIN MIXTURES

The Floseal Hemostatic Matrix (Baxter Healthcare Corporation, Hayward, CA) and the Surgiflo Hemostatic Matrix (Ethicon, Patterson, NJ), among others, are composed of human-derived thrombin and bovine-derived gelatin. They stop bleeding fast (approximately 2 minutes) in up to 97% of cases.[19,31] The thrombin-gelatin matrix begins to break down in 3 to 5 days and is fully dissolved by 7 days. Floseal does not require platelet activation, allowing it to function in patients taking aspirin and other antiplatelet medications. Floseal and Surgiflo are provided in components that must be mixed. The process is more complicated than other thrombin products and can take several minutes. The kit contains all the required components and supplies. Attach one of the two applicator tips to the syringe containing the gelatin-thrombin solution.

Apply a small mound of the Floseal to the bleeding source. The Floseal must remain at the site for 2 minutes. Place a sterile saline-moistened gauze sponge over the Floseal mound to ensure it maintains a seal against the bleeding site. Remove the gauze after 2 minutes and inspect the site. If the gauze adheres to the clot or the Floseal, moisten it with sterile normal saline. Insert the applicator tip through the Floseal mound and deliver fresh Floseal to the bleeding site if bleeding persists. Remove any excess Floseal by gentle irrigation after the bleeding is controlled. Consider applying a piece of Gelfoam to the newly formed clot to support it.

The procedure for using Surgiflo is different. Reconstitute it in sterile water and draw it into a syringe. Apply the applicator tip onto the syringe. Place the applicator tip into the nasal cavity. Fill the nasal cavity from the back to the front and fill the nasal cavity while withdrawing the syringe. Allow the mixture to gel and stop the bleeding.

WOUNDSEAL

WoundSeal powder (Biolife LLC, Sarasota, FL) was previously marketed as NoseBleed QR and WoundSeal for Nosebleeds. It is an over-the-counter product that comes in a variety of applications to control bleeding (**Figure 205-16**). It is marketed to control epistaxis and external wounds of all types. It consists of a powder containing a hydrophilic polymer and potassium ferrate. The powder is loaded onto an applicator swab and applied to the site of bleeding. When the powder comes in contact with blood, the polymer absorbs liquid and concentrates the red blood cells and plasma proteins under the powder to form a clot. The potassium ferrate releases iron to bind the blood proteins into a clot.

The WoundSeal powder is simple to apply. Open the blister pack. Roll the tip of the applicator stick in the powder to completely coat it. Roll the powder-coated applicator onto the nasal mucosa. Remove the applicator stick from the nasal cavity. Pinch the nostrils closed for 30 seconds. Reassess the nasal cavity for continued bleeding and the need for additional applications of the powder. Consider applying a piece of Gelfoam to the newly formed clot to support it.

ANKAFERD BLOOD STOPPER

Ankaferd Blood Stopper (ABS, Ankaferd Health Products LTD, Istanbul, Turkey) is a medicinal plant extract. It is approved in Turkey for the management of postsurgical dental bleeding and external hemorrhage.[32] ABS is not currently available in the United States and many other countries. It induces the rapid formation of a hemostatic protein network to control hemorrhage that is that is not dependent upon coagulation factors and platelets.[18] It has been successfully used to control epistaxis.[33] ABS is available in tampons and liquid spray that may provide a new method to control epistaxis in the future.

FIGURE 205-16. The WoundSeal and applicator.

TRANEXAMIC ACID (TXA)

Tranexamic acid (TXA) has recently emerged as a potential primary or adjunctive treatment for epistaxis. TXA is an antifibrinolytic agent that reversibly binds to plasminogen. It inhibits plasminogen from binding to fibrin. This interaction prevents plasminogen activating thrombin and prevents clot dissolution.[34] TXA is a synthetic analog of lysine. It is available in 100 mg/mL solution for injection and oral tablets. An Internet search revealed liquid TXA to be inexpensive (e.g., $1.37/mL) and more cost effective than other equipment used to treat epistaxis.

A study evaluated 216 patients with presumed anterior epistaxis and randomized them to receive packing soaked in TXA (500 mg in 5 mL) or packing soaked in lidocaine with epinephrine followed by anterior nasal packing for 3 days.[35] This study demonstrated a statistically significant decrease in time to epistaxis resolution and discharge from the Emergency Department in the TXA group. Topical TXA is largely considered safe for use without complications.[36,37] A Cochrane review evaluated the effects of topical TXA administration to control bleeding in patients undergoing surgery or those with epistaxis.[36] This review suggested that topical application of TXA reduces bleeding with no significant increase in thromboembolic events. TXA has been used successfully in epistaxis for patients taking oral anticoagulants.[38]

POSTERIOR EPISTAXIS MANAGEMENT TECHNIQUES

A posterior source of the bleeding must be considered when epistaxis is bilateral, brisk, or not controlled with anterior nasal packing. It is estimated that 5% of all cases of epistaxis originate from a posterior source.[39] **The placement of a posterior nasal pack is extremely uncomfortable.** The patient usually requires intravenous sedation and analgesia (Chapter 159) in addition to the topical anesthesia. Consider spraying the patient's soft palate, uvula, and oropharynx with a topical anesthetic spray (e.g., Cetacaine). This will minimize their gag reflex during the placement of the posterior pack. **Endotracheal intubation is sometimes required to appropriately visualize and control posterior epistaxis.**

The rationale behind placing a posterior pack is that the occlusion of the choanal arch provides a semirigid buttress against which anterior nasal packing may be placed, allowing adequate hemostasis to be achieved. This buttress may be formed from either a gauze pack, a 30 mL Foley catheter (e.g., size 12 to 14 French), an expandable nasal sponge/tampon, or an inflatable nasal balloon catheter. The Foley catheter and inflatable nasal balloon catheter are most easily tolerated by the patient. The inflatable nasal balloon catheter is the easiest to place and has two balloons that serve as anterior and posterior packs. **An anterior nasal pack is always required on the side of a posterior pack.** Strongly consider inserting a contralateral anterior nasal pack to maintain the septum in the midline. **Epistaxis that requires posterior packing should be managed in conjunction with an Otolaryngologist. Admission to a monitored setting is warranted due to the potential for complications (e.g., dysrhythmias, hypoxia, hypoventilation, and airway compromise)**.

TRADITIONAL (GAUZE ROLL) PACKING

The traditional technique of posterior nasal packing involves using rolled gauze, dental rolls, or tonsil packs placed through the oropharynx (**Figure 205-17**). This technique is difficult, time-consuming, and messy; requires many supplies; and is not well tolerated by the awake patient. Easier and quicker techniques exist and for these reasons this technique is not often performed.

Prepare the equipment. Gather the required equipment on a bedside procedure table. Use 3 inch long dental rolls or tonsil packs. An alternative is to use 3 inch wide and 36 inch long petrolatum gauze formed into a tight cylindrical roll (**Figure 205-17*A***). Tie two pieces of umbilical tape or 0 silk suture around the pack to divide it into thirds (**Figure 205-17*A***).

Anesthetize and vasoconstrict both nasal cavities. Apply a topical anesthetic spray to the soft palate, uvula, and oropharynx. Lubricate a red rubber catheter. Pass the red rubber catheter through one nostril and along the floor of the nasal cavity. Advance the catheter so that the tip is visible in the patient's oropharynx. Grasp the tip of the catheter with a hemostat or forceps and pull it out the patient's mouth (**Figure 205-17*B***). Clamp the free ends of the catheter together. Pass a second red rubber catheter through the other nostril and out the patient's mouth.

Insert the posterior pack. Tie the free end of one piece of the umbilical tape or silk surrounding the packing to the distal end of one red rubber catheter exiting the patient's mouth (**Figure 205-17*C***). **Tie the knots tightly.** Tie the second piece of umbilical tape or silk to the second red rubber catheter. Pull the proximal ends (i.e., the portion exiting the nostrils) of both red rubber catheters until the packing is against the choanae (**Figure 205-17*D***). It may be necessary to place a finger into the patient's mouth and push the pack behind the soft palate and uvula if it gets caught (**Figure 205-17*D***). Place a hemostat on both pieces of umbilical tape exiting the nostrils. Apply slight traction with the hemostat to maintain the posterior pack against the choanae. Instruct an assistant or the patient to hold the hemostat. Untie or cut the red rubber catheters from the umbilical tape or silk sutures.

Place an anterior pack and secure the posterior pack. **An anterior nasal pack is always required when placing a posterior nasal pack.** The anterior pack may be an expandable sponge/tampon, petrolatum gauze, or a balloon catheter (**Figure 205-17*E***). Tie the umbilical tape or silk exiting each nostril together (**Figure 205-17*F***). **Always place a piece of cotton or gauze between the knot and the columella to prevent pressure necrosis (Figure 205-17*F*). Tie the umbilical tape or silk snugly but not too tight to hold the posterior pack in place and minimize pressure on the choanae and the columella.** Tape the umbilical tape or silk exiting the patient's nostril and mouth to their face (**Figure 205-17*F***).

Removal of this packing is simple and requires several stages. Remove the anterior packing. Thoroughly examine the mucosa to make sure that the epistaxis has not restarted. Suction any clots and blood from the nasal cavity. Rebleeding requires the placement of a new anterior pack if it cannot be controlled with an absorbable dressing or silver nitrate. Cut the knot securing the umbilical tape or silk around the columella. Pull the pieces of umbilical tape or silk exiting the patient's mouth to remove the posterior pack completely.

FOLEY CATHETER TECHNIQUE

A Foley catheter may be used to provide a posterior buttress. Using a Foley catheter to control posterior epistaxis is relative quick, easy, and simple as it does not require the preparation of specialized equipment. Select a 12 or 14 French Foley catheter with a 30 mL balloon. Inflate the balloon with air and ensure the integrity of the balloon. Some Emergency Physicians cut off the portion of the Foley catheter distal to the balloon as they believe that the distal tip is irritating to the patient and may stimulate their gag reflex. The practice of cutting off the distal tip is based purely on Emergency Physician preference. Be careful to not cut the balloon or its attachment to the catheter.

Lubricate the distal third of the Foley catheter. Insert the Foley catheter into the nostril and along the floor of the nasal cavity.

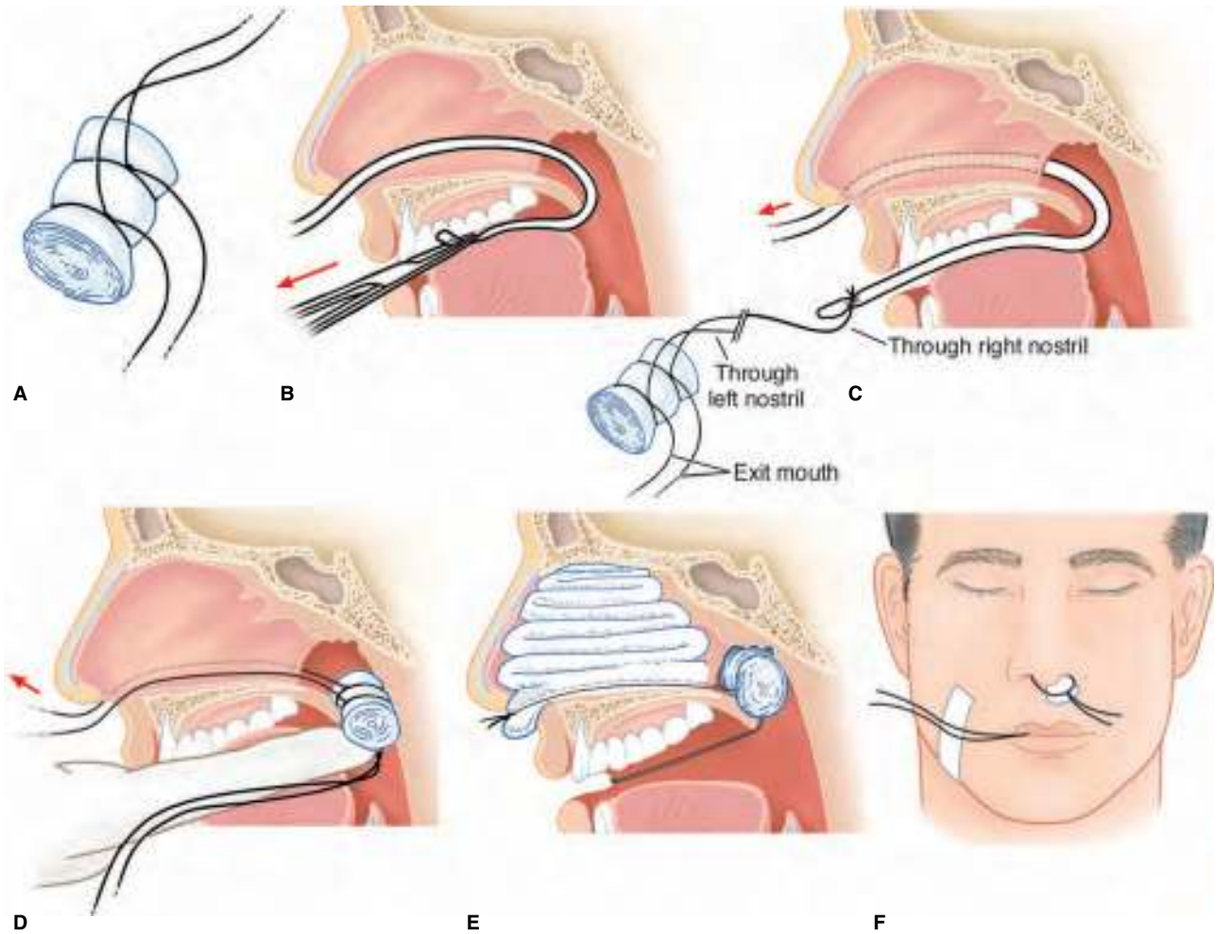

FIGURE 205-17. The traditional technique of placing a posterior nasal pack. ***A.*** Preparation of the pack. ***B.*** A red rubber catheter inserted through the nostril and pulled out the mouth. ***C.*** The pack is attached to the two red rubber catheters. ***D.*** The pack is pulled into place. Use a finger to pass the pack around the soft palate and uvula. ***E.*** An anterior nasal pack has been placed. ***F.*** The ties of the posterior pack are secured.

Advance the catheter until the tip is visible in the patient's oropharynx. Inflate the balloon with 7 to 10 mL of air. **Do not use saline as the fluid can result in aspiration if the balloon ruptures.** Withdraw the catheter to lodge the balloon against the choanal arch (**Figure 205-18*A***). If the balloon withdraws into the nasal cavity, advance it back into the nasopharynx. Add an additional 3 to 5 mL of air and withdraw the catheter. Continue the process by adding 3 to 5 mL aliquots of air until the balloon lodges against the choanal arch. Inflate the balloon with an additional 3 to 5 mL of air and until the soft palate just begins to bulge. **The balloon is overinflated if the soft palate bulges or the patient experiences pain.**

Place an anterior pack and secure the Foley catheter. Apply slight traction to maintain the balloon against the choanal arch. Instruct an assistant or the patient to hold the Foley catheter. Place the anterior pack using an expandable sponge/tampon, petrolatum gauze, or a balloon catheter. **Place a piece of cotton or gauze against the columella and nasal ala to prevent pressure necrosis.**[40] Place an umbilical clamp on the Foley catheter and over the protective

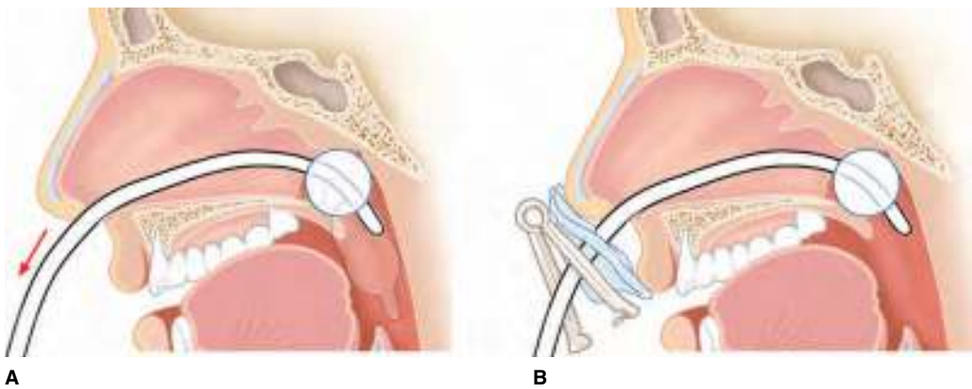

FIGURE 205-18. The Foley catheter technique. Insert a Foley catheter along the floor of the nasal cavity until the tip is visible in the patient's oropharynx. ***A.*** Inflate the balloon and withdraw the catheter to lodge the balloon against the choanal arch. ***B.*** Secure the Foley catheter.

padding (**Figure 205-18*B***). **The clamp must hold the balloon against the choanal arch without applying pressure to the choana or columella.**

Removal of this packing is simple and requires several stages. Cut the open loop of the umbilical clamp with a scissors. Open the jaws of the clamp and remove them from the Foley catheter. **Always maintain a grasp of the catheter and apply slight traction to keep the balloon in place.** Remove the anterior packing. Thoroughly examine the mucosa to make sure that the epistaxis has not restarted. Suction any clots and blood from the nasal cavity. Rebleeding requires the placement of a new anterior pack if it cannot be controlled with an absorbable dressing or silver nitrate. Deflate the balloon and pull the catheter out the patient's nostril.

EXPANDABLE NASAL SPONGES/TAMPONS

Many Emergency Physicians prefer to use an 8 or 10 cm sponge/tampon rather than the other techniques of posterior packing as this technique is easy, quick, simple, and inexpensive. The technique for insertion and removal is the same as for the anterior packing technique described previously. A combination device with an anterior nasal sponge/tampon and a posterior balloon is available. It provides better posterior hemostasis than a sponge/tampon alone.

INFLATABLE DUAL-BALLOON NASAL CATHETERS

A dual-balloon catheter can be used when a posterior pack is required. These catheters come in many types and styles (**Figure 205-13**). They are expensive, easy to use, and quickly placed. The smaller distal balloon obstructs the choanal arch and acts as a posterior pack (**Figure 205-19**). The larger proximal balloon fills the nasal cavity and acts as an anterior pack (**Figure 205-19**). The distal balloon holds 10 mL and the anterior balloon holds 30 mL in most dual-balloon systems. Note the manufacturer's maximum volume recommendations on the package and on the inflation ports of the balloons.

Prepare the equipment. Fully inflate both balloons with air. Observe and palpate the balloons for leaks. They may also be inflated in a cup of water to look for leaks. Completely deflate both balloons and apply a lubricant over the catheter and balloons.

Insert the catheter with the distal bevel toward the nasal septum. This prevents the distal end of the catheter from getting caught on the turbinates, damaging the mucosa overlying the turbinates, and causing a second source of epistaxis. Advance the catheter along the floor of the nasal cavity until the distal tip is visible in the patient's oropharynx.

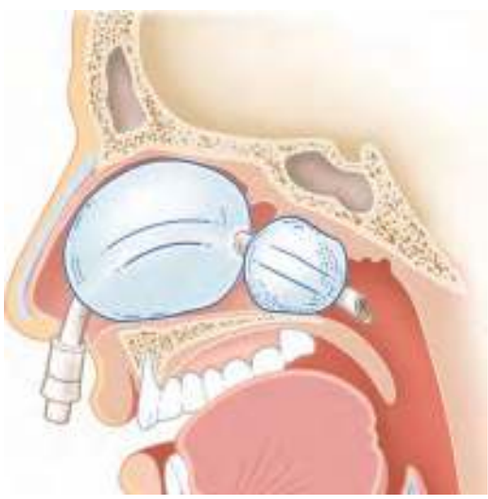

FIGURE 205-19. Placement of a dual-balloon catheter.

Inflate the distal balloon with 4 to 5 mL of air. **Do not use saline or water to inflate the balloon.** Rupture of the balloon can result in aspiration if it is filled with liquid. Withdraw the catheter to lodge the balloon against the choanal arch (**Figure 205-19**). If the balloon withdraws into the nasal cavity, advance it back into the nasopharynx. Add an additional 2 to 3 mL of air and withdraw the catheter. Continue this process until the balloon lodges or the maximum balloon volume is reached. Inflate the anterior balloon with 20 to 25 mL of air.

Observe the patient for bleeding. Inflate both balloons with additional aliquots of air until the bleeding stops or the maximum balloon volume is reached. The balloon does not always fill the high anterior nasal cavity. Deflate the anterior balloon, pack the high anterior nasal cavity with Vaseline gauze, and reinflate the anterior balloon if the bleeding continues. Place an anterior pack on the contralateral side if the bleeding continues to maintain the septum in the midline and apply pressure to the ipsilateral nasal cavity.

SPHENOPALATINE ARTERY BLOCK

This technique is used only as a last resort when an Otolaryngologist is not available, the hemorrhage is unremitting, and other methods to control the epistaxis have failed. Local anesthetic solution is injected into the pterygopalatine canal to occlude the sphenopalatine artery. The local anesthetic solution can cause pressure necrosis of the adjacent nerves.

Place the patient supine with their mouth open. Apply a topical anesthetic spray to the hard palate. Identify the greater palatine foramen. Insert a 27 gauge needle into the mucosa of the hard palate, 1 cm medial to the gum line between the junction of the second and third maxillary molars. Probe this area with the needle until it falls into the greater palatine foramen. Inject 0.25 mL of local anesthetic solution containing epinephrine into the mucosa overlying the greater palatine foramen. Arm a 3 or 5 mL syringe containing local anesthetic solution with epinephrine with a 22 or 25 gauge needle. Insert the needle approximately 25 to 28 mm into the greater palatine foramen and inject 3 mL of the local anesthetic solution.

SURGICAL INTERVENTION

Consult an Otolaryngologist when the epistaxis is difficult to control by the described methods above. Surgical therapy may include septoplasty, endoscopic cauterization, arterial ligation (e.g., internal maxillary artery, sphenopalatine artery, anterior and posterior ethmoid arteries, external carotid artery), or embolization by an Interventional Radiologist.[41-45] Surgical and angiographic measures have not been shown to have benefits in regard to morbidity and mortality when compared with packing.[46-48] **Packing should be attempted first with surgical and angiographic measures reserved for refractory cases.**

PEDIATRIC CONSIDERATIONS

Epistaxis is a common childhood symptom that may prompt an evaluation in the Emergency Department.[8] The management of a child with acute epistaxis is straightforward. The source of bleeding is typically from the anterior one-third of the nasal septum just posterior to the squamous-mucosal junction in most children. The etiology is often due to digital manipulation, infection, allergic rhinitis, a coagulopathy, a foreign body, trauma, medications, a neoplasm, or recent surgery.[49] Epistaxis in the neonate and infant is rare.[50] Consider nonaccidental trauma (i.e., abuse) in this group.[50]

The principles of management in the pediatric population are like those of adult patients. Active bleeding requires immediate attention. Assess the child for any hemodynamic instability or airway compromise. Obtain a brief history during the initial stages of treatment. The minimal equipment required for initial evaluation

includes a headlight, Yankauer suction, Frazier tip suction, a small nasal speculum, and a tongue depressor. Additional supplies may be required for the clinical situation. A more comprehensive list of equipment and medications has been outlined earlier in this chapter.

Manage any life-threatening injuries and ensure hemodynamic stability. The goal is to identify the bleeding source. Apply an anesthetic and vasoconstrictor. Evacuate any remaining clot from the nasal airway and inspect the mucosa. Cauterize the vessel with silver nitrate if the bleeding source is identified. Apply a thin piece of oxidized cellulose (i.e., Surgicel) or Gelfoam that has been impregnated with an antibiotic ointment over the site to help stabilize the clot. Place an anterior nasal pack if chemical cauterization fails to control the bleeding. Expandable nasal sponges/tampons are readily available and are relatively easy to place. Trim the sponge/tampon to an appropriate length prior to insertion. Consult an Otolaryngologist if the bleeding cannot be controlled, if there are concerns that the bleeding source is from a posterior location, or for any postsurgical patient.

The underlying cause should be identified and treated.[3,51] Antibiotic ointment and nasal saline should be used to minimize crusting and to hydrate the mucosa for patients with epistaxis secondary to digital manipulation. Antibiotic therapy is indicated in cases of acute sinusitis and adenoiditis. Coagulopathy must be considered when a patient presents with recurrent epistaxis. Up to 30% of children with recurrent epistaxis have a coagulopathy, with Von Willebrand's disease the most common disorder identified.[52] Laboratory testing should include a complete blood count, prothrombin time, partial thromboplastin time, and international normalized ratio in patients whose history suggests heavy bleeding, recurrent bleeding, or a personal or family history of bleeding abnormalities.[20]

AFTERCARE

The postprocedural care of patients with anterior epistaxis is just as important as the initial control of bleeding. The recommendation to place patients on prophylactic antibiotics after nasal packing remains highly controversial and is not routinely indicated.[53-57] The historic rationale for prophylactic antibiotics is due to a perceived risk of developing sinusitis and toxic shock syndrome. No randomized controlled trials exist evaluating the utility of prophylactic antibiotics after epistaxis. One study demonstrated no changes in microbiological flora before and after anterior nasal packing.[55] Another observational study found no association between the use of prophylactic antibiotics after anterior nasal packing and infectious complications (e.g., sinusitis or toxic shock syndrome).[56] **The authors do not recommend routine prophylactic antibiotics in most cases of anterior packing**. Consider antibiotic prophylaxis in cases of posterior packing and preference of the consulting Otolaryngologist. Routine use of antibiotics may select for resistant organisms.[59] Antibiotics are recommended for immunocompromised patients with nasal packing. Choices for prophylactic antibiotic therapy include amoxicillin-clavulanate, cephalexin, or trimethoprim-sulfamethoxazole to cover *Staphylococcus aureus, Streptococcus pneumoniae, Haemophilus influenzae,* and *Moraxella catarrhalis.*

Patients with significant epistaxis should follow up with an Otolaryngologist within 24 to 48 hours. Malignancies or other serious etiologies of epistaxis remain rare. Patients presenting with epistaxis should have follow-up care with an Otolaryngologist for a thorough nasopharyngeal examination. Remove the packing in 48 to 72 hours if the patient experienced only minimal blood loss and is hemodynamically stable.

Inform the patient that the anterior pack is uncomfortable. Acetaminophen will typically provide adequate analgesia. **Avoid aspirin-containing compounds and nonsteroidal anti-inflammatory drugs that can contribute to further bleeding.** Opiates should also be used with caution as they can cause nausea, sedation, and vomiting. Patients on warfarin may generally continue their current regimen. Active reversal of anticoagulation is generally not needed provided their international normalized ratio is not supratherapeutic. **Instruct the patient to apply saline nasal spray to the packing or each nostril three or four times per hour while awake.** The use of a humidifier at home will aid in preventing drying of the packing or the nasal mucosa. **The patient must avoid nose picking and nose blowing.** Instruct the patient on the proper technique to apply pressure on the nose if bleeding restarts. Such pressure should be maintained continuously for 20 minutes. Instruct the patient to return to the Emergency Department if bleeding persists. The patient should also return for increased nasal pain, fever, or any symptoms related to blood loss (e.g., chest pain, dyspnea on exertion, dizziness, lightheadedness, shortness of breath, or syncope).

Patients with unstable vital signs, uncontrollable bleeding, posterior packing, or serious concomitant medical problems will require hospitalization. Patients with posterior nasal packs require consultation with an Otolaryngologist and admission to a telemetry or intensive care unit. They must be monitored for respiratory distress, hypoxia, hypotension, anemia, and cardiac sequelae. It has been estimated that 40% of patients with posterior nasal packing eventually require intubation.[58] The nasal ala and columella must be evaluated continually to prevent pressure necrosis. Consider the use of antibiotics in posterior packing.[59]

Patients with a posterior nasal pack may experience hypoxemia and hypercarbia. The etiology of this phenomenon, called the nasopulmonary reflex, is unknown. The PaO_2 may decrease 7 to 15 mmHg and the $PaCO_2$ may increase 7 to 15 mmHg. The nasopulmonary reflex is more worrisome in patients with underlying lung disease or comorbid conditions.

The association between hypertension and epistaxis remains complicated as patients with epistaxis frequently present with an elevated blood pressure.[3] Epistaxis also appears to be more common in hypertensive patients.[60] Hypertension is rarely the direct cause of epistaxis and more commonly the associated anxiety with epistaxis results in an abrupt increase in blood pressure.[61,62] Therapy should initially focus on control of the hemorrhage, reducing discomfort, and reducing anxiety rather than reduction of the blood pressure.

Local vasoconstriction, cautery, and/or packing will accomplish resolution of epistaxis for most patients. Administration of appropriate blood products (i.e., fresh frozen plasma, platelets, or recombinant factor VIIa) may be useful in patients with inherited or acquired bleeding disorders or platelet dysfunction. A risk-benefit analysis and discussion should occur on a case-by-case basis. A transfusion of packed red blood cells must also be considered in patients with significant bleeding, anemia, and ongoing blood loss.

COMPLICATIONS

The complications associated with epistaxis are variable, wide ranging, and estimated to be from 2% to 69%.[47] Epistaxis may be complicated by hemorrhage, hypoxemia, hypovolemia, circulatory collapse, and airway compromise. Complications resulting from the treatment of epistaxis include nasal septal perforation, sinusitis, otitis media, toxic shock syndrome, aspiration, alar necrosis, and hypoxia from intrapulmonary shunting due to the nasopulmonary reflex.[63] A single-center retrospective study showed recurrence of epistaxis and return to the Emergency Department occurred in approximately 26% of patients.[64] Silver nitrate cauterization had the highest success rate in cessation of epistaxis at 80% while recurrence occurred the most often in those treated with nasal clips at 59%, Merocel packing at 26%, and petroleum gauze packing at 42%.[64]

Most complications can be prevented by using proper technique, providing supplemental oxygen when not contraindicated, arranging appropriate hospitalization if indicated, obtaining an Otolaryngology consultation, and arranging for adequate follow-up.[65] Topical lidocaine in the nasal cavity can cause complications.[66] Always calculate the dose used and assume that the entire dose will be absorbed. The nasal mucosa is very absorbant and the lack of a vasoconstrictor can result in increased absorption.

SUMMARY

Epistaxis is a common condition that affects 10% to 13% of the general population. The key to successful management includes a prompt and thorough evaluation of the patient, an accurate diagnosis of the problem, and rapid control of the bleeding. There has been a significant expansion in therapeutic options (e.g., TXA) to treat epistaxis. Almost all cases of epistaxis stem from an anterior source and can be controlled with either direct pressure, chemical cautery, or nasal packing. Posterior bleeding requires the use of a posterior pack and anterior packing. There is insufficient evidence supporting the routine use of prophylactic antibiotics in patients with anterior packing. Consider prophylactic antibiotics in all patients with nasal packing to prevent sinusitis and toxic shock syndrome. Follow-up is required in 24 to 72 hours with an Otolaryngologist or in the Emergency Department to remove the nasal packing.

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206 Laryngoscopy

Steven Charous

INTRODUCTION

Evaluation of the larynx can be crucial in the diagnosis and management of common and life-threatening disorders. The approach to the patient with laryngeal dysfunction begins with obtaining a complete history. Symptoms may be related to any of the three primary functions of the larynx. These are protection of the lower airway from aspiration, a conduit of the airway, and phonation. Symptoms may include aspiration, cough, dysphagia, odynophagia, dyspnea, or hoarseness. Otalgia may be a referred symptom from the larynx and transmitted by a branch of the vagus nerve. Information regarding patient age, onset, duration, severity, and progressive nature of the process is necessary. Determine the patient's past medical history including prior intubations, neck trauma, reflux esophagitis, similar previous episodes, and other systemic diseases. The social history, including smoking and alcohol usage, needs to be investigated. Medications, allergies, and over-the-counter drugs should be reviewed.

Perform a physical examination, including a complete head and neck examination, once the history has been obtained.[1] Listen for stridor and watch for accessory muscle breathing. Consciously and critically evaluate the patient's voice to hear breathiness, clarity, and volume. Inspect the ears, nose, oral cavity, oropharynx, and nasopharynx. Careful palpation of the neck is extremely important. Note any lymphadenopathy and neck masses. This must include their size, location, tenderness, and mobility. Palpate the larynx and note any crepitus. The lack of crepitus on lateral movement of the larynx over the vertebral bodies can be indicative of a laryngeal or hypopharyngeal mass. Note any movement with swallowing and asymmetry. This can help in determining the extent of a disease process.

Visualize the larynx after performing a complete history and physical examination with the exception of true airway emergencies. This allows the Emergency Physician to examine the larynx in context to the patient's symptoms and other physical findings. It also allows a rapport to develop between the patient and Emergency Physician prior to undergoing a mildly invasive procedure.

There are four methods of performing indirect laryngoscopy: mirror laryngoscopy, nasal flexible fiberoptic laryngoscopy, oral flexible fiberoptic laryngoscopy, and rigid telescopic laryngoscopy. The following is a complete description of the procedure involved in performing each of these techniques. An excellent pictorial source for viewing normal and pathologic conditions of the larynx may be found in Bruce Benjamin's publications.[2,3] **Table 206-1** reviews the advantages and disadvantages of each procedure. Each method allows visualization of the larynx with different degrees of distortion (**Figure 206-1**).

ANATOMY AND PATHOPHYSIOLOGY

The larynx occupies the central neck and is located within the hypopharynx (**Figures 206-2 and 206-3**).[4-6] Lateral to the larynx are the pyriform sinuses and the pharyngeal recesses that are the primary route for food to pass into the esophagus. The basic framework of the larynx consists of the thyroid cartilage, cricoid cartilage, epiglottic cartilage, arytenoid cartilage, and the hyoid bone. The shield-like thyroid cartilage supports the soft tissues of the larynx. It is connected to the hyoid bone via the thyrohyoid membrane and is attached to the cricoid cartilage via the cricothyroid membrane and at the cricothyroid joint. **The signet ring–shaped cricoid cartilage is the only complete cartilaginous ring in the larynx.** On top of its posterior portion sits the paired arytenoid cartilages. The arytenoid cartilages are somewhat shaped like an inverted "T." Each has a body, a muscular process, and a vocal process. The aryepiglottic folds connect the epiglottis to the top portion of the arytenoid body. The vocal ligament attaches the vocal process to the thyroid cartilage. The cricoarytenoid muscles attach to the muscular process. The epiglottic cartilage is leaf-shaped and forms the anterior wall of the laryngeal entranceway. Its main portion projects posterior to the tongue base. It folds downward over the larynx during swallowing to aid in protecting the laryngeal opening from aspiration.

The muscles associated with the larynx may be divided into extrinsic muscles and intrinsic muscles. The extrinsic muscles move the larynx as a unit and can be further subdivided into those muscles that elevate the larynx (i.e., the stylohyoid, digastric, geniohyoid, and stylopharyngeus) and those that depress the larynx (i.e., the omohyoid, sternohyoid, and sternothyroid). The intrinsic muscles are involved with vocal cord mobility and all cause adduction with the exception of the cricoarytenoid muscle that causes abduction. Innervation to the intrinsic laryngeal muscles is via the recurrent laryngeal nerve, a branch of the vagus nerve (cranial nerve X). The cricothyroid muscle is the only muscle innervated by the external branch of the superior laryngeal nerve, a branch of the vagus

TABLE 206-1 Summary of the Advantages and Disadvantages of the Different Techniques Used to Perform Indirect Laryngoscopy

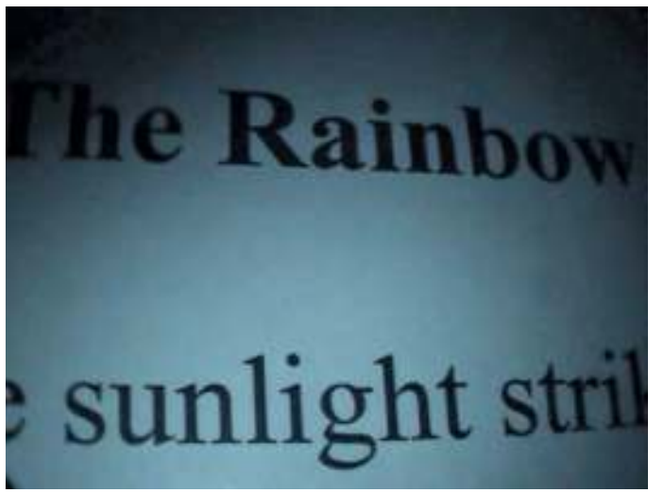
	Hand-held mirror	Per-oral flexible endoscopy	Per-oral rigid endoscopy	Nasal flexible endoscopy
Gag reflex	Moderate	Moderate	Moderate	Minimal
Visual clarity	Good	Good–distorted	Superior	Good–distorted
Anesthetic	Occasionally	Yes	Occasionally	Yes
Observe larynx during speech	No	No	No	Optional
Patient cooperation	Necessary	Necessary	Necessary	Not necessary
Approximate equipment cost	Minimal	$4500	$4500	$4500
Documentation possible?	No	Yes	Yes	Yes

nerve. All of the laryngeal muscles and cartilages are covered with respiratory epithelium.

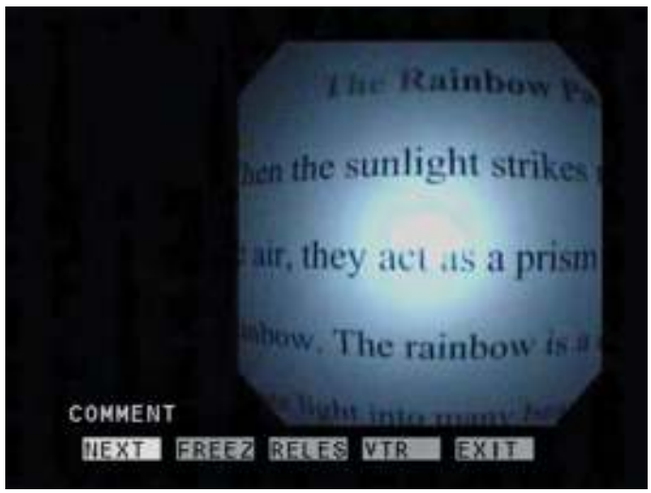
Just superior to the vocal cords is a recess called the laryngeal ventricle. Just superior to the ventricle are the false vocal folds. These are rounded protrusions rich in mucus-secreting glands. The supraglottic larynx is defined as that portion of the larynx extending from the tip of the epiglottis to the laryngeal ventricle. The glottic larynx contains the true vocal cords and extends approximately 5 to 7 mm inferiorly. The subglottis extends from the inferior glottis to the inferior edge of the cricoid cartilage.

The primary function of the larynx is to protect the airway from the aspiration of food particles. A complex reflex arc, with the glossopharyngeal nerve (cranial nerve IX) mediating the sensory arm and the vagus nerve (cranial nerve X) mediating the motor arm, occurs with swallowing. With each swallow the larynx elevates, the aryepiglottic folds squeeze medially, the epiglottis folds posteriorly over the larynx, and the true and false vocal folds close tightly. These actions allow the food bolus to pass around the larynx, into the pyriform sinuses, and subsequently into the esophagus. Any alteration or disturbance in the reflex arc may predispose a patient to aspiration.

Phonation occurs with adduction of the vocal cords as air passes from the trachea through the vocal cords. The mucosa overlying the muscles of the vocal cords undulates and the two vibrating vocal cords produce sound. Anything that alters the mucosal wave of the vocal cords, impairs adduction, or changes the configuration of vocal cord alignment will result in decreased phonatory



A



B

C

FIGURE 206-1. Visualization through the endoscopes. ***A.*** Endoscopic view through the 90° rigid telescope. Note the magnification, clarity, and breadth of view. ***B.**** Endoscopic view through the "chip-in-tip" scope. Note the larger field of vision with minimal distortion. ***C.*** Endoscopic view through flexible fiberoptic scope. Note the distortion and limited view as compared to the view through the rigid scope.

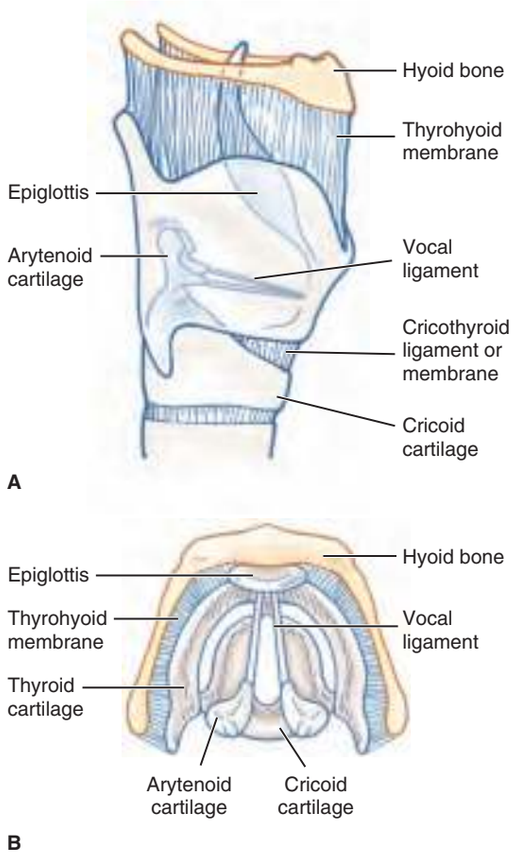


FIGURE 206-2. The laryngeal framework. ***A.*** Lateral view. ***B.*** Superior view.

performance. Note that mucosal wave abnormalities can only be observed with videostroboscopy of the larynx. Many things can change a person's voice. This includes but is not limited to inflammation, thick mucus, polyps, granulomas, vocal cord paralysis, and tumors. Be careful and define hoarseness as a change in the patient's vocal quality. What may be normal for one patient may not be for another.

The larynx is crucial in respiratory activity. Inspiration signals the recurrent laryngeal nerve to stimulate vocal cord abduction. Impairment in abduction, unilaterally or bilaterally, can lead to respiratory compromise. Tumors, polyps, or edema located in the supraglottis, glottis, or subglottis can lead to airway compromise.

INDICATIONS

Have a very low threshold for examining the larynx. The diagnostic value of laryngoscopy greatly outweighs the minimal discomfort associated with the quick procedure. Any patient presenting with an allergic reaction, angioedema, acute hoarseness or voice changes, symptoms of aspiration, shortness of breath, a foreign body sensation, hemoptysis, stridor, exposure to ingested caustic agents, exposure to hot fumes or gasses, exposure to caustic fumes or gasses, or any other symptom that may be related to the larynx should have laryngoscopy performed as part of a complete physical examination including predicting a difficult airway.[1-10]

CONTRAINDICATIONS

There are no absolute contraindications for laryngoscopy. Patients with severe respiratory compromise, such as a child with suspected epiglottitis, should have laryngoscopy performed in the Operating Room (or in a controlled area) with an Anesthesiologist, intubation equipment, and tracheotomy instrumentation ready for use. **Performing laryngoscopy in the respiratory distressed patient can lead to increased distress and ventilatory collapse if laryngospasm ensues. Experienced personnel should perform laryngoscopy in a patient with a high-grade airway obstruction with caution and preparedness for intubation, but the exam should most likely still be performed as the information gained is crucial for planning patient care.** In general, the patient must be able to follow instructions and cooperate with the examination.

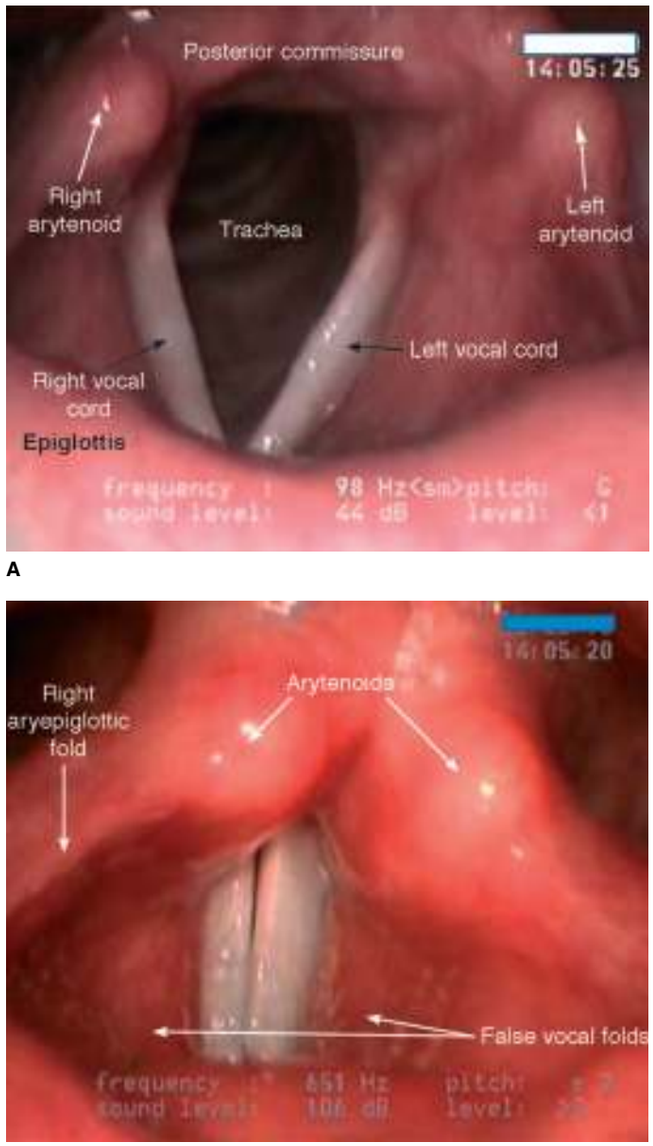


FIGURE 206-3. Endoscopic view of the laryngeal anatomy. ***A.*** Vocal cords open. ***B.*** Vocal cords closed.

EQUIPMENT

ANESTHESIA AND VASOCONSTRICTION

- 4% lidocaine (preferred for nasal anesthesia)
- 10% lidocaine spray

- 20% benzocaine spray
- 2% tetracaine spray
- 4% cocaine
- 3% ephedrine, Neo-Synephrine spray, or oxymetazoline spray
- Cotton pledgets

SCOPES

- #4 or #5 dental mirror, with or without magnification
- 3 to 5 mm diameter flexible fiberoptic laryngoscope; 3 mm is better for children
- 90° rigid laryngoscope
- Video laryngoscope (Chapter 20, optional)

LIGHT SOURCES

- Headlight for mirror examinations
- 125 to 250 watt halogen or xenon light sources for fiberoptic laryngoscopes

MISCELLANEOUS

- Alcohol lamp, heated beads, or glass of warm water for mirror examination
- 4×4 gauze squares
- Water-soluble lubricant
- Antifog solution
- Video camera and equipment for teaching and photodocumentation (optional)

Set up all of the required equipment on a bedside procedure table (**Figure 206-4**). Prepare several different endoscopes, if available. If one fails, or is inadequate, another will be immediately available. The availability of multiple endoscopes allows the Emergency Physician to choose the scope and technique of their choice.

The flexible fiberoptic scope is composed of fiberoptic strands and lenses along its length. Do not manipulate or bend it into acute angles. Do not place objects onto the scope. Do not use abrasive materials (i.e., gauze, paper towels) to wipe the lens at the end of the fiberoptic cord.

FIGURE 206-4. Basic equipment required for examination of the larynx in the ambulatory setting. Included are the rigid 90° telescope, dental mirror, flexible fiberoptic scope, gauze, oral anesthetic, antifog solution, and a light source. Not included is topical spray for nasal decongestion and anesthesia.

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. **Patient reassurance and relaxation are of the utmost importance in obtaining excellent visualization of the larynx.** This can be achieved by reviewing what can be expected from the patient's perspective, by reassuring them of the minimal discomfort, and by reassuring them of the short duration of the procedure. Topical anesthesia to the nasal or oral mucosa can induce the sensation of not being able to breathe or swallow because of the lack of sensory input. Informing the patient of this sensation prior to its use is extremely helpful in preventing a panicked patient. Alternatively, using only a vasoconstrictive agent and not a topical anesthetic may be more appropriate and tolerated for transnasal endoscopies as the procedure itself is usually very well tolerated with minimal discomfort.

Patient positioning is crucial in obtaining laryngeal visualization in any per-oral technique. Place the patient sitting upright in a multipositional procedure chair or on a gurney with their legs together. Instruct the patient to lean slightly forward and to draw their chin forward. This aligns the larynx and the oropharynx in a vertical plane to allow visualization of the anterior portion of the larynx. Raise or lower the multipositional chair, or the gurney, so that the patient's mouth is at the examiner's eye level.

Patient cooperation and positioning are not crucial for flexible fiberoptic examination of the larynx performed through the nose. Place the patient, optimally, sitting upright with their head against a headrest. The headrest will prevent the common occurrence of the patient's head backing away during the examination. The examination with the patient in the supine position is technically more challenging as gravity pushes the tongue posteriorly and the scope falls against the posterior pharyngeal wall, both of which make visualization of the anteriorly placed larynx more difficult. However, in most instances, it can still be performed without significant problems.

The use of good lighting cannot be overemphasized when performing a mirror examination. Apply a headlamp if one is available. An alternative is an overhead adjustable light source. Position the light so that it is aimed in the patient's mouth. The overhead light often hits the examiner in the head, casts shadows, and is too bright for the patient's eyes. It is also difficult to properly position as both of the examiner's hands must be used for the procedure.

TECHNIQUES

A systematic evaluation of the larynx and hypopharynx is necessary no matter what technique is used. Assess all of the airway structures including the base of the tongue, vallecula, epiglottis, aryepiglottic folds, true and false vocal cords, arytenoids, posterior pharyngeal wall, and pyriform sinuses. Visualize adduction and abduction of the vocal cords during phonation and inspiration. Visualize, if possible, the subglottis through the vocal folds looking for a narrowing or stenosis of the airway in the stridorous or respiratory compromised patient without other laryngeal findings. Use care to minimize contact with the laryngeal mucosa with whatever device is used to avoiding excessive gagging and allowing a comfortable and thorough examination.

THE MIRROR EXAMINATION

Determine whether the patient has a significant gag reflex. If so, apply topical anesthesia. Spray a topical local anesthetic agent (e.g., benzocaine, lidocaine, or tetracaine) onto the patient's palate, tonsillar pillars, posterior pharyngeal wall, and base of the tongue. The application of a topical anesthetic agent is optional if the patient

FIGURE 206-5. Proper positioning for an indirect mirror examination of the larynx. Both of the examiner's hands are braced against the patient for stability. Some patients require elevation of the upper lip with the examiner's left, or nondominant, middle finger.

does not have a significant gag reflex. Instruct the patient to keep their eyes open and focus on a distant object to diminish the gag reflex. Practicing the entire procedure once with the patient, without inserting the mirror, is often a more reassuring and efficient manner of performing indirect laryngoscopy.

Instruct the patient to protrude their tongue. Grasp the tongue firmly between the nondominant thumb and index finger with a neatly folded gauze square (**Figure 206-5**). **Do not apply excessive traction on the tongue.** This is counterproductive as it elevates the tongue and makes the patient uncomfortable. Place the nondominant middle finger against the upper teeth as a brace. It may also be used to elevate the upper lip if needed. Instruct the patient to breathe through their mouth in a slow "panting-like" manner and to try to relax their tongue. This maneuver diminishes the gag reflex, elevates the palate, and lowers the tongue giving better access and visualization of the oropharynx.

Warm the mirror over an alcohol lamp, in heated beads, or in a cup of warm water to prevent fogging during the examination. Test the mirror back, if any type of heat is used, on the examiner's wrist for excessive heat that can injure the patient. An alternative is to use a mirror that has antifog solution placed on it to prevent fogging during the examination. Grasp the mirror with the dominant hand midway down the shaft like a pen (**Figure 206-5**). Brace the dominant fifth finger against the patient's face.

Introduce the mirror into the oral cavity with the glass surface parallel to the tongue (**Figure 206-5**). Instruct the patient to say a high-pitched "e-e-e" and hold it for 5 seconds just before the mirror touches and elevates the uvula and soft palate. The high-pitched "e-e-e" tilts the epiglottis forward and brings the vocal cords into view and apposition. Inform the patient that the "e-e-e" may sound like "ahhhhhh" while their tongue is held. This is helpful to them as they try to cooperate fully with the instructions. The Emergency Physician should simultaneously demonstrate the high-pitched sound as patients invariably will phonate in too low of a pitch for too short of a time period. Prevent gagging by asking the patient to phonate before the mirror actually touches the soft palate, avoiding touching the base of the tongue, and avoid touching the anterior tonsillar pillars.

Focus the headlight (or overhead light), as the patient phonates, on the mirror. Gently and slightly maneuver the mirror until the larynx is visualized. **Remember the orientation through the mirror. Left and right are the same but anterior and posterior have reversed orientation.** Perform a quick and systematic evaluation of the larynx and hypopharynx. Several reinsertions of the mirror are often required to obtain a complete examination.

PER-ORAL FLEXIBLE FIBEROPTIC LARYNGOSCOPY

The examiner will often have a more leisurely view of the larynx with this technique as compared to the mirror exam. Patients usually gag less and can tolerate this examination for longer periods of time.

Position the patient the same as for the mirror examination. All patients undergoing this procedure, in contrast to the mirror examination, must be topically anesthetized with an aerosolized local anesthetic agent as described above. Connect the light source to the fiberoptic scope and turn it on. Turn off the overhead room lights, if possible, to provide better contrast.

Instruct the patient to protrude their tongue and to grasp it with a gauze square (**Figure 206-6**). Hold the eyepiece of the scope in the dominant hand. Use the dominant thumb to manipulate the tip controller. Grasp and hold the middle portion of the fiberoptic scope with the thumb and index finger of the nondominant hand. Brace the remaining fingers of the nondominant hand against the patient's face (**Figure 206-6**). Instruct the patient to breathe slowly through their mouth in a "panting-like" manner.

Advance the scope with both hands until the tip is situated within the middle of the mouth and over the base of the tongue. Use the hand control to direct the tip of the scope downward. Look through the scope and visualize the base of the tongue, the vallecula, and the tip of the epiglottis. Direct the scope posteriorly over the epiglottis. The larynx and hypopharynx will come into view (**Figure 206-3**). Adjust the eyepiece, if necessary, to focus the scope. **Avoid touching the epiglottis as this may induce gagging.** Entry into the laryngeal vestibule will allow a more detailed examination but may also induce laryngospasm if the vocal cords are touched. Perform a systematic examination of the laryngeal and hypopharyngeal anatomy. Instruct the patient to say "e-e-e" while viewing abduction and adduction of the vocal cords.

PER-ORAL RIGID FIBEROPTIC LARYNGOSCOPY

Position the patient the same as for the mirror examination. All patients undergoing this procedure, in contrast to the mirror examination, must be topically anesthetized with an aerosolized local anesthetic agent as described above. Connect the light source to the fiberoptic scope and turn it on. Turn off the overhead room lights, if possible, or dim them to provide better contrast. Apply antifog solution onto the laryngoscope lens.

Instruct the patient to protrude their tongue. Either the patient or the examiner may grasp the patient's tongue with gauze as it

FIGURE 206-6. Positioning for an oral flexible fiberoptic examination of the larynx. The patient grasps their tongue with a gauze square. The examiner's left hand is braced against the patient's face.

FIGURE 206-7. Positioning for a rigid telescopic examination of the larynx. Note that the telescope is stabilized on the left thumb and the left fifth finger braces against the patient's face.

protrudes (**Figure 206-7**). Hold the scope with both hands while stabilizing the nondominant hand against the patient's face if the patient holds their own tongue. Hold the scope with the dominant index finger and thumb if the examiner is grasping the patient's tongue (**Figure 206-7**). Stabilize the scope with the dominant fifth finger against the patient's face. Instruct the patient to breathe slowly through their mouth in a "panting-like" manner.

Insert the laryngoscope into the center of the mouth. Advance it straight backward and over the tongue. Stop advancing the scope when the circumvallate papillae are reached. Instruct the patient to phonate as described above. Advance the scope until the larynx is visualized (**Figures 206-3 and 206-8**). Tilt and gently rotate the scope to visualize the entire larynx and hypopharynx during phonation and quiet breathing.

FLEXIBLE NASOPHARYNGOSCOPY OR NASOLARYNGOSCOPY

A relatively new technology for laryngeal visualization is the "chip-in-tip" laryngoscopes. Rather than fiberoptic cables, these flexible scopes have the video and lens apparatus at the tip of the scope and transmit the data to a processor. The processor converts the data into video images onto a screen. This scope produces a picture that is significantly brighter and less distorted (**Figure 206-1*B***). It is an excellent tool for teaching and in children.[7] However, the cost for a complete digital unit is three to four times the cost of a traditional fiberoptic laryngoscope.

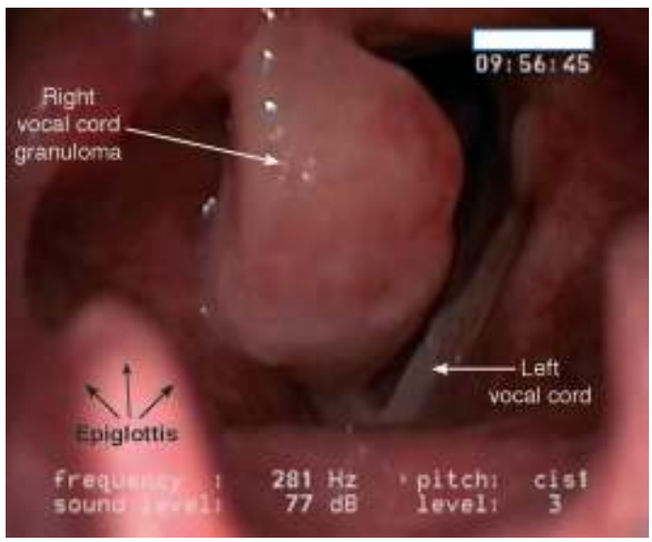


FIGURE 206-8. Endoscopic view of the larynx through a rigid 90° telescope.

Patient positioning is not crucial with this technique. Anesthetize the nasal passage if deemed necessary so that the procedure is best tolerated. Visualize both nasal cavities with a nasal speculum to determine which nasal passageway will be easier to pass the scope through. Decongest the nasal mucosa with aerosolized oxymetazoline, 3% ephedrine, or cocaine. Instruct the patient to sniff in deeply after the spray is applied. Allow 3 to 5 minutes to pass for the vasoconstriction to occur. Apply a topical anesthetic spray to the nasal passageway. Spraying achieves excellent vasoconstriction and anesthesia as the agents diffuse through the entire nasal cavity and pharynx. Alternatively, place cocaine or lidocaine with oxymetazoline-soaked pledgets into the nasal cavity inferior and superior to the inferior turbinate for 10 minutes to achieve excellent anesthesia and vasoconstriction.

Connect the light source to the fiberoptic scope and turn it on. Turn off the overhead room lights, if possible, to provide better contrast. Apply water-soluble lubricant onto the fiberoptic cord. **Do not get the lubricant on the lens or it will blur visualization.** Hold the eyepiece of the scope in the dominant hand (**Figure 206-9**). Use the dominant thumb to manipulate the tip controller. Grasp and hold the middle portion of the fiberoptic scope with the thumb and index finger of the nondominant hand. Brace the remaining fingers of the nondominant hand against the patient's cheek (**Figure 206-9**).

Insert the tip of the scope into the nose. Check both nasal airways with the scope minimally advanced in the nostril to see which side has a more patent airway and will thus allow an easier exam. Advance the scope in the easier nostril with both hands and directed either along the floor of the nose or along the superomedial aspect of the inferior turbinate depending upon the nasal anatomy (**Figure 206-10**). Instruct the patient to breathe through their nose as the nasopharynx is encountered. This lowers the soft palate and opens the nasopharynx. Direct the tip of the endoscope downward and advance it past the oropharynx. Slide the tip of the scope behind the epiglottis, along the posterior pharyngeal wall, and into the hypopharynx and larynx. **Avoid touching the epiglottis to prevent gagging and the vocal cords to prevent laryngospasm.** Almost all patients can tolerate this procedure. Gagging is unusual. Apply a topical anesthetic agent to the oral cavity and oropharynx if the patient gags. Perform a leisurely and thorough examination of the airway. **This is the only laryngoscopy technique in which normal speech can be observed.**

FIGURE 206-9. Positioning for a nasal flexible fiberoptic examination of the larynx. Note that the left, nondominant, hand is stabilized against the patient's face.

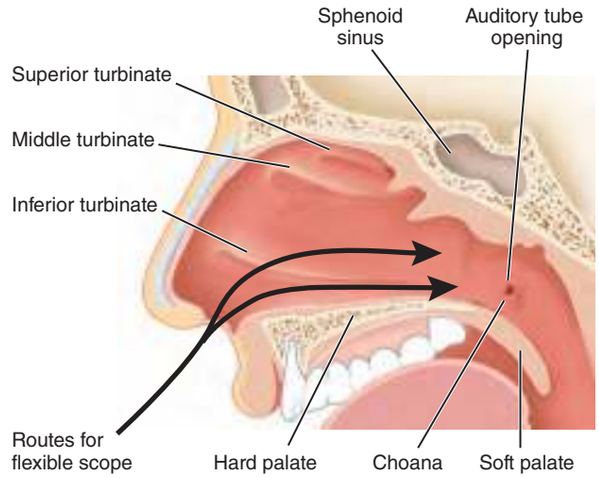


FIGURE 206-10. Anatomy of the lateral nasal wall and the two routes that the flexible scope can follow to gain access to the nasopharynx and subsequently the larynx.

ALTERNATIVE TECHNIQUES

The Emergency Physician must be looking through the rigid or flexible scope during the procedure. This in combination with holding, stabilizing, and advancing the scope makes fiberoptic laryngoscopy awkward if the Emergency Physician does not have much experience with this technique. An alternative, if available, is to connect the scope to a portable monitor (**Figure 206-11**). This makes the procedure less cumbersome and easier to perform. This type of setup is especially helpful in training situations (e.g., medical students, residents, and midlevel providers) as well as educating the patient and their family to the examination findings. A final alternative is to use a video laryngoscope (Chapter 20) to examine the airway.[8,9] The Emergency Physician is usually more familiar with the video laryngoscope than the flexible or the 90° scopes.

AFTERCARE

Inform the patient that the effects of the topical anesthetic persist an average of 30 to 45 minutes. Alert the patient that they may experience symptoms of aspiration, such as coughing or choking, when swallowing. Liquids are more likely to cause problems than solids.

FIGURE 206-11. Nasopharyngoscopy using an external monitor for visualization of endoscopic images.

Instruct the patient not to ingest any solid or liquid substances until the topical anesthetic agent wears off. Mild bleeding from the scope abrading the mucosa is usually minimal and self-limited. The patient will need reassurance as rarely is any treatment required.

Scopes require cleaning and disinfection between uses. Gently wipe any blood, lubricant, mucus, and other body fluids off the scope. Disinfect the scope per the manufacturer's recommendations and hospital guidelines.

COMPLICATIONS

Few complications arise from laryngoscopy. Epistaxis is possible with nasal endoscopy. It is uncommon when using vasoconstrictive agents and careful manipulation of the scope. Emesis is rare, even in the patient with an extremely sensitive gag reflex. Laryngospasm can be avoided as long as care is taken to avoid direct contact with the vocal cords. Although rare, the patient can have an adverse reaction to the local anesthetic agent or the decongestant. These should be evaluated and managed similar to any other allergic reaction.

Aspiration during or after the procedure is extremely rare. Aspiration can be minimized by performing laryngoscopy on patients with an empty stomach, although this is often impractical in the Emergency Department.

Other complications are minor and result from mechanical trauma. The tip of the scope can rub against and abrade the mucosa. This can result in mild irritation, rhinorrhea, and hemorrhage. Inappropriate technique or patient movement during the procedure can result in mucosal lacerations.

SUMMARY

Visualization of the larynx is a basic and often a crucial component in the physical examination of the patient. A variety of techniques have been described, all of which are adequate in evaluating the larynx and hypopharynx. Risks and complications are rare. Maintain a very low threshold when deciding whether or not to perform laryngoscopy. Patient education and preparation are as important, if not more important, than equipment and instrumentation in obtaining a thorough examination.

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207 Airway Foreign Body Removal

Guy Shochat, Jacqueline Nemer, David L. Walner, and Martin E. Anderson, Jr.

INTRODUCTION

The presence of an airway foreign body is a common cause of morbidity and mortality in children, especially those younger than 3 years of age. Children use their mouths to explore their surroundings. Over 70% of foreign body aspirations occur in children.[1,2] Boys are twice as likely as girls to experience an airway foreign body.[3] The incidence of airway foreign bodies has decreased over the past four decades due to public awareness campaigns. The mortality rate following foreign body aspiration is estimated to be 1% to 2%. This rate has remained fairly constant since the beginning of the 20th century. The mortality rate was approximately 24% prior to the advent of endoscopic techniques for airway foreign body removal.[4] In the year 2000, ingestion or aspiration of a foreign body resulted in more than 17,000 Emergency Department (ED) visits in the United States.[5] The death rate has remained at approximately 4000 deaths per year for the past two decades.[6-8] **The most serious and life-threatening scenario is complete airway obstruction. This usually occurs at the level of the larynx or trachea.**

Common airway foreign bodies are food items, coins, jewelry, and toys. Food objects have been associated with 41% of deaths and nonfood substances have been associated with 59% of reported deaths.[5] The leading causes of food foreign body airway obstruction are hard candy and nuts.[5,9] Other globular objects such as hot dogs, soft candies, chewing gum, and grapes are commonly aspirated food objects.[10] Coins and toys are commonly aspirated nonfood objects.[5,9,11-13] Rubber balloons, jewelry, and toys are the most commonly aspirated nonfood objects.[12,14] Vegetable matter accounts for 55% to 95% of airway foreign bodies, with nuts accounting for around 39% alone.[15]

Parents and caregivers should be educated and aware of the types of food and objects that pose a choking risk for children.[12,16-18] They should become familiar with the methods to reduce this risk. All parents and caregivers should learn the techniques to treat a choking child. Basic life support classes are often available free or at a minimal cost at hospitals, churches, and community centers.

The management of airway foreign bodies in the hospital requires an emergency specialist capable of managing acute airway compromise. Airway foreign bodies located in the larynx, trachea, or pulmonary tree require an Otolaryngologist, Pulmonologist, or other qualified specialist with experience in airway endoscopy and the availability of that equipment in the Operating Room or an Intensive Care Unit. Cases involving children require specialized expertise and equipment for pediatric airway endoscopy. **The ED must manage the airway foreign body if the airway becomes unstable or if the consultants are not immediately available.** The morbidity and mortality associated with airway foreign body retrieval have greatly declined due to the development of safe endoscopic techniques, rod-lens telescopes, and optical forceps.

The burden of proof lies with the Emergency Physician (EP) to diagnose an airway foreign body. Diagnosis of an airway foreign body can range from the obvious to very difficult and unexpected. This diagnosis may be challenging because information gained from the history, physical examination, and radiologic studies may not clearly confirm the presence of an airway foreign body.[11,19-23] An airway foreign body may not even be initially suspected in some cases. This is especially true in young children, patients with intoxication, or patients with cognitive dysfunction. Physical examination and radiographic studies may appear normal. In some cases, the only definitive test will be endoscopy to evaluate the entire laryngotracheobronchial tree. **Many airway foreign bodies are neither observed nor suspected.**

The EP must often rely on clinical suspicion guided by knowledge of the epidemiology of airway foreign bodies.[22,23] A choking crisis is the most sensitive clinical parameter of an airway foreign body. Coughing, choking, gagging, and throat clearing are reflexes designed to protect the airway.[20,24] These signs indicate that an airway obstruction is incomplete. Complete airway obstruction is evident by sudden respiratory distress followed by the inability to speak or cough.

The physical examination may be normal in up to 39% of patients. Radiographic studies may be normal in up to 20% of the patients. **The only definitive test when considering the diagnosis of an airway foreign body is endoscopy to evaluate the entire laryngotracheobronchial tree.** This chapter reviews airway foreign body removal with an emphasis on foreign bodies located within the trachea or lower airway. Refer to Chapter 224 for a discussion regarding upper airway foreign bodies.

ANATOMY AND PATHOPHYSIOLOGY

The airway is divided into three anatomic regions: the larynx, the trachea, and the bronchi. The laryngeal aditus is formed by the epiglottis anteriorly, the aryepiglottic folds laterally and posteriorly by the corniculate cartilages and upper border of the arytenoid muscle. The larynx extends from the level of the aditus to the lower border of the cricoid cartilage where it is continuous with the trachea.[9,11,19,25] The infant larynx is located higher in the neck than the adult larynx. The cricoid cartilage descends in the neck through childhood and is the narrowest part of the infant airway. The epiglottis is located with the tip often resting on the soft palate due to the superior position of the infant larynx.[19,20,26] The infant larynx is approximately one-third the size of the adult larynx. Laryngeal foreign bodies are most common in infants due to the small size of the inlet and subglottis. Refer to Chapter 9 for a more complete discussion regarding the differences between the child and adult larynx.

It is important to be familiar with the anatomy of the pediatric and adult airway.[19] The trachea begins at the lower border of the cricoid cartilage, extending downward from about the level of the sixth cervical vertebra in adults or the fourth cervical vertebra in infants. The trachea extends inferiorly to the level of the carina. The inferior end of the trachea is located at the level of the fifth thoracic vertebra or the sternal angle. The trachea is 4 cm long in a full-term newborn infant and 11 to 13 cm long in an adult. The diameter of the trachea is 3.6 mm in a newborn and 12 to 23 mm in an adult.[27]

The trachea divides into two mainstem bronchi.[27] The right mainstem bronchus is shorter, straighter, and larger in diameter than the left mainstem bronchus. **This explains why right mainstem foreign bodies are more common than left mainstem foreign bodies.** The mainstem bronchi divide into three lobar bronchi on the right and two on the left. The lobar bronchi divide into segmental bronchi. There are 10 segmental bronchi on the right and 8 on the left.[27]

The anatomic location of airway foreign bodies in pediatric patients varies by age.[9,20,24,25] Airway foreign bodies commonly become lodged in the larynx, the trachea, or the bronchi. Laryngeal foreign bodies account for 4% to 5% of airway foreign bodies.[24] Laryngeal foreign body has a high mortality rate due to complete airway obstruction.[28,29] Many survivors of transient airway obstruction suffer from hypoxic encephalopathy.

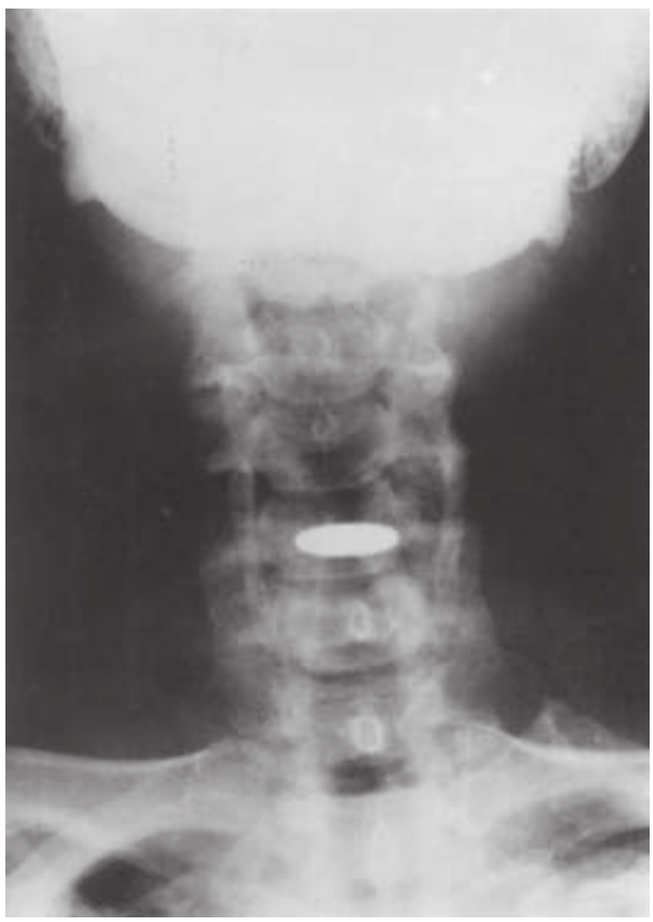
Most choking victims are able to generate a forceful cough to expel an airway foreign body. Some patients are unable to relieve the

obstruction themselves. The use of the Heimlich maneuver has further decreased mortality.[30] **It should be emphasized that the relief of an airway obstruction should only be attempted if signs of a complete airway obstruction are observed. The American Heart Association since 2005 no longer advocates blind finger sweeps as they may further impact foreign bodies into the larynx or esophagus.[31] Only perform oral cavity finger sweeps if a foreign body is seen within the oral cavity.[32] Immediate intervention is unnecessary and potentially dangerous if a patient is able to breathe, speak, or cough.[33]**

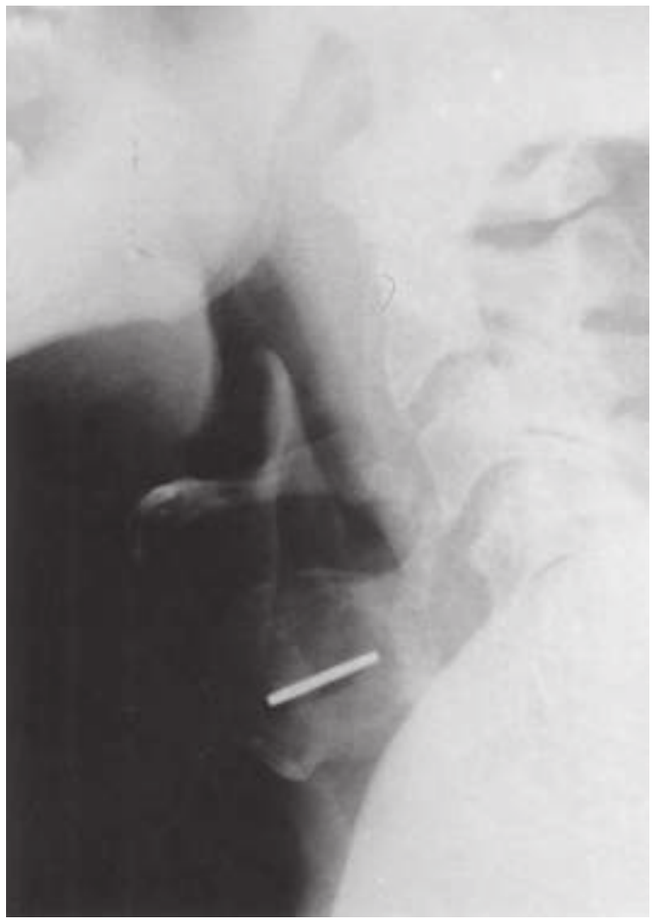
Laryngeal foreign bodies can present with only mild or moderate respiratory distress. They may be located, or wedged, between the laryngeal ventricles, the true vocal folds, or the immediate subglottis (**Figure 207-1*A***). Plain radiographs of the neck will detect radiopaque foreign bodies (**Figures 207-1*B* and 207-1*C***). Flexible awake fiberoptic laryngoscopy allows visualization of the larynx and supraglottic structures.

Tracheal foreign bodies account for 9% and bronchial foreign bodies account for 81% of airway foreign bodies.[34] The "classic" diagnostic

A



B

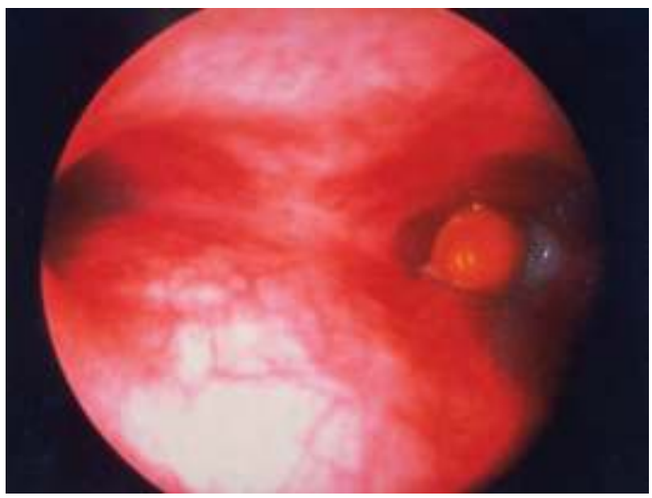


C

FIGURE 207-1. Laryngeal foreign body. ***A.*** Coin lodged within the laryngeal ventricles resulting in a partial airway obstruction. ***B.*** Anteroposterior neck radiograph. ***C.*** Lateral neck radiograph.

triad of a tracheobronchial foreign body consists of the sudden onset of paroxysmal coughing, wheezing, and unilateral diminished breath sounds on the affected side.[20,24] However, these symptoms may only be present in approximately half of cases. Approximately 33% of airway foreign bodies do not demonstrate any coughing, choking, or wheezing.[35] Tracheal foreign bodies may present with audible biphasic or expiratory stridor. Audible expiratory wheezing is more likely associated with a main bronchus obstruction. Tachypnea and cyanotic episodes may occur when larger obstructing objects are present. Small foreign bodies may travel distally to a secondary bronchus and produce the more subtle symptoms of mild wheezing, cough, pneumonia, or fever. No abnormalities are found in up to 39% of patients.[36] It is estimated that only 70% of patients with a foreign body aspiration seek treatment within the first week of the aspiration.[29,37] Long-standing airway foreign bodies are associated with a considerable increase in morbidity. Early diagnosis is the key to successful management and risk reduction.[1]

The most common airway foreign bodies are food items and toys. Peanuts account for nearly 39% of tracheobronchial foreign bodies (**Figure 207-2**).[38] Other common foreign bodies include plastic toys, pins, tacks, watermelon seeds, sunflower seeds, nails, screws, carrots, and popcorn. More than 80% of airway foreign bodies are radiolucent and can be difficult to diagnose.[20,39,40]



A

B

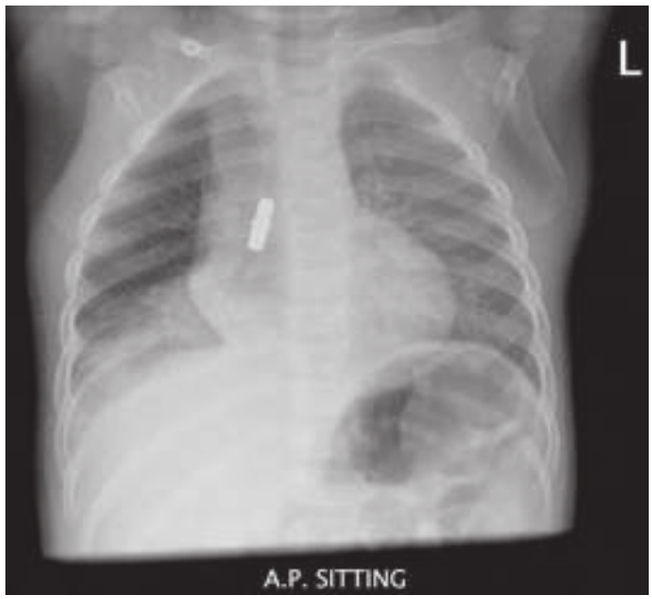
FIGURE 207-2. Endoscopic view of a peanut in the right mainstem bronchus. ***A.*** Endoscopic view from just superior to the carina. Note that both mainstem bronchi are visible. ***B.*** Close-up view of the foreign body.

FIGURE 207-3. Chest radiograph of a patient with a left mainstem bronchus foreign body and a check valve-type of obstruction. Marks delineate the trachea and the mainstem bronchi.

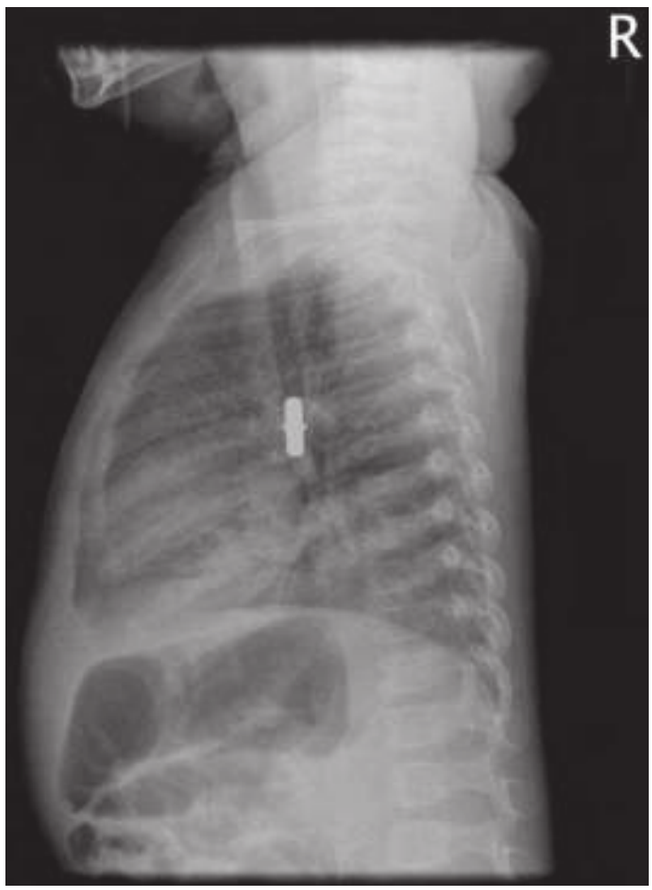
The most common radiologic findings are identified using both inspiratory and expiratory chest radiographs.[41] The acute airway foreign body can be in the form of a check valve with postobstructive hyperinflation and mediastinal shift to the contralateral side (**Figure 207-3**). Inspiratory and expiratory chest radiography may identify a ball valve phenomenon with atelectasis, collapse of the distal airways, and mediastinal shift to the ipsilateral side with chronic foreign bodies. Lateral decubitus films are often used as an adjunct imaging study as young children are not often able to cooperate for inspiratory and expiratory chest radiographs. The lateral decubitus radiograph uses the patient's body weight to promote expiratory excursion. The use of decubitus films is no better than the use of upright films.[42] The foreign body can be in the mainstem bronchus (**Figure 207-4**). Pneumonia can be present when the foreign body has been present for weeks or months. Chest radiographs can be normal in up to 20% of patients with tracheobronchial foreign bodies.[11,20,24,25]

INDICATIONS

The indications for airway endoscopy must take into account the patient's history, physical examination, radiologic findings, and the suspected location of the foreign body. An accurate history is of



A



B

FIGURE 207-4. Chest radiograph of a patient with a right mainstem bronchus foreign body. ***A.*** Posteroanterior view. ***B.*** Lateral view. (Courtesy of www.lifeinthefastlane.com.)

the utmost importance in the diagnosis of foreign body aspiration as the remainder of the assessment, physical examination, and radiographic studies can be deceptively unremarkable.[39,43] Many aspiration events are unwitnessed. The characteristic history consists of an incipient choking or gagging episode. Caretakers often describe subsequent coughing spells when the event was witnessed. Aspiration must be assumed if the patient was eating peanuts, seeds, or beans during the episode. All witnessed aspirations with nuts or nondissolvable food matter require endoscopy and removal if the foreign material is identified.

The EP must have a high index of suspicion for an airway foreign body based upon history, physical examination, and radiologic findings.[9,20] **However, the diagnosis is not always clear cut.[21] It is the responsibility of the EP to suspect an airway foreign body.** Consult with the appropriate specialist and strongly suggest endoscopy consisting of laryngoscopy and/or bronchoscopy. Endoscopy remains the gold standard to manage or rule out an airway foreign body.[24,39,43]

CONTRAINDICATIONS

The majority of patients presenting to the ED with airway foreign bodies are in stable condition. This allows time to adequately access the patient and formulate the best possible treatment plan. Infants and children with airway foreign bodies require an institution with the capability for comprehensive pediatric care. This includes a physician who is experienced with airway endoscopy and foreign body retrieval in children and a facility with pediatric endoscopic equipment, pediatric anesthesia capabilities, and pediatric intensive care capabilities. This often will require transfer to a specialized pediatric center. **Attempting removal of airway foreign bodies in a less-than-adequate environment can be catastrophic for the patient and is not advised.** This same philosophy applies to adult airway endoscopy and to having qualified personnel who are experienced in this area.

Follow basic life support protocols in the case of an acutely obstructed airway and prepare to manage the airway if needed.[44,45] A patient with a suspected laryngeal foreign body and a concern for impending airway obstruction should be closely observed and monitored while facilitating emergent endoscopy and control of the airway in the Operating Room. If urgent endoscopy is not available, direct laryngoscopy in the ED and removal of a visualized foreign body with a forceps (e.g., Magill, Boedeker, or Tylke) can be attempted. Options include plain radiographs to attempt to localize and assess radiopaque objects and fiberoptic or direct laryngoscopy by the EP if impending airway obstruction does not appear to be an issue and a laryngeal foreign body is suspected.[24,43] A cricothyrotomy or percutaneous transtracheal jet ventilation may be required in an unstable patient with a laryngeal foreign body if attempts at resuscitation and/or removal are unsuccessful. **These two procedures are unlikely to be beneficial if the foreign body is located in the distal trachea or bronchi.**

The EP may attempt a lifesaving temporizing measure to dislodge the total airway obstruction distally if urgent endoscopy is not available. This lifesaving temporizing measure is orotracheal intubation. The endotracheal tube can be advanced as deeply as it will go or a bougie can be placed through the endotracheal tube and advanced to force the foreign body deeper into a mainstem bronchus. A flexible fiberoptic scope can be used to position the endotracheal tube into the contralateral mainstem bronchi. This mainstem positioning can be helpful in the case of a contralateral mainstem bronchus obstruction. A final alternative is the application of extracorporeal membrane oxygenation (Chapter 73).

EQUIPMENT

ED EQUIPMENT

- Intubation equipment
- Percutaneous transtracheal jet ventilator

- Cricothyroidotomy kit
- Suction source, tubing, and catheter
- Magill, Boedeker, or Tylke forceps (**Figure 29-2**)
- Laryngoscope with a variety of blades or a video laryngoscope
- Topical anesthetic
- Atomizer for topical anesthetic
- Fiberoptic nasopharyngoscope

Atomizer devices are usually not available in the ED. Many devices are available commercially. These are disposable devices that are single patient use (**Figure 29-1 and Table 29-1**). A Popular device is the MAD line of mucosal atomizer devices (LMA North America, San Diego, CA). They make multiple devices for all topical needs.

An airway forceps is used to remove an airway foreign body. The Magill forceps is the standard used by most institutions (**Figure 29-2*A***). It is available in infant, child, and adult sizes. The forceps tips twin blades for gripping are offset with serrated tips. It is curved at an oblique angle between the handles and the blades to prevent the view of the patient's airway from being obscured. The ends of the blades are rounded to minimize trauma. The Boedeker forceps (Karl Storz Endoscopy, El Segundo, CA) is an alternative to the Magill forceps (**Figure 29-2*B***).[46] It is also curved but does not put the working end into the view of the intubator. The Tylke forceps (Rodinia LLC, Jupiter, FL) is a new device that is simple to use, safe, and an effective alternative to the Magill forceps (**Figure 29-2*C***). The twist and bend of the Tylke forceps prevents visual obstruction and provides improved access to the trachea. It is available in three sizes (i.e., adult, adolescent, and child).

OPERATING ROOM EQUIPMENT

The proper equipment must be selected based upon the patient's age, the patient's size, and the suspected composition of the foreign body. Laryngoscopes are selected to allow visualization of the larynx and the passage of a bronchoscope. Rigid ventilating bronchoscopes with fiberoptic telescopes provide optimal visualization. This allows direct access to the airway, excellent visualization, continuous administration of an anesthetic agent and oxygen, and a conduit for the introduction of instruments (e.g., forceps) to retrieve the foreign object. Multiple sizes of laryngoscopes and bronchoscopes are essential to have available in the Operating Room. Numerous extraction instruments of different sizes and shapes must be available and include smooth, toothed, cupped, angled, open mouth, and optical forceps. The optical forceps allow a magnified and direct view through the forceps improving visualization and ease of foreign body removal.

PATIENT PREPARATION

Timing of endoscopy and airway foreign body retrieval must be based upon each individual patient. **Do not waste time if impending airway obstruction exists.** Immediately notify and mobilize an Anesthesiologist, Otolaryngologist, and the Operating Room as this is an emergent situation. It is appropriate to wait for NPO (nothing by mouth) status to be present and the stomach to empty prior to proceeding to the Operating Room if the diagnosis of an airway foreign body is highly suspected and the patient is stable. This is considered a timely approach and may take up to 6 hours for children or 8 hours for adults. Waiting this time in the stable patient decreases the risk of aspiration and further compromising the situation. It is also appropriate to wait in a stable patient to assemble the appropriate and best nursing and anesthesia team to care for the patient. Using personnel who are unfamiliar with endoscopy can create a compromised and stressful situation.

TECHNIQUES

ED TECHNIQUES

The foreign body airway obstruction protocol based upon Pediatric Basic Life Support treats conscious infants younger than 1 year of age with four back blows while the infant is in a prone position on the rescuer's forearm, face down, and their head lower than their trunk.[37,47,48] This is combined with four rapid chest thrusts, if the obstruction persists, while the infant is supine with their head lower than their body.[32,44] Treat unconscious infants by opening the airway and attempting rescue breathing based on basic life support protocols.[32,44] Treat children older than 1 year of age and adults with the standard Heimlich maneuver. **Use gentle thrusts in smaller children to decrease the likelihood of injury to the abdominal organs.[44,49] Finger sweeps to remove a foreign body in the oral cavity should only be performed if the foreign body is directly visualized. Blind finger sweeps are not recommended as they can further impact the foreign body and obstruct the airway.[44,45]** Refer to Chapter 224 for more information on relief of choking and acute airway foreign body removal.

The Heimlich maneuver can be a lifesaving procedure but it is also associated with a wide range of serious complications. Reported Heimlich complications include solid organ injury to the liver, spleen, and pancreas; perforation of the esophagus or stomach; diaphragmatic rupture; fracture of sternum, ribs, or vertebra; hemothorax; pneumothorax; or a pulmonary contusion.[30,49-52] Any patient who is symptomatic following the Heimlich maneuver must be thoroughly evaluated and closely observed for potential complications.

It is always best not to manipulate the airway or attempt intubation in a stable patient with an airway foreign body and who is moving air and breathing. The airway is best controlled in the Operating Room at the time of the actual foreign body removal. Once the airway is manipulated a foreign body can become dislodged, turning a partial airway obstruction into a complete airway obstruction. If this occurs in the Operating Room, the bronchoscopy equipment is available for urgent use by the Endoscopist.

Attempt orotracheal intubation in the ED if the airway obstruction progresses rapidly and the patient cannot be ventilated. Proceed to direct or video laryngoscopy. Intubation can be used to force the foreign body into one mainstem bronchus and allow ventilation of the other lung. One-lung ventilation will keep the patient alive until the foreign body can be removed in the Operating Room. Position the laryngoscope to visualize the larynx. Grasp the foreign body with a forceps (e.g., Magill, Boedeker, or Tylke) and remove it if a foreign body is visualized. Intubate the patient if no foreign body is visualized. Insert and advance the endotracheal tube as far as it will advance distally if the foreign body is not visualized or unable to be grasped. Attempt to pass a smaller endotracheal tube if the original endotracheal tube will not pass. Withdraw and position the endotracheal tube with the tip above the carina to optimize ventilation. As an alternative, insert and position the endotracheal tube above the carina and then advance a bougie through the endotracheal tube in an attempt to move the foreign body distally.

Always be prepared to perform a cricothyrotomy or transtracheal jet ventilation. Transtracheal jet ventilation allows for short-term oxygenation, is temporary, and may allow time for safe transport to the Operating Room so that endoscopy and foreign body retrieval can be performed in a more controlled environment with appropriate equipment at hand. Refer to Chapters 18, 32, and 31 regarding the details of orotracheal intubation, cricothyroidotomy, and transtracheal jet ventilation, respectively.

Direct laryngoscopy and bronchoscopy in a child or adult with an airway foreign body is a dangerous situation. The procedure

may result in a partial airway obstruction becoming a complete airway obstruction. **Always have a cricothyroidotomy tray immediately available.** All equipment must be selected, assembled, and ready for use.

It is possible to remove foreign bodies located within the hypopharynx in the ED. Typical foreign bodies that may be removed include pieces of food and bones. **The patient must be stable and in no risk of airway compromise.** Obtain anteroposterior and lateral soft tissue radiographs of the neck to localize, if possible, the foreign body. There has been an increase in the use of computed tomography (CT) scans to attempt to identify potential fish or chicken bones that may be lodged in the pharynx, hypopharynx, or esophagus in the stable patient. The CT scan is superior to plain radiographs but the use needs to be weighed against the risk of radiation exposure. Perform a laryngoscopy (Chapter 206) or awake direct intubation to identify the foreign body and its location.

Place the patient in full monitoring (e.g., pulse oximeter, cardiac monitor, and noninvasive blood pressure cuff). Apply a topical anesthetic spray to the oropharynx and the base of the tongue. Alternatively or additionally, administer a nebulized local anesthetic (e.g., 5 mL of lidocaine 2% in a standard nebulizer unit). **Avoid Cetacaine spray due to concerns of methemoglobinemia from the benzocaine.** Administer a small dose of an intravenous sedative if required.

Place the patient supine. Slowly and gently insert the laryngoscope blade. An alternative to a traditional laryngoscope, if available, is a video laryngoscope. The video laryngoscope may provide a better field of view with less manipulation. **Do not immediately insert the laryngoscope blade all the way.** Stop frequently to lift the laryngoscope and look for the foreign body. This slow insertion and frequent looks will prevent the laryngoscope blade from pushing the foreign body further into the airway. Elevate the patient's tongue and jaw. Grasp the foreign body with a forceps (e.g., Magill, Boedeker, or Tylke) if it is visualized. Withdraw the forceps followed by the laryngoscope.

OPERATING ROOM TECHNIQUES

The procedure begins with the induction of general anesthesia. It cannot be overemphasized that anesthesia should only be administered by an Anesthesiologist who is competent and comfortable with the situation. Pediatric patients require an Anesthesiologist with pediatric airway experience. Full monitoring and mask induction allow the patient to maintain spontaneous respiration. Muscle relaxants are avoided as they can induce complete airway obstruction.

The Otolaryngologist begins the procedure. Place the patient supine with a shoulder roll to position the airway. Insert the laryngoscope into the larynx. Expose the larynx by elevating the laryngoscope. Topical anesthetic is applied to the larynx to avoid laryngospasm. The bronchoscope with a telescope is then passed under direct vision through the mouth and into the laryngeal introitus. Ventilation can continue via a port on the scope. The foreign body is visualized. Forceps are inserted through the scope and used to grasp the foreign body. Small objects can be removed directly through the scope whereas larger objects require simultaneously removing the bronchoscope along with the forceps and foreign body. The bronchoscope is passed again after the removal of the foreign body to identify any mucosal injury or a second foreign body which may occur in as many as 5% of patients.[36] A culture may be obtained and antibiotics administered if purulent secretions are noted.

A specific type of foreign body (e.g., a tack or sharp object) may become lodged in the larynx or upper trachea. Extraction with the forceps using standard endoscopic techniques may not be possible. Patients may require a tracheotomy and an open approach (e.g., a laryngotomy) to remove the foreign body.[53] A distal airway foreign body that cannot be removed may require a thoracotomy but this incurs a significant increase in surgical morbidity.

ALTERNATIVE TECHNIQUE

The use of a flexible fiberoptic bronchoscope to retrieve bronchial foreign bodies may be acceptable for EPs who are well trained with this technique. The mainstay of tracheobronchial foreign body retrieval remains rigid bronchoscopy.

AFTERCARE

Most patients should be breathing spontaneously after the retrieval of an airway foreign body. An endotracheal tube may rarely (e.g., in the presence of significant laryngeal or tracheobronchial edema) need to remain in place. This would require admission to an Intensive Care Unit. Humidified oxygen is helpful to keep the airway moist and prevent mucous crusts from forming. A postprocedural radiograph will help to determine any subcutaneous emphysema, pneumomediastinum or pneumothorax, or any changes to the lung fields following the extraction.

All patients who have undergone foreign body removal require at least a few hours of airway observation in a monitored setting. Racemic epinephrine treatments, Heliox, and/or intravenous dexamethasone can be administered as adjunctive therapies. Discharge from the hospital is acceptable when the patient is breathing comfortably and no longer in danger of airway compromise.[54] Some patients may be discharged home the same day while others may require multiple days of airway support and observation. Educate parents and caregivers about the prevention of foreign body aspiration.[12,16-18]

Granulation tissue, severe inflammation, and a pneumonia can form around the foreign body making removal difficult or impossible when a bronchial foreign body has been present for a prolonged period of time.[55] It may be necessary to treat the patient with intravenous steroids and antibiotics for 48 hours, with or without intubation, followed by a repeat bronchoscopy and a repeat attempt to remove the foreign body.

COMPLICATIONS

Complications from the foreign bodies themselves (e.g., a button battery) or attempts at their retrieval can potentially lead to hypoxia or cerebral anoxia. Intraoperative complications can occur in the hands of both experienced and inexperienced Endoscopists. Loss of control of the foreign body can turn a partially obstructed foreign body into a completely obstructive foreign body and lead to a respiratory arrest. Cardiac arrhythmias can occur from hypoxia or direct pressure on the left mainstem bronchus. Bradycardia may also result from stimulating the trachea or from prolonged hypoxia. Postoperative problems can include laryngeal or tracheobronchial edema from the foreign body or the instrumentation of the airway. Mucosal irritation can instigate a tracheitis or bronchitis. Pneumonia can develop. Pneumomediastinum has been reported in up to 13% of aspirations and a pneumothorax slightly less frequently.[56]

A foreign body pulled up from a mainstem bronchus can become dislodged in the larynx or trachea and cause a complete airway obstruction. A foreign body in the hypopharynx can be pushed distally into the airway and result in a total airway obstruction or can be pushed distally into the esophagus causing obstruction of the esophagus and anterior displacement of the membranous trachea. The foreign body needs to be quickly removed or pushed back down into one of the mainstem bronchi to allow ventilation of at least one lung or a surgical airway needs to be performed. Failure to react appropriately in this situation can result in asphyxiation and death.

FUTURE CONSIDERATIONS

A three-pronged instrument has been developed to grasp a round foreign body.[57] This may be used through a fiberoptic endoscope or freehanded in the ED. It was designed similar to devices available at the local hardware store. More research is needed before this device may be used in the ED.

SUMMARY

Airway foreign bodies can pose a diagnostic and therapeutic challenge. The presentation may be obvious or require review of any historical fact, physical abnormality, or radiographic abnormality that may lead to a definitive or presumed diagnosis of an airway foreign body. Suspicion of the presence of a foreign body must be followed up with visualization of the airway in the ED or in the Operating Room. Transfer may be required for stable patients if the endoscopy team and equipment are not available, especially in the case of children. Hypopharyngeal foreign bodies may be safely removed in the ED by an EP. The EP must anticipate and be prepared to perform critical airway management if there is clinical degradation including complete airway obstruction. These airway skills include intubation, forcing the foreign body into a mainstem bronchus to facilitate temporary one lung ventilation, cricothyrotomy, and percutaneous transtracheal needle ventilation.

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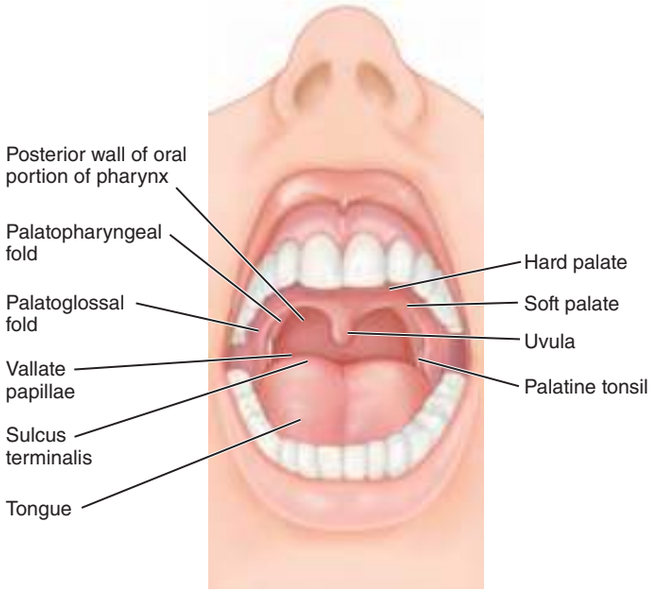


FIGURE 208-1. Anatomy of the oropharynx as seen through the open mouth.

208 Peritonsillar Abscess Incision and Drainage

Daniel S. Morrison and Chirag N. Shah

INTRODUCTION

A peritonsillar abscess is the most common deep infection of the head and neck encountered in young adults in the Emergency Department.[1,2] The incidence has remained stable in the United States at 30 cases per 100,000 people.[3] The incidence in the United Kingdom has increased 18% over 10 years.[3,4] This infection can occur in all age groups. It is relatively rare before the age of 5 years. The highest incidence occurs in teenagers and young adults, and incidence gradually declines after the age of 40.[5,6] There is a female-to-male predominance until the age of 14.[7] There remains controversy in the literature regarding the optimal antibiotic choice and the mechanism of drainage. The objective for the Emergency Physician remains to make an accurate diagnosis, to institute appropriate care, and to arrange timely follow-up.

ANATOMY AND PATHOPHYSIOLOGY

Knowledge of oropharynx anatomy is imperative. The anatomy of the oral cavity is relatively simple (**Figure 208-1**). The peritonsillar abscess can be found posterolateral to the palatine tonsil and posterior to the palatoglossal fold (i.e., arch). **Note the close proximity of the internal carotid artery and the facial artery to the peritonsillar abscess (Figure 208-2). Use extreme care to not penetrate too deeply and puncture or lacerate these arteries.**

The duration of reported symptoms ranges from 2 to 7 days for patients with ultrasound (US)-proven peritonsillar abscesses.[8,9] The most common symptoms include fever, sore throat, dysphagia, muffled voice (i.e., the "hot potato" voice), and trismus.[10] Physical examination will reveal a nonexudative pharyngitis in the majority of cases, soft palate edema, a bulging prominent tonsil, and uveal deviation away from the abscessed tonsil (**Figure 208-3**). The differential diagnosis includes intratonsillar abscess, peritonsillar cellulitis, infectious mononucleosis, malignancy, leukemia, odontogenic infections, aberrant carotid arteries, internal carotid artery pseudoaneurysms, and aneurysms of the internal carotid artery. The frequency of bilateral peritonsillar abscesses has been reported to be between 1.9% and 24% with the unsuspected contralateral abscess being discovered during surgery for the acute abscess tonsillectomy.[11] The Emergency Physician should maintain a high index of suspicion even if uvular deviation is not noted.

Intraoral US for a peritonsillar abscess has been performed since the 1990s. It was first described in the Otolaryngology literature and subsequently in the Emergency Medicine literature. The first case series of patients whose peritonsillar abscesses were drained under US guidance was described by Blaivas and colleagues.[12] US guidance has been shown to have a high degree of sensitivity (85% to 92%) and specificity (80% to 100%).[13,14] US offers the advantage of confirming the presence of an abscess prior to aspiration attempts, allowing visualization of important neighboring structures (e.g., the internal carotid artery), and using no ionizing radiation.[15-19] Bedside US reduces the delay of consulting an Otolaryngologist, admission, and drainage of the abscess when compared to a computed tomography scan.[20] Some patients with a peritonsillar abscess may be misdiagnosed with cellulitis and not undergo drainage. The use of US can prevent this. Up to 24% of patients have false-negative results on blind aspiration because the peritonsillar abscess can be multilocular and its location can vary.[13,21,22] US will identify the location of the abscess to limit the number of false-negative aspirations. US decreases the Otolaryngologist consult rate.[23] Transcutaneous US can be used to assist with the diagnosis of a peritonsillar abscess in patients in whom intraoral US may not be possible (e.g., pediatric population).[24-26]

The peritonsillar abscess has been attributed to progression and direct extension of an acute exudative pharyngitis. It has also been proposed that the Weber's glands located in the supratonsillar space are the actual site of bacterial invasion and subsequent abscess

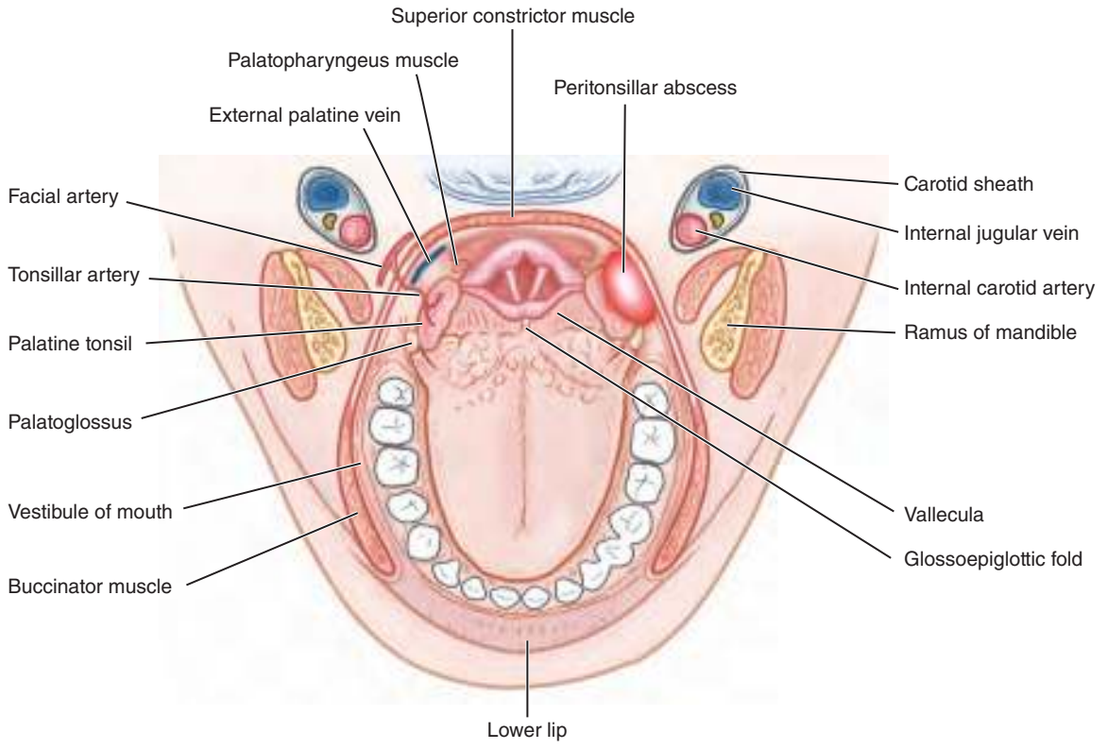


FIGURE 208-2. Horizontal section through the mouth and oropharynx. Note the close proximity of the peritonsillar abscess to the internal carotid artery and the facial artery.

formation.[27-29] The glands clear the tonsillar area of debris and assist with the digestion of food particles trapped in the tonsillar crypts. A local cellulitis can develop if the Weber's glands become inflamed.[28] The bacterial inoculum results in tissue necrosis and pus formation between the tonsillar capsule and the lateral pharyngeal wall and/or the supratonsillar space. Progression of pus formation and cellulitis within the supratonsillar space results in a gradual involvement of the surrounding musculature, particularly the internal pterygoids, and leads to muscle spasm and trismus.

Most peritonsillar abscesses are polymicrobial infections containing common oropharyngeal microflora with an average of three isolates per specimen.[6,30,31] Group A streptococci and *Fusobacterium necrophorum* are the most commonly isolated organisms if the Weber's glands become inflamed.[6] Pure cultures containing *Streptococcus milleri* group, *Staphylococcus aureus, Nocardia asteroids, Haemophilus influenzae, Arcanobacterium haemolyticum, Streptococcus pneumonia,* and *Streptococcus pyogenes* have also been recorded.[6,30] There can be a higher incidence of group A beta-hemolytic streptococci in the winter and spring compared to the summer.[7] Patients aged 15 to 24 have a higher risk of peritonsillar abscess due to *Fusobacterium necrophorum*.[7,31] Children up to 9 years of age are at increased risk of being affected by group A streptococci.[7,31] There can be an increased prevalence of beta-lactamase–producing organisms.[32]

Treatment options for a peritonsillar abscess have undergone a significant amount of debate in the literature. Most treatment guidelines recommend appropriate antibiotic selection and removal of purulent fluid. Techniques of fluid removal range from simple or single aspiration of the abscess to repeated aspirations to incision and drainage. Patients treated with aspiration alone have success rates ranging from 85% to 100%.[33-35] The cure rates for simple aspiration versus incision and drainage are similar.[36-40] The overall recurrence rate is less in patients undergoing incision and drainage.[33-35] Patients with less severe symptoms and smaller abscesses may be treated medically without significant outcomes from those treated with surgical intervention.[41] A younger age, fewer episodes of acute tonsillitis, and small abscess pockets are significant predictors in the pediatric population of a good response to nonsurgical treatment.[42] The response to nonsurgical treatment significantly deteriorates after 7.5 years of age.[42]

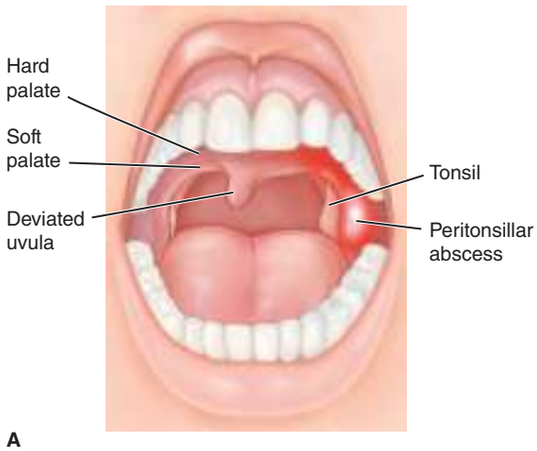
INDICATIONS

Most peritonsillar abscesses require either aspiration or incision and drainage. **The gold standard to diagnose a peritonsillar abscess remains the collection of pus through needle aspiration or the identification of a peritonsillar fluid collection using US.** The decision regarding the drainage technique is left to the Emergency Physician's preference and, if needed, in consultation with an Otolaryngologist.

US may be used to confirm the presence of a peritonsillar abscess, to guide needle aspiration of a confirmed peritonsillar abscess, and/or rescue a failed blind needle aspiration. The use of US can confirm the presence of a cellulitis with no underlying abscess. It will also confirm that the "mass" is not an aberrant internal carotid artery.[19,43] US can distinguish a simple peritonsillar abscess from more complex masses that may have parapharyngeal spread.[44]

CONTRAINDICATIONS

There are no absolute contraindications to draining a peritonsillar abscess. Patients with severe trismus limiting visibility, limiting access, and limiting intraoral US may require intravenous analgesics and muscle relaxants, procedural sedation (Chapter 159), or intraoperative drainage. **Consult an Otolaryngologist for all patients who are coagulopathic, taking oral anticoagulants, or with a known bleeding disorder.** These patients are at risk for significant



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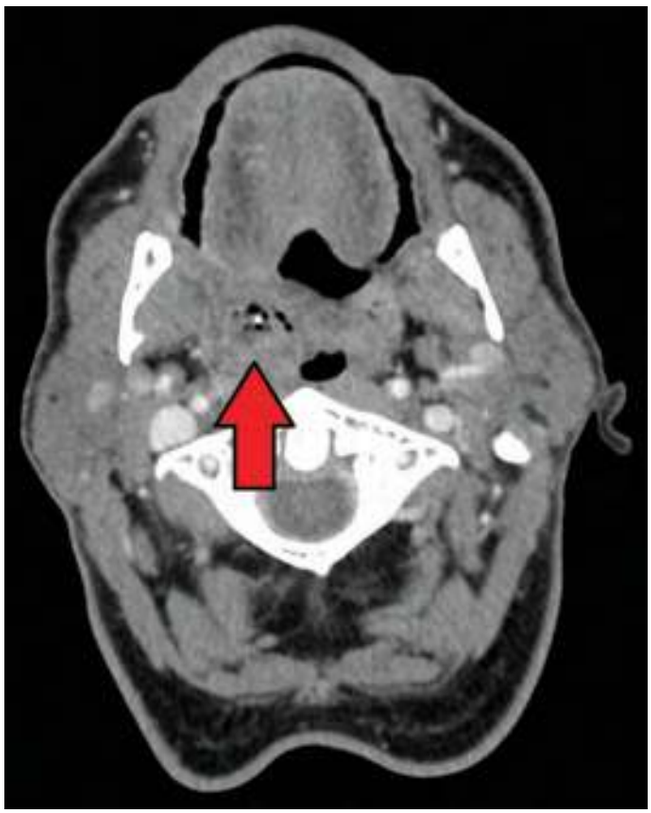


C

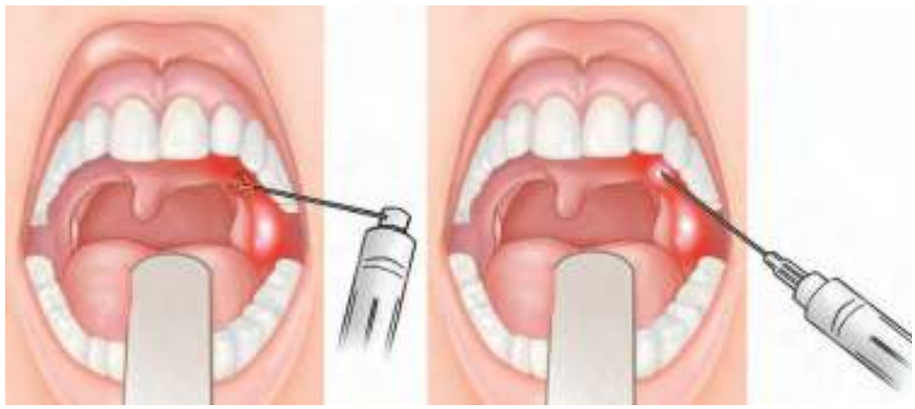
FIGURE 208-3. A peritonsillar abscess. The abscess displaces the tonsil forward and medially. The uvula is deviated toward the contralateral side. ***A.*** Artist illustration. ***B.*** Clinical photo. The black arrow identifies the abscessed tonsil. ***C.*** A computed tomography scan through the abscess. The red arrow identifies the abscess. (Parts B and C used from www.wikimediacommons.com.)

bleeding and associated complications. Admit children to the hospital for intravenous antibiotics, possible incision and drainage under general anesthesia, and possible tonsillectomy. The procedure should be avoided in patients who are uncooperative, unable to follow instructions, unable to sit upright, and those who are very young to prevent iatrogenic complications.

EQUIPMENT

GENERAL SUPPLIES

- #11 scalpel blade on a handle
- Curved hemostat
- Frazier suction catheter
- Suction source and tubing
- Tongue depressors
- Topical anesthetic spray (e.g., Cetacaine, lidocaine, tetracaine, or benzocaine)
- Syringe, 3 or 5 mL
- 25 or 27 gauge needle, 2 inches long
- Local anesthetic solution with epinephrine
- 10 mL syringe
- 18 gauge needle
- Culturettes or culture bottles
- Headlamp or adjustable overhead light source
- Other light sources (e.g., a pelvic speculum or curved laryngoscope blade) can be used as options
- Gloves



A B

FIGURE 208-4. Anesthesia techniques. ***A.*** Topical spray anesthesia. ***B.*** Infiltrative anesthesia.

- Face mask with an eye shield
- Gown
- Oral rinse solution (e.g., hydrogen peroxide or Peridex)

US GUIDANCE

- US machine
- High-frequency microconvex endocavitary or hockey-stick US transducer[45]
- Sterile US gel
- US transducer cover

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Obtain an informed consent for the procedure and place this in the medical record. When performing the procedure on a neonate or child, it is advisable to give the parent the option to leave the room or look away as the procedure can be disconcerting to some parents. The discussion should include proximate structures as well as hand signals that should be used if the patient requests the procedure to be stopped once it starts. Ensure that the patient has a thorough understanding of the postprocedural care instructions and follow-up requirements.

Position the patient sitting in an upright multipositional procedure chair. Alternatively, place the patient sitting upright on a gurney with the back elevated. Prepare the wall suction unit to ensure it is working. Apply suction tubing and a suction catheter to the suction source. The Emergency Physician should wear gloves, a gown, and a face mask with eye protection. This protective wear will prevent against becoming exposed, contaminated with oral secretions, or contaminated with abscess contents during the procedure if the patient coughs.

The use of good lighting cannot be overemphasized. Apply a headlamp if one is available. An alternative is an overhead adjustable light source. Position the light so that it is aimed in the patient's mouth. The overhead light often hits the examiner in the head, casts shadows, is too bright for the patient's eyes, and is also difficult to properly position because both of the Emergency Physician's hands must be used for the procedure. An additional alternative is to use a vaginal speculum with a fiberoptic light source (**Figure 88-3*F***). This may require an assistant to position and hold the speculum against the tongue and apply downward pressure. A further alternative is to use a curved laryngoscope blade to depress the tongue and provide adequate lighting.

Incision and drainage must be preceded by adequate anesthesia to the abscess site. Determine the most fluctuant region of the abscess. Anesthetize this area. Spray topical anesthetic over the abscess (**Figure 208-4*A***). Dry the mucosa overlying the peritonsillar abscess with a gauze square. Arm a 3 mL syringe with a 25 or 27 gauge needle. Inject 1 mL of local anesthetic solution containing epinephrine through the area of topical anesthesia and just under the mucosal surface (**Figure 208-4*B***). Allow 3 to 5 minutes for the anesthetic to work.

Atomizer devices are usually not available in the Emergency Department. Many devices are available commercially. These are disposable devices that are single patient use (**Figure 29-1 and Table 29-1**). A Popular device is the MAD line of mucosal atomizer devices (LMA North America, San Diego, CA). They make multiple devices for all topical needs.

TECHNIQUES

ASPIRATION

Identify the area of maximal fluctuance at the upper pole of the abscess. Anesthetize the area as described previously. Prepare the equipment. Apply an 18 gauge needle onto a 10 mL syringe. **A smaller needle may not allow thick pus to be aspirated**. Break the bead of the syringe. Trim the needle cap and place it over the needle to act as a depth gauge (**Figure 208-5*A***). The needle should project only 1 cm from the distal end of the needle cap. Alternatively, apply a piece of tape onto the needle to mark a point 1 cm from the tip of the needle (**Figure 208-5*B***). **The guard cap or tape serves as a marker for the maximum allowable depth to insert the needle during the procedure. Limiting of the depth of insertion of the needle should help to prevent injury to the carotid artery.** The mean distance from the abscess to the mucosal surface can range from 6 to 15 mm and the distance from the wall of the abscess to the internal carotid artery can vary from 5 to 25 mm.[46]

Depress the tongue with a tongue depressor held in the nondominant hand (**Figure 208-6*A***). A pelvic speculum or curved laryngoscope blade may be used. Insert the prepared needle attached to the syringe into the upper pole of the abscess and into the point of maximal fluctuance (**Figures 208-6*A* and 208-6*B***).[36,39,47,48] Hold and advance the needle parallel to the floor and direct posteriorly. **Do not direct the needle laterally where it can injure the carotid artery.** Aspirate while advancing the needle. Approximately 70% to 90% of abscesses occur in the upper pole of the tonsil.[9,36,39] Continue to aspirate and remove as much purulent material as possible

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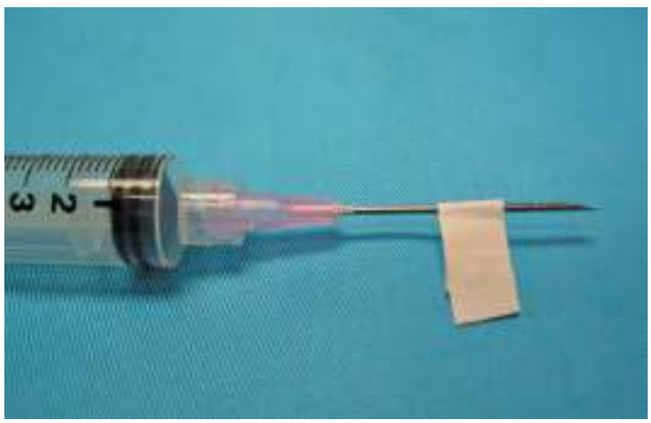

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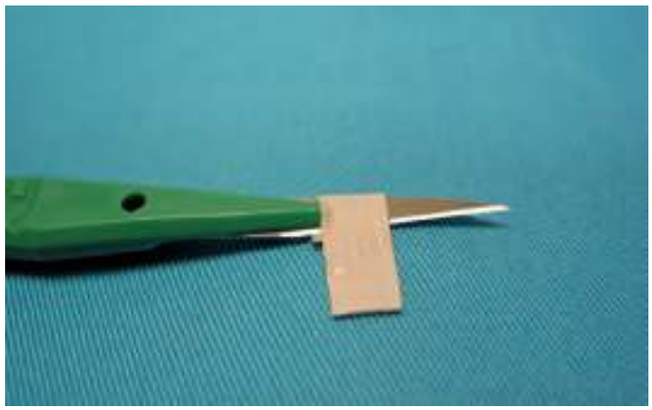


C

FIGURE 208-5. Safety techniques to prevent injury to the internal carotid artery. ***A.*** The needle cover is cut and placed over the needle as a guard. ***B.*** Tape applied to an 18 gauge needle. ***C.*** Tape applied to a #11 scalpel blade.

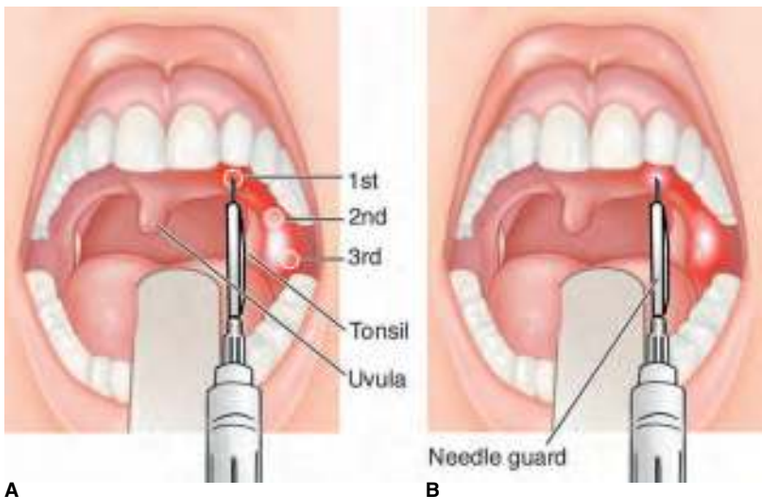
if purulent fluid is obtained. Consider obtaining a culture and sensitivity of the aspirated material. Allow the patient to rinse and spit several times with Peridex solution or half-strength hydrogen peroxide solution.

Reattempt the procedure if the initial aspirate is negative by inserting the needle into the middle pole and then the inferior pole of the abscessed tonsil until purulent material is obtained (**Figure 208-6*A***). **A completely negative aspiration, while more consistent with a tonsillar cellulitis, does not rule out the existence of an abscess.** Gentle palpation will reveal fluctuance when an abscess is present.

Almost 89% of patients were successfully treated with a single aspiration.[49] Just over 11% needed an additional aspiration or an incision and drainage in the immediate short term. High fever was a predictor of the need for additional procedures.

INCISION AND DRAINAGE

It is recommended to always perform a needle aspiration prior to the incision and drainage technique. Aspiration will localize the collection of pus and allow a more accurate incision and drainage.



A B

FIGURE 208-6. Needle aspiration of a peritonsillar abscess. ***A.*** Recommended sites for needle aspiration. ***B.*** Aspiration in the first area.

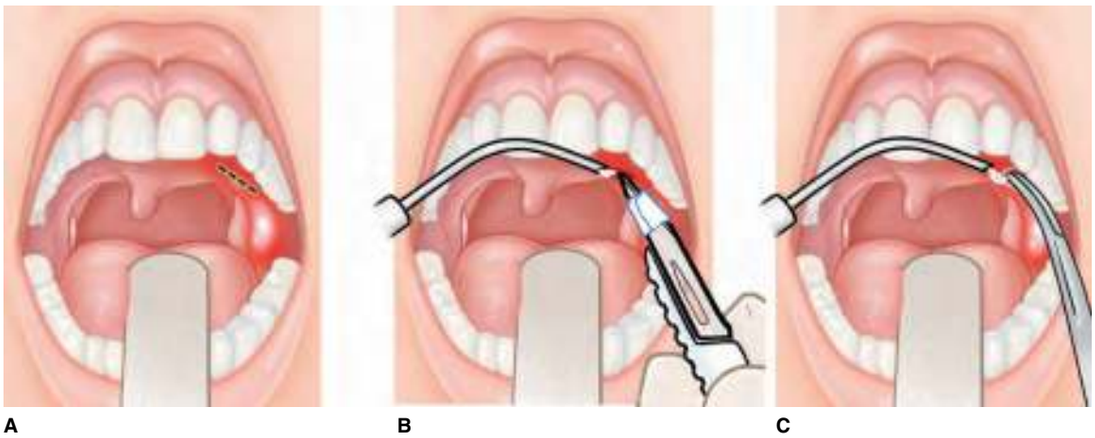


FIGURE 208-7. Incision and drainage of a peritonsillar abscess. ***A.*** The incision site is the same site for the first aspiration. ***B.*** Incision with a #11 scalpel blade. Note the tape marking the maximum insertion depth of the scalpel blade. Suction any bloody or purulent fluid that escapes from the incision. ***C.*** Hemostat gently inserted to break any loculations.

A negative aspiration at all three sites (Figure 208-6*A*) is a contraindication for an incision and drainage procedure. It may be too early and an abscess has not yet formed. A negative aspiration at all three sites suggests the patient has a tonsillar cellulitis requiring oral antibiotics, gargles with hydrogen peroxide, and follow-up in 24 hours for reevaluation.

Identify the area of maximal fluctuance at the upper pole of the abscess. Anesthetize the area as described previously. Prepare the equipment. Place a piece of tape on the #11 scalpel blade so that only 0.75 to 1 cm is exposed (**Figure 208-5*C***). Place the Frazier suction catheter near the incision site. Insert the scalpel blade to make a horizontal stab wound to a maximum depth of 1 cm in the same area noted for the aspiration technique (**Figures 208-7*A* and 208-7*B***). The Frazier suction catheter will remove any blood and purulent material and prevent the patient from aspirating. **The depth of the stab should be no more than 1 cm. Extend the length of the incision to a maximum of 1.0 to 1.5 cm**.

Insert a curved hemostat into the wound (**Figure 208-7*C***). Gently spread apart the jaws of the hemostat to break up any loculations in the abscess. Continue to simultaneously suction the area during the procedure. Packing of the abscess cavity is not required. Obtain a culture and sensitivity of the purulent material. Allow the patient to rinse and spit several times with Peridex solution or half-strength hydrogen peroxide solution. Leave the suction in the patient's hand so they can use it as needed.

ULTRASOUND GUIDANCE

US has the ability to identify the location of the abscess and prevent unnecessary "dry aspirations." The use of US should be strongly considered whenever available. Anesthetize the area as described previously. Prepare the equipment. Place sterile US gel on the scanning surface of the US transducer. Apply the US transducer cover. Take care to avoid trapping air bubbles between the cover and the US transducer as they can seriously degrade the image. Apply sterile US gel over the covered US transducer surface.

Gently insert the US transducer into the patient's oral cavity. Direct the US transducer to the peritonsillar area and until it rests lightly against the posterior pharynx (**Figure 208-8**). Maintain the US transducer in a transverse orientation to maximize visualization of the posterior pharynx. Note the location of the palatine tonsil, a small oval structure with low-level echoes, and the internal carotid artery (**Figure 208-9**). A peritonsillar abscess can have variable appearances on US. They usually are heterogeneous, cystic, and adjacent to the tonsil (**Figure 208-10**). The carotid artery usually lies 5 to 20 mm posterolateral to the tonsil. Note the depth and location of the abscess.

FIGURE 208-8. Placement of the endocavitary US transducer inside the oral cavity and directed against the peritonsillar area.

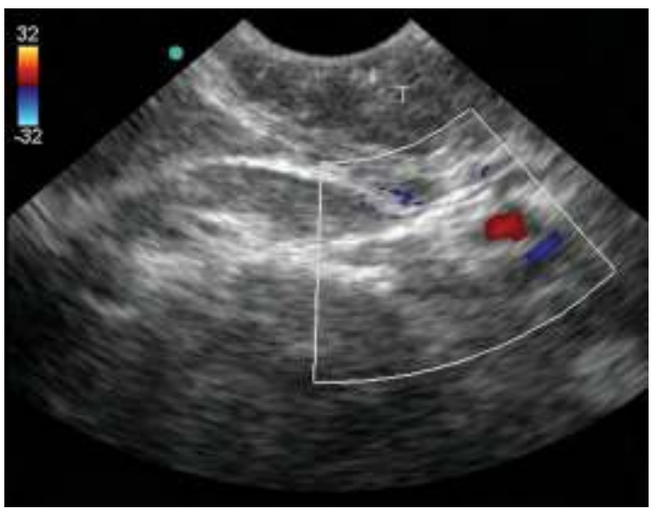


FIGURE 208-9. Transverse color Doppler scan of the normal posterior pharynx. The tonsil (T) is visible on the top of the image. The internal carotid artery is noted by its red color Doppler signal.

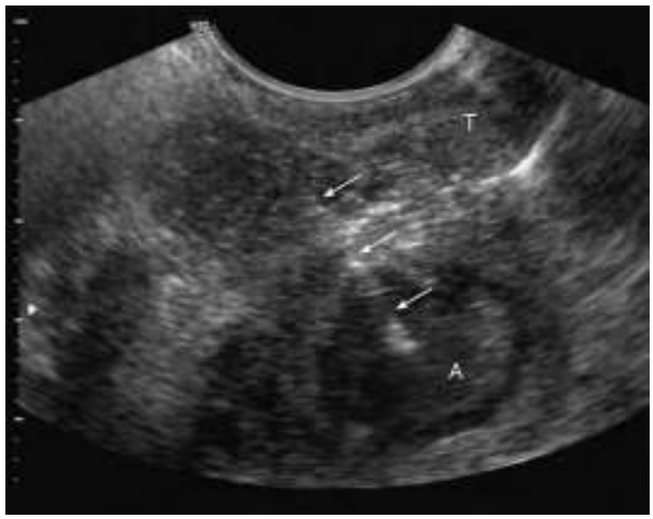


FIGURE 208-10. Intraoral ultrasound image. The needle (arrows) is inserted into the peritonsillar abscess (A). The tonsil (T) is visible on the top of the image. The internal carotid artery is not visualized in this image.

Confirm the presence of a peritonsillar abscess and map its location. Use the previously described techniques for aspiration or incision and drainage. Alternatively, perform a needle aspiration under real-time US guidance. Use the long axis approach to direct the needle into the peritonsillar abscess (**Figure 208-10**). Leave the suction in the patient's hand so they can use it as needed. Ensure the endocavitary transducer is cleansed per institutional guidelines.

ASSESSMENT

Aspiration or incision and drainage will result in significant relief of the patient's pain and trismus. Allow the patient to rinse and spit with either Peridex solution or half-strength peroxide solution. Observe the patient for any evidence of continued bleeding or upper airway symptomatology. Assess the patient's ability to tolerate oral fluids prior to discharge.

AFTERCARE

Discharge the patient with oral antibiotics and analgesics.[50,51] Antibiotic resistance to clindamycin and erythromycin is common across streptococcal strains and staphylococcal strains.[31] Penicillin resistance is common in staphylococcal strains responsible for a peritonsillar abscess.[31] The emergence of beta-lactamase–producing organisms has to be considered in antibiotic selection.[52] Many Otolaryngologists will add to the penicillin either clindamycin or metronidazole due to the increasing incidence of penicillin-resistant organisms.[33,53] Penicillin or a cephalosporin plus metronidazole appears to be effective in vitro in 98% of isolates.[54] Nonsteroidal anti-inflammatory drugs (NSAIDs) should adequately control pain and fever. Administration of 3 mg/kg, to a maximum of 250 mg, of methylprednisolone intramuscularly has been shown to decrease pain and inflammation.[55] Alternatively, the use of a single dose of 10 mg of intravenous or intramuscular dexamethasone has been shown to reduce pain levels and improve patient quality of life at 24 hours.[56,57]

Recommend to the patient to follow a soft diet and drink plenty of fluids during the first 48 hours after the intervention. Instruct the patient to gargle with half-strength hydrogen peroxide or Peridex after each meal, at a minimum, and several other times per day. Arrange follow-up within 48 hours. **The patient should follow-up sooner if they do not improve.** Instruct the patient to return to the Emergency Department immediately if they develop bleeding, shortness of breath, difficulty swallowing, drooling, or have any concerns.

Admission is generally required for patients who appear toxic, pediatric patients, dehydrated patients, immunocompromised patients, patients with recurrent abscesses, and patients who are unable to tolerate oral fluids. These patients require observation for 23 hours and intravenous antibiotics. The abscess is often drained in the Operating Room by the Otolaryngologist.

Patients may develop recurrent peritonsillar abscesses. Predictors for recurrence include the need for a repeated procedure at the initial presentation, younger age, female sex, and patients known to have recurrent tonsillitis.[49,58] Tonsillectomy has been shown to be curative for recurrence of peritonsillar abscess.[49]

COMPLICATIONS

There are few complications associated with the management of a peritonsillar abscess. Potential complications include aspiration pneumonitis, airway obstruction, hemorrhage, or extension of infection into deep tissue of the neck. Aspiration pneumonitis or lung abscess can occur secondary to abscess rupture.[28] Protect against aspiration of purulent material and subsequent pulmonic infection by having the patient sit upright during the procedure and using suction as the abscess is opened. Making an incision that is too large or too deep can injure the carotid artery or a carotid artery aneurysm. This could result in prolonged bleeding or hemorrhage. Always limit the depth of the needle or scalpel insertion and strongly consider US guidance. Certain patients may be sensitive to topical anesthetic spray and develop methemoglobinemia.

The use of US to locate and assist in the drainage of a peritonsillar abscess is associated with several pitfalls. Failure to recognize the internal carotid artery can be catastrophic for the patient. Always note the relationship of the tonsil and the peritonsillar abscess to the internal carotid artery.[8] The internal carotid artery lies posterolateral to the tonsil. The use of color Doppler can assist in the identification of the carotid artery and differentiate it from the peritonsillar abscess. US may fail to identify a peritonsillar abscess. Fluid within a peritonsillar abscess is usually hypoechoic. It can appear isoechoic or hyperechoic.

SUMMARY

A peritonsillar abscess is commonly encountered in the Emergency Department. Diagnosis and treatment result in rapid symptom resolution in the majority of patients. Admission may be required in a few instances for observation and intravenous antibiotics. Appropriate antibiotics after the procedure can prevent a recurrence of the peritonsillar abscess. The use of steroids in conjunction with appropriate drainage improves the patient's pain and quality of life.

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SECTION 14

Dental Procedures

209 Dental Anesthesia and Analgesia

Eric F. Reichman

INTRODUCTION

Dental anesthesia techniques are used by Emergency Physicians for a variety of intraoral and extraoral conditions. This includes dental caries, jaw fractures, dry sockets, intraoral hemorrhage, laceration repair, and tooth fractures. These techniques are simple to learn, easy to perform, and provide temporary pain relief for the patient. **The Emergency Physician can provide pain-free intraoral manipulations, extraoral manipulations, facial manipulations, and simple pain control until the patient receives definitive evaluation and treatment by a Dentist or Oral Surgeon.** The fundamental principles of dental anesthesia and anatomy will be discussed so that the Emergency Physician will feel knowledgeable and comfortable performing dental anesthetic techniques.

ANATOMY AND PATHOPHYSIOLOGY

An understanding of the anatomy of the fifth cranial nerve is essential to performing dental nerve blocks (**Figure 209-1**).[1-3] The fifth cranial nerve is also referred to as CN V or the trigeminal nerve. It is the largest cranial nerve. It is a mixed cranial nerve containing primarily sensory fibers to the skin of the face and scalp, the nasal cavity, and the oral cavity. The motor fibers innervate the muscles of mastication.

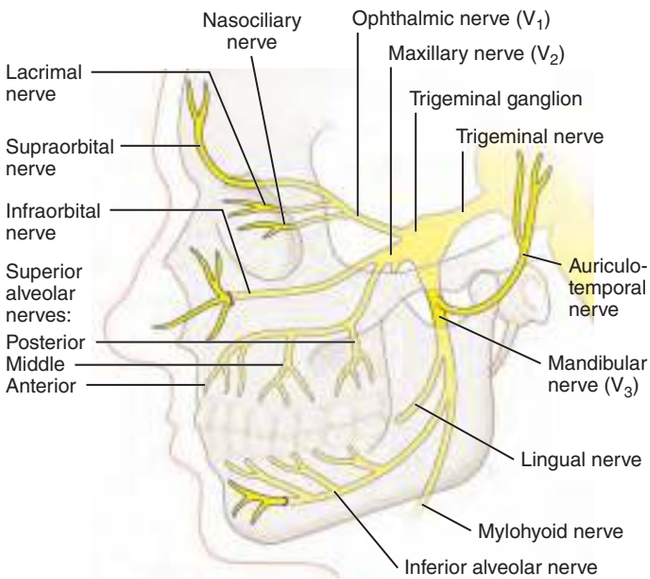

FIGURE 209-1. The anatomy of the trigeminal nerve.

The trigeminal nerve originates in the brainstem as a small motor root and a large sensory root. These roots fuse as they leave the brainstem. The trigeminal nerve travels forward into the middle cranial fossa where it expands into the large and crescent-shaped trigeminal ganglion. The trigeminal ganglion divides to give rise to the three divisions of the trigeminal nerve: the ophthalmic nerve (V_1), the maxillary nerve (V_2), and the mandibular nerve (V_3) (**Figure 209-1**). Each of these nerves leaves the middle cranial fossa through its own foramen.

OPHTHALMIC NERVE

The ophthalmic nerve is the smallest branch of the trigeminal nerve. It travels forward in the lateral wall of the cavernous sinus and enters the orbit via the superior orbital fissure. It provides sensory innervation to the forehead, scalp, upper eyelid, cornea, nasal cavity, sinuses, and the orbit. This nerve is not discussed further because it does not innervate any oral or dental structures.

MAXILLARY NERVE

The maxillary nerve is purely sensory. It travels forward in the lateral wall of the cavernous sinus and exits the cranial vault via the foramen rotundum into the pterygopalatine fossa. It then enters the orbit through the inferior orbital fissure to continue on as the infraorbital nerve and emerge on the face. The infraorbital nerve terminates as a sensory nerve to the lower eyelid, upper cheek, nose, and upper lip. The infraorbital nerve gives off the anterior superior alveolar nerves prior to its termination. These nerves supply the maxillary sinus, the maxillary incisors, the maxillary canine teeth, and the maxillary premolar teeth. The anterior superior alveolar nerve occasionally crosses the midline to supply the contralateral maxillary incisors.

The maxillary nerve forms numerous branches in the pterygopalatine fossa. The zygomaticofacial and zygomaticotemporal nerves are cutaneous to the face and temple. The nasal and nasopalatine nerves supply the nasal cavity and floor of the nasal cavity. The nasopalatine nerve exits the nasal canal through the midline incisive foramen just posterior to the central incisors. It provides sensory innervation to the anterior hard palate and associated soft tissues. The greater palatine nerve exits the greater palatine foramen to provide sensory innervation to the posterior two-thirds of the hard palate. The lesser palatine nerve exits the lesser palatine foramen and provides sensory innervation to the soft palate. The greater and lesser palatine nerves exit 1 cm medial to the junction of the second and third molar on the hard palate. The middle superior alveolar nerve provides sensory innervation to the premolars, and occasionally the canine and the first molar teeth. The posterior superior alveolar nerve provides sensory innervation to the molars and occasionally the premolars.

MANDIBULAR NERVE

The mandibular nerve is the largest division of the trigeminal nerve. It is the only division of the trigeminal nerve to contain motor fibers. The mandibular nerve exits the middle cranial fossa via the foramen

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ovale. It provides branches to the meninges, the small muscles of the palate, and the medial pterygoid muscle. It divides into a small anterior division and a large posterior division.

The anterior division of the mandibular nerve is primarily motor. It innervates the muscles of mastication (i.e., masseter, temporalis, and lateral pterygoid muscles). The sensory portion of the anterior division is the buccal nerve. This nerve travels between the two heads of the lateral pterygoid muscle, under the masseter muscle, and emerges from the anterior border of the masseter muscle. It travels forward to innervate a small and variable portion of the skin of the cheek. It primarily innervates the mucous membranes of the cheek.

The posterior division of the mandibular nerve is purely sensory. It divides into the auriculotemporal, inferior alveolar, and lingual nerves. The auriculotemporal nerve supplies sensory innervation to the skin of the auricle, external auditory canal, scalp, and temporomandibular joint. It conveys postganglionic parasympathetic fibers to the parotid gland.

The lingual nerve descends into the mouth and travels along the lateral surface of the tongue. It supplies sensory innervation to the anterior two-thirds of the tongue and the floor of the mouth. The lingual nerve receives near its origin and conveys the chorda tympani from the facial nerve. The chorda tympani provides taste sensation to the anterior two-thirds of the tongue and preganglionic fibers to the submandibular ganglion for the submandibular and sublingual glands.

The inferior alveolar nerve descends immediately posterior and adjacent to the lingual nerve. It enters the upper one-third of the ramus of the mandible posterior to the lingula to enter the mandibular canal. It provides sensory innervation to the mandible, the mandibular teeth, and the adjacent mucous membranes. The inferior alveolar nerve gives origin to the mental nerve. The mental nerve exits the mental foramen located on the outer surface of the mandible between the first and second premolars. It supplies sensory innervation to the lower lip, the skin of the chin, and the mucous membrane of the chin.

INDICATIONS

Dental nerve blocks can be performed to provide temporary relief of pain. They are often used to provide relief from alveolar ridge fractures, dental caries, dry sockets, mandible fractures, and tooth fractures. Nerve blocks can be performed prior to painful intraoral procedures such as incision and drainage of dental abscesses and laceration repair to the cheek, lips, oral mucosa, and tongue. Nerve blocks do not distort the local anatomy when compared to infiltration of the surrounding soft tissue with local anesthetic solution and allow better approximation of the wound edges during suturing. Nerve blocks are an excellent alternative if narcotic analgesics are contraindicated or to be avoided. Local anesthetic solutions containing epinephrine can be utilized to provide longer pain relief and vasoconstriction along the nerve distribution (**Table 209-1**).

CONTRAINDICATIONS

The two absolute contraindications to dental anesthesia include a known hypersensitivity to the anesthetic agent and gross distortion of the anatomic landmarks required to perform the nerve block. Other relative contraindications include an uncooperative patient such as an anxious adult, scared child, or any patient with altered mental status. These patients place themselves and the Emergency Physician at significant risk for injury. It may be most prudent to abort the procedure and perform procedural sedation and analgesia (or general anesthesia in the Operating Room) to ensure the safety of the patient and the Emergency Physician.

The needle used to inject local anesthetic solution should not traverse infected tissue. Injection of the local anesthetic solution into or through infected tissue is also a relative contraindication. These processes may result in spread of the infection into other adjacent tissues or tissue planes. It is possible to cause bacteremia when injecting into infected tissues. Local anesthetic solutions are less effective when injected into areas of infection or inflammation. An infection that tracks along a nerve and into a bone of the face and/or skull is extremely difficult to treat. Evaluate the risks and benefits of injecting through infected tissue and attempt an alternative method of anesthesia if possible.

EQUIPMENT

- Nonsterile gloves
- Antiseptic mouth rinse; any of the following:
  - Hydrogen peroxide
  - Ethanol (7%) with chlorhexidine (0.5%)
  - Povidone iodine solution
  - 0.12% chlorhexidine or Peridex solution
- 4×4 gauze squares
- Cotton-tipped applicators
- Aspirating dental syringe (**Figure 209-2**)
- Local anesthetic solution (**Table 209-1**)

TABLE 209-1 Local Anesthetic Solutions Commonly Used in Dental Anesthesia Procedures

Anesthetic solution	Proprietary name	Time of onset (minutes)	Pulpal duration of action (minutes)	Soft tissue duration of action (minutes)	Maximum dose (mg)*	Maximum adult or pediatric weight-based dose (mg/kg)
1% procaine	Novocaine	6–10	10	15–90	500	7.0
1% procaine with epinephrine		6–10	15	15–120	600	9.0
2% lidocaine	Xylocaine	2–5	10	30–45	300	4.5
2% lidocaine with epinephrine		2–5	60	180–300	500	7.0
3% mepivacaine	Carbocaine	5	5–10	90–120	400	6.6
2% mepivacaine with epinephrine		5	45–60	120–140	400	6.6
4% prilocaine	Citanest	3–5	10–60	60–240	400	6
4% prilocaine with epinephrine		3–5	60–90	180–480	400	6
4% articaine with epinephrine	Septocaine	1–3	45–75	180–300	500	7
0.5% bupivacaine	Marcaine	5	60–90	240–280	90	1.3
0.5% bupivacaine with epinephrine		5	90–120	240–720	90	1.3

*Do not exceed this quantity if the maximum weight-based dose is larger than this number.

FIGURE 209-2. The aspirating dental syringe and local anesthetic cartridges.

- Syringes, 1 mL and 3 mL
- Suction source and tubing
- Yankauer suction catheter
- Topical anesthetic (e.g., viscous lidocaine, viscous benzocaine, or aerosolized benzocaine)
- Overhead light source or headlamp
- 25 and 27 gauge needles, 2 inches long[2]

The above-listed supplies are required to provide dental anesthesia. They are contained in every Emergency Department. An aspirating dental syringe and anesthetic cartridges, if available, are ideal to perform the nerve blocks (**Figure 209-2**).[2] Standard 1 to 3 mL syringes armed with a 25 or 27 gauge, 2 inch long needle will work as a substitute. The aspirating dental syringe allows better control of the syringe and the ability to simultaneously aspirate and insert the needle with one hand. This allows the nondominant hand to be used to identify landmarks, retract the cheek or tongue, adjust the light source, and/or use the suction catheter. Many local anesthetic solutions are available in 1.8 mL carpules that fit into the dental aspirating syringe. The carpules are available with the local anesthetic solution and also with or without a vasoconstrictor (usually epinephrine). A dental or multipositional procedure chair would be preferred to the use of a standard cart or gurney.[4,5] Unfortunately, this may not be available in many Emergency Departments.

Numerous injectable agents are available to provide anesthesia (**Table 209-1**). The most commonly used agents are lidocaine and lidocaine with epinephrine (1:100,000). Longer acting agents are commonly available in the Emergency Department (e.g., bupivacaine and mepivacaine, with and without epinephrine) when a prolonged period of anesthesia is required. Avoid using long-acting local anesthetic agents if the tongue (i.e., inferior alveolar or lingual nerve blocks) or mucosa (i.e., buccal nerve block) is anesthetized, especially in children, to prevent the patient from biting the area and causing injury.

Relatively new devices are the Accupal (Accupal, Little Rock, AR) and the DentalVibe (Bing Innovations LLC, Boca Raton, FL). These devices are injection preparation tools that prepare the gums before the anesthetic needle is inserted. These devices vibrate and produce ultrasonic tissue stimulation to reduce pain sensation at the needle injection site. The devices are usually not available in the Emergency Department.

Numerous jet injectors for the delivery of local anesthetic agents are commercially available. These devices inject the local anesthetic solution under pressure and in a fine stream into the soft tissues without the use of a needle. The small volume of injected solution and its superficial penetration into the soft tissues only anesthetizes the soft tissues and not the teeth. **Thus, jet injectors cannot be used to anesthetize teeth.** They can be used to provide topical anesthesia.

Several studies have used ultrasound to visualize the nerves involved with dental blocks.[6,7] There was no more success using ultrasound than the traditional landmark technique.[7] With more studies and practice, ultrasound may become more widespread in its use for dental anesthesia.

PATIENT PREPARATION

Perform a thorough history and a directed physical examination, dependent on the clinical situation, in any patient undergoing dental anesthesia. Give special attention to the past medical history, past surgical history, current medications, and any history of allergic or adverse reactions to an anesthetic agent. A patient with severe systemic disease may be better served by rescheduling the procedure after appropriate consultation or referral. This will clarify functional reserve and treatment limitations.[4,5] Any patient with cardiac valvular disease, congenital heart anomalies, artificial heart valves, or other indications should receive antibiotic prophylaxis to help prevent bacterial endocarditis caused from transient bacteremia. Please refer to Chapter 210 for more complete details of antibiotic prophylaxis.

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain an informed consent for the specific technique to be performed. A formal consent may exist depending upon the institution in which one practices. Some physicians desire a formal signed and dated consent form. Other physicians choose to chart: "All the risks, benefits, and complications were described and discussed with the patient. They understood the procedure described and gave verbal consent for the procedure." This is physician and institution dependent. Place the patient in a well-lighted environment. Reassurance often alleviates a patient's anxiety regarding injections or manipulations and puts them at ease.[4]

Prepare the mucosa at the injection site. Completely dry the mucosa with gauze squares. Apply an antiseptic solution (e.g., 7% ethyl alcohol [ETOH] with 0.5% chlorhexidine solution, diluted povidone iodine, 0.12% chlorhexidine solution, or hydrogen peroxide) to the working area with a cotton ball or gauze square for 15 seconds. Alternatively, the patient can swish 0.12% chlorhexidine mouth rinse or hydrogen peroxide for 30 seconds and then spit it out.

Apply a topical anesthetic agent (e.g., viscous lidocaine or benzocaine with a cotton-tipped applicator or 20% benzocaine spray) for added patient comfort. **It is not recommended to use TAC (tetracaine, adrenalin, and cocaine) or other topical anesthetic combinations that are used for cutaneous laceration repair on mucosal surfaces.** Their use may lead to significant absorption and systemic toxicity.

TECHNIQUES

The general procedure of a dental nerve block will be described. The specific details are contained within each nerve block described below. Identify the anatomic landmarks required to perform the nerve block. Clean the mucous membrane at the injection site with a gauze square. Apply an antiseptic solution. Apply a topical anesthetic and allow it to work for 2 to 3 minutes. Reidentify the anatomic landmarks. Insert a 25 or 27 gauge needle into the appropriate area to deliver the local anesthetic agent. **If the patient experiences paresthesias, do not inject the local anesthetic solution. Paresthesias signify that the tip of the needle is within the nerve bundle.** Withdraw the needle 1 to 2 mm and allow the paresthesias to resolve. This usually takes 5 to 20 seconds. Inject the local anesthetic solution. Allow up to 10 minutes for the local anesthetic solution to take effect. Some Dentists prefer to apply pressure to the area immediately next to the site of the anesthetic injection with a cotton-tipped applicator. This aids in distracting the patient from the pain of injection. Other Dentists "jiggle" the mucous membrane to and fro rapidly as they simultaneously introduce the needle.[4,5]

SUPRAPERIOSTEAL INFILTRATION (FIELD BLOCK)

This technique is commonly used in dentistry. Excellent anesthesia can be achieved with this technique when it is used to anesthetize a branch of the anterior or middle superior alveolar nerve.[5,8] This technique deposits local anesthetic agent against the periosteum of the alveolar ridge adjacent to a tooth (**Figure 209-3*A***). The local anesthetic agent then infiltrates through the periosteum, the cortical plate of the maxilla, and the medullary bone to anesthetize the nerve root as it leaves the apex of the tooth. This technique works best for teeth with associated thin cortical bone. This includes the maxillary incisor, canine, and premolar teeth. The molars of the maxilla in an adult are less likely to be anesthetized with this technique as the cortical bone in which they lie is relatively thick and a poor conduit for the anesthetic. Supraperiosteal infiltration is also a poor technique for anesthesia of mandibular teeth in the adult patient for the same reasons. In children, the cortical bone of the maxillary molars and the mandible is thin and may allow this technique to be effectively utilized to anesthetize a tooth.

Anatomy: The anterior superior alveolar nerve provides sensory innervation to the ipsilateral medial and lateral incisors, canine, and sometimes the first premolar teeth. The middle superior alveolar nerve provides sensory innervation to the ipsilateral premolars, canine, and first molar teeth.

Patient positioning: Place the patient recumbent in a dental chair with their neck extended 45°. Alternatively, position the patient sitting upright with their back and head firmly set against an examination chair or table.

Landmarks: Use the nondominant hand to grasp and pull the upper lip outward and upward (**Figures 209-3*B* and 209-3*C***). Identify the mucobuccal fold above the tooth to be anesthetized.[5]

Needle insertion and direction: Clean, prep, and apply a topical anesthetic agent to the mucobuccal fold above the tooth to be anesthetized. Firmly grasp the upper lip. Pull it outward and upward to tighten the tissues and allow a clear identification of the maxillary mucobuccal fold (**Figures 209-3*B* and 209-3*C***). Insert a 27 gauge needle through the mucobuccal fold over the center of the tooth to be anesthetized (**Figures 209-3*B* and 209-3*C***). Aim the tip of the needle toward the maxilla. Advance the needle 1.0 to 1.5 cm until it contacts the maxilla (**Figure 209-3*A***). Withdraw the needle 1 mm. Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 1 to 2 mL of local anesthetic solution.

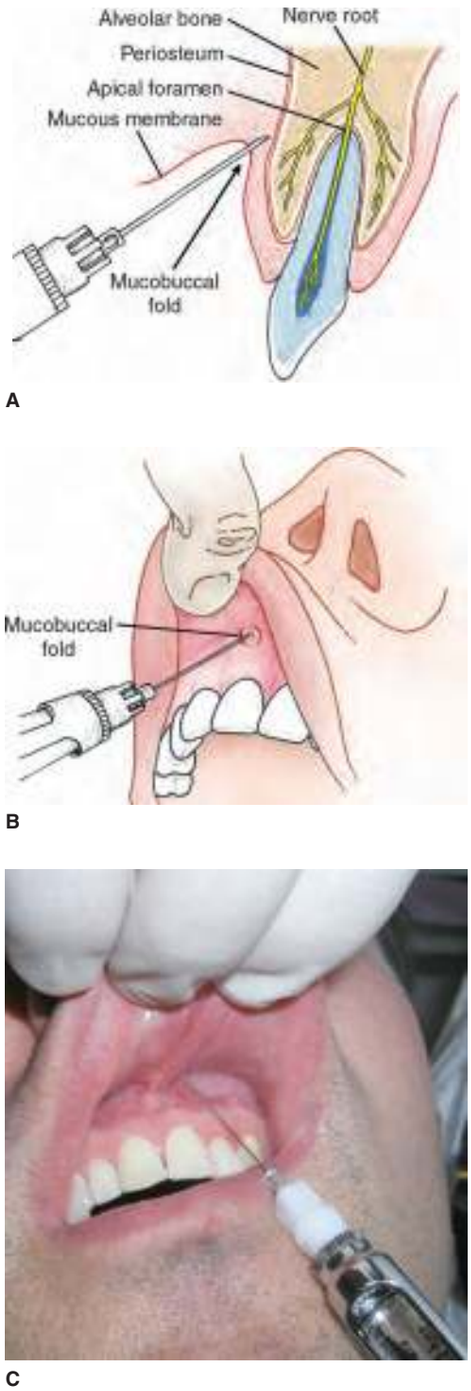


FIGURE 209-3. Supraperiosteal infiltration of local anesthetic solution. ***A.*** Illustration of the correct needle position. ***B.*** Elevate the upper lip and insert the needle through the mucobuccal fold. ***C.*** Photo. (Used with permission from Hadzic A: *New York School of Regional Anesthesia Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw Hill; 2007.)

Remarks: The anesthetic will be deposited in a nonoptimal location if the needle is too deep or too shallow. It may take as long as 10 minutes to achieve anesthesia as the local anesthetic solution diffuses through the cortical bone and to the nerve root. **Be careful when using this technique for anesthesia of the incisor or canine teeth because advancing the needle too far may breach the nasal cavity or maxillary sinuses.**

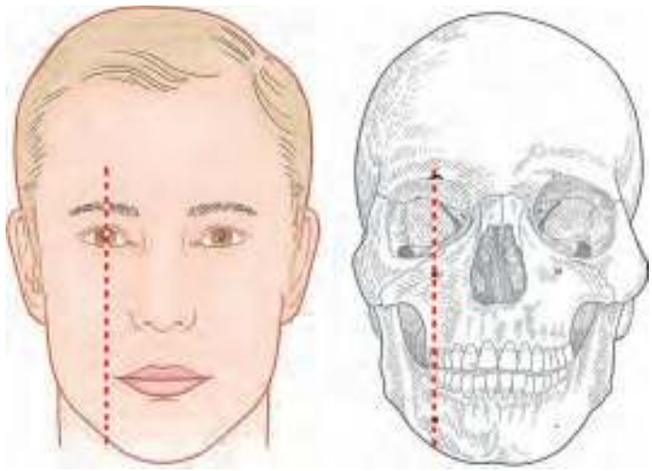


FIGURE 209-4. The supraorbital foramen, infraorbital foramen, and the mental foramen all lie along a straight line drawn through the pupil in the midposition.

INFRAORBITAL NERVE BLOCK

Anatomy: The infraorbital nerve is the terminal branch of the maxillary nerve. It exits the maxilla via the infraorbital foramina and supplies sensation to the ipsilateral upper lip, cheek, lateral nose, and lower eyelid. It may be blocked by either an extraoral or intraoral approach.

Patient positioning: Place the patient recumbent in a dental chair with their neck extended 30°. Alternatively, position the patient sitting upright with their head and back against an examination chair or table with the neck extended 30°. Instruct the patient to slightly open their mouth.

Landmarks: Identify the infraorbital foramen by palpation. It is located below the infraorbital ridge in the mid-pupillary line (**Figure 209-4**). The mid-pupillary line is a line drawn in the sagittal plane (i.e., vertical) through the pupil while the patient is staring straight ahead.

Needle insertion and direction (extraoral approach): Identify the infraorbital foramen as above. Clean and prep the skin over the infraorbital foramen. Instruct the patient to close their eyes. Insert a 25 or 27 gauge needle through the skin overlying the infraorbital foramen (**Figure 209-5**). Advance the needle to just beneath the subcutaneous tissue. **Do not enter the infraorbital canal as this may damage the nerve.** Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 1 to 2 mL of local anesthetic solution. Massage the area over the infraorbital foramen for a few seconds to ensure optimal infiltration.

A B

FIGURE 209-5. The extraoral approach to the infraorbital nerve block. ***A.*** Location of the nerve. ***B.*** Insertion of the needle.

A B

C

FIGURE 209-6. The intraoral approach to the infraorbital nerve block. ***A.*** Location of the nerve. ***B.*** Insertion of the needle. ***C.*** Photo. (Used with permission from Hadzic A: *New York School of Regional Anesthesia Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill; 2007.)

Needle insertion and direction (intraoral approach): Clean, prep, and apply a topical anesthetic agent to the mucosa opposite the first maxillary premolar. Place the nondominant index finger over the infraorbital foramen (**Figure 209-6**). Retract the upper lip using the nondominant thumb. Identify the mucobuccal fold above the first premolar. Insert a 25 or 27 gauge needle through the mucobuccal fold. Advance the needle toward the nondominant index finger situated over the infraorbital foramen (**Figures 209-6*B* and 209-6*C***). Stop advancing the needle when the tip is felt beneath the index finger. The estimated depth of penetration of the needle tip is 1.0 to 1.5 cm in an older child or an adult and 0.5 to 1.0 cm in a younger child. Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 1 to 2 mL of local anesthetic solution.

Remarks: **Be careful not to penetrate too deeply when performing the intraoral approach.** The infraorbital venous plexus may be disrupted and result in a hematoma. **The globe may also be accidentally penetrated.** Avoid these complications by positioning the nondominant index finger over the infraorbital foramen and using it to palpate and track the advancing needle tip. The intraoral approach is the preferred technique.

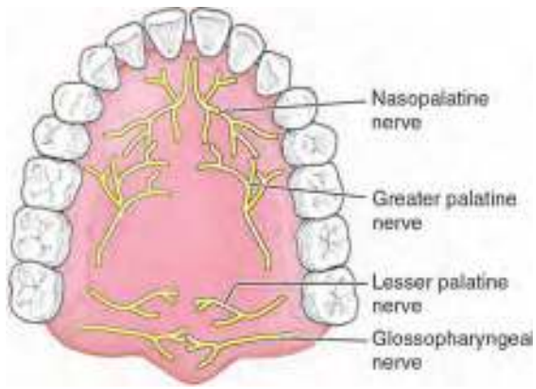
NASOPALATINE NERVE BLOCK

Anatomy: The nasopalatine nerve provides sensory innervation to the anterior one-third of the hard palate (**Figure 209-7*A***). It exits the maxilla via the incisive foramen in the midline and 0.5 cm posterior to the central incisors.

Patient positioning: Place the patient recumbent in a dental chair with their head extended 45°. Alternatively, place the patient supine with a rolled sheet beneath their shoulder blades to assist in neck extension. Instruct the patient to fully open their mouth.

Landmarks: The incisive foramen lies in the midline and approximately 5 mm posterior to the central incisors of the maxilla. Overlying the incisive foramen is the incisive papilla, a soft tissue elevation.

Needle insertion and direction: Clean, prep, and apply a topical anesthetic agent to the mucosa on the anterior one-third of the hard palate. Identify the incisive foramen by first identifying the incisive papilla. Insert a 27 to 30 gauge needle, with the bevel facing the hard palate, from a position immediately lateral to the edge of the incisive papilla (**Figures 209-7*B* and 209-7*C***). Advance the needle 3 to 4 mm toward the midline or until bone is identified. Aspirate to



A

B

C

D

E

FIGURE 209-7. Anesthesia of the palate. ***A.*** Sensory innervation of the palate. ***B.*** Nasopalatine nerve block. ***C.*** Photo. (Used with permission from Hadzic A: *New York School of Regional Anesthesia Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill; 2007.) ***D.*** Greater palatine nerve block. **E.** Photo. (Used with permission from Hadzic A: *New York School of Regional Anesthesia Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill; 2007.)

confirm that the tip of the needle is not within a blood vessel. Inject 0.25 to 0.35 mL of local anesthetic solution. The area surrounding the injection site will blanch upon deposition of the local anesthetic solution.[4,5]

Remarks: This is a particularly painful injection due to the adherent nature of the mucosa to the underlying hard palate. Topical anesthetics will provide adequate preinjection anesthesia. Some clinicians use a cotton-tipped applicator or a blunt instrument to put pressure on the incisive papilla for 30 seconds prior to and during the injection.[5] This seems to defer the attention of the patient and make the injection more bearable. **Be careful not to penetrate too deeply with the needle and enter the incisive foramen.** Insertion into the incisive foramen will cause severe pain. **Injection into the incisive foramen can result in permanent nerve damage.**[4,5] The mucosa of the hard palate receives its blood supply from the hard palate. Injection of more than 0.4 mL will elevate the mucosa from the hard palate and result in mucosal necrosis. This block may be performed to repair lacerations of the mucosa of the anterior hard palate.

GREATER PALATINE NERVE BLOCK

Anatomy: The greater palatine nerve provides sensory innervation to the ipsilateral posterior two-thirds of the hard palate (**Figure 209-7*A***). It enters the oral cavity via the greater palatine foramen. The greater palatine foramen lies between the second and third maxillary molar and approximately 1 cm onto the hard palate.

Patient positioning: Place the patient recumbent in a dental chair with their head extended 45°. Alternatively, place the patient supine with a rolled sheet beneath their shoulder blades to assist in neck extension. Instruct the patient to fully open their mouth.

Landmarks: The greater palatine foramen lies 1 cm medial to the gingival junction of the second and third maxillary molar (**Figure 209-7*D***).

Needle insertion and direction: Clean, prep, and apply a topical anesthetic agent to the hard palate adjacent to the second and third maxillary molars. Insert a 27 to 30 gauge needle 1 cm medial to the junction of the second and third maxillary molars (**Figures 209-7*D* and 209-7*E***). Ensure that the tip of the needle is held at 90° to the curve of the palate. Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 0.25 to 0.35 mL of local anesthetic solution. The area surrounding the injection site will blanch upon deposition of the local anesthetic solution.[4,5]

Remarks: This block may be performed to repair lacerations of the mucosa of the hard palate. The mucosa of the hard palate receives its blood supply from the hard palate. Injection of more than 0.4 mL will elevate the mucosa from the hard palate and result in mucosal necrosis. The position of the lesser palatine foramen is 2 to 4 mm posterior to the greater palatine foramen. The lesser palatine nerve provides sensory innervation to the soft palate and uvula. If anesthetized, as it often is when blocking the greater palatine nerve, the patient may experience a feeling of dysphagia or throat closure. Reassurance is usually adequate to alleviate the patient's anxiety until the anesthesia wears off.

POSTERIOR SUPERIOR ALVEOLAR NERVE BLOCK

Anatomy: The maxillary nerve exits the skull via the foramen rotundum. It then courses anteriorly into the pterygopalatine fossa and divides into its constituent branches. The posterior superior alveolar nerve provides sensory innervation to the maxillary molar teeth and their associated mucosal tissues.

Patient positioning: Place the patient semi-recumbent in a dental chair with their head extended 30°. Alternatively, place the patient sitting upright with their head and back firmly against the examination chair or table and their head extended 30° to 45°. Instruct the patient to fully open their mouth.

Landmarks: Pull the buccal mucosa laterally and identify the inferior-most posterior portion of the zygoma (**Figure 209-8*A***). It lies posterior, lateral, and superior to the third maxillary molar. The pterygomaxillary fissure lies posterior, medial, and superior to the vestibule between the third maxillary molar and the posterior zygoma. The pterygopalatine fossa can be reached by following the pterygomaxillary fissure superiorly and medially.[4,5]

Needle insertion and direction: Clean, prep, and apply a topical anesthetic agent to the recess posterior and lateral to the maxilla. Insert the nondominant index finger between the maxillary molars and the cheek (**Figure 209-8*A***). Palpate the zygomatic process of the maxilla with the index finger. Rotate the index finger 180° so that the pad is against the patient's cheek (**Figure 209-8*B***). Apply outward pressure to move the cheek away from the teeth. Place the needle along the middle of the nail plate of the index finger. Aim the needle and syringe along the index finger (**Figure 209-8*B***). The needle and syringe should be aimed posteriorly, superiorly, and medially (**Figures 209-8*C* and 209-8*D***). Insert and advance the needle 2.5 cm along the index finger. If the needle contacts bone, withdraw the needle completely and direct it more laterally.[4,5] Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 3 mL of local anesthetic solution.

Remarks: Bend the needle 30° at the hub to assist in achieving a medial direction of the needle. **Do not bend the needle more than 30° as the needle may fracture. It is extremely important to never change the direction of a needle once it is inserted.** This is associated with an increased risk of needle breakage requiring an operative procedure to recover the needle segment. **Never force the needle.** The needle is inappropriately positioned if it is meeting resistance. Abort the procedure, reidentify the landmarks, and reattempt the procedure.[4,5] Occasionally, the first molar is only partially anesthetized by this block. Consider supplementation of this block with a supraperiosteal infiltration of the first molar.

MENTAL NERVE BLOCK, INTRAORAL APPROACH

Anatomy: The mental nerve is one of the two terminal divisions of the inferior alveolar nerve. It provides sensory innervation to the ipsilateral skin and mucosa of the lower lip and chin. It exits the bony mandible at the mental foramen.

Patient positioning: Place the patient recumbent in a dental chair. Alternatively, place the patient sitting upright or supine with their head against the examination table and in the neutral position. Instruct the patient to slightly open their mouth.

Landmarks: The mental foramen lies in the same plane as the infraorbital foramen and the mid-pupillary line (**Figure 209-4**). The mental foramen is located approximately 1 cm beneath the gum line between the first and second premolar.

Needle insertion and direction: Clean, prep, and apply a topical anesthetic agent to the oral mucosa overlying the mental foramen. Grasp the lower lip with the nondominant hand. Pull it outward and downward (**Figures 209-9*A* and 209-9*B***). Insert a 27 gauge needle into the mucobuccal fold between the first and second premolar (**Figures 209-9*A* and 209-9*B***). Advance the needle medially until it contacts the mandible. Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 1.5 to 2.0 mL of local anesthetic solution.

Remarks: The mental nerve block, as the infraorbital nerve block, has an intraoral and an extraoral approach. The extraoral approach will not be discussed as it is more painful and there is no benefit to

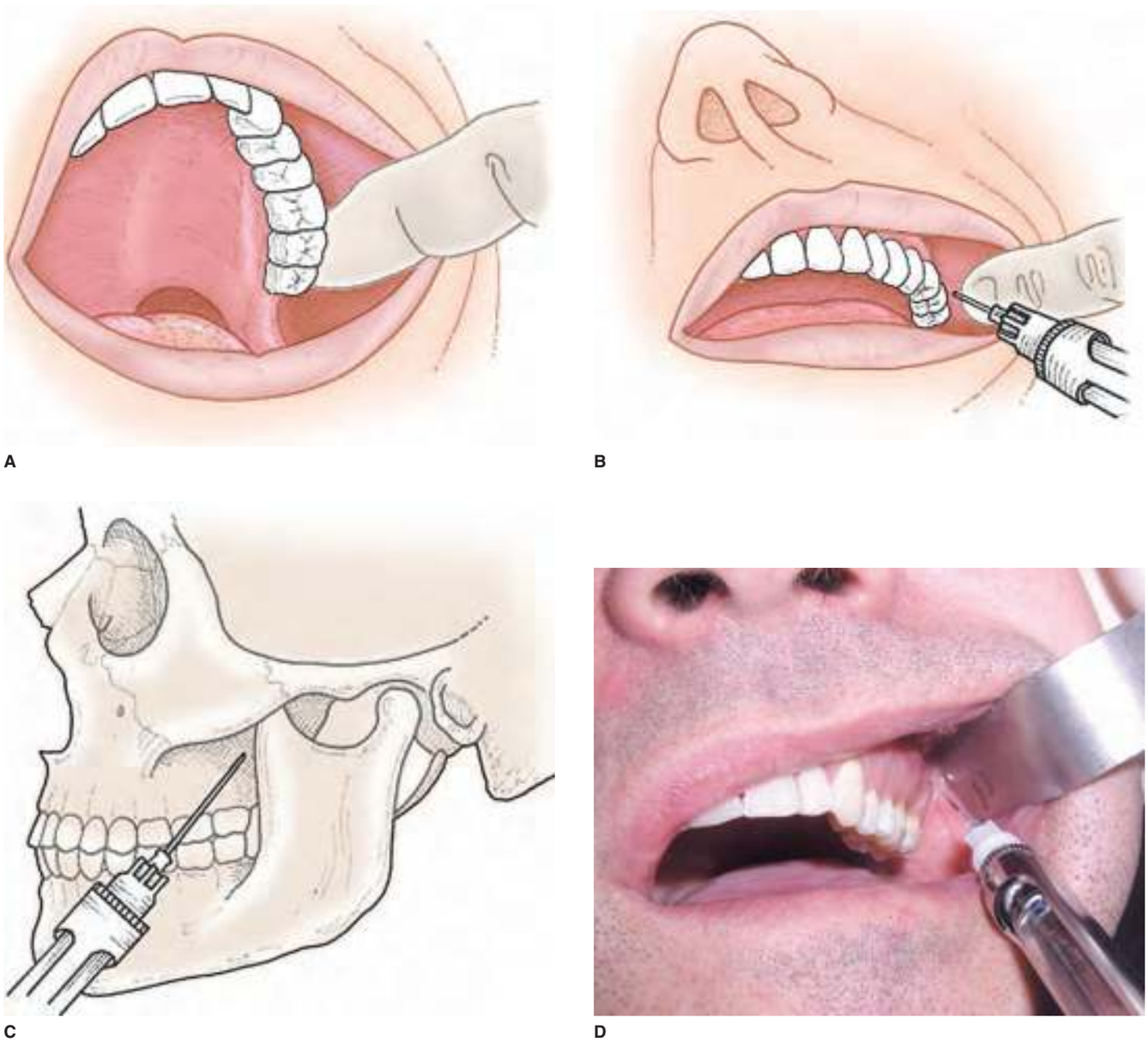


FIGURE 209-8. The posterior superior alveolar nerve block. ***A.*** The nondominant index finger is inserted and positioned. ***B.*** The cheek is retracted. ***C.*** The proper direction for insertion and advancement of the needle. ***D.*** Photo. (Used with permission from Hadzic A: *New York School of Regional Anesthesia Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill; 2007.)

its use over the intraoral approach. A description of the extraoral approach to the mental nerve block is found in Chapter 156. A near midline lower lip or chin injury may necessitate bilateral mental nerve blockade due to the midline crossover from each of the mental nerves.[4]

BUCCAL NERVE BLOCK

Anatomy: The buccal nerve is one of the main branches of the mandibular nerve. It travels down the medial aspect of the ramus of the mandible anterior to the inferior alveolar neurovascular bundle. It crosses from the medial mandible into the soft tissue of the cheek at the level of the occlusive plane. It supplies the sensory innervation to the mucous membrane of the cheek and vestibule.[1] It innervates, to a variable degree, a small patch of skin over the cheek.

Patient positioning: Place the patient recumbent in a dental chair with their head extended 30°. Alternatively, place the patient sitting with their head and back firmly against an examination chair or upright table with their head extended 30° to 45°. Instruct the patient to fully open their mouth.

Landmarks: Visually identify the third mandibular molar. Palpate the anterior border of the ramus of the mandible. The buccal nerve traverses the anterior border of the ramus of the mandible, posterior and slightly lateral to the third molar at the level of the occlusive plane.

Needle insertion and direction: Clean, prep, and apply a topical anesthetic agent to the oral mucosa over the anterolateral border of the ramus of the mandible. Place the thumb of the nondominant hand on the inner surface of the cheek. Pull the cheek outward (**Figures 209-10*A* and 209-10*B***). Insert a 27 gauge needle 1 mm lateral to the anterior border of the ramus of the mandible and at

A

B

FIGURE 209-9. The intraoral approach to the mental nerve block. ***A.*** Artist illustration. ***B.*** Photo. (Used with permission from Hadzic A: *New York School of Regional Anesthesia Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill; 2007.)

A

B

FIGURE 209-10. The buccal nerve block. ***A.*** Artist illustration. ***B.*** Photo. (Used with permission from Hadzic A: *New York School of Regional Anesthesia Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill; 2007.)

the level of the occlusal plane (**Figures 209-10*A* and 209-10*B***). Advance the needle 3 to 4 mm into the soft tissues. Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 2 mL of local anesthetic solution.

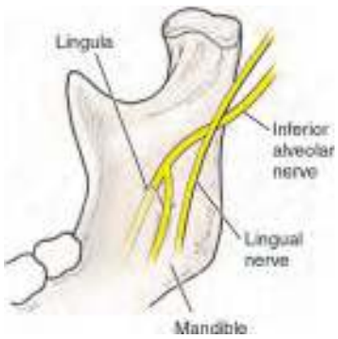
Remarks: Buccal nerve blocks are used when extensive intraoral manipulation is anticipated, when buccal manipulation or repair is required, or for the incision and drainage of an abscess. It provides additional patient comfort. The block is nearly always performed as an adjunct to an inferior alveolar, maxillary, or posterior superior alveolar nerve block.[5]

INFERIOR ALVEOLAR NERVE BLOCK

Anatomy: The lingual and inferior alveolar nerves are two of four branches of the mandibular nerve. The nerves initially travel together and inferiorly on the medial side of the mandibular ramus (**Figure 209-11*A***). The lingula is a palpable bony landmark immediately anterior to the mandibular foramen. The inferior alveolar nerve courses posterior to the lingula and enters the mandibular canal via the mandibular foramen. It continues to travel anteriorly within the mandible to provide sensory innervation to the body of the mandible, the mandibular teeth, and the overlying oral mucosa. One of the terminal branches of the inferior alveolar nerve is the mental nerve. The inferior alveolar nerve may be blocked by the classic open-mouth approach or the closed-mouth approach.

Patient positioning: Place the patient in a dental chair with their head neutral such that the occlusive surface is parallel to the floor. Alternatively, place the patient sitting upright in an examination chair or on a gurney with their head positioned firmly against the back of the gurney or chair. Instruct the patient to fully open their mouth. Perform the open-mouth approach if the patient can fully open their mouth. Perform the closed-mouth approach if the patient has trismus or cannot fully open their mouth.

Landmarks: Identify by palpation the anterior border of the ramus of the mandible within the mouth, the coronoid notch within the mouth, and the posterior border of the ramus of the mandible externally (**Figure 209-11*B***). Approximately equidistant from these two points lie the lingual and the inferior alveolar nerves. Palpate



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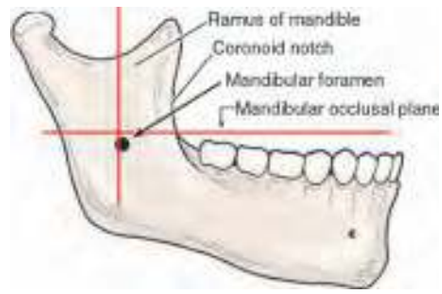

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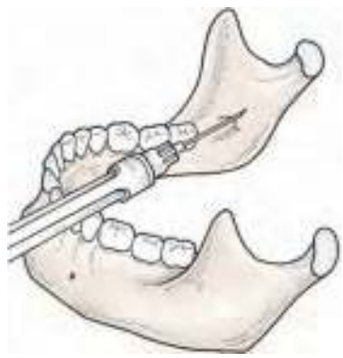

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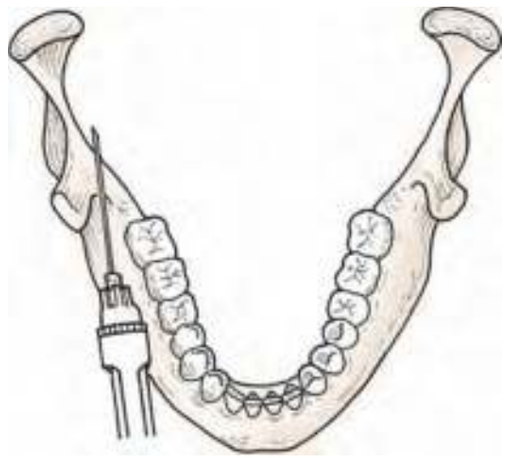

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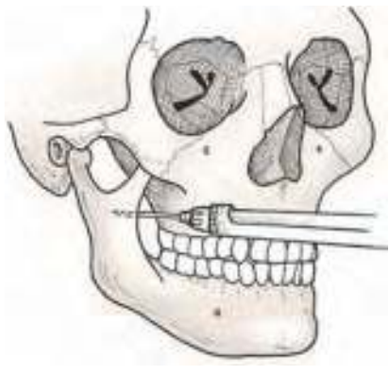


G

FIGURE 209-11. The inferior alveolar nerve block. ***A.*** The course of the inferior alveolar nerve and the lingual nerve along the ramus of the mandible. ***B.*** The anatomy of the external surface of the mandible. ***C.*** Positioning for the open-mouth approach. ***D.*** Proper needle insertion and direction for the open-mouth approach. ***E.*** Photo. (Used with permission from Hadzic A: *New York School of Regional Anesthesia Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill; 2007.) ***F.*** Superior view of the closed-mouth approach demonstrating the proper needle direction. ***G.*** The closed-mouth approach.

the lingula of the ramus of the mandible. It is a bony projection on the medial surface of the ramus of the mandible and 1 cm above the occlusive plane.

Needle insertion and direction (open-mouth approach): Clean, prep, and apply a topical anesthetic agent to the inner surface of the ramus of the mandible. Stand opposite the side to be blocked. Place the thumb of the nondominant hand on the anterior border of the ramus of the mandible. Move the thumb posteromedially to identify the lingula. Place the index finger of the nondominant hand against the extraoral border of the mandibular ramus just above the angle of the mandible. Grasp the ramus between the thumb and the forefinger (**Figure 209-11*C***). Pull the cheek outward using the nondominant thumb as a lever. Place a 27 gauge, 2 inch needle on a 3 mL syringe that contains local anesthetic solution. A 5 mL syringe is too large for this approach. A syringe smaller than 3 mL will not carry enough anesthetic.

Introduce the needle from the opposite side (**Figures 209-11*D* and 209-11*E***). Align the tip of the needle toward the lingula with the barrel of the syringe between the contralateral first and second premolars (**Figures 209-11*D* and 209-11*E***). Hold the syringe parallel to the occlusal plane and 3 to 4 mm above the premolars. Insert the needle into the oral mucosa just superior and posterior to the lingula. Advance the needle until the tip contacts the ramus of the mandible. Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 2 mL of local anesthetic solution.

The above technique is optimal if the operator is right-handed and a right-sided inferior alveolar block is attempted. If the operator is right-handed and attempting a left-sided inferior alveolar nerve block, it is still necessary to stand opposite the side to be anesthetized with the syringe in the dominant hand. Place the nondominant arm over and around the patient's head so that the thumb of the nondominant hand can contact the anterior border of the mandibular ramus and the index finger can grasp the posterior border above the angle of the mandible. The remainder of the technique is the same.

Needle insertion and direction (closed-mouth approach): This method can be used when the patient cannot fully open their mouth due to an abscess, edema, mandible fractures, trismus, or if the mandible is wired closed to the maxilla. This approach deposits the local anesthetic solution superior to the site of the classic open-mouth approach. The local anesthetic solution will descend due to gravity to bathe the inferior alveolar nerve and provide adequate anesthesia.

Place the nondominant thumb on the inner surface of the cheek. Pull the cheek outward. Place a 27 gauge needle on a 3 mL syringe that contains local anesthetic solution. Place the needle and syringe parallel to the occlusal plane and aligned along the junction of the maxillary molars and their gingiva (**Figure 209-11*F***). Direct the needle just medial to the ramus of the mandible (**Figures 209-11*F* and 209-11*G***). Advance the needle 3 cm through the mucosa. Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 2 mL of local anesthetic solution.

Remarks: **It is crucial that the tip of the needle contacts the mandible in the open-mouth approach.** The needle is usually advanced in the tissue 0.5 to 1 cm before the mandible is encountered. The needle is most likely inappropriately placed and deposition of anesthesia will not produce the desired results if the mandible is not encountered. Remove the needle, reidentify the appropriate anatomic landmarks, and reattempt the procedure if the mandible is not encountered. The buccal, inferior alveolar, and lingual nerves must be blocked on one side to achieve complete anesthesia of the hemimandible. Facial nerve paralysis can occur if the needle is inserted too far posterior and enters the capsule of the parotid gland. This paralysis is usually transient and resolves as the anesthetic wears off.

LINGUAL NERVE BLOCK

Anatomy: The lingual nerve is a branch of the mandibular division of the trigeminal nerve. It travels with the inferior alveolar nerve until the inferior alveolar nerve enters the mandible. The lingual nerve leaves the medial aspect of the mandibular ramus and penetrates the posterior tongue at the level of the occlusive plane just medial to the third mandibular premolar. It courses anteriorly to provide sensory innervation to the anterior two-thirds of the tongue, the floor of the mouth, and the lingual mucous membrane.[1]

Patient positioning: Place the patient in a dental chair with their head neutral such that the occlusive surface is parallel to the floor. Alternatively, place the patient sitting upright in an examination chair or on a gurney with their head firmly against the back of the gurney or chair. Instruct the patient to fully open their mouth.

Landmarks: Identify the lingual side of the second mandibular molar. The injection site is 1 cm medial to the second mandibular molar.

Needle insertion and direction: Approach the patient from the contralateral side. Move the tongue upward or toward the contralateral side with a tongue blade. Insert a 27 gauge, 2 inch needle into the mucosa 1 cm medial to the second mandibular premolar (**Figures 209-12*A* and 209-12*B***). Advance the needle posteriorly 1 cm. Aspirate to confirm that the tip of the needle is not within a blood vessel. Inject 1.0 to 1.5 mL of local anesthetic solution.

Remarks: The inferior alveolar nerve and the lingual nerve can be, and usually are, blocked simultaneously during an inferior alveolar nerve block. The lingual nerve, however, can be blocked in an isolated fashion. Perform an isolated lingual nerve block only when the initial combined block has failed or for isolated tongue lacerations.[1,5] This is an optimal block for tongue laceration repair. However, bilateral lingual nerve blocks may be necessary.

ASSESSMENT

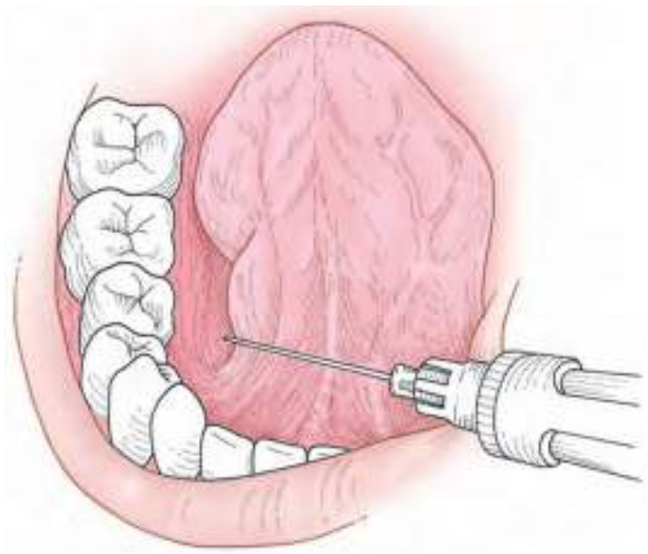
Anesthesia is usually achieved within 5 minutes of the injection. However, depending upon the particular injection and the local anesthetic used, anesthesia can be achieved anywhere from 20 seconds to 10 minutes. The block was properly performed if the patient experiences anesthesia. Repeat the block if anesthesia is not achieved by 10 minutes.

AFTERCARE

The aftercare of dental anesthesia is minimal. Reexamine the area of the local anesthetic injection before the patient is discharged to ensure that a hematoma has not developed. **Instruct the patient to use caution as there is no sensation in the area anesthetized.** Encourage them to refrain from meals, chewing gum, hot beverages, aggravated scratching, placing foreign bodies in the mouth, or anything that may cause injury to the anesthetized area. Parents must be informed to discourage children from testing the anesthetized area by biting or chewing. Many Dentists and Physicians place a cotton roll, or rolled 2×2 gauze, between the area anesthetized and the teeth to provide added protection from a self-inflicted bite injury.[4,5]

COMPLICATIONS

Any patient receiving dental anesthesia has the potential to develop complications. The key is to have knowledge of these complications and be prepared to deal with them should they occur. Most severe complications will declare themselves in a rapid fashion. These severe complications include intravascular injection of local anesthetic, allergic reactions to the local anesthetic, cardiovascular toxicity, neurologic toxicity, seizures, and unintentional overdosage.



A

B

FIGURE 209-12. The lingual nerve block. ***A.*** Artist illustration. ***B.*** Photo. (Used with permission from Hadzic A: *New York School of Regional Anesthesia Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill; 2007.)

Allergic reactions can occur from the latex vial/carpule seal or the preservative in the local anesthetic.[9] These complications can be avoided by aspiration before injection, obtaining an appropriate history of prior anesthesia, and cautious calculation of anesthetic dosages, respectively.[4,5,8]

Late complications of dental anesthesia include hematomas, neuropathy, infection, and trismus.[4,5] Hematomas after dental anesthesia occur after the inadvertent puncture of an artery or vein. Arterial hematomas enlarge more rapidly and are usually more painful than venous hematomas. They can cause a significant facial deformity. Hematomas following dental anesthesia are usually of little significance, require no intervention, and resolve spontaneously with time. A proper preprocedural history should determine if the patient is using any anticoagulants or blood thinners, has a real or potential bleeding disorder, or had adverse bleeding complications from previous procedures. Treatment initially involves applying cold packs. Heat in the form of an externally placed heating pad should be used to help disintegrate the clot after 24 hours.[4,5]

A peripheral neuropathy can occur in the form of prolonged anesthesia, paresthesias (i.e., burning or itching sensation), hyperesthesias (i.e., increased sensitivity to noxious stimuli), and dysesthesias (i.e., painful sensation to non-noxious stimuli). This may be a result of direct damage to the nerve from traumatic needle insertion, hemorrhage or hematoma within the nerve sheath, deposition of local anesthetic into a foramen or canal causing pressure injury to the nerve, or chemical injury to the nerve.[10,11] Reduce the risk of causing a neuropathy by avoiding injections into a foramen, using a small gauge needle, and withdrawing the needle slightly before injection if the patient should feel a shock or paresthesias with needle insertion.[4,5] While many nerve injuries are temporary and will resolve with time, some can be permanent.[11]

Infection due to dental anesthesia is rare but does exist. Follow several simple measures to minimize potential infections. Never inject through an infected area. Doing so may carry bacteria through facial planes into deeper compartments. Refrain from multiple injections (i.e., needle misadventures) as this increases the risk of iatrogenic infection. This is easily resolved by appropriately identifying the anatomic landmarks prior to needle insertion.

Needle breakage can occur as a result of manufacturing defects or operator imprudence. **To prevent needle breakage, never exert force against resistance.** Resistance signifies that the tip of the needle is against bone or a tooth. Withdraw the needle and reattempt insertion if this occurs. **Never advance the needle to the hub.** The greatest percentage of needle breaks occur when the needle is inserted to the hub.[4,5] **Never redirect the needle after it is inserted through the skin or mucosa.** This puts abnormal force on the needle and potentiates breakage. The needle may inadvertently be in an undesired anatomic location. Withdraw the needle completely, identify the appropriate anatomic landmarks, and reinsert the needle. Do not bend the needle as this can weaken it and make it more prone to breakage. If a needle breaks, grasp the fragment with a forceps or hemostat and remove it from the soft tissue. If the needle fragment is not palpable with an instrument, consult a Dentist or Oral Surgeon for possible removal. The consultant may choose to see the patient in the Emergency Department, see the patient in their office within 24 to 48 hours, or leave the needle fragment in place and not remove it.

SUMMARY

Dental anesthetic techniques are easy to learn, simple to perform, and effective in providing temporary pain relief. The required equipment is readily available in every Emergency Department. Consider using local anesthetic agents that contain epinephrine to provide significantly longer analgesia than those without epinephrine. A rapidly performed local anesthetic injection goes a long way toward patient satisfaction.

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210 Dental Abscess Incision and Drainage

Richard Dean Robinson and Peter S. Kim

INTRODUCTION

Patients frequently present to the Emergency Department complaining of a "toothache." There are multiple common causes of toothache pain and similarly multiple etiologies for a dental abscess (**Table 210-1**).[1] Distinguishing the type of dental abscess can have an impact on the treatment decisions, prognosis, and patient morbidity.[2-5] Accurate diagnosis and proper treatment of these maladies requires that the Emergency Physician have a basic understanding of dental anatomy, pathophysiology, and simple dental treatment protocols. There are many complications of odontogenic infections (**Table 210-2**).[6-8] Many of these conditions can be managed initially though the Emergency Department. The prudent Emergency Physician must have a clear understanding that these infections can rapidly become complicated requiring timely consultation or referral.

ANATOMY AND PATHOPHYSIOLOGY

Teeth are essentially composed of three layers: the outermost enamel, underlying dentin, and the pulp which is the deepest layer (**Figure 210-1**). The dentin and pulp are living tissues that are sensitive to noxious stimuli. The crown is covered with enamel while the root is covered with a substance known as cementum. Cementum helps attach the tooth to the surrounding alveolar bone via the periodontal ligament (PDL). The neurovascular supply enters the pulp through the apical foramen at the root apex. The pulp contains only pain transmitting neuronal fibers while the PDL contains both pain-sensitive and pressure-sensitive fibers.[9] Anatomic structures in the body limit and direct the spread of infection in a predictable way. These structures include fascial layers, ligaments, tendons, muscles, and bone.[10]

Dental abscesses arise when bacteria penetrate the normal anatomic and physiologic barriers of the tooth and surrounding structures. This can lead to a localized collection of purulence contained within the tooth (i.e., a pulp abscess) or around the apex of the tooth (i.e., a periapical abscess) (**Figure 210-2**). Alternatively, a dental abscess may localize to the supporting structures of the tooth (i.e., a periodontal abscess) or strictly to the adjacent soft tissues (i.e., a pericoronitis) (**Figure 210-2**).

TABLE 210-1 Common Etiologies for a Dental Abscess

Cysts that become infected
Gingival infections
Mixed periodontal/periapical infections
Periapical infections
Periodontal infections
Postoperative infections
Root fracture that becomes infected

TABLE 210-2 Complications of Odontogenic Infections[6-8]

Acute respiratory distress syndrome	Lemierre's syndrome
Aspiration pneumonia	Mediastinitis
Brain abscess	Necrotizing fasciitis
Carotid sheath involvement	Orbital abscess
Cavernous sinus thrombosis	Pericarditis
Cervical spine osteomyelitis	Pleuropulmonary suppuration
Coagulation problems	Pneumothorax
Disseminated intravascular coagulation	Respiratory obstruction
Endocarditis	Sepsis
Facial abscess	Spondylitis
Hematogenous dissemination	Systemic immune response syndrome
Internal jugular thrombophlebitis	Thoracic empyema
Jaw osteomyelitis	

PULPAL OR PERIAPICAL ABSCESSES

Dental abscesses often arise from pulp necrosis secondary to dental caries or a defective restoration.[1,3,4,7-9,11,12] Dental caries are commonly known as dental decay or "cavities." This process represents direct destruction of the tooth substance by acidic bacterial products of normal oral flora. A carious tooth may not initially be painful. However, as bacterial inflammatory products invade the dental pulp during disease progression, the tooth will consequently become sensitive.[1-3,9,13,14] This is known as pulpitis which typically presents with nonlocalizable and intermittent symptoms. This process may initially be reversible by routine dental treatment (e.g., a filling) but the pulp will rapidly necrose and die if it becomes infected. If the infectious process is allowed to continue, products from the necrotic pulp may escape the confines of the tooth via the apical foramen and begin to involve the PDL and surrounding alveolar bone. This is known as a periapical abscess and makes the infected tooth easily localizable with intermittent or constant sensitivity, and/

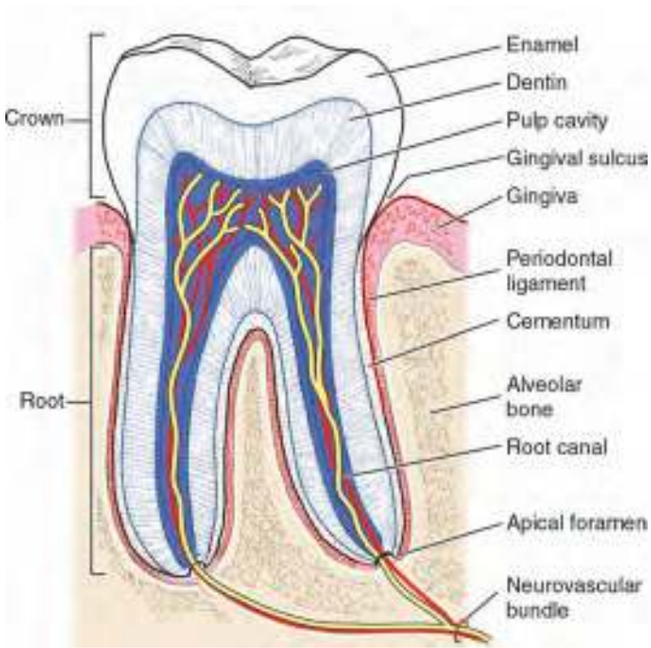

FIGURE 210-1. The anatomy of a tooth.

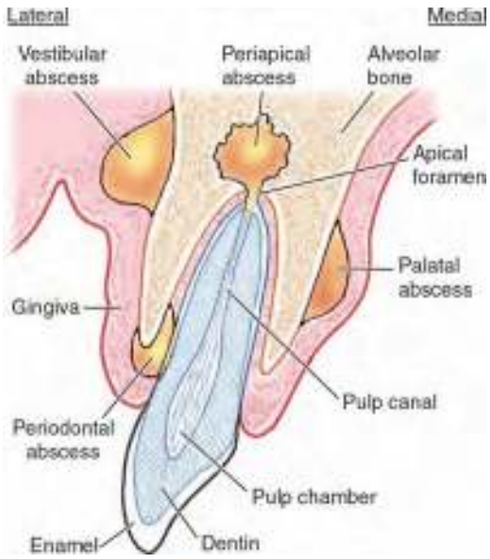

FIGURE 210-2. Locations of common dental abscesses.

or tenderness to gentle percussion (**Figure 210-3**).[1-3,5,9,11,14] At this stage of the disease process, a root canal would be indicated which is essentially an incision and drainage procedure performed on the inside of a tooth by an Endodontist.[9,11]

If left unchecked, however, the bacterial products of a periapical infection and the host's immune response to it can lead to progressive destruction of dental supporting tissues including the alveolar bone. At this point the tooth will become increasingly mobile. The infection will follow the path of least resistance as it penetrates through the alveolar bone into the surrounding soft tissues.[9,11] It may perforate laterally to form a vestibular abscess (**Figures 210-2 and 210-4*A***), or alternatively, it may perforate medially to form a palatal or lingual abscess (**Figures 210-2 and 210-4*B***). Further spread will be dictated by the proximity of muscle attachments and fascial planes.[3,5,9,11,14-16] The appropriate treatment for an abscessed tooth depends on the extent of the infection. It may include endodontic treatment, incision and drainage, extraction, or a combination of these.[9,11] An incision and drainage procedure is warranted if the infectious process extends outside the alveolar bone and involves proximal soft tissues.[5,9,11,14,15]

The extension of infectious products beyond the root apex can lead to a multitude of clinical signs and symptoms involving the

A

B

C

FIGURE 210-3. Periapical abscess. ***A.*** A patient with an abscess. (Used from www.commons.wikimedia.com.) ***B.*** Sinus tract opening lateral to the maxillary teeth. ***C.*** Tooth x-ray with well-defined radiolucent area. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. New York: McGraw-Hill; 2016. Part B: Photo contributor: Alan B. Storrow, MD. Part C: Photo contributor: James L. Kretzschmar, DDS, MBS.)

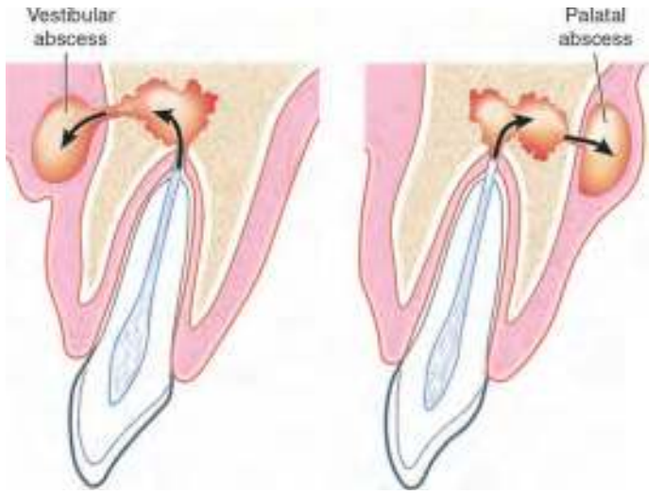

FIGURE 210-4. The spread of dental abscess. A pulpal abscess progresses to a periapical abscess that perforates the alveolar plate. ***A.*** Labial or buccal perforation leads to a vestibular abscess. ***B.*** Palatal or lingual perforation leads to a palatal or lingual abscess, respectively.

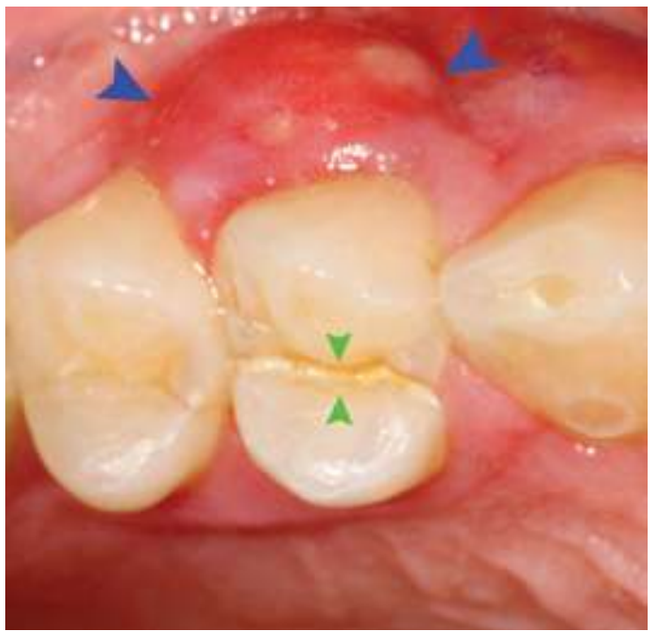

FIGURE 210-5. A cracked tooth (*green arrowheads*) led to the lateral periodontal abscess (*blue arrowheads*). (Used from www.commons.wikimedia.org/wiki/Category:Periodontal_abscess#/media/File:Cracked_tooth_lateral_periodontal_abscess.jpg).

head and neck. Swelling, erythema, warmth, fluctuance, and spontaneous drainage of purulence via a sinus tract or fistula may be seen intraorally or extraorally (**Figure 210-3*B***). A localized or generalized cellulitis may be present. There is often a foul breath odor.[1,5,11,15] One or more of the numerous fascial spaces of the head and neck may become involved via direct extension.[3,5,9,11,13,15-21] Systemic symptoms may occur including fever, malaise, anorexia, and leukocytosis.[5,6,11,13,15,17] Structures immediately adjacent to the oral cavity may also become involved. This can result in complications such as trismus, reactive sinusitis, lymphadenopathy, osteomyelitis, cavernous sinus thrombosis, airway compromise, and/or a brain abscess.[3,6,16-21]

Dental infections extending into any of the fascial compartments of the head and neck are dangerous, can rapidly progress, and are considered complicated infections.[3,5,6,9,11,15,17-21] The classic example is Ludwig's angina, a potentially life-threatening cellulitis involving the sublingual, submental, and submandibular spaces bilaterally. **This represents an immediate threat to the airway.** Patients typically will appear toxic with systemic signs and symptoms. Classically, patients will present with an elevation of the mouth, submandibular swelling, dysphagia, and voice changes.[6,10,16,18,20,21] Infectious extensions into the lateral pharyngeal, retropharyngeal, and prevertebral spaces are rare and can lead to disastrous complications such as aspiration, mediastinitis, and airway compromise. A retropharyngeal space measuring greater than 3 to 5 mm on lateral soft tissue neck radiographs or computed tomography (CT) scans is indicative of airway compromise.[15] **Consider rapid and aggressive management with prompt surgical consultation, broad-spectrum antibiotics, and early airway intervention in patients who are suspected of manifesting any of these processes.[3,13,15-20]**

PERIODONTAL ABSCESSES

Poor dental hygiene and poor nutrition lead to local inflammation of the tissues surrounding and attaching the tooth to its socket allowing bacterial penetration.[7,8,12] Early periodontal disease is isolated to the gingiva and is known as gingivitis.[5,22] Alveolar bone may be destroyed as the disease progresses leading to gingival pockets and tooth mobility.[1,5,23] Food debris or plaque may become trapped within these pockets and create a localized infection known as a periodontal abscess (**Figure 210-5**).[4,5,22] Periodontal disease is very common in pregnant women.

Patients may complain of bleeding, foul oral odor, bad taste, loose teeth, pain, or swelling. The physical examination will reveal gingival tissue that may be erythematous or necrotic and bleed easily. Heavy accretions of dental plaque and calculi may be present. An abscess may present as a focal swelling, tooth mobility, pain on percussion, and/or purulence that is expressible from the gingival sulcus.[1,5,22,23] It may be impossible with current dental caries to differentiate a periapical abscess from a periodontal abscess without radiographs.[3,5] In fact, both lesions may occur simultaneously.[23] Periodontal disease may be sufficiently advanced such that teeth may be so mobile they cannot be salvaged and require extraction for definitive therapy.[5]

True periodontal abscesses rarely spread beyond the local dentoalveolar structure. If present, urgent referral may be required.[3,4] Treat an isolated periodontal abscess with dental anesthesia, incision and drainage, and dilute peroxide (e.g., a 1:5 solution or 5%) or chlorhexidine rinses. Prescribe oral antibiotics for advanced disease evidenced by spread to adjacent tissues, if there is anticipated delay to definitive care, and/or for systemic manifestations.[1,3-5,23] Appropriate antibiotic options include penicillin, clindamycin, or erythromycin. Refer patients with these lesions to a Dentist within 24 to 48 hours for definitive care and to avoid recurrence of disease.[1,3,5]

PERICORONITIS

Inflammation can occur around the crown of any erupting tooth and is common around impacted teeth, especially the third molars.[1,3,4,7,8,12,24] This condition is known as pericoronitis (**Figure 210-6**). It is often exacerbated by impaction of food under soft tissues. Progression of the primary process or overzealous treatment can easily lead to extension of the infection posteriorly to multiple contiguous spaces, including the retropharyngeal space.[1,3,24,25] Simple cases are easily managed in the Emergency Department. Always maintain a very low threshold for consultation and referral of these patients with complicated presentations.

FIGURE 210-6. Soft tissue (*green arrowhead*) over the partially erupted left lower third molar with inflammation and pus is known as a pericoronitis. (Used from www.commons.wikimedia.org/wiki/Category:Pericoronitis#/media/File:38_pericoronitis_with_pus.jpg).

Treatment of pericoronitis may include dental anesthesia, direct saline irrigation, warm salt water rinses, dilute peroxide or chlorhexidine rinses, and oral analgesics. The presence of swelling, trismus, and inflammation may be severe enough to warrant a course of oral antibiotics.[1] Some authors advocate antibiotic coverage in essentially all cases.[1,15,24] Definitive treatment requires the completed eruption or extraction of the tooth. Refer the patient to a Dentist or Oral Surgeon for definitive care within 24 to 48 hours.

RADIOLOGIC EVALUATION

Odontogenic infections often require a radiologic evaluation. This may include mandibular plain films, a Panorex film, an ultrasound, and a CT scan. This is because the clinical examination does not always lead to a definitive diagnosis. Panorex x-rays, if available, are traditionally used to evaluate the teeth. This is time-consuming because the order must be written, the order must be put in the order system, the patient needs to be transported to the Radiology Department for the study, the patient needs transportation back to the Emergency Department, and the Radiologist needs to read the film. Mandibular films or a CT scan will work if a Panorex unit is not available, with the same time-consuming process. In addition, there is the issue of ionizing radiation. Bedside ultrasonography has been used to determine if an abscess is present and to perform an incision and drainage.[26] This is an accurate, inexpensive, and quick technique that does not use ionizing radiation.

INDICATIONS

Incision and drainage of a periapical or periodontal abscess is indicated when there is clinical evidence of alveolar penetration and soft tissue spread.[14,15] Incision and drainage achieves several key objectives including symptomatic relief via decompression, creation of a portal for irrigation, means of obtaining a sample for culture, and exposure of anaerobic bacteria to air thus inhibiting growth.[10] Some authors recommend incision and drainage for purely cellulitic processes.[4,5,16] Standard recommendation is to undergo incision and drainage sooner rather than later as progression of abscess development increases the incidence of complications and morbidity.[27] **Extraoral incision and drainage is indicated only for dental infections progressing toward inevitable spontaneous extraoral drainage.[3,15] Drain all other dental abscesses intraorally.**

CONTRAINDICATIONS

A review of the current literature reveals no direct contraindications to the incision and drainage of a dental abscess. Maintain a low threshold for consultation and referral to a surgical specialist for any patient with rapidly progressing infections, dyspnea, dysphagia, fascial space involvement, a temperature greater than 101°F (38.3°C), white blood cells >10,000, severe trismus manifested by mandibular opening less than 10 mm or the inability to adequately visualize the hypopharynx, a toxic appearance, compromised host defenses, or children.[3,11,15,28]

EQUIPMENT

- #15 scalpel blade on a handle
- #11 scalpel blade on a handle
- Povidone iodine or chlorhexidine solution
- Minnesota retractor
- Weider tongue retractor
- Frazier suction catheter
- Suction source and tubing
- Needle driver
- Hemostat
- Suture, 4–0 and 5–0 silk
- Gelfoam
- Penrose drain, 0.25 inch wide
- Light source, overhead or headlamp

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain dental radiographs if the infection is from a dental source.[5,11,15] Obtain an informed consent for the procedure. Provide adequate anesthesia, ideally in the form of a dental block. Obtain a separate informed consent for the dental block. **Direct infiltration into the area of purulence does not achieve adequate anesthesia and risks spreading the infection by inoculation.[3,5]** Dental blocks may require augmentation with direct infiltration anterior and posterior to the abscess. Anesthetize the areas adjacent to the abscess last to avoid seeding. Refer to Chapter 209 for a complete discussion of dental analgesia and anesthesia. The application of procedural sedation (Chapter 159) may be required if adequate local anesthesia is not possible.[15]

Prophylaxis to prevent infective bacterial endocarditis should be addressed in appropriate patients prior to incision and drainage of a dental abscess in accordance with the policy put forth by the American Dental Association. Further information and specific antibiotic regimens can readily be found on the websites of the American Heart Association and American Dental Association (**Table 210-3**).[29,30]

TECHNIQUES

SIMPLE INTRAORAL INCISION AND DRAINAGE

There are two techniques for the intraoral incision and drainage. The first technique is to make a simple stab incision with a #11 scalpel blade in the area of greatest fluctuance and in the

TABLE 210-3 Prophylactic Regimens for Endocarditis in Patients Undergoing Dental or Oral Surgical Procedures

Situation	Antibiotic choice***	Adult dose	Pediatric dose**
Standard general prophylaxis and able to take oral medications	Amoxicillin	2000 mg PO	50 mg/kg PO
Standard general prophylaxis and unable to take oral medications	Ampicillin Or Cefazolin Or Ceftriaxone	2000 mg IV or IM 1000 mg IV or IM 1000 mg IV or IM	50 mg/kg IV or IM 50 mg/kg IV or IM 50 mg/kg IV or IM
Penicillin-allergic patient able to take oral medications	Clindamycin Or Cephalexin or cefadroxil* Or Azithromycin or clarithromycin	600 mg PO 2000 mg PO 500 mg PO	20 mg/kg PO 50 mg/kg PO 15 mg/kg PO
Penicillin-allergic patient unable to take oral medications	Clindamycin Or Cefazolin* Or Ceftriaxone*	600 mg IV or IM 1000 mg IV or IM 1000 mg IV or IM	20 mg/kg IV or IM 50 mg/kg IV or IM 50 mg/kg IV or IM

IM, intramuscular; IV, intravenous; PO, oral.
*Administer the antibiotics 30 to 60 minutes prior to the procedure.
**The pediatric weight-based dose should not exceed the adult dose.
***Do not use cephalosporins in a patient with a history of a penicillin allergy and any of the following: anaphylaxis, angioedema, respiratory difficulties, urticaria, or unknown reactions.

area that best facilitates dependent drainage (**Figure 210-7*A***). **Do not make the incision more than 1 cm in length.** Gently insert a closed curved hemostat into the incision (**Figure 210-7*B***). Gently spread the jaws of the hemostat in several different directions to break up any loculations (**Figure 210-7*C***). Express and suction any remaining purulence. Insert a sterile rubber drain cut from a 0.25 inch wide Penrose drain or from a sterile surgical glove (**Figure 210-7*D***). **Place one to two silk sutures though the drain and the oral soft tissues. This will ensure that the drain does not fall out and result in premature closure of the incision and/or aspiration of the drain.**

The second technique for simple intraoral incision and drainage differs only in the location of the incision. Make an incision with a #15 scalpel blade at the alveolar crest within the gingival sulcus, scalloping around the teeth. Extend the incision one tooth medial and distal to the tooth in question or the area of the abscess. Gently elevate the attached gingiva from the bone with a blunt instrument. Continue the blunt dissection until the abscess cavity is penetrated. This typically occurs just within the level of unattached gingiva. The remainder of the procedure is as described above. This technique affords some mechanical advantages to the operator and is well tolerated by the patient postoperatively. It can be performed in both arches and in the buccal, palatal, and/or lingual directions. It is often useful for draining a periodontal abscess.

EXTRAORAL INCISION AND DRAINAGE

Extraoral incision and drainage is indicated when a dental infection appears to be progressing toward inevitable spontaneous extraoral drainage.[15] This procedure requires more attention and skill because of the numerous underlying vital structures and the possible cosmetic consequences.[3,15] Make every effort to avoid extraoral incision and drainage.[3] Consider consulting a Dentist or Oral Surgeon before performing an extraoral incision and drainage.

Extraoral incision and drainage differs from the intraoral approach.[3,15,16] **Advise the patient that there will almost certainly be a visible scar after the incision heals.[3,15]** Extraoral incision and drainage requires the use of sterile technique. Prepare the area with povidone iodine or chlorhexidine solution and allow it to dry. Apply sterile drapes to isolate a field. Perform local subcutaneous infiltrative anesthesia of the skin. Use a separate needle and syringe for any intraoral anesthetic techniques.

Make a 1.0 to 1.5 cm long incision with a #15 scalpel blade in an area of healthy skin proximal to the site of expected breakdown and in a location that facilitates dependent drainage (**Figure 210-8**). Extend the incision into the subcutaneous tissues. This is another critical difference from the intraoral technique. Insert a hemostat to penetrate and drain the abscess cavity. Spread the hemostat in several directions to break up any loculations. The remainder of the procedure is performed as described as above. Scarring with extraoral incisions is virtually universal.

PEDIATRIC CONSIDERATIONS

Facial infections in children tend to develop and progress more rapidly. They are more likely to present with systemic signs and symptoms.[31,32] Facial infections in children are also more likely to be associated with complications such as dehydration or bacteremia.[31] Unlike adults, the anatomic location of a facial infection in a child can be a more useful guide in determining its source and management.[31] In general, upper facial infections tend to be more common in children less than 5 years of age and frequently have no identifiable source. Children older than 5 years of age more commonly have lower face infections and typically have an odontogenic or wound-related source.[31] According to the American Academy of Pediatric Dentistry, a child presenting with facial swelling secondary to a dental infection should receive immediate dental attention.[33] Lastly, in sharp contrast to adults, children with facial infections in either upper or lower locations more frequently respond to antibiotics alone and may not require an incision and drainage procedure.[31]

ASSESSMENT

Basic postoperative assessment includes inspecting the operative site for those minor complications described below. **Assess adequate drain retention. A loose intraoral drain may represent an aspiration risk.** Observe the patient for postprocedural bleeding. It can be controlled with the application of pressure, injection of a local anesthetic agent containing epinephrine, or topical Gelfoam. Wound cultures are not indicated unless the patient is immunocompromised or the infection is recurrent. Wound cultures usually demonstrate mixed oral flora with no predominate organism.

AFTERCARE

The application of warm moist compresses, oral fluids, rest, and good nutrition are mainstays in the treatment of any inflammatory process. Patients may benefit from a soft bland diet and frequent oral rinses with a warm saltwater solution. All patients require postoperative oral analgesics.[3,5,15,16] Patients with an extraoral incision require daily or twice daily dressing changes depending on the dressing soilage.[3,5,15,16] Provide the patient with adequate supplies and education. Instruct the patient on the application of pressure if postprocedural bleeding occurs. Refer the patient to a Dentist within 24 to 48 hours.

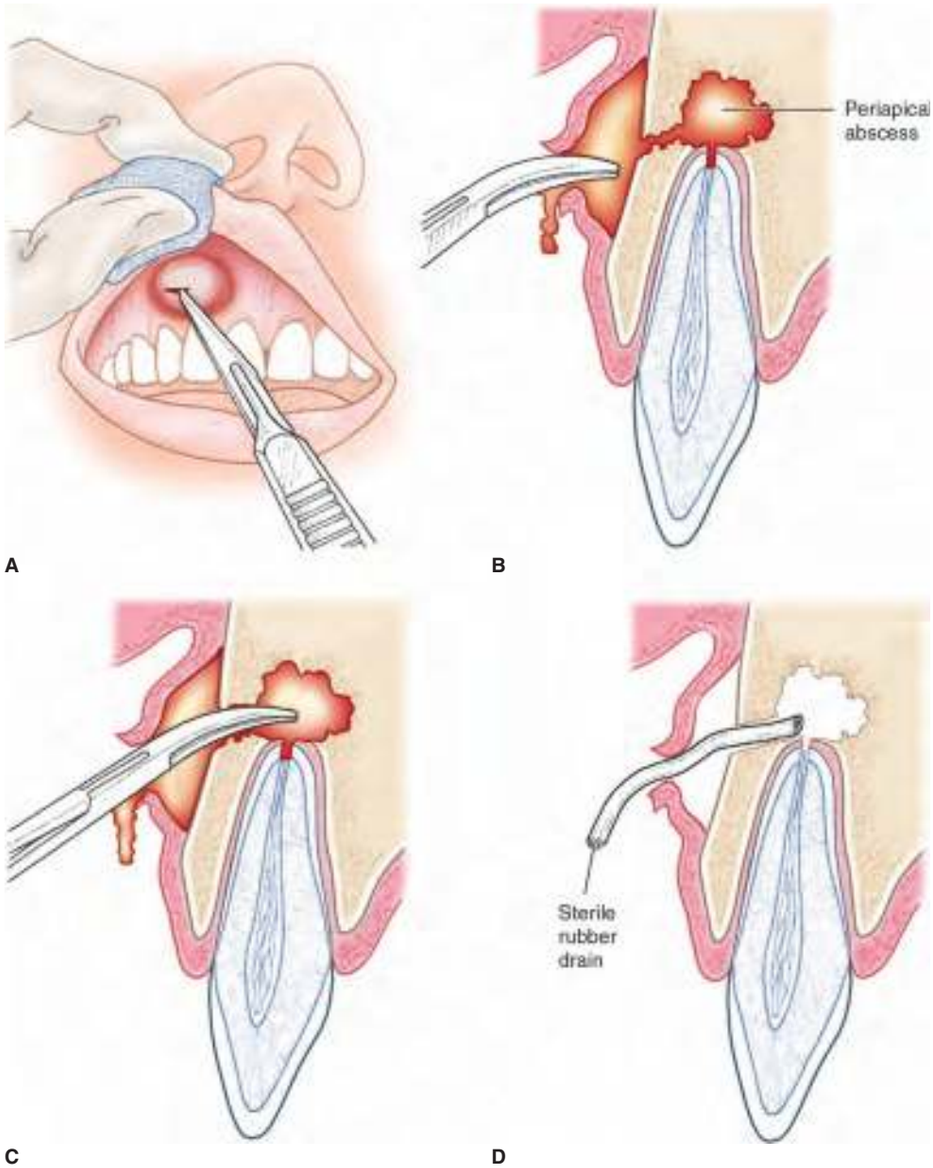

FIGURE 210-7. Incision and drainage of a periapical abscess that has extended into a vestibular abscess. ***A.*** Make a stab incision with a #11 scalpel blade. ***B.*** Insert a hemostat into the incision. ***C.*** Advance the hemostat into the abscess cavity to lyse any adhesions. ***D.*** Place a sterile drain into the abscess cavity.

Nearly half of all Medicare patients seen in the Emergency Department for dental complaints will not follow-up with a Dentist within 6 months which may result in inadequate care overall.[34]

The use of antibiotics in light of adequate drainage is somewhat controversial.[8,35,36] Clear-cut indications for oral antibiotics in orofacial infections include cellulitis, extraoral incision, systemic symptoms, persistent infection, pericoronitis, fascial space involvement, and immunocompromised patients.[4,9,15,21] The prudent Emergency Physician is advised to err on the side of caution while the academics quarrel over antibiotic indications. Penicillin is the drug of choice for the empiric treatment of dental-related infections. Alternatives to penicillin include erythromycin, clindamycin, or a cephalosporin. Clindamycin is the first alternate choice. It is especially useful when anaerobes are suspected or in recalcitrant infections where sensitivities are lacking.[3,5,11,15]

Allow the intraoral drain to remain in place until the swelling has resolved and purulent drainage has ceased. Evaluate the patient for drain removal or advancement in 24 to 48 hours. Prescribe oral antibiotics and analgesics during this time.

Occasional patients need to be admitted to the hospital for intravenous antibiotics.[35] This includes airway concerns, facial cellulitis, large abscesses, and many of the complications noted in **Table 210-2**.[6-8] This should be done in consultation with the Dentist or Oral Surgeon and the admitting physician.

COMPLICATIONS

Minor postoperative pain, swelling, bleeding, drainage, and possibly bruising can be expected following incision and drainage of a dental abscess. Significant postprocedural bleeding can be controlled with pressure, a vasoconstricting local anesthetic agent, or topical Gelfoam.[5,11,22,23] An incision and drainage tract that communicates freely between the oral cavity and the external face represents a significant complication. Refer these patients to a Plastic or Oral Surgeon.

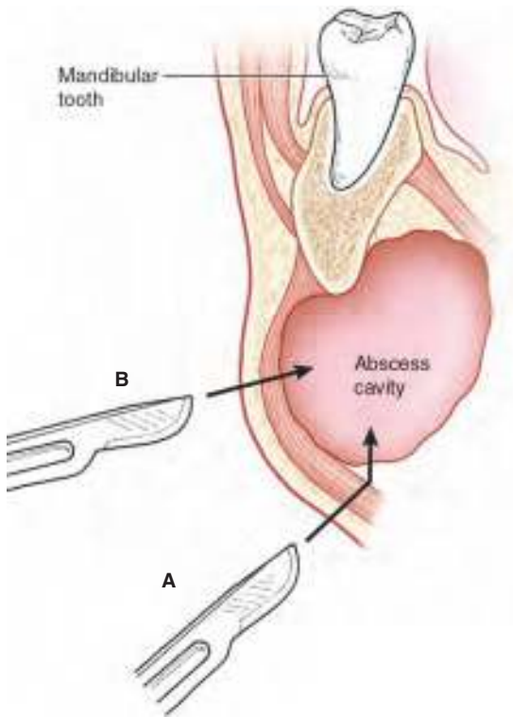

FIGURE 210-8. Extraoral drainage of a dental abscess. Choose a location that has healthy skin and that will allow dependent drainage.

SUMMARY

The recognition of common dental-related infections (i.e., pericoronitis, periodontal abscesses, and dental abscesses) can impact patient prognosis and morbidity. The diagnosis and treatment of these maladies, and their complications, requires knowledge of dental anatomy and pathophysiology. The Emergency Department management of these infections is quick and simple. Refer all patients to a Dentist or Oral Surgeon for timely definitive dental care.

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211 Post-Extraction Pain and Dry Socket (Alveolar Osteitis) Management

Eric F. Reichman

INTRODUCTION

Post-extraction pain, or periosteitis, begins as the local anesthetic agent wears off. The pain begins to diminish, most of the time, within 12 hours. The prescription of nonsteroidal anti-inflammatory drugs will provide analgesia and comfort while the pain subsides over 1 to 2 days. Narcotic analgesics may occasionally be required for the first 24 to 48 hours.

Pain that develops 2 to 4 days after the tooth extraction most likely indicates a localized alveolar osteitis or a dry socket. A dry socket occurs most commonly with the extraction of the third mandibular molar but can be associated with any tooth that has been extracted. The pain is quite severe in nature and is localized to the area of the extraction site. The extraction site may emit a foul odor and the patient often complains of a bad taste in their mouth.[1,2] Physical examination may reveal the socket is missing a clot but this is sometimes difficult to identify. The signs of an infection are absent.

There has been much work in the prevention of a dry socket.[3-8] This prevention focuses on methods in surgical technique and injections that occur at the time of tooth extraction. These methods are not in widespread use yet. Despite these methods, patients still present to the Emergency Department with a dry socket. The Emergency Physician must know how to solve this simple condition.

ANATOMY AND PATHOPHYSIOLOGY

The exact etiology or the pathogenesis of a dry socket is not clear.[1,2,9-12] It may be multifactorial due to smoking, a localized infection, a poor blood supply, traumatic extractions, a foreign body in the socket, and certain medications. These factors result in an increased level of fibrinolysis of the blood clot in the socket before the clot has had the time to be replaced by granulation tissue. The clot falls out of the socket and exposes the bony surface of the socket to the oral cavity. The exposed bone is extremely sensitive to air resulting in severe pain.[1,2,9,10]

INDICATIONS

The single and utmost therapeutic goal of alveolar osteitis is to relieve the patient's pain during the healing process. This procedure should be performed on all patients with a dry socket.

CONTRAINDICATIONS

There are no contraindications to the management of a dry socket.

EQUIPMENT

- Dental mirror
- 2×2 gauze squares
- Scissors
- Dry socket paste or Dressol-x
- Gelfoam
- Irrigating syringe
- Normal saline solution
- Frazier suction catheter
- Suction source and tubing
- Forceps
- Iodoform ribbon gauze
- Zinc oxide eugenol (ZOE)-impregnated ribbon gauze
- Oil of cloves
- Tranexamic acid
- Neocone (not approved or available in the United States)
- Alvogyl (eugenol, iodoform, and butamen)

PATIENT PREPARATION

Explain the risks, benefits, potential complications, and aftercare to the patient and/or their representative. A signed consent is not required for this procedure. Place the patient sitting upright or reclining. A multipositional dental chair is ideal and allows for a variety of positions to visualize the affected tooth. This procedure may be accomplished with no anesthesia. Some may consider performing a dental block to temporarily alleviate the patient's pain, allow the procedure to be accomplished with minimal discomfort, and increase the level of patient satisfaction. If performed, use lidocaine without epinephrine because the procedure is quickly performed and the anesthesia wears off while the patient is still in the Emergency Department. Refer to Chapter 209 for the complete details regarding dental anesthesia and analgesia. Consider obtaining a plain radiograph or Panorex to rule out a retained root tip or foreign material in the socket.

TECHNIQUES

Identify the defective tooth. Gently and thoroughly irrigate the socket with warm normal saline and low-level suction to remove any debris.[13,14] Use a forceps to pack dry socket paste into the socket. Dry socket paste is composed of balsa wood fragments saturated with eucalyptol and looks like chewing tobacco. Completely fill the socket with the dry socket paste. The patient will experience almost instant pain relief if a dental block was not performed. Place a piece of Gelfoam on top of the dry socket paste. Compress the Gelfoam and dry socket paste into the socket. Instruct the patient to bite down against a 2×2 gauze square placed over the socket for 5 to 10 minutes. Dressol-x may be used instead of dry socket paste if available.

ALTERNATIVE TECHNIQUES

Unfortunately, few Emergency Departments stock dry socket paste or Dressol-x. An alternative is ribbon gauze or Gelfoam impregnated with eugenol, iodine, or oil of cloves.[13,14] Ribbon gauze tends to dry out and fall out sooner than commercially available dry socket paste. Use a forceps to pack the socket completely with the impregnated ribbon gauze (**Figures 211-1*A*, 211-1*B*, and 211-1*C***) or Gelfoam. Place a piece of plain Gelfoam on top of the socket (**Figure 211-1*D***). Compress the plain Gelfoam and the underlying impregnated ribbon gauze or impregnated Gelfoam into the socket. Instruct the patient to bite down against a 2×2 gauze square placed over the socket for 5 to 10 minutes. Place a figure-of-eight stitch through the soft tissue and over the packed socket to prevent the Gelfoam from falling out (**Figure 211-2**).

There are several other successful techniques described in the literature to manage the patient who presents with a dry socket.[3,8,14-16] These techniques are also used immediately after extraction of the tooth. These include Oraquix gel (2.5% prilocaine, 2.5% lidocaine thermosetting agents, hydrochloric acid, and purified water), low-level laser treatment (LLLT), the SaliCept patch derived from aloe vera, and vitamin C.[14] Plasma-rich growth factors (platelets,

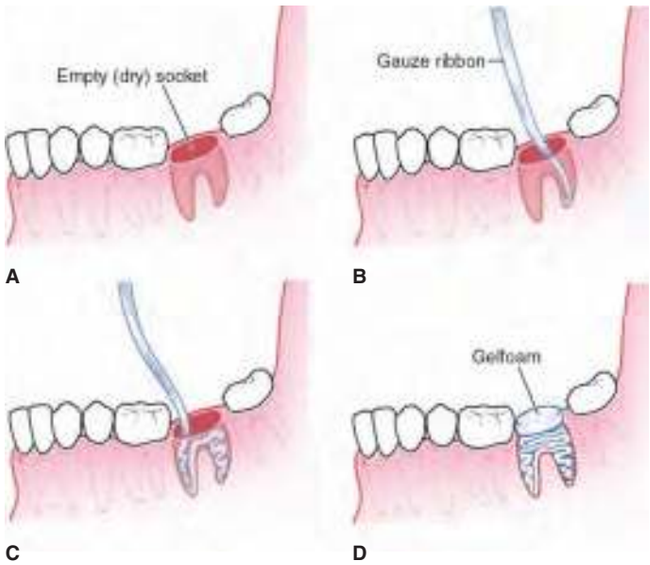

FIGURE 211-1. Packing a dry socket with ribbon gauze. ***A.*** The empty socket. ***B.*** Pack the socket starting inferiorly and working upward. ***C.*** Completely fill the socket. ***D.*** Apply Gelfoam over the socket and the ribbon gauze.

fibrogen, platelet-derived growth factor, and tissue growth factor) and G.E.C.B. pastille (3% guaiacol, 3% eugenol, chlorobutanol, and balsam peru) have been placed in the empty socket.[8] Neocone was compared to Alvogyl and zinc oxide eugenol (ZOE).[15] All three relieved the pain of a dry socket. Neocone resulted in faster healing. Neocone is not U.S. Food and Drug Administration approved for availability in the United States. A honey-coated dressing has been packed in a pocket.[16] This dressing halts necrosis, prevents infection, tastes good, and has no adverse effects. Soak Gelfoam in 160 mg of tranexamic acid. Pack the Gelfoam into the socket and secured with a stitch (**Figure 211-2**).[3] Only the use of tranexamic acid is feasible in the Emergency Department.

ASSESSMENT

The patient should experience almost immediate pain relief after the socket is packed. If a dental block was performed, allow the local anesthetic to wear off to ensure the patient's pain truly has resolved.

AFTERCARE

The patient may be discharged immediately after the procedure or after the local anesthetic has worn off. Nonsteroidal anti-inflammatory drugs are usually adequate to provide analgesia. Narcotic analgesics are not needed or required. Arrange follow-up as soon as possible with the Dentist or Oral Surgeon who performed the extraction procedure. The dressing must be replaced daily, or as needed, until the patient is pain free. Instruct the patient to begin a soft diet, to not ingest extremely hot or cold substances, and to not play with the packing with their tongue. **Instruct the patient to also not suck anything, use a straw, gargle, spit, or smoke.** All these activities will produce negative pressure within the oral cavity and remove the clot from the extraction site.[8]

The decision to prescribe antibiotics to cover the oral flora is physician dependent. There is no literature to support or refute this practice. Consult the Dentist or Oral Surgeon who extracted the tooth. Oral penicillin VK (500 mg four times a day) is the preferred antibiotic if they are prescribed. Clindamycin (300 mg four times a day) is an alternative for patients allergic or intolerant to penicillin.

FIGURE 211-2. The figure-of-eight stitch over a packed dry socket.

COMPLICATIONS

There are no complications associated with this procedure. A potential complication is the aspiration of the material used to pack the socket. This has never been reported in the literature. The packing may fall out and result in the patient's pain recurring. Instruct the patient to return to the Emergency Department if the packing falls out prior to their follow-up appointment.

SUMMARY

A dry socket can be extremely painful. Packing an empty socket is easy, quick, simple, and provides the patient with significant relief. The packing needs to be changed every 24 to 48 hours for several days and then less frequently after that until the patient is free of pain. Consider prescribing antibiotics to cover oral flora and analgesics to manage pain in consultation with the Dentist or Oral Surgeon who extracted the tooth.

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212 Post-Extraction Bleeding Management

Eric F. Reichman

INTRODUCTION

Post-extraction bleeding is a common problem after the removal or extraction of a tooth. It is often seen in the Emergency Department in the late evening or night when the patient is unable to contact their Dentist. Bleeding that occurs within a few hours of the extraction is often due to the wearing off of the vasoconstrictor effect of the local anesthetic solution used for anesthesia.[1] The application of direct pressure over the bleeding site by having the patient bite down on a folded piece of moist gauze almost always controls post-extraction bleeding.[1] Many patients, however, will report that they have been doing this prior to coming to the Emergency Department and require additional assistance. Post-extraction bleeding can be classified depending on when it occurs (**Table 212-1**).

ANATOMY AND PATHOPHYSIOLOGY

A careful history may reveal that the patient inadvertently caused the extraction site to bleed by drinking through a straw, spitting, gargling, or smoking. All these activities will produce negative pressure within the oral cavity and remove the clot from the extraction site. Ask the patient if they are touching the extraction site with their tongue causing a mechanical disruption of the clot. Obtain information about any significant medical history, any history of bleeding, and current medications. This includes use of aspirin products, anticoagulants, broad-spectrum antibiotics, alcohol, and antineoplastic medications. These all may contribute to prolonged bleeding. Ask about the symptoms and examine for the signs of liver disease, hypertension, or hematologic disorders.[2,3] Post-extraction bleeding may be a sign of an underlying coagulopathy due to anticoagulant use, clotting factor disorders, thrombocytopenia, or vascular abnormalities.[1]

INDICATIONS

All post-extraction bleeding must be managed carefully and methodically. The techniques are easy to perform, simple, and straightforward.

TABLE 212-1 Types of Post-Extraction Hemorrhage

Type	Timing	Most likely etiologies
Primary	At time of extraction	Blood vessels Bone Tissue damage
Reactionary	2–3 hours after extraction	Loss of vasoconstriction from local anesthetic solution wearing off
Secondary	Days 1–14 after the extraction	Dry socket Infection

Modified from McCormick et al.[1]

CONTRAINDICATIONS

There are no contraindications to the management of post-extraction bleeding.

EQUIPMENT

- 2×2 gauze squares
- Irrigating syringe
- Dental mirror, optional
- Local anesthetic solution containing 1:100,000 epinephrine
- 23 to 25 gauge, 1.5 inch needle
- 5 mL syringe
- Silk, plain gut, or Vicryl sutures, 3–0 on a half-circle needle
- Absorbable gelatin sponge (Gelfoam)
- Oxidized regenerated cellulose (Surgicel)
- Topical thrombin
- Topical collagen
- Tranexamic acid
- Ankaferd Blood Stopper
- Hemocoagulase (Reptilase)
- Silver nitrate matchsticks
- Suture set
- Dental forceps
- Tea bag
- Bone rongeur
- Bone wax (beeswax, paraffin, and a softening agent)
- Headlamp
- Yankauer suction catheter
- Suction source and tubing
- Electrocautery unit

PATIENT PREPARATION

Explain the risks, benefits, potential complications, and aftercare to the patient and/or their representative. Document this discussion in the medical record. A signed consent form is usually not required for these procedures. Consider obtaining a radiograph of the affected area to rule out a retained root or a bony spur.

Position the patient to visualize the extraction site. Place the patient in a multipositional dental chair, if available, or on a gurney. Do not place the patient in a chair as they may become presyncopal and require being placed supine to prevent injury. An overhead light source or a headlamp is ideal to illuminate the field. Suction any blood and oral secretions from the mouth. Visualize the extraction site for any signs of bleeding. Thoroughly irrigate the site with warm saline and remove all clots with the aid of suction. It may be necessary to perform a dental block if the patient complains of pain upon irrigation. Refer to Chapter 209 for the complete details regarding dental anesthesia and analgesia.

TECHNIQUES

The management of post-extraction bleeding is simple. Numerous methods to control the bleeding have been described and tested (**Figure 212-1, Tables 212-1 and 212-2**). These techniques are often performed in a sequential manner as described below. The techniques may, of course, be performed in any order depending on the physical examination and physician preference.

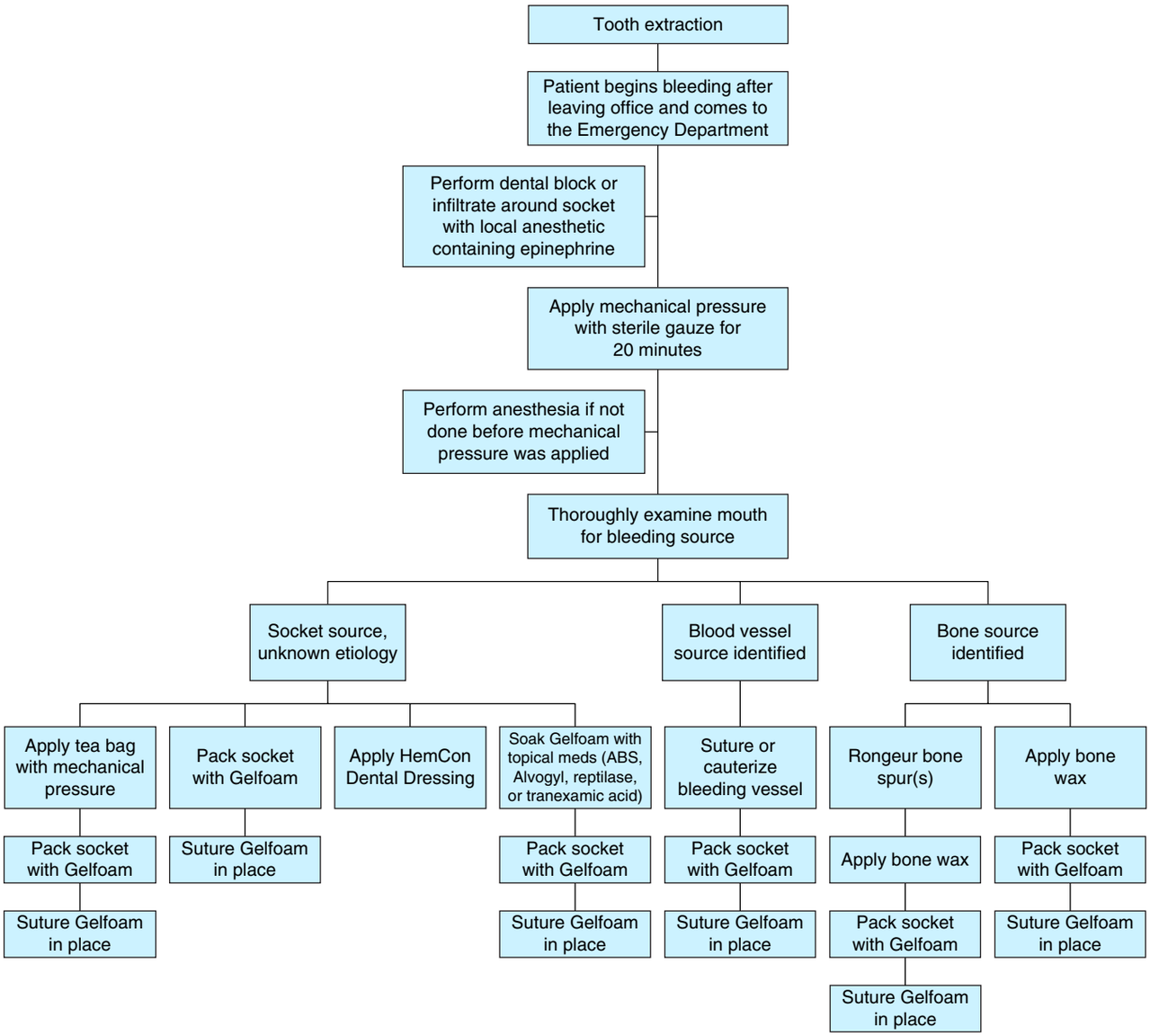

FIGURE 212-1. Treatment algorithm.

TABLE 212-2 Methods to Control Post-Extraction Bleeding

Absorbable dressing into socket and mechanical pressure with gauze squares
Absorbable dressing into socket and stitch gingival tissue closed
Ankaferd Blood Stopper
Bone wax to bone spurs
Cauterize bleeding granulation tissue
Cauterize or stitch bleeding blood vessels
Gelfoam
Gingival infiltration with local anesthetic containing epinephrine
HemCon Dental Dressing
Hemocoagulase (reptilase)
Mechanical pressure with gauze squares
Moist tea bag and pressure
Rongeur followed by bone wax to bone spurs
Stitch gingival tears
Surgicel
Topical collagen
Topical thrombin
Topical tranexamic acid

MECHANICAL PRESSURE

Place saline-moistened 2×2 gauze squares over the socket. Instruct the patient to apply firm pressure by biting down on the gauze for 20 minutes.[1] Instruct the patient to maintain pressure for 20 minutes despite initial bleeding. Place the suction catheter intermittently into the vestibule of the patient's mouth to remove any blood and secretions. Allow the patient to self-suction during the 20 minutes they are biting down on the gauze. Show the patient the proper way to use the suction prior to leaving the examination room. The application of pressure will control most post-extraction bleeding. It may be necessary to perform a dental block, if not performed during the irrigation phase, if the patient cannot bite down due to pain.

TEA BAG APPLICATION

Place a saline-moistened tea bag in the socket if mechanical pressure does not control the bleeding.[4] The tannins in the tea leaves will assist with the coagulation process. Instruct the patient to bite down on the tea bag for 15 minutes. Place the suction catheter

intermittently into the vestibule of the patient's mouth to remove any blood and secretions. Allow the patient to self-suction during the 20 minutes they are biting down on the gauze. Show the patient the proper way to use the suction prior to leaving the examination room.

ABSORBABLE DRESSINGS

The two most commonly used absorbable dressings are Gelfoam and Surgicel. Gelfoam is an absorbable gelatin sponge that is readily available and inexpensive. It forms a scaffold for the formation of a blood clot. Surgicel is composed of oxidized and regenerated cellulose. It promotes coagulation better than Gelfoam and can be packed into the socket under pressure. Unfortunately, Surgicel results in delayed healing of the socket and its use should be reserved for persistent bleeding or when Gelfoam is not available.

Place an absorbable dressing (Gelfoam or Surgicel) in the socket if the extraction site continues to bleed.[2-4] The author prefers to use Gelfoam because it is easier to manipulate and it is absorbed more rapidly than Surgicel. Work the Gelfoam in your fingers until it resembles the shape of the socket. Insert the Gelfoam into the socket and compact it with a dental forceps. Insert additional pieces of Gelfoam into the socket, as necessary, to obtain a solid mass of Gelfoam filling the socket. An alternative is to pack the socket with Surgicel. Once the socket is packed, apply a 2×2 gauze square over the socket. Instruct the patient to bite down for approximately 20 to 30 minutes. Place the suction catheter intermittently into the vestibule of the patient's mouth to remove any blood and secretions. Allow the patient to self-suction during the 20 minutes they are biting down on the gauze. Show the patient the proper way to use the suction prior to leaving the examination room.

The author prefers to place a figure-of-eight stitch using 3–0 silk suture, plain gut suture, or Vicryl suture over the socket (**Figure 212-2**). The suture applies pressure over the socket and ensures that the Gelfoam or Surgicel will not prematurely fall out of the socket. This technique will usually stop most post-extraction bleeding.

Two additional absorbable dressings are topical thrombin and collagen. They are expensive, not usually available in the Emergency Department, and their use should be limited to circumstances where other hemostasis methods have failed. Topical thrombin is made from bovine thrombin. Place a piece of Gelfoam or Surgicel saturated with thrombin into the socket. Thrombin converts fibrinogen to fibrin, bypassing the coagulation cascade, to form a clot within the socket. Collagen is available in multiple forms from a variety of sources. It promotes platelet aggregation and forms a scaffold for the formation of a clot. Pack the socket with collagen and cover it with a piece of Gelfoam. Place a figure-of-eight suture over the thrombin or collagen filled socket to secure it in place.

FIGURE 212-2. Pack the socket with Gelfoam followed by a figure-of-eight stitch to control the bleeding.

The use of chitosan bandages to control battlefield and prehospital hemorrhage has crossed over into the dental realm. Chitosan dental bandages, also known as HemCon Dental Dressing (HemCon Medical Technologies, Portland, OR), have been designed to control post-extraction hemorrhage (**Figure 212-3**). Chitosan is a naturally occurring polysaccharide derived from shellfish. It attracts red blood cells. When placed over the bleeding extraction site, the

A

B

FIGURE 212-3. The HemCon Dental Dressing. ***A.*** The dressing. ***B.*** The dressing placed in a bleeding tooth socket. (Photos courtesy of HemCon Medical Technologies.)

red blood cells attach to the bandage to form a clot and tamponade the bleeding.

LOCAL ANESTHETIC INFILTRATION

Infiltrate the soft tissue surrounding the socket with local anesthetic solution containing epinephrine if the bleeding is not controlled by the above methods. The most commonly used local anesthetic solutions are lidocaine and bupivacaine. Infiltrate the soft tissues surrounding the socket with the local anesthetic solution until the tissue blanches. This usually requires 2 to 3 mL of the local anesthetic solution. Irrigate the socket. Apply a piece of moist gauze over the socket. Instruct the patient to bite down and to exert pressure on the socket. The patient is often able to bite down much harder on the tissues after the infiltration of the local anesthetic solution. Place the suction catheter intermittently into the vestibule of the patient's mouth to remove any blood and secretions. Allow the patient to self-suction during the 20 minutes they are biting down on the gauze. Show the patient the proper way to use the suction prior to leaving the examination room.

The effect of mechanical pressure combined with the vasoconstrictive effects of epinephrine control the bleeding. Occasionally, after the vasoconstrictive effect of epinephrine wears off, there is a rebound effect and the persistence of bleeding. This may be prevented by routinely placing Gelfoam or Surgicel into the socket after the bleeding is controlled.

ANKAFERD BLOOD STOPPER

Ankaferd Blood Stopper or ABS (Ankaferd Health Products, Istanbul, Turkey) is an extract of five plants. It was developed by Huseyin Cahit Firat, a Turkish entrepreneur and an economist with an interest in herbology. It is available in a spray, liquid ampule, and pad form. ABS induces the rapid formation of a protein network structure that acts as an anchor for erythrocyte aggregation. It is independent of the coagulation cascade and works with defects in the coagulation cascade and platelet abnormalities.[5] It is ideal for patients with a coagulopathy. It is approved by the Turkish Ministry of Health and used both prehospital in ambulances and hospitals. It is applied by soaking Gelfoam with the ABS and packing the socket.

TRANEXAMIC ACID

Tranexamic acid, or TXA, is used for traumatic bleeding, hyphemas, subdural and intracranial hemorrhage, epistaxis, and menstrual bleeding to name a few of its uses. TXA is a synthetic analog of the amino acid lysine. It has the desirable properties of being an antifibrinolytic. TXA has been used for post-extraction bleeding prior to the extraction intravenously, topically as a mouthwash prior to or after the extraction, or for dental surgery in hemophiliacs.[6-10] Dissolve a 650 mg tablet in 20 mL of sterile water to make a 3.25% mouthwash solution. Instruct the patient to swish and swallow four times a day. One can also dissolve a 500 mg tablet in 20 mL of sterile water to make a more dilute mouthwash. As an alternative, mix 1 gm per 10 mL of the intravenous formulation of TXA with 10 mL of sterile water to make a 5% mouthwash solution. It can also be applied by soaking Gelfoam with the TXA and packing the socket.

HEMOCOAGULASE (REPTILASE)

The reptilase time (RT) is a blood test used to detect deficiency or abnormalities in fibrinogen. Reptilase is an enzyme found in the venom of *Bothrops* snakes. It has an activity similar to thrombin. Reptilase is resistant to inhibition by antithrombin III unlike thrombin. Reptilase also differs from thrombin by releasing fibrinopeptide A but not fibrinopeptide B in its cleavage of fibrinogen to form fibrin. It is applied by soaking Gelfoam with the hemocoagulase and packing the socket.[11]

MISCELLANEOUS TECHNIQUES

Reexamine the extraction site if the bleeding is not controlled by the above methods. The source of bleeding may be new granulation tissue, gingival tears, a bone spur, or a partially transected vessel. Cauterization of granulation tissue with silver nitrate or electrocautery will control the bleeding. Hand-held, disposable, single patient use electrocautery units work well but require the patient to be anesthetized. Use a blunt instrument to feel for the presence of a bone spur. This may be a source of significant bleeding. Remove the bone spur with a rongeur or cover it with bone wax to control the bleeding. Cover the bone wax with Gelfoam and suture it in place. An exposed and bleeding arteriole or venule can be controlled with cauterization (i.e., silver nitrate or electrocautery) or the application of a plain gut suture through the vessel. Suture any tears in the gingiva.

ASSESSMENT

Observe the patient for 30 to 60 minutes after the bleeding has terminated. Do not give the patient anything by mouth (NPO). Reevaluate the socket for signs of bleeding. Continued bleeding requires further attempts at termination.

AFTERCARE

Discharge the patient home after the bleeding has been terminated and a brief observation period. **Instruct the patient to avoid any liquids or solids for 2 hours and rinsing their mouth for 24 hours. Stress the importance of not spitting, gargling, drinking through a straw, smoking, using aspirin-containing products, or playing with the site with their tongue.** Instruct the patient to apply gauze squares and bite down for 20 minutes if the bleeding returns. They should return promptly to the Emergency Department if the bleeding continues after 20 minutes. Additional instructions should include a soft diet for 24 hours, avoidance of extremely hot or cold substances, avoidance of chewing gum, avoidance of other such "sticky" foods, and minimization of physical activity for 24 hours. The patient may clean (brush and floss) teeth as normal except those teeth immediately adjacent to the extraction site. Arrange follow-up as soon as possible with the Dentist or Oral Surgeon who performed the extraction.

COMPLICATIONS

There are no documented complications associated with the termination of post-extraction bleeding. The complications are associated with the bleeding itself. Obtain screening labs (e.g., prothrombin time, partial thromboplastin time, platelet count, and bleeding times) if hemostasis is not achieved by any of the aforementioned methods. Early consultation with a Dentist or Oral Surgeon and a Hematologist should be considered if the patient is coagulopathic or has a bleeding disorder.

SUMMARY

A careful history and physical examination will often provide the reasons for most post-extraction bleeding that presents to the Emergency Department. Most patients without complicating medical conditions will be easily managed in a simple and systematic manner with a minimal amount of equipment (**Figure 212-1**). All patients should follow-up with their Dentist or Oral Surgeon after the bleeding is terminated.

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213 Defective Dental Restoration Management

Richard Dean Robinson and Daisha McLarty

INTRODUCTION

The field of restorative dentistry is a complex specialty that derives from many disciplines. Patients have often invested considerable time, money, and quite possibly "blood, sweat, and tears" in their dental work. This may be particularly true with the advent of both cosmetic and implant dentistry which involve long and complicated treatment plans that are often not covered by insurance. A patient's investment in their dental work and the technical complexity of today's dental appliances should not be taken lightly. In fact, the treatment of common dental emergencies was published in the *Emergency Medicine Clinics of North America* under the heading "Difficult and Advanced Procedures."[1]

The urgent management of an acutely problematic dental restoration can be as simple as relieving discomfort, treating injury and infection, and employing temporizing measures until definitive treatment can be rendered by the appropriate specialist. This requires a basic understanding of dental anatomy, the pathophysiology of various dental states, and their typical treatment modalities.[1,2] It goes without saying that one should also recognize the inherent and all too frequent limitations of treating these problems in the Emergency Department. **The key to the successful emergent or urgent management of an acutely problematic defective dental restoration is stabilization and timely referral with great care to "first, do no harm."**

A few basic principles serve as a useful guide in treating patients with defective dental restorations. First, know your limitations. Dental pain in general, and a defective dental restoration in particular, is rarely if ever a true emergency.[2-4] Chronic problems should be treated with equal measures of conservatism and reluctance. Refer the patient to a Dentist if you are hesitant to treat or are unfamiliar with an appliance or a presentation. Always consider a secondary or comorbid process. Consider the utility of dental radiographs when in doubt. Never remove a fixed appliance without first discussing it with a specialist, preferably the one who placed it. Save anything and everything that belongs to the patient, whether it be appliance or tissue, as it may have utility for the definitive treatment process.[5,6] Treat pain, inflammation, and evidence of infection. Consider a reversible or temporary solution over all others. Remember to appropriately treat a traumatized (e.g., fracture, luxation, or subluxation) dental restoration when the situation presents. Always consider ingestion or aspiration when dealing with a multiply fragmented tooth or appliance. Consider facial, neck, chest, and abdominal radiographs in cases where all dental fragments cannot be accounted.[5,7-10] Whenever a patient is actively involved in an ongoing treatment process, it is probably best to do as little as possible. Always use caution and tact when discussing the possibility of a defective dental restoration with a patient. Remember that you are not an expert. The treating specialist may have insight into the patient's current condition of which you are unaware. Attempt to consult before treating. Always arrange follow-up within 24 to 48 hours.

ANATOMY AND PATHOPHYSIOLOGY

Patients frequently present to the Emergency Department with some sort of dental complaint and their primary concern is typically pain related.[1,2,11,12] The perioral tissues are exceptionally sensitive to noxious stimuli. This is particularly true for the oral mucosa, periodontal ligament, dentin, and pulp. This concept is paramount to the effective management of any dental-related complaint. Although a patient's chief complaint may be directed at a particular tooth, be diligent in searching the entire mouth for alternative primary, comorbid, or secondary problems.

A meaningful discussion of the management of defective dental restorations requires a brief outline of the available types of dental appliances. In general, dental appliances are either fixed or removable. Fixed dental appliances are considered permanently attached to the teeth. They include crowns, bridges, implants, some forms of dentures, orthodontic bands and brackets, interdental wiring, and any type of filling (e.g., silver amalgam, gold, porcelain, or tooth-colored composite material). Removable dental appliances are those that are not permanently attached to the teeth and include partial dentures, complete dentures, space-maintenance devices, and other orthodontic devices.

Understanding whether a defective dental device is permanent or removable is often the first step in managing an acutely problematic appliance. Detaching a broken or defective removable appliance is a simple and easy solution if it can be done safely and without significantly damaging the appliance. If removal would significantly alter or damage an appliance and there is no other reasonable option but to remove it, document the discussion with the patient and their consent to proceed. This is true whether the appliance is permanent or removable.

Removable dental appliances can predispose patients to several easily treatable mucosal conditions such as ulcerations, abrasions, or mucosal infections.[13-15] Compromised cellular immunity, broad-spectrum antibiotics, low salivary flow, poor oral hygiene, and trauma from poorly fitting dentures can all contribute to oral *Candida* overgrowth.[13,16] Patients often complain of burning

pain. Treat the affected oral mucosa with nystatin or clotrimazole troches. The appliance itself may harbor the organism and must be treated. Instruct the patient to take the appliance out of their mouth for 24 to 48 hours and soak it overnight in a nystatin suspension (5 mL nystatin in 250 mL [8 ounces] of tap water). Instruct the patient to scrub the denture daily with an approved product to clean it.[13-15]

Oral candidiasis must be differentiated from other mucosal ulcerating conditions such as simple traumatic ulcers, aphthous ulcers, and oral herpes. Herpetic lesions occur on attached mucosa only whereas aphthous ulcers occur on unattached mucosa.[13-16] Traumatic ulcers, minor aphthae, and major aphthae can be treated with topical corticosteroid ointment, such as triamcinolone, applied with a cotton-tipped applicator. The symptoms associated with herpetic outbreaks can be diminished if treated with an antiviral agent.[13-15] Orthodontic or interdental wiring that is impinging upon the oral mucosa may lead to traumatic ulcerations. Apply soft dental wax directly to the irritating appliance to relieve the impingement. This can be easily removed or replaced and is available over the counter at many local pharmacies.

In general, all patients with painful mucosal conditions may benefit from soft and bland diets, frequent use of ice chips for discomfort, and frequent warm saltwater rinses to avoid superinfection.[13,14] A stomatitis cocktail or BMX solution may be helpful for patients with severe mucosal pain. Mix equal amounts of Benadryl (12.5 mg/5 mL), Maalox, and Xylocaine (2% viscous lidocaine). Instruct the patient to swish 30 mL in their mouth for 1 minute and then spit it out. **Instruct the patient not to swallow so they will not get anticholinergic or local anesthetic toxicity.**

The repair of broken or defective removable appliances in the Emergency Department is not recommended even though multiple over-the-counter products are available for home use (Figure 213-1).[16] Under the best of circumstances these procedures can be tedious, time consuming, and fraught with complications, even for the trained dental professional.[3] Most importantly, a substandard repair attempt can result in patient discomfort, morbidity, and irreversible damage to an otherwise salvageable appliance. **In this light, it seems best to simply recommend removal and a dental referral to the patient seeking care for a broken removable appliance.[16]** Although far less than ideal, a home repair kit may be a viable option for some patients under certain circumstances.

The temporary urgent repair of defective fixed dental restorations by nondental personnel is advocated throughout the literature.[2,3,11,12,14,17-19] Use caution, however, when evaluating a new or recently placed fixed restoration because sensitivity from recent dental procedures can occur due to pulp or periodontal ligament (PDL) irritation.[20] This may be a normal and expected sequela of the dental procedure and not necessarily an indication that the restoration is defective.[1,20,21] Trauma from occlusion can result if a new dental restoration is left "too high" and does not fit properly with the opposing dentition.[3,14,18-20] These patients complain of pain with mastication and are sensitive to percussion secondary to PDL irritation. **Do not alter a fixed restoration if it is firmly in place.** Simple temporizing measures include dental blocks (Chapter 209), oral analgesics, a soft diet, and possibly removing the tooth from occlusion by placing a small amount of soft dental wax (or something similar) between the teeth on the opposite side.[3]

Long-standing fixed dental restorations may be defective secondary to microleakage or recurrent decay around the margins of the restoration.[1,20] Estimates suggest that approximately one in three restorations in existence may be defective in some fashion.[20,21] A faulty restoration that sits within or upon a tooth can lead to dentin exposure regardless of the etiology. Exposed dentin is highly sensitive and prone to further decay. Dentin sensitivity (also known as reversible pulpitis) is typically nonspontaneous and fleeting.[1,16,18,22] Patients who complain of spontaneous and lasting sensitivity have an irreversible pulpitis that is most likely due to recurrent decay. Sensitivity to percussion is indicative of periapical involvement and suggests the possibility of periapical periodontitis or an absc ess.[1,14,16,18,22]

Evaluate the tooth and its restoration for recurrent decay or possible dentin exposure if a patient presents complaining of sensitivity associated with a tooth that has a long-standing restoration. **A temporary restoration can easily be fabricated in the Emergency Department if the restoration in question is missing, broken, or easily removed.**

Consideration should be given to possible pulp pathology, as outlined above, if a restoration is firmly in place. Provide dental anesthesia, oral analgesics, antibiotics as necessary, and referral to a Dentist. Refer to Chapter 210 for details regarding the management of dental abscesses.

FIGURE 213-1. Some of the commercially available home repair kits for the fractured denture.

INDICATIONS

Replace any previously fixed, permanent or temporary, dental restoration that has completely or partially fallen out or that is easily removed with a dental explorer. Replace any previously fixed, permanent or temporary, crown that has fallen out or is easily removed with a dental explorer and is in the patient's possession.

CONTRAINDICATIONS

Relative contraindications for the placement of a temporary dental filling, or temporarily recementing a crown, include patients who are involved with extensive ongoing dental treatment plans, have a consulting specialist readily available, are at a significant aspiration risk, or have obvious extensive comorbid or secondary processes including antecedent trauma.

EQUIPMENT

- 10 mL syringe
- 18 gauge angiocatheter
- Normal saline solution
- Local anesthetic solution containing epinephrine
- Dental mirror
- Dental explorer
- 2×2 gauze squares
- Sterile cotton rolls
- Dental floss
- Clear nail polish
- Cavit-G
- IRM (zinc oxide and eugenol)
- Dycal (calcium hydroxide paste)
- Copalite (cavity varnish) or clear acrylic nail polish
- Tin foil
- Cotton-tipped applicators
- Discoid-cleoid dental carver
- Articulating paper
- Fraser suction catheters
- Suction source and tubing
- Petrolatum-based lubricant (Vaseline)
- Good overhead lighting or a headlamp

PATIENT PREPARATION

Explain the procedure, its risks, complications, and aftercare to the patient and/or their representative. Obtain an informed consent for the procedure. The simple placement of a temporary filling does not usually require local anesthesia. However, consider the use of a dental block (Chapter 209) if the patient is uncomfortable.

Prepare the patient. Seat the patient in a multipositional procedure chair. Gently irrigate the area with a syringe that contains warm normal saline and is armed with an 18 gauge angiocatheter to remove any food debris. Warm saline is usually less sensitive to the exposed dentin.[1] Gently remove any debris with a dental explorer that does not irrigate away. **Do not attempt to remove any decay because doing so may lead to a complicating pulp exposure.**[1] Remove the loose portion of the restoration. **Do not remove any firmly fixed portion of the restoration.**

It is mandatory to have a dry field when performing this procedure. Dry the area to be filled with sterile cotton pellets or compressed air. Remember that the tooth may be sensitive. A nasal cannula was successfully used as an alternative by some to keep an area dry that was constantly oozing blood. Aim the nasal cannula at a flow rate of 10 to 15 L of air or oxygen at the site prior to and during the procedure to keep the area dry. If the flow interferes with the procedure, intermittently use the nasal cannula to dry the area.

TECHNIQUES

REPLACING A TEMPORARY OR PERMANENT FILLING

Treatment of defective fillings depends upon the relative size of the defect. There are no specific guidelines of what size (i.e., how many millimeters) the defect must be to perform each of these techniques. Paint small dentin exposures with calcium hydroxide paste followed by cavity varnish or clear acrylic nail polish to relieve sensitivity.[1,10,14,19] Alternatively, place a simple tin foil dressing over the tooth to act as a bandage following placement of the calcium hydroxide paste.[10,14,19]

Larger defects require a filling to avoid food impaction and other sequelae. Cavit-G is an excellent choice for temporary filling material, especially in inexperienced hands.[1,10,12,23] This material is premixed, nonirritating, and sets in approximately 30 minutes upon contact with saliva. IRM, or a mixture of zinc oxide and eugenol, is similar material with the benefit of pulp sedative properties. The use of IRM is operator dependent and requires a longer setting time.[23] Oddly enough, multiple mixtures of zinc oxide and eugenol are available over-the-counter for home use (e.g., Dentemp, Tempanol, Thin Set; **Figure 213-2**).

Cavit-G is recommended for Emergency Department use. Determine whether the missing filling exposes an open previous endodontic repair (i.e., root canal). Place a small sterile cotton pellet into the canal prior to placing the filling material if the pulp cavity is exposed.[23] Express a small amount of Cavit-G from the container. Apply it onto the cavity and condense it into the cavity with the moistened end of a cotton-tipped applicator or a dental explorer (**Figure 213-3*A***). **A temporary restoration placed by nondental personnel is always better "short" and out of occlusion with the opposing tooth for patient comfort. Work quickly because Cavit-G can set rapidly and it may be difficult to remove once it sets.** Remove any excess Cavit-G with the stick end of the cotton-tipped applicator. Instruct the patient to fully occlude on the new restoration and grind their teeth back and forth in all directions for 5 to 10 minutes. This will form the occlusal aspect of the filling to fit the opposing teeth if it is not made "short."

Cyanoacrylate is used in the Emergency Department for wound repair and has the additional properties of being hemostatic and bacteriostatic.[24,25] Cyanoacrylate also has many applications in periodontics. It can be used as a desensitizing agent, for pulp capping, pit and fissure sealant, to seal dentin, for retrofilling material in endodontic surgeries, and to replace avulsed teeth. Some patients have used cyanoacrylate to fix their own dentures, especially dental prostheses composed of an acrylic base. Cases have also been reported of cyanoacrylate being used to repair fractured molar teeth.[24-26]

Using cyanoacrylate to fill a tooth is similar to using dental cement as noted above. Once the cavity is prepared and dried, drip the cyanoacrylate into the cavity until filled. Ensure there are no air bubbles. Use smoothing equipment to quickly smooth out the filling. Cyanoacrylate dries very quickly. Instruct the patient to occlude the teeth and grind similar to the above instructions.[26]

There is some concern that cyanoacrylate may be harmful to the oral mucosa. Documented effects include urticarial reaction and skin irritation. Rarely, its vapors have been noted to induce asthma.

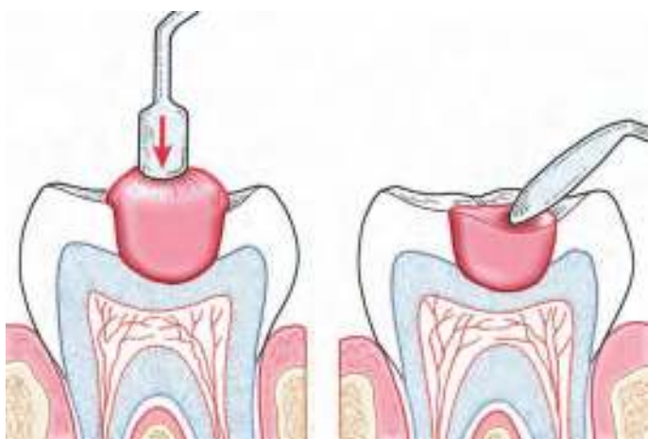
FIGURE 213-2. Some of the commercially available home use products for temporary dental filling and crown restoration.

Avoid unnecessary direct skin contact and use cyanoacrylate in rooms with ventilation.[24]

Remove any excess material with the stick end of the cotton-tipped applicator. Use the discoid-cleoid dental carver to remove excess material once it begins to harden (**Figure 213-3*B***). Use dental floss to contour a proper embrasure and clear excess material from the gingival tissues. **Use the dental floss in a downward direction only. Never bring the dental floss back up toward the occlusal surface because doing so risks dislodging the new restoration.** Simply pull the dental floss through after one downward pass around the tooth.

REPLACING A TEMPORARY OR PERMANENT CROWN

The patient preparation required for this procedure is essentially the same as that for replacing a temporary filling. However, avoid dental anesthesia if possible to allow better patient proprioception. This will provide the Emergency Physician with a crucial aid in assessing the orientation and occlusion of the final restoration.[1] The same over-the-counter mixtures for repairing a filling may be used to temporarily replace a crown (**Figure 213-2**).



A B

FIGURE 213-3. Application of temporary restorative material. ***A.*** Pack the material into the defect and condense it with a dental explorer (or a cotton-tipped applicator). ***B.*** Remove any excess material.

Reapply the crown to ensure that it fits properly. It is often necessary to remove a small amount of the existing cement from within the patient's existing restoration in order to provide an adequate seal and a proper occlusion.[1] This may or may not be possible to do with the discoid-cleoid dental carver. Do not use rotary instruments as these may irreversibly damage the inside of the coping. Reinsert the patient's crown after removing some of the cement to assure a proper fit and proper occlusion. Make an attempt using the alternative technique described below if sufficient preexisting cement cannot be removed and the crown seats properly. Treat dentin sensitivity as outlined above (i.e., as replacing a filling) if the appliance does not seat at all.

Proceed with this technique if the restoration appears to fit and a small amount of the preexisting cement can be removed from the inside. Prepare the area. Apply a thin layer of petrolatum-based lubricant (e.g., Vaseline) to the mucosal tissues surrounding the tooth to aid in the cleanup. **Do not contaminate the prepared tooth with the lubricant.**

Prepare or mix a thin consistency of zinc oxide and eugenol cement. Place a very small amount of the mixture into the preexisting crown with a cotton-tipped applicator.[3,14,18] Place the crown on the tooth and in the proper orientation. Firmly and fully seat the crown with firm finger pressure. Remove any excess cement material from around the margin of the newly cemented restoration. This may be easier once the material has hardened. Instruct the patient to gently and fully occlude on the restoration. **Ensure proper occlusion.** Ask the patient to provide a subjective report as to whether their occlusion feels like baseline (i.e., does their bite feel funny or normal). Confirm this using articulating paper to ensure that the contact marks are light or minimal. Wipe the petrolatum-based lubricant from the mucosal tissues.

A simpler alternative and far more temporary method of replacing a crown involves using Vaseline as the cementing agent.[1] Apply a thin layer of Vaseline to the inside of the coping. It may be necessary to remove a small amount of the preexisting cement. Seat the crown on the tooth in the proper orientation. Ensure proper occlusion. Warn the patient that the crown may come off again and about the risk of aspiration. Instructions may be given to the patient for repeating the procedure at home.

Another simple approach for replacing a crown is to use cyanoacrylate. Similar to the Vaseline method, place a thin layer inside the coping and seat the crown on the tooth in the proper orientation. Have the patient gently and fully occlude on the restoration.[26]

ASSESSMENT

Check the patient's occlusion. It may be necessary to use articulating paper to ensure minimal or no contact with the opposing teeth with the temporary filling. The newly cemented crown restoration must primarily be assessed for proper occlusion. The patient's subjective opinion is invaluable in this regard.

AFTERCARE

Instruct the patient not to eat or chew on their new restoration for at least 1 hour. Additional instructions should include a soft diet, avoidance of extremely hot or cold substances, avoidance of chewing gum, and avoidance of other such "sticky" foodstuffs. They may brush their teeth normally but should not floss adjacent to the new restoration. Warn them that they may experience some continued sensitivity. Nonsteroidal anti-inflammatory drugs will provide any needed analgesia. Arrange follow-up within 24 to 48 hours.

COMPLICATIONS

Typical complications of replacing a filling include improper occlusion and poor retention of the restoration. The solution for both problems is replacing the restoration. Adjust the occlusion with the discoid-cleoid dental carver. Treat the restoration as a dental restoration that is "too high" if using the discoid-cleoid dental carver is not effective. Treat the tooth as a simple dentin exposure if a temporary restoration is continually falling out. An unlikely complication would be pulp exposure manifested as minimal bleeding from within the tooth defect.[1,10,12] Manage this as a dental trauma or fractured tooth (Chapter 215).

Typical complications for replacing a crown are similar to those listed in replacing a temporary filling. These restorations are often easily removed with a slight twisting motion. A restoration that is seated "too high" should be replaced. Treat the tooth as a simple dentin exposure if a restoration is consistently "too high."

SUMMARY

Management of the patient with a dental complaint may initially seem intimidating to the Emergency Physician. The recognition and treatment of dental pain, minor defective dental restorations, and painful mucosal conditions can be relatively simple provided the Emergency Physician possesses a minimal understanding of basic dentistry. Emergency Physicians must be cognizant of the inherent limitations involved in treating these patients in the Emergency Department. Have a low threshold for referral. Pain, inflammation, and infection should always be treated. Refer patients to the appropriate specialist within 24 to 48 hours.

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214 Subluxed and Avulsed Tooth Management

Richard Dean Robinson, Adam Flink, and Ryan Nathaniel Krech

INTRODUCTION

Traumatic dental injuries are a common presentation to the Emergency Department.[1-6] They can have significant lasting cosmetic, functional, and psychological consequences for the patient. Recent estimates indicate over three quarters of a million annual Emergency Department visits in the United States occur for dental-related complaints.[7] Nearly 12% of these are related to some form of trauma.[7] Approximately 50% of children will sustain traumatic dental injuries, the majority of these to the permanent dentition.[8-10] Violence of

a suspicious nature, such as domestic adult abuse and child abuse, must always be considered when evaluating dental injuries.

The appropriate Emergency Department management of dental trauma depends heavily upon the type of tooth (i.e., permanent versus primary), the age of the tooth, the time elapsed since the dental trauma, and the extent of the damage. Successful treatment of dental injuries requires a basic understanding of dental anatomy, terminology, and pathophysiology. **The goals of the emergent treatment of dental trauma are to maintain patient comfort and tooth viability while ensuring prompt dental follow-up for definitive care.**

ANATOMY AND PATHOPHYSIOLOGY

TOOTH ANATOMY

There are significant differences in the adult and pediatric dentition that impact their treatment in the Emergency Department (**Figure 214-1**). The pediatric dentition is known as the primary or deciduous dentition and consists of 20 teeth. These include 8 incisors, 4 canines, and 8 molars. The adult dentition consists of 32 teeth and is composed of 8 incisors, 4 canines, 8 premolars, and 12 molars. The variable absence of a tooth or the addition of an extra tooth is common in either dentition. Teeth in both pediatric and adult dentitions erupt in a predictable sequence, albeit with considerable individual variation (**Figure 214-1**). Treatment strategies differ for permanent versus deciduous (i.e., primary) teeth as well as by the age of the adult tooth. **Exercise great care when evaluating patients with a "mixed" dentition, roughly between the ages of 6 and 12 years.**

The anatomy of a tooth is rather simple (**Figure 214-2**). The tooth itself consists of a neurovascular pulp surrounded by supportive dentin which is surrounded by a hard thick crown of enamel. The

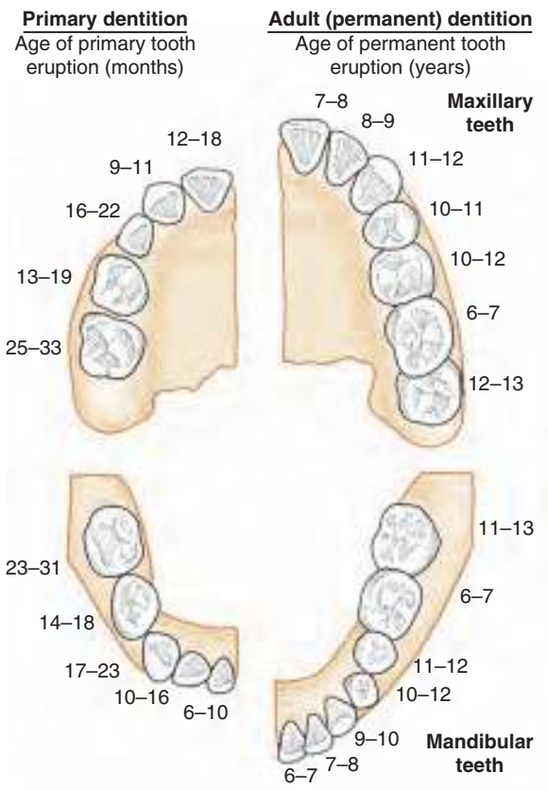


FIGURE 214-1. The normal eruptive patterns of the pediatric and adult dentition.

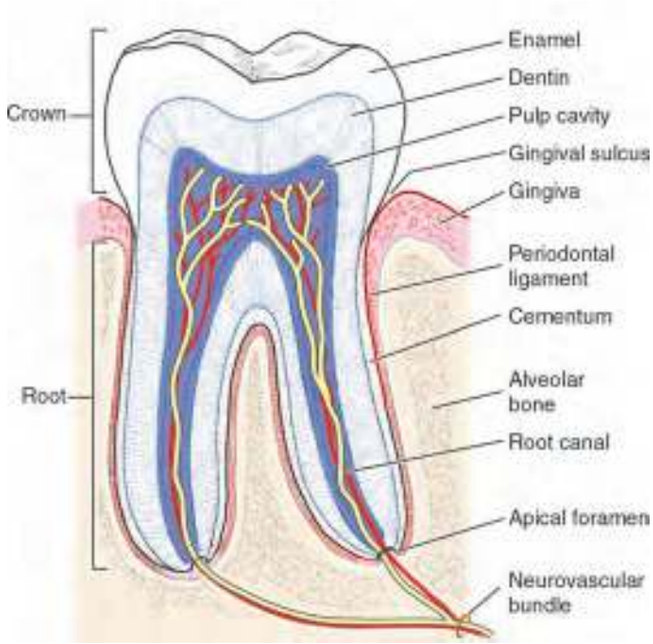


FIGURE 214-2. The dental anatomic unit (i.e., the tooth) and its supporting structures.

crown portion lies above the gum line or gingiva. The root portion lies embedded within the alveolar bone of the jaw anchored by a thin layer of cementum and the periodontal ligament. The alveolar bone, periodontal ligament fibers, and fragile cementum cell layer taken together are considered a functional unit known as the attachment apparatus. A complete attachment apparatus requires an intact cementum cellular layer and a fully formed root apex. Immature adult teeth do not have a fully formed apex and necessitate special attention to maintain pulp viability.[9,11-15]

TOOTH INJURY

Mechanisms of tooth injury include direct trauma (i.e., a blow) or occlusive trauma (i.e., biting on a hard object or a seizure). These mechanisms can result in a spectrum of injury patterns that vary from simple sensitivity to complete tooth avulsion. Crown and root fractures are discussed in Chapter 215. This chapter focuses on the diagnosis and management of dental subluxations and avulsions.

Appropriate treatment of dental injuries requires a thorough history and meticulous examination of the oral cavity including subsequent radiographs after ruling out more serious injuries. Important points in the history include the age of the patient, the time of the trauma, the mechanism of injury, the location of teeth or tooth fragments, subjective disturbance of bite, and treatments provided since the time of the incident. The physical examination must include an assessment of the extraoral and intraoral soft tissues, bony displacement, missing teeth, crown fractures, pulp exposures, tooth sensitivity, and tooth mobility.

The need for radiographs with dental trauma is worth emphasizing. A tooth that is missing, both by history and physical examination, may be found completely intruded below the gum line, embedded in the perioral soft tissues, floating within the maxillary sinus or stomach, or aspirated into the airway.[16-21] **Obtain facial films if a tooth, or a portion of the tooth, cannot be unequivocally located by history or physical examination. Strongly consider obtaining chest and abdominal radiographs if the tooth, or the portion in question, is not visualized on the facial films.**

CONCUSSED AND SUBLUXED TEETH

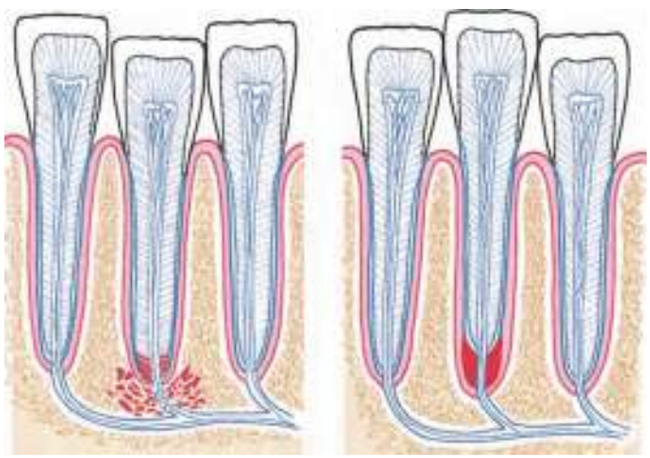
Mild injuries to teeth are common and cause concussions and subluxations. Concussed teeth are essentially injured, not mobile, and not fractured teeth. These teeth have suffered a direct blow and are sensitive with no concrete clinical or radiographic evidence of injury. Concussions are often mild injuries to the periodontal ligament with associated inflammation. Subluxed teeth may or may not be sensitive, are not displaced, but are perceptively mobile when manipulated between two cotton applicators or other instruments. Subluxations have definite damage to the periodontal ligament with associated inflammation. Mild gingival bleeding may be present. **Both a concussion and a subluxation imply an injury to the attachment apparatus.** Pain control, soft diet instructions, and follow-up with a Dentist are all that is required in the management of most of these injuries. Concussed and subluxed primary and permanent teeth are generally treated in the same manner.[8,13-15]

Excessive mobility from a severe subluxation may be irritating, painful, and damaging. These injuries require a temporary splint for relief.[8,9,11-15] Definitive treatment for severely subluxed permanent teeth requires splinting for 2 weeks or more.[15] Definitive treatment for severely subluxed primary teeth is extraction.[15]

LUXATED TEETH

Luxated teeth are displaced or dislocated from their usual position within the alveolar bone. The periodontal ligament is torn. Luxated teeth are commonly associated with other injuries such as alveolar fractures, root fractures, and gingival lacerations.[8,22] Subcategories of injury within this class are described by the direction of the dislocation. Luxated teeth may be displaced laterally, intruded, or extruded (**Figure 214-3**). Lateral luxations may be mesial, distal, buccal, or lingual in direction. An alveolar fracture is self-evident when several teeth are luxated as a solid segment.

Laterally luxated permanent teeth should be repositioned and temporarily splinted.[13-15] Laterally luxated primary teeth can be treated in the same fashion.[15] A general word of caution regarding primary teeth is warranted. **The apices of the primary teeth are in close anatomic proximity to the developing permanent tooth buds within the alveolus. This close relationship can lead to numerous developmental disturbances in the permanent dentition whenever there is trauma to a primary tooth.[23] Therefore, it is recommended that the Emergency Physician defer any manipulation of a primary tooth to the care of a Dentist or at least consult them first.**



A B

FIGURE 214-3. Luxation injuries with neurovascular damage to the apex. ***A.*** Intrusive luxation injury. ***B.*** Extrusive luxation injury.

Extruded teeth represent a partial avulsion from the alveolar socket and a damaged attachment apparatus. They typically appear clinically longer than the surrounding teeth (**Figure 214-3*B***). Patients may complain of occlusal prematurity. There may be associated gingival bleeding. A hematoma surrounding the apex may preclude complete repositioning. Extruded permanent teeth are treated with repositioning and temporary splinting.[8,9,11,13-15] Treatment decisions for extruded primary teeth are complex and best left to a Dentist. The majority of these teeth will likely require extraction.[15]

Intrusion is a severe form of luxation injury with the tooth driven inward in an axial direction. These injuries are manifested by displacement of the tooth into the alveolar socket with a corresponding fracture of the alveolar bone surrounding the apex (**Figure 214-3*A***). Adjacent structures, such as the floor of the nose or maxillary sinus, may be involved or damaged.[19,20] These injuries may be so profound that the tooth is not visible within the oral cavity and believed to be avulsed. **It is worth reiterating that a tooth that cannot be unequivocally located on physical examination requires radiographic localization.** Immature adult teeth suffering intrusion injuries generally have the best prognosis. They are often left alone and allowed to re-erupt. Mature adult teeth often require surgical or orthodontic assisted re-eruption and root canal therapy. The intrusion of primary teeth frequently leads to damage of the developing permanent tooth buds and requires close dental follow-up. Rule out more serious injuries, arrange an expedited appointment for definitive care, prescribe appropriate analgesics, and give strong consideration to the prescription of antibiotics.[8,9,11,13-15,24]

AVULSED TEETH

Avulsed teeth are teeth that have been completely torn from their alveolar sockets. **The teeth have suffered profound attachment and neurovascular damage that progresses in a time-dependent fashion.** There is a high likelihood of associated injuries with this type of trauma. Perform a thorough evaluation of the entire oral cavity after any dried blood, clots, and debris have been removed. Bleeding can generally be controlled with firm digital pressure or local infiltration of an epinephrine-containing local anesthetic solution. Patients may present with the tooth in hand or may not be aware of the location of the tooth. **The onus is on the Emergency Physician to determine the exact whereabouts of the tooth. Treat the patient's pain, control the bleeding, and provide tetanus prophylaxis.** Prescribe antibiotics for these patients if there are significant concomitant injuries or as the situation warrants. Arrange follow-up with a Dentist at their convenience. If available, a permanent tooth can be treated with replantation. **As a rule, avulsed primary teeth are not replanted to avoid damage to the developing permanent teeth and possible growth disturbances.[11,13-15]** Exercise great care in evaluating patients in the mixed dentition stage roughly between the ages of 6 and 12 years.[8,9,11]

An attempt can be made at replantation in order to preserve patient comfort, cosmesis, and function when a permanent avulsed tooth is available. **The objective for the emergency treatment of these injuries is to maintain viability of the torn periodontal ligament fibers on the external root surface as pulp necrosis is inevitable for the majority of these teeth.[8]** A successfully replanted tooth may be fully functional with little or no cosmetic impact following root canal therapy. **Periodontal ligament fibers are extremely sensitive to desiccation. The most critical factor in the successful replantation of avulsed teeth is the speed with which the tooth is replanted.[8,9,22,25]** Patients, parents, or Emergency Medical Service personnel can be instructed to replant an avulsed tooth in the field in order to improve the prognosis.[8,9]

Take great care in the handling of an avulsed tooth. They should be handled minimally and only by the crown. The root surface should not be manipulated in any way other than gentle cleansing with sterile saline or tap water as a substitute. This will prevent further damage to the cementum and periodontal ligament. Treat the socket in a similar fashion (i.e., cleansed of any obstructing clots or debris with gentle irrigation and suction only) following anesthesia.[9,25]

When it is not possible to immediately replant an avulsed tooth, it can be transported or stored in such a way as to prevent desiccation of the fragile periodontal ligament fibers and to improve salvage rates.[8,11,13-15] The best possible transport and storage solutions are Hank's balanced salt solution (HBSS) and Viaspan, a special cell culture medium (SCCM) used to preserve transplant tissues. The SCCM seems to show some benefit over HBSS.[26] Several commercial products are readily available and specifically designed for tooth transportation and storage. They include EMT Tooth Saver (SmartPractice, Phoenix, AZ) and Dentosafe (Medice, Iserlohn, Germany) which utilize SCCM or Save-a-Tooth (Phoenix-Lazerus, Pottstown, PA) which utilizes HBSS.[26] These products have great utility in the field and the Emergency Department. They should be considered standard and essential equipment for the Emergency Department. This is particularly true where definitive dental care is not readily available. Simply providing a patient with one of these products may allow successful replantation by a dental specialist hours or even days later.[8,27] Fresh pasteurized whole milk and sterile normal saline are alternatives but carry diminishing returns for salvaging the tooth. Saliva can be employed as a transport medium by placing the tooth in the buccal vestibule of a conscious and cooperative adult for very brief periods, again with diminishing hopes for salvage. Tap water or plastic wrap may prevent desiccation for a brief period if all else fails.[9,11,13-15,27]

The literature indicates that irreversible periodontal ligament cell damage occurs within minutes following total tooth avulsion.[9,11,25] Common clinical practice is typically to abandon attempts at salvaging the tooth if it has been out of the socket for more than an hour. **However, the notion that replantation is not possible for extraoral times beyond 1 hour is a myth.** The dental trauma literature is replete with case reports of successful outcomes following very long extraoral times (e.g., up to a week) and under extremely suboptimal storage conditions such as being kept completely dry.[27,28] An aesthetic and functional, albeit less than ideal, result may be possible for avulsed teeth with extraoral times over an hour through a process known as ankylosis if appropriately and aggressively treated by a skilled Dentist. **Therefore, it is never acceptable to discard an avulsed tooth.** Avulsed teeth with extraoral times greater than 1 hour require special treatment with topical fluoride and antibiotic soaks that are typically beyond the scope of care in the Emergency Department.[15,25] Consult a Dentist prior to considering these techniques. At the very least, always give the patient their tooth back in a suitable storage or transport device and advise them on their options for definitive care.

REDUCTION OF SEVERELY SUBLUXED, LATERALLY LUXATED, AND EXTRUDED TEETH

The emergent treatment of a severely subluxed tooth, a laterally luxated tooth, or an extruded tooth involves obtaining adequate anesthesia, reduction of the tooth, and a temporary splint for stabilization.

INDICATIONS

Any severely subluxed, laterally luxated, or extruded tooth is a candidate for reduction.

CONTRAINDICATIONS

There are no absolute contraindications to the reduction of a subluxed, laterally luxated, or extruded permanent tooth. Laterally luxated and primary teeth can be repositioned and splinted. However, their manipulation may damage the permanent tooth bud growing beneath the primary tooth root. Therefore, all subluxed, luxated, or extruded primary teeth should be considered for extraction in consultation with a Dentist or Oral Surgeon.[11,13-15,22,29] Consult a Dentist or Oral Surgeon if there is a significant alveolar bone fracture. Do not attempt to reduce teeth that are fractured or grossly carious.

EQUIPMENT

- Local anesthetic solution, with and without epinephrine
- Dental aspirating syringe or a 3 mL syringe with a 2 inch, 25 or 27 gauge needle
- Sterile saline
- Sterile 2×2 gauze squares
- Suction source and tubing
- Frazier suction catheter
- Cotton-tipped applicators
- Overhead lighting or headlight

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain a signed informed consent for the procedure. Place the patient in a multipositional procedure chair with good overhead lighting. Administer dental anesthesia (Chapter 209). Cleanse the oral cavity with warm saline or tap water and gentle suction. Thoroughly examine the entire oral cavity. Obtain radiographs as indicated. Provide tetanus prophylaxis if required.

TECHNIQUES

SEVERELY SUBLUXED TOOTH REDUCTION

A severely subluxed tooth is not displaced from its socket but is excessively mobile. Ensure that the tooth is in its proper anatomic location. Apply a temporary dental splint as described below.

LATERALLY LUXATED TOOTH REDUCTION

A laterally luxated tooth has its roots displaced laterally and out of the socket (**Figure 214-4**). It is often associated with a fracture of the surrounding alveolar bone. Place the dominant thumb over the medial surface of the tooth and the index finger overlying the root end of the tooth (**Figure 214-4**). Apply downward and inward pressure with the index finger (**Figure 214-4*A***) followed by application of outward pressure with the thumb (**Figure 214-4*B***) to reduce the tooth. Apply a temporary dental splint as described below.

EXTRUDED TOOTH REDUCTION

An extruded tooth is a partially avulsed tooth that protrudes above the adjacent teeth. Apply gentle pressure to the crown of the tooth to reduce the tooth. **Do not force the tooth into the socket to avoid an iatrogenic fracture at its base.** Consult a Dentist if the tooth will not reduce. A hematoma in the base of the socket often prevents reduction. Insert a piece of gauze over the tooth and instruct the patient to gently bite down.

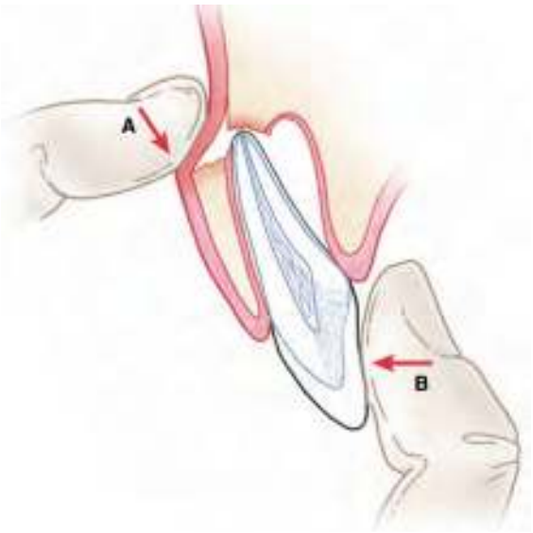


FIGURE 214-4. Manually repositioning a laterally luxated tooth. Apply downward and inward pressure with index finger (*A*) followed by outward pressure with the thumb (*B*).

ASSESSMENT

Obtain postreduction radiographs to verify the correct tooth position. Radiographs may be delayed until after splinting. Reassess the patient for pain, occlusal discrepancies, and stability of the reduction. Manage any soft tissue injuries.

AFTERCARE

Prescribe appropriate analgesics. Nonsteroidal anti-inflammatory drugs supplemented with occasional narcotic analgesics will provide adequate analgesia. Prescribe empiric antibiotics (e.g., penicillin or clindamycin). Twice daily rinses with chlorhexidine may also be useful. Instruct the patient to avoid extremely hot or cold substances, to eat a liquid or soft diet, and to avoid chewing in the area of the injury. Provide specific instructions regarding interim dental splint care as discussed below. Arrange follow-up with a Dentist or Oral Surgeon within 24 hours. Remind the patient that any dental injury can result in the loss of tooth vitality and, ultimately, the loss of the tooth despite the best of efforts to maintain it.[8,9,24]

COMPLICATIONS

Immediate complications of any dental trauma include pain and cosmetic deformity. Additionally, instability may be an issue following the application of a temporary splint. Delayed complications can be variable and include tooth mobility, root resorption, pulp necrosis, infection, and abscess formation. Extension of untreated infections into alveolar bones can cause osteomyelitis and/or systemic infectious complications. A permanent tooth may develop abnormally in a younger child if injured. Bleeding is minimal and often self-limited. Refer to Chapter 212 for details of post-extraction bleeding management. Ensuring prompt dental follow-up, adequate outpatient analgesics, and empiric antibiotics can limit most of these complications.

REPLANTATION OF AN AVULSED TOOTH

Permanent teeth that have been avulsed should be handled gently and only by the crown. Time is a critical factor in the successful treatment of these injuries and every effort should be made to expedite the care of these patients. Replantation consists of gently reinserting the tooth in the proper orientation and fully seating it with gentle pressure.

INDICATIONS

Any intact and avulsed permanent tooth is a candidate for replantation.[30-32]

CONTRAINDICATIONS

There are no absolute contraindications to the replanting of permanent teeth by nondental personnel. Concerns for the ABCs (airway, breathing, and circulation), concomitant major morbidity, and aspiration risk in acutely or chronically debilitated patients should be considered prior to tooth replantation. Primary teeth are never replanted. Do not attempt to replant teeth that are fractured or grossly carious.

EQUIPMENT

- Local anesthetic solution, with and without epinephrine
- Dental aspirating syringe or a 3 mL syringe with a 2 inch, 25 or 27 gauge needle
- Sterile saline
- Hank's balanced salt solution
- Sterile 2×2 gauze squares
- Sterile cotton rolls
- Suction source and tubing
- Frazier suction catheter
- Cotton-tipped applicators
- Overhead lighting or headlight

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain a signed informed consent for the procedure. Place the patient in a multipositional procedure chair with good overhead lighting. Administer dental anesthesia (Chapter 209). Cleanse the oral cavity with warm saline or tap water. **Use gentle suction but never near the injured tooth.** Thoroughly examine the entire oral cavity. Obtain radiographs as indicated. Provide tetanus prophylaxis and antibiotics if required. Gently irrigate the avulsed tooth and socket with warm sterile saline or Hank's solution. Remove any clots and debris using a Frazier suction catheter. **Take great care to avoid touching or contaminating the tooth root.** Soak the tooth in Hank's solution for 30 minutes prior to replantation if the extraoral dry time exceeds 30 minutes. Consult a Dentist skilled in dental trauma care prior to replanting if the extraoral time exceeds 1 hour.

If the apex of the tooth is open, consider soaking the tooth for 5 minutes prior to replantation in a solution of 1 mg of doxycycline in 20 mL of normal saline which may improve pulp revascularization. The soaking of avulsed monkey teeth in doxycycline for 5 minutes prior to reimplantation showed improvement in revascularization of the pulp and root development.[33] However, a more recent study showed no increased benefit in pulp survival with soaking the tooth in doxycycline compared to saline.[34]

TECHNIQUE

Grasp the avulsed tooth gently and only by the crown. Replace the avulsed tooth into the socket in an anatomically correct position. Seat the tooth fully with gentle but firm digital pressure. **Never force the tooth into the socket.** Evaluate the patient's occlusion. Instruct the

patient to gently bite together several times while observing for any prematurity. Occasionally, a tooth cannot be completely seated or its position is uncertain. Instruct the patient to temporarily bite on a gauze roll until the dental specialist arrives or store the tooth in a storage or transport medium until definitive dental care can be rendered.[9,24] Address any soft tissue injuries once the tooth's position has been verified. Insert a piece of gauze over the tooth and instruct the patient to gently bite down. Apply a temporary dental splint as described below.

ASSESSMENT

Obtain postreplantation radiographs to verify the correct tooth position. Radiographs may be delayed until after splinting. Reassess the patient for pain, occlusal discrepancies, and stability of the replanted tooth prior to discharge. Manage any soft tissue injuries.

AFTERCARE

Prescribe appropriate analgesics. Nonsteroidal anti-inflammatory drugs supplemented with occasional narcotic analgesics will provide adequate analgesia. Prescribe empiric antibiotics. Doxycycline is recommended as the drug of choice in patients over 12 years of age. Penicillin and clindamycin are useful substitutes. Twice daily rinses with chlorhexidine are useful but not required. Instruct the patient to avoid extremely hot or cold substances, to eat a liquid or soft diet, and to avoid chewing in the area of the injury. Provide specific instructions regarding interim dental splint care as discussed below. Arrange follow-up with a Dentist or Oral Surgeon within 24 hours. Remind the patient that any dental injury can result in the loss of tooth vitality and, ultimately, the loss of the tooth despite the best of efforts to maintain it.[8,9,24]

COMPLICATIONS

Immediate complications of any dental trauma include pain and cosmetic deformity. Additionally, instability may be an issue following the application of a temporary splint. Delayed complications can be variable and include tooth mobility, root resorption, pulp necrosis, infection, and abscess formation. Extension of untreated infections into alveolar bones can cause osteomyelitis and/or systemic infectious complications. A permanent tooth may develop abnormally in a younger child if injured. Bleeding is minimal and often self-limited. Refer to Chapter 212 for the details of post-extraction bleeding management. Ensuring prompt dental follow-up, adequate outpatient analgesics, and empiric antibiotics can limit most of these complications.

PREPARING A TEMPORARY DENTAL SPLINT

Concussed teeth, subluxed primary teeth, and subluxed permanent teeth usually do not require splinting. A temporary splint may prevent further damage and improve patient comfort if severe mobility or subluxation is present. Manually reposition laterally luxated and extruded permanent teeth using gentle and firm digital manipulation following adequate anesthesia. A Dentist or Oral Surgeon will typically extract laterally luxated or extruded primary teeth. Intruded teeth, both primary and adult, are associated with considerable comorbidity and complications. These injuries require consultation with a Dentist or Oral Surgeon after defining the extent of the injuries with appropriate radiographs.

INDICATIONS

Any severely traumatized and grossly mobile, luxated, repositioned, or replanted tooth requires temporary splinting. This will prevent further damage, promote patient comfort, preserve form, and preserve function.

CONTRAINDICATIONS

There are no absolute contraindications to the temporary splinting of mobile teeth. The aspiration risk in acutely or chronically debilitated patients should be considered prior to tooth splinting.

GENERAL SPLINTING EQUIPMENT

- Local anesthetic solution, with and without epinephrine
- Dental aspirating syringe or a 3 mL syringe with a 2 inch, 25 or 27 gauge needle
- Sterile saline
- Coe-Pak or Perio-Pak
- Dental utility wax or beeswax
- Sterile 2×2 gauze squares
- Sterile cotton rolls
- Applicator sticks; tongue depressors or the wooden end of cotton swabs will substitute
- Frazier suction catheter
- Suction source and tubing
- Overhead lighting or headlight

ADVANCED SPLINTING EQUIPMENT

- General splinting equipment as described above
- Wire cutters
- Tooth etching gel
- Composite resin material (e.g., Centrix Tempit Ultra-F, Centrix Inc., Shelton, CT)
- Single-component adhesive (e.g., Optibond Solo Plus Kit, Kerr Corp., Orange, CA)
- Bondable reinforcement ribbon (e.g., Ribbond, Ribbond Inc., Seattle, WA)
- Orthodontic wire, 0.025 inch
- Tubing and regulator to attach to wall air supply
- Visible curing light

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain a signed informed consent for the procedure. Place the patient in a multipositional procedure chair with good overhead lighting. Administer dental anesthesia, cleanse and thoroughly examine the entire oral cavity, obtain radiographs as indicated, and provide tetanus prophylaxis and antibiotics if required if not done earlier. Manually reposition any luxated and avulsed teeth. **Manage any soft tissue injuries prior to splint placement to avoid wound contamination by the splint material.**

TECHNIQUES

Cold-curing periodontal packing material (i.e., Coe-Pak or Perio-Pak) is an ideal splinting material for practitioners without dental experience. Measure out equal amounts (i.e., lengths) of the catalyst and the epoxy onto a paper pad (**Figure 214-5*A***). A 2 to 3 inch long ribbon of each substance is usually adequate. Thoroughly mix the catalyst and epoxy compounds together with a tongue depressor to form a putty-like consistency (**Figure 214-5*B***). Using tap water- or saline-wetted gloved hands, roll the material into a log (**Figure 214-5*C***). Apply the material to frame both aspects, medial

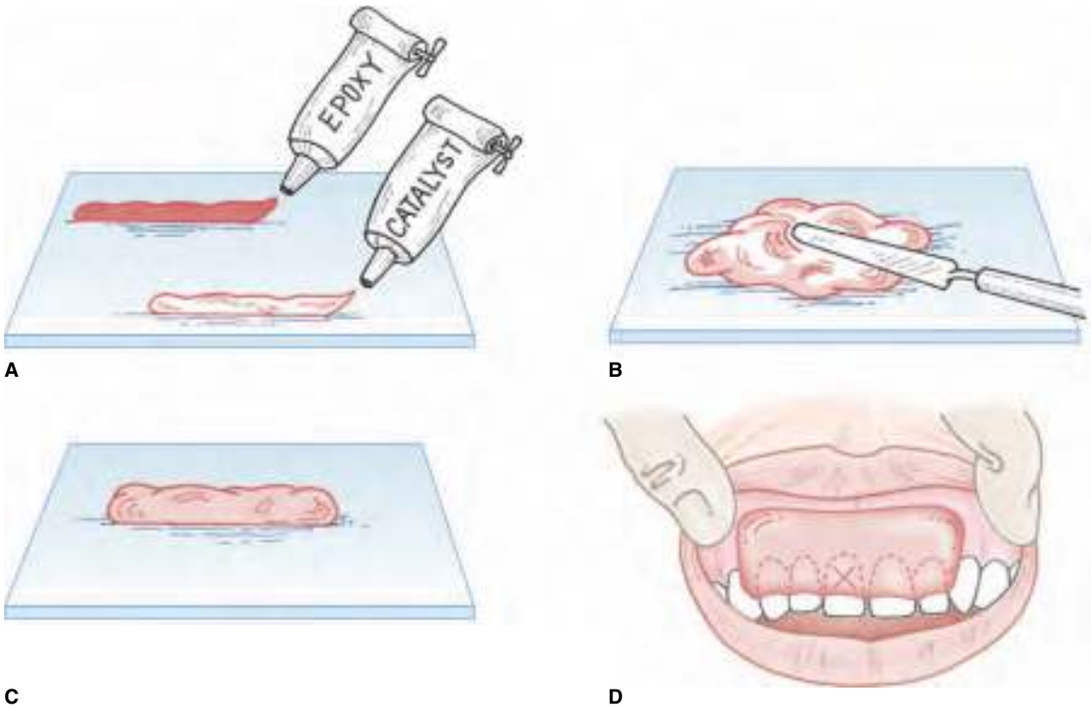


FIGURE 214-5. Preparation of the dental bonding resin and repair of the injured tooth. ***A.*** Equal amounts of the epoxy and catalyst are measured. ***B.*** The epoxy and catalyst are mixed together. ***C.*** The hardening dental paste is molded into a supportive bridge. ***D.*** The dental bridge is applied over the injured tooth and adjacent two uninjured teeth (both sides) for support while hardening.

and lateral, of the injured tooth and two to three adjacent stable teeth on either side of the injured tooth (**Figure 214-5*D***). Use finger pressure to squeeze the material between the teeth medially and laterally to create a single splint unit. **To allow proper occlusion, do not place the packing material on the masticatory surfaces of the teeth. The material must be kept dry and uncontaminated to cure which is achieved within minutes.**

ALTERNATIVE TECHNIQUES

Numerous alternative techniques have also been used to temporarily splint a tooth. A simpler technique employs softened dental utility wax or beeswax in a similar fashion as described above. The wax splint is not nearly as stable as the cold-curing periodontal packing. Both the medial and lateral surfaces of the teeth can be splinted in this fashion. Ligature splinting with suture material has been described but rarely provides any significant stability.

Advanced techniques include bondable reinforcement ribbon, acid-etched composite resin, direct interdental wiring, resin-wire combinations, arch bars, and stabilization with a figure-of-eight stitch to the adjacent tooth.[35-37] These are excellent materials in experienced hands. Unfortunately, they are difficult to use, fraught with complications, and cost prohibitive for routine Emergency Department use.[8,9,11,12,21,22,24,29] Two of these methods, which can be performed by the Emergency Physician if supplies are available, are described below. In an emergency with no immediate dental supplies or available Dentist, skin wound glue (2-octyl cyanoacrylate) and a metal nasal bridge from a face mask have been used to splint a tooth.[38]

Teeth may be splinted using bondable reinforcement ribbon. Open the Ribbond kit. Use the included scissors to cut a piece of the splinting fabric long enough to span the length of the injured tooth and one tooth on each side of the injured tooth. Apply a small drop of the acid etching solution onto all three teeth (i.e., the injured tooth, the one behind it, and the one in front of it). Allow the acid etching solution to remain on the teeth for 20 seconds. Thoroughly and gently rinse the acid etching solution using warm tap water or warm sterile saline in a syringe while using suction to capture the liquid. It is often easier to etch and rinse one tooth at a time instead of all three simultaneously. Thoroughly dry the teeth with compressed air. Remove the Optibond Solo Plus container from its packaging. Twist and remove the tip from the Optibond Solo Plus container. Apply a layer of the Optibond Solo Plus to the etched surface of all three teeth using the kit's micro brush. Apply the curing light to the three teeth for 20 seconds each. Apply Optibond Solo Plus onto the previously cut splinting fabric until it is saturated. Apply the tip onto the syringe containing the composite resin. Apply the composite resin to each of the three teeth. Apply and embed the splinting fabric into the composite resin on each tooth. Use a wooden stick (e.g., tongue depressor or cotton-tipped applicator) to ensure the fabric is embedded in the resin as well as covered by the resin. Apply the curing light onto the three teeth for 40 seconds each. The procedure is the same if multiple adjacent teeth require splinting.

A tooth may be splinted using wire and composite resin. Use a wire cutter to cut a piece of orthodontic wire long enough to span the length of the injured tooth and one tooth on each side of the injured tooth. The remainder of the procedure is exactly as described above except the orthodontic wire is substituted for the splinting fabric.

ASSESSMENT

Allow the patient to wait in the Emergency Department until the splinting material has hardened. The splint material should impinge minimally on the soft tissues. The patient must be able to open and close their mouth and lips freely without any obstruction. Reassess the patient for pain, occlusal discrepancies, and stability of the

replanted or subluxed tooth prior to discharge. Obtain postsplinting radiographs to verify the proper tooth position.

AFTERCARE

Prescribe appropriate analgesics. Nonsteroidal anti-inflammatory drugs supplemented with occasional narcotic analgesics will provide adequate analgesia. Prescribe empiric antibiotics. Instruct the patient to avoid extremely hot or cold substances, to eat a liquid or soft diet, and to avoid chewing in the area of the injury. Provide specific instructions regarding interim dental splint care. Arrange follow-up with a Dentist or Oral Surgeon within 24 hours. Remind the patient that any dental injury can result in the loss of tooth vitality and, ultimately, the loss of the tooth despite the best of efforts to maintain it.[8,9,24]

COMPLICATIONS

The complications of temporary dental splinting are minimal. The splint material may not stabilize the tooth. A tooth allowed to move within the socket may result in damage to the cementum or the periodontal ligament. An improperly splinted tooth may fall out and result in an aspiration risk. To prevent irritation and bleeding, do not apply splinting materials over the soft tissues. Do not leave the etching acid on longer than 20 seconds or it can penetrate too deep and damage the tooth.

SUMMARY

Traumatic dental injuries are a common presentation to the Emergency Department, especially in pediatric patients during the mixed dentition stage. These injuries may have significant cosmetic, functional, and psychological consequences for the rest of the patient's life. The appropriate Emergency Department management of dental trauma depends heavily upon the type of tooth involved (i.e., primary versus permanent), the time elapsed since the incident, and the extent of the damage. A basic understanding of dental anatomy, terminology, pathophysiology, and treatment protocols will facilitate an accurate description of the extent of the injuries to the dental consultant and be of great aid in providing temporizing emergent dental care when no specialist is readily available. In the future, a subluxed or avulsed tooth may be easily replaced with a 3D-printed tooth made of antimicrobial plastic.[39]

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215 Fractured Tooth Management

Richard Dean Robinson, Mahmuda Farha, and Bharti Chaudhari

INTRODUCTION

Traumatic dental injuries are common and can have significant lasting consequences for the patient. Recent estimates indicate over three quarters of a million annual Emergency Department visits in the United States for dental-related complaints.[1] Nearly 12% of these are related to some form of trauma.[1] It has been estimated that approximately 50% of children will sustain traumatic dental injuries, and the majority of these are to permanent dentition.[2-4] **Violence of a suspicious nature such as domestic adult abuse, child abuse, and child neglect must always be considered when evaluating dental injuries. The goals of the emergent treatment of dental trauma are to maintain patient comfort and tooth viability while ensuring prompt dental follow-up for definitive care.**

ANATOMY AND PATHOPHYSIOLOGY

TOOTH ANATOMY

There are significant differences in adult and pediatric dentition that impact their treatment in the Emergency Department (**Figure 215-1**). The pediatric dentition is known as the primary or deciduous dentition and consists of 20 teeth, which includes 8 incisors, 4 canines, and 8 molars. The adult dentition consists of 32 teeth and is composed of 8 incisors, 4 canines, 8 premolars, and 12 molars. The variable absence of a tooth or the addition of an extra tooth is common in either dentition. The teeth in both the pediatric and adult dentitions erupt in a predictable sequence, albeit with considerable individual variation (**Figure 215-1**). Treatment strategies differ for permanent versus deciduous (i.e., primary) teeth as well as by the age of the adult tooth.[5-8] **Exercise great care when evaluating patients with a "mixed" dentition, roughly between the ages of 6 and 12 years.**

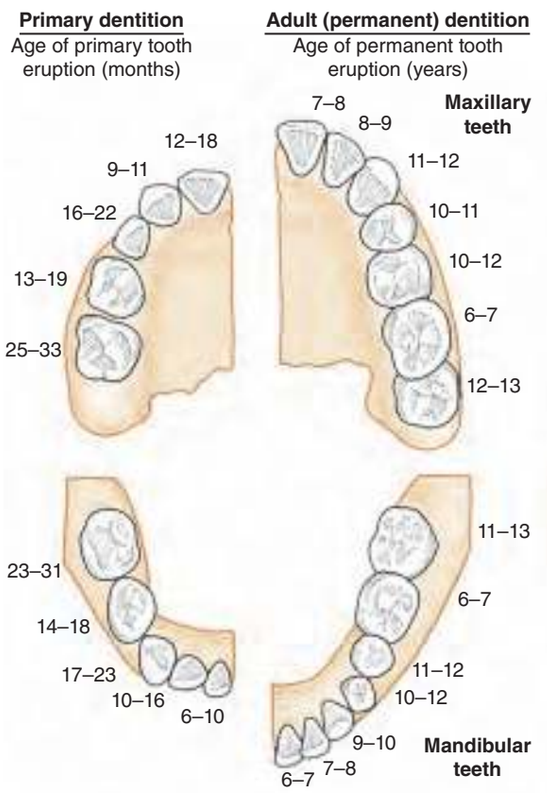

FIGURE 215-1. The normal eruptive patterns of the pediatric and adult dentition.

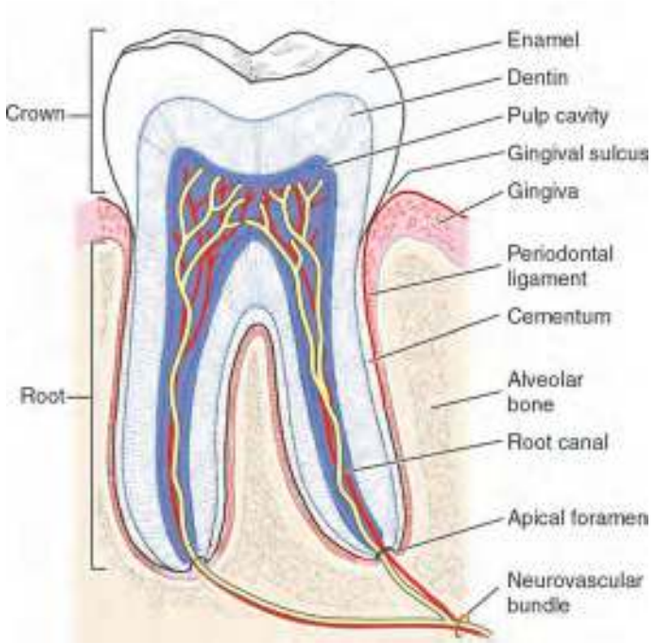

FIGURE 215-2. The dental anatomic unit (i.e., the tooth) and its supporting structures.

The anatomy of a tooth is rather simple (**Figure 215-2**). The tooth itself consists of a neurovascular pulp surrounded by supportive dentin which is surrounded by a hard thick crown of enamel. The crown portion lies above the gum line or gingiva. The root portion lies embedded within the alveolar bone of the jaw anchored by a thin layer of cementum and the periodontal ligament. The alveolar bone, periodontal ligament fibers, and fragile cementum cell layer taken together are considered a functional unit known as the attachment apparatus. A complete attachment apparatus requires a fully formed root apex. Immature adult teeth do not have a fully formed apex and necessitate special attention to maintain pulp viability.[2-4,9]

TOOTH INJURY

Mechanisms of tooth injury include direct trauma (e.g., a blow) or occlusive trauma (e.g., biting on a hard object or a seizure). These mechanisms can result in a spectrum of injury patterns that vary from simple sensitivity to complete tooth avulsion. The fracture of any portion of the tooth, whether the crown or the root, falls in the middle of this spectrum and is frequently seen in the Emergency Department.[4]

Appropriate treatment of dental injuries requires a thorough history and meticulous examination of the oral cavity including subsequent radiographs after ruling out more serious injuries. Important points in the history include the age of the patient, the time of the trauma, the mechanism of injury, teeth or tooth pieces at the scene, subjective disturbance of bite, and the treatments provided since the time of the incident. The physical examination must include an assessment of the extraoral and intraoral soft tissues, bony

displacement, missing teeth, crown fractures, pulp exposures, tooth sensitivity, and tooth mobility. This chapter focuses primarily on tooth fractures while luxation and avulsion injuries are dealt with in Chapter 214.

The need for radiographs with dental trauma is worth emphasizing. A tooth that is missing, both by history and physical examination, may be found completely intruded below the gum line, impacted within the perioral soft tissues, floating within the maxillary sinus or stomach, or even aspirated. **Obtain facial films if a tooth, or a portion of a tooth, cannot be unequivocally located by history or physical examination. Strongly consider obtaining chest and abdominal radiographs if the tooth, or portion in question, is not visualized on the facial films.**[10-15] Any available tooth fragments whether retrieved from the scene, the patient's perioral soft tissues, or the patient's pocket should be saved and stored for potential use during the definitive care process by the Dentist.[13,16-18] Bonding techniques in the Emergency Department are not prudent due to multiple potential complications including bond failure and tooth fragment aspiration.

TOOTH FRACTURES

Fractures involving the crown of the tooth are commonly described in the emergency literature using the Ellis classification system (**Figure 215-3**).[2,4,9,10,19,20] **An Ellis I fracture involves only the enamel portion of the tooth (Figures 215-3 and 215-4*A*).** These injuries typically are not sensitive or painful. They can result in a sharp edge of enamel that may irritate the tongue and other adjacent soft tissues. Emergency treatment may be as simple as smoothing the rough edge with an emery board or similar instrument.[2,4,18,21] These injuries frequently involve the prominent anterior teeth and may be cosmetically unappealing. Reassure patients with these concerns that aesthetic restorations are possible by their Dentist.[4,9,10,19,22] **Forewarn patients with even minor trauma and sensitivity that unseen or undiagnosed trauma at the apex of any traumatized tooth, even with an appropriately treated crown fracture, can compromise blood flow to the pulp and obviate the need for root canal therapy.[4,22] Both primary and permanent teeth with these fractures can be treated in a similar fashion.[18]**

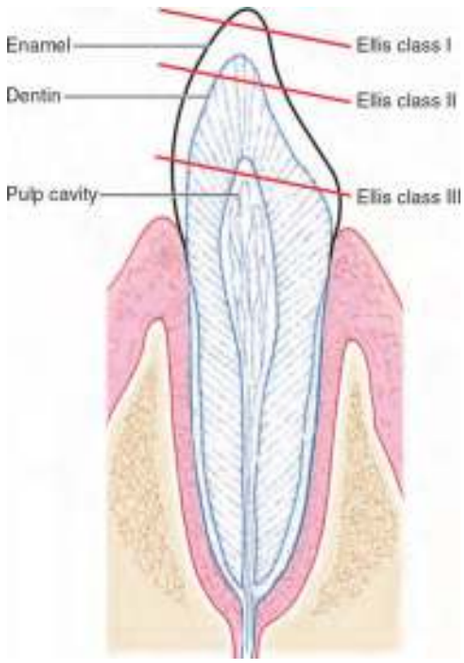

FIGURE 215-3. The Ellis classification scheme of dental fractures through the crown.

A

B

C

FIGURE 215-4. Photos of tooth fractures. ***A.*** Ellis class I fracture. ***B.*** Ellis class II fracture. ***C.*** Ellis class III fracture. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine,* 4th ed. New York: McGraw-Hill; 2016. Parts A and B: Photo contributor: James F. Steiner, DDS. Part C: Photo contributor: Kevin J. Knoop, MD, MS.)

An Ellis II fracture involves the dentin (Figures 215-3 and 215-4*B*). It can be recognized by the yellow to pink hue of the dentin in contrast to the white of enamel. This fracture allows for potential contamination of the dentin microtubular network by oral bacteria that may eventually compromise the pulp if not treated. Dentin is alive, formed by the pulp, sensitive to temperature, sensitive to osmotic gradients, and sensitive to mechanical forces. Dentin is laid down concentrically from within the pulp chamber as the tooth ages. Therefore, children have less dentin than pulp (as compared to adults) and their pulp is less insulated against trauma and subsequent infection. Children under the age of 12 years with Ellis II fractures have a higher risk of complications and require more expeditious follow-up.[2,9,18,22] **Refer these patients to a General or Pediatric Dentist as soon as possible.**

Emergency treatment for Ellis II fractures consists of applying a protective dressing that is also sedative to the pulp. Examples include Dycal (L.D. Caulk Co., Milford, DE) and IRM (L.D. Caulk Co.,

Milford, DE). These materials need to cover the entire exposed dentin (and therefore the dentin microtubules) in order to protect the pulp from contamination. These materials are then often covered with a sealant such as Copalite (Cooley & Cooley, Houston, TX), clear acrylic nail polish, or a dental bonding resin.[2,3,9,10,18,19,22,23] Some authors have suggested that a non–light cured glass ionomer cement replace the long-held standard of Dycal.[3] While these materials may offer some advantages, they can be expensive and difficult to use.[22]

Tissue adhesives such as Dermabond (Ethicon, Inc., Somerville, NJ) have been suggested as alternative dressings in the treatment of Ellis II fractures.[24-26] This should be discouraged as its effects on pulp tissues via exposed dentin microtubules have not been studied and are unknown. Physicians may actually be causing harm by using this material on exposed dentin.

Both primary and permanent teeth with Ellis II fractures can be treated in a similar fashion. However, like immature permanent teeth, primary teeth with Ellis II fractures require special care and more expeditious follow-up.[18]

An Ellis III fracture involves exposure of both the dentin and the pulp (Figures 215-3 and 215-4*C*). This is identified as a reddish tinge or subtle bleeding from the exposed dentin. Frank pulp exposures are obvious. The pulp is highly vascular and exquisitely sensitive due to exposed nerve endings. The pulp is exceedingly vulnerable to bacterial infection if exposed. **These fractures constitute a true dental emergency and should be evaluated immediately by a Dentist or Oral Surgeon for possible emergent root canal therapy or extraction.** Although less than ideal, minimal pulp exposures (i.e., those less than 1 to 2 mm) may be treated as Ellis II fractures with dental follow-up within 24 hours.[2-4,9,10,18,19,22] Complete coverage of the fractured crown may be difficult in these cases. Dental dry foil or tin foil may provide adequate coverage. Any root canal manipulation is fraught with complications, even in the hands of Endodontists. Emergency Physicians are well advised to avoid these procedures.[2,19,22]

The pulp may be tricky to see in some cases of tooth fracture. A slight hint of pink may be seen under the dentin. **If pink is seen, do not explore deeper as you may open a thin layer of dentin covering the pulp.** Exploration may turn an Ellis II into an Ellis III fracture.

Fractures of the root are much less common than crown fractures and occur in less than 7% of dental injuries.[2,4] Root fractures are uncommon in primary teeth as they have short roots.[2] Root fractures may be described as either horizontal or vertical. Horizontal root fractures are described according to their location along the tooth root (**Figure 215-5**). Vertical root fractures occasionally extend into the crown. All root fractures are prone to infection, impaired healing, and may ultimately lead to pulp necrosis and tooth loss.

The clinical diagnosis of root fractures is challenging at best, even with the aid of radiographs readily available in the Emergency Department (i.e., a Panorex). Root fractures classically present with pain, mobility, and sometimes displacement of a tooth fragment. However, these fractures are often insidious and found only on dental radiographs after follow-up reveals continued sensitivity. **Emergency Physicians must maintain a high level of clinical suspicion for these injuries and should probably err on the side of cautious overtreatment.**[2,4]

Vertical root fractures and root fractures in the coronal portion of the root have a poor prognosis. Horizontal root fractures elsewhere along the tooth root have a good prognosis if treated within 24 to 72 hours before a coagulum can develop between the fragments.[2,19] Immediate reduction and immobilization with one of the various splinting techniques is the treatment of choice. Refer to Chapter 214 for details regarding dental splinting techniques. Root fractures in primary teeth require extraction.[18]

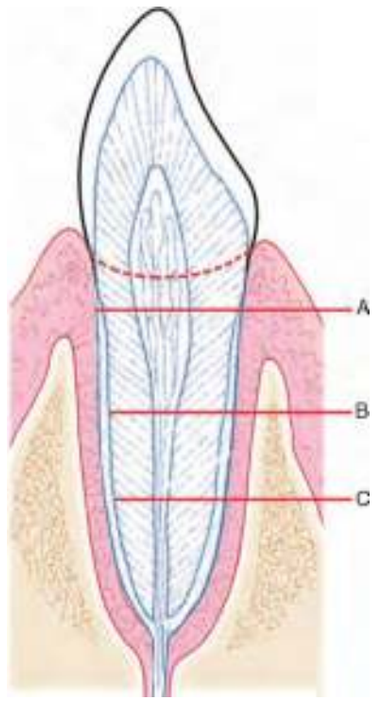

FIGURE 215-5. Classification of root fractures according to their location. ***A.*** Incisal or coronal third fracture. ***B.*** Mid-root fracture. ***C.*** Apical third fracture.

INDICATIONS

Fractured teeth may require no treatment or a significant amount of treatment based upon the type of injury as described by the Ellis classification system. Fractured roots must be treated based upon the level of clinical suspicion.

CONTRAINDICATIONS

There are no absolute contraindications to the temporary repair of a fractured tooth or tooth root. Concerns for the ABCs (airway, breathing, and circulation), concomitant major morbidity, and aspiration risk in acutely or chronically debilitated patients should be considered prior to tooth repair.

EQUIPMENT

- Local anesthetic solution, with and without epinephrine
- Dental aspirating syringe, or a 3 mL syringe with a 2 inch, 25 or 27 gauge needle
- Dental explorer
- Discoid-cleoid carver
- Dental drill or emery board
- Sterile saline
- Sterile 2×2 gauze squares
- Sterile cotton rolls
- Applicator or molding sticks; tongue depressor or the wooden end of cotton swabs will substitute
- Calcium hydroxide paste (e.g., Dycal), mixable or premixed (preferred)
- Copalite cavity varnish or clear acrylic nail polish
- Zinc oxide eugenol paste (e.g., ZOE or IRM)
- Dental dry foil or tin foil

- Frazier tip suction catheter
- Suction source and tubing
- Good overhead lighting or headlight
- Dental floss

PATIENT PREPARATION

Explain the risks, benefits, and aftercare to the patient and/or their representative. Obtain an informed consent for the procedure. Position the patient upright in a multipositional procedure chair in a well-lighted environment. Provide tetanus prophylaxis as required. Provide anesthesia to the patient. Refer to Chapter 209 for the complete details regarding dental anesthesia and analgesia.

Irrigate the oral cavity and dental repair region with warm saline to remove any gross contaminants or clotted blood. Warm gentle irrigation is usually less sensitive to exposed dentin.[19] **A dry and uncontaminated field is mandatory.** This can be achieved via dabbing with sterile cotton pellets or gently blowing with compressed air. Maintain a dry working environment by isolating the traumatized tooth with sterile cotton rolls on either side. Inject a small amount of local anesthetic solution containing epinephrine directly into the pulp if continued bleeding from a large pulp exposure is problematic.

A nasal cannula was successfully used as an alternative by some to keep an area dry that was constantly oozing blood. Aim the nasal cannula at a flow rate of 10 to 15 L of air or oxygen at the site prior to and during the procedure to keep the area dry. If the flow interferes with the procedure, intermittently use the nasal cannula to dry the area.

TECHNIQUES

ELLIS I FRACTURES

Ellis I fractures are clinically minor injuries (**Figures 215-3 and 215-4*A***). Management includes smoothing of any sharp edges with a dental drill or a nail file to prevent injury or irritation to the soft tissues of the oral cavity. **Use care to avoid overly aggressive smoothing and exposure of the dentin (i.e., iatrogenic Ellis II injury).**

ELLIS II FRACTURES

Ellis II fractures are clinically more important than Ellis I fractures (**Figures 215-3 and 215-4*B***). They require coverage of the exposed dentin to prevent infection and reduce sensitivity. Paint small or large dentin exposures with calcium hydroxide paste covering all of the exposed dentin.[10,19,21] Apply the premixed calcium hydroxide paste in a thin layer with any small blunt instrument or the wooden handle of a cotton-tipped applicator. If not premixed, apply equal amounts of the calcium hydroxide base and catalyst on a piece of paper. Mix these two components thoroughly with a blunt instrument or wooden stick and then apply it to the tooth. **Allow the calcium hydroxide paste to completely dry.** Apply three to four coats of cavity varnish or clear acrylic nail polish over the dry calcium hydroxide paste. **Allow adequate drying time between the coats.** This will relieve any sensitivity. As an alternative to the cavity varnish or clear nail polish, place a tin foil dressing over the tooth to act as a bandage following placement of the calcium hydroxide paste.[10,19,20] With cautious care, a calcium hydroxide dressing will typically last 2 to 3 days.[22]

ELLIS III FRACTURES

Ellis III fractures are true dental emergencies and should be treated by a Dentist or Oral Surgeon (Figures 215-3 and 215-4*C*). Treat pulp exposures, if no specialist is immediately available, by applying a saline-moistened or lidocaine-moistened cotton pledget over the exposed pulp and holding it there until the bleeding stops. This may take 2 to 5 minutes. Apply a thick mixture of calcium hydroxide paste over the exposed pulp and dentin. Apply the calcium hydroxide paste over an adjacent tooth also to form a temporary hold. Contour the calcium hydroxide paste so that it does not irritate the surrounding tissues. The majority of these injuries will require root canal therapy. Refer the patient to a Dentist within 24 hours if they are not seen by a specialist in the Emergency Department.

ASSESSMENT

Reassess the patient for pain and any occlusal discrepancies prior to discharge.

AFTERCARE

Most of these patients will have some degree of sensitivity until definitive treatment by a Dentist or Oral Surgeon. Prescribe appropriate outpatient analgesics. Nonsteroidal anti-inflammatory drugs supplemented with a narcotic analgesic will provide adequate pain control for Ellis II and III fractures. Antibiotics are generally not necessary unless the initial presentation had been significantly delayed, suppuration is present, or a significant delay is expected in obtaining dental follow-up. Instruct the patient to avoid extremely hot or cold substances, to begin a liquid or soft diet until seen by a Dentist or Oral Surgeon, to avoid chewing in the area of the injured tooth, and to avoid topical analgesics (e.g., oil of cloves) due to the propensity for sterile abscess formation.[2,4,19] Always warn patients about continued sensitivity. Provide instructions to maintain good dental hygiene, to avoid further trauma, and to follow up with a Dentist or Oral Surgeon for serial examinations.[27]

Inform the patient and/or their representative what will happen upon follow-up. If a fractured tooth is a primary tooth, the patient usually receives a pulpotomy with removal of the nerve tissue in the crown of the tooth. The primary tooth will be replaced by a permanent tooth. Permanent teeth have a worse prognosis and it should be made clear. Another tooth will not replace the fractured tooth and it will likely receive a pulpotomy. The pulpotomy involves removal of a portion of the nerve tissue followed by sealing the hole. Follow-up is frequent after a pulpotomy to make sure there are no problems involving the root of the tooth.

COMPLICATIONS

Complications of dental trauma include pain, cosmetic deformity, loss of tooth viability, and unsuspected or unrecognized injury to adjacent teeth with later complications. A permanent tooth may develop abnormally in a younger child if injured. Infection can form locally and advance to an abscess, osteomyelitis, and/or systemic infectious complications. Ensuring prompt follow-up with a Dentist can abate the majority of these complications.

SUMMARY

Dental fractures are relatively common traumatic injuries. Appropriate clinical assessment and treatment requires an understanding of basic dental anatomy, terminology, and pathophysiology. Management includes addressing patient discomfort, dental stabilization, and coverage of exposed vulnerable tooth components. Arrange prompt follow-up with a Dentist or Oral Surgeon for definitive care of any dental injury. The Emergency Physician should be ever vigilant for cases suspicious for domestic adult abuse, child abuse, or child neglect.

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216 Temporomandibular Joint Dislocation Reduction

Marilyn M. Hallock

INTRODUCTION

Mandible or temporomandibular joint (TMJ) dislocations usually occur in the setting of prior musculoskeletal problems of the jaw.[1-5] This includes joint laxity, prior injury or dislocation, inherent hypermobile syndromes (e.g., Marfan's, Ehlers-Danlos), or neuromuscular problems (e.g., dystonic reactions) that pull the mandible out of its joint. The mandibular dislocation typically results from TMJ hyperextension or trauma. The Emergency Physician must be able to reduce a TMJ dislocation. The procedure is easy, simple, and straightforward.

ANATOMY AND PATHOPHYSIOLOGY

The TMJ is an unusual joint (**Figure 216-1**). It is composed of two joints separated by an articular disk.[6] The TMJ functions as a hinge and gliding joint. A discussion of the mechanics of the TMJ is beyond the scope of this chapter. Anterior dislocations are most commonly seen in the Emergency Department. The etiology of the dislocation includes laughing, chewing, iatrogenic from procedures, opening the mouth wide (e.g., eating, for procedures, yawning, vomiting, singing), seizures, and trauma.[7-10] These actions can result in the mandibular condyle sliding forward and anterior to the articular eminence of the temporal bone. Anatomic abnormalities of the TMJ have a greater predisposition for mandibular dislocation. These include a shallow articular eminence, weak or torn temporomandibular ligaments, an overstretched joint capsule, previous TMJ dislocations, or hypermobile syndromes (e.g., Marfan's or Ehlers-Danlos syndrome).[11] The muscular attachments of the mandible result in a pulling of the condyle superiorly and in front of the articular eminence (**Figure 216-2**).[10] This causes the mandible to become fixed in dislocation and rarely spontaneously reduces.[12]

TMJ dislocations are commonly anterior but may be in any direction.[13] Anterior TMJ dislocations may occur spontaneously in normal individuals and can occasionally reduce spontaneously. Dislocations of the TMJ can be unilateral or bilateral. Posterior, superior, and lateral dislocations are much rarer. They are seen in the context of direct trauma to the mandible with or without an associated mandible fracture, cervical spine fracture, or skull fracture.[14,15]

The diagnosis can often be made clinically in a cooperative patient with a nontraumatic history. The patient will present in pain with an open mouth, pain anterior to the ear, protruding mandible, and malocclusion.[16,17] **A depression, both palpable and visible, will be noted in the preauricular area.[16]** The mandible appears symmetrical in bilateral anterior dislocations and deviated to the opposite side of the dislocation in the case of a unilateral anterior dislocation.[10]

Mandibular radiographs are indicated when trauma is involved to rule out an associated fracture. The dislocation is best seen on the Panorex view of the mandible (**Figure 216-3**). TMJ views, if available, are also useful. Computed tomography (CT) scanning (**Figure 216-4**) is warranted if an associated basilar skull fracture, intracranial injuries, or facial fractures are suspected.

INDICATIONS

Attempt to reduce a closed anterior TMJ dislocation without a concomitant mandible fracture in an alert, cooperative, and consenting patient.[18]

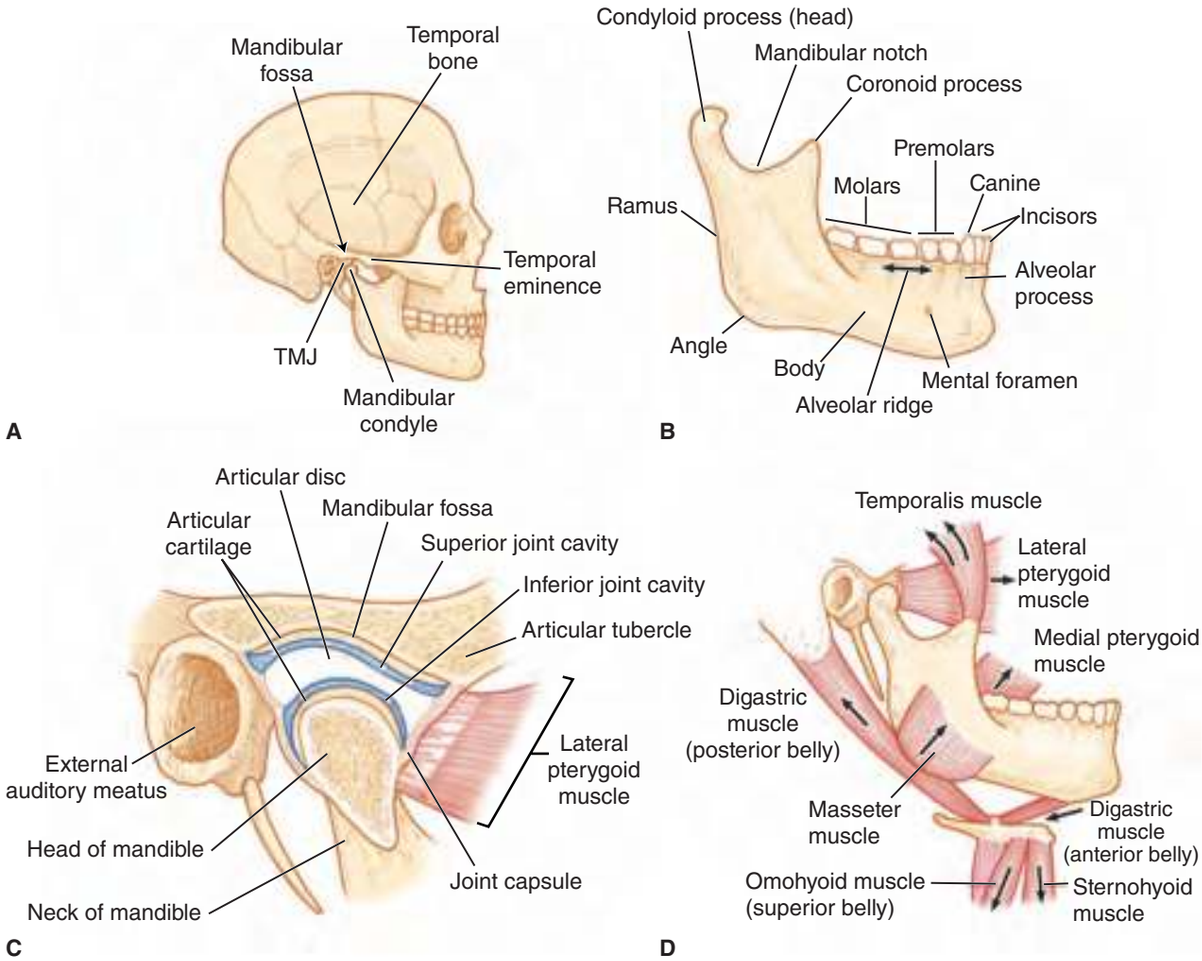


FIGURE 216-1. Anatomy of the TMJ. ***A.*** Lateral view of the head and TMJ. ***B.*** Anatomy of the mandible. ***C.*** Sagittal section through the TMJ. ***D.*** The attachment of the muscles of mastication to the mandible. The arrows represent the direction of pull of the muscles.

CONTRAINDICATIONS

Mandibular dislocations that are open, superior in direction, lateral in direction, or posterior in direction require an Oral Surgeon or Otolaryngology consultation prior to reduction attempts. Dislocations associated with mandible fractures require consultation prior to reduction attempts. The inability to reduce an anterior mandible dislocation by the closed method requires consultation and reduction under general anesthesia. Patients presenting with cranial nerve injuries associated with the dislocation require emergent consultation prior to the reduction.

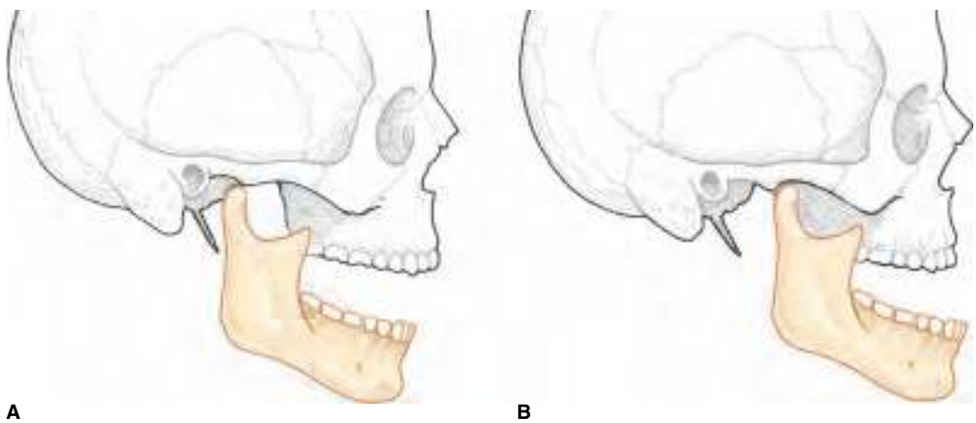


FIGURE 216-2. Anatomic relationships of the mandible. ***A.*** The fully opened mandible. ***B.*** An anterior TMJ dislocation.

FIGURE 216-3. Panorex radiograph of a bilateral mandibular dislocation. Note the mandibular condyle is anterior to the articular eminence of the temporal bone.

EQUIPMENT

- 25 gauge needle
- 3 mL syringe
- Gauze 4×4 squares or rolls
- Gloves
- Povidone iodine or chlorhexidine solution
- Local anesthetic solution without epinephrine
- Equipment and supplies for procedural sedation (Chapter 159)

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain a signed informed consent for the procedure. Place the patient sitting in a multipositional procedure chair with a solid headrest to support their head. Alternatives include placing the patient supine on a gurney or in a chair with an assistant standing behind the patient to stabilize their head (**Figure 216-5**).

The mandible can often be reduced without anesthesia. This is not recommended. **Adequate analgesia and muscle relaxation will allow easier manipulation of the mandible back into its anatomic position.** Strongly consider the use of parenteral analgesics, sedatives, and/or muscle relaxants. Procedural analgesia and sedation (Chapter 159) may be required to overcome the patient's pain and masticatory muscle spasm. This is especially true to relax the muscles of mastication and allow reduction if the mandible has been dislocated for more than 6 to 8 hours.

FIGURE 216-4. Sagittal computed tomography scan image demonstrating the mandibular condyle (arrow) is dislocated anteriorly to the articular eminence (arrowhead). (Used with permission from reference 10.)

FIGURE 216-5. Alternative positioning of the patient.

An alternative, or adjunct, to the administration of parenteral medications is to inject local anesthetic solution into the TMJ space (**Figure 216-6**). This injection is simple, quick, and relieves significant discomfort. The local anesthetic blocks the pain receptors from the TMJ causing less muscle of mastication spasm. Locate the depression 2.5 cm anterior to the tragus of the pinna and just above the head (i.e., condyle) of the mandible. This is the location of the TMJ space. Clean the skin of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the skin over the TMJ space and allow it to dry. Insert a 25 gauge needle perpendicular to the skin and directed medially. Advance the needle 0.5 cm and

FIGURE 216-6. Anesthesia of the TMJ. Insert the needle 2.5 cm anterior to the tragus of the auricle and just above the mandibular condyle.

into the TMJ space. Inject 1 mL of local anesthetic solution without epinephrine. Inject the local anesthetic in the subcutaneous tissues as the needle is removed. Inject the contralateral TMJ space if the dislocation is bilateral.

Consider injecting the contralateral TMJ space in a unilateral TMJ dislocation. The patient may experience pain in their contralateral TMJ from muscle spasm due to the increased pressure on it from the contralateral TMJ dislocation. The TMJ injection may reduce some dislocations.[19]

TECHNIQUES

CLASSIC OR BIMANUAL INTRAORAL TRACTION TECHNIQUE

The actual reduction requires no specialized equipment beyond nonsterile gloves and gauze 4×4 squares. Position the patient as mentioned previously. Apply gloves and then wrap several layers of gauze around each thumb. **The gauze squares on the thumbs are to prevent possible lacerations to the Emergency Physicians thumbs when the mandible reduces.**

This technique is the most commonly used method to reduce an anterior TMJ dislocation and is described as bimanual intraoral traction.[20] Stand or sit in front of the patient (**Figure 216-5**). Place both thumbs into the patient's mouth and onto the most posterior molars of the mandible bilaterally (**Figure 216-7**). Alternatively, place the thumbs on the mandibular ridge immediately posterior to the molars. Wrap the index, middle, ring, and little fingers below the mandible with the index fingers behind the angles of the mandible (**Figures 216-7**). Slowly apply inferior (i.e., downward) and then posterior (i.e., backward) pressure to allow the muscles of mastication to stretch and overcome the muscle spasm (**Figure 216-7**). The downward pressure releases the mandibular condyle from the articular eminence of the temporal bone while manipulating the condylar head and seating it back in the articular glenoid fossa.

Instruct the patient to open their mouth further to relax the elevators of the mandible and to accentuate the deformity. This may disengage the impacted mandibular condyle from the anterior articular eminence of the TMJ. The masseter muscle will cause a rapid and sudden closing of the patient's jaw when the condyle of the mandible snaps back and over the articular eminence.[21]

This classic technique has many disadvantages. It requires much force to reduce the dislocation. The risk of a bite injury to the Emergency Physician is real and might lead to a transmittable disease (e.g., hepatitis, herpes, or HIV). This technique often requires procedural sedation to overcome the muscles of mastication. Repeated attempts may be required to reduce the dislocation.

ALTERNATIVE TECHNIQUES

Numerous alternative techniques have been developed to reduce a TMJ dislocation. They are described below and may work to reduce the TMJ dislocation. Many of these were case reports or a small series of patients. These are too small to make a recommendation and are not endorsed by the author of this chapter and book editor. Nonetheless, they are worth trying.

CORK TECHNIQUE

This technique uses a cork to reduce the TMJ dislocation.[22,23] Place a cork between the posterior molars on each side. Push up on the mental process of the mandible and use the corks as fulcrums. A cork is hard to find today in the modern hospital. Try a rubber stopper from a test tube as a substitute for the cork. The technique risks the aspiration of the corks or rubber stoppers.

SYRINGE TECHNIQUE

A syringe can be used in place of the cork or rubber stopper.[9] The size of the syringe will vary based on the ability to fit the syringe in the patient's mouth and the space between the molars. Use as large of a syringe as possible. This technique has a high failure rate with traumatic TMJ dislocations.[9] Place the syringe as far back in the patient's mouth and between the posterior molars on the dislocated side. Instruct the patient to gently bite the syringe while rolling it back and forth in their mouth. The syringe is used as a rolling fulcrum.

POSTERIOR INTRAORAL TECHNIQUE

The technique requires the Emergency Physician to stand behind the sitting or lying patient.[24,25] Place inferiorly (i.e., downward) and posteriorly (i.e., backward) pressure on the lower molars (**Figure 216-8**).[24] Alternatively, place the thumbs on the retromolar gums and apply inferiorly (i.e., downward) and anteriorly

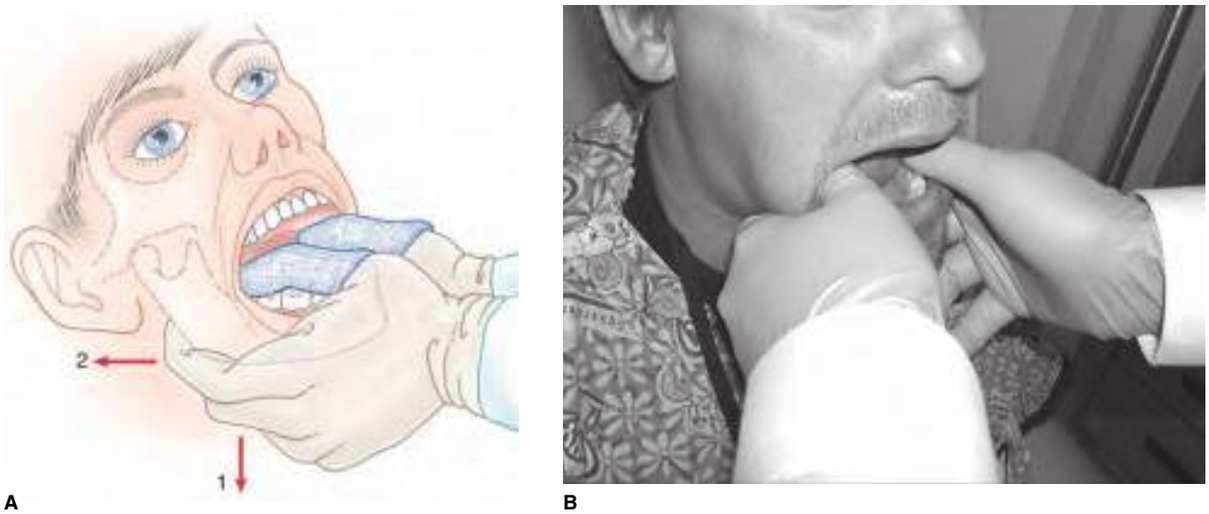


FIGURE 216-7. The classic or bimanual intraoral technique. Proper thumb and hand placement for the reduction of an anteriorly dislocated mandible. ***A.*** Artist illustration. Apply downward pressure with the thumbs (1) followed by posteriorly directed pressure (2) to reduce the dislocation. ***B.*** Clinical photo. (Used with permission from reference 4.)

A

B

FIGURE 216-8. An alternative method to perform the classic or bimanual intraoral technique to reduce an anterior TMJ dislocation. ***A.*** Artist illustration. The patient is supine. Apply inferiorly (i.e., downward) and posteriorly (i.e., backward) pressure on the molars with the thumbs (1) followed by posteriorly directed pressure (2) to reduce the dislocation to reduce the dislocation. ***B.*** Clinical photo. (Used with permission from reference 4.)

(i.e., forward) pressure on the lower molars.[25] This technique has the same disadvantages as listed for the classic technique.

ALTERNATIVE INTRAORAL TECHNIQUE

An alternative technique is to place the thumbs on the buccal aspect of the molars while the patient is sitting or lying. This avoids potential injury to the thumbs when the mandible relocates. It is performed by standing in front of the patient. This method limits the force placed upon the mandible and may not result in a reduction. It is hard to grasp the wet gingiva and the sides of the molars.

GAG REFLEX TECHNIQUE

Inducing a gag reflex with a tongue depressor may relax the spasm of the muscles of mastication and free the condyle and has been used to successfully reduce an anterior TMJ dislocation.[26] The mandible snaps back into anatomic position.[26] The rapid and forceful snap of the mandible may result in tooth fractures.

UNIFIED HANDS TECHNIQUE

This technique was first described by Cheng.[27] Place both thumbs on the occlusal surface of the molars on the dislocated side (**Figure 216-9**). This may allow additional pressure to reduce the dislocation that is not reduced using the traditional method. This technique has the same disadvantages as listed for the classic technique.

FIGURE 216-9. The unified hands technique to reduce an anterior TMJ dislocation. (Used with permission from reference 27.)

WRIST PIVOT METHOD

The wrist pivot method has been described to reduce a TMJ dislocation (**Figure 216-10**).[16] Wrap the index and middle fingers of both hands with gauze. Place both thumbs under the patient's chin and the wrapped fingers of both hands on the patient's premolars and molars (**Figure 216-10**). Apply upward pressure with the thumbs while simultaneously applying downward pressure on the mandible with the fingers to unlock the mandible and reduce the dislocation. The mandibular angle acts as a fulcrum. This technique has the same disadvantages as listed for the classic technique.

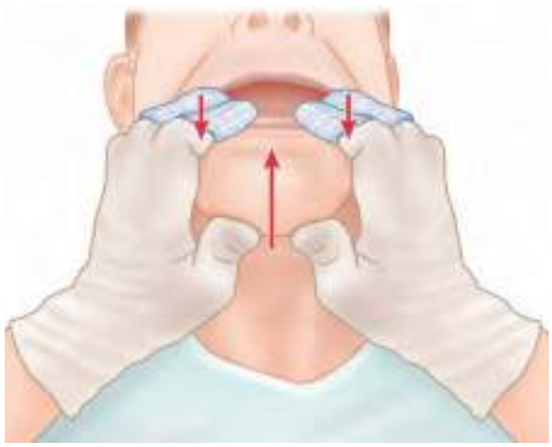
EXTRAORAL APPROACH

An extraoral approach has been described to reduce a TMJ dislocation (**Figures 216-11 and 216-12**).[28] The main advantage is the avoidance of hand bites and potential disease transfer. Each TMJ joint is reduced separately.

Place the patient sitting or supine. Stand in front of the patient. Place a hand on each of the patient's cheeks. Place a thumb above the dislocated coronoid process and the fingers of that hand behind the mastoid process. The fingers act as a countertraction force. Place the other thumb over the malar eminence with the fingers of that hand over the angle of the mandible. Pull the nondislocated side anteriorly with the fingers behind the mandibular angle and use the malar-applied thumb as a fulcrum. Simultaneously apply steady posteriorly directed pressure on the dislocated coronion process until the mandible relocates. The reduction on one side is often followed by spontaneous reduction on the other side.[28] This technique is less successful than the traditional technique.[28]

HIPPOCRATIC TECHNIQUE

The Hippocratic technique involves the mental process pulled inferiorly (i.e., downward) and simultaneously pushing the mandible posteriorly (i.e., backward).[29,30] There is little information on this technique.



A

B

FIGURE 216-10. The wrist pivot method to reduce an anterior TMJ dislocation. ***A.*** Artist illustration. ***B.*** Clinical photo. (Used with permission from reference 4.)

WEDGE METHOD

This method puts wood or cork in between the posterior molars to reduce a TMJ dislocation.[31] This method may result in tooth fractures.[31]

FIGURE 216-12. The extraoral approach to reduce an anterior TMJ dislocation from behind the patient. (Used with permission from reference 4.)

FORCEPS METHOD

A method was developed using specialized forceps.[32] Place the horseshoe-shaped jaws of a forceps between the upper and lower teeth on the dislocated side. Open the jaws of the forceps to force the teeth apart and apply posteriorly (i.e., backward) directed pressure to reduce the dislocation. This method is not recommended for the Emergency Physician.

HOOK REDUCTION

Some consultants openly reduce the dislocation in the Emergency Department. It is usually performed under general anesthesia in the Operating Room with the patient intubated.[33,34] A small incision is made under the zygomatic arch. The neck of the mandible is grasped with a hook. The hook is pulled to reduce the dislocation. This method is not recommended for the Emergency Physician.

ALTERNATIVE EXTERNAL METHOD

This technique requires a lot of force on the part of the Emergency Physician.[35] The patient is placed supine. Stand above the

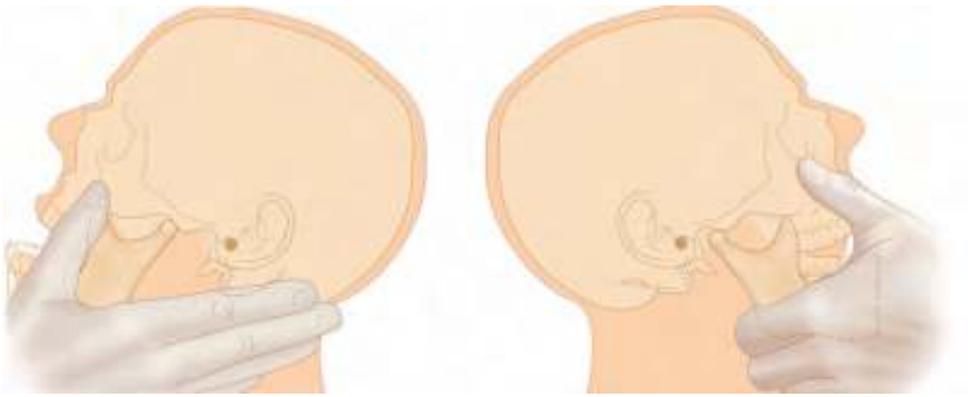


FIGURE 216-11. The extraoral approach to reduce an anterior TMJ dislocation.

patient's head. Hold the body of the mandible from the opposite side (i.e., under the chin). Instruct the patient to open and close their mouth. Move the mandible up and down in phase with the patient's opening and closing movements. Push on the dislocated condyle with a thumb and simultaneously push it obliquely downward while moving the body of the mandible upward. The dislocated condyle moves over the articular eminence and slips into the fossa.

TONGUE DEPRESSOR TECHNIQUE

Iserson described a technique used by many Oral Surgeons to reduce TMJ dislocations.[36] It is a progressive technique using tongue depressors to gradually reduce the dislocation. This technique can be used without anesthesia. Place tongue depressors on top of each other and between the molars on the dislocated side. Insert another tongue depressor when the spasm (i.e., pain) decreases. Keep adding tongue depressors, one at a time, until the dislocation reduces.

GONAI EXTRAORAL AUTOREDUCTION

This autoreduction technique uses the extraoral approach.[37] It stands for guided by physicians, opening and closing movements of the mandible, nonstress, autoreduction, and information sheet. This technique involves two steps. The first step is the patient identifying the mandibular condyle using an information sheet. Step two is the patient reducing the mandible. It has the advantages of being easy to explain to the patient, being performed with one hand, and not requiring equipment.

ASSESSMENT

The patient should be able to open and close their mandible without any difficulty after a successful reduction. Postreduction radiographs are not necessary unless a fracture was present or suspected on prereduction radiographs.

AFTERCARE

Refer the patient to an Oral Surgeon or Otolaryngologist within 24 to 48 hours.[38] Chronic or recurrent dislocations may require surgical fixation, intermaxillary wiring, surgical alteration of the articular eminence (e.g., eminectomy or eminoplasty), sclerosing of the TMJ, or the injection of botulinum toxin into the lateral pterygoid muscles.[39] Instruct the patient to avoid excessive jaw opening (i.e., over 2 cm), to avoid "gummy" foods and hard foods, and to begin a soft diet to avoid excessive strain on the TMJ. Instruct the patient to support their mandible with their hand when yawning so that it does not open widely and dislocate. Nonsteroidal anti-inflammatory drugs will provide adequate analgesia, if needed at all. The application of warm compresses to the TMJ may provide additional pain relief.

A Barton's bandage can be applied around the head to keep the mandibular condyle in its fossa and minimize mandible opening (**Figure 216-13**). This bandage is often applied for 2 weeks in patients with chronic dislocations or acute recurrences. Only apply a Barton's bandage in consultation with an Oral Surgeon or Otolaryngologist. Patients often do not like this bandage, are noncompliant, and remove it before follow-up. A soft cervical collar can be used to support the mandible and remind the patient to rest their jaw.[40]

COMPLICATIONS

Complications of the initial injury include fractures, intrusion of the mandibular condyle into the external auditory canal (i.e., posterior dislocation) or basal skull (i.e., superior dislocation), cerebral contusions, facial nerve injuries, and middle or inner ear injuries with hearing and balance impairments. Recurrent dislocations are possible in the future after the mandible has been dislocated once.

FIGURE 216-13. The Barton bandage.

The reduction technique is rarely associated with complications. Significant pain after a successful reduction may signify a fracture or articular cartilage avulsion. Fractures are rarely iatrogenic. They are often present on initial radiographs but not identified until retrospectively examined. A fracture may be displaced during the reduction. The Emergency Physician's thumbs can be crushed and/or lacerated by the patient's teeth.

Closed reduction may be unsuccessful. The patient will require reduction under general anesthesia. This is particularly true of chronic dislocations, dislocations for longer than 12 hours, and recurrent dislocations.[41,42] An avulsed articular cartilage or articular disc may be interposed and prevent closed reduction.

SUMMARY

Uncomplicated anterior mandible dislocations can be managed with basic anesthesia and sedation techniques. A gentle and progressive reduction will allow the mandibular condyle to relocate into the TMJ space without significant complications. Dislocations that are complicated by fractures or by overlying skin damage, that are nonanterior in location, or that have associated neurologic injuries require an emergent consultation and reduction by an Oral Surgeon or an Otolaryngologist.

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217 Arch Bar Separation

Richard Dean Robinson, Amanda Catherine Pientka, and Phuc Ba Duong

INTRODUCTION

The evaluation and management of the airway is a crucial component of any critically ill medical or trauma patient. Any airway has the potential to be challenging. Certain situations complicate our standard approach to securing the airway and warrant attention. Mandibular and maxillofacial trauma complicate the standard approach to securing an airway. Patients presenting with hardware in place due to previous mandibular and/or maxillofacial fixation and requiring airway protection and/or intubation represent a potentially challenging situation. The technique for emergent mandibular and/or maxillofacial fixation release to facilitate airway rescue will be discussed in this chapter.

ANATOMY AND PATHOPHYSIOLOGY

Dental anatomy differs between pediatric and adult populations (Chapter 215). Variations among individuals are not uncommon. The basic anatomy of a tooth consists of the crown covered with enamel which sits above the gum line. The tooth root is embedded in the alveolar bone. The fully formed root apex present in adult patients consists of alveolar bone, periodontal ligament fibers, and the cementum cell layer.

The musculoskeletal anatomy of the mandible and maxilla is shown in **Figure 216-1**. Fractures of the mandible and/or maxilla can occur at several different locations (e.g., alveolar ridge, alveolar process, body, angle, and symphysis). The mandibular condyle interacts with the articular fossa of the maxilla to form the temporomandibular joint. The zygomatic process connects to the body of the maxilla superior to the alveolar process. Mandibular or maxillofacial trauma can cause instability at any of these sites.

Mandibular fixation with arch bars consists of a series of wires. These wires run horizontally across the alveolar bone of the mandible and maxilla to fixate and stabilize the dental arches (**Figure 217-1**). There are also vertical wires connecting the maxilla and mandible to eliminate any mandibular opening and closing motion (**Figure 217-2**). Cut the vertical fixation wires in the setting of airway compromise to facilitate emergent airway access or significant vomiting that may result in aspiration. The horizontal running arch bars are not involved in jaw separation and should be left intact.

The fixation technique (i.e., arch bars) used to stabilize mandibular and maxillary fractures carries a significant associated expense.[1] This depends on the facility and specialist placing the hardware. The mean total charges can range from $13,000 to over $25,000. It can take between 0.6 to 1.4 hours to place arch bars depending on the specific injury.

INDICATIONS

The decision to remove the hardware calls for careful consideration of the urgency of the patient's presenting symptoms.[2] The indications for emergent arch bar separation include the need for

FIGURE 217-1. Arch bar horizontal fixation apparatus. (Photo courtesy of Phuc Ba Duong, DDS.)

FIGURE 217-3. The wire cutters. ***A.*** Standard version from the maintenance department or a hardware store. ***B.*** A sterilizable hospital wire cutters.

emergent access to airway, vomiting with the potential for aspiration, or aspiration.

CONTRAINDICATIONS

There are no absolute contraindications to the separation of standard wired arch bars. Consider a surgical airway if arch bars are severely damaged and prohibit the proper separation of the patient's jaws and a patient's airway is an immediate concern.

FIGURE 217-2. Arch bar complete horizontal and vertical fixation apparatus. (Photo courtesy of Phuc Ba Duong, DDS.)

EQUIPMENT

- Wire cutters
- Hemostat
- Bone wax, optional
- Sterile 2×2 gauze squares
- Frazier suction catheter
- Suction source and tubing
- Light source, overhead or headlamp

Several sources are available for the wire cutters. The maintenance department may be contacted to borrow wire cutters (**Figure 217-3*A***). The Operating Room or central supply can be contacted for sterile wire cutters (**Figure 217-3*B***). These are no help due to the delays involved if the wire cutters are needed emergently. It is recommended to keep a pair of wire cutters in a secure location within the Emergency Department for easy access and use in an emergency.[2]

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain an informed consent for the procedure from stable patients. Document that an emergent procedure was necessary when unable to obtain informed consent due to impending airway compromise. Place the patient on a monitor. Insert and secure a large-bore intravenous catheter to facilitate parenteral medication administration and as a precursor to advanced airway management should the need arise. Medication to prevent nausea

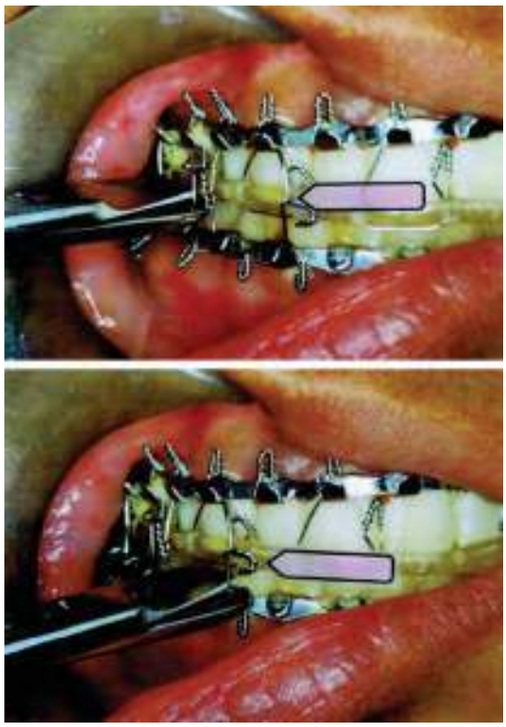

FIGURE 217-4. Cutting of the vertical wires (pink arrow) connecting the maxilla and mandible. (Used with permission from reference 2.)

may be useful. Be prepared to roll the patient onto their side to avoid complications (e.g., aspiration). Consider the use of local anesthetic in patients experiencing pain (Chapter 209). Use an overhead light or a headlamp to provide adequate illumination.

TECHNIQUES

Clamp the middle of the vertical wire with a hemostat for safety. Use a pair of wire cutters. Clip the vertical fixation wires in two places (**Figure 217-4**). Cut the wire near the attachment to the maxillary and mandibular arch bars. **Do not cut the horizontal arch bars or wires. They are not involved in jaw separation and should be left intact.** Remove the hemostat and cut wire. Cutting the wires from the maxilla to the mandible permits jaw opening, facilitates access to the airway, and allows the patient to expel any oral contents (e.g., vomitus).

PEDIATRIC CONSIDERATIONS

Pediatric patients may or may not be treated with conventional arch bars depending on their age. Children from ages 6 to 12 often have mixed dentition. Use caution when evaluating children in the setting of any dental or maxillofacial trauma.[3]

ASSESSMENT

Secure the airway and address the emergency after cutting the wires. Inspect the oral cavity for retained wires, broken dentition, and foreign bodies. Damage to oral structures or the arch bars may have occurred if the patient sustained trauma while the arch bars were in place.

AFTERCARE

Ensure no sharp edges from cutting the vertical wires that can cause injury to the patient. Cut these sharp wires or cover them with bone wax. Consult the Oral Maxillofacial Surgeon, Otolaryngologist, or Plastic Surgeon who placed the arch bars. Discuss the replacement of vertical wires after the emergency has been stabilized or resolved, whether additional operative interventions or procedures are required, and any additional care. Ensure adequate follow-up if the patient is discharged from the Emergency Department and provide appropriate analgesia.

COMPLICATIONS

Exercise caution while separating and cutting the wires. Cut wires can puncture gloves, puncture or cut skin, and potentially transmit infection. Use care to minimize iatrogenic trauma during arch bar separation. Approach wire cutting as if the wires and wire cutters were sharp needles. The patient can receive intraoral punctures or cuts from the wire cutters or the cut wires. Routine plain radiography is not indicated in the setting of arch bar removal.[4]

The cutting of wires can result in pieces of the wire being aspirated or lost. Clamping the vertical wire with a hemostat before cutting can prevent this. **Ensure the removal of the cut wire if not using a hemostat.** Obtain a Panorex or bilateral mandible radiographs to evaluate for missing wires. Obtain chest and abdomen radiographs if the dental radiographs are negative. The missing fragment seen on radiography requires an emergent consult with the appropriate specialist for removal.

SUMMARY

Patients with arch bars present a unique challenge to the Emergency Physician. Arch bars limit the ability to evaluate and access the airway. The cost of mandibular fixation is significant. The decision to remove or separate vertical wiring must be weighed against the urgency of the patient's presentation. Identification of the proper wires to cut are vital in a situation where immediate lifesaving action is undertaken. Wire cutters should be readily available in the Emergency Department for use in these situations. Care should be taken during and after arch bar separation to prevent injuries from the wire cutters and cut wires. Handle the wires as if they were needles. Examine the patient for loose hardware or foreign bodies. Arrange adequate consultation and follow-up.

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SECTION 15

Podiatric Procedures

218 Ingrown Toenail Management

Sean Dyer and Jeff Schaider

INTRODUCTION

An ingrown toenail (onychocryptosis) is a common affliction that can occur in any toe. It most commonly afflicts the great toe, occurring when the lateral edge of the nail plate penetrates the soft tissue of the lateral nail fold. There are three stages of ingrown toenails.[1] Stage I includes erythema, slight edema, and pain when pressure is applied to the lateral nail fold. Stage II includes the stage I findings plus signs of infection and a purulent drainage. Stage III is a magnification of the two previous stages with the addition of granulation tissue formation and lateral nail fold hypertrophy. Most ingrown toenails can be definitively managed in the Emergency Department by the Emergency Physician.

ANATOMY AND PATHOPHYSIOLOGY

The toenail usually does not grow into the soft tissue. Instead, the soft tissue overgrows and obliterates the nail sulcus in response to external pressure and irritation.[2-6] The toenail itself is usually normal, although some older patients may have incurved nails. The causes of an ingrown toenail are multiple and include trimming the toenails too short, using sharp tools to clean the toenail gutters, wearing improperly fitted (i.e., too tight) shoes, rotated digits, and bony deformities. Improper toenail trimming can result in a small nail spike on the lateral aspect of the toenail (**Figure 218-1**). As the toenail continues to grow, the spike will irritate the soft tissue causing the end result of chronic inflammation and an infection.

INDICATIONS

Warm soaks, oral antistaphylococcal antibiotics, and shoes with an adequate toe box may be curative in mild cases (stages I and II). Elevate and maintain the nail edge above the soft tissues or trim the edge of the nail (**Figure 218-2**). More severe cases (stage III) require partial toenail removal. For those with incurved toenails or abnormally wide toenails, partial nail avulsion and phenol treatment are usually indicated as simply instructing on proper toenail cutting is not sufficient.[7] When caused by nail spikes from improper nail trimming, usually in younger populations, these infrequently require partial nail removal and can be treated conservatively with instruction on proper nail trimming and proper fitting shoes. Have a lower threshold for toenail removal in diabetic patients to prevent a more severe infection from forming. Other indications for removal of an ingrown toenail include chronic or recurring ingrown toenails, failure of conservative therapy, fungal infections of the toenail, and severe pain.

A

B

FIGURE 218-1. An ingrown toenail. ***A.*** Photograph. (Used with permission from Sherman S: *Simon's Emergency Orthopedics*, 7th ed. New York: McGraw-Hill; 2015.) ***B.*** Artist drawing noting the nail spicule and the overgrowth of the adjacent soft tissues.

A B

FIGURE 218-2. Management of stage I and II ingrown toenails. ***A.*** Trimming the lateral nail edge. ***B.*** Elevation of the lateral nail edge.

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CONTRAINDICATIONS

The only relative contraindication to toenail removal is a decreased vascular supply to the toe. Trim the toenail edge if possible and minimize any injury to the adjacent soft tissues. These patients require an evaluation by a Podiatrist and a Vascular Surgeon to minimize future complications.

EQUIPMENT

GENERAL SUPPLIES

- Povidone iodine or chlorhexidine solution
- Sterile drapes
- Sterile gloves
- Curved hemostat
- Freer or another periosteal elevator
- Cotton
- Scissors or nail splitter
- Tourniquet or sterile Penrose drain
- Curette
- Topical antibacterial ointment
- 4×4 gauze squares
- Tape, 1 inch wide

CHEMICAL MATRIX ABLATION

- Above listed general supplies
- Cotton-tipped applicators
- 89% phenol solution
- 70% isopropyl alcohol solution
- Silver nitrate matchsticks

SURGICAL MATRIX EXCISION

- Above listed general supplies
- #15 scalpel blade on a handle
- Needle driver
- 5–0 nylon suture

NAIL MATRIX CAUTERIZATION

- Above listed general supplies
- Electrocautery unit, disposable

PATIENT PREPARATION

Explain the risks, benefits, complications, and aftercare of the procedure to the patient and/or their representative. Obtain an informed consent for the procedure. Place the patient supine on a gurney or procedure table. Flex the patient's hip and knee so that the plantar surface of their foot is flat against the gurney. An overhead light source is essential to provide appropriate illumination. Perform a digit block using aseptic technique. Refer to Chapter 156 regarding the details of digital anesthesia. Although rare, an occasional nervous or uncooperative patient may require an intravenous anxiolytic or even procedural sedation prior to the digital anesthesia. The young child will require physical restraint with a sheet or commercial device (e.g., Papoose board) and procedural sedation. Apply povidone iodine or chlorhexidine solution over the involved toe and allow it to dry. Apply a sterile drape to delineate a sterile field.

TECHNIQUES

Manage early ingrown toenails (stages I and II) with conservative therapy with simple trimming of the distal nail or elevation of the nail. Remove the medial or lateral one-quarter of the toenail along with the germinal matrix at the base of the toenail for stage III ingrown toenails. The entire nail may be removed if both sides are ingrown. It may be necessary to prevent any new nail growth in the area once the nail has been removed. Three options include chemical ablation of the matrix, surgical excision of the matrix, and electrocautery of the nail matrix.[1] There is a recurrence rate of less than 5% when using the excision and phenol method.[7] This is the most effective treatment in a recent clinical review.[7] When the ingrown toenail is caused from hypertrophy of surrounding tissue, a referral to a Podiatrist is indicated for a debulking treatment. The treatment of the pediatric patient is no different than that of the adult patient.[6,7]

TOENAIL ELEVATION AND TRIMMING

Ingrown toenails in the first two stages can be trimmed or elevated to relieve the patient's symptoms (**Figure 218-2**). Trim the distal edge of the nail plate to remove the ingrown portion (**Figure 218-2*A***). Remove the distal one-third to one-half of the nail plate. Smooth the nail plate edge so that it will grow out freely. Remove any debris along the lateral nail fold (i.e., the paronychia) or nailbed.

An alternative is to elevate the edge of the nail plate (**Figure 218-2*B***). Insert the jaws of a hemostat so that one is above and the other is below the ingrown nail edge. Clamp the jaws of the hemostat onto the nail plate. Slowly rotate the hemostat up and away from the ingrown side to elevate the edge of the nail plate above the adjacent soft tissues (**Figure 218-2*B***). Insert a wad of cotton under the nail edge to maintain it above the adjacent soft tissues. Release the hemostat. Teach the patient and/or their representative this technique so that they can replace the cotton wad daily until the nail plate grows out and past the soft tissues. The main disadvantages of this technique are that the patient or their representative must elevate the nail edge and replace the cotton daily as well as maintain the nail plate elevation for 3 to 6 weeks. This can be quite a challenge, if it is even possible, in the young child.

TOENAIL REMOVAL

Apply a tourniquet along the base of the afflicted toe (**Figure 218-3*A***). The tourniquet may be a commercially available product for the digits or a Penrose drain. Refer to Chapter 129 for several examples of digital tourniquets. Separate the nail from the underlying nail bed. Grasp and stabilize the toe with the nondominant hand. If available, use a Freer periosteal elevator to lift the soft tissue off the lateral and proximal toenail. The elevator can also be used to separate the nail plate from the underlying nail bed, but this is optional. Insert one jaw of a curved hemostat under the distal toenail margin and along the medial or lateral side of the nail plate, depending upon which side is ingrown (**Figure 218-3*B***). Advance the hemostat until the jaw is at the proximal corner of the involved side of the ingrown nail (**Figure 218-2*C***). Grasp the nail by clamping the jaws of the curved hemostat on the toenail.

Dislodge the ingrown nail from the skin, the nail bed, and the nail matrix by rotating the hemostat away from the ingrown portion (**Figure 218-3*D***). Continue to rotate the hemostat until the entire ingrown portion of the nail is separated from the skin, the nail bed, and the nail matrix. A large and complete portion of the underlying toenail will emerge from under the skin fold (**Figure 218-3*E***). The nail plate might have broken and a significant piece may still be under the inflamed skin border if only a small amount of the nail is visible after rotating the hemostat. Expose this area and use the curved hemostat to remove any remaining nail plate.

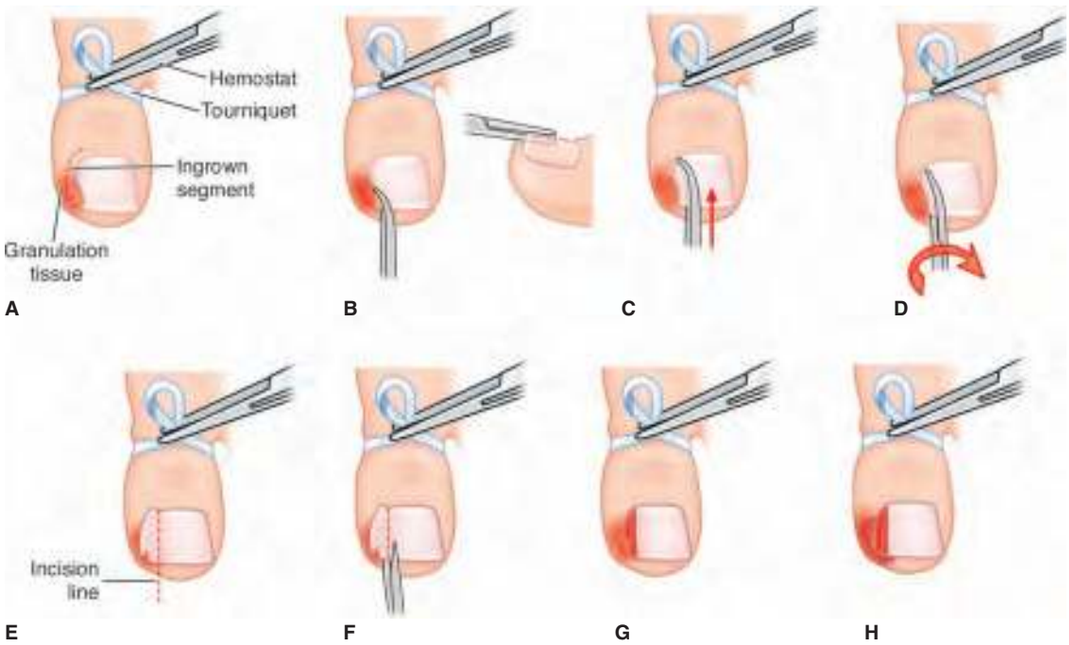

FIGURE 218-3. Ingrown toenail removal. ***A.*** Placement of a tourniquet. ***B.*** Place one jaw of the hemostat above the nail plate and one jaw beneath the nail plate. ***C.*** Advance the hemostat toward the base of the nail plate. ***D.*** Rotate the hemostat to elevate the edge of the toenail. Be sure to elevate and expose the proximal nail segment at the base of the nail in the region of the nail matrix. ***E.*** Determine where to cut the nail plate. ***F.*** Cut the nail plate with a heavy scissors or a nail splitter. ***G.*** The lateral one-fourth of the nail plate has been removed. ***H.*** The granulation tissue has been trimmed away.

Cut away the ingrown portion of the toenail, from distal to proximal, down to the nail plate in a straight line with a heavy scissors or nail splitter (**Figure 218-3*F***). **Make sure that the points of the scissors or nail splitter are facing upward to prevent injury to the nail bed.** The granulation tissue overlying the nail bed must be removed to prevent another ingrown toenail (**Figure 218-3*G***). Trim the granulation tissue using a #15 scalpel blade or a curette (**Figure 218-3*H***). Remove the tourniquet and control any bleeding.

Some Emergency Physicians prefer to perform the above described procedural steps in a slightly different order. They cut away the ingrown portion of the toenail before dislodging the remainder of the toenail. This is left to physician preference.

CHEMICAL NAIL MATRIX ABLATION

Chemical ablation of the nail matrix with phenol has several advantages.[1,8,9] The procedure is easy, quick, and simple to perform. No special equipment is required. The use of an incision or electrocautery and their associated complications is avoided.

Chemical ablation of the matrix with phenol is the author's preferred method. Remove the ingrown part of the toenail as described above. Remove any obvious remaining nail matrix and nail bed with a blunt instrument such as a curette. **Completely dry the field of any blood and fluid.** Dip a cotton-tipped applicator in a phenol solution. **Avoid excessive saturation of the swab.** Introduce the swab between the roof and the root matrix (i.e., under the eponychium) of the removed nail section (**Figure 218-4**). Rotate the cotton-tipped applicator slowly for 30 seconds in adults and 20 seconds for children. Remove the cotton-tipped applicator. Repeat the phenol application two additional times using a fresh phenol-soaked cotton-tipped applicator. **Do not allow the phenol to contact normal skin.[10] Immediately wipe off any phenol that contacts the skin.** The phenol will turn the tissue pale or gray. Dip a cotton-tipped applicator in isopropyl alcohol. Swab the area in similar fashion as the phenol swab. The isopropyl alcohol neutralizes the necrotizing effect of the phenol.[8-11]

An alternative to phenol is silver nitrate. The preferred technique is a phenol matrixectomy. Silver nitrate may be used if phenol is not available. The main disadvantage of silver nitrate is that it turns the tissues black. Insert the silver nitrate matchstick under the eponychium (**Figure 218-4**). Roll the matchstick around for 5 to 10 seconds to ablate the matrix.

SURGICAL NAIL MATRIX ABLATION

Surgical excision of the toenail matrix requires more time and experience than chemical ablation.[5] This technique is usually reserved for the Podiatrist or the Orthopedic Surgeon. An experienced

FIGURE 218-4. Chemical nail matrix ablation.

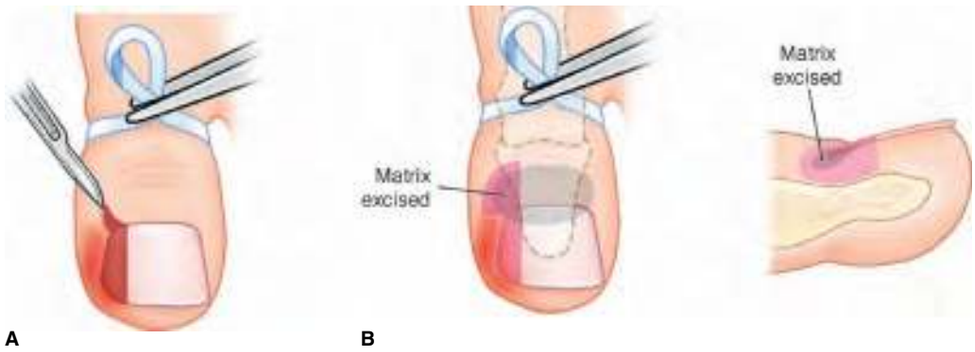

FIGURE 218-5. Surgical nail matrix ablation. ***A.*** An incision in the eponychium to expose the matrix. ***B.*** Removal of the matrix. The pink shading represents the areas of tissue to be removed.

Emergency Physician can easily perform this technique in the Emergency Department.

Remove the ingrown part of the toenail as described above. Expose the nail matrix by retracting the adjacent overlying skin. Make an oblique proximal incision from the proximal corner of the nail if necessary to fully expose the nail matrix in the ingrown area (**Figure 218-5*A***). Make an incision with a #15 scalpel blade to separate the nail matrix to be removed from the remaining nail and matrix (**Figure 218-5*B***). Grasp the corner of the matrix with a hemostat. Use the scalpel blade to separate the matrix from the underlying tissues. Remove the nail matrix. Do not forget to remove the dorsal and deep matrix that envelops the base of the toenail under the skin fold. Remove any remaining nail matrix and nail bed with a curette. Close the skin incision with 5–0 nylon suture.

ELECTROCAUTERY NAIL MATRIX ABLATION

Electrocauterization of the nail matrix is rapidly performed but requires access to an electrocautery instrument. Apply electrocautery between the roof and the root matrix of the removed nail section to destroy the matrix in this area. Avoid excessive burning of the surrounding tissues. This technique can cause significant damage to normal tissue and should be reserved for the Podiatrist or the Orthopedic Surgeon.

AFTERCARE

Apply a topical antibiotic ointment and a small compressive gauze dressing over the toe. The patient requires follow-up in 24 to 48 hours for a dressing change and evaluation of the wound. Saturate the dressing with saline or sterile water to make the removal process less painful. Instruct the patient to elevate the foot for the first 2 to 3 days to prevent bleeding and edema. Large shoes, sandals, or cast shoes are best used in the immediate postprocedure days. The use of oral antibiotics is restricted for patients who are immunocompromised, have an associated cellulitis, have a postprocedure infection, or whose vascular supply to the toe is decreased. Prescribe nonsteroidal anti-inflammatory drugs supplemented with narcotic analgesics as needed for pain control. Instruct the patient to return to the Emergency Department immediately if they experience increased pain, develop a fever or a purulent discharge, or notice increased redness of the toe or foot. Demonstrate the proper method to trim toenails (**Figure 218-6**).

Chemical ablation of the matrix with phenol induces a chemical burn.[10] The patient may experience a serous drainage for a few days up to 2 weeks.[2] The use of nonsteroidal anti-inflammatory drugs can limit the duration and the amount of drainage.[2] Instruct the patient to soak the foot in warm water three times a day for 10 to 15 minutes each time. Apply a topical antibiotic ointment after each soak. Prolonged drainage may be due to a superficial infection and requires evaluation.

COMPLICATIONS

The most common complication is the persistence of toenail horns or spikes due to incomplete ablation of the nail and matrix. These can be managed with nail trimming if mild or en bloc excision of the area if severe. Ensure that the portion of the nail is completely removed and that no fragments remain under the nail folds. Use care to not lacerate the nail bed when elevating or cutting the nail plate. Lacerations of the nail bed can bleed significantly, cause chronic pain once healed, and result in a deformed nail. Repair any nail bed lacerations. Refer to Chapter 129 for a complete discussion regarding nail bed repair.

Phenol will deteriorate if it is exposed to air or light. Store the phenol solution in a cool, dark place. Replace the solution frequently. The field must be dry before applying phenol. Phenol mixed with blood results in an alteration of the pH of the phenol, decreasing its effectiveness and turning the tissues black. The ingrown toenail will recur if the phenol is old or exposed to light before it is used, if the phenol is not properly applied, or if fragments of the toenail or matrix remain. The patient may experience a chemical burn if too much phenol is applied or it is not neutralized with isopropyl alcohol.

SUMMARY

An ingrown toenail can be managed easily, quickly, and definitively in the Emergency Department. Perform a partial toenail removal on patients with clinical stage III toenails characterized by pain,

FIGURE 218-6. The technique of toenail trimming. ***A.*** Correct. ***B.*** Incorrect.

overgrowth of inflamed and infected tissue, and drainage. Remove the lateral or medial one-quarter of the nail. Apply phenol solution to the nail matrix to prevent further growth of the nail and a recurrence.

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219 Plantar Puncture Wound Management

Vishal Bhuva and Dhara Amin

INTRODUCTION

Plantar puncture wounds are frequently encountered in the Emergency Department (ED).[1] They account for approximately 7% of patients presenting to the ED with lower extremity trauma.[2] Wounds are more likely to occur in the warmer summer months from May through October when people spend more time outdoors and barefoot. Nails produce the majority of puncture wounds followed less commonly by wood, glass, and other metal objects.[2-5] Patients seek medical treatment following puncture wounds for many reasons including tetanus immunization, pain relief, retained foreign bodies, and treatment of established infections.

There are very little data regarding the proper management of plantar puncture wounds but it is clear that complications can and do arise.[1-17] Infection and retained foreign bodies remain the most serious of these complications. The true risk of infection and osteomyelitis remains unknown. Rates of infection are estimated to be in the range of 6% to 11%, with only a fraction of these complications proceeding to osteomyelitis.[2,4] Risk factors for the development of infectious complications have been identified and include puncture wounds to the forefoot, punctures through rubber-soled shoes, deep penetration, and diabetics (particularly those with neuropathy).[6,7]

One of the difficulties in reporting the incidence of plantar puncture wounds is that patients do not always present to the Emergency Department. Most patients remove the offending foreign body and never seek medical treatment. They present to the ED when there is a suspicion of a retained foreign body or an infection.[8] Despite the high frequency with which Emergency Physicians are faced with patients who have sustained a plantar puncture wound, there is no standard of care for treating these injuries. The pathophysiology and management of plantar puncture wounds are dependent on a host of factors including the location of the wound, the penetrating material, the depth of penetration, the footwear at the time of injury, the time to presentation, and any concomitant illnesses. This chapter summarizes the approach to the management of the plantar puncture wounds.

ANATOMY AND PATHOPHYSIOLOGY

The foot is a complex structure (**Figures 219-1 and 219-2**). The plantar surface is composed of the skin and a thin subcutaneous layer. The skin has a thickened stratum corneum layer making it one of the thickest areas of epidermis in the body. This thickened epidermal layer gives the plantar surface protection against mechanical forces. The plantar aponeurosis extends over the base of the foot and forms the plantar fascia. Deep to the fascia are various muscles and tendons with their sheaths. The longitudinal arch of the foot extends from the metatarsal heads to the calcaneus. The dorsal surface of the foot has a thin skin layer without much subcutaneous tissue. Under the thin subcutaneous layer is the superior dorsal fascia and the dorsal aponeurotic layer which encompasses the extensor tendon sheaths.

The thickened plantar epidermal layer prevents minor mechanical insults from penetrating the skin. An object can puncture the plantar surface of the foot and compromise the deeper layers of the foot. The impaling object may just breach the plantar fascia or even go through the foot depending on the depth of penetration. Puncture wounds occurring over the metaphalangeal joints are associated with greater complications as they often involve the joint spaces.[9,10]

The most common organisms causing soft tissue infections are *Staphylococcus* and *Streptococcus*. However, when osteomyelitis or osteochondritis occurs from a puncture through the sole of the shoe, *Pseudomonas* is the most frequent causative agent.[2,9] Studies have shown that *Pseudomonas* lives in actively used shoes and will enter the avascular cartilage layers of the foot during a puncture through a shoe.[9]

INDICATIONS

Determine if the penetrating object is small, such as a needle or pin, and if a portion may still be within the foot. If the patient was able to remove the object intact, no further treatment is required. The patient may be discharged with appropriate wound care instructions and asked to return if there are any signs of an infection. **Obtain soft tissue radiographs if the impaling object is broken off or the possibility of a retained foreign body exists.** Whereas metallic and glass foreign bodies are visible on plain radiographs, wood and plastic foreign bodies are generally not.[11,12] They require evaluation by ultrasound, computed tomography (CT) scanning (**Figure 219-3**), magnetic resonance imaging (MRI), or wound exploration.[2,3,5] Ultrasound continues to emerge as a powerful bedside tool for Emergency Physicians (**Figure 219-4**). However, the dense tissue planes of the foot limit its usefulness in detecting plantar foreign bodies.[11] **Blind wound exploration, blind probing, and blind irrigation are no longer supported.** These techniques have never been shown to improve outcomes, they are painful, and they are potentially harmful. A sample algorithm for penetrating objects is noted in **Figure 219-5**.

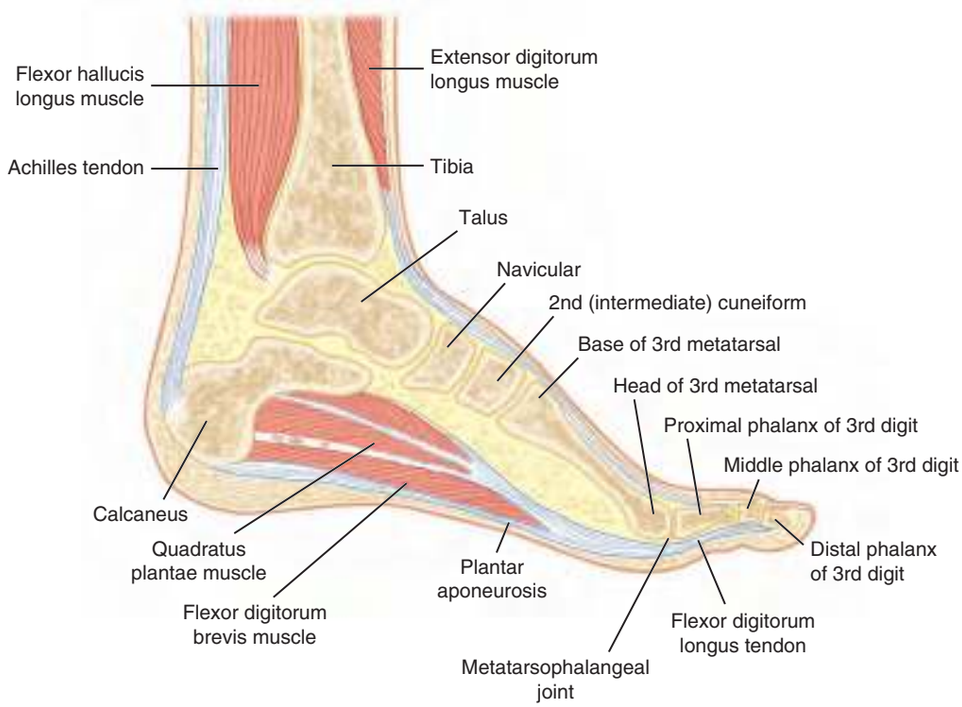

FIGURE 219-1. The anatomy of the foot in midsagittal section.

CONTRAINDICATIONS

Evidence of deep penetration, extensive penetration, or multiple retained foreign bodies should be evaluated in consultation with a Podiatrist or Orthopedic Surgeon. Additionally, manage patients who present more than 72 hours after the injury with evidence of an infection while taking antibiotics, a draining tract, or dorsal foot symptoms (e.g., erythema, pain, or swelling) in consultation with a foot specialist.[5,8,12] Surgical exploration and irrigation are required for penetration of any joint space or wounds overlying the metatarsal heads. Do not explore plantar puncture wounds if you are not experienced in or comfortable with the procedure.

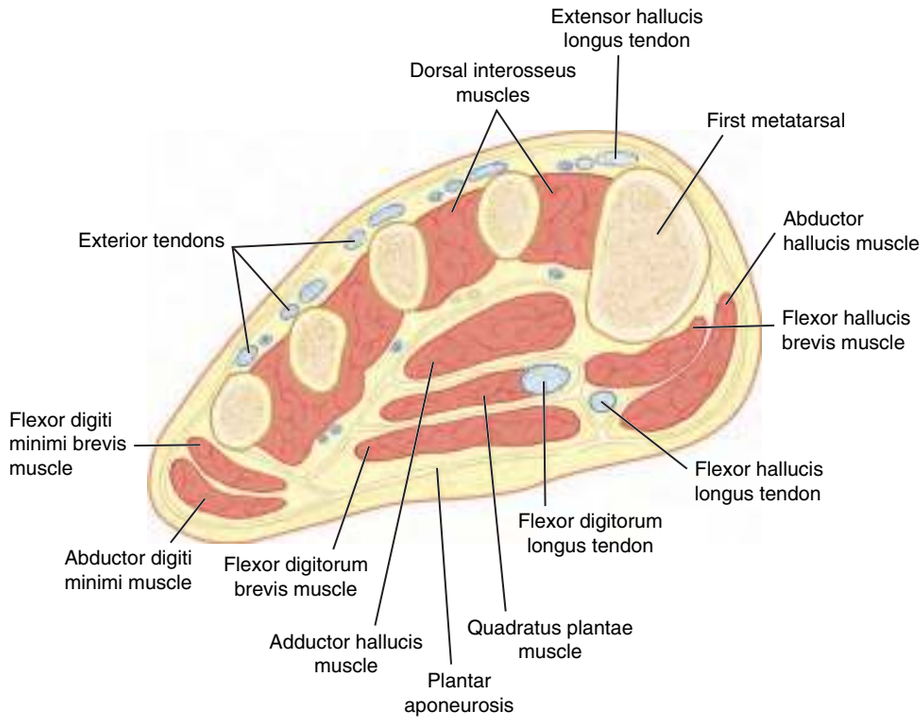

FIGURE 219-2. The anatomy of the foot. Cross-section just proximal to the metatarsal heads.

FIGURE 219-3. An example of a CT scan showing a wood foreign body in the left foot (arrow). (Used with permission from Schwartz DT: *Emergency Radiology: Case Studies*. New York: McGraw-Hill; 2008.)

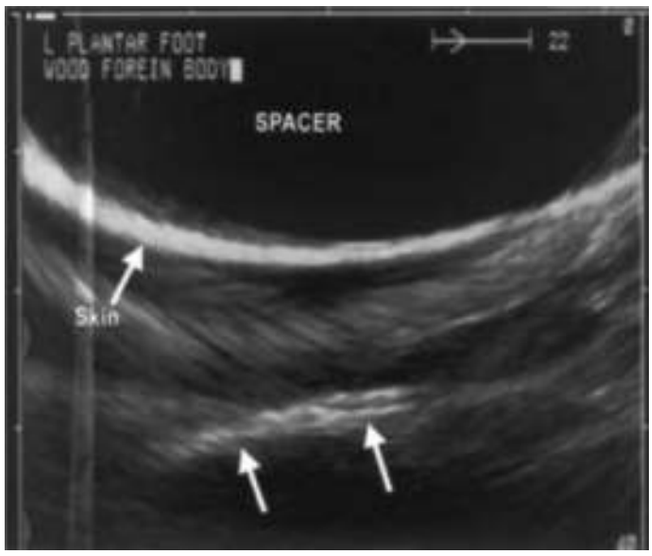

FIGURE 219-4. An example of an ultrasound scan showing a wood foreign body in the foot (arrows). (Used with permission from Dean AJ, Gronczewski CA, Costantino TG. Technique for emergency medicine bedside ultrasound identification of a radiolucent foreign body. *J Emerg Med* 2003;24(3):303-308.)

EQUIPMENT

- Povidone iodine solution
- 10 mL syringe
- Local anesthetic solution with or without epinephrine
- 23 to 27 gauge needle
- 4×4 gauze squares
- Eye protection or splash guard
- Gloves
- 15 mL syringe
- Sterile normal saline for irrigation
- 18 gauge plastic angiocatheter without the needle
- Surgical scalpel blades, #11 and #15, on a handle
- Cuticle clippers or fine iris scissors
- Curved mosquito hemostat
- Blunt probe
- Toothed forceps
- Circular corn pad
- Tubular cling
- Tape, 1 inch wide

PATIENT PREPARATION

Assess every patient with a puncture wound individually. Determine whether the penetrating object remains embedded in the foot and the cleanliness of the object. Was it contaminated or rusty? Identify the site of the incident. Was it outdoors or indoors? What type of footwear was worn at the time of the incident? Where in the foot is the puncture wound? Estimate the depth of penetration. Ascertain if the patient has any comorbid health conditions that may affect wound healing.[6] Does the patient need tetanus immune globulin (TIG) or a tetanus booster? If the patient has received less than three doses of tetanus toxoid, they should be given tetanus booster and TIG.[9]

Obtain plain soft tissue radiographs or a bedside ultrasound if there is suspicion of a retained foreign body, if an infection is present, if the patient presents more than 72 hours after incurring the injury, if the patient stepped on glass, or if the patient feels that a foreign body is present. Glass is radiopaque and visible upon plain radiographs.[6,8-12] The patient is often correct if they have a foreign body sensation and feel that a foreign body is "still there."[12]

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. Obtain a signed informed consent for the procedure. Place the patient supine or prone on a gurney. Remove any superficial foreign bodies. Clean the skin overlying the wound of any dirt and debris. Clean the skin posterior to the medial malleolus if a posterior tibial nerve block is to be performed. Apply povidone iodine solution to the skin and allow it to dry. Apply sterile drapes to delineate a sterile field.

Anesthetize the area with a posterior tibial nerve block if exploration and retrieval are indicated (**Figures 219-5 and 219-6**). Do not inject directly into the plantar surface of the foot. Perform a posterior tibial nerve block if anesthesia is required.[5] The posterior tibial nerve is superficial at the ankle midway between the medial malleolus and the heel (**Figure 219-6**). It lies between the tendons of the flexor digitorum longus and flexor hallucis longus muscles. It travels with and slightly posterior to the posterior tibial artery. The posterior tibial nerve divides at the inferior border of the calcaneus to form the medial and lateral plantar nerves which provide motor innervation to the intrinsic foot muscles. The lateral plantar

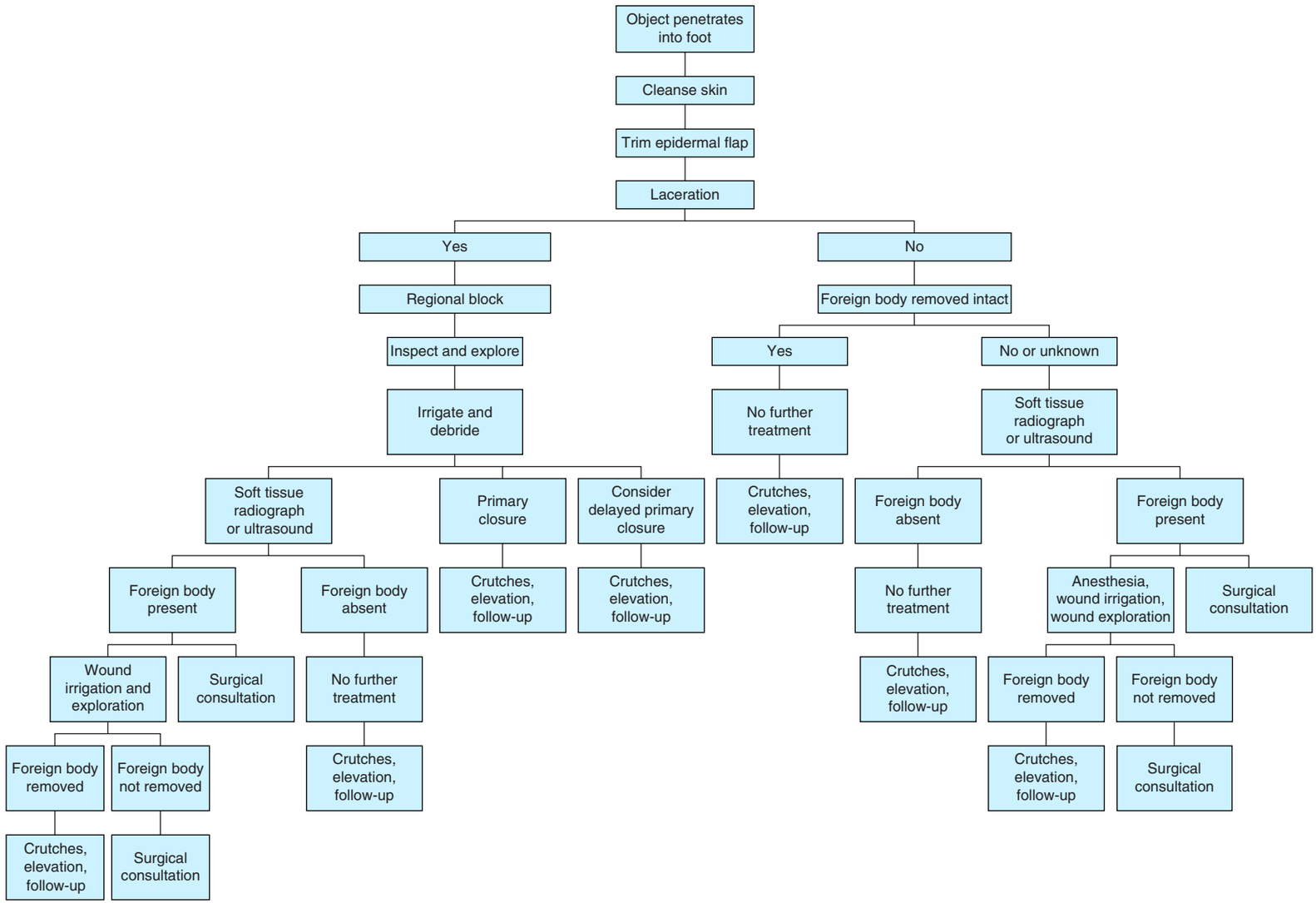

FIGURE 219-5. An example of a management algorithm for plantar puncture wounds. (Adapted from references 4 and 13.)

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A

B

FIGURE 219-6. The posterior tibial nerve block. ***A.*** The course of the posterior tibial nerve. ***B.*** Needle insertion and direction.

nerve provides sensory innervation to the lateral one-third of the sole and plantar surface of the lateral 1½ toes (**Figure 156-27**). The medial plantar nerve provides sensory innervation to the medial two-thirds of the sole and the plantar surface of the medial 3½ toes (**Figure 156-27**).

Place the patient supine with the ankle supported on a pillow or blanket and the leg externally rotated. Identify, by palpation, the medial malleolus and the posterior tibial artery by its pulsation. Place a skin wheal of local anesthetic solution at the level of the upper border of the medial malleolus and just posterior to the posterior tibial artery or medial to the Achilles tendon. Insert a 25 gauge needle through the skin wheal and perpendicular to the skin (**Figure 156-40**). Advance the needle to the tibia or until paresthesias are elicited. If paresthesias are elicited, withdraw the needle 2 mm and allow them to resolve. Inject 3 to 5 mL of local anesthetic solution. If paresthesias are not elicited, infiltrate 5 to 7 mL of local anesthetic solution starting against the posterior tibia as the needle is withdrawn.

TECHNIQUES

After cleaning the area carefully, debride any devitalized skin and callused edges of the stratum corneum overlying the wound with a pair of fine scissors or cuticle clippers. Obtain a culture swab of the wound if there is any evidence of infection.

It is often difficult to determine whether a joint space has been breached by the foreign body. Assume any puncture wound in the metatarsophalangeal joint area involves a joint space and consider consulting a Surgeon. Try to remove a foreign body within the wound. Avoid extensive exploration to prevent secondary injury to a joint space or tendon sheath. Refer deeply embedded foreign bodies to a Surgeon for exploration and removal in the Operating Room. Consider conservative therapy in patients presenting within 24 hours of the injury without preexisting impediments to wound healing or wound assessment.[4,9] Clean the area locally and keep patients non–weight bearing for 24 hours with follow-up within 48 hours.

AFTERCARE

Apply a donut-shaped corn pad around the puncture site. Apply topical antibiotic gel followed by a gauze dressing. The gauze may be held in place with tubular gauze or a gauze roll wrapped about the foot.

The use of prophylactic antibiotics is controversial. **There is no good evidence supporting the use of prophylactic treatment of plantar puncture wounds with antibiotics.**[4-6,8,9] Physician discretion is advised. Certain at-risk groups or injuries (e.g., diabetics, wounds over metatarsophalangeal joint, immunocompromised, or deep wounds) may be appropriate for treatment, although evidence is lacking even in these high-risk groups. Institute empiric antibiotic coverage for *Staphylococcus* and *Streptococcus* if this is a late presentation and there is evidence of cellulitis. Prescribe penicillinase-resistant antibiotics for 7 to 10 days guided by sensitivities. Patients who have diabetes or who are immunocompromised require broad-spectrum antibiotics, such as a third-generation cephalosporin or quinolone, and close follow-up. Consider antibiotic coverage that includes that for methicillin-resistance *Staphylococcus aureus*.

Patients who present more than 24 hours after a puncture wound need to be assessed for the retention of a foreign body.[14] If they fail a course of outpatient antibiotics, they require a full work-up to rule out osteomyelitis and a deep foreign body, not just a change to a new antibiotic. One study looking at pediatric puncture wounds advised that all children with an established infection 24 to 36 hours after a plantar puncture wound should be admitted for parenteral antibiotics.[12] They found that delayed presentation is significant for deep-seated infection. **Further infection or relapse after an initial treatment is suspicious for a retained foreign body or osteomyelitis.** A bone scan and an erythrocyte sedimentation rate (ESR) are advised for all patients suspected of having osteomyelitis. **A CT scan or an MRI scan is indicated for patients suspected of having a retained foreign body.**

Prescribe nonsteroidal anti-inflammatory drugs for analgesia. Instruct the patient to elevate the foot as much as possible in the next 48 hours. Adjunctive treatments such as epsom salts or tepid water soaks may have some benefit, but this has not been proven in clinical trials. Give the patient a wound care sheet and instruct them to return to the Emergency Department if they experience worsening pain, swelling, erythema along the dorsal surface of the foot, or a draining sinus. Patients should be non–weight bearing with crutches for 48 hours at which point they should be reassessed by a Primary Care Physician, Podiatrist, or Orthopedist.

COMPLICATIONS

The most frequently encountered complications of a puncture wound to the foot are cellulitis, soft tissue abscesses, osteomyelitis, foreign body granulomas, tendon lacerations, and nerve injury.[15] Symptoms persisting beyond 24 hours of the injury increase the possibility that complications will develop.[2-6,8] Late presenters with signs of cellulitis despite appropriate antibiotics may have a retained foreign body.[2,16] It is estimated that 8% to 15% of all puncture wounds of the foot progress to a cellulitis or a localized abscess. Cellulitis is often due to normal skin flora. Approximately 0.6% to 1.8% of patients develop osteomyelitis within 16 months of the injury.[2]

Osteomyelitis is a rare complication and is difficult to treat. ***Pseudomonas aeruginosa* is the most common organism isolated.** It is thought that the use of prior prophylactic antibiotics, increased regional humidity from wearing shoes, or the presence of a serous exudate from the wound may facilitate the growth of *Pseudomonas*.[4,6] The diagnosis of osteomyelitis can be made from clinical features such as a draining sinus visualized to bone or radiographic evidence of disruption of the bony cortex. Consider obtaining a CT scan, a bone scan, or radiolabeled white blood cell scan to make the diagnosis. Treatment involves surgical debridement and/or intravenous antibiotics for 4 to 8 weeks.[9,10]

SUMMARY

The paucity of clinical trials in this area of Emergency Medicine makes the management of plantar puncture wounds controversial. Simple and small plantar puncture wounds have a low incidence of complications. The presence of a retained foreign body and unknown depths of penetration make puncture wounds vulnerable to complications. A thorough history and physical examination are crucial to stratify patients for risk and to start initial therapy. Basic cleansing and debridement of devitalized tissue along with good follow-up and discharge instructions are the cornerstones of treatment. Tetanus prophylaxis should be reviewed with all patients. Prophylactic antibiotics are discouraged unless the wound is grossly contaminated.

Late presenters often have established wound infections requiring antibiotic treatment and appropriate referrals. Retained foreign bodies are a common cause of infections and require a thorough search. Osteomyelitis is a rare but serious complication. Involving Podiatric or Orthopedic Surgery, as well as using investigations (e.g., CT scan, bone scan, and ESR), can be important adjuncts for the treatment of these wounds. Frequent and timely follow-up remains the current standard of care to avoid infectious complications until further studies of antibiotic prophylaxis and initial puncture wound management can resolve the controversies.

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220 Toe Fracture Management

JoAnna Leuck and Tyler Hedman

INTRODUCTION

Toe fractures are the most common fracture of the foot. They result most commonly from a direct blow (e.g., from an object falling on an unprotected toe) or a "stubbing" injury.[1,2] The incidence of toe fractures has been estimated at 140 cases per 100,000 people per year, with the first and fifth toes being the most commonly affected.[1,3] The significance of toe fractures depends greatly upon which digit is affected. Most significant is a fracture of the great toe due to being the main propulsive segment of the forefoot.[4] Many patients do not present to the Emergency Department as they consider the injury trivial. Those who do present often do so because of severe pain and/or a large subungual hematoma.

Toe fractures are common injuries that rarely require surgical treatment. **The primary goals of treating toe fractures are reestablishing and maintaining alignment, regaining range of motion, preventing complications, and managing pain.** They may be completely and definitively managed in the Emergency Department. Most toe fractures require only a properly placed splint. Between 91% and 97% of toe fractures are nondisplaced or minimally displaced. These fractures often require a closed reduction.[1,5] An intraarticular fracture with severe displacement of the great toe may require an open reduction and internal fixation to prevent deformity and arthritis in the joint. Complications of toe fractures include damage to the articular cartilage, hypermobility of fracture segments, malposition, and malunion.

ANATOMY AND PATHOPHYSIOLOGY

The foot can be anatomically divided into the forefoot, the midfoot, and the hindfoot (**Figure 220-1**). The forefoot is composed of the metatarsals and their respective phalanges. Sesamoid bones often lie along the plantar surface of the metatarsal heads. The sesamoid bones of the great toe lie in a groove on the plantar surface of the metatarsal head and within the tendon of its respective flexor hallucis brevis muscle belly. Each toe has two pairs of digital nerves that course along the superior and inferior aspects of the digit. The

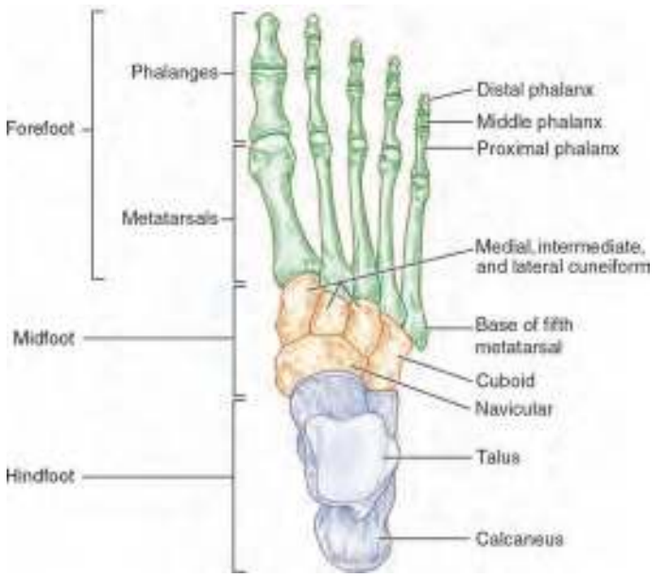


FIGURE 220-1. The bony anatomy of the foot.

digital artery and vein accompany the nerve. The great toe often receives superficial cutaneous nerves along its dorsal surface.

Anteroposterior and oblique radiographic views will demonstrate most fractures. Lateral views may be necessary to identify phalangeal fractures of the great toe. Obtain the lateral projection with toes 2 through 5 passively dorsiflexed to avoid overlap. An alternative method to achieve adequate radiographic views of the great toe in the lateral projection is to insert dental x-ray film between the first and second toes and direct the x-ray beam laterally.[6]

INDICATIONS

The indications for simple splinting of toe fractures are to relieve pain and allow for healing. The management of closed fractures depends upon the digit involved. Nondisplaced phalangeal fractures of the great toe can be managed with buddy taping to the adjacent normal toe as a splint. Some sources recommend a short leg cast with a toe plate or a short leg walking boot for 2 to 3 weeks for a great toe fracture.[4] Mildly displaced phalangeal fractures of the great toe can be reduced using local anesthesia, gentle traction, and then placement of buddy tape versus a short leg cast or boot.[4] Manage nondisplaced phalangeal fractures of toes 2 through 5 with buddy taping. Mildly displaced phalangeal fractures of toes 2 through 5 can be reduced using local anesthesia, gentle traction, and buddy taping. Exact anatomic reduction of toes 2 through 5 is not a concern if the general alignment of the toe is satisfactory.[7]

CONTRAINDICATIONS

Consult an Orthopedic Surgeon or Podiatrist for open fractures, extensive crush injuries to the forefoot, injuries with the potential to progress toward a compartment syndrome, intraarticular fractures, or severely displaced toe fractures. The incidence of arthritis and malunion is quite high with these injuries. Fractures of multiple toes on one foot cannot be treated with buddy taping.

EQUIPMENT

- Povidone iodine or chlorhexidine solution
- Local anesthetic solution without epinephrine
- 25 or 27 gauge needle
- 5 mL syringe
- 2×2 gauze squares or a soft corn pad
- Finger trap, optional
- Bunion pad
- Metatarsal bar or wooden tongue depressors
- Permeable tape, ½ inch wide
- Fluoroscopy unit, optional

The use of a fluoroscopy unit, if available, will facilitate an easier and quicker reduction technique. It allows for immediate evaluation of the reduction, multiple reduction attempts without the waiting time associated with serial plain radiography after each reduction attempt, and the reduction to be completed and splinted before the anesthesia wears off.

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. Obtain a signed informed consent for the procedure. Obtain plain radiographs or fluoroscopic images to assess the severity of the injury. Place the patient sitting upright so that the affected foot is suspended above the floor to allow adequate space for manipulation and splinting of the toe. An alternative is to place the patient supine with their hip and knee flexed so that the sole of the foot is flat against the gurney. Drain a subungual hematoma (Chapter 127) to relieve the pressure on the nail bed and improve patient comfort.

The use of anesthesia is Emergency Physician and patient dependent. Buddy taping of nondisplaced fractures of toes 1 through 5 or minimally displaced fractures of toes 2 through 5 requires no anesthesia. Consider performing a digital block (Chapter 156) if the patient is significantly tender and a displaced fracture must be reduced. Clean the web spaces of the affected toe of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the web space and surrounding skin. Allow the solution to dry. Arm a 5 mL syringe containing local anesthetic solution (e.g., lidocaine or bupivacaine) without epinephrine with a 25 or 27 gauge needle.

Insert the needle into the dorsal surface of the web space. Aim the needle 45° downward and toward the posterior aspect of the phalanx (**Figure 220-2*A***). Advance the needle while injecting 1 to 2 mL of local anesthetic solution. **Do not puncture the plantar surface of the toe.** The local anesthetic solution will easily inject into the areolar tissue of the web space. Withdraw the needle. Shift the needle so that it is aimed along the dorsal surface of the toe (**Figure 220-2*B***). Advance the needle while injecting 1 to 2 mL of local anesthetic solution over the dorsal surface. Completely withdraw the needle. Inject local anesthetic solution into the contralateral side of the affected toe in the same way as in the first web space. Allow 5 to 10 minutes for the block to take effect. Reassess the patient to determine whether the block was successful. An additional injection along the plantar surface of the toe may be required to provide total anesthesia, especially of the great toe.

An alternative is to perform a hematoma block (Chapter 155) by injecting local anesthetic solution directly into the fracture site. Clean and prepare the skin. Insert the needle over the fracture site. Advance the needle until it enters the fracture. Aspirate a small amount of blood to confirm that the tip of the needle is properly positioned within the fracture. Inject 2 to 3 mL of local anesthetic solution into the hematoma. Allow 5 to 10 minutes for the local anesthetic to take effect before proceeding.

A B

FIGURE 220-2. Digital block of the toe. ***A.*** Needle position and direction for infiltration of the lateral surface. ***B.*** Needle position and direction for infiltration of the dorsal surface.

TECHNIQUES

FRACTURE REDUCTION

Closed reduction can be achieved with the use of the finger trap or straightforward axial traction. Place the anesthetized toe in the finger trap (**Figure 220-3**). Elevate and suspend the foot by the affected toe in the finger trap (**Figure 220-3**). Allow the weight of the leg to slowly distract the fracture site.

Reduce the fracture (**Figure 220-4**). Grasp the forefoot near the base of the affected toe or fractured phalanx with the nondominant index finger and thumb. Grasp the distal aspect of the fractured phalanx with the dominant index finger and thumb. Apply distally directed inline traction with the dominant hand and apply simultaneous countertraction with the nondominant hand to distract the fracture site. Remove the toe from the finger trap. Obtain a radiograph or fluoroscopic image to confirm the reduction. Buddy tape the toe to the adjacent toe as described below.

FIGURE 220-3. Use of the finger trap to aid fracture reduction.

The alternative is to reduce the fracture manually without the use of a finger trap (**Figure 220-4**). Grasp the forefoot near the base of the affected toe or fractured phalanx with the nondominant index finger and thumb. Grasp the distal aspect of the fractured phalanx with the dominant index finger and thumb. Apply distally directed inline traction with the dominant hand and apply simultaneous countertraction with the nondominant hand to distract the fracture site. Reduce the fracture. Obtain a radiograph or fluoroscopic image to confirm the reduction. Buddy tape the toe to the adjacent toe as described below.

BUDDY TAPING

Buddy tape the toe after the reduction (**Figure 220-5**). Place a piece of folded 2×2 gauze or a corn pad between the fractured toe and its neighboring toe (**Figure 220-5*A***). The gauze or corn pad will prevent maceration and pressure necrosis of the skin. Tape the toes together (**Figure 220-5*B***).

ASSESSMENT

Reassess the toe's perfusion by checking capillary refill time after any attempt at reduction. Obtain a postreduction radiograph or fluoroscopic image to verify proper alignment. Mild to moderate displacement in phalangeal fractures of toes 2 through 5 is quite acceptable if the toes are not rubbing together. Repeat the reduction process as necessary to reduce the fracture. Consult an Orthopedic Surgeon or Podiatrist if the fracture cannot be adequately reduced.

FIGURE 220-4. Manual fracture reduction.

FIGURE 220-5. Buddy taping the fractured toe. ***A.*** Place gauze or a corn pad between the fractured toe and an adjacent toe. ***B.*** Tape the toes together.

AFTERCARE

All toe fractures are persistently painful. They require analgesia and splinting for 2 to 3 weeks. Prescribing nonsteroidal anti-inflammatory drugs (NSAIDs) supplemented with narcotic analgesics has been the standard of care. Research may shift future management from the use of NSAIDs as these medications can delay fracture healing.[8] The patient can apply ice packs for 10 to 15 minutes every 3 hours to help decrease pain and swelling. Provide the patient with a hard-soled shoe, wooden-soled shoe, or Reese orthopedic shoe. It is felt that dorsiflexion of the forefoot in walking causes the most pain in toe fractures.[5] The use of these shoes, as opposed to the patient's own shoes, minimizes pain with walking.

An alternative to the orthopedic shoe is to place a metatarsal bar for patient comfort.[9] Use a commercially available unit or make a metatarsal bar by taping wooden tongue depressors together (**Figure 220-6**).[6] Obtain four tongue depressors and cut one in half (**Figure 220-6*A***). Place two tongue depressors side by side (**Figure 220-6*B***). Place the third tongue depressor to cover the seam of the adjacent tongue depressors (**Figure 220-6*B***). Tape the tongue depressors together to form longitudinal support (**Figure 220-6*C***). Apply the cut tongue depressor to form transverse support (**Figure 220-6*D***). Tape the transverse support to the longitudinal support (**Figure 220-6*E***). Apply the metatarsal bar to the sole of the patient's shoe (**Figure 220-7**). Additional support and pain control can be achieved by immobilizing the foot in a short leg walking cast with a toe plate or a short leg boot for 2 to 3 weeks.[4-7] This is true for great toe fractures or if the patient complains of persistent pain.[4-7]

Instruct the patient to continue weight bearing as tolerated and to elevate the foot above the level of their heart when not walking to minimize swelling. Teach the patient how to reapply the buddy taping splint as it must be changed every 2 to 3 days for up to 6 weeks. Inform the patient that swelling may last up to 3 months, especially with return to activity or wearing of tight-fitting shoes. All patients with great toe fractures should follow up with an Orthopedic Surgeon or Podiatrist within 2 to 7 days for reevaluation. Follow-up in several weeks for all fractures of toes 2 through 5 toe fractures has previously been the standard of care. Recent data showing very low complication rates for simple nondisplaced fractures demonstrate that these can be treated definitively from the Emergency Department. Other than great toe fractures, as previously discussed, the indications for Orthopedic Surgeon or Podiatrist follow-up include intraarticular fractures, phalangeal fractures with gross deformity upon presentation, and fractures that involve the growth plate.[5]

COMPLICATIONS

Long-term sequelae from toe fractures are rare. Persistent angulation at the fracture site with a malunion may result in a "sore area" on the plantar surface of the toe. Refer the patient to an Orthopedic Surgeon or Podiatrist if such areas remain symptomatic and functionally disabling. A simple surgical procedure may be indicated to correct the problem. Any fractures involving the joint space will result in some degree of arthritis. Warn the patient of this possible complication before they are discharged.

Few complications are associated with the management of toe fractures. Incomplete reduction can result in an angulated toe. Persistent angulation can result in the toe pushing against adjacent toes, skin irritation, and possible skin ulceration. Always place a pad between the toes before buddy taping to prevent irritation, pressure necrosis, ulceration, and maceration of the skin.

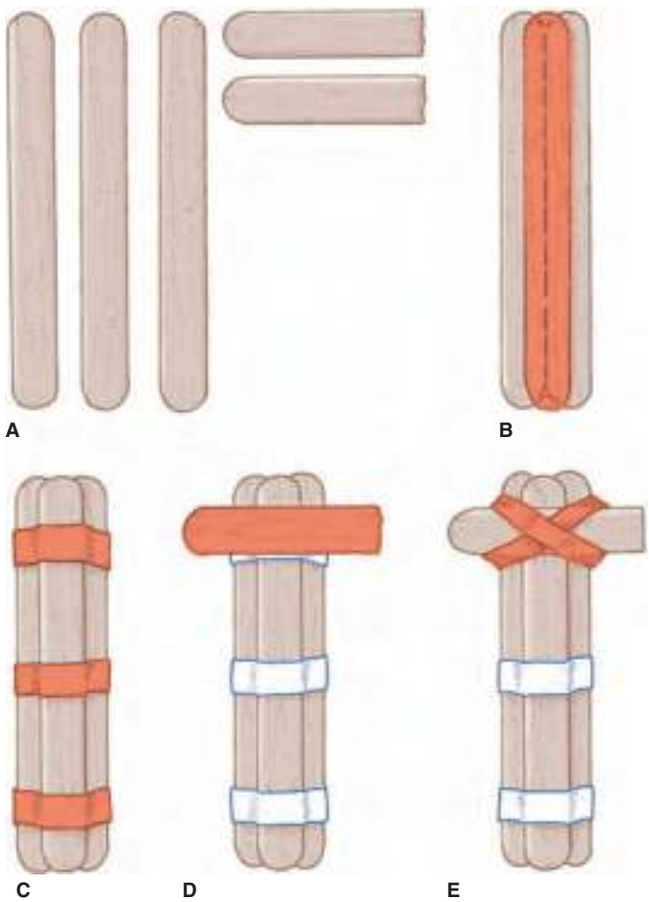
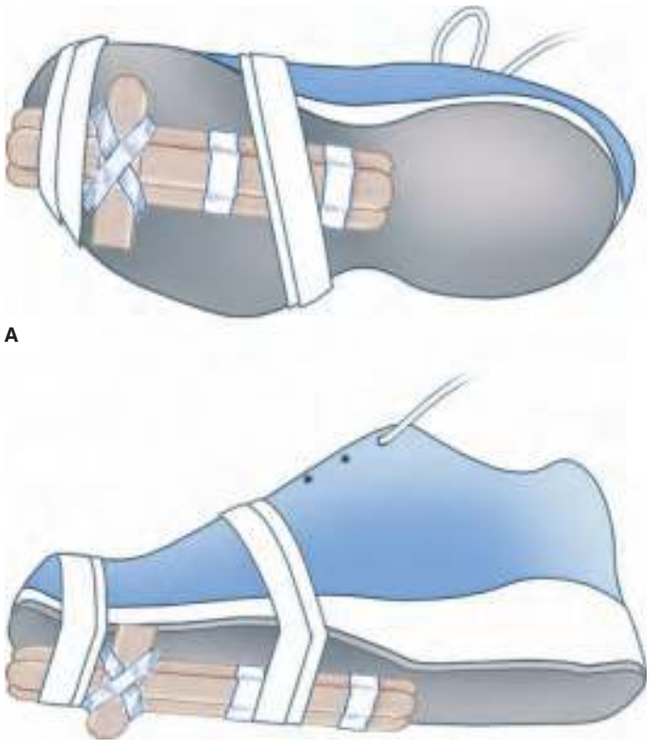


FIGURE 220-6. Fabricating a metatarsal bar from tongue depressors. ***A.*** Lay out four tongue depressors, one of which is cut in half. ***B.*** Place two tongue depressors side by side. Place the third tongue depressor to cover the seam between the first two. ***C.*** Tape the tongue depressors together to form the longitudinal support. ***D.*** Apply the cut tongue depressor to form the transverse support. ***E.*** Tape the transverse support to the longitudinal support.



A

B

FIGURE 220-7. Application of the metatarsal bar to the patient's shoe. ***A.*** Inferior view. ***B.*** Lateral view.

SUMMARY

Toe fractures are commonly seen in the Emergency Department. They may cause the patient significant pain and discomfort. Simple conservative management with the use of buddy taping and appropriate footwear helps the fracture heal in 3 to 6 weeks. Open or closed surgical reduction of phalangeal fractures may be required to achieve proper reduction. Intraarticular or severely displaced toe fractures, especially those of the great toe, should be referred to an Orthopedic Surgeon or Podiatrist. Most toe fractures can be satisfactorily and definitively managed by the Emergency Physician with minimal complications.[1]

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221 Plantar Wart Management

Kevin O'Rourke

INTRODUCTION

Plantar warts (i.e., verruca pedis) are caused by infection of the epidermal skin layer with the human papillomavirus (HPV). Plantar warts were discussed as far back as the ancient Greeks and Romans. They were subsequently identified as being caused by infectious agents in the late 1800s. HPV, a member of the Papovaviridae family, was identified in 1949 and is composed of double-stranded DNA. The peak incidence of plantar warts occurs in the teenage years.[1] They are estimated to occur in 10% of children and young adults, with a greater prevalence in females. HPV enters the host keratinocyte through an epidermal abrasion with the help of a transiently impaired immune system. It is found in the upper epidermis and results in squamous epithelial cell hyperplasia. Numerous types of HPV exist. Simple plantar warts are mainly due to HPV types 1, 2, 4, 27, or 57.

Two-thirds of untreated common warts in children regress spontaneously within 1 to 2 years.[1,2] Patients seek treatment despite these high remission rates in part because of their location. Large warts on weight-bearing areas can be painful and disabling. They can be transmitted to adjacent or distant body areas if left untreated. Patients often present with the plantar wart and request its removal. No single therapy has been proven effective at achieving complete remission and there are many different approaches to therapy. Warts typically continue to increase in size and distribution and may become more resistant to treatment over time.[3]

This chapter summarizes the pathogenesis of plantar warts and three techniques for their removal. The Dermatologist has other treatments they can apply to remove plantar warts.[4-6] This includes ablation, the injection of a variety of substances, laser removal, and needling.

ANATOMY AND PATHOPHYSIOLOGY

One must recall the layers of the skin to understand the pathogenesis of the HPV (**Figure 221-1**). The skin is composed of the epidermis, the dermis, and the subcutaneous tissue or hypodermis. The epidermis on the sole of the foot consists mainly of stratified squamous epithelium. The thickness of the epidermis ranges from 0.05 mm on the dorsal surface of the foot to 1.5 mm on the plantar surface.[7] HPV replication occurs in the most superficial epidermal layer. The dermis is composed of connective tissue and serves as a scaffold for the skin. The blood vessels, nerves, glands, and hair follicles are in the dermis.

HPV is a DNA virus. It gains access to host cells and uses the host's replicating cell proteins to manufacture viral proteins and DNA. This replication process induces epithelial cell hyperplasia and results in the mound of thickened skin observed in a plantar wart. The HPV remains in the epidermis but spreads laterally from the mound of verrucous tissue into what appears to be normal-looking epidermis. Intracellular HPV can remain in the dormant state as the host's cell-mediated immunity holds it in check. Untreated, solitary, and small to mid-sized warts have a remission rate of approximately 70% in 2 years, with only 20% of the initial lesions persisting in immunocompetent individuals.[1,2]

INDICATIONS

The task force of the American Academy of Dermatology's Committee on Guidelines of Care has evaluated the indications for treatment of any wart.[2] They include patient request for therapy, symptomatic warts (e.g., pain, bleeding, itching, or burning), lesions that are disabling or

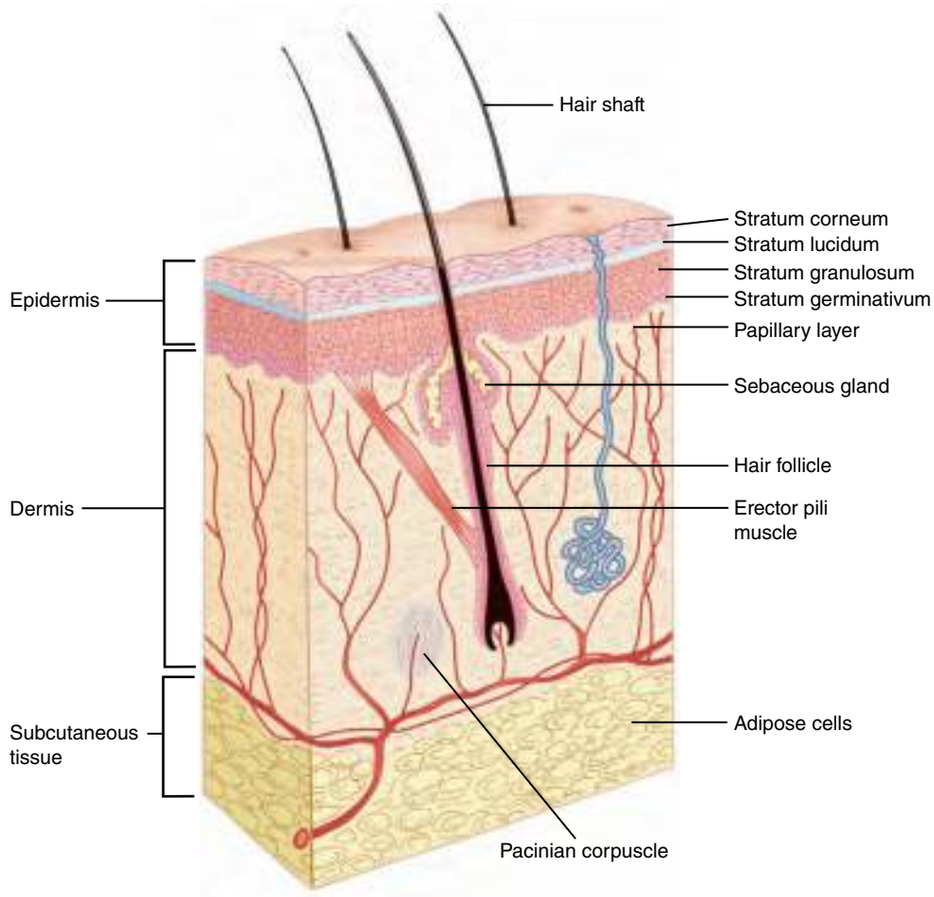

FIGURE 221-1. The anatomy of the skin.

disfiguring, many or large-sized lesions, to prevent spread to other body areas, and warts in patients who are immunocompromised.

CONTRAINDICATIONS

There are few contraindications to the removal of a plantar wart. **Any lesion whose diagnosis is uncertain requires a biopsy and/or an evaluation by a Dermatologist prior to any ablative removal.** An asymptomatic plantar wart should not be treated in the Emergency Department simply based on its presence. Relative contraindications for cryotherapy include patients who suffer from cold urticaria, cold intolerance, cryofibrinogenemia, cryoglobulinemia, diabetes, or peripheral vascular disease. Patients with diabetes or peripheral vascular disease have poor plantar blood circulation, may take longer to heal, and are at increased risk of developing a subsequent infection. Patients with hepatitis B, inflammatory bowel disease, or active collagen vascular disease may have an exaggerated freeze response and poor wound healing.[7] Patients taking corticosteroids or who are immunocompromised may have an exaggerated freeze response and poor wound healing.[7] Renal transplant patients have a higher incidence of warts after transplantation and their immunocompromised state should be kept in mind before doing the procedure.

EQUIPMENT

GENERAL SUPPLIES

- Povidone iodine or chlorhexidine solution
- 25 or 27 gauge needle
- 3 mL syringe
- Local anesthetic solution without epinephrine
- EMLA cream
- #15 surgical scalpel blade on a handle or straight razor blade
- Nonsterile gloves
- 4×4 gauze squares
- Pumice stone
- Petroleum-based lubricant (i.e., Vaseline)

CRYOTHERAPY

- Liquid nitrogen stored in a Dewer tank
- Handheld, self-contained spray devices (i.e., CRY-AC in 300 or 500 mL canisters)
- Cotton-tipped applicators
- Foam cup
- Otoscope earpiece

SALICYLIC ACID THERAPY

- Cotton-tipped applicators
- 15% to 40% salicylic acid ointment in collodion, with optional 20% lactic acid
- Clear nail polish or petroleum-based lubricant
- Occlusive dressing, Band-Aid or Tegaderm

DUCT TAPE THERAPY

- Duct tape
- Emery board

PATIENT PREPARATION

Explain the risks, benefits, and potential complications of the procedure to the patient and/or their representative. Obtain a signed informed consent for the procedure. Mention that the patient might experience a "burning" discomfort with the liquid nitrogen application and a possibility of throbbing pain for up to 24 hours. Consider administering acetaminophen or a nonsteroidal anti-inflammatory drug (NSAID) before or after the procedure.

The area of the wart must be clean, dry, and well exposed. Place the patient in a position of comfort. This may be prone, supine, or standing with the ipsilateral knee and leg resting on a chair. Clean the plantar surface of the foot of any dirt and debris. There is no need for sterile preparation or draping of the foot. Position an overhead light so that the beam is focused onto the wart. **Adequate lighting is essential.**

Anesthesia is rarely required for cryotherapy or the application of salicylate acid to plantar warts. Children, nervous adults, or persons with large warts may require anesthesia. One option is to apply EMLA cream to the wart followed by an occlusive dressing for 1 hour prior to the procedure.

Local injection of an anesthetic agent is an alternative. Clean the skin overlying and surrounding the wart site of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. Inject a small volume of 1 to 2 mL of a local anesthetic solution to create a wheal under the plantar wart. The editor believes this to be cruel and unusual punishment for the patient. Local infiltration is extremely painful as there are numerous sensory nerve fibers in the sole. The tough and tight skin of the plantar surface quickly comes under tension with the injection of the local anesthetic solution and results in undue discomfort.

Perform a posterior tibial nerve block (Chapter 156) if injection anesthesia is required. The posterior tibial nerve is superficial at the ankle, midway between the medial malleolus and the heel (**Figure 156-40**). It lies between the tendons of the flexor digitorum longus and flexor hallucis longus muscles. It travels with and slightly posterior to the posterior tibial artery. The posterior tibial nerve divides at the inferior border of the calcaneus to form the medial and lateral plantar nerves. The nerves provide motor innervation to the intrinsic foot muscles. The lateral plantar nerve provides sensory innervation to the lateral one-third of the sole and plantar surface of the lateral 1½ toes (**Figure 156-27**). The medial plantar nerve provides sensory innervation to the medial two-thirds of the sole and the plantar surface of the medial 3½ toes (**Figure 156-27**).

Place the patient supine with the ankle supported on a pillow or blanket and the leg externally rotated. Identify by palpation the medial malleolus and the posterior tibial artery by its pulsation. Place a skin wheal of local anesthetic solution at the level of the upper border of the medial malleolus and just posterior to the posterior tibial artery or medial to the Achilles tendon (**Figure 221-2**). Insert a 25 gauge needle through the skin wheal and perpendicular to the skin (**Figure 221-2**). Advance the needle to the tibia or until paresthesias are elicited. If paresthesias are elicited, withdraw the needle 2 mm and allow them to resolve. Inject 3 to 5 mL of local anesthetic solution. If paresthesias are not elicited, infiltrate 5 to 7 mL of local anesthetic solution starting against the posterior tibia as the needle is withdrawn.

FIGURE 221-2. The posterior tibial nerve block. Note the course of the posterior tibial artery (A), the course of the nerve (N), and the needle position.

TECHNIQUES

WART PREPARATION

The plantar wart often has a thicker epidermal layer than the adjacent normal epidermis. Trim the wart to allow easier and more efficient penetration of the liquid nitrogen or salicylic acid (**Figure 221-3**). Use a #15 blade or a straight razor blade to shave the wart's surface. Place a 4×4 gauze square under the patient's foot to catch the shavings. Approach the plantar wart at its base and at a 30° to 45° angle to the skin. Gently move the blade in a smooth sawing motion. Trim the wart to reduce it to a flat structure contiguous with the adjacent normal epidermis. It may take several passes of the blade to trim the wart. Soak the affected foot in lukewarm water for 5 to 10 minutes if the wart is very hard and difficult to trim. **Thoroughly dry the wart and the surrounding skin after the soak. The skin must be dry before the application of liquid nitrogen or salicylic acid.** Proceed with the cryotherapy or salicylic acid treatment as these agents can now easily penetrate through the trimmed plantar wart.

CRYOTHERAPY

Cryotherapy may affect wart clearance by simple necrotic destruction of HPV-infected keratinocytes or possibly by inducing local

FIGURE 221-3. Trimming the wart with a scalpel blade.

inflammation conducive to the development of an effective cell-mediated response. Cure rates for cryotherapy vary widely depending on the treatment regimen. The highest cure rates are achieved when treatment occurs at a frequency of every 2 to 3 weeks.[2] This technique is not suitable for children under 10 years of age.

Cryotherapy can be achieved using one of two methods. A self-contained liquid nitrogen spray can (e.g., CRY-AC) makes the job very easy (**Figure 221-4*A***). Use a spot-freeze technique. Hold the spray tip 1 to 2 cm from the skin surface and tangential to the wart (**Figure 221-4*B***). Gently pull the spray trigger to form a circular "ice field" overlying the wart (**Figure 221-4*C***). **Avoid spraying liquid nitrogen onto the normal epidermis.** A spot-freeze of approximately 30 seconds will form a circular ice field of roughly 2 to 3 mm in diameter (**Figure 221-4*D***). This corresponds to a depth of freeze of approximately 2 to 3 mm and epidermal temperatures of –5°C to –150°C.[8] Place an otoscope earpiece over the wart to zone in a more concentrated cryospray for thicker or irregularly shaped plantar warts. This also prevents a "blast" effect and freeze damage to the

A

B

C

D

FIGURE 221-4. Cryotherapy for plantar warts. ***A.*** A self-contained liquid nitrogen spray can. ***B.*** Positioning of the spray tip 1 to 2 cm from the skin surface. ***C.*** Spray liquid nitrogen to form a circular ice field over the wart. ***D.*** A 2 to 3 mm ice field after a 30 second spray.

surrounding normal tissue. The cure rates approach 97% depending upon the type and number of treatments used.[7]

The lack of liquid nitrogen spray should not impede the use of liquid nitrogen if it is available at your institution. Obtain 5 to 10 mL of liquid nitrogen from the stock tank and place it into a foam cup. Dip a cotton-tipped applicator into the liquid nitrogen. Firmly apply the liquid-nitrogen-soaked cotton-tipped applicator to the plantar wart. Hold the applicator to the wart until a narrow halo of white ice forms around the swab. Larger warts require multiple dips of the cotton-tipped applicator into liquid nitrogen followed by its application onto the wart. **Do not place the cotton-tipped applicator onto the same spot more than once.** Apply the cotton-tipped applicator to cover the entire wart. This technique of touching the plantar wart with a liquid nitrogen-dipped cotton applicator results in a tissue temperature of only –20°C. **It is not as effective as the liquid nitrogen spray at eliminating the plantar wart.**

SALICYLIC ACID THERAPY

Topical therapy with salicylic acid is a safe and effective method. No clear evidence exists to prove that other therapies have an advantage with higher cure rates or fewer adverse effects.[8] Another guideline lists salicylic acid as the first-line therapy for flat warts on the face, hand, and plantar warts.[9] Salicylic acid is cheaper, easily available, and can be used by the patient themselves. Salicylic acid ointment is available in a variety of concentrations ranging from 15% to 40%. Salicylic acid ointments can be made in any concentration. The ideal concentration is 40% with or without 20% lactic acid. The lactic acid serves to lower the pH of the ointment and increase the penetration of the salicylic acid into the plantar wart.[10] Topical salicylic acid ointment is recommended over the liquid as it is easy to store and more accurate to apply.

Place a rim of petroleum-based lubricant (e.g., Vaseline) or clear nail polish around the wart (**Figure 221-5**). This will protect the surrounding normal skin from the effects of the salicylic acid. Allow the nail polish to dry. Apply a coat of salicylic acid ointment up to 2 mm beyond the margins of the plantar wart with a cotton-tipped applicator (**Figure 221-6**). Completely cover the wart so that the underlying skin does not show through. Apply an occlusive dressing on top of the wart. A clear dressing (e.g., Tegaderm) is useful, thin, allows visualization of the area, and does not affect walking. Daily treatments have an 84% cure rate at 3 months.[11]

The Mediplast or Duofilm patch of 40% concentration is particularly useful in the management of plantar warts. The plaster is best applied to the wart and a few millimeters of surrounding skin, taped in place with duct or athletic tape, and kept dry for 48 to 72 hours. The patch is then removed and the wart pared down. This process can be repeated. **The patch must be taped securely in place because it destroys all skin it contacts and must be reapplied if it gets wet.**

FIGURE 221-5. Application of nail polish around the plantar wart. A clear nail polish was used to circumscribe the region of the plantar wart for this photograph.

FIGURE 221-6. The application of a salicylic acid ointment to cover the plantar wart.

Adequate paring of dead skin is an essential component of successful treatment. Paring is performed with a nail file or a pumice stone by the patient in between treatments and/or with a scalpel blade in the Emergency Department immediately before cryotherapy or application of topical agents.

DUCT TAPE THERAPY

Clean and dry the plantar surface of the foot. Apply duct tape to the surface of the foot overlying the wart. Leave the duct tape in place for 6 days before it is removed. Soak the wart in warm water. Instruct the patient to use an emery board to gently remove dead tissue. Allow the wart to remain exposed for about 12 hours. Repeat the duct tape application. Instruct the patient to do this every 6 days for up to 2 months. One study found an 85% cure rate in the duct tape arm versus 60% in the cryotherapy arm.[12]

AFTERCARE

A clear or hemorrhagic blister may form at the site of cryotherapy. The blister is sterile. Leave it intact if it does not interfere with function. It may be punctured with a sterile needle, covered with topical antibiotic ointment, and bandaged if the patient finds the blister a nuisance. Instruct the patient to not pick at the blister. Arrange follow-up in 2 to 3 weeks and after healing has occurred by secondary intention.

Salicylic acid therapy does not produce any pain. The patient's complaints of pain indicate excessive irritation or infection that requires prompt assessment. Teach the patient or their caretaker the application procedure. The application procedure must be performed daily until the wart is gone and for at least a week after that. Be aware that over-the-counter salicylic acid preparations are 15% to 17% in concentration and prescription strength preparations are 30% to 70% in concentration. The most commonly used concentrations for the removal of plantar warts are 40% to 50%.[8,13]

General instructions include advising the patient to return to the Emergency Department if any signs of an infection develop or there is persistent pain. Mild pain can be managed with acetaminophen

or NSAIDs. Educate the patient on the prevention of future warts and the possibility of recurrence.

COMPLICATIONS

One must understand the normal process of healing after cryotherapy before the complications can be understood.[14] The wound is initially raw and erythematous. It may develop a clear or hemorrhagic bulla. Edema and exudate are expected in the first 24 hours. The use of nonadherent gauze or a bandage is often helpful. Cryotherapy pain lasts no more than 24 hours after the procedure.

The main complications of cryotherapy are pain, delayed bleeding, and infection.[14] Hypertrophic scar formation is rare. Discuss this prior to the procedure if the patient is prone to developing hypertrophic scars. The healing process results in mild skin contractures and depigmentation.[7] This is less of an issue with the plantar warts compared to lesions on more visible body parts.

Both techniques have the potential to damage normal skin.[14] Do not allow the liquid nitrogen or salicylic acid to extend more than 1 to 2 mm onto the adjacent tissue. Consider using an otoscope cone to prevent blast damage from liquid nitrogen spray. The use of a protective barrier with petrolatum-based lubricant or nail polish will prevent salicylic acid runoff.

There are no known complications with duct tape therapy. This is a cost-effective therapy for patients who are unable to afford a physician. Many people have a roll of duct tape in a drawer at home.

SUMMARY

Plantar warts are extremely common. They can be readily managed in the Emergency Department. Treatment consists of a one-time liquid nitrogen application, daily salicylic acid ointment applications, or weekly duct tape therapy. The key to success is patient education. Patients need to know the entire aftercare plan due to the possible complications and recurrence.

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222 Neuroma Management

Justin Bosley and Eric R. Snoey

INTRODUCTION

Morton's neuroma, also referred to as an interdigital neuroma, is one of the most common painful disorders of the forefoot. It was first described in 1835 by Civinni.[1,2] It is named after Thomas Morton who presented a case series of patients afflicted with this disorder in 1876.[1,2] Patients with an established Morton's neuroma are usually cared for by a Podiatrist or an Orthopedist. They may present to the Emergency Department with a previously undiagnosed neuroma or with a painful exacerbation of a previously diagnosed neuroma.

The term neuroma is a misnomer. Histologic investigation does not reveal the typical proliferation of axons found in true neuromas. Instead, there is a fibrosis and thickening of the perineural tissue with corresponding degeneration of the underlying nerve.[3] This condition most commonly affects the third plantar common digital nerve located in the third interspace between the third and fourth metatarsal heads. The second interspace may be affected less commonly. It is rare for neuromas to involve the first or fourth interspaces.

Morton's neuroma disproportionally affects women between their fourth and sixth decades.[4] It is especially common in those who wear high-heeled shoes, poorly fitting shoes, shoes with poor or no padding, or shoes that are narrow at the forefoot. Persons with pronated or pes cavus feet are similarly at risk.[5] Neuromas do not usually become symptomatic until their transverse diameter reaches more than 5 mm.[6]

ANATOMY AND PATHOPHYSIOLOGY

Neuromas form just proximal to the bifurcation of the plantar common digital nerves (**Figure 222-1**) and below the deep transverse intermetatarsal ligament (**Figure 222-2**).[7] The deep transverse intermetatarsal ligament connects the metatarsal heads on the plantar aspect of the foot (**Figure 222-2**).[7] The neuroma is made up of branches from both the medial and lateral plantar nerves (**Figure 222-1**). Most commonly affected is the third interdigital nerve. It is the largest of the interdigital nerves which may explain the increased frequency of neuroma formation in this location.

FIGURE 222-1. Morton's neuroma most commonly occurs in the third intermetatarsal space beneath the transverse metacarpal ligament.

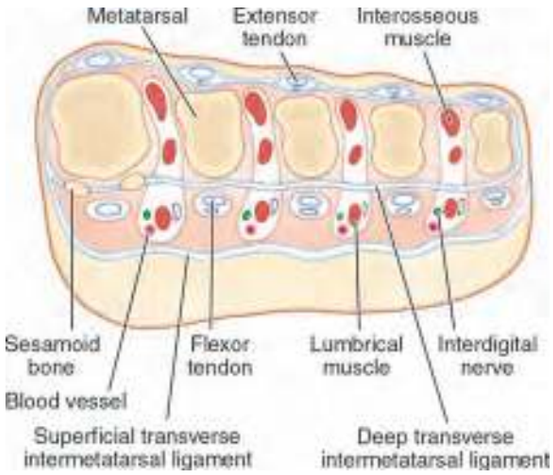


FIGURE 222-2. Anatomy of the forefoot. Cross-section through the distal metatarsals.

FIGURE 222-3. Examination for Mulder's click. The thumb and forefinger of the examiner's dominant hand are used to compress the interdigital space. The nondominant hand then performs medial and lateral compression of the metatarsal heads (*arrows*). A palpable click is diagnostic while pain alone is suggestive of a neuroma.

Morton and others postulated that the increased mobility of the fourth and fifth metatarsal heads relative to the more fixed medial portion of the foot results in disproportionate trauma to the third interdigital nerve. Mechanical factors combined with the impingement and stretching from a tight transverse intermetatarsal ligament results in repetitive microtrauma. Histologic evaluation reveals perineural fibroma formation consistent with compression-induced trauma.[4] Injury begins with edema of the endoneurium, followed by fibrosis beneath the perineurium, axonal degeneration, and finally neuronal necrosis.

Some authors believe that a more significant contributor to neuroma formation is enlargement of the interphalangeal component of the intermetatarsophalangeal bursa. This leads to microvascular trauma.[8] Movements of the bursa result in minor compressive effects on the adjacent digital arteries leading to ischemia of local neural tissue.

The diagnosis of a Morton's neuroma is usually made clinically based upon classic historical features and physical examination findings.[3,9,10] The pain of a neuroma is initially intermittent, increases in frequency over time, and eventually becomes constant. The patient may complain of pain, burning, electric shocks, or tingling over the involved intermetatarsal space and occasionally the adjacent toes. Attacks typically occur suddenly after a period of walking, running, or standing. Some patients may complain of pain disturbing their sleep.[8] The involved toes and web space commonly have hyperesthesia or hypoesthesia. Patients often complain of the unilateral feeling of "walking on a lump." Symptoms are relieved by rest and shoe removal.

Physical examination localizes the pain to the involved interspace with minimal involvement of the adjacent metatarsal heads.[11] Moderate pressure applied proximally in the affected web space reproduces the pain. A small mass, representing the neuroma, can be palpated in approximately one-third of the cases. **A positive Mulder's sign is diagnostic.**[10,12] This is a click palpated in the interspace during medial and lateral compression of the metatarsal heads (**Figure 222-3**).

Radiographic imaging may help to confirm the diagnosis of a Morton's neuroma and exclude alternative etiologies.[9] Plain radiographs will not demonstrate a neuroma but may reveal splaying of the involved toes when the neuroma is especially large or show alternative diagnoses (e.g., stress fractures or arthritis). Ultrasound (US) represents the most efficient and effective imaging modality.[4,13-15] The US was not shown to be an improvement over the clinical examination.[10] An experienced Emergency Physician may identify hypoechoic neuromas as small as 2.9 mm.[16] Magnetic resonance imaging (MRI) is quite effective at identifying the presence of a neuroma. The cost and access issues make it a less practical choice in the Emergency Department.[3] MRI was shown to be equal to US in identifying the neuroma.[17] Computed tomography (CT) may be useful if an MRI is contraindicated or not available. The sensitivity of CT scanning is less than that of MRI or US.[4]

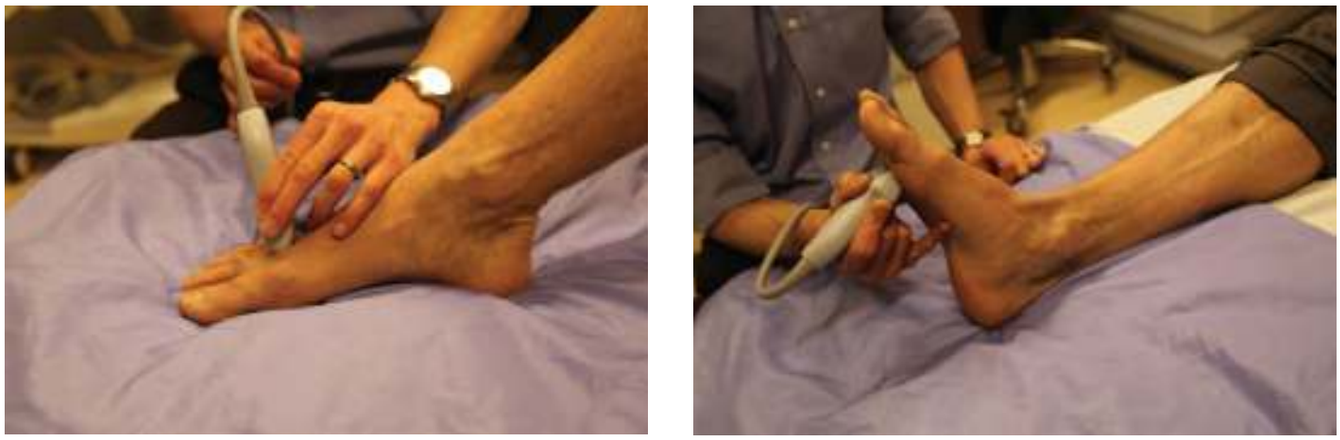
US evaluation of the foot is best accomplished with a high-frequency linear transducer scanning the region in which the patient identifies as the foci of pain.[13] The plantar and dorsal placement of the US transducer can be used to evaluate the area for a Morton's neuroma (**Figures 222-4 and 222-5*A***). The classical finding involves identifying the fibrillar echogenic nerve coursing into a heterogeneous hypoechoic mass (**Figures 222-6 and 222-7**).[18] **Careful attention should be paid when scanning to minimize artifacts that can falsely suggest abnormal findings. The US appearance of nerves and their surrounding tissues can vary depending on the angle of scanning.** Scanning perpendicular to the target structure can help minimize this issue. Scanning the contralateral foot provides a helpful means of identifying abnormal pathology on the affected side.

INDICATIONS

Injection of the neuroma can provide significant pain relief to the patient and confirm a Morton's neuroma as the etiology of the patient's complaints. Injection is indicated after less invasive methods have failed.[19-23] These include foot elevation and rest, nonsteroidal anti-inflammatory drugs, foot manipulation, arch supports, orthoses, and changing footwear to prevent forefoot compression (i.e., low heels and wide toe boxes). Corticosteroid injections can provide partial or complete relief in up to 80% of patients.[19,24]

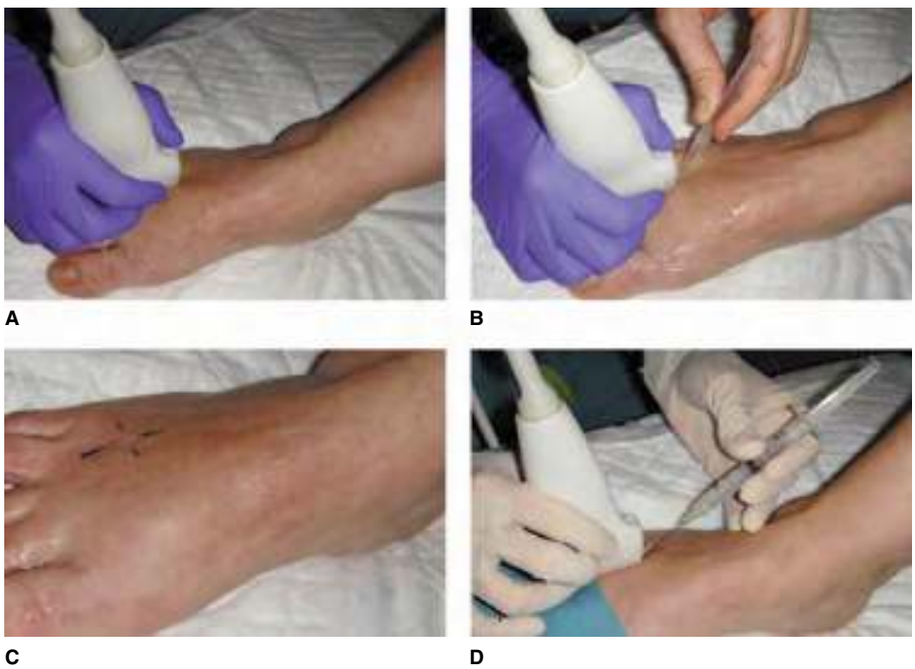
CONTRAINDICATIONS

The primary contraindication to injection of a Morton's neuroma is failure to make a correct initial diagnosis. The differential diagnosis for forefoot pain includes a wide variety of pathologies that may closely mimic the presentation of a neuroma. Alternative diagnoses include tarsal tunnel syndrome, peripheral neuropathy, capsulitis, bursitis, rheumatoid arthritis, foreign bodies, avascular necrosis,



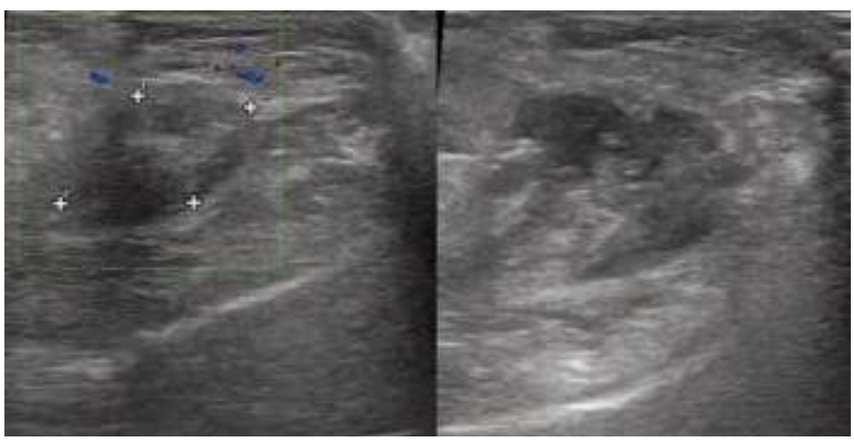
A B

FIGURE 222-4. Demonstration of US transducer positions for the evaluation of a possible neuroma. ***A.*** Dorsal. ***B.*** Plantar.



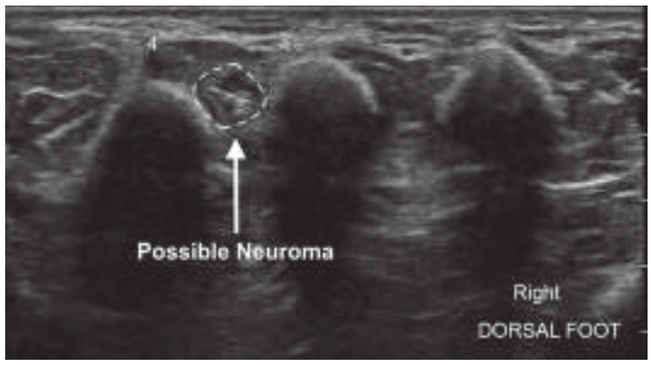
A B C D

FIGURE 222-5. The neuroma injection. ***A.*** Positioning of the transducer to identify the neuroma. ***B.*** Identifying the neuroma. ***C.*** The neuroma is marked on the skin. ***D.*** Needle insertion under US guidance. (Photo used with permission from reference 14.)

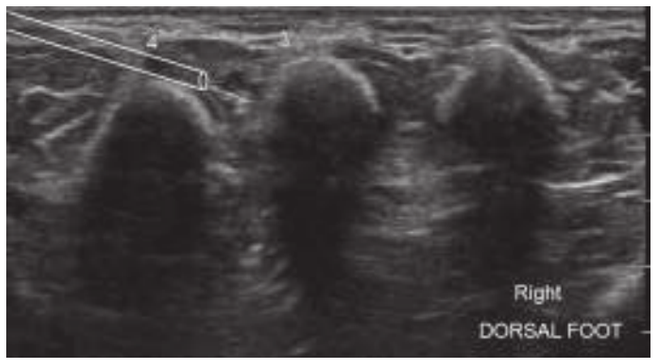


A B

FIGURE 222-6. The neuroma. ***A.*** Baseline identification. ***B.*** After injection. (Photo used with permission from reference 14.)



A



B

FIGURE 222-7. The transverse plane US appearance of a neuroma. ***A.*** A patient with a suggestive clinical history. ***B.**** US visualization of the needle approaching the neuroma during the injection. The numbers note the metatarsal heads.

stress fractures, and peripheral vascular insufficiency. A careful history and physical examination with discretionary use of imaging modalities will lead to a correct diagnosis.

Injection therapy may be contraindicated in serious athletes.[5] The steroid injection may result in fat pad atrophy, degeneration of the volar plate, and degeneration of the collateral ligaments. A discussion of alternative therapies to injection therapy is provided in the "Aftercare" section of this chapter.

EQUIPMENT

- Sterile gloves
- Sterile drapes
- Povidone iodine or chlorhexidine solution
- 3 mL syringe
- 22 or 25 gauge needle
- 0.5% bupivacaine without epinephrine
- Injectable methylprednisolone or triamcinolone
- Sterile bandage
- US machine, optional
- US high-frequency transducer, 5 to 12 MHz
- Sterile US transducer cover
- Sterile US gel

PATIENT PREPARATION

Explain the risks, benefits, complications, and aftercare of the procedure to the patient and/or their representative. Place the patient supine on a gurney with their hip and knee flexed so that the sole of the affected foot is flat on the gurney. Identify the neuroma by palpation and/or US (**Figure 222-5*A***). Mark the location of the neuroma on the patient's skin (**Figures 222-5*B* and 222-5*C***). Clean the skin overlying the neuroma of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. Apply sterile drapes to isolate a sterile field.

Prepare for the procedure. Set up a sterile field on a bedside table. Use aseptic technique to open the needle and 3 mL syringe onto the sterile field. Don sterile gloves. Have an assistant hold the medication bottles upside down while sterilely drawing out the medications. Draw 1 mL of 0.5% bupivacaine without epinephrine and 10 mg of methylprednisolone or triamcinolone into the syringe. Another local anesthetic agent without epinephrine may be used instead of bupivacaine. Long-acting local anesthetic agents are preferred as they provide the patient with longer pain relief after the injection.

Prepare the US transducer if using US with the procedure. Have an assistant apply US gel over the footprint of the transducer. Instruct the assistant to place the gel-coated US transducer in the sterile cover while you hold it. Have the assistant place sterile US gel over the dorsum of the patient's foot. Reidentify the neuroma (**Figures 222-5*A*, 222-5*B*, and 222-5*C***).

TECHNIQUE

TRADITIONAL TECHNIQUE FOR A PALPABLE NEUROMA

Instruct the patient to moderately dorsiflex their toes to separate the metatarsal heads. **Use a dorsal approach to the neuroma injection as this is less painful than penetrating the sole with a**

FIGURE 222-8. Traditional technique for injection of the neuroma.

FIGURE 222-9. Placement of a metatarsal pad.

needle.[25] Insert the needle perpendicular into the skin overlying the neuroma.

Advance the needle perpendicular to the skin and into the neuroma or adjacent into its fascial plane (**Figure 222-8**). Inject the affected interspace with 1 to 2 mL of the combination long-acting local anesthetic agent and steroid. Remove the needle. Apply pressure to the skin puncture site followed by a bandage.

TRADITIONAL TECHNIQUE FOR A NONPALPABLE NEUROMA

Sometimes the neuroma is not palpable but the physical examination is positive for a neuroma. Insert the needle approximately 1 to 2 cm proximal to the web space. Direct the needle into the facial space between the deep and superficial transverse metatarsal ligaments. Note that this space is deep to the metatarsal heads on the plantar aspect of the foot (**Figure 222-2**). Inject the affected interspace with 1 to 2 mL of the combination long-acting local anesthetic agent and steroid. Remove the needle. Apply pressure to the skin puncture site followed by a bandage.

ULTRASOUND TECHNIQUE

The use of bedside US by the Emergency Physician has made the diagnosis and procedures more accurate. US guidance may improve the efficacy of steroid injection.[26-28] Use the US transducer with the sterile cover to identify the neuroma (**Figures 222-5*A*, 222-5*B*, and 222-5*C***). Hold the US transducer in place to visualize the neuroma on the US machine monitor. Insert the needle (**Figure 222-5*D***). Advance the tip of the needle under direct visualization until it is abutting the target structure (**Figure 222-7**). Inject the affected interspace with 1 to 2 mL of the combination long-acting local anesthetic agent and steroid. The injected medication can be seen adjacent to the target structure when performed correctly (**Figures 222-6 and 222-7**).[14] Remove the needle. Apply pressure to the skin puncture site followed by a bandage.

ALTERNATIVE TECHNIQUES

Various types of conservative treatment can be initiated once a Morton's neuroma is diagnosed. Success rates have been reported from 20% to 80% with a combination of injections with other conservative therapy including massage, physical therapy, orthoses, metatarsal pads, shoes with wider toe boxes, and physical therapy.[21-23,29,30] Orthoses are used to help control abnormal pronation but may be ineffective in treating a neuroma.[30] Metatarsal pads serve to spread the metatarsal heads at the involved interspace and decrease compressive trauma (**Figure 222-9**). The types of physical therapy employed include massage, US, electrical stimulation, and whirlpool immersion. A trial of acetaminophen or nonsteroidal anti-inflammatory drugs may be helpful in the short term.

Studies have described the injection of dilute alcohol into the site of the neuroma using US guidance.[31-35] The effect is to sclerose and harden the perineural tissues. The injection is typical repeated every 1 to 2 weeks for four to seven times with treated patients experiencing a significant reduction in symptoms and neuroma size. Sclerosing agents (e.g., ethanol) injected under US have demonstrated more consistent results when compared to blind landmark-based approaches.[31-33] The long-term results of alcohol injection does not appear to be effective.[36] The neuroma regrows and causes pain.[36]

Podiatrists and Orthopedic Surgeons may offer more aggressive and definitive therapy when conservative measures fail. Other techniques available include the injection of capsaicin, cryoablation, and radiofrequency ablation.[37-40] Surgical excision using a dorsal approach has success rates of 84% to 93%.[41-43] The literature offers increasing support for endoscopic decompression of the deep transverse intermetatarsal ligament and leaving the nerve intact.[4] The advantage is a shorter recovery time which may make this especially desirable for athletes. No long-term results have been reported but short-term results appear quite promising.[4] Excision using a carbon dioxide laser may allow for an even shorter recovery.[8]

ASSESSMENT

Allow 10 to 20 minutes for the local anesthetic agent to take full effect. The patient will experience a rapid resolution of symptoms if the injection was successful. The relief of pain confirms that the corticosteroid has been injected into the correct location.

AFTERCARE

Pain may be controlled with the use of acetaminophen or nonsteroidal anti-inflammatory drugs. Instruct the patient to wear flat shoes, shoes with flat and wide toe boxes, and to avoid shoes that elevate the heel more than 1 to 2 cm above the metatarsal heads. The patient should avoid activities that place repeated pressure on the forefoot (e.g., bicycling, jogging, and impact sports). Apply a metatarsal pad to decrease compressive forces on the neuroma. Place the dome of

the pad between the third and fourth metatarsals just posterior to the metatarsal heads (**Figure 222-9**). Cold packs can be applied for 10 to 15 minutes every 3 hours to decrease inflammation and pain. This is helpful after activities that aggravate the patient's symptoms. Refer the patient to a Podiatrist or Orthopedic Surgeon for follow-up, additional injection therapy, orthoses, physical therapy, and possible surgical management.

COMPLICATIONS

Complications associated with neuroma injection are exceedingly rare. They include the introduction of infection, damage to neurovascular structures, local fat pad and skin atrophy from the steroid, skin depigmentation or hyperpigmentation, telangiectasias, and thinning of the skin.[32,33] Fat pad atrophy on the dorsal foot is primarily a cosmetic issue. Fat pad atrophy on the plantar aspect of the foot can result in painful ambulation and gait disturbances.[44] Skin changes and fat pad atrophy can be prevented by ensuring the injection is deep and properly placed so that the corticosteroids do not leak into the subcutaneous tissues.[45] It is common for the pain to recur in days to weeks, even when the injection is successful. Single injections may not totally relieve the patient's symptoms. The patient often requires a series of injections every 1 to 2 weeks to be successful.

SUMMARY

Morton's neuroma is one of the most common causes of forefoot pain. Injection therapy is useful in alleviating the symptoms of a neuroma and in confirming the diagnosis, particularly when aided by US guidance. Arrange Podiatric or Orthopedic follow-up for all patients given this diagnosis. More invasive procedures may be needed for the long-term resolution of symptoms.

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223 Management of Select Podiatric Conditions

JoAnna Leuck, Jacob Hurst, and Anant Patel

INTRODUCTION

Socrates once said "To him whose feet hurt, everything hurts." Patients with foot pain and deformities are commonly seen in the Emergency Department complaining of compromised mobility. The decreased mobility causes a decreased quality of life. The frequency of these disorders increases with age. This chapter addresses common presentations and procedures for the management of the painful foot. Numerous other podiatric procedures (e.g., local anesthesia, ingrown toenails, plantar warts, puncture wounds, toe fractures, ganglionic cysts, neuromas) are discussed in other chapters of this book.

The foot is divided into the forefoot, midfoot, and hindfoot (**Figure 223-1**).[1-3] The forefoot is composed of the five metatarsals and the phalanges. The great toe has two phalanges and each of the remaining toes has three phalanges. The forefoot is important in normal gait. The gait consists of a heel strike, forefoot contact, heel lift, peak forefoot loading with metatarsophalangeal joint extension, and toe lift. The first metatarsal bears a large force during walking and running. The first metatarsal supports the longitudinal arch of the foot. The great toe provides stability during forefoot loading. The great toe is involved in the biomechanics of walking. The bones of the midfoot and hindfoot are referred collectively as the tarsus. The midfoot comprises the cuboid, cuneiform (i.e., medial, intermediate, and lateral), and navicular bones. The hindfoot is composed of the talus and calcaneus. These bones are stressed during walking and running.

Foot injuries are very common. The chief complaint of foot pain often is followed by the clinical examination and radiologic imaging. A detailed knowledge of imaging anatomy is essential for an accurate diagnosis of an injury and to avoid missing an injury. The radiographic superimposition of the midfoot and hindfoot can make the identification of injuries difficult. These injuries are commonly missed during the clinical examination and subsequent imaging.

MANAGEMENT OF PLANTAR LESIONS

INTRODUCTION

The skin on the sole (i.e., plantar surface) of the foot is the thickest skin on the entire body. It is adapted to protect the internal structures from environmental demands. A 150-lb person has dissipated 60 tons of force with each foot after walking a mile.[4] Hyperkeratosis (i.e., callosity) occurs when the process of keratinization becomes overactive due to shearing forces and pressure points over bony prominences (**Figure 223-2**). This is a normal protective response as the body attempts to protect the irritated skin. It may be seen on the hands of a laborer or on the plantar surfaces of feet in those who

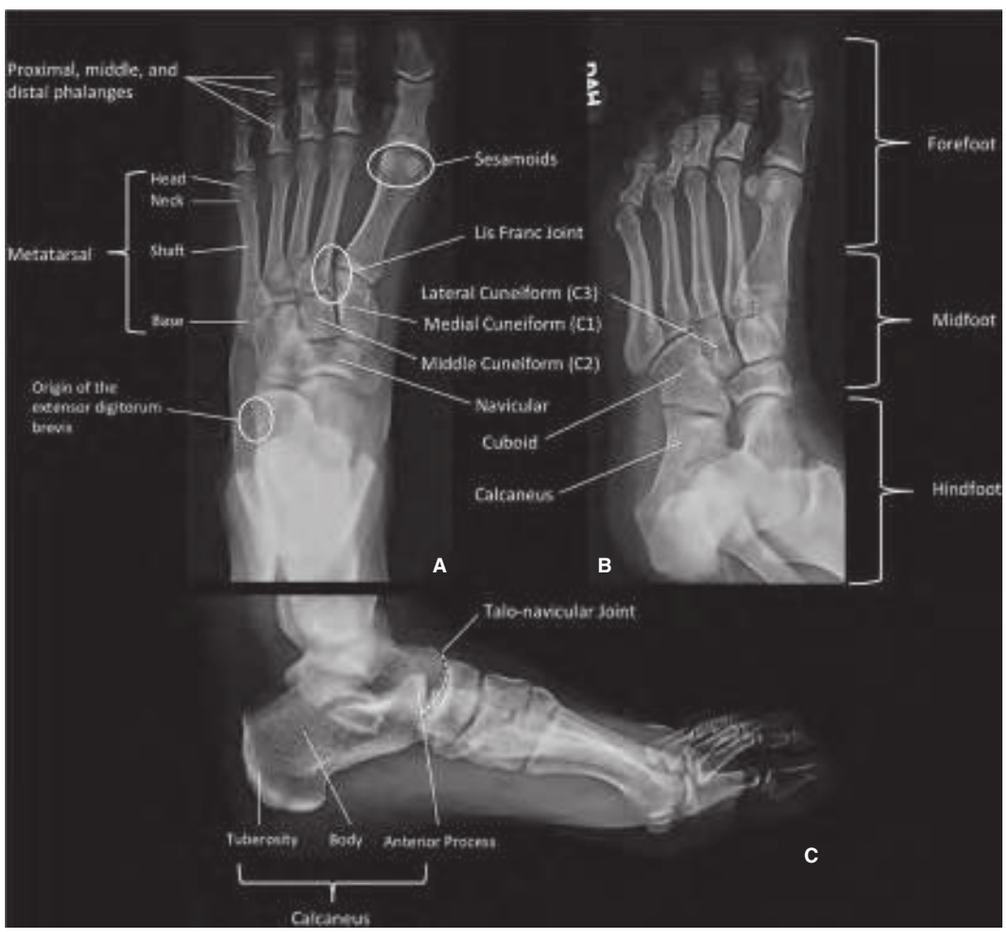

FIGURE 223-1. Foot radiographic anatomy. ***A.*** Anteroposterior view. ***B.*** Oblique view. ***C.*** Lateral view. (Used with permission from reference 1.[1])

FIGURE 223-2. Calluses of the heel.

walk barefoot. A vicious cycle begins over time. The hyperkeratotic area becomes prominent, increases the pressure in a tight shoe, produces further discomfort, and results in further keratinization.

The ubiquitous verruca virus can invade the plantar skin of the foot and produce a wart or a hyperkeratotic response. It is sometimes difficult to differentiate this condition from other hyperkeratoses (e.g., calluses and corns). Warts may occur at any site on the plantar skin, unlike a pressure- or friction-induced callus or corn. Warts are less likely to occur on the digits and seldom occur on the dorsal skin. **The surface appearance of these conditions may be identical yet their treatment strategies are radically different.** The wart is treated with epidermal eradication (Chapter 221). Other hyperkeratoses are treated with simple paring and rebalancing of the weight-bearing surfaces.

A corn is known as a helomata or a clavus. It represents a well-circumscribed traumatic hyperkeratosis caused by friction or pressure on the skin (**Figure 223-3**). It has a visibly translucent core that presses deeply into the dermis. Corns may be so painful as to be disabling.[5] A hard corn is known as a heloma durum. It forms primarily on the exposed surfaces of the toes from extrinsic pressure of the footwear.[6-9] It is commonly found on the dorsolateral aspect of the fifth toe or the dorsum of the interphalangeal joints of toes 2 to 5.

A soft corn is known as a heloma molle. It is extremely painful and often misdiagnosed as warts or fungal infections. They are macerated and whitish-appearing hard lesions resulting from the corn's absorption of large amounts of sweat. They occur interdigitally, usually in the fourth web space, from wearing tight shoes that press the condyle of a metatarsal against its neighbor. A soft corn will reveal its central "core" if pared.

Another skin lesion that mimics a wart is porokeratosis plantaris discreta. This small cyst-like lesion is a sweat duct filled with a keratin plug. It is located on the plantar surface of the foot. This lesion often goes by the misnomer "seed wart." Their treatment includes curettage or keratolytic agents similar to wart treatment.

Calluses are known as tylomata. They are broad-based, poorly circumscribed, diffuse areas of hyperkeratosis that develop at pressure sites (**Figure 223-2**).[5] Calluses are usually larger than corns, do not have a central core, and may or may not be painful. A large area of hyperkeratosis that occurs in combination with a centrally located plug of keratin is called an intractable plantar keratosis (IPK). These lesions usually require podiatric evaluation for debridement, protective padding, and rebalancing of the weight-bearing surfaces with orthotics.

Hammertoes are a contracture of the toes from of a muscle imbalance between the tendons on the top and the bottom of the toe (**Figure 223-4**). Hammertoes can be flexible or rigid. Hammertoes occur when the intrinsic muscles of the foot lose their stabilizing effect on the interphalangeal joints allowing the extrinsic muscles of the leg to overpower and flex the interphalangeal joints. This deformity may be associated with the formation of painful hyperkeratotic lesions. The patients develop corns on the top of the toe from rubbing and a callous on the ball of the foot from the toe pressing downward (**Figure 223-5**). The callous becomes prominent from the pressure of the bone. These lesions may be overlying or adjacent to any or all the interphalangeal joints or on the distal tip of the digit.[10]

The most common cause of hammertoes are tight shoes. The shoe forces a toe to stay bent, the muscles tighten, and the tendons contract. Other causes of hammertoes are congenital abnormalities, contractures (e.g., bedridden or cerebral palsy), joint diseases (e.g., rheumatoid arthritis), nerve injury, peripheral neuropathy, peripheral vascular disease, spinal cord injury, and stroke. Treatment of a

FIGURE 223-3. Numerous painful corns on the toes of a 51 year old female. (Used from Marionette www.commons.wikimedia.org.)

FIGURE 223-4. Hammertoes. (Photo courtesy of RoseMary Mabo, DPM.)

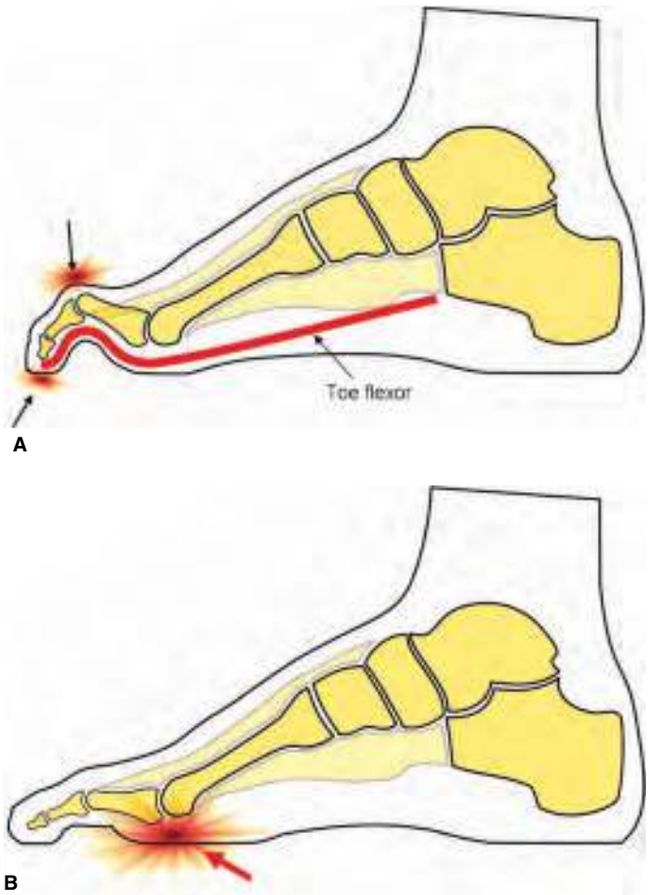

FIGURE 223-5. Hammertoes can cause calluses and corns. The arrows represent some of the pressure points. (Used with permission from www.thion-medical.com.)

hammertoe is multifactorial. Instruct the patient to wear shoes with a roomy toe box to allow the toes to function without excessive pressure. Treat all corns. Use padding to relieve pressure from the toe. Surgery is an option if conservative treatment is unsuccessful.

The first step in treating a patient with a plantar skin lesion is to differentiate a wart (i.e., verruca) from a plantar hyperkeratosis. Warts and plantar corns are best distinguished by paring the outer layer of epidermis with a sharp scalpel blade. Warts may or may not be localized under a bony prominence (**Figure 223-6**). Apply alcohol to the plantar lesion to enhance the skin lines. **The skin lines of a hyperkeratosis pass through the lesion whereas those from a wart pass around the lesion. End arteries are visible when a wart is pared.** These appear as multiple black dots that hurt and bleed if cut (**Figure 223-6**). Paring the hyperkeratotic lesion reveals only yellowish, translucent, firm keratin, even after many shaves. **A firm keratin core is a corn and not a wart.** The patient's pain response to pressure on the lesion may help in differentiating a wart from a corn. Pain is elicited with direct pressure on the hyperkeratotic lesion. Squeezing the wart side to side with a similar force elicits maximal pain.

INDICATIONS

Perform paring to provide temporary relief of pain associated with corns and calluses. **Make the patient aware that paring is not a permanent treatment. The first step in treatment is to suggest a better-fitting shoe.** Identify the lesion type by shaving the outer layers of keratinized epidermis. Examine the shaved surface looking

FIGURE 223-6. Plantar wart on the underside of the great toe. (Used from Dewdude www.commons.wikimedia.org.)

for the identifying characteristics of a wart versus a corn or callus. Trimming the callused hyperkeratotic surface will provide temporary relief from the discomfort of a corn, a callus, and even a wart. Definitive treatment of a wart or porokeratosis requires curettage, acid, freezing, or referral to a Podiatrist. Perform a more definitive shaving if a corn or callus is identified.

CONTRAINDICATIONS

Severe peripheral vascular disease, impaired wound healing, and overlying cellulitis are contraindications to shaving a plantar lesion. A variety of neoplasms may appear as a callused plantar lesion. If there is any suspicion that the plantar lesion is not a simple callus or corn, do not shave the lesions and refer to a foot specialist. **Biopsy is mandatory if there is any question about the nature of the lesion.**[5]

EQUIPMENT

- #10 or #15 surgical scalpel blade on a handle
- Isopropyl alcohol swabs
- 18 gauge needle
- 25 or 27 gauge needle
- Local anesthetic solution without epinephrine
- Cotton ball or 2×2 gauze square
- Tape, 1 inch wide
- Lamb's wool
- Aperture pads

Podiatrists and Orthopedic Surgeons have specialized equipment to debride warts and hyperkeratotic lesions. This includes curettes, mini-blades, chisel blades, and tissue nippers. This equipment is not commonly available in Emergency Departments, clinics, or offices. The techniques described below use commonly available equipment to manage foot lesions.

PATIENT PREPARATION

Explain the procedure, its risks, and benefits to the patient and/or their representative. Obtain an informed consent to perform the procedure. Soak the foot in warm water for 10 to 15 minutes. Place the patient supine or prone on a gurney. Heel lesions are often best

FIGURE 223-7. Technique for using a scalpel blade in the paring of a hyperkeratotic lesion (*curved arrow*).

addressed with the patient in the prone position. Sit on a stool so that the lesion is at eye level. Visualize the hyperkeratotic lesion under a bright light. Anesthesia is usually not required. Consider the use of a posterior tibial nerve block (Chapter 156) if the patient is significantly sensitive.

TECHNIQUES

Support the foot to be debrided with the nondominant hand. Wipe the hyperkeratotic area with an alcohol swab. Grasp the skin surrounding the lesion. Place the index finger of the nondominant hand above the hyperkeratotic area and the thumb below it (**Figure 223-7**). Grasp a #10 or #15 scalpel blade on a handle with the dominant hand. Rest the base of the dominant hand against the patient's foot. This allows a measure of safety for both the Emergency Physician and the patient. All materials move in mass with the patient if they move. Place the scalpel blade almost parallel to the lesion (**Figure 223-7**). Remove superficial amounts of the hyperkeratotic lesion using a semicircular slicing motion.[11] Continue this process of debriding repeatedly until healthy pink tissue is noted.[11] **A central core, if uncovered during the debridement, must be removed to relieve the patient's symptoms.** Insert the tip of the scalpel blade into the core. Carefully and slowly twist the scalpel blade in a circular motion to remove the core. **Avoid injury to the underlying and adjacent healthy tissue.** Treat the common corn with periodic debridement.

Adjunctive therapy includes felt corn pads with a central aperture or a horseshoe to relieve pain and disperse pressure at bony prominences to the surrounding skin (**Figure 223-8**). Place pads under the metatarsals for protection and pressure dispersal from any or all the metatarsal heads. Tape the pads to secure them to the skin.

The macerated soft corn is frequently misdiagnosed on visual inspection as an interdigital fungal infection or wart. Superficial debridement will aid in the diagnosis. Perform a digital or metatarsal block using local anesthetic solution without epinephrine.[12] The digital arteries are end arteries and local anesthetics containing epinephrine can theoretically induce digital ischemia. Pare the hyperkeratotic lesion with a #10 scalpel blade to provide pain relief and a diagnosis.

The toes must be separated while the lesion is healing. Place a cotton ball or a small gauze square between the toes, either distal or proximal to the lesion. Tape the dressing in place. **Never circumscribe the digit with tape due to the risk of circulatory impairment from constriction.** Apply lamb's wool around the affected digit. The advantage of lamb's wool is that tape is avoided and the patient can change the dressing daily. A foam interdigital cushion is a good choice for pressure dispersion and can be applied later.

FIGURE 223-8. The use of self-adherent aperture pads on the dorsal surface to relieve pressure from a pared hyperkeratotic lesion overlying a bony prominence. Pads are further secured to the skin with tape.

AFTERCARE

The patient can expect 6 to 8 weeks of pain relief after a corn or callus is pared. The procedure may be repeated after this period. Patients must realize that hyperkeratotic lesions are a result of lifestyle choices (e.g., wearing constricting, improperly fitted, and/or high-heeled shoes).[13] Refer the patient to a Podiatrist if conservative therapy fails.

Provide all patients with verbal and written instructions regarding the signs of infection and lifestyle modification. This includes immediate return to the Emergency Department for increasing pain, progressive swelling, redness, tenderness, and warmth. Follow-up treatment consists of frequent trimming of the keratotic lesion. Consider using shock-absorbing inserts. Metatarsal pads are an excellent choice for submetatarsal pressure dispersion and the protection of pared calluses (**Figure 223-9**). Recommend lifestyle modification

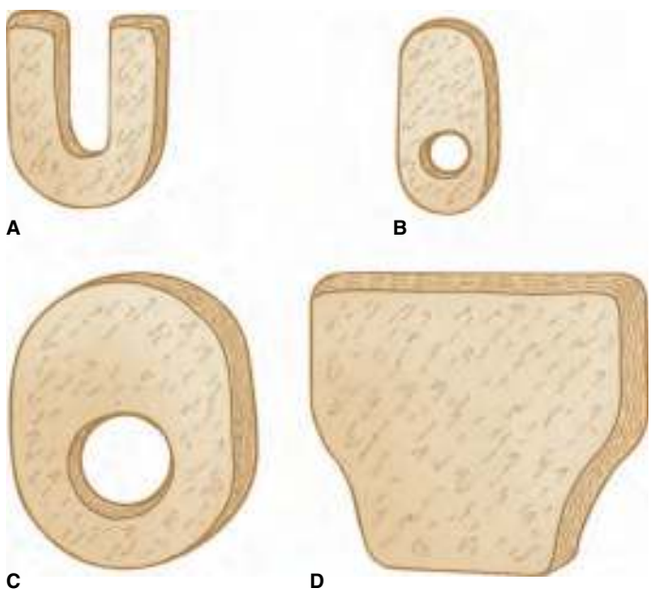

FIGURE 223-9. Examples of self-adherent commercially available padding. ***A.*** The ⅛ inch horseshoe digital corn pad. ***B.*** The ¼ inch interdigital soft corn pad. ***C.*** The ¼ inch bunion shield. ***D.*** The ¼ inch submetatarsal pad.

and the use of appropriate footwear. Home care of the macerated corn includes daily astringent soaks with boric acid solution and a topical antibiotic applied with dressing changes.

PADDING

Padding offers temporary and immediate relief for many patients with foot pain associated with bursitis, fasciitis, and tendonitis. Padding relieves pressure around bony prominences by dispersing it to the surrounding skin. The type of pad and its placement depend on the underlying problem (**Figure 223-9**). Many commercial pads are available in felt, gels, and silicone. They are useful for the management of hyperkeratosis secondary to hammer toes, corns, and calluses. The following section discusses the use of padding for common forefoot and hindfoot disorders.

BUNIONS AND BUNIONETTE DEFORMITIES

The bunion affects the first metatarsal head and is known as hallux abducto valgus.[14] The bunionette affects the fifth metatarsal head and is known as the tailor's bunion, as tailors often sit in a cross-leg position with pressure on the lateral foot leading to this lesion. Deformities are identified as painful prominent metatarsal heads and surrounding soft tissues (**Figures 223-10 and 223-11**). Common causes of the bunion and bunionette deformities are complex and multifactorial. These include anatomic and physiologic abnormalities, hereditary conditions, and extrinsic footwear elements.[14,15] Biomechanical abnormalities lead to dysfunction of the metatarsophalangeal (MTP) joint.

The bunion deformity consists of lateral deviation of the great toe and medial deviation of the first metatarsal (**Figures 223-10 and 223-11**). The bunionette deformity is a bunion on the lateral aspect of the foot overlying the fifth metatarsal head. The fifth digit is medially deviated with lateral deviation of the fifth metatarsal (**Figure 223-11**). Pain results from shoes causing pressure against the bony deformity and the surrounding soft tissues. This can result in subsequent pressure hyperkeratosis. Clinical examination reveals pain upon palpation along the border of the metatarsal head.

The differential diagnosis of a bunion includes bursitis of the first MTP joint, which commonly occurs over the medial bunion bump.[16] Consider other conditions (e.g., infectious and inflammatory diseases). Associated foot complaints may include lesser metatarsalgia, intermetatarsal neuroma, hammertoes, corns, and calluses.

FIGURE 223-11. The bunion and bunionette deformity. The dotted lines represent the normal metatarsal positions.

TREATMENT

The initial treatment consists of removing the offending footwear. Shoes with a spacious toe box minimize pressure against inflamed soft tissues and bony prominences. Carefully pare any hyperkeratotic lesions while avoiding injury to surrounding healthy tissue. Padding and accommodative shields help to disperse pressure and may be beneficial. Precut bunion shields are available (e.g., felt, gels, plastic, or silicone) or may be fashioned from 1 cm thick felt (**Figure 223-12**). Prescribe nonsteroidal anti-inflammatory drugs (NSAIDs) for analgesia. Refer the patient to a Podiatrist or

FIGURE 223-10. A bunion. (Used from Lamiot, www.commons.wikimedia.org.)

FIGURE 223-12. The application of a bunion shield.

Orthopedic Surgeon when conservative measures fail.[14] Surgical techniques can be used to correct the underlying bony deformity.

A painful bursa overlying the medial aspect of the first MTP joint is common. Local aspiration of bursal fluid followed by the injection of a local anesthetic agent and a short-acting steroid may relieve pain and decrease the size of the bursa. Clean the skin overlying the bursa of any dirt and debris. Apply povidone iodine or chlorhexidine solution to the skin and allow it to dry. Aspirate the bursa fluid with a 22 gauge needle on a 3 to 5 mL syringe. Inject the bursa with a mixture containing 0.5 mL of local anesthetic solution and 4 mg of dexamethasone.

METATARSALGIA

Metatarsalgia is a general term used to describe a diffusely painful area directly beneath the metatarsal heads (**Figure 223-13**).[17] The patient usually complains of forefoot pain that is insidious in onset with walking, worsens throughout the day, and is relieved with rest. The pathophysiology of metatarsalgia is complex. A common denominator is weight transfer from the first ray during ambulation to the lesser metatarsals. This may promote a bursitis, tendonitis, tenosynovitis, and capsulitis of the lesser MTP joints. The patient can usually point to the painful spot. The second metatarsal head is most commonly involved followed by the third and fourth metatarsal heads.

The clinical diagnosis can be made with direct palpation of each individual metatarsal head. Repetitive force induces a capsulitis or metatarsalgia beneath the metatarsal head that may progress to a stress fracture of the metatarsal. Radiographs may be negative initially with stress fractures. Repeat the radiographs in 2 to 4 weeks. A hyperkeratotic lesion may be associated with this syndrome. It can be located either directly beneath a metatarsal head or diffusely under the metatarsal heads. Paring of the lesion is important for the diagnosis and treatment. Sufficient debridement may provide immediate pain relief in the Emergency Department.

TREATMENT

All hyperkeratotic lesions require debridement for the diagnosis and treatment. Pad the lesion to disperse pressure to the surrounding tissues. Place a 1 cm thick pad beneath an inner sole under the first metatarsal for discrete pain just beneath the second metatarsal head

FIGURE 223-13. Metatarsalgia. (Used with permission from reference 17.)

FIGURE 223-14. A ¼ inch-thick felt pad placed beneath the first metatarsal to redistribute weight and to protect the second metatarsal.

or second proximal phalangeal shaft (**Figure 223-14**). This helps to redistribute the weight-bearing surface and protect the second metatarsal. NSAIDs may provide analgesia. Instruct the patient to wear a well-cushioned good-quality athletic or walking shoe. Arrange follow-up with a Podiatrist or Orthopedic Surgeon to address the underlying reason for the hyperkeratotic reaction.

SESAMOIDITIS

Two sesamoid bones lie beneath the first metatarsal. Their structure and function are similar to those of the patella. The clinical examination reveals pain with palpation beneath the sesamoid bones. This may develop into the more painful conditions of chondromalacia, osteoarthritis, and stress fractures. Radiography can be important in the initial evaluation of the pain in the sesamoid area.[18] The radiographs may be normal, may demonstrate sclerosis, or may demonstrate fragmentation of the involved sesamoid. Radiographic evaluation includes the anteroposterior and lateral radiographs. A sesamoid view can be ordered as an oblique coronal radiograph and allows direct imaging of the joint without osseous overlap.[18,19] Initial radiographs may be negative.[18] Serial radiographs taken 2 to 4 weeks later may reveal a fracture.

TREATMENT

Nonsurgical treatment that aims at reducing the load under the sesamoids is preferred for sesamoid problems. This includes restriction of activities, modification of footwear, and a moulded orthosis with metatarsal pads.[20] Pare any hyperkeratotic lesions. Instruct the patient to wear a well-cushioned good-quality athletic or walking shoe. Apply padding with a central aperture cut out beneath the sesamoid bones to help disperse pressure to the surrounding skin and away from the sesamoid bones. NSAIDs will provide analgesia. Arrange follow-up with a Podiatrist or Orthopedic Surgeon if the patient experiences persistent symptoms.

HEEL PAIN SYNDROMES

The painful heel is a common complaint of the middle-aged and elderly.[21] An in-depth discussion on the myriad of etiologies for heel pain is beyond the scope of this chapter. Patients most often have no associated disease other than obesity. Consider screening young

men for ankylosing spondylitis or reactive arthritis. Most cases respond to modification of physical activities, the use of NSAIDs, injection of local anesthetic agents and steroids, and the use of specially designed insoles.[21] The use of heel padding as adjunctive therapy is addressed later.

HEEL SPUR SYNDROME

The process of spur formation is termed heel spur syndrome.[22,23] The etiology is presumed to be chronic traction on the plantar fascia and intrinsic muscles. This results in repeated microtrauma along the medial calcaneal tuberosity, periostitis, and calcification. The patient complains of heel pain progressing over months. Palpation reproduces the pain along the medial border of the calcaneus. Radiographs reveal the spur emanating from the calcaneal tuberosity (**Figures 223-15 and 223-16**). **Absence of a spur suggests plantar fasciitis as the etiology of the pain.**

EQUIPMENT

- Povidone iodine solution
- 3 mL syringe
- 25 gauge needle, 1¼ inch long
- Ethyl chloride spray
- Local anesthetic solution without epinephrine, lidocaine or bupivacaine
- Dexamethasone
- Felt pad, 1 cm thick
- Tape, 2 inches wide

TREATMENT

Treatment is aimed at conservative measures. NSAIDs will provide analgesia. Padding can temporarily relieve heel pain by reducing central pressure on the more painful part of the heel. Apply a 1 cm thick felt pad or high-grade foam rubber pad cut in the form of a horseshoe directly to the affected heel (**Figure 223-17**). Secure the pad in place with two or three strips of 2 inch wide tape. Instruct the patient to wear the pad for 1 to 3 days. Remove the pad if it becomes wet to avoid skin maceration.

FIGURE 223-15. Lateral hindfoot radiograph demonstrating a heel spur. (Used from Lucien Monfils, www.commons.wikimedia.org.)

FIGURE 223-16. Sites of potential heel pain.

The patient's shoes may be modified to help provide pain relief. Place a self-adherent sponge pad into the heel of the shoe to decrease pressure on the calcaneus. Have a cobbler place a cut-out into the heel of the patient's shoe if the patient still experiences pain (**Figure 223-18**). Place a self-adherent sponge pad over the cut-out before the patient wears the shoe.

Injection of local anesthetic solution and corticosteroids is controversial as a treatment in the Emergency Department for heel spur syndrome. **This is due to the risks and complications (i.e., infection, increased pain, and fat pad atrophy).** Treatment with injection of any type is often reserved for a Podiatrist if conservative treatment fails.[24]

Injection of local anesthetic solution and corticosteroids is useful in reducing acute inflammation. Mix 1 mL of 1% lidocaine hydrochloride, 1 mL of 0.5% bupivacaine hydrochloride, and 0.8 to 1.0 mg of dexamethasone in a 3 mL syringe. Thoroughly mix the contents of the syringe together. Clean the skin overlying the medial border of the calcaneus of any dirt and debris. Apply povidone iodine or chlorhexidine solution and allow it to dry. Spray ethyl chloride to anesthetize the skin. Insert a 25 gauge perpendicular to the skin and at the level of the heel spur. Advance the needle in a dart like motion. Infiltrate the local anesthetic-steroid mixture along the medial calcaneal tubercle and the central plantar calcaneus beneath the spur

FIGURE 223-17. Relief of heel pain from a heel spur. A ¼ inch thick pad is cut in the form of a horseshoe and applied directly to the affected heel.

FIGURE 223-18. Shoe modification to relieve heel spur pain.

fascia junction. **Avoid infiltration into the plantar fat pad as this area is extremely sensitive and the steroid can result in fat pad atrophy.** The injection procedure can be repeated in 2 to 4 weeks. Refer the patient to a Podiatrist or Orthopedic Surgeon for persistent symptoms despite steroid injection or for an anatomically supinated or pronated foot.

PLANTAR FASCIITIS

Plantar fasciitis is one of the most common causes of heel pain.[22,25,26] It is an overuse syndrome like the heel spur syndrome. Excessive traction on the plantar fascia results in localized inflammation and acute pain at its origin on the calcaneus (**Figure 223-19**).[25] The inflammation may eventually result in the formation of a heel spur. The pain can progress distally along the fascial course in the chronic form.

The patient generally describes an insidious onset of unilateral heel pain that is characteristically worst first thing in the morning and eases after the first few steps. Plantar fasciitis is common in obese patients and can be caused by the chronic wearing of high-heeled shoes. Women in the 40 to 60 year age group are afflicted more than men. **Clinical examination reveals pain upon palpation along the plantar fascia in the midfoot but not over the area of a heel spur if present.**

Radiographs are generally not helpful at the initial presentation but rather 4 to 6 weeks after the onset if the symptoms persist. Radiographs will often confirm the presence of a heel spur that may or may not be involved in plantar fasciitis. One study demonstrated that 46% of patients with plantar fasciitis had no spur and 50% of patients who had bilateral spurs had pain in only one heel.[27]

TREATMENT

Treatment is aimed at conservative measures.[26,28] NSAIDs will provide analgesia. Instruct the patient to obtain well-fitted and cushioned footgear that supports the medial longitudinal arch, supports the plantar fascial band, and has good heel shock-absorbing qualities. Demonstrate a regimen of active stretching of the medial fascial band (**Figure 223-20*A***). It is important to perform this daily. Stand facing a wall and approximately 2 to 3 ft from the wall. Lean forward and place both palms on the wall. Keep both heels firmly pressed on the ground. The stretch should be felt in the Achilles tendon and longitudinal arch. Dynamic stretches such as rolling the foot arch over a 15-oz size can or a tennis ball are also useful.[28,29] Instruct the patient to stretch three times a day for 3 to 5 minutes.

The use of padding can provide relief for some patients. Place medial longitudinal pads to unload the anteromedial aspect of the heel and support the medial longitudinal arch (**Figure 223-20*B***). Make the pad from 0.2 inch thick felt and fashion it to fit the patient's longitudinal arch. Secure the pad in place with two or three strips of 2 inch wide tape. Instruct the patient to wear the pad for 1 to 3 days. Remove the pad if it becomes wet to avoid skin maceration. Numerous commercial pads are available over-the-counter and through a Podiatrist.

Night splints can be helpful in the treatment of plantar fasciitis. They are designed to keep the patient's ankle in a neutral position. A night dorsiflexion splint allows passive stretching of the calf and the plantar fascia. It potentially allows any healing to occur while the plantar fascia is in an elongated position and creates less tension with the first step in the morning. A night splint can be made from plaster, fiberglass, casting material, or a prefabricated commercially produced plastic brace.[28,29]

Injection of corticosteroids and local anesthetic will relieve any pain refractory to conservative treatment. Injections are relatively invasive and carry a risk of complications (e.g., fat pad atrophy, osteomyelitis of the calcaneus, and iatrogenic rupture of the plantar fascia).[30] Limited evidence supports the use of corticosteroid injections to manage plantar fasciitis. Results of a Cochrane review showed that corticosteroid injections improved plantar fasciitis symptoms at 1 month but not at 6 months when compared with control groups.[31]

Prepare the skin and the injection solution as described with the heel spur syndrome. Inject the solution deep into the plantar fascia (**Figures 223-20*C* and 223-21**). Use of a careful injection technique, use of an aseptic technique, avoidance of impact activities for

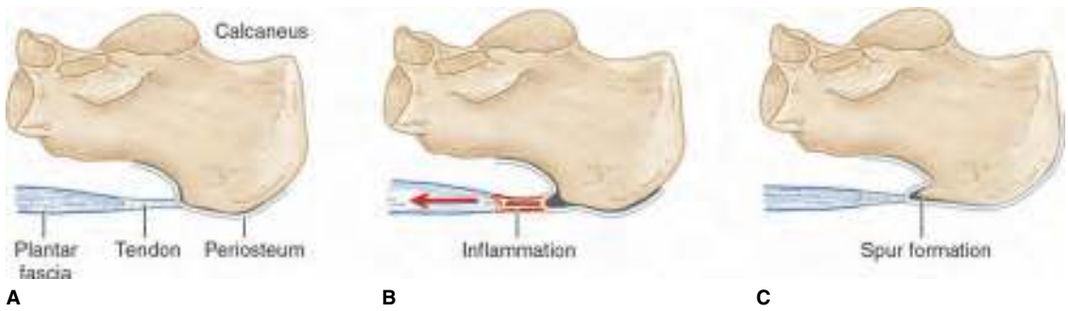

FIGURE 223-19. The mechanism of plantar fasciitis. ***A.*** Normal anatomic relationships. ***B.*** Traction on the tendon elevates the periosteum from the calcaneus and promotes the invasion of inflammatory tissue. ***C.*** The inflammatory tissue and periosteal elevation can result in the formation of a heel spur.

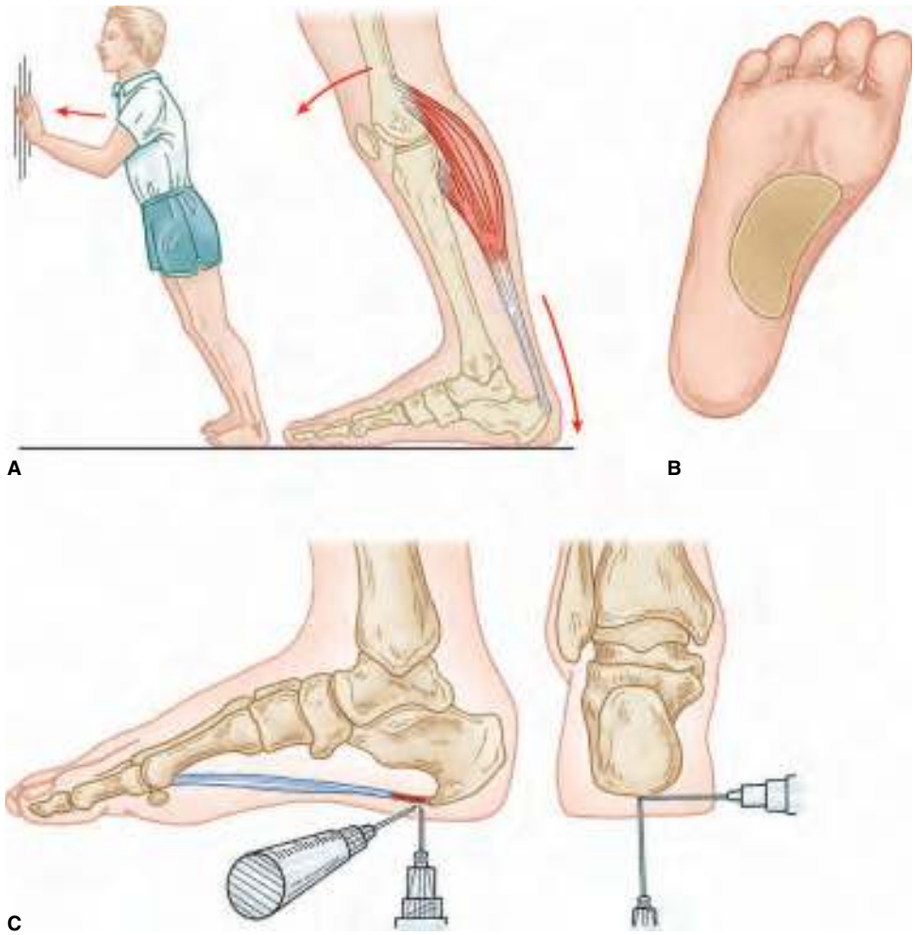

FIGURE 223-20. The treatment of plantar fasciitis. ***A.*** Active stretching of the medial fascial band. ***B.*** Placement of a medial longitudinal arch pad. ***C.*** Injection therapy.

10 days after injection, and ultrasound-guided injection may minimize these risks.[32,33]

The injection procedure can be repeated in 2 to 4 weeks if required. **Do not inject more than two to three times into the plantar fascia. Multiple injections can lead to rupture of the plantar fascia. Avoid injection into the plantar fat pad.** This area is extremely sensitive. Steroids injected into the fat pad can result in atrophy and loss of the intrinsic shock-absorbing qualities. Apply a medial longitudinal pad as described above. Relief from a combination treatment may last 6 to 8 weeks. Refer the patient to a Podiatrist or Orthopedic Surgeon for persistent symptoms greater than several months and if conservative therapy fails. No evidence strongly supports the effectiveness of any treatment for plantar fasciitis and most patients improve without specific therapy or by using conservative measures.[31]

FIGURE 223-21. Inferior view of injection therapy for plantar fasciitis. (Used with permission from www.gulfshoreapothecary.com.)

RETROCALCANEAL BURSITIS

Retrocalcaneal bursitis is a painful condition. It results from inflammation of one or both bursae at the Achilles tendon insertion on the calcaneus (**Figure 223-16**).[22] The pain in the posterior heel is insidious in onset. It is aggravated by increased activity and shoes with tight heel counterpoints. Palpate the bursae. Place the patient's foot in plantarflexion to relax the Achilles tendon. Squeeze the anterior part of the tendon just proximal to its bony insertion site in the depression above the calcaneus. Tenderness elicited with a squeezing force applied anterior to the Achilles tendon suggests inflammation of the anterior, retrocalcaneal, or subtendinous bursae. Always exclude the possibility of a systemic inflammatory disease when bilateral. There is an association of retrocalcaneal bursitis with rheumatoid arthritis and is seen in up to 10% of patients.[34] Tenderness elicited with pressure on the posterior surface of the Achilles tendon insertion site suggests inflammation of the posterior or subcutaneous calcaneal bursa. Erythema and swelling may be evident at the lateral borders of the Achilles tendon.

TREATMENT

Treatment is conservative and mirrors the treatment for heel spurs. NSAIDs are recommended. Steroid injections are controversial. Many studies have found that the use of injectable steroids is contraindicated as it may lead to tendon rupture. A one-time steroid injection directly into the retrocalcaneal bursa is sometimes helpful in refractory cases of retrocalcaneal bursitis.[34,35] **Extreme care must be taken to not inject any steroid solution into the tendon. Never perform more than one or two injections. This reduces the risk of tendon atrophy or rupture.** Occasionally bursal fluid is aspirated before the injection of the corticosteroid.[34] Apply a horseshoe-shaped heel pad made from 0.25 inch thick felt or foam rubber to the affected heel.

CALCANEAL APOPHYSITIS (SEVER'S DISEASE)

This condition is a common cause of heel pain in skeletally immature patients, particularly in 8 to 14 year old active males.[22,36-39] It results from excessive traction and microtrauma on the calcaneal apophysis by the Achilles tendon. This typically occurs before or during a growth spurt and shortly after the beginning of a new season or new sport, especially during running and jumping. Clinical examination reveals tenderness at the Achilles tendon insertion site on the posterior calcaneus. Radiographs may reveal an elevated apophysis with scleritis and fragmentation (**Figure 223-22**).[40]

A

B

FIGURE 223-22. Lateral radiograph of the foot with Sever's disease. ***A.*** Saw-tooth appearance of the calcaneus before the appearance of the apophysis. ***B.*** Irregular apophysis with sclerosis before fusion with the calcaneus. (Used with permission from reference 38.)

TREATMENT

Treatment is aimed at conservative measures.[41,42] Rest, NSAIDs, and heel lifts fashioned like the pads for heel spurs are recommended. Instruct the patient to perform Achilles tendon stretching exercises three times a day for 3 to 5 minutes (**Figure 223-20*A***). **Steroid injections are not recommended.** Preventative and proactive measures include education of coaches, education of parents, and well-fitted supportive cushioned athletic footwear worn by an active adolescent during activities.[43]

ACHILLES TENDINITIS

Repeated trauma to the Achilles tendon from faulty footwear, faulty landing techniques, hard landings, or the use of fluoroquinolone antibiotics can lead to inflammation of the tendon and its sheath due to prolonged friction.[22] Patients complain of pain with prolonged standing and with climbing stairs that is relieved with rest. Clinical examination reveals erythema and localized pain upon palpation just proximal to the Achilles tendon insertion site on the calcaneus. (**Figure 223-23**).

TREATMENT

There is no consensus on the best form of treatment for Achilles tendinitis.[44-46] An array of nonsurgical interventions (e.g., NSAIDs,

FIGURE 223-23. Erythema and thickening of the left Achilles tendon demonstrating tendonitis. (Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ (eds): *The Atlas of Emergency Medicine*, 4th ed. McGraw-Hill; 2016. Photo contributor: Kevin J. Knoop, MD, MS.)

rest, stretching and strengthening exercises, heel lifts, and other orthotic devices) are commonly applied. Surgery is often considered for refractory cases.[44] **Steroid injections of the Achilles tendon are absolutely contraindicated.**[47] Inform the patient regarding shoe modifications (e.g., cutting out the heel counter point [**Figure 223-18**] or recommending an open-backed shoe). Apply a 0.25 inch thick felt pad fashioned to fit the heel or a commercial cushioned heel lift as appropriate adjunctive therapy. Refer the patient to a Podiatrist or Orthopedic Surgeon for recalcitrant or severe cases.

ACHILLES TENDON RUPTURE

Achilles tendon rupture is one of the most common tendon failures affecting the lower extremity. It occurs primarily in middle-aged adults who participate in occasional athletic activities. Most of Achilles tendon ruptures occur in the serious athlete during competition or in the relatively sedentary former athlete during demonstrations of former skills on the basketball or volleyball court. Rupture of the Achilles tendon involves a significant force (e.g., push-off), a forceful drive, a direct blow, or landing in sudden dorsiflexion with the foot plantarflexed and extended.[22] Rupture of the Achilles tendon can occur at its attachment to the calcaneus, along the tendon, or more commonly at the musculotendinous junction.

Patients typically report a history of sudden pain associated with an audible snap and difficulty tiptoeing or climbing stairs. Clinical findings include a positive Thompson squeeze test (**Figure 223-24**), a positive heel resistance test (i.e., easy dorsiflexion of the heel and foot against plantarflexion), and a positive gap sign (i.e., a palpable, tender gap in the tendon).[48] The Thompson squeeze test is performed with the patient in the prone position and the examiner squeezing the calf (**Figure 223-24**). It normally produces plantarflexion. The Thompson test produces no motion or markedly decreased motion if the Achilles tendon is ruptured (**Figure 223-25**). Active range of motion and plantarflexion are preserved due to intact extrinsic muscles of the foot. It is always important to compare against the uninjured side as some motion may be retained even with injury. Obtain radiographs to rule out a fracture. This includes bedside ultrasonography of the Achilles tendon (**Figure 223-26**) to detect a partial or complete rupture.[34] This is especially useful when the differential diagnosis includes tendinitis versus a partial tendon rupture.

FIGURE 223-25. Achilles tendon rupture of the right foot. Note the edema and lack of plantarflexion. (Used with permission from Sherman SC: *Simon's Emergency Orthopedics*, 7th ed. New York: McGraw-Hill; 2014.)

A

B

FIGURE 223-24. The Thompson test. ***A.*** Squeezing the calf with an intact Achilles tendon causes plantarflexion of the foot. ***B.*** Squeezing the calf with a ruptured Achilles tendon causes no plantarflexion of the foot. (Used with permission from Sherman SC: *Simon's Emergency Orthopedics*, 7th ed. New York: McGraw-Hill; 2014.)

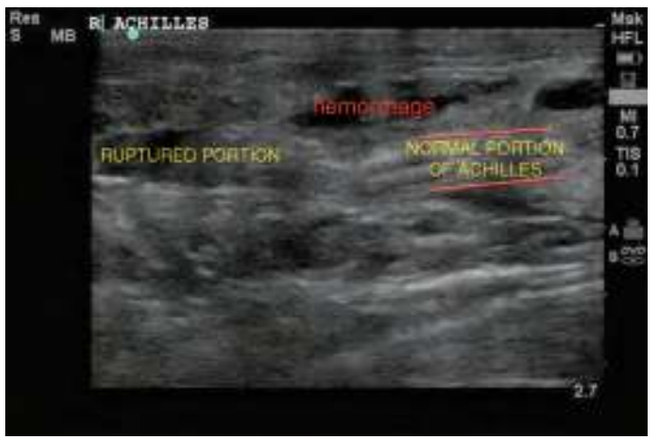

FIGURE 223-26. Ultrasound of an Achilles tendon rupture. (Used with permission of Emory Emergency Medicine Ultrasound Section.)

TREATMENT

Treatment in the Emergency Department is aimed at conservative measures.[48] It consists of NSAIDs, RICE (i.e., rest, ice, compression, and elevation), crutch use, and non–weight bearing. Apply a posterior long leg splint with the foot in 20° to 25° of plantarflexion (i.e., the equinus position) to oppose the tendon ends. Many Achilles tendon ruptures require surgical repair.[48,49] Arrange follow-up with a Podiatrist or Orthopedic Surgeon within 24 to 48 hours.

DIABETIC FOOT CARE

Diabetic patients are vulnerable to many systemic complications of their disease.[50,51] The diabetic foot syndrome is a major complication due to the pathogenic effects of plantar pressure on the foot. The three main risk factors leading to the development of serious cutaneous injury and infection include microcirculatory insufficiency, neuropathic changes, and an altered immune system. Diabetic foot syndrome is commonly triggered by ill-fitting shoes, poor foot hygiene, overlooked foreign bodies, callus formation, preexisting dry scaly skin with fissures, and possible underlying structural deformities.[52] The goal of diabetic foot care is preventive to avoid plantar pressure and formation of plantar lesions, especially ulcers.[53]

The diabetic patient faces a much higher likelihood of complications from the development of usually benign conditions due to plantar pressure (e.g., corns, calluses, paronychia, ingrown toenails, and ulcers).[51] The diabetic patient may not be aware of the development of these conditions because of the loss of protective sensory functions. The impact of these complications to the diabetic foot is staggering. A significant percentage of diabetics with a foot ulcer will eventually require an amputation.[54] **Early and aggressive diagnosis and treatment of common diabetic foot disorders is essential to maximize the chances for wound healing and reduce the likelihood of complications.**

The medical history and clinical examination are the most important aspect of evaluating the diabetic foot.[50] **Perform a careful dermatologic, neurologic, orthopedic, and vascular examination as well as an assessment of the patient's footwear.** A thorough review of the physical examination and assessment of the diabetic foot is beyond the scope of this chapter.

Radiographic evaluation is mandatory if osteomyelitis is suspected. Characteristic changes include soft tissue swelling, osteolysis, and periosteal elevation. The formation of a sequestrum (i.e., a highly opaque and smooth island of bone surrounded by areas of decreased bone density) may be seen. Radiographic evaluation often lags behind the clinical evidence. A bone scan or magnetic resonance imaging is more sensitive than plain radiographs in making an early diagnosis of osteomyelitis.

TREATMENT

Educate the diabetic patient regarding foot care. Instruct the patient on proper footwear (e.g., good fit, high toe box, a round toe, good support of the heel and arch), inserts to accommodate any plantar lesions, and soft leather. Instruct the patient to check their shoes for any foreign bodies, rough spots, or any pressure points at the time of purchase and daily before donning the shoe. Caution the patient to avoid extreme temperatures from the environment or when cleansing the feet. Limit standing to 20 to 30 minutes at a time if a foot lesion is present.

Treat hyperkeratotic lesions the same as for nondiabetics with periodic debridement. Apply a corn pad with a central aperture or a horseshoe-shaped pad to relieve pain and disperse pressure at bony prominences to the surrounding skin. Cut a hole in the patient's shoe over any nonweightbearing bony prominence to prevent irritation of the surrounding soft tissue. Apply a felt pad to cover the tops of any hammertoes. Apply a commercially available adhesive bunion pad or cut a bunion shield from 1 cm thick felt over a bunion or bunionette deformity.

Diabetic patients must be extra vigilant in caring for their toenails.[50,51] Some authors recommend that diabetics should not cut their own nails, corns, or calluses. Ingrown toenails (Chapter 218) are treated the same as for nondiabetics. **Treat mycotic infections aggressively.** These conditions are often unnoticed by diabetic patients because of their peripheral neuropathy and the loss of protective sensation. Mycotic lesions often present at an advanced stage. A simple but thorough vascular examination is mandatory if an infected or advanced lesion is present. Absent or diminished pedal pulses (i.e., posterior tibial and dorsalis pedis) may indicate lower extremity ischemia. This requires further vascular evaluation, either through noninvasive testing or outpatient management from a Vascular Surgeon.

The most common location for foot ulcers is the plantar surface of the forefoot. These are essentially pressure ulcers. Instruct the patient to avoid mechanical stress on the injured extremity as ongoing trauma prevents healing. Treatment from the Emergency Department is simple and consists of the application of a wound dressing, antibiotics if there is infection, bed rest or the use of crutches, and referral to a Podiatrist or Orthopedic Surgeon. The consultant may use sharp debridement of devitalized tissue from the wound area to promote wound healing.

A moist wound environment is important for wound healing. There are many commercially available foot care products with little proven efficacy. They may confuse the patient. A simple and clear-cut discharge strategy is wet-to-dry dressing changes twice a day. This can be continued in the home. There is controversy regarding the use of topical agents and foot soaks. Neither one has been shown to be beneficial in the healing of a diabetic foot ulcer. Cover the lesions with a pad to disperse pressure to the surrounding skin and protect the ulcer. Instruct the patient to place a pillow under the calf and allow the heel to overhang if there is a heel ulcer.

Treat infected ulcers aggressively with systemic antibiotics. Hospital admission is often required. Mild and most moderate infections can be treated with oral antibiotics on an outpatient basis with an appropriate candidate (e.g., reliable patient and good home support). The selected antibiotic must achieve good tissue levels and sufficiently cover most skin pathogens, especially aerobic gram-positive cocci. Wound infections are commonly polymicrobial with some drug resistance. Wound cultures may help guide therapy and focus the antibiotic choice. These patients require close follow-up within 24 to 48 hours for a wound check and a review of antibiotic therapy when the culture and sensitivity results are available.

Severe infections, with or without systemic symptoms, require hospital admission for parenteral antibiotics and surgical consultation. An experienced consultant will decide if the wound can be debrided at the bedside or necessitates the Operating Room. **Administer broad-spectrum intravenous antibiotics immediately after wound and blood cultures are obtained. The antibiotic regimen must cover aerobic and anaerobic gram-positive cocci as well as gram-negative organisms.** Examples include imipenem-cilastatin or vancomycin plus aztreonam plus metronidazole. A beta-lactamase inhibitor (e.g., ampicillin-sulbactam) or clindamycin plus a fluoroquinolone is recommended for less severe infections.

Osteomyelitis is a difficult infection to cure and requires a long course of antibiotics. A 6 week course of antibiotics is recommended, including 1 to 2 weeks of parenteral therapy. Surgical treatment may

include debridement of the infected bone if this will not compromise long-term foot function. Debridement will increase the likelihood of a cure and shorten the course of required antibiotics. Removal of the infected bone may correct any underlying bony deformity that may have originally caused the ulcer. Vascular reconstruction and/or amputation are considered for appropriate candidates.

Adjunctive medical therapy includes improving blood glucose, control of comorbid conditions, and medical nutrition to improve the healing potential of foot wounds. A discussion of these topics is beyond the scope of this chapter.

ONYCHOMYCOSIS (FUNGAL TOENAIL INFECTIONS)

Approximately 20% of the U.S. population between the ages of 40 and 60 have fungal nail disease.[55] Onychomycosis is most often caused by dermatophytes of the *Trichophyton* genus. Nondermatophytes (e.g., molds or yeasts) are also causative organisms. Onychomycosis may occur posttraumatically. It is commonly seen in the first and fifth toenails where the greatest shoe friction occurs. The infection occurs under or within the nail plate and causes proliferation of keratinized debris under the nail. The nail will become opaque, discolored, thickened, and brittle if the infection progresses (**Figure 223-27**). Formal diagnosis is made by identifying hyphal fragments upon microscopic examination of nail scrapings placed in a potassium hydroxide solution. Onychomycosis is usually diagnosed based upon the clinical examination and therapy is often empiric. "Skipped" normal nails are often seen in fungal toenail infections. Psoriasis or other inherited nail dystrophies involve all 10 nails.

Four major types of mycotic nail infection have been identified. These include distal subungual onychomycosis, white superficial onychomycosis, proximal white subungual onychomycosis, and candidal onychomycosis. These various entities differ in the pattern of fungal invasion of the nail plate and the causative organism. Clinical symptoms of mycotic toenail infections include brittleness, color changes, hyperkeratosis, onycholysis (i.e., separation of the nail from its bed), and paronychial inflammation (**Figure 223-27**).[56] Distal subungual onychomycosis is the most common type. It affects the most distal part of the nail bed and nail plate. White superficial onychomycosis is seen mainly in toenails. It produces a white, brittle appearance due to direct invasion of the nail plate's surface. Proximal white subungual onychomycosis is very rare. It is due to fungus invading the cuticle and turning the proximal nail plate white.

FIGURE 223-27. Onychomycosis. (Used from James Heilman, www.commons.wikimedia.org.)

The morphology of the nail plate infection at the time of presentation may help determine the need for additional therapy. Dermatophytoma or longitudinal streaking with nail plate changes is produced by keratin debris and filled with dermatophytes. This produces a relatively inaccessible foci of infection. Lateral nail plate involvement or onycholysis results in a separation of the nail plate from the nail bed, reduces the vascular access to the nail plate, and limits the penetration of systemic therapy. Thick nails > 2 mm may indicate matrix involvement and keratinaceous debris. This can limit drug diffusion regardless of the delivery system. These presentations all limit the access of topical and systemic agents to the site of infection and all require adjunctive therapy that is directed to the physical removal of the keratinaceous debris. Mechanical or chemical debridement is essential for effective therapy.[57]

TREATMENT

Treatment of onychomycosis may be divided into topical, systemic, and surgical therapies.[58] Topical agents have been found to be effective if the fungal infection is limited to the distal portion of the nail plate.[59] Simple mechanical filing of the nail plate, curettage of the necrotic subungual tissue, trimming of the nail plate, and topical antimycotic therapy are often effective.

Treatment of more advanced infections begins with an empiric trial of an oral antifungal agent.[56,58,60] Itraconazole (e.g., Sporanox) is a newer member of the azole family and has a very broad antimycotic spectrum of action. It is effective in the treatment of dermatophytes, yeasts, and some nondermatophyte molds. Terbinafine (e.g., Lamisil) is an orally active allylamine that works against dermatophytes but not yeast. Both itraconazole and terbinafine are associated with a significantly shorter treatment time and higher clinical cure rate over the older systemic agents (e.g., griseofulvin). Studies have shown that pulse therapy is an effective treatment regimen for itraconazole and terbinafine.[61-65] This consists of 1 week of daily treatment each month for 3 to 4 consecutive months. Inform patients of any potential symptoms, side effects, and drug reactions. Obtain a baseline liver profile, chemistry panel, and complete blood count. **There is a small risk of hepatotoxicity from oral antifungal agents.** It is recommended that a Primary Care Physician, Podiatrist, or Orthopedist prescribe these medications rather than an Emergency Physician. Any patient receiving long-term antifungal therapy for onychomycosis must undergo periodic laboratory monitoring.

More extensive involvement or failure of outpatient therapy may require removal of the nail plate, an oral antifungal agent, and local wound care. Apply a topical antifungal agent twice daily while the new nail plate develops. Refer the patient to a Podiatrist for refractory chronic onychomycosis, especially the patient who has diabetes or vascular disease. Surgical treatment may include nail plate removal with nail bed debridement (with or without chemical destruction of the nail bed matrix). Close follow-up is mandatory.[66]

SUMMARY

Foot complaints are commonly encountered in the Emergency Department. Many of these conditions can be managed using easy, simple, and straightforward techniques. Refer the patient to a Podiatrist or Orthopedic Surgeon for plantar lesions in high-risk patients (e.g., poor wound healing, diabetes, peripheral neuropathy), conditions needing a specialist, or chronic conditions needing continuing care.

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SECTION 16

Miscellaneous Procedures

224 Relief of Choking and Acute Upper Airway Foreign Body Removal

Guy Shochat and Jacqueline Nemer

INTRODUCTION

The National Safety Council reports that airway foreign body (FB) obstruction accounts for over 4800 adult and pediatric deaths per year in the United States.[1] Accidental FB obstruction of the airway is the fourth leading cause of accidental death in children less than 6 years of age.[1] Contemporary data are lacking regarding the type of obstructing object in fatal cases. Older research data for nonfatal choking episodes in children reported a more common association with food to nonfood objects by a 2:1 ratio. There is some variation but the most common objects are candy (e.g., hard candy which leads softer candy and gum); peanuts which lead the food items category; and coins which lead the nonfood group.[2] Mortality due to airway FB obstruction is bimodal and increases again in the elderly.[1] Food impaction is the primary etiology in the elderly, institutionalized, and those with cognitive impairment or intoxication.[2-4]

ANATOMY AND PATHOPHYSIOLOGY

Developmental differences in the pediatric airway make visualization and removal of FBs more difficult than in the adult patient. The pediatric tongue occupies a larger percentage of the oral cavity and the oropharynx. The pediatric epiglottis is larger, U-shaped, and more cephalad. The pediatric epiglottis does not attain the adult position until age 4. The narrowest portion of the pediatric airway and a likely site of obstruction is below the vocal cords at the level of the cricoid cartilage, making removal more difficult and more likely to require endoscopy.[3-6]

The clinical presentation and patient management are dependent on the anatomic site of obstruction, the degree of obstruction, and the size of the FB. Patients with a supraglottic obstruction classically present with inspiratory stridor while those with an infraglottic obstruction present with expiratory wheezes. Infraglottic FBs usually lodge in the trachea or the main stem bronchus and will require instrument removal. A FB simply contacting the vocal cords while moving through the glottis may result in laryngospasm which can completely obstruct the airway, even after the expulsion of the FB.[5,6]

Partial airway obstructions generally allow at least limited amounts of air passage and the potential for removal by the patient's cough reflex. Complete airway obstructions can result in a silent cough followed by loss of consciousness if not cleared by coughing or assistive maneuvers. A larger FB is more likely to lodge at or above the vocal cords where it can cause a complete airway obstruction. Sharp, small, and thin FBs are more likely to partially obstruct between or below the vocal cords and result in difficulty breathing and dysphonia.[5,7-9]

Pediatric and adult patients provide different clues to airway FB obstruction. Adults and older children typically indicate the "universal choking sign" by clutching or pointing to their neck and nodding affirmatively when asked if choking. The symptoms in an infant or toddler may range from tachypnea, subtle stridor, wheezing to respiratory distress, cyanosis, and unconsciousness.[10,11]

DIGITAL REMOVAL (FINGER SWEEP)

Perform digital removal of an intraoral FB only when it is directly visualized in the patient's mouth or oropharynx.[10] Blind finger sweeps are contraindicated to prevent inadvertently pushing the FB into a more distal location and converting an incomplete supraglottic obstruction into a difficult to remove and complete infraglottic obstruction.[12]

Open the patient's mouth and airway using the thumb and fingers of the nondominant hand to grab the tongue and mandible and then lift it anteriorly. Use a hooking action with the index finger of the dominant had to dislodge and remove the FB.

The most important potential complication is the conversion of a partial airway obstruction into a complete airway obstruction. Local trauma can result in intraoral abrasions, bleeding, and dental trauma. The rescuer can sustain digital abrasions and lacerations from the patient's teeth. **Do not place your fingers into the mouth of a conscious patient as this can result in a significant bite injury.[13,14]**

BACK BLOWS AND CHEST THRUSTS IN INFANTS

An uncoordinated swallow mechanism and the lack of molar dentition predispose the infant to an airway FB obstruction within their narrow and pliable glottis and trachea.[15] **Perform back blows and chest thrusts on the infant with a witnessed or suspected airway FB obstruction who suddenly develops respiratory distress, appears cyanotic, or becomes unconscious.[14]**

Initiate back blows and chest thrusts to attempt to dislodge the obstruction in the conscious infant showing signs of airway obstruction.[16] **Use this technique on infants less than 1 year of age. Use abdominal thrusts if the patient is over 1 year of age. Closely observe the infant but do not intervene if they are maintaining their airway as indicated by clear breathing and an effective cough. Their cough reflex may allow them to dislodge and expel the FB.[10]**

Support the infant's head and neck with one hand. Place the choking infant with their head lower than their torso in a prone position over the arm and resting on the thigh or the lap of the Emergency Physician (**Figure 224-1*A***). **This head-down position allows gravity to assist in the expulsion of the dislodged FB.** Perform five firm back blows with the heel of a hand between the infant's shoulder blades (**Figure 224-1*A***). Turn the infant face up in the supine position with their head still below the torso (**Figure 224-1*B***). Use the heel of the hand (**Figure 224-1*B***) or two or three fingers (**Figure 224-1*C***) to deliver up to five chest thrusts over the sternum and depress the sternum ½ to 1 inch per thrust while avoiding the lower tip of the sternum. (**Figure 224-1*B***). An alternative is to

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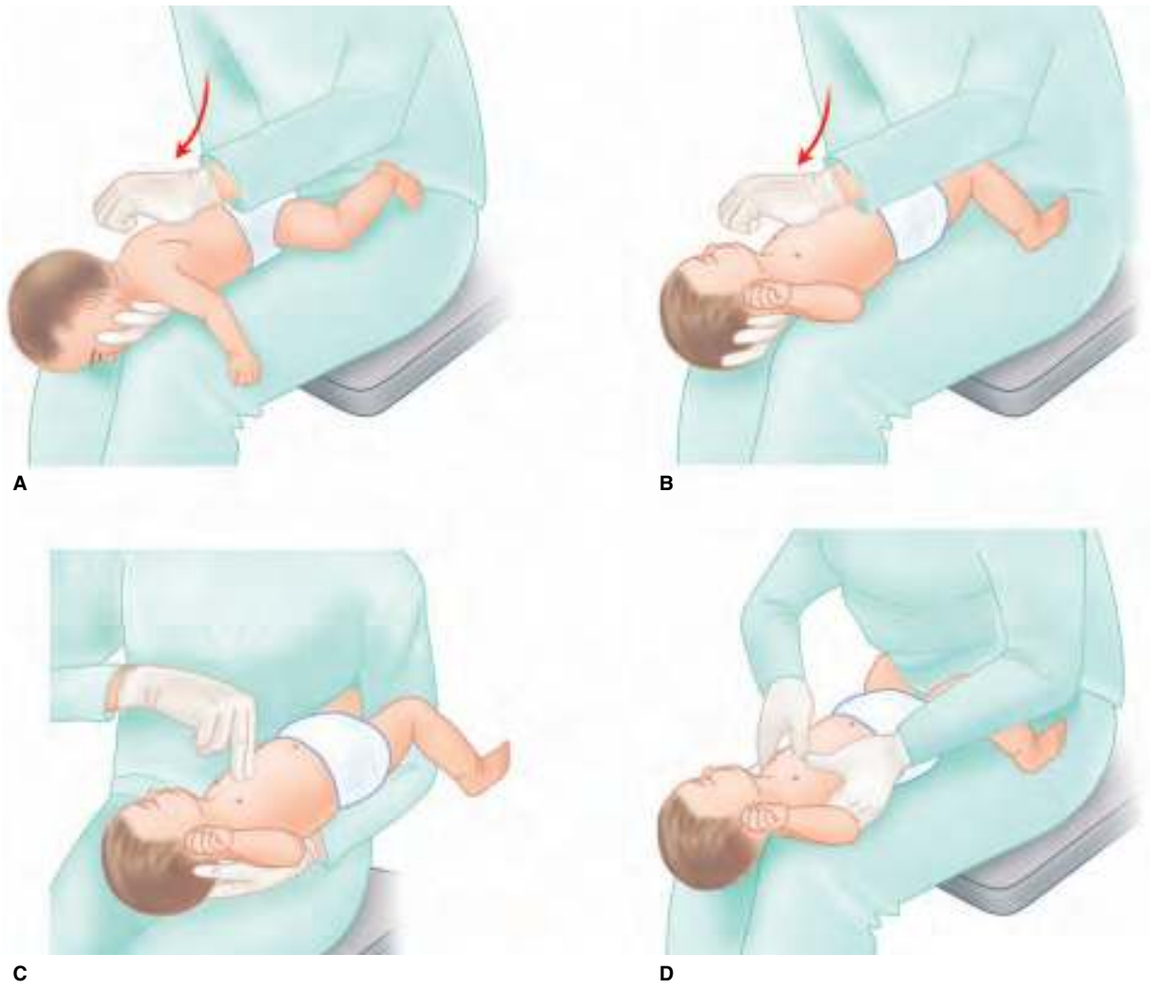

FIGURE 224-1. Relief of an airway foreign body obstruction in the infant. ***A.*** Back blows using the heel of a hand. ***B.*** Chest thrusts using the heel of a hand. ***C.*** Chest thrusts using two fingers. ***D.*** Chest thrusts using both thumbs.

use both thumbs (**Figure 224-1*D***). The use of thumbs is performed when one or two rescuers are present. Place both thumbs together over the lower half of the infant sternum just below the intermamillary line. Encircle the infant's chest with both hands, spreading the fingers around the posterior thorax. Compress the sternum while squeezing the thorax to apply counterpressure. Repeat this sequence of back blows and chest thrusts until the FB is expelled or the infant becomes unresponsive.[10]

The procedure is successful when the FB is expelled. Observe the infant closely following the expulsion of the FB for any signs of persistent respiratory distress due to an incompletely expelled FB or injury from the removal techniques.

The Emergency Physician must first check the mouth and remove any obvious FB in the unresponsive infant. **Blind finger sweeps are always contraindicated.** Case reports describe wedging of FBs into more distal locations and converting a partial airway obstruction into a complete airway obstruction.[7] The Emergency Physician must immediately initiate bag-valve-mask ventilation in the apneic infant. Continue the cycles of back blows and chest thrusts with bag-valve-mask ventilation while preparing for immediate intubation.

Injuries may result from back blows and chest thrusts (e.g., back and chest wall contusions). Vigorous back blows can result in rib fractures and lung trauma (e.g., pulmonary contusion). Nausea and vomiting can occasionally result. Chest thrusts may result in rib and sternal fractures, myocardial contusions, lung puncture or contusion, pneumothoraces, and intraabdominal injury.[7,10] **These potential complications are serious. Failure to relieve the airway FB obstruction can result in death.**

The Emergency Physician must maintain a low threshold for performing back blows and chest thrusts to relieve an airway FB obstruction in infants. Airway FB obstruction in an infant is more common, more difficult to assess, and more often fatal than in an older child, adolescent, or adult. **The key steps include positioning the infant head down over the Emergency Physician's arm or leg with their head lower than their trunk, performing five back blows alternating with five chest thrusts, and repeating this sequence continuously until the FB is expelled.**

HEIMLICH MANEUVER

Dr. Henry Heimlich first proposed the Heimlich maneuver in 1974.[17] Controversy soon followed as he publicly denounced the recommendations made by the American Red Cross and the American Heart Association. He claimed that back blows previously listed as a first-line treatment were "death blows" and that various national organizations were involved in "Watergate cover-ups" intended to prevent acceptance and widespread use of his maneuver.

No prospective clinical trials have been reported on the various techniques used to relieve an airway FB obstruction. Physiologic data demonstrate that each technique produces varying effects on

intrathoracic pressure and airflow to overcome the static resistance of the obstructing FB.[18] Back blows generate a substantial increase in intrathoracic pressure over a very short time and potentially dislodge an airway FB without its expulsion from the airway. Abdominal thrusts generate more prolonged increases in airway pressure and flow rates, theoretically allowing for expulsion of the dislodged FB. The Emergency Physician must be prepared to quickly utilize a combination of these techniques for the dislodgement and expulsion of an airway FB obstruction.[10,11,13]

Abdominal thrusts can be a lifesaving procedure but they are also associated with a wide range of serious complications. Reported complications include solid organ injury (i.e., liver, spleen, and pancreas), perforation of the esophagus or stomach, diaphragmatic rupture, fractures (e.g., ribs, sternum, or vertebra), hemothorax, pneumothorax, or a pulmonary contusion.[19-22] **Any patient that is symptomatic following this maneuver must be thoroughly evaluated and closely observed for potential complications.**

ABDOMINAL THRUSTS IN THE PATIENT OVER 1 YEAR OF AGE

Abdominal thrusts may be considered in the conscious patient over 1 year of age with a witnessed or presumed aspiration who has findings of complete airway obstruction (i.e., cannot speak, cough, or breathe effectively).[10] Older children and adult patients will often present and be grasping or pointing to their neck with the "universal choking sign" (**Figure 224-2**).[11] Young children will often have a history or presentation consistent with an airway FB obstruction. This technique can be performed from behind the patient if they are conscious and have signs of a complete airway obstruction. **Abdominal thrusts should NOT be performed in patients less than 1 year of age due to the significant risk of liver injury.[10,14]** The patient's cough reflex may expel the partial obstruction if they are able to breath, cough, and speak. A patient with a partial FB obstruction must be closely observed. The partial FB obstruction may convert into a complete airway obstruction. **Abdominal thrusts should be performed only for a complete airway obstruction.**

Stand directly behind the conscious patient (**Figure 224-2**). Wrap both arms around the patient's waist. Make a fist with one hand and place this hand with the thumb side of the fist against the midline of the patient's upper abdomen between the ribcage and umbilicus. The other hand is placed over the fisted hand and grasps the fisted hand (**Figure 224-2**). Press inward with a deliberate and quick upward thrust. **Limit the force of the thrust to the hands without squeezing the ribcage. Repeat these quick upward thrusts until the FB is expelled or the patient becomes unresponsive.** Observe the patient for signs of respiratory distress from persistent obstruction or from injury sustained during abdominal thrusts after the FB is expelled.

The next step in the Basic Life Support (BLS) protocol is to perform abdominal thrusts if the patient loses consciousness. The Emergency Physician should intubate and provide advanced care if the patient become unresponsive.

Incorrect hand placement can result in sternal and rib fractures. Significant but rare complications may occur including thoracic and intraabdominal injuries. Perform close observation and evaluation on any patient who is symptomatic after undergoing abdominal thrusts. Failure to relieve an airway FB obstruction can result in death.

ABDOMINAL THRUSTS IN THE UNCONSCIOUS PATIENT

Perform abdominal thrusts in the patient with a witnessed or suspected airway FB obstruction, if the previously described maneuvers have failed, or in the unresponsive patient.[14] Do not perform this maneuver in patients less than 1 year of age due to the significant risk of liver injury.

Place the patient supine on the floor. A bed is not firm enough to allow the appropriate generation of intraabdominal pressure. Open their mouth. **Perform a finger sweep only if a FB is clearly visible.** Place the heel of one hand in the midline of the abdomen and just above the umbilicus. Place the other hand over the first (**Figure 224-3**). Apply

FIGURE 224-2. Abdominal thrusts in a conscious patient.

FIGURE 224-3. Abdominal thrusts in the unconscious patient.

five rapid and forceful thrusts downward and upward, forcing the diaphragm upward and compressing the lungs. Reassess the patient's airway. Inspect their mouth for a FB. Remove the intraoral FB if it is visible. If the FB is not visible, repeat the abdominal thrusts or attempt another technique.

One case study reported that increased airway pressure was generated using a knees-to-chest approach in the supine position. While there are no further studies to support this technique, it may be a simple adjunct when other methods have failed.[23]

Closely observe the patient after the FB is expelled for signs of respiratory distress from persistent obstruction or from injury from the abdominal thrusts. Incorrect hand placement can result in sternal and rib fractures. Place the patient in the semirecumbent position or sitting upright if no contraindications exist after the FB is removed.

OBSERVATION UNTIL REMOVAL IN THE OPERATING ROOM

It is best not to manipulate the airway or attempt intubation in a stable patient with an airway FB who is moving air and breathing. The airway may be best controlled in the Operating Room at the time of the actual FB removal. Manipulation of the airway can result in a FB becoming dislodged and turn a partial airway obstruction into a complete airway obstruction. The bronchoscopy equipment is available for urgent use if needed in the Operating Room.

The timing of endoscopy and airway FB retrieval must be based upon each individual patient. It is essential not to waste time if impending airway obstruction exists or the patient is unstable. This is an emergent situation warranting immediate notification and mobilization of an Anesthesiologist, Otolaryngologist and/or Pulmonologist, and the Operating Room.

It is appropriate to wait for the NPO (nothing by mouth) status to decrease the risk of aspiration which can further compromise the situation if the patient is stable. It is appropriate to wait in a stable patient to assemble the appropriate and best team to care for the patient. Using personnel who are unfamiliar with endoscopy can create a compromised and stressful situation.

DIRECT LARYNGOSCOPY

It is possible to remove FBs located within the hypopharynx in a stable patient.[24] **It may sometimes become necessary to attempt FB removal by direct laryngoscopy in a decompensating patient when the specialized consultants are not available.** Typical FBs that may be removed include pieces of food and fishbones. Anteroposterior and lateral soft tissue radiographs of the neck may localize a radiopaque FB. There has been an increase in computed tomography (CT) scans to identify FBs lodged in the pharynx, hypopharynx, or esophagus due to their superiority over plain radiographs. Their use needs to be weighed against the risk of radiation exposure.[3,4,9] Fiberoptic laryngoscopy can be used to locate and identify a FB.[5,7-9] Experienced and trained Emergency Physicians can retrieve a FB with graspers via an instrument channel on a flexible fiberscope (Chapter 28). The technique of awake topically anesthetized direct laryngoscopy can be used to locate and remove a supraglottic FB.

EQUIPMENT

- Intubation equipment (Chapter 18)
- Percutaneous transtracheal jet ventilator
- Cricothyroidotomy equipment (Chapter 32)
- Suction source, tubing, and catheter
- Airway forceps (i.e., Boedeker, Magill, or Tylke)
- Laryngoscope with a variety of blades (Chapter 18) or a video laryngoscope (Chapter 20)
- Topical or nebulized anesthetic
- Fiberoptic nasopharyngoscope or bronchoscope (Chapter 28)
- Oxygen

Direct laryngoscopy and bronchoscopy in a child or adult with an airway FB is a dangerous situation. The procedure may result in a partial airway obstruction becoming a complete airway obstruction. **Always have a cricothyroidotomy tray and/or percutaneous transtracheal jet ventilation device immediately available.**

All equipment must be selected, assembled, and ready for use. Place the patient in full monitoring (pulse oximeter, cardiac monitor, and noninvasive blood pressure cuff). Apply a topical anesthetic spray to the oropharynx and the base of the tongue to blunt the gag reflex. Place the patient supine or semirecumbent. Administer a small dose of an intravenous sedative if required and not contraindicated.

Slowly and gently insert the laryngoscope blade. An alternative to a traditional laryngoscope is a video laryngoscope.[25] The video laryngoscope may provide a better field of view with less manipulation. **Do not immediately insert the laryngoscope blade all the way.** Stop frequently to lift the laryngoscope and look for the FB. Slow insertion and frequent looks will prevent the laryngoscope blade from pushing the FB further into the airway. Elevate the patient's tongue and jaw. Grasp the FB with an airway forceps.[24,26] If the FB is too large or impacted, it may need to be crushed with the forceps to securely grasp it as one piece.[27] Withdraw the forceps with the FB followed by the laryngoscope.

One case report describes passing a Foley catheter distal to the FB under direct visualization with a laryngoscope.[27] The balloon was then inflated with 5 mL of air and subsequently pulled back until the FB could be grasped and removed with a Magill forceps. There were no reported complications. It should be noted the FB in this case was impacted within the esophagus at the level of the cricoid cartilage. Do not use this technique with subglottic airway foreign bodies since retraction of a dilated catheter balloon through the glottic structures may cause injury, edema, bleeding, and further worsen the obstruction.

One review reported no complications with subglottic FB removal when utilizing Magill forceps under direct laryngoscopy.[28] Potential complications include oropharyngeal trauma resulting in glottic edema and bleeding.

All patients who have undergone FB retrieval require at least 4 to 6 hours of airway observation in a monitored setting to assess for persistent or delayed respiratory distress resulting from incomplete removal or procedural complications. Administer racemic epinephrine treatments and intravenous dexamethasone as needed. Prolonged observation is warranted along with consideration of an admission to the Intensive Care Unit if these treatments are needed. Discharge from the hospital is acceptable when the patient is breathing comfortably and no longer in danger of airway compromise following several hours of observation. Some patients may be discharged to home the same day. Others may require multiple days of airway support and observation. Potential complications include oropharyngeal trauma resulting in glottic edema and bleeding.

OROTRACHEAL INTUBATION

Orotracheal intubation can be attempted if the airway obstruction progresses rapidly and the patient cannot ventilate. Intubation can be used to force the FB into one main stem bronchus and allow ventilation of the other lung. One-lung ventilation will keep the patient alive until the FB can be removed.

Position the laryngoscope to visualize the larynx. Grasp the FB with an airway forceps and remove it if it is visualized. Intubate the patient if no FB is visualized. Insert the endotracheal tube if the patient is unable to ventilate. Advance the endotracheal tube as far as it will advance. Use a smaller endotracheal tube or a bougie if the first endotracheal tube will not advance. The patient will be able to be ventilated if this is successful. The endotracheal tube can then be withdrawn and positioned with its tip above the carina to try to optimize ventilation. **As an alternative, properly insert and position the endotracheal tube above the carina and then advance a bougie through the endotracheal tube in attempt to move the FB distally.**

Always be prepared to perform a cricothyrotomy (Chapter 32) or transtracheal jet ventilation (Chapter 31). Transtracheal jet ventilation allows for short-term oxygenation, is temporary, and may allow time for endoscopy for the FB retrieval.

MECHANICAL DEVICES

The LifeVac device was developed for clearing a FB obstruction from the upper airway (LifeVac LLC., Springfield Gardens, NY).[29] The kit contains the LifeVac device, adult and pediatric face masks, and instructions for use (**Figures 224-4*A* and 224-4*B***). It is a single-use suction device that is portable and used with the included face mask (**Figure 224-4*C***). The device works like a toilet plunger (**Figure 224-4*D***). The face mask makes a tight seal to the face and uses pressure to dislodge the FB from the airway. It can be used by the lay public or health-care workers. The company website notes it generates 300 mmHg of suction and three times the force of any choking pressure.

The Dechoker device was developed for clearing a FB obstruction from the upper airway (Dechoker, Prospect, KY). It looks like a big syringe with a face mask and a tube at its distal end (**Figure 224-5*A***). The company makes a wall mount that is often placed by first aid kits (**Figure 224-5*B***). The device is available in size-specific kits of toddler, child, and adult size. The kit contains the Dechoker device with a size-specific face mask and instructions for use (**Figures 224-5*A* and 224-5*B***). It is a single-use suction device that is portable and used with the attached face mask (**Figure 224-5*C***). The face mask makes a tight seal to the face and uses pressure to dislodge the FB from the airway. It can be used by the person choking, lay public, or health–care professionals (**Figures 224-5*D* and 224-5*E***).

FIGURE 224-4. The LifeVac. ***A.*** The packaged kit. ***B.*** The contents of the kit. ***C.*** The device with a face mask. ***D.*** The use of the device. (Photos courtesy of LifeVac LLC.)

A

B

C

FIGURE 224-5. The Dechoker. ***A.*** The device. ***B.*** The wall-mounted kit with the device and labels of the size. ***C.*** The device on a manikin. ***D.*** The instructions for use of the device. ***E.*** The instructions for self-use of the device. (Photos courtesy of Dechoker.)

D

E

FIGURE 224-5. (*Continued*)

These above mechanical devices have not undergone clinical trials. Their use, for now, must be a last effort to relieve the choking victim if other methods fail. First try abdominal thrusts, back blows, digital removal, laryngoscopy if the equipment is available, and intubation if the equipment is available.

AFTERCARE

Most patients should be breathing spontaneously after the extraction, removal, or retrieval of an airway FB. An endotracheal tube may rarely, in the presence of significant laryngeal or tracheobronchial edema, need to remain in place temporarily. This would require admission to an Intensive Care Unit. Humidified oxygen is helpful to keep the airway moist and prevent mucous crusts from forming. A postprocedural radiograph will help to determine any complications (e.g., subcutaneous air, air in the soft tissues, a pneumothorax, or any changes to the lung fields) following the extraction. The radiograph will confirm the endotracheal tube position.

All patients who have undergone FB extraction, removal, or retrieval require at least a few hours of airway observation in a monitored setting.[30] Racemic epinephrine treatments and intravenous Decadron can be administered as needed. Discharge from the hospital is acceptable when the patient is breathing comfortably and no longer in danger of airway compromise. Some patients may be discharged home the same day while others may require multiple days of airway support and observation.[30]

COMPLICATIONS

Complications from the foreign bodies themselves include hypoxia leading to cerebral anoxia if not identified. Cardiac arrhythmias can occur from hypoxia or direct pressure on the left mainstem bronchus. Other complications include laryngeal or tracheobronchial edema from the FB or the instrumentation of the airway. Mucosal irritation can instigate a bronchitis, pneumonia, or tracheitis. A pneumomediastinum and/or a pneumothorax is possible. The complications specific for each technique have been described previously in the respective sections.

A~~n~~ FB in the hypopharynx can be pushed distally and result in a total airway obstruction. The FB needs to be quickly removed or pushed back down into one of the mainstem bronchi to allow ventilation of at least one lung or a surgical airway needs to be performed. Failure to react appropriately in this situation can result in asphyxiation and death.

SUMMARY

Airway FB obstruction is a significant cause of morbidity and mortality, especially in pediatric, debilitated, and elderly patients. Presentations can range from the obvious to the very subtle. Close observation is indicated to determine if the patient's own cough reflex can expel the FB for the awake patient. Stable patients can be observed closely. Manage a stable patient with an airway FB with a planned endoscopic removal in a setting that allows for endoscopic or surgical management if a complete obstruction occurs.

Age-appropriate BLS maneuvers should be performed in conscious patients with a complete airway obstruction or partial airway obstruction with severe respiratory distress.[10,11] The patient requires emergent intubation for ventilation and airway management if they become unconscious. The Emergency Physician may attempt FB removal if it is visualized. A temporizing measure is to force the FB distally in the airway and allow for partial ventilation.

The flexible fiberoptic endoscopy can be used to localize FBs. Consultants are not always immediately available. Direct laryngoscopy can be used to localize and facilitate forceps removal of the FB. This is especially true for the FB when it is low risk for fully obstructing the airway (e.g., a fish bone). Direct laryngoscopy can be used if a patient is deteriorating and requires management to prevent imminent complete airway obstruction. Observe all patients who have airway FB removal for complications if not admitted for observation.

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225 Induction of Therapeutic Hypothermia (Targeted Temperature Management)

Eleanor Dunham

INTRODUCTION

Sudden cardiac death claims more than 350,000 victims in the United States every year.[1] The mortality rate for out-of-hospital cardiac arrest remains a staggering 65% to 95%, with only 10% to 20% of survivors discharged from the hospital with a good neurologic outcome.[2] **Therapeutic hypothermia for the treatment of comatose survivors of cardiac arrest is a proven therapy to improve survival and neurologic outcome.**[3-5]

Many consider therapeutic hypothermia to be a relatively new concept. Experiments with deep therapeutic hypothermia began in the 1940s with initially mixed results. In the 1950s, studies examined moderate hypothermia in the range of 26°C to 32°C (i.e., 78.8°F to 89.6°F) in comatose survivors of cardiac arrest and reported a trend toward improved outcomes but were complicated by difficult-to-control side effects. Additional animal studies of mild hypothermia in the range of 32°C to 35°C (i.e., 89.6°F to 95°F) in the 1980s and small clinical trials in the 1990s demonstrated that even mild hypothermia provided protective benefits with far fewer side effects.[2]

Two landmark randomized trials published in 2002 specifically examined the use of mild hypothermia in comatose survivors of witnessed cardiac arrests with initial rhythms of pulseless ventricular tachycardia or ventricular fibrillation.[6,7] **Meta-analyses report that only seven patients need to be treated to save one life and only five patients need to be treated to prevent one poor neurologic outcome.**[8,9] Utilizing strict screening criteria of these studies resulted in only 10% of screened patients being eligible. Follow-up studies suggest a wider benefit in patients with other rhythms at presentation, patients with cardiogenic shock, and those requiring percutaneous cardiac intervention (i.e., angioplasty and stenting).[2,3,10-18]

Preliminary studies of therapeutic hypothermia for other indications including anaphylaxis, acute respiratory distress syndrome, heatstroke, myocardial infarction, near-drowning, near-hanging, pregnancy, strokes, status epilepticus, subarachnoid hemorrhages, toxins, and traumatic brain injury have reported only limited data or conflicting results.[19-32] Recent studies have shown that therapeutic hypothermia in comatose pediatric patients who initially survived out-of-hospital arrest does not improve survival or neurologic outcome after 1 year.[33] The exception is studies of neonates with perinatal asphyxia that reported similar results to adult cardiac arrest with a number needed to treat of six for one favorable outcome.[3] Extensive research since 2002 has been conducted to determine the effectiveness of therapeutic hypothermia for other indications as well as the optimal timing, duration, target temperature, and techniques.

The International Liaison Committee on Resuscitation Recommendations (ILCOR) and American Heart Association (AHA) Resuscitation Guidelines for 2015 state that targeted temperature management is recommended between 32°C and 36°C and is to be maintained for at least 24 hours.[34] The exact temperature for induction and maintenance of hypothermia is unknown.[35-40] ILCOR recommends against routine use of prehospital cooling with rapid infusion of large volumes of cold intravenous fluids immediately after return of spontaneous circulation (ROSC).[34]

The AHA recommends that active rewarming should be avoided in comatose patients who spontaneously develop a mild degree of hypothermia after resuscitation from cardiac arrest during the first 48 hours after ROSC.[41] They cited reasons such as not enough data, not part of Advanced Cardiac Life Support (ACLS) protocol, too technically difficult, or that it had not been considered.[17] The induction of therapeutic hypothermia for comatose survivors of cardiac arrest is relatively straightforward in any Emergency Department. **The creation of collaborative protocols between Emergency Physicians, Intensivists, Cardiologists, and Neurointensivists is essential to producing the best outcomes at each institution.**

ANATOMY AND PATHOPHYSIOLOGY

The postcardiac arrest syndrome has recently been defined as a complex pathophysiologic process involving brain injury, myocardial dysfunction, systemic ischemia and reperfusion response, and the underlying persistent precipitating pathology.[3] Brain injury is the reported cause of death in two-thirds of patients after an out-of-hospital arrest and one-quarter of patients after an in-hospital cardiac arrest.[18] Multiple cellular mechanisms contribute to neuronal apoptosis and necrosis including the formation of free radicals, disruption of calcium homeostasis, excitotoxicity, altered gene expression, mitochondrial dysfunction, and inflammation. These cellular mechanisms at the tissue level lead to failure of cerebral autoregulation and ultimately hypotension, hypoxemia, brain edema, pyrexia, hyperglycemia, and seizures.[3]

The post–cardiac arrest state does not result in myocardial infarction but only myocardial stunning for 24 to 48 hours, producing a reduced ejection fraction and a reduction in end organ oxygen delivery.[3] The accumulated oxygen debt activates an inflammatory endothelial response further worsening oxygen delivery. The vicious cycle of oxygen debt and resultant inflammation does not end with reperfusion. The burst of reactive oxygen intermediates further exacerbates the inflammatory response and organ injury. The resultant state of systemic ischemia with reperfusion response leads to intravascular volume depletion, changes in vasoregulation, decreased oxygen utilization and delivery, and an increased risk of infection.

The underlying persisting pathology that caused the cardiac arrest must be addressed. Acute coronary syndrome is strongly implicated in up to 50% of out-of-hospital cardiac arrests.[12] Elevations of troponin T are present in up to 40% of patients at the time of cardiac arrest suggesting an ongoing cardiac ischemia or injury hours prior to the cardiac arrest. Additional etiologies of the cardiac arrest to consider include pulmonary embolism, primary pulmonary disease, sepsis, drug toxicity, or severe hemorrhage.

Hypothermia improves outcomes through several proposed mechanisms.[39,42] Metabolic demand is reduced 5% for each degree Celsius reduction in core body temperature. At the cellular level hypothermia decreases adenosine triphosphate demand preventing intracellular acidosis and stabilizing cell membranes. At the tissue

level, hypothermia leads to decreased vascular permeability at the blood-brain barrier thus decreasing edema. Hypothermia interrupts the inflammatory cascade by inhibiting neutrophils, reduces the production of proinflammatory cytokines, and helps to prevent the free radical production associated with reperfusion injury.

INDICATIONS

Hypothermia is most effective in patients meeting the strict inclusion criteria of the initial landmark studies. Begin the induction of hypothermia in the Emergency Department for comatose survivors of cardiac arrest with a presumed cardiac etiology, an initial rhythm of ventricular fibrillation or pulseless ventricular tachycardia, and between 18 and 75 years of age.[3,9] Studies have demonstrated improved outcomes in patients with other initial rhythms (e.g., pulseless electrical activity and asystole), comatose survivors of in-hospital cardiac arrest, patients requiring percutaneous cardiac intervention, and those suffering from cardiogenic shock.[12,13,15,16,39,43] Consider the induction of therapeutic hypothermia when the cardiac arrest is witnessed, there is < 30 minutes from the time of the collapse to the start of resuscitative efforts, it is < 60 minutes from collapse to ROSC, the patient has a core body temperature > 35°C (i.e., 95°F), and blood pressure can be maintained > 80 mmHg with or without intravenous (IV) fluid boluses and pressors. The benefits have not been shown for in-hospital arrest.[44] The use of therapeutic hypothermia in this group is currently being studied further. Refer to **Figure 225-1** regarding a sample induction of therapeutic hypothermia checklist and clinical pathway.

CONTRAINDICATIONS

Hypothermia is contraindicated in patients with a known status of do not resuscitate (DNR) or do not intubate (DNI), a terminal illness, severe comorbidities, multiorgan failure before arrest, or who are comatose due to a noncardiac etiology. Patients with a preexisting coagulopathy or pregnancy have been excluded from most studies. One case of successful therapeutic hypothermia in pregnancy has been reported.[45] Other contraindications include patients in whom prehospital or in-hospital resuscitation was initiated greater than 15 to 30 minutes after collapse, patients with > 60 minutes from the time of arrest to the ROSC, patients who are persistently hypoxic (e.g., SpO_2 < 85%) for greater than 15 minutes after the ROSC, patients with an arrest that is related to blunt or penetrating trauma, patients in whom greater than 6 hours have elapsed since the ROSC, and patients who spontaneously awaken with a normal mental status. Do not induce hypothermia during cardiopulmonary resuscitation as this decreases ROSC.[46,47] Wait until the patient has ROSC.

EQUIPMENT

GENERAL SUPPLIES

- Arterial line equipment and supplies (Chapter 72)
- Nasogastric tube equipment and supplies (Chapter 75)
- Cardiac monitor
- Ventilator
- Orotracheal intubation equipment and supplies (Chapter 18)
- Venous access equipment and supplies (Chapters 59 and 61)
- Analgesics (e.g., fentanyl, morphine, or meperidine)
- Sedatives (e.g., propofol, Ativan, or Versed)
- Neuromuscular blockers (e.g., pancuronium, vecuronium, or rocuronium)
- Defibrillator-cardioverter unit and supplies (Chapter 40)

COOLING SUPPLIES

- 4°C sterile normal saline or lactated Ringer's
- Ice packs or ice water-soaked blankets
- Core temperature probe and monitor (e.g., esophageal, rectal, or urinary)

EXTERNAL COMMERCIAL COOLING DEVICES (FIGURES 225-2 AND 225-3)

- Excel Cerebral Cooling System (Cryothermic Systems Inc., Broadview Heights, OH)
- Gaymar Medi-Therm III (Stryker, Orchard Park, NY)
- Arctic Sun Temperature Management System (Medivance Inc., Louisville, CO)
- Cincinnati Sub-Zero Blankets (Cincinnati Sub-Zero Products Inc., Cincinnati, OH)
- InnerCool STx Surface Pad Systems (Zoll Medical Corp., Chelmsford, MA)
- Thermosuit (Life Recovery Systems, Waldwick, NJ)
- CoolGuard 3000 (Zoll Medical Corp., Chelmsford, MA)
- MediTherm Cooling Blankets (Stryker, Orchard Park, NY)
- Altrix Cooling Unit (Stryker, Orchard Park, NY)
- EMCOOLS FlexPad Medical Cooling System (EMCOOLS, Austria)
- Mistral Cooling Unit (37 Degree Co., Netherlands)

INTERNAL ENDOVASCULAR COOLING DEVICES (FIGURE 225-4)

- Accutrol Catheter (Zoll Medical Corp., Chelmsford, MA)
- Cool Line, Icy, & Quattro catheters (Stryker, Orchard Park, NY)

There is a nasal cooling system called Rhinochill (BeneChill, San Diego, CA) (**Figure 225-5**). These systems take advantage of the large surface area of the nasal cavity with an abundant capillary supply just below the mucosa as a heat exchanger. These systems may be used prehospital as well as in-hospital for the cooling of the post–cardiac arrest patient. This system is used in Europe and not currently available in the United States.

PATIENT PREPARATION

Carefully consider inclusion and exclusion criteria for each individual patient utilizing preestablished institution-specific protocols. The risks, benefits, potential complications, and aftercare of the procedure should be explained to the patient's representative and an informed consent obtained whenever possible. The postarrest patient will or should be intubated, have continuous pulse oximetry and cardiac monitors, and have frequent automated cuff or continuous arterial line blood pressure monitoring.

Perform a detailed examination to document a postarrest comatose state and the patient's baseline neurologic status (e.g., Glasgow coma scale, pupillary light reflex, corneal reflex, facial movements, eye movements, gag, cough, and motor response to painful stimuli). The retention of any neurologic function during or after cardiopulmonary resuscitation suggests a good prognosis. **The absence of neurologic function immediately after the ROSC is not a reliable predictor of a poor outcome.** The reliability of the neurologic exam to predict a poor outcome is time dependent. The absence of pupillary light reflexes, corneal reflexes, or motor responses to painful stimuli at day 3 provides the most reliable predictor of poor outcome.[3]

Prepare the patient for therapeutic hypothermia. Turn the ventilator's warm humidified air function off so as not to warm the patient. Perform an electrocardiogram (ECG). Obtain basic laboratory analyses to include electrolytes, renal function, calcium, magnesium,

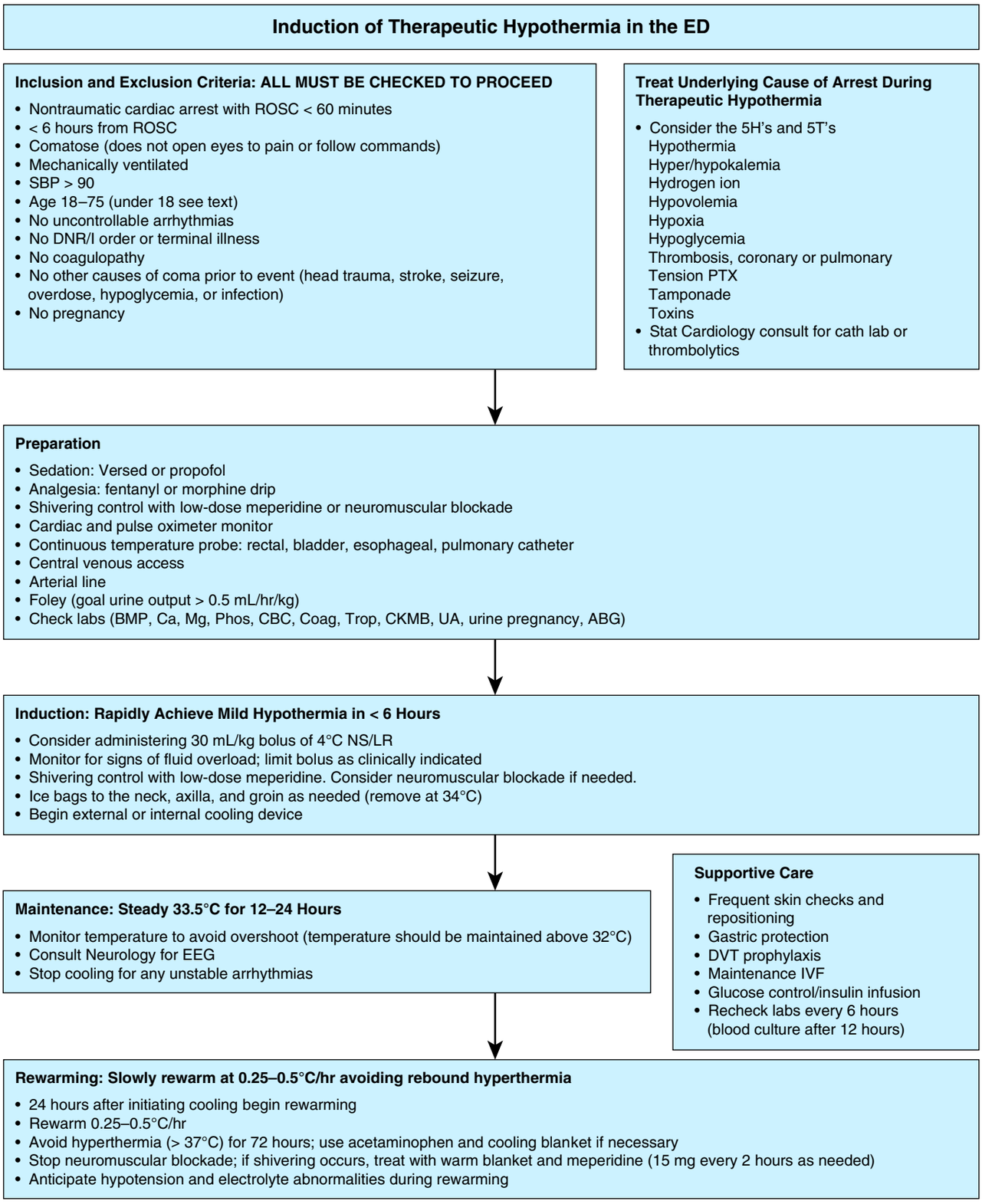

FIGURE 225-1. A sample induction of therapeutic hypothermia checklist and clinical pathway. ABG, arterial blood gas; BMP, basic metabolic panel; CBC, complete blood count; DVT, deep vein thrombosis; ED, Emergency Department; IVF, intravenous fluid; LR, lactated Ringer's; NS, normal saline; PTX, pneumothorax; SBP, systolic blood pressure; UA, urinalysis.

phosphorus, complete blood count, coagulation profile, cardiac markers, urinalysis, pregnancy, and arterial blood gas. Other laboratory analyses may include cortisol levels and thyroid function tests at the discretion of the admitting team. Titrate intravenous sedation and analgesia for comfort with mechanical ventilation. Shivering increases oxygen consumption, requiring control with low-dose meperidine (e.g., 15 mg IV every 2 hours) and possibly neuromuscular blockade. Place a urethral catheter (Chapter 173) and monitor the urine output

A

C

B

D

E

FIGURE 225-2. Examples of externally applied cooling devices. ***A.*** The Excel Cerebral Cooling System. (Photo courtesy of Cryothermic Systems Inc., Broadview Heights, OH.) ***B.*** The Arctic Sun Temperature Management System. (Photo courtesy of Medivance Inc., Louisville, CO.) ***C.*** The Cincinnati Kool-Kit. (Photo courtesy of Cincinnati Sub-zero Products Inc., Cincinnati, OH.) ***D.*** The Thermosuit. (Photo courtesy of Life Recovery Systems, Waldwick, NJ.) ***E.*** The Medi-Therm. (Photo courtesy of Stryker Medical, Orchard Park, NY.) ***F.*** The CoolGard 3000. (Photo courtesy of Zoll Medical Corp., Chelmsford, MA.) ***G.*** The Arctic Sun 5000. (Photo courtesy of Medivance Inc., Louisville, CO.) ***H.*** The Altrix. (Photo courtesy of Stryker, Orchard Park, NY.) ***I.*** The MediTherm Blankets (Photo courtesy of Stryker, Orchard Park, NY.) ***J.*** The Mistral Cooling Unit. (Photo courtesy of 37 Degree Co., The Netherlands.)

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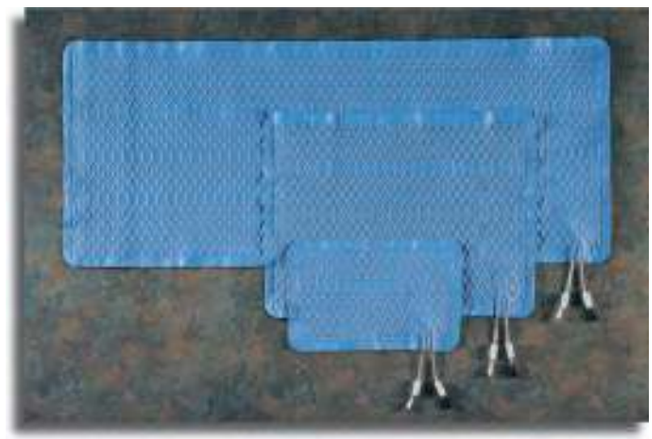

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FIGURE 225-2. (*Continued*)

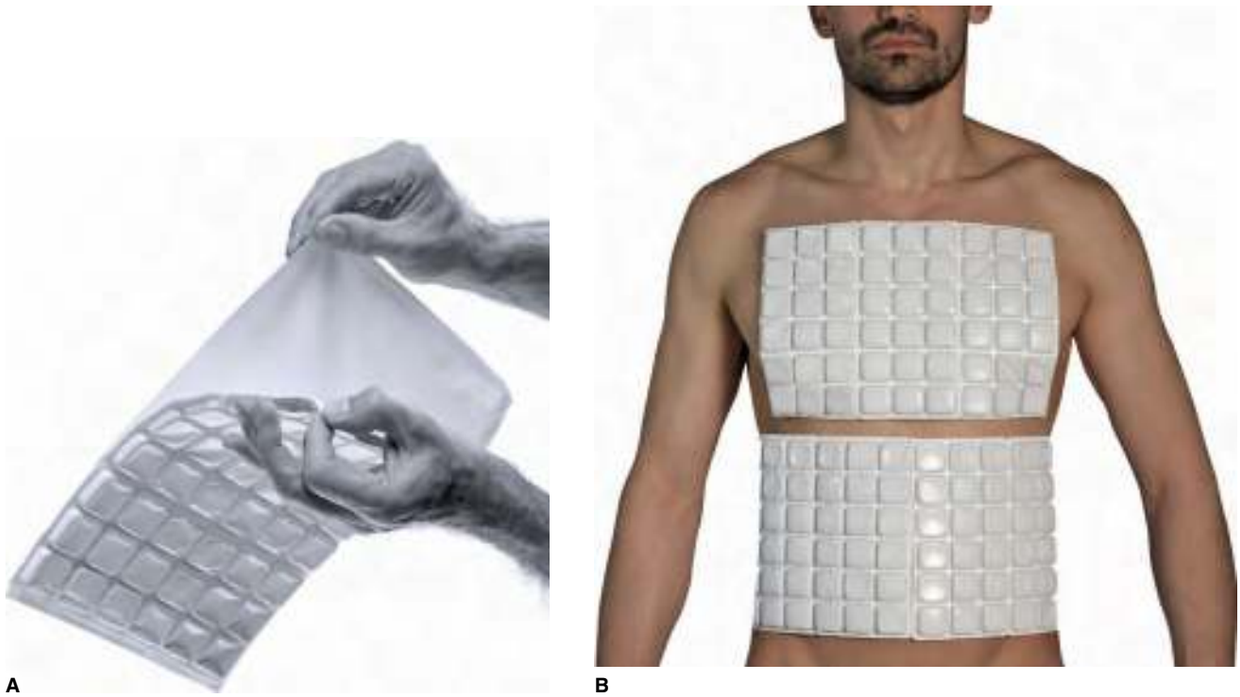

FIGURE 225-3. The EMCOOLS FlexPad Medical Cooling System. ***A.*** The pad being removed from its protective backing. ***B.*** The pads on a patient. (Photos courtesy of EMCOOLS, Austria.)

with a goal of > 0.5 mL/hr/kg. Continuously monitor the core temperature with an esophageal, rectal, or urinary temperature probe. Place a nasogastric tube (Chapter 75). Apply defibrillator/pacing pads (Chapter 40) in case they are required during the therapeutic hypothermia process. Standard acute coronary syndrome and postresuscitation protocols otherwise apply.

TECHNIQUE

Therapeutic hypothermia is performed in three phases: induction, maintenance, and rewarming. There are multiple methods to perform all three phases. Detailed protocols from several institutions are available through the University of Pennsylvania's Center for Resuscitation Science at www.med.upenn.edu/resuscitation/hypothermia/protocols.shtml. This site lists over 50 protocols for therapeutic hypothermia from various hospitals. **The goals of each phase always remain the same. Rapidly achieve a hypothermic state of 32°C to 36°C (i.e., 89.6°F to 96.8°F), maintain hypothermia for at least 12 to 24 hours, and then slowly rewarm at no faster than 0.25°C to 0.50°C (i.e., 0.45°F to 0.9°F) per hour.**

INDUCTION

Beginning the induction phase of therapeutic hypothermia in the Emergency Department is straightforward and inexpensive. The typical temperature decrease seen in survivors of cardiac arrest begins the process even before it starts. The infusion of 4°C Hartmann's solution, lactated Ringer's, or normal saline stored

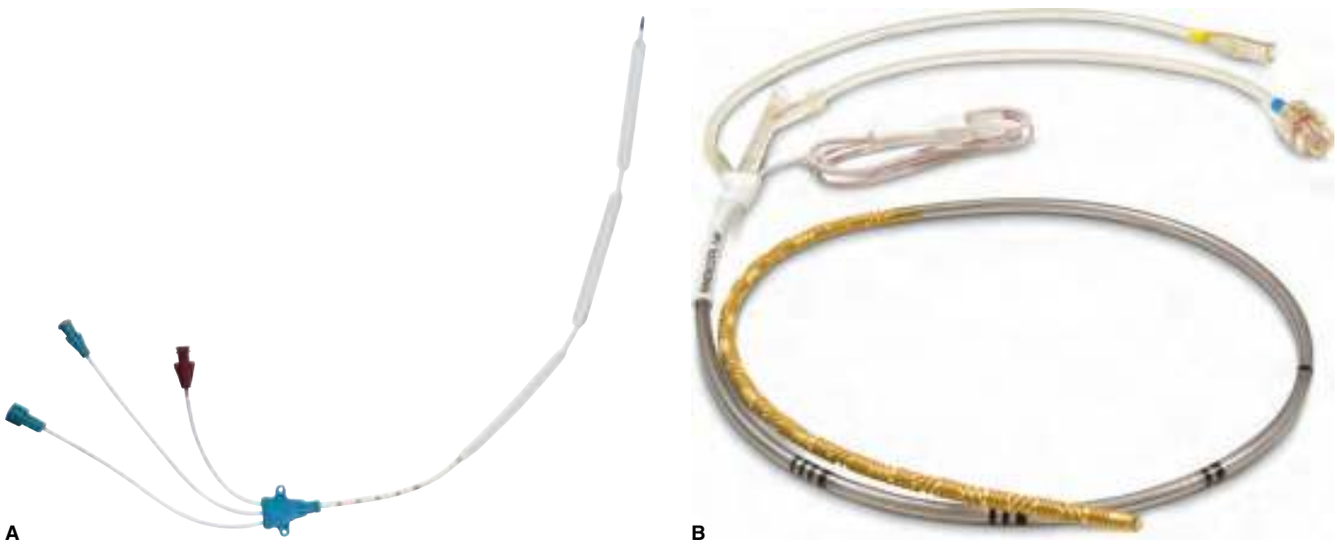

FIGURE 225-4. Examples of internally applied cooling devices. ***A.*** The Icy Catheter. ***B.*** The Accutrol Catheter. (Photos courtesy of Zoll Medical Corp., Chelmsford, MA.)

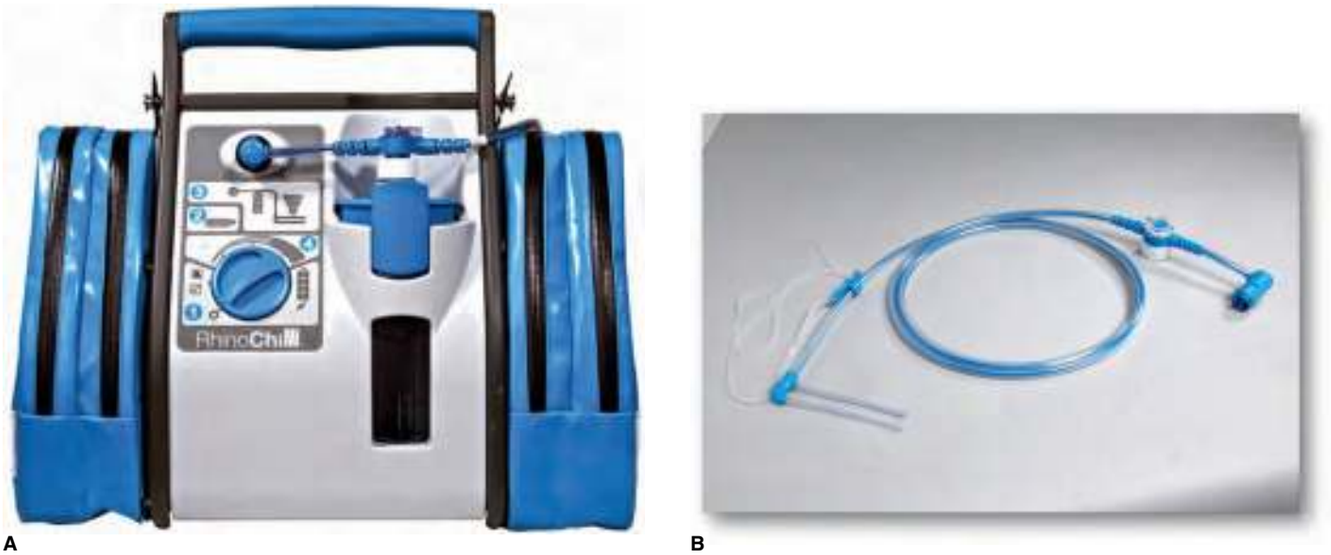

FIGURE 225-5. The RhinoChill. ***A.*** The unit. ***B.*** The disposable nasal prongs that attach to the base unit. (Photos courtesy of BeneChill, San Diego, CA.)

in a temperature-controlled refrigerator provides a reliable and safe method for rapid induction.[47-51] Bolus 30 mL/kg or 1.5 to 2 L of fluid to create a 1.5°C to 2.3°C (2.7°F to 4.1°F) decrease in temperature.[52-54] **Monitor the patient for signs of fluid overload and adjust the rate and total volume accordingly.** Smaller repeat boluses of 500 mL may be given when required.[42] The application of ice packs to the head, neck, torso, and groin provides another safe and reliable method for rapidly decreasing temperature.[55] Patients requiring immediate angiography or admission to other locations may be transferred with these methods in place and without delay.[12,15]

Several commercially available external and internal cooling devices listed in the equipment section allow the Emergency Physician to set and maintain a target temperature through continuous feedback from the patient's core temperature monitor. External systems utilize cooled circulated air, pads, or blankets. Internal systems require the placement of a femoral or subclavian central venous catheter with an endovascular cooling device. Initiate these devices while the fluid boluses and/or ice packs are applied.

To date there have been no studies comparing external versus internal cooling devices in cardiac arrest patients.[55] A manufacturer-sponsored study of therapeutic hypothermia in neurosurgical patients demonstrated that the endovascular device reached target temperature much faster (i.e., 35 versus 204 minutes), maintained a tighter control of target temperature, and had no significant increase in complications versus surface cooling.[56]

Beginning the induction in the Emergency Department with cooled IV fluids and ice packs, followed by the immediate transfer of the patient to the Intensive Care Unit for the application of more expensive and delicate commercial cooling devices for maintenance of therapeutic hypothermia, provides a clinically effective and cost-effective approach in any institution. **Apply a commercial cooling device and continue the process of therapeutic hypothermia in the Emergency Department if there is any delay in transferring the patient to the Intensive Care Unit.**

MAINTENANCE

Stop cooling at 33.5°C (i.e., 92.3°F) to prevent overshoot.[42] Use an external or internal cooling system with feedback control to maintain a core temperature of 32°C to 36°C (i.e., 89.6°F to 96.8°F).[57] Internal cooling devices have less variability.[57,58] Other methods include the use of cold wet blankets and ice packs placed around the head, neck, torso, and extremities. Although less expensive, these methods are more labor intensive, result in greater temperature fluctuations, and do not allow for controlled rewarming.[59] Maintaining hypothermia with fluid boluses alone is not possible.[59]

Supportive therapy continues in the Intensive Care Unit (e.g., continuous cardiac monitoring, consideration of arterial monitoring, analgesics, sedatives, and neuromuscular blockade). Maintain a goal mean arterial pressure of 65 to 100 mmHg with a central venous pressure of 8 to 12 mmHg. Monitor urine output and laboratory values to best manage the resulting cold diuresis. Maintain tight glucose control (e.g., 108 to 144 mg/dL) to improve survival and neurologic outcomes.[3] Decreased fever with therapeutic hypothermia increases the survival.[60]

REWARMING

Rewarming typically begins 24 hours after the initiation of cooling. Gradual rewarming at a rate of 0.25°C to 0.5°C (i.e., 0.45°F to 0.9°F) an hour over a 6 to 8 hour period helps prevent rapid changes in metabolic rate, electrolyte levels, and hemodynamics.[61] Techniques include passive rewarming, resetting the temperature on commercial devices, and the application of heated-air blankets.

Discontinue analgesics, sedatives, and neuromuscular blockade during the rewarming phase. In landmark studies, Bernard stopped the neuromuscular blockade before 24 hours while Holzer maintained blockade for 32 hours.[6,7] Other published protocols call for medications to be held when the core temperature reaches 35.5°C to 36.5°C (i.e., 95.9°F to 97.7°F). Treat shivering during the rewarming with a warm blanket and low-dose meperidine.[62] Care must be taken to avoid hyperthermia/pyrexia (i.e., > 37.5°C or 99.5°F) by treating with antipyretics and active cooling for 72 hours postarrest. Hypotension and hyperkalemia commonly occur during rewarming.[3] This requires close monitoring of the patient, the ECG tracings, and periodic laboratory analysis.[3]

TECHNIQUE FOR PEDIATRIC PATIENTS

Patients under the age of 18 were excluded from the landmark studies. The 2010 ILCOR guideline recommends that the induction of hypothermia should be considered for 12 to 24 hours in children

who remain comatose after resuscitation from a cardiac arrest.[63] There are now pediatric studies and reviews that have shown that there is mixed benefit to induced hypothermia.[31,33,64-67] Neonates with perinatal asphyxia have been treated successfully with external cooling elements for 72 hours with impressive results.[68] Consult a Pediatric Intensivist and Pediatric Cardiologist prior to inducing hypothermia in a patient under the age of 18 years. The adverse events are different than in adults.[64]

AFTERCARE

Consult an Intensivist and Cardiologist. Admit the patient to the appropriate Intensive Care Unit. Therapeutic hypothermia requires Intensive Care Unit admission for ventilator management as well as the continuous monitoring of core temperature, cardiac rhythm, blood pressure, and central venous pressure. Sedatives, paralytics, and orotracheal intubation are often required for patient comfort and shivering control.[69-71] Obtain laboratory analyses every 4 hours or as clinically indicated. Further bedside cardiac testing or cardiac catheterization laboratory intervention may be performed by a Cardiologist to help determine and treat the precipitating coronary artery disease. Consult a Neurologist for an electroencephalogram (EEG) and other neurologic testing to detect seizures and determine patient prognosis.[72]

Begin standard Intensive Care Unit and post–cardiac arrest/acute coronary syndrome care in all patients after completion of therapeutic hypothermia. Consider the use of deep venous thrombosis prophylaxis with sequential compression devices, subcutaneous heparin, or subcutaneous low-molecular-weight heparin. Apply Lacrilube to the patient's eyes every 4 to 6 hours to prevent a corneal abrasion or corneal ulceration from dry corneas.[69] Consider the use of stress ulcer prophylaxis. Once the 24 hour therapeutic hypothermia period is completed, begin to rewarm the patient and wean any neuromuscular blockade and sedation. Extubation is determined and evaluated on a case-by-case basis.

The patient may not become responsive until 48 hours or later after rewarming.[73] Do not make early prognostic judgements regarding the patient.[74-77] These are often wrong. Defer to the Intensive Care Unit teams all judgements regarding the patient outcome.

COMPLICATIONS

Emergency Physicians must be aware of several complications despite the proven safety and benefit of therapeutic hypothermia. Shivering during the induction phase increases core temperature and oxygen consumption. Low-dose buspirone, meperidine, opioids, and 4 gm of magnesium have been reported to lower the shivering threshold in awake hypothermic patients.[62,78] Magnesium sulfate has the added benefits of improving cooling rates through vasodilatation while acting as an antiarrhythmic.[79] The use of orotracheal intubation and paralytics may be required to stop the shivering.

Pulmonary edema may occur with cooled IV fluid boluses. Arrhythmias may occur, especially with the use of endovascular cooling techniques. Other complications of bradycardia, decreased cardiac output, and increased systemic resistance are of unknown clinical significance. Hypothermic patients may be more susceptible to bacteremia and infection (e.g., pneumonia and sepsis).[80] The landmark studies showed only an insignificant trend toward increased sepsis. Post–cardiac arrest patients are at high risk of developing pneumonia in the first 48 hours, with aspiration being a key risk factor.[3]

A coagulopathy may occur due to changes in platelet function, clotting factor enzyme function, and fibrinolytic activity.[81,82] Thrombolytics, heparin, and aspirin may be safely administered during induction when clinically indicated.[16,83] A cold diuresis may lead to electrolyte abnormalities including hypophosphatemia, hypomagnesemia, hypocalcemia, hypokalemia, or hyperkalemia. Hyperglycemia must be avoided to improve neurologic outcomes after cardiac arrest and in all critically ill patients. Increases in amylase and a transient rise in renal markers have been reported but are of unclear significance. Decreased core temperature often prolongs the duration of action of neuromuscular blockers (e.g., vecuronium) and sedatives (e.g., midazolam or propofol) by impairing metabolism.[84-86] Rhabdomyolysis and subsequent renal failure are preventable by monitoring urine output and hydration.[87]

The cooling devices themselves may be a source of complications. The endovascular catheter of internal cooling devices increases risk of infections and venous thrombosis similar to any central venous catheter (Chapter 63).[56] Right atrial thrombus has been reported with the use of internal cooling catheters.[88] External cooling devices may cause skin breakdown and tissue necrosis.[55]

FUTURE CONSIDERATIONS

The use of esophageal cooling may be used in the future to induce and maintain therapeutic hypothermia.[89-91] The Esophageal Cooling Device (Advance Cooling Therapy, Chicago, IL) has been approved for use in the United States (**Figure 225-6**). It has been used for several years in Australia, Canada, and Europe. A single-use catheter attaches to an external cooling unit. The catheter is inserted into the esophagus like a nasogastric tube. It contains three lumens. Two of the lumens are for the circulation of cooling fluid. The third lumen allows gastric decompression and drainage.

SUMMARY

The staggering number of sudden cardiac deaths and the poor neurologic outcomes of survivors place an enormous burden on the health-care system. Two well-done landmark studies in 2002 clearly demonstrated improved survival and neurologic outcomes in comatose survivors of an out-of-hospital cardiac arrest with an initial rhythm of pulseless ventricular tachycardia or ventricular fibrillation treated with mild therapeutic hypothermia of 32°C to 34°C (i.e., 89.6°F to 93.2°F) for 12 to 24 hours. These results were incorporated into the recommendations from ILCOR and the AHA. These organizations recommend therapeutic hypothermia for comatose survivors of an out-of-hospital cardiac arrest with an initial rhythm of ventricular fibrillation or pulseless ventricular tachycardia. They also recommend no IV cold saline boluses and to cool to a targeted temperature between 32°C and 36°C (i.e., 89.6°F to 96.8°F) for

FIGURE 225-6. The Esophageal Cooling Device. (Photo courtesy of Advanced Cooling Therapy, Chicago, IL.)

at least 24 hours. No definite proven benefit has been shown for the use of target temperature management for the pediatric population. The induction of therapeutic hypothermia is a straightforward, inexpensive, safe, and effective procedure in any Emergency Department.

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226 Hypothermic Patient Management

Jessen Schiebout

INTRODUCTION

The Centers for Disease Control and Prevention (CDC) reported 13,419 deaths from hypothermia in the United States between 2003 and 2013.[1] Approximately 67% of deaths were in males. Many think these figures underestimate the number of cases because standard thermometers do not measure low temperatures.[1,2] Hypothermia is defined as a core body temperature below 35°C.[2] The normal physiologic thermoregulatory responses fail below this level and lead to an inability to generate enough heat to maintain bodily functions.[3,4] Accidental hypothermia is further classified as primary or secondary.[5] Primary accidental hypothermia occurs when a healthy individual's heat production is overcome by environmental factors.[6-9] Secondary

accidental hypothermia occurs when predisposing factors lead to disruption of temperature homeostasis and increase the individual's susceptibility to lesser environmental stresses (e.g., drug intoxication, endocrine disorders, and trauma).[3] Traumatic injury is a common example of an acquired condition associated with hypothermia.[5]

There are multiple reasons why trauma patients are at an increased risk for hypothermia.[10] These include extended prehospital time, resuscitation with room temperature intravenous fluids, exposure to environmental factors, and physiologic characteristics of the trauma. Bleeding and hypoperfusion alter thermoregulation. Hypothermia is an independent risk factor for increased morbidity and mortality in trauma patients because of its association with coagulopathy and multiple organ failure.[11]

Significant emphasis has been placed on the prevention of hypothermia as well as early recognition and correction of hypothermia in the prehospital setting. **Hypothermia can progress after the patient arrives in the Emergency Department.**[12] Many studies have demonstrated this phenomenon in traumatically injured patients.[13-15] This recognition has prompted development of multidisciplinary approaches to maintaining normothermia as the patient moves through the hospital.[16] **The importance of continuity and communication in dealing with hypothermia cannot be overstated.**

There are many other groups at risk for hypothermia. Infants lack a shivering response and exhibit a larger surface-to-volume ratio leading to rapid heat loss.[17,18] Hypothermia is seen more frequently in southern states and during colder months.[19] Malnourished or very thin individuals contain less insulating adipose tissue and less energy for metabolic heat generation.[18,20-22] Endocrine disorders (e.g., hypothyroidism, hypoglycemia, diabetes, and adrenal insufficiency) lower metabolic heat production.[17,20] Underlying infections, poverty, dementia, and conditions that impair peripheral vasoconstriction (e.g., burns and psoriasis) predispose to hypothermia. Drugs of abuse lead to diminished awareness of cold surroundings while others (e.g., opiates, clonidine, and tramadol) directly block the shivering mechanism.[20,22,23]

Alcohol has been cited as the most common factor associated with hypothermia.[18] Alcohol decreases shivering, causes hypoglycemia, and inhibits metabolic heat production.[24] Alcohol affects the hypothalamus. It changes the thermoregulatory set point and cold perception.[18,25,26] Peripheral vasodilation increases heat loss and perceived comfort at cold temperatures and diminishes the drive to find warmer environments or clothing.[24,26]

Hypothermia is common in the elderly population. They exhibit limited peripheral vasoconstriction and mobility.[27] Humans lose muscle mass during aging and this leads to decreased heat production from shivering. The elderly have impaired thermal sensation from frequently taken medications (e.g., beta-blockers or clonidine) that increase cold susceptibility.[17-21,28,29]

Increased age and many other factors are associated with worse outcomes.[30] Serum lactate levels greater than 12.5 mmol/L or potassium levels greater than 10 to 12 mmol/L are considered terminal. Asystole as a presenting rhythm, drowning mechanism, and traumatic injury are all associated with worse outcomes.[30-32] Patient's with prolonged hypothermia, secondary hypothermia, lower core temperatures, severe acidosis, concomitant infections, rhabdomyolysis, alcohol use, and increased comorbidities have a poorer prognosis.[27,28,33,34]

ANATOMY AND PATHOPHYSIOLOGY

Heat is lost by conduction, convection, evaporation, and radiation. Conduction is the loss of heat energy through direct contact with a cooler entity (e.g., air, snow, water). The rate of heat loss is proportional to the temperature gradient and size of the contact area. Convection is heat loss when liquids or gases flow over the body surface (e.g., Bair Hugger or wind). **Wet clothing can increase heat loss by convection and conduction up to 5 times and up to 25 times if a person is submerged in water.**[35] As wind speeds increase, heat loss by convection increases. Evaporation is heat loss through conversion of water to vapor (e.g., sweating). The rate of fluid evaporation and heat loss increases as humidity falls. Radiation is heat transfer from warmer to cooler objects via infrared rays and is proportional to the difference between body temperature and surrounding temperature. The hypothalamus detects falling blood temperature or cold signals from skin thermoreceptors, increases sympathetic drive, causes peripheral vasoconstriction, and causes shivering. The net effect is an increase in metabolic heat production to offset heat loss.[36]

The Swiss Staging Model for hypothermia was invented to classify hypothermia based on clinical presentation and to predict core body temperature.[37] Shivering but lucid patients are classified as stage 1 and are predicted to be between 32°C and 35°C. Confused patients who are not shivering are predicted to be between 28°C and 32°C and meet stage 2 criteria. Unconscious patients with vital signs are predicted to be between 24°C and 28°C and are considered to be in stage 3. Stage 4 patients are predicted to be lower than 24°C and have no vital signs. This staging system is useful to classify patients but is not accurate in core temperature prediction.[37]

Hypothermia is more commonly characterized as mild, moderate, and severe (**Table 226-1**).[38] Mild hypothermia is defined as a core temperature of 32°C to 35°C. Moderate hypothermia is defined as a core temperature of 28°C to 32°C. Severe hypothermia is defined as a core temperature of less than 28°C. The scale can be amended in the trauma patient to 34°C to 36°C for mild hypothermia, 32°C to 34°C for moderate hypothermia, and less than 32°C for severe hypothermia.

Mild hypothermia is associated with shivering, elevated heart rates, and elevated respiratory rates. The body increases heat production by increasing the shivering and increasing the metabolic rate. Peripheral vasoconstriction may result in acrocyanosis. Neurologic symptoms may include confusion, dysarthria, and impaired judgment.

Moderate hypothermia causes lethargy, hallucinations, loss of coordination, and diminished or dilated pupillary reflex. The patient stops complaining of "feeling cold" and the shiver mechanism is lost. Further temperature drop leads to peripheral autonomic nerve dysfunction and causes peripheral vasodilation. This creates a warm sensation and may explain the "paradoxical undressing" that is often seen.[20] Hyperreflexia is initially present. The muscles become stiff and tendon reflexes are lost as temperatures fall below 32°C.[35,39] With every 1°C drop in core temperature, cerebral blood flow diminishes 6% to 7%.[35,40,41] Cerebrovascular autoregulation is lost at 25°C.[35,40,41] Brain wave changes are notable on an electroencephalogram (EEG) by 34°C and correlate with a cold narcosis. A cardiac standstill is seen at 29°C.[22,35,42] Severe hypothermia is often associated with absent pupillary reflexes and coma.

Falling temperatures correlate with worsening bradycardia and decreased cardiac contractility.[42] Reduced cardiac output may generate adequate blood flow due to deceased metabolic demands of the body.[31] The heart rate typically falls by 30 to 40 bpm at 28°C. Lower heart rates may suggest other comorbidities (e.g., hypoglycemia or endocrine disorders).[40] The cardiac rhythm commonly converts from normal sinus to atrial fibrillation.[43]

The patient is usually comatose by the time severe hypothermia is present, often with cardiopulmonary collapse. Ventricular irritability becomes evident and slight disturbances (e.g., bed transfers, orotracheal intubation, and nasogastric tube insertion) may lead to ventricular fibrillation. Intubation is frequently mentioned as a possible

TABLE 226-1 The Effects of Mild, Moderate, and Severe Hypothermia on Various Physiologic Systems

	Mild hypothermia (> 32°C)	Moderate hypothermia (28–32°C)	Severe hypothermia (< 28°C)
Cardiovascular system	Hypertension Sinus tachycardia	Atrial fibrillation Bradycardia Decreased P- and T-wave amplitude Hypotension Increased electrocardiogram intervals Osborne J waves	Asystole Heart blocks Pulseless electrical activity Severe bradycardia Ventricular fibrillation
Fluid and electrolytes	Cold diuresis	Hyperglycemia Hyperkalemia Lactic acidosis	Acute renal failure Hyperglycemia Hyperkalemia Lactic acidosis
Gastrointestinal (GI) system		GI bleeding Hepatic dysfunction Ileus Pancreatitis	GI bleeding Hepatic dysfunction Ileus Pancreatitis
Hematologic system		Decreased coagulation cascade Decreased platelets Hemoconcentration	Decreased coagulation cascade Decreased platelets Disseminated intravascular coagulation
Neurologic system	Ataxia Disorientation Dysarthria Hyperreflexia	Agitation Dilated pupils Hallucinations Hyporeflexia Stupor	Coma Nonreactive pupils
Respiratory system	Tachypnea	Aspiration Bradypnea Loss of cough reflex Shallow respirations	Apnea Acute respiratory distress syndrome Severe bradypnea
Thermoregulatory and musculoskeletal systems	Hypertonia Shivering	Loss of shivering Muscle rigidity	Pseudo-rigor mortis Rhabdomyolysis
Rewarming method	Passive Active external	Active external Noninvasive internal	Active external Invasive internal Noninvasive

inciting event for ventricular fibrillation. **The actual incidence is rare and intubation should not be withheld if clinically indicated.**[40,44] Myocardial irritability results from associated hypoxia, acid-base disturbances, and increased sympathetic stimulation.[31] Temperature gradients within the myocardium cause heterogeneous repolarization and fibrillation.[45] Ventricular fibrillation becomes refractory to conversion below 28°C. Asystole occurs at 20°C.

The characteristic electrocardiographic (ECG) finding of an Osborne J wave occurs at approximately 32°C (**Figure 226-1**). This is a positive deflection between the QRS and the ST segment typically seen in lateral or inferior chest leads. The Osborne wave is often only seen in one ECG lead.[46] The Osborne wave is produced by repolarization abnormalities correlating with temperature and pH.[40,47,48] **The Osborne waves are not pathognomonic for hypothermia and can be seen in other conditions (e.g., brain injury, hypercalcemia, tricyclic overdoses, Brugada syndrome, and cardiac ischemia).**[46,49] The PR, QT, and QRS intervals on the ECG lengthen as core temperature falls. The P wave amplitude decreases, heart blocks develop, and medications (e.g., atropine) become ineffective.[40,46,48,49]

Oxygen consumption (VO_2) increases dramatically in mild hypothermia due to shivering. A core temperature decrease of 0.3°C is associated with a 7% increase in VO_2. Temperature reductions between 0.3°C and 1.2°C have been reported to result in a 92% increase in VO_2 with proportional increases in minute ventilation.[50] The resultant increase in oxygen utilization may result in anaerobic metabolism, acidosis, and significant cardiopulmonary stress.[51] However, temperatures below 33°C cause decreased VO_2, tidal volume, and respiratory rates. Oxygen consumption and carbon dioxide production fall by 50% at 30°C. Every additional 1°C fall in temperature causes a 6% drop in oxygen consumption.[40,41] The ciliary action and the cough reflex are depressed below 33°C predisposing to atelectasis and aspiration.[33,35,41] Moderate hypothermia leads to increased bronchial secretions and rewarming may cause pulmonary edema.[22,33,41,44,52]

Metabolic problems associated with hypothermia include both hyperglycemia and hypoglycemia. Hyperglycemia results from elevated sympathetic drive and cold-induced pancreatic inhibition. Hypoglycemia is frequently seen and thought to be a predisposing factor instead of a cause for hypothermia.[20,33] Hypothermia initially shunts blood from the periphery causing an elevation in blood pressure, an elevation of renal blood flow, and a cold diuresis.[53] Antidiuretic hormone release and distal tubule water reabsorption are directly affected as core temperature falls.[41,43] This often results in blood urea nitrogen elevation, creatinine elevation, and electrolyte shifts.[18] Further kidney injury may result from hypothermia-induced rhabdomyolysis.[54]

Serum potassium is often slightly increased because of renal tubular dysfunction, acidosis, and the breakdown of liver glycogen. Hyperkalemia results from tissue necrosis at more advanced stages. It is recommended that uncorrected values be used to guide therapy due to the complexities of the acid-base balance associated with hypothermia when interpreting blood test results. These derangements are reversible with rewarming.

Multiple gastrointestinal organs are directly affected by hypothermia. Liver metabolism and the P450 system slow as temperature falls.[55] Gastrointestinal motility slows at temperatures below 34°C and an ileus may develop below 28°C. Gastrointestinal bleeding occurs from ulcer formation (i.e., Wischnevsky ulcers) and

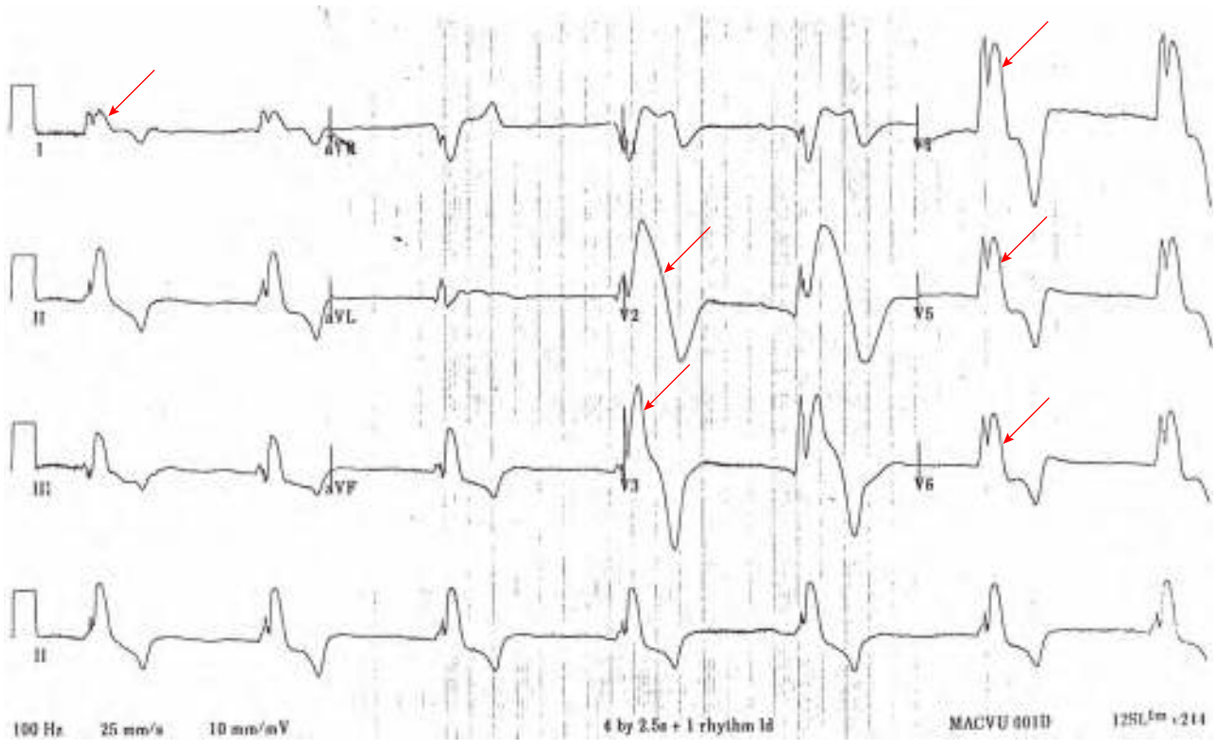


FIGURE 226-1. The Osborne J wave associated with hypothermia is indicated by the arrows. This patient had a core temperature of 34°C.

pancreatitis.[20,22,27,33,40,41] Gastrointestinal absorption is inhibited. Avoid medication administration orally or via nasogastric tube.

Coagulopathy is one of the most clinically significant metabolic derangements seen with hypothermia. Increased clotting times occur at core temperatures less than 35°C. Temperatures less than 32°C lead to severe platelet dysfunction.[5,13] Cold platelets undergo morphologic changes affecting adherence and motility. Worsening hypothermia may lead to reversible platelet sequestration. Prostacyclin may become inhibited and promotes inappropriate platelet activation, disseminated intravascular coagulation (DIC), and thrombosis.[35,40]

Lowering core temperature to 34°C decreases clotting factor activity up to 40% due to the direct effect on physiochemical activity.[35,56] Cold-induced slowing of the enzymatic reaction rate of thromboxane synthetase results in decreased production of thromboxane A2, a potent vasoconstrictor necessary for normal platelet aggregation.[57,58] These derangements are reversible with rewarming. It is important to remember that clinical tests of coagulation are temperature standardized to 37°C. Fibrometers contain a thermal block that heats the plasma and reagents to 37°C before initiating the assay. **The tests of coagulation reflect clotting factor deficiencies and are corrected for any potential effect of hypothermia on clotting factor function.**

Dehydration and increased vascular permeability often result in hemoconcentration. The hematocrit increases by 2% for every 1°C drop in temperature. Low hematocrit may suggest an underlying anemia and/or blood loss. Leukopenia may be seen due to splenic or hepatic sequestration.[18,20,40]

INDICATIONS

Patients presenting with hypothermia must be identified and treated (**Table 226-2**). **Hypothermia should be easily identified since a temperature is obtained in all patients presenting to the Emergency Department.** Patients may develop hypothermia while in the Emergency Department. **Cover all patients with warm sheets or blankets as soon as their initial evaluation and any necessary procedures are completed.**

CONTRAINDICATIONS

There are few contraindications for treating hypothermia. **Serum potassium greater than 10 to 12 mmol/L and lactates greater than 12.5 mmol/L suggest futile resuscitative efforts.**[30-33,53,59-62] Those

TABLE 226-2 Rewarming Techniques

Passive External Rewarming
Remove wet clothing
Dry clothing and blankets
Active External Rewarming
Circulated fluid warmers
Forced air warmers
Heat lamps
Hot water immersion
Warm blankets
Active Core Rewarming
Arteriovenous rewarming
Bladder lavage
Cardiac bypass
Gastric lavage
Heated humidified oxygen
Heated intravenous fluids
Hemodialysis
Open chest (thoracotomy) lavage
Peritoneal lavage
Pleural lavage
Venovenous rewarming

with do not resuscitate (DNR) orders should not receive cardiopulmonary resuscitation (CPR) if they arrest. Many suggest withholding transport and care if rigor mortis is present; however, severe hypothermia causes muscle stiffness making this unreliable. Good neurologic outcomes have been demonstrated in prolonged extraction and intermittent CPR for multiple hours.

Warming faster usually produces better outcomes.[61] Hypothermic brain injury patients warmed at slower rates did not show benefit and hypothermic patients with cardiac arrest do not benefit from additional therapeutic hypothermia.[62] Patients have worse neurologic recovery if temperature correction overshoots.[63] Stop active rewarming at 33°C to 35°C.[20,62] There are circumstances where induced therapeutic hypothermia may be of benefit (Chapter 225).[64-67]

SPECIAL RESUSCITATION CONSIDERATIONS

All patients with hypothermia are identified by measurement of core body temperature on admission. Close subsequent monitoring is necessary and required. Poor peripheral perfusion may result in difficulty assessing the pulse and blood pressure. This makes the use of Doppler ultrasound (US) necessary. Cutaneous probes for temperature and pulse oximetry have difficulty picking up signals in hypothermic patients. Oral and auricular temperature probes are often inaccurate. Consider using bladder, esophageal, or rectal temperature probes. Esophageal temperature probes will be inaccurate if using heated oxygen. Rectal or bladder temperature probes are less accurate if peritoneal or bladder lavage is used. Place rectal temporal probes at a depth of 15 cm to obtain an accurate core temperature measurement.[68]

Airway management is often required in patients with moderate and severe hypothermia. Standard orotracheal intubation may be difficult due to jaw muscle rigidity. Neuromuscular blocking agents are ineffective with core body temperatures less than 30°C and succinylcholine may acutely worsen acute hyperkalemia.[33,53,68] The use of topical vasoconstricting agents, smaller endotracheal tubes, and blind nasotracheal intubation has been suggested.[33] Fiberoptic-guided orotracheal intubation can be utilized if available. Periodically monitor the endotracheal cuff pressure as it may increase with patient rewarming. Chest wall elasticity and pulmonary compliance diminish in hypothermia and ventilatory pressures increase.[18] Ventilate patients at 8 to 12 breaths per minute to avoid respiratory alkalosis.[35]

Management of the circulatory system with moderate to severe hypothermia often requires fluid resuscitation as these patients are volume depleted. Dextrose-containing fluids are contraindicated because the warmed solution will caramelize. Ringer's solution is not recommended due to decreased hepatic metabolism of lactate when hypothermic. **The heart is susceptible to arrhythmias. Take special care when transferring patients on beds or performing procedures. Avoid transthoracic central venous catheters and pulmonary artery catheters until rewarming has been achieved.** Use peripheral intravenous catheters or a femoral central venous line as the guidewire does not reach the heart.

Treat nonperfusing cardiac rhythms using Advanced Cardiac Life Support (ACLS) guidelines. The presence of bradycardia, atrial fibrillation, and other dysrhythmias that do not reduce perfusion do not require pharmacologic support and these will reverse with rewarming. The pulse is clinically difficult to detect in severe hypothermia due to decreased cardiac rate, decreased cardiac contractility, and severe vasoconstriction. Perform bedside US to look for cardiac activity, especially if the presenting rhythm is pulseless electrical activity. Do not initiate CPR unless no central pulse is palpable for 1 minute or pulses are noted to disappear.

TABLE 226-3 Tests Indicated in Hypothermia

Arterial blood gas
Chest x-ray
Coagulation panel
Complete blood count
Cortisol
Creatine kinase
Creatinine
Electrolytes, including magnesium
Ethanol
Glucose
Lactate
Lipase
Liver function tests
Thyroid studies
Toxicology screen

Hold CPR unless there is true cardiac arrest due to lower metabolic demands and predisposition of ventricular fibrillation.[18,44] **Defibrillation at core body temperatures less than 30°C to 32°C may not be effective. Up to three defibrillations may be given. Hold further defibrillation until a core temperature of 30°C has been achieved. Continue resuscitative efforts in conjunction with rewarming.** Those with severe hypothermia may require more forceful compressions due increased stiffness of cardiac and chest wall tissues.[18,31,33,35,44,69] Attempt to pace the heart transcutaneously if needed.[70]

Resuscitation drugs are often ineffective at core body temperatures below 30°C. Lidocaine, verapamil, diltiazem, and neuromuscular blocking agents may have decreased efficacy. Procainamide has been suggested to increase ventricular fibrillation.[35,69] Hypothermia increases the duration of action for rocuronium and vecuronium.[71-74] **Hypothermia elevates serum concentrations of propofol, fentanyl, and midazolam.**[71,75,76] The overall effect on opiate administration is unclear because the opiate receptor exhibits diminished responsiveness.[77] Epinephrine metabolism slows and may accumulate to toxic levels if administered at standard ACLS doses.[31] **It is recommended that medications be administered at one-half the normal dose during the resuscitation of a moderately or severely hypothermic patient to avoid drug toxicity.** Avoid vasopressors due to their potential to cause arrhythmias.[35] Repeat the resuscitation cycle with every increase in core body temperature of 1°C to 2°C until normothermia is achieved.[69] **Patients should not be pronounced dead until they are warm (i.e., greater than 32°C) and dead. Table 226-3 lists pertinent tests in hypothermic patients.**

FLUID MANAGEMENT

The administration of fluids or blood products can exacerbate hypothermia. Infusion of 1 L of crystalloid solution at room temperature (i.e., 21°C) results in a 0.3°C decrease in core body temperature.[5] **Take active measures to prevent hypothermia when patients are receiving large volumes of resuscitation fluid. This is even more important in patients receiving blood products that are kept at 4.0°C.** Transfusion of 1 L of blood products leads to a decrease in core body temperature of 0.54°C.[5] **A normothermic patient can be rendered hypothermic quickly when subjected to an aggressive fluid resuscitation.**

One of the mainstays in the hypothermia prevention during resuscitation is the use of fluid warmers to infuse intravenous fluids. Conventional fluid warmers use a coil or sleeve system in a water bath and pose problems at both slow and rapid infusion rates.[78] Slow infusion rates allow time for the fluid to cool in the

FIGURE 226-2. Examples of warming devices. ***A.*** Smiths Medical Level 1 H-1200. ***B.*** Hotline Level 1. ***C.*** TheraCor 1200 (Smisson-Cartledge Biomedical, Macon, GA.)

tubing between the warmer and the patient. Fast infusion rates leave inadequate time to warm the intravenous fluid.

These problems have been addressed with devices that are useful in the Emergency Department (**Figure 226-2**). They warm infused fluid to a temperature of 40°C. The first uses a jacket in which circulating warmed fluid covers the entire length of the intravenous tubing from the warming device to the patient (Hotline, Smiths Medical, Norwell, MA). This device is more effective than conventional fluid warmers at flow rates between 50 and 6000 mL/hr.[78] The second device uses an inline heating unit that warms the fluid rapidly and allows for substantially higher infusion rates (ThermaCore 1200, Smisson-Cartledge Biosystems, Macon, GA). This device can infuse crystalloid solutions or blood products at rates up to 1200 mL/min and is extremely useful in the resuscitation of patients in severe hemorrhagic shock. Similar devices are available from a variety of manufacturers. **Use these devices to administer intravenous fluid to hypothermic patients or to those at risk for developing hypothermia.** These devices may be used in conjunction with the techniques described below.

PASSIVE EXTERNAL REWARMING

Perform passive external warming on all patients who are mildly hypothermic or who have normal body temperatures and are undressed.[79,80] Approximately 90% of heat loss occurs through the skin.[33,53,71] Cover the patient with warm sheets and blankets as soon as they have been examined and any necessary procedures have been performed.

TECHNIQUES

Remove any wet and/or cold clothing. Dry the patient before applying blankets (Figure 226-3). Blankets and sheets may be kept in a blanket warmer as insulating material cooler than the patient (i.e., room temperature) will extract heat. **Prevent secondary injury**

FIGURE 226-3. Passive external rewarming. Remove any wet and/or cold clothes. Cover the patient with warm blankets.

to the patient from blankets and sheets that are too hot. There is no reason for the blankets to be any warmer than normal body temperature for passive external rewarming.

ASSESSMENT

An otherwise healthy and normal patient will increase their core body temperature by 0.5°C/hr to 2.0°C/hr with passive external rewarming. The body attempts to regain normothermia by increasing its metabolic rate. The patient may manifest evidence of increased cardiopulmonary stress and lactic acidosis during this time.[5]

COMPLICATIONS

Complications occur if attempts are made to place inappropriately heated objects directly on the patient. **Under no circumstances should heated intravenous fluid bags be placed in prolonged contact with the skin.** This may result in thermal injuries ranging from superficial blistering to full-thickness burns. Blankets and sheets that are too warm will also cause damage to the skin.

SUMMARY

Use passive external rewarming as a sole agent in otherwise healthy patients with normothermia who are undressed, patients with mild hypothermia, and as an adjunct to more aggressive rewarming techniques. **Always be aware of the possibility of a patient developing hypothermia during their Emergency Department stay.**

ACTIVE EXTERNAL REWARMING

INDICATIONS

Perform these maneuvers as sole therapy for patients with mild hypothermia, on awake and otherwise stable patients with moderate hypothermia, and in conjunction with more aggressive rewarming techniques.

CONTRAINDICATIONS

Do not use active external rewarming as the sole therapy for patients with moderate hypothermia who have altered mental status or hemodynamic instability. Active external rewarming is contraindicated as the sole therapy for patients with severe hypothermia. **There is no significant difference in the rates of rewarming between the different devices used for external rewarming. The use of radiant heaters and heating packs is not currently recommended due to the risk of potential complications and limited efficacy.**[39,81]

EQUIPMENT

- Warm blankets
- Forced air rewarming blankets (**Figures 226-4*A*, 226-4*B*, and 226-4*C***)
- Fluid circulating rewarming blankets (**Figure 226-4*D***)
- Water bath and associated supplies

TECHNIQUE

Examine the patient. Determine if the patient is a candidate for active external rewarming. Place a forced air rewarming blanket or a fluid circulating rewarming blanket on the patient (**Figure 226-5**). **Do not place these blankets, particularly the fluid circulating ones, underneath the patient.** Placing the blanket under the patient may lead to excessive thermal transfer to areas with decreased blood flow due to pressure and prolonged time of contact. A thin sheet may be placed between a fluid circulating blanket and the skin. This is not necessary with forced air blankets. Warmed blankets and sheets may be placed over the warming blankets.

Active external rewarming can be accomplished by immersion of the patient in a warm water bath (**Figure 226-6**). This technique is easy, effective, and simple. Immersion therapy in the Emergency Department is not practical. The equipment is bulky, not portable, and occupies a large fixed space. Monitoring leads (i.e., electrocardiography and pulse oximetry) will not adhere to wet skin and water submersion precludes care for concomitant medical issues.

ASSESSMENT

Care must be taken to reassess any wounds for recurrent bleeding, any catheters or monitors for placement, and any pressure areas that might be in prolonged contact with the heating elements as the patient is covered with the warming device. **Check the air source connection to the blanket to make sure that the hot air is not blowing directly onto the patient.** A directed flow of hot air onto the patient under the blanket may lead to thermal injury.

COMPLICATIONS

Thermal injuries are associated with active external rewarming. They result from improper use of the devices. Maintain vigilance regarding prevention of a focused delivery of heat to a region of skin, particularly one poorly perfused, to essentially eliminate these burns.

Afterdrop refers to a decrease in core temperature that occurs 15 to 20 minutes after active external rewarming begins.[69] This is thought to be due to vasodilatation of the peripheral tissues as they rewarm and a subsequent washout of cooler peripheral blood back to the central areas. Peripheral vasodilatation can lead to hypotension if fluid resuscitation is inadequate or the patient has cardiovascular compromise. Afterdrop can be attenuated by the concomitant use of core rewarming techniques.

SUMMARY

Active external rewarming is useful in treating moderately hypothermic patients who are awake and alert. Warming rates of 1.0°C/hr to 2.5°C/hr have been reported.[35] Frequent monitoring for potential thermal injuries is required to prevent secondary injury. Monitoring is easier when the patient is a cooperative partner who can participate in the process. Active external rewarming, like passive external rewarming, is often used in conjunction with more aggressive techniques or core rewarming. The problem of afterdrop is lessened when active external rewarming is used in conjunction with core warming techniques.

AIRWAY REWARMING

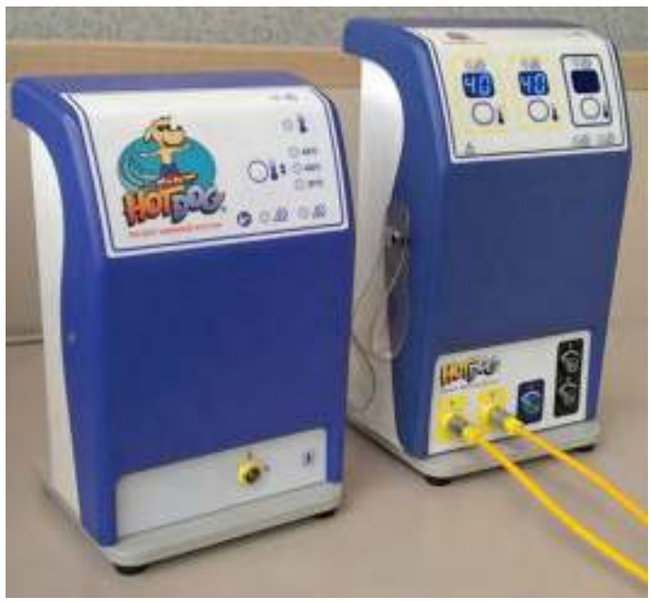
Airway rewarming with warmed and humidified oxygen is safe and easily achieved (**Figure 226-7**). It can be applied to any hypothermic patient. However, airway rewarming is never used as a sole means of rewarming.

INDICATIONS

The technique of airway rewarming may be used in any patient with hypothermia and in conjunction with other techniques. Any patient receiving supplementary oxygen can receive warmed and humidified oxygen. Its efficacy via endotracheal tube is greater than via mask or nasal prongs.

A

B



C

D

FIGURE 226-4. Active external rewarming systems. ***A.*** Stryker Mistral-Air Forced Air warming system. (Photo courtesy of 37 Company, The Netherlands.) ***B.*** Bair Hugger forced air circulator. (Photo courtesy of 3M, Minneapolis, MN.) ***C.*** HotDog air circulating patient warmers. (Photo courtesy of Augustine Temperature Management, Eden Prairie, MN.) ***D.*** Stryker Altrix fluid circulating warmer. (Photo courtesy of Stryker Medical, Portage, MD.)

CONTRAINDICATIONS

There are no contraindications to airway rewarming. The more severely hypothermic patient will often need their airway controlled by orotracheal intubation due to a depressed level of consciousness. **Intubation for the sole purpose of airway rewarming is contraindicated.**

EQUIPMENT

- Nasal cannula
- Face mask
- Endotracheal tubes
- Humidifier tank

FIGURE 226-5. Active external rewarming with a forced air heating blanket.

- Heating circuit
- Oxygen source

TECHNIQUES

Consult the Emergency Department Respiratory Therapist. They will gather, set up, administer, and monitor the required equipment (**Figure 226-7**). Connect the humidifier tank and heating circuit in line with the oxygen source. Humidify and warm the inhaled oxygen to 40°C to 45°C. A rewarming rate of 1.0°C/hr to 2.5°C/hr has been reported.[35] **Avoid reliance on esophageal temperature probes as these may give falsely elevated readings.**

SUMMARY

Airway rewarming can be applied to any hypothermic patient. The oxygen must be humidified to prevent fluid loss from evaporation.

GASTRIC AND BLADDER LAVAGE

These techniques are based on techniques familiar to Emergency Physicians, are minimally invasive, and can be readily performed with minimal interruption of continued resuscitative efforts.[82]

FIGURE 226-6. Active external rewarming with a water bath.

FIGURE 226-7. Airway rewarming equipment with humidified and warmed oxygen. The Opti-Flow cannula and humidifier. (Photo courtesy of Fisher and Paykel Healthcare Ltd., Irvine, CA.)

INDICATIONS

These techniques are usually reserved for patients with severe hypothermia. They are rarely used in stable patients and generally reserved for situations where extracorporeal warming is not available. Gastric lavage can be used for decontamination of stomach contents in the patient who overdoses and is hypothermic.

CONTRAINDICATIONS

The only contraindications to these techniques are the contraindications to the placement of a urethral catheter (Chapter 173) and a nasogastric tube (Chapter 75). Do not perform gastric lavage concurrently with cardiac compressions. Gastric and bladder lavage are of limited value in the management of the hypothermic patient. They are generally bypassed in favor of peritoneal lavage, pleural lavage, and extracorporeal warming.

EQUIPMENT

- Warmed (40°C) intravenous fluid, normal saline or Ringer's lactate
- 60 mL catheter-tip syringe
- Urethral catheterization supplies (Chapter 173)
- Nasogastric intubation supplies (Chapter 75)

TECHNIQUES

Place a Foley catheter (Chapter 173) and/or nasogastric tube (Chapter 75). Confirm their proper placement. Instill sterile saline or Ringer's lactate warmed to 40°C. Inject 60 mL aliquots into the tubes using the catheter-tipped syringe. Instill a total of 300 to 400 mL into the stomach and 200 to 250 mL into the bladder. **Do not overdistend the respective organ.** Gastric overdistention can result in rupture or aspiration. Bladder overdistention can result in rupture and extravasation of fluid. Clamp the Foley catheter and nasogastric tube. Allow the fluid to dwell within the organ for 5 to 10 minutes. Remove the fluid by gravity drainage from the bladder or suction from the stomach. Repeat the process as necessary until the core body temperature increases. Bladder and gastric lavage each add 0.5°C/hr to 1°C/hr.[68]

COMPLICATIONS

Gastric lavage may be complicated by perforation of the stomach by the nasogastric tube or overdistention. Aspiration is a potential complication with gastric lavage. Nasogastric tube placement may provoke arrhythmias in severe hypothermia. These techniques make esophageal and rectal temperatures less accurate. Refer to Chapters 75 and 77 for a more complete discussion of the complications associated with nasogastric intubation and gastric lavage, respectively.

Bladder irrigation is safe if the bladder is not overdistended. The small surface area of the bladder makes it an inefficient means of rewarming. Refer to Chapter 173 for a more complete discussion of the complications associated with urethral catheterization. Use normal saline as sterile water can result in significant volume and electrolyte shifts.

SUMMARY

Perform gastric or bladder irrigation if other types of body cavity rewarming are contraindicated. These techniques are easily initiated using standard equipment in an Emergency Department. These organs have relatively small surface areas which makes these techniques inefficient. Overdistention of the bladder or stomach can result in serious complications.

PERITONEAL LAVAGE

This technique is invasive, is based on techniques familiar to Emergency Physicians, and can be readily performed with minimal interruption of resuscitative efforts.

INDICATIONS

This technique is usually reserved for patients with severe hypothermia. It is rarely used in stable patients and generally reserved for situations where extracorporeal warming is not available.

CONTRAINDICATIONS

Suspicion of injury to a hollow viscus is a contraindication to this technique. The presence of a previous laparotomy scar is a strong relative contraindication. The use of an open peritoneal lavage technique does not work well with the continuous infusion of fluid required for this procedure. Other contraindications are those associated with performing a diagnostic peritoneal lavage (Chapter 84).

EQUIPMENT

- Warm (40°C) intravenous fluid, normal saline or Ringer's lactate
- Warm (40°C) peritoneal dialysis fluid
- Fluid infuser device, optional
- Peritoneal lavage kits
- Peritoneal lavage supplies (Chapter 84)
- Nasogastric tube supplies (Chapter 75)
- Foley catheter supplies (Chapter 173)

A commercially available, disposable, single patient use peritoneal lavage kit is available from numerous manufacturers. The kit includes all the material required to perform a closed or semi-open diagnostic peritoneal lavage except lavage fluid. An example is the Arrow kit (Arrow International, Reading, PA). It contains 10% povidone iodine or chlorhexidine swabs, gauze squares, fenestrated drape, tubing for the administration of intravenous fluid, 1% lidocaine, 5 mL syringes, 22 and 25 gauge needles, an 18 gauge × 2.5 inch introducer needle, an 0.89 mm × 45 cm J-tipped guidewire, an 8 French lavage catheter, and a #11 scalpel blade on a handle. Refer to Chapter 84 for a more complete list of equipment.

TECHNIQUE

Prepare the patient by placing a nasogastric tube (Chapter 75) and a Foley catheter (Chapter 173). Place two peritoneal lavage catheters (Chapter 84), one supraumbilical and one infraumbilical (**Figure 226-8**). Confirm the position of the peritoneal catheters. Clamp the infraumbilical peritoneal catheter. Infuse warm (40°C) intravenous fluid or peritoneal dialysis fluid through the supraumbilical peritoneal catheter using the Level 1 infuser or a fluid warmer. The use of dialysate has the added benefit of potentially correcting some electrolyte abnormalities.[35,69] The fluid may be infused manually but this technique is much less efficient. Infuse 1 to 2 L of fluid. Allow the fluid to bathe the peritoneal cavity for 10 minutes. Remove the clamp on the infraumbilical peritoneal catheter and allow the fluid to drain from the peritoneal cavity. Repeat the procedure. Up to 10 L/hr of fluid may be infused in this fashion.[35] An alternative technique is to allow the fluid to flow at a moderate rate continuously through the supraumbilical peritoneal catheter, into the peritoneal cavity, and out the infraumbilical peritoneal catheter. This can

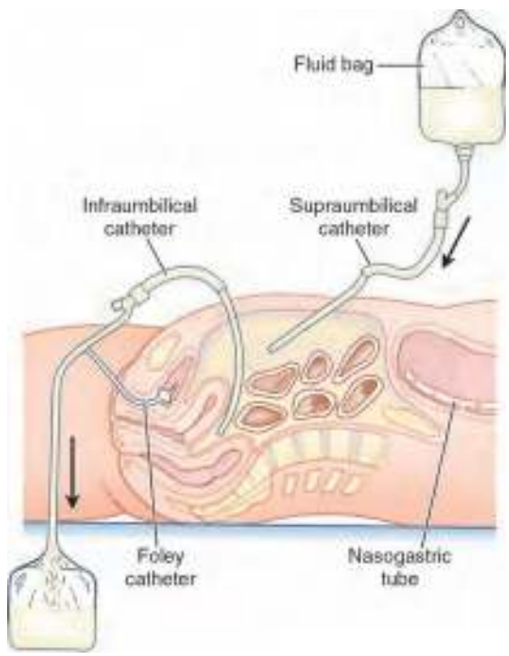


FIGURE 226-8. Active core rewarming with peritoneal lavage.

FIGURE 226-9. The Veratherm Portable Hypothermic Perfusion System.

be performed manually or with a commercial device developed for this purpose (Veratherm Portable Hyperthermic Perfusion System, Thermal Therapeutic Systems Inc., Pittsburg, PA) (**Figure 226-9**).

COMPLICATIONS

The potential complications are primarily associated with the placement of the peritoneal lavage catheter (Chapter 84). The infusion and removal of the fluid are safe. Avoid using highly hyperosmolar peritoneal dialysates in patients with persistent hypovolemia or with a questionable perfusion status as this may worsen existing hypovolemia. Do not use sterile water as it can result in significant volume and electrolyte shifts.

SUMMARY

Peritoneal lavage is the most widely recognized means of body cavity lavage. This is due in part to the familiarity with the diagnostic peritoneal lavage procedure. It has the advantage of being able to be performed concurrently with chest compressions and other resuscitative efforts. This technique can raise the core body temperature by 1°C/hr to 3°C/hr.

PLEURAL LAVAGE

This technique is invasive, is based on techniques familiar to most Emergency Physicians, and can be readily performed with minimal interruption of resuscitative efforts. Pleural lavage can be performed closed (i.e., tube thoracostomy) or open (i.e., a thoracotomy).

INDICATIONS

This technique is usually reserved for patients with severe hypothermia. It is rarely used in stable patients and generally reserved for situations where extracorporeal warming is not available. Pleural lavage is superior to gastric and bladder lavage.[63]

CONTRAINDICATIONS

The presence of a pneumothorax and/or hemothorax is the only contraindication to pleural lavage. This procedure is difficult when performed in conjunction with chest compressions.

EQUIPMENT

- Tube thoracostomy supplies (Chapter 51)
- Warm (40°C) intravenous fluid, normal saline or Ringer's lactate
- Warm (40°C) peritoneal dialysate
- Fluid infuser device, optional

TECHNIQUE

Place two chest tubes (Chapter 51) into the same pleural space, either the left or the right side, to perform closed pleural lavage (**Figure 226-10**). The left side is preferred by those who believe that the heart, aorta, and blood within the aorta will be warmed quicker than with right-sided chest tubes. Place one tube anteriorly in the second or third intercostal space at the midclavicular line (**Figure 226-10**). Place the other tube posteriorly in the fifth or sixth intercostal space at the posterior axillary line (**Figure 226-10**). Secure the tubes to the chest wall with suture and tape. Attach the fluid infuser to the anterior chest tube using a Christmas tree adapter. Apply a pleural drainage device to the posterior chest tube in the usual fashion. Infuse warm (40°C) fluid via the fluid infuser device at a rate of 180 to 550 mL/min.[69] Allow the fluid to exit into the pleural drainage device. The fluid will flow into the anterior chest tube, bathe the structures in the hemithorax (i.e., great vessels, heart, lung, and mediastinum), and exit via the posterior chest tube. This can be performed manually or with a commercial device developed for this purpose (Veratherm Portable Hyperthermic Perfusion System, Thermal Therapeutic Systems Inc., Pittsburg, PA) (**Figure 226-9**).

Pleural lavage may be performed using an open technique (**Figure 226-11**). This technique is used when a thoracotomy (Chapter 54) is indicated for other purposes (e.g., penetrating trauma or to perform open cardiac massage). **Do not use this technique for the sole purpose of rewarming if other less invasive or extracorporeal techniques are available.** The advantage of open pleural lavage is that it allows direct cardiac rewarming and open cardiac massage.

COMPLICATIONS

The complications are primarily associated with the placement of the chest tube (Chapter 51) or the thoracotomy (Chapter 54). Maintain free drainage of the irrigation fluid to avoid increasing the intrathoracic pressure.

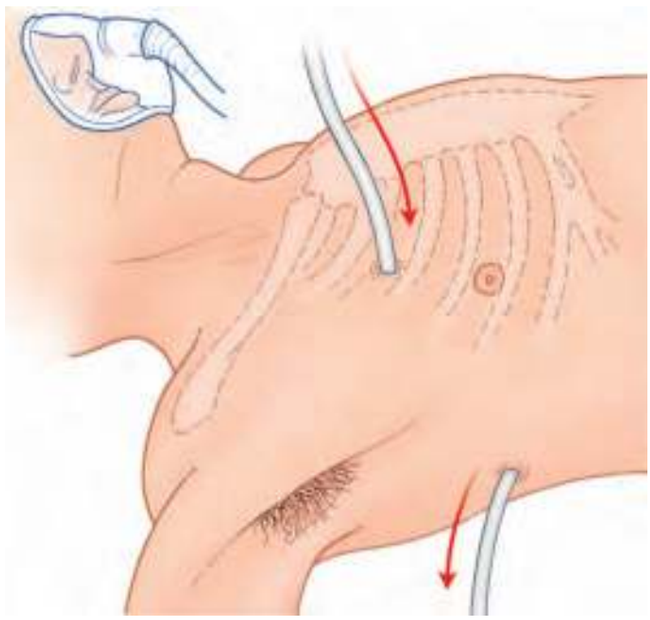


FIGURE 226-10. Active core rewarming with closed pleural lavage.

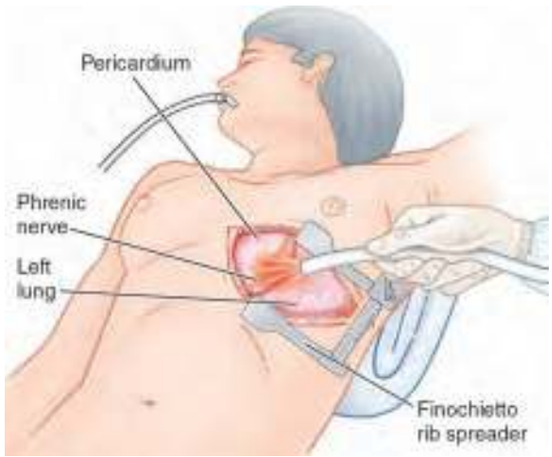


FIGURE 226-11. Active core rewarming with open pleural lavage.

SUMMARY

Closed pleural lavage is an effective means of rewarming. It can increase the core body temperature by 1°C/hr to 5°C/hr. It is very similar in efficacy to peritoneal lavage. Chest tube placement is slightly more challenging than placement of a peritoneal lavage catheter. Free drainage is usually easier to achieve and maintain with a chest tube than with a peritoneal lavage catheter. The two techniques may be employed simultaneously and carried out in conjunction with other resuscitative measures.

EXTRACORPOREAL REWARMING

Extracorporeal rewarming is the most aggressive, effective, and efficient technique to correct severe hypothermia (Chapter 73). It has been shown to improve outcomes in those with hypothermic cardiopulmonary arrest and there is growing evidence supporting its use in patients with severe hypothermia.[83,84] It is suggested that Emergency Medical Services transport severely hypothermic patients to centers capable of these procedures even if transportation times are increased.[31,59,68]

Extracorporeal rewarming includes the use of hemodialysis, arteriovenous rewarming, venovenous rewarming, continuous venovenous hemofiltration, cardiopulmonary bypass, and extracorporeal membrane oxygenation. The choice between the techniques is made based on availability of resources and the Emergency Physician's familiarity with each technique. Cardiopulmonary bypass is discussed but should not be performed by anyone who is not a Surgeon familiar with the procedure.

HEMODIALYSIS

Hemodialysis is readily available in most institutions. It requires the placement of a specialized double-lumen central venous line. The only contraindications to hemodialysis are those associated with central venous line placement (Chapter 63) and hemodialysis. An advantage of hemodialysis is that associated drug toxicity or significant electrolyte abnormalities can be simultaneously managed as the patient is being rewarmed.[85,86]

EQUIPMENT

- Central line supplies (Chapter 63)
- Dual-lumen percutaneous hemodialysis (e.g., Quinton or Mahurkar) catheter
- Dialysis machine
- Dialysis nurse or technician
- Warmed (40°C) dialysis fluid

TECHNIQUE

Place a double-lumen central venous hemodialysis catheter into a femoral vein (Chapter 63). The catheter may be placed into a subclavian vein if femoral vein access is contraindicated. Femoral vein placement decreases the risk of inducing an arrhythmia with the guidewire.[35] There is a tendency to have fewer problems related to the positioning of the catheter and achieving appropriate flow rates with femoral catheters. Instruct the hemodialysis technician to set the temperature control on the dialysis machine to deliver a blood temperature of 40°C. Attach the dialysis machine tubing to the lumens of the catheter. Hemodialysis warms patients at 2°C/hr to 3°C/hr.[68]

COMPLICATIONS

The potential complications are primarily associated with the placement of the central venous line (Chapter 63). The hemodynamic status of hypotensive patients may become worse upon initiation of hemodialysis but this technique has been successfully used on hypothermic patients.[35]

SUMMARY

Hemodialysis is available at most institutions and is effective in rewarming a patient.[85,86] The limitations of this technique include the need to have a dialysis technician or nurse available, the time required to obtain on-call personnel, and the time required to set up the equipment. Hemodialysis is a viable option for the rewarming of a hypothermic patient when other forms of extracorporeal warming are not available.

ARTERIOVENOUS REWARMING

This technique was initially described in 1992.[87,88] The authors described placing one large-bore catheter in the femoral artery and another in a central vein (i.e., femoral, internal jugular, or subclavian). This method allows for rewarming in the Emergency Department without the need for specialized equipment (e.g., dialysis or bypass machine) or heparin administration (**Figure 226-12**).[89]

CONTRAINDICATIONS

This method requires the patient's own blood pressure to circulate blood through the warming system. Hypotension or cardiac arrest makes this method ineffective. Known occlusive arterial disease, particularly in the iliofemoral region, is a relative contraindication.

EQUIPMENT

- Central line supplies (Chapter 63)
- Two 8.5 French introducer catheters
- Two 10 French single-lumen arteriovenous hemofiltration catheters
- Fluid warmer
- Luer-lock adapter, male-to-male
- Warmed (40°C) saline

TECHNIQUE

Place two catheters, one in the femoral artery and one in a central vein (Chapter 63). The placement of the arterial catheter is like that of the venous catheter.

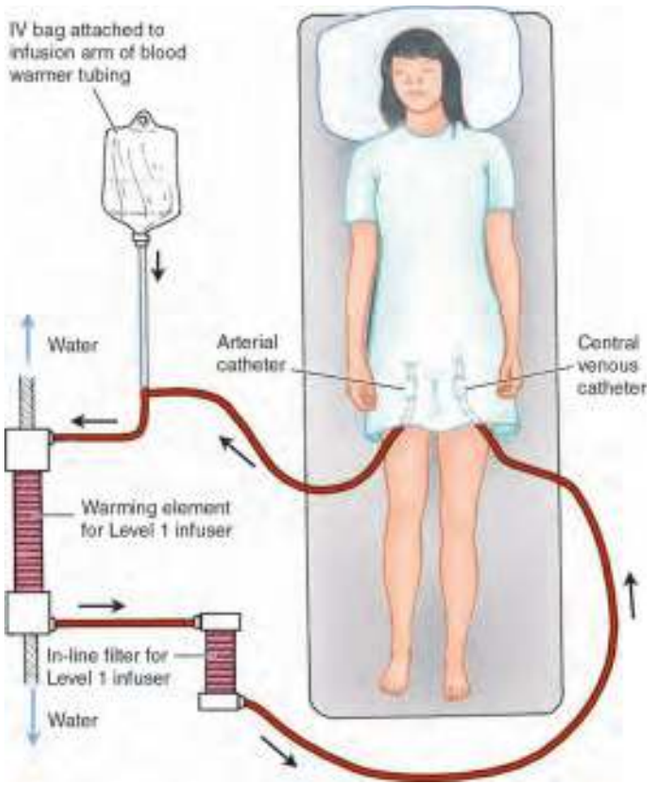


FIGURE 226-12. Arteriovenous rewarming.

The tubing for the Level 1 rapid infuser consists of two intravenous spike-tipped lengths that come together in a "Y." The "Y" runs to a countercurrent warming chamber and then to the patient. Cut off the spike tip from one of the limbs of the "Y" and attach the male-to-male Luer-lock adapter. An additional length of wide-bore intravenous tubing may be needed to bridge the distance between the arterial catheter and the fluid-warmer tubing. Connect this limb to the arterial catheter. Connect the other arm of the "Y" to a bag of intravenous fluid with the roller clamp closed.

Allow the system to prime with blood under the patient's arterial pressure. Connect the primed distal end of the tubing to the central venous catheter. The blood will travel via the arterial catheter to the extracorporeal circuit and through the proximal limb of the tubing to the warming chamber of the Level 1 infuser, through the heating elements to the distal tubing, and back to the patient via the venous catheter (**Figure 226-12**).

The driving force in this circuit is the patient's systemic blood pressure. Low-flow states will limit the efficacy of the system. A pressure infusion of intravenous fluid via the other limb of the "Y" augments flow. This fluid will be warmed as it reaches the patient, since this other limb of the "Y" is proximal to the warming element. Systemic anticoagulation is not required as the tubing for the Level 1 infuser is heparin-bonded. Arteriovenous rewarming is faster and more efficient than venovenous rewarming.[90]

COMPLICATIONS

The complications outlined in the initial description of the procedure include the inability to achieve vascular access, a small hematoma at the arterial puncture site, and ischemia of the limb distal to the arterial catheter.[87] Refer to Chapters 63 and 72 for a full description of the complications that may arise in placing a central venous catheter and an arterial catheter, respectively.

SUMMARY

Arteriovenous rewarming is an innovative means by which severely hypothermic patients may be rapidly rewarmed without the resources of a dialysis or a bypass pump technician. This mechanism utilizes the patient's own blood pressure gradients and can warm efficiently by approximately 3°C/hr to 4°C/hr.[53]

VENOVENOUS REWARMING

This technique is rarely utilized by Emergency Physicians due to the lack of a venous roller pump and inexperience with this piece of equipment. The technique is briefly described for the sake of completeness. The venovenous technique has two main advantages over arteriovenous rewarming.[91] First is the lack of arterial cannulation and its potential for limb ischemia. The second is not relying on systemic blood pressure to drive the system. Flow may be independently determined and delivered by the roller pump. This may be particularly important in the severely hypothermic patient who presents in full cardiorespiratory arrest. The primary disadvantage of this technique is the requirement of expertise in using the roller pump, although advances in design may obviate this in the future.

CONTRAINDICATIONS

Persons not familiar with the function and operation of a venous roller pump should not attempt this technique.

EQUIPMENT

- Central line supplies (Chapter 63)
- Two 8.5 French introducer catheters
- Two 10 French single-lumen arteriovenous hemofiltration catheters
- Fluid warmer
- Luer-lock adapter, male-to-male
- Venous roller pump
- Warmed (40°C) saline

TECHNIQUE

The setup for this technique is like that for arteriovenous rewarming.[91] Place two central venous catheters (Chapter 63), one in the femoral vein and one in another central vein. Alternatively, place a dual-lumen hemodialysis catheter.[92] Ensure that the "outflow" port of the hemodialysis catheter is upstream and that the "inflow" port is downstream. This helps to reduce the amount of warmed fluid returning from the circuit that is immediately withdrawn by the roller pump. Place the venous roller pump in line with this circuit between the patient and the warming elements. Adjust the flow rate within the tolerances of the device.

COMPLICATIONS

The primary complication associated with use of venous roller pumps is an air embolus. This usually happens when the roller pump is operated by a person unfamiliar with it. Recent advancements in roller pump technology have made their operation simpler and safer. Refer to Chapter 63 for a complete description of the complications that may arise due to the placement of a central venous catheter.

SUMMARY

Venovenous rewarming is one step away from cardiopulmonary bypass. Its use in the Emergency Department is limited due to lack of equipment availability and familiarity. It is a viable option if a

roller pump is available as well as a Physician familiar with this procedure. Warming rates of 4°C/hr to 10°C/hr have been noted.[68,91]

CONTINUOUS VENOVENOUS HEMOFILTRATION

Continuous venovenous hemofiltration (CVVH) machines have become more widely available. This is an attractive means of rewarming. The use of CVVH machines has largely replaced the venovenous rewarming technique. This technique has two main advantages over arteriovenous rewarming. First is the lack of arterial cannulation with its potential for limb ischemia. The second is not relying on systemic blood pressure to drive the system. The use of CVVH in the management of moderate-to-severe accidental hypothermia has been shown to be rapid, safe, and effective.[88,93]

CONTRAINDICATIONS

Persons not familiar with the function and operation of a CVVH machine should not attempt this technique.

EQUIPMENT

- Central line supplies (Chapter 63)
- Dual-lumen percutaneous hemodialysis (e.g., Quinton or Mahurkar) catheter
- CVVH dialysis machine
- Dialysis nurse, dialysis technician, or trained Intensive Care Unit nurse
- Warmed (40°C) dialysis fluid

TECHNIQUE

Rewarming with CVVH utilizes a single intravenously inserted, dual-lumen catheter placed in a central vein and connected to the CVVH dialysis machine. Some CVVH dialysis machines have a built-in thermostat allowing for temperature control while others depend on circulating already warmed dialysis fluid bags. Passive and/or active external rewarming maneuvers should be used in conjunction to the CVVH technique. Continuously monitor cardiopulmonary monitoring and core body temperature.

COMPLICATIONS

Recent advancements in CVVH technology have made their operation simpler and safer in terms of the prior concern over an air embolism. Their use should be restricted to individuals familiar with the operation of these devices. Refer to Chapter 63 for a complete description of the complications that may arise due to the placement of a central venous catheter.

SUMMARY

CVVH is one step away from cardiopulmonary bypass. Its use in the Emergency Department is limited due to lack of equipment availability and familiarity. It is a viable option if the equipment is available as well as a Physician familiar with this procedure. It has a reported warming rate of 1.5°C/hr to 3°C/hr.[68,83,94]

CARDIOPULMONARY BYPASS

Discussion of this technique is limited to some basic properties of rewarming with cardiopulmonary bypass. This technique should not be attempted by anyone who does not regularly use bypass modalities and without the support of an experienced pump technician. Bypass is usually achieved using femoral-femoral bypass. The cardiopulmonary bypass pump can provide complete hemodynamic and respiratory support during the resuscitation.[87,95,96] Core body temperatures can be raised 1°C to 2°C every 3 to 5 minutes with flow rates of 2 to 3 L/min. Use heparin-bonded tubing if available to avoid systemic anticoagulation. This technique is extremely resource intensive, not readily available in many institutions, and requires specific expertise with respect to surgical and mechanical techniques.

EXTRACORPOREAL MEMBRANE OXYGINATION

There are multiple case reports of extracorporeal membrane oxygenation (ECMO) success in severe hypothermia (Chapter 73).[8,97-99] Patients with core temperatures as low as 13.8°C, with 5 hours of CPR, and with 83 minutes of complete cold water submersion have been reported to have a good neurologic outcome after ECMO.[97,100] Some are reporting successful use in trauma cases.[98] Hypoxemia, cardiac arrest, and asphyxia are recognized as major predictive adverse factors for survival and/or poor neurologic outcome in the hypothermic patient.[99,101] ECMO provides appropriate support for cardiac and pulmonary functions with the added benefit of allowing for hemodialysis for restoration of electrolyte abnormalities and acid-base imbalances. ECMO administration in severe hypothermia has significantly improved survival and neurologic recovery when compared to lavage techniques. **Patients with severe hypothermia should be preferentially transported to hospitals where ECMO is available even if this prolongs transport times.**

The main obstacle to initiation of ECMO is the process of cannulation and setting up the apparatus (Chapter 73). Starting ECMO takes 134 minutes on average and requires specialized personnel.[83,84] A new device and technique termed portable and percutaneous cardiopulmonary bypass (PPCPB) has been under investigation. PPCPB does not require heparinized tubing or advanced surgical technique. It can be initiated by an Emergency Physician. The process involves simple large vessel arterial and venous access. Set up requires 48 minutes on average. PPCPB has all the benefits of ECMO and warms patients by approximately 4°C/hr.[83,84] PPCPB has improved outcomes in severe hypothermia with and without cardiac arrest when compared to other invasive techniques (e.g., lavage) and comparable outcomes to ECMO.[84] Refer to Chapter 73 for the complete details of ECMO.

ALTERNATIVE TECHNIQUES

There has been much research regarding the induction of therapeutic hypothermia. This has resulted in the development of a variety of external and internal cooling devices. They can easily be used to manage the hypothermic patient while not specifically designed to treat hypothermia.[102] Reports have been published using endovascular cooling catheters to warm hypothermia patients with good outcomes.[103] These devices use a special central catheter that is placed the same way as central venous lines and warm patients by 0.5°C/hr to 2.5°C/hr.[68] New esophageal cooling devices can be used to warm patients but no data on warming accidental hypothermia patients have been published.[104-106] These devices are placed in a similar manner to nasogastric tubes.

SUMMARY

The management and prevention of hypothermia in the Emergency Department is a challenging and perpetual task. The resuscitation of a severely hypothermic patient in full cardiorespiratory arrest can push the Emergency Physician and the Emergency Department

to the limit of their abilities and resources. The challenge in dealing with hypothermia lies in recognizing and managing the risk of hypothermia in the "routine" patient who presents every day. It is not possible to overstate the importance of continuity of care and the necessity of good communication as the patient is moved from the Emergency Department to either the Operating Room or the Intensive Care Unit so that rewarming efforts may continue.

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227 Hyperthermic Patient Management

Jessica Mann and J. Elizabeth Neuman

INTRODUCTION

The evaluation and management of hyperthermia and heat stroke can be difficult and challenging in the Emergency Department (ED). **Heat stroke is defined as a core body temperature equal to or greater than 40°C with associated central nervous system dysfunction.[1] Heat stroke is a multisystem insult.** There are two types of heat stroke, the classic nonexertional heat stroke and the exertional heat stroke. Classic heat stroke affects individuals most often over 70 years of

age with underlying chronic medical conditions that impair thermoregulation, prevent removal from a hot environment, interfere with access to hydration, or interfere with attempts at cooling.[2] Exertional heat stroke generally occurs in young healthy individuals who engage in heavy exercise during periods of high ambient temperature and humidity. Typical patients are athletes and military recruits in basic training.[3] More than 300 people die of heat-related illness in the United States each year.[4,5] This can more than double in years with prolonged heat waves.[6] Exertional heat illness is one of the leading causes of death in young athletes each year.[5,7] Heat stroke is an uncommon medical emergency. It is considered one of the most important of all the environmental illnesses because of its potential for high morbidity and mortality in large numbers.[8] Major complications of heat stroke include seizures, adult respiratory distress syndrome (ARDS), acute renal failure, liver disease, rhabdomyolysis, disseminated intravascular coagulation, and death.[9]

The patient must be exposed adequately. Cooling must be initiated in the quickest and most efficient manner possible as stabilization is occurring. Rapid cooling is the most effective strategy for minimizing morbidity and mortality from heat stroke and should be initiated as soon as possible and within 30 minutes of presentation.[10] The most effective means of cooling remains controversial. Few controlled studies are available to determine the best method for achieving rapid cooling of patients. The techniques rely upon prompt recognition of symptoms, immediate intervention in the prehospital setting, a complete diagnostic evaluation in the ED, and the continued care in the ED. Begin cooling the patient in the prehospital setting by removing the patient from the heat stress, removing any excess clothing, keeping the skin wet, and fanning the patient in transport.

Continuous core temperature monitoring with a rectal or esophageal probe is mandatory. **Cooling measures should be stopped once a temperature of 38°C to 39°C has been achieved to reduce the risk of iatrogenic hypothermia.[11] Cooling must precede the investigation for the cause. Evaporation and convection are the simplest and most efficient means of cooling victims of heat stroke or heat exhaustion.** Skin blood flow is preserved as compared with the use of ice because evaporation and convection are much more efficient modes of heat exchange.[12] Evaporation of 1 gm of water dissipates approximately seven times more heat than melting the same quantity of ice.[4]

The information in this chapter primarily pertains to hyperthermia from heat-related illness. Some of these techniques may be also used for other causes of hyperthermia. These include endocrine disorders, head injury, infection, intracranial hemorrhage, malignant hyperthermia, neuroleptic malignant syndrome, sepsis, serotonin syndrome, stroke, toxins, or other causes. Consider discussing the use of these techniques to treat hyperthermia with the appropriate consultant if the etiology is not heat-related.

ANATOMY AND PATHOPHYSIOLOGY

The classification of heat illness is controversial and there continues to be much disagreement about the general categories of illness. The International Classification of Diseases (ICD) published by the World Health Organization (WHO) offers a logical approach to categorizing the various forms of exertional heat illnesses.[13] Diagnoses include heat edema, heat cramps, heat syncope, heat exhaustion, and heat stroke (**Figure 227-1**). Heat edema is self-limited. The patient presents with edema of the hands, feet, and ankles. This usually occurs in the first few days of heat acclimatization. Heat cramps occur most often in individuals who sweat profusely and are exercising or walking. Numerous factors contribute to the development of muscle cramps that include dehydration, loss of sodium, loss of potassium, extreme environmental conditions, and neurogenic fatigue.[14] Heat syncope is dizziness or syncope after exposure

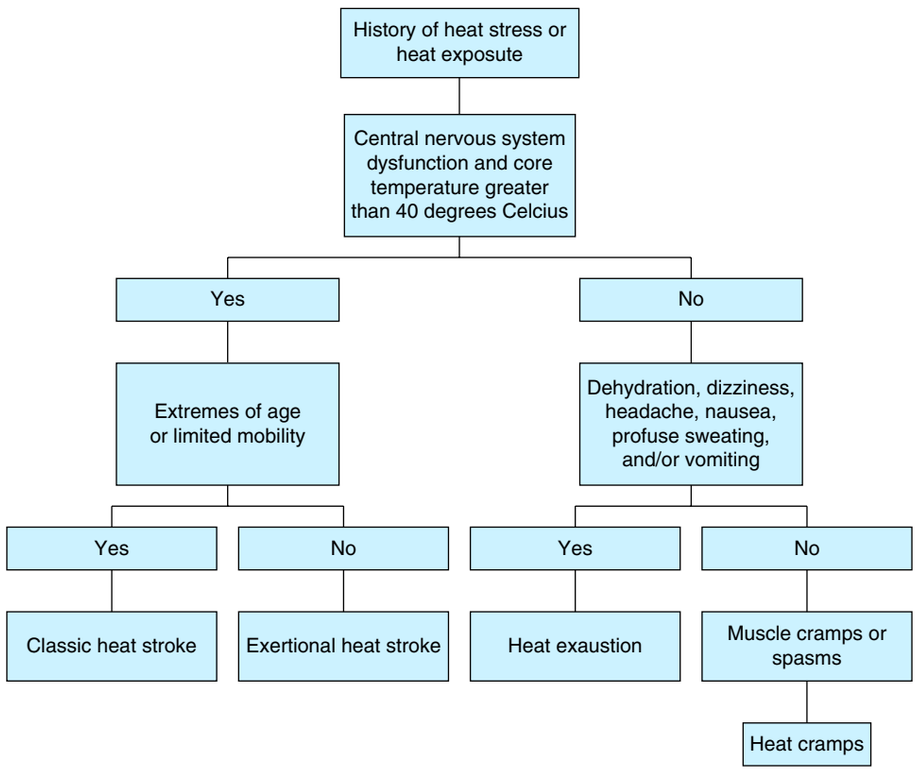

FIGURE 227-1. The types of heat-related illness.

to high temperatures. It is caused by vasodilatation with subsequent decrease in venous return and postural tone. Heat exhaustion is due to the inability to maintain adequate cardiac output. This can result from the excessive loss of body water, electrolytes, or both. The patient may complain of headache, nausea, vomiting, malaise, and myalgias. Heat exhaustion is distinguished from heat stroke in that there is no significant dysfunction of the central nervous system. Any mental status changes associated with heat exhaustion are mild and resolve quickly with rest.

Management of the heat stroke victim can be difficult. This is even after the successful initial resuscitation and stabilization. **Heat stroke is a multisystem insult that affects virtually every organ system.**[11,15-23] Neurologic complications of hyperthermia include seizures, cerebral edema, delirium, and localized brain hemorrhages. Irreversible brain damage often occurs above 42°C or 108°F. Cerebellar impairment may persist after recovery. Cardiac complications include tachydysrhythmias, high-output heart failure, and myocardial injury. Cardiac dysfunction and tachydysrhythmias usually resolve with cooling. Pulmonary edema may be cardiogenic in patients with a limited cardiac reserve or secondary to ARDS. Pulmonary aspiration can be encountered in obtunded patients. Patients may also have bronchospasm, pulmonary infarction, and pulmonary hemorrhage. Acute renal failure may be due to direct heat damage, renal hypoperfusion, or rhabdomyolysis. Mucosal ulceration of the gastrointestinal tract is common and can lead to hemorrhage. Hepatic damage and dysfunction are common. The extent of hepatic necrosis and cholestasis may not be apparent until 48 to 72 hours after the heat injury. Hepatic injury is usually self-limited but may progress to fulminant failure and require a liver transplant. Hematologic complications include hemolysis, thrombocytopenia, and disseminated intravascular coagulation. Disseminated intravascular coagulation, triggered by diffuse endothelial and organ damage, is usually delayed 2 to 3 days in onset and is associated with a high mortality. Replacement of clotting factors with fresh frozen plasma and platelets may be necessary.

Heat stroke must be suspected in any patient who has acute mental status changes or other signs of central nervous system dysfunction in the setting of a high core temperature and a history of heat exposure. The initial temperature is usually at least 40°C to 42°C or 104°F to 108°F. Central nervous system dysfunction may manifest as varying degrees of confusion, obtundation, seizures, delirium, or focal neurologic deficits.

Core body temperature is maintained within close limits by a balance between heat production and heat dissipation. Muscular and metabolic activity along with absorption of heat from the environment results in a heat load on the body. Most fevers encountered are a response to microbial invasion. Some fevers are due to exposure to high temperatures or abnormalities in the thermoregulatory apparatus.[24] The body temperature is controlled by the preoptic nucleus of the anterior hypothalamus. Factors that may compromise protective thermoregulatory mechanisms include chronic illness, psychiatric disorders, cardiovascular disease, endocrine disorders, nutritional deficiencies, metabolic disorders, infection disorders, medications, substance abuse, poor judgment, or extremes of age (**Table 227-1**).[9,25,26]

It has been speculated that oxidative phosphorylation becomes uncoupled and enzyme systems cease to function when temperature regulation is lost and the core body temperature exceeds 42°C or 108°F. Energy stores become depleted as maximum metabolic demands escalate. This causes the cell membranes to become increasingly permeable thus increasing sodium influxes. These phenomena have been postulated to be part of a positive feedback loop involving progressive depletion of adenosine triphosphate (ATP), increased ion flux, increased rates of membrane depolarization and neurotransmitter activity, and a subsequent increase in heat production. Cell membranes eventually lose their integrity. Heat production accelerates, proteins denature, and widespread necrosis occurs as temperature-control mechanisms fail and leads to organ failure. Cellular damage occurs due to the elevated temperatures and the length of exposure to heat.[9,27-29] **Tissues at greatest risk for heat-related damage include the vascular endothelium, neural tissue, hepatocytes, and kidneys.**

There are numerous mechanisms of heat dissipation including evaporation, radiation, convection, and conduction. Evaporation is the principal mechanism. It is the conversion of a liquid to the gaseous phase. Evaporation becomes the dominant mechanism of heat loss as ambient temperatures rise.[27,30] Radiation is the transfer of heat to objects not in direct contact with the subject. Radiation accounts for approximately 65% of heat loss in cool environments. It is a major source of heat gain in hot climates. Convection is the transfer of heat to circulating fluid or gas. The amount of heat dissipated by convection becomes minimal as ambient temperature rises. Convective heat loss varies directly with wind velocity. Conduction is the direct transfer of heat to another object by direct contact.

INDICATIONS

Cooling will not harm patients who are hyperthermic regardless of the etiology. Cooling can be lifesaving in those with true heat stroke. Heat stroke may be difficult to identify if cooling was initiated in the field and the patient's mental functioning is intact. **Initiate cooling immediately if there is any doubt.**[9]

TABLE 227-1 Factors Related to Hyperthermia

Illness	Medications	Behaviors	Groups	Toxins
Autonomic neuropathies	Anticholinergics	Confining garments	Athletes	Alcohol
Cardiovascular disease	Antihistamines	Hot environments	Elderly	Amphetamines
CNS tumors	Antipsychotics	Inability to care for self	Infants	Cocaine
Dehydration	Diuretics	Injudicious exertion	Military recruits	Hallucinogens
Delirium tremors	Lithium	Poor fluid intake	Neonates	
Diabetes	MAO inhibitors	Social isolation	Prior heat stroke	
Hyperthyroidism	Neuroleptics		Psychiatric	
Major burn scarring	Phenothiazines			
Parkinson's disease	Salicylates			
Pheochromocytoma	Serotoninergics			
Psychosis	Sympathomimetics			
Thyroid storm	Thyroid replacements			
	Tricyclic antidepressants			

CNS, central nervous system; MAO, monoamine oxidase.

CONTRAINDICATIONS

Evaluation and resuscitation of every patient always begins with assessment of the patient's airway, breathing, and circulation despite the need for immediate cooling. **Any life-threatening or limb-threatening conditions must be addressed simultaneously or before proceeding to cooling measures.**

Typical fever treatments are not indicated in the treatment of hyperthermia. The use of antipyretics such as acetaminophen and nonsteroidal anti-inflammatory drugs is not effective in treating hyperthermia due to heat-related illness. Do not administer medications used to treat malignant hyperthermia, neuroleptic malignant syndrome, or serotonin syndrome.[31] These treatments do not work for heat-related hyperthermia.

There are specific contraindications to the individual cooling techniques. Ice water immersion is harmful to elder patients. It is difficult in patients with certain intravenous (IV) access (e.g., intraosseous) or monitor connections. Ice water immersion is also contraindicated in patients with seizures or airway compromise. Iced gastric lavage is contraindicated in patients with altered mental status or depressed airway reflexes unless they are endotracheally intubated or if nasogastric intubation (Chapter 77) is contraindicated. Iced peritoneal lavage should not be performed if the placement of a peritoneal lavage catheter (Chapter 84) is contraindicated.

EQUIPMENT

- Core thermometer (esophageal, rectal, or urinary)
- Large fans
- Spray bottles with a misting nozzle
- Water
- Ice
- Immersion tub
- Ice packs
- Wet sheets and towels
- Cooling blanket
- Peritoneal lavage kits
- Nasogastric tubes
- Iced saline

PATIENT PREPARATION

Immediately remove the patient from the heat-stress environment. Explain the cooling techniques to be employed to the patient and/or their representative. Remove the patient's clothes. Stabilize the patient's airway, breathing, and circulation as necessary. **Patient outcome is directly related to the length of time the tissues are exposed to the thermal challenge.** Apply cardiac rhythm monitoring, a noninvasive blood pressure cuff, and pulse oximetry. Place an esophageal (if the patient is intubated), rectal, or urinary temperature probe to monitor the patient's core body temperature. **Do not use any other routes for obtaining temperature as they are not accurate.**[32]

TECHNIQUES

EVAPORATION

This technique is simple, does not interfere with patient care, and is noninvasive. It causes very little to no discomfort to the conscious patient. An additional benefit to evaporation is the access to the patient should other interventions be necessary.[30] This method is the most common for treatment of classic heat stroke. Completely wet the exposed patient with tepid water. Place large and powerful fans to strategically direct high-flow air currents toward the patient's head, feet, and torso (**Figure 227-2**). Spray the patient with a mist of tepid water from the spray bottles. Keeping the patient "wet and windy" provides a state-of-the-art cooling method for even the smallest ED without the need to purchase new or specialized equipment. Place ice packs wrapped in wet towels on the patient's groin, axilla, and neck.

Discontinue evaporative cooling when the core body temperature reaches 39°C or 102°F. Continued cooling can result in hypothermia as the core temperature continues to decrease after evaporative cooling is discontinued. Monitor the patient to ensure that their core body temperature does not increase.

The concept of evaporative cooling gained popularity with the Makkah Body Cooling Unit (BCU) (**Figure 227-3**). The Makkah BCU is used to provide field treatment for people participating in the annual pilgrimage to Mecca who may develop heat stroke. The Makkah BCU has been found to provide the fastest core temperature cooling rates of 0.31°C/min.[27] Today there are multiple options

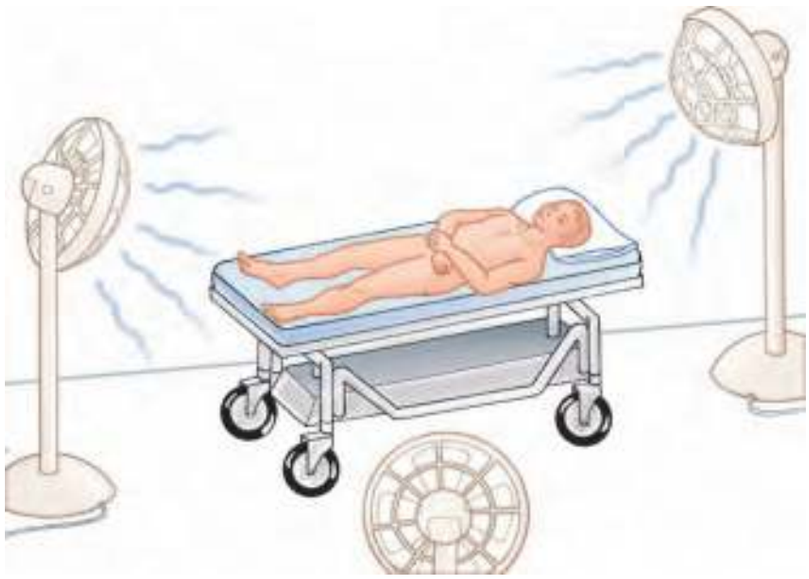

FIGURE 227-2. Large fans at the head, foot, and side of the bed to direct high-flow air currents.

FIGURE 227-3. The first body cooling unit. (Photo courtesy of Science & Society Picture Library.)

of special beds called body cooling units available. They are expensive, require special training, take a significant amount of room for storage, and are usually not available in the ED.

ICE WATER IMMERSION

Immersion in an ice water bath is the most common and best method of cooling exertional heat stroke patients.[33-35] Immerse the patient in a tub of ice water for 10 to 40 minutes (**Figure 227-4**). Water temperature should be between 2°C and 15°C (35°F and 60°F). Cover the patient's body as much as possible. Circulate the ice water vigorously while cooling. Briskly massage the patient's extremities to maintain peripheral circulation and promote heat loss. Monitor the patient's core temperature carefully. Rectal temperatures will decrease approximately 0.16 to 0.21°C/min.[9,27]

Ice water immersion has limited use in the ED.[27,30] Preparing an ice bath can result in a delay in cooling the patient. Contact with the ice water results in intense vasoconstriction which blocks heat exchange, paradoxically increases core body temperature, induces shivering, and is uncomfortable for the patient. Heat transfer from the core to the surface is reduced. The practical issues of impairing access to the patient, monitoring the airway, and initiating resuscitative measures are challenging when the patient is in an ice water bath.[27,30,36] The tub and patient lift device are large, bulky, and not often available in the ED. Multiple caregivers are required to prevent the patient from drowning and to maintain their head above water.

FIGURE 227-4. Ice water immersion to cool a hyperthermic patient.

A simpler and easier method is immersion of just the patient's hands and forearms.[37] This method requires less patient cooperation than full-body immersion. It also allows for full patient monitoring during the cooling process. Another alternative is iced water therapy. The patient is placed supine on a porous stretcher that is positioned on top of a tub of ice water. Ice water is continuously poured onto the patient, ice packs are placed onto the patient (in the axillae, neck, and groin), and major muscles are massaged to increase skin vasodilation.[38]

ICE PACKING

Packing the heat stroke patient in ice is an alternative to ice water immersion. It can result in conductive heat loss that is as effective as evaporative cooling. Patients who are awake and alert do not often tolerate ice packing. Its use may require parenteral sedation. The complications and practical limitations are similar to those of ice water immersion.

Place the undressed patient in the center of the gurney. Completely cover the patient with ice. This technique requires a large quantity of ice which may be unavailable in the ED. Consider placing plastic sheets or trash bags, with the edges curved upward, under the patient to prevent the ice and water from spilling onto the floor. Health-care personnel may otherwise slip on the wet floor, fall, and sustain injuries. Ice should be replenished as soon as there is notable melting present. An easier alternative is to place the patient in a body bag and fill this with ice. Discontinue cooling when the core temperature reaches 39°C or 102°F.

ICE PACKS

The placement of ice packs over select body areas is a more reasonable option than ice water immersion or ice packing. It may be used as the sole cooling method or in conjunction with evaporative cooling. Place plastic bags filled with ice or ice water in the patient's neck, axilla, and groin areas. These regions are near large blood vessels. The ice should be exchanged once notable melting is present. Remove the ice packs when the core temperature reaches 39°C or 102°F.

An alternative to ice packs is the EMCOOLS pads (EMCOOLS, Vienna, Austria).[39-42] These products are surface cooling pads that are nontoxic and designed for easy cooling, mobile cooling, and efficient cooling without the mess of ice water or the need for ice (**Figure 227-5**). The HypoCarbon technology provides better thermal conductivity when compared to water or ice. They are disposable, single-use, and frozen in a regular freezer ahead of being needed. The pads come with a peel-and-stick adhesive on the back for easy contouring to the patient's body. The pads can be used prehospital and in the ED. They are radiologically transparent allowing the cooling to continue while the patient is in the computed tomography scanner, magnetic resonance imaging scanner, or Radiology Department.

ICED PERITONEAL LAVAGE

Peritoneal lavage in hyperthermia has been investigated in canine models but used infrequently in humans. The advantages of this technique include the large surface area exposed to iced saline and direct cooling of the core. This technique is invasive and time consuming, requires significant equipment and expertise, and is associated with significant complications. A case report of a comatose patient with rectal temperature exceeding 42.5°C indicated that iced peritoneal lavage with 2 L of normal saline was successful in decreasing the rectal temperature to 39.4°C.[12] The patient had responded only partially to external evaporative cooling and iced gastric lavage.

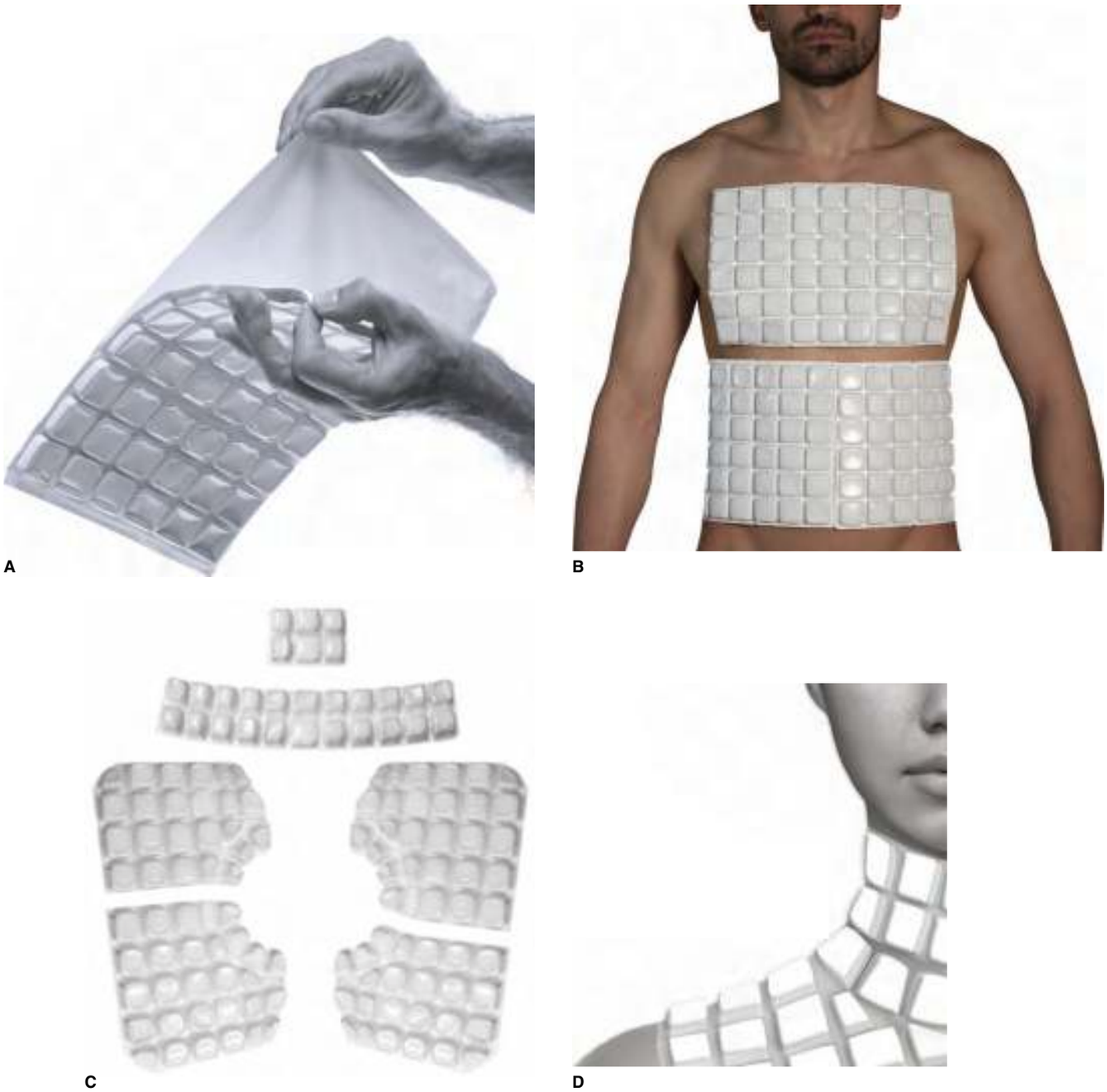

FIGURE 227-5. The EMCOOLS pads. ***A.*** The self-adhesive pads. ***B.*** The pads on a person. ***C.*** Contoured pads for the neck and shoulders. ***D.*** The use of contoured pads. (Photos courtesy of EMCOOLS.)

Place 1 L bags of normal saline or Ringer's lactate solution into an ice water bath to cool down. The technique is similar to that used for warming the hypothermic patient (Chapter 226). Iced peritoneal lavage should be reserved for the patient who is not responding to other methods of cooling.

ICED GASTRIC LAVAGE

Central cooling techniques have not been studied extensively. An alternative core cooling technique may be iced gastric lavage.[24,43] This method has been compared to room-air cooling in an anesthetized canine heat stroke model.[24] Gastric lavage with iced tap water cooled the canine models rapidly and safely. The technique yielded cooling rates five to six times faster than in controls. Significant differences in hemodynamic parameters were noted to return to baseline more quickly in the iced gastric lavage group than in the control group. The technique is similar to that used for warming the hypothermic patient (Chapter 226). Instill 10 mL/kg of iced sterile saline, allow it to dwell for 30 to 60 seconds, and suction out the fluid. Repeat the procedure as necessary. Potential hazards using this technique include aspiration, gastric mucosal injury, electrolyte imbalances with water lavage, water intoxication, and dysrhythmias.[24]

ALTERNATIVE TECHNIQUES

There is active research regarding the induction of therapeutic hypothermia by intravenous cooling to produce a rapid reduction in core temperature. This research has primarily focused on those

with brain injury, post-cardiac arrest, and stroke. This has resulted in the development of a variety of externally applied and internally applied cooling devices (Chapter 225). While not specifically designed to treat hyperthermia, they can easily be used to manage the hyperthermic patient. Some of these devices may be favorable in the prehospital environment or when other supplies are not readily available. The Emergency Physician may be using esophageal cooling in intubated patients in the future.[44] The first esophageal cooling device has been cleared in Canada and Europe (Advance Cooling Therapies, Chicago, IL). This technology is pending U.S. Food and Drug Administration approval at the time of this writing.

ASSESSMENT

Monitor the patient's core body temperature closely with an esophageal, rectal, or urinary temperature probe. Monitor the patient's vital signs, cardiac function, neurologic function, urine output, and laboratory measurements (e.g., arterial blood gases and serum electrolytes). Examination of muscle compartments and all orifices for bleeding is essential. Consider monitoring central venous pressure for assessing volume status to guide fluid resuscitation, especially in patients with heat stroke and limited cardiac reserve. Volume deficits may not be more than 2 to 3 L in patients with classic heat stroke. Hypotension usually responds to intravenous fluids. Use an inotrope, if required, that produces little vasoconstriction such as dobutamine. The use of H_2 blockers may decrease the incidence of gastrointestinal bleeding.[28] Treat rhabdomyolysis aggressively with saline diuresis, alkalinization of the urine, and an infusion of 0.5 gm/kg of mannitol with or without furosemide.[28] Treat seizures with benzodiazepines.[28]

AFTERCARE

Patients with minor heat-related illnesses may be safely discharged home after cooling. Instruct the patient to rest, drink plenty of fluids, and avoid the heat. Discuss with the patient the methods to remain cool if they are exposed to heat. This requires adequate rest, hydration before physical exertion, periods of rest during exertion when the individual can cool off, and adequate oral fluid intake during exertion. Arrange follow-up within 24 hours for a reevaluation.

Admission is prudent in several circumstances. Consider admitting patients with heat exhaustion for 23-hour observation if they have abnormal vital signs or abnormal laboratory investigations or if they remain symptomatic after fluid therapy. Patients with heat stroke should be admitted to an intensive care unit. Permanently altered and unstable thermoregulatory mechanisms have been observed in survivors of heat stroke which increases their susceptibility to future heat illness. **Prevention through education should be a part of every discharge plan.**

COMPLICATIONS

Complications can occur during the cooling of the hyperthermic patient. **Discontinue all cooling techniques when the core body temperature reaches 39°C or 102°F. Continued cooling can result in hypothermia as the core temperature continues to decrease after cooling is discontinued.**[11,45] Monitor the patient to ensure that their core body temperature does not increase. The patient may experience muscle fasciculations, shivering, and agitation during the cooling process. These symptoms can be managed with intravenous benzodiazepines. Ice water immersion and ice packing make patient monitoring and management difficult if not impossible. Iced peritoneal lavage (Chapter 84) and iced gastric lavage (Chapter 77) are invasive procedures associated with significant potential complications.

SUMMARY

The best intervention for heat-related illness is prevention. Heat illness caught early responds to rest, exposure to shade and a breeze, and the ingestion of cool liquids. External evaporative cooling is acknowledged to be the safest means of cooling the hyperthermic patient. The tragedy of heat stroke is that it so frequently strikes highly motivated young individuals under the discipline of work, military training, or sporting endeavors. Heat-illness precautions should be implemented even in temperate zones during the hot summer months.

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228 Autotransfusion

Carlos J. Roldan and Amit Mehta

INTRODUCTION

Trauma is the leading cause of death in children and adults under 44 years of age.[1] Exsanguination plays a significant role in as many as half of these deaths, with hemorrhagic shock the most common cause for potentially preventable death.[2] Hemorrhagic shock is a primary indication for the transfusion of homologous blood products. Transfusions with homologous blood products carry the possibility of associated complications including transfusion reactions, transmission of infectious diseases, and sensitization to antigens.[3] Massive transfusions are associated with the additional complications of acidosis, dilutional coagulopathy, and hypothermia. Transfusion of homologous blood products in the trauma patient has been independently associated with an increase in both morbidity and mortality, particularly when transfusing older stored blood products.[4-6] Blood centers have more than doubled the prices of blood products in recent years due to a decline in blood donors, higher skilled labor costs, and increases in the cost of testing and processing blood.[7] There is evidence that the best replacement for whole blood loss may be fresh whole blood.[8]

Alternative transfusion strategies have been developed for elective surgeries. These include autologous (i.e., acute) normovolemic hemodilution, autologous preoperative donation, autologous priming on bypass initiation during extracorporeal membrane oxygenation (ECMO), and intraoperative cell salvage with autotransfusion.[9] The first two are not possible in the Emergency Department (ED). Cell salvage with autotransfusion represents a viable alternative to autologous transfusion in the ED but has received limited attention in the trauma patient.

An autotransfusion was first performed in the trauma setting by Elmendorf during the First World War on a soldier with a traumatic hemothorax.[10] It was subsequently used sporadically as a lifesaving procedure.[11] A modified autotransfusion for a traumatic hemothorax was described in 1978.[12] This technique was simple, safe, and easy to practice. It was used in approximately 400 patients with a traumatic hemothorax without any noticeable complications. This technique is still applicable to current practice with preliminary studies showing that transfusion of a patient's own blood is safe.[8,13-15]

Many of the patients with a life-threatening hemothorax either die before reaching the ED or experience severe hemodilution that accounts for some deaths in the ED and the Operating Room. **Hypovolemic shock secondary to trauma is the most frequent indication for a massive blood transfusion in the ED. It is an indication for an autotransfusion.** Much of the controversy surrounding practice paradigms centers on the disagreement as to what constitutes the proverbial "perfect procedure."

An autotransfusion may occur in the ED, Operating Room, and Surgical Intensive Care Unit with increasing frequency. **The shed blood is collected, mixed with an anticoagulant, concentrated, washed or filtered, and then returned through an intravenous (IV) line to the patient. Harmful contaminants (e.g., fat, free hemoglobin, and potassium) are removed from the salvaged blood.** This blood is returned through a 40 micron blood filter to collect particulate matter and microthrombi. An autotransfusion in the ED is usually limited to an acute traumatic hemothorax associated with clinically significant hypovolemia. **Utilization of blood from the chest cavity is ideal for autotransfusion since the bacterial contamination of this shed blood is minimal compared to shed blood in other areas.[8]**

INDICATIONS

Consider an autotransfusion with the anticipated loss of at least 20% of the patient's estimated total body blood volume. Consider an autotransfusion if the ED cannot obtain or provide cross-matched compatible blood. It can be used for the patient unwilling to accept homologous blood but willing to accept salvaged blood. The average amount of blood to be autotransfused should be expected to be greater than 1 unit to make this procedure cost effective.[16-18]

There are numerous advantages to an autotransfusion.[15,19] It is convenient and immediately available as a blood product replacement. The blood is already warmed and at, or just below, body temperature. It addresses any special needs of the patient (e.g., religious preferences or a rare blood type). There is no chance of allergic reactions, alloimmunization, disease transmission, febrile reactions, and transfusion reactions. An autotransfusion requires no work on the part of the blood bank, has no compatibility issues, and ensures that the patient receives compatible blood. It conserves homologous

blood products for other patients. An autotransfusion results in the blood having a normal pH, viable platelets, and viable clotting factors when compared to homologous blood supplies. Higher levels of 2,3-diphosphoglycerate in autologous blood results in the easier release of oxygen from hemoglobin in the tissues. There is the positive psychological benefit to the patient when they are informed that they received their own blood.

CONTRAINDICATIONS

There are numerous contraindications to the use of an autotransfusion. The presence of a coagulopathy regardless of its etiology or when it occurs (i.e., in the ED, preoperative, intraoperative, or postoperative) or evidence of disseminated intravascular coagulopathy (DIC) may be more optimally managed with the administration of specific blood component therapy. Evidence or suspicion of serious infectious processes (e.g., mediastinal, pericardial, respiratory, or systemic) precludes the use of an autotransfusion. Do not autotransfuse blood if there is the presence of a malignant neoplasm at the collection site (i.e., abdominal or chest cavity) that can lead to possible contamination of the blood with malignant cells. Do not autotransfuse blood that is collected from a potentially contaminated abdominal or thoracic cavity (e.g., esophageal tear). Do not autotransfuse from a cavity that was irrigated with a solution that can cause lysis of red blood cells prior to blood collection and potentially precipitate end-organ damage if the salvaged product is administered. This includes alcohol, hydrogen peroxide, hypotonic irrigation solutions, povidone iodine antiseptic gels or solutions, or sterile water. Do not autotransfuse blood collected from body cavities that have had the administration of topical thrombin or microfibrillar hemostatic agents. Patients with renal or hepatic failure may not tolerate the increased levels of plasma-free hemoglobin or potassium that occur with an autotransfusion. Relative contraindications include cesarean delivery and sickle cell disease.

EQUIPMENT

- Sterile gloves and gown
- Face mask with a face shield or goggles
- Cap
- Povidone iodine or chlorhexidine solution
- Chest tube drainage apparatus with either a water seal or a dry one-way valve
- In-line autotransfusion system blood collection bag
- Whole blood in-line microaggregate filter for reinfusion (usually 40 microns, but can vary between 20 and 170 microns)[20]
- Nonvented blood compatible IV administration set
- 250 mL bag of sterile normal saline for priming of IV tubing and microaggregate filter prior to blood infusion
- Pressure bag for rapid blood infusion
- Blood warming system if it is anticipated that multiple units of blood will need to be infused to help prevent hypothermia[21-23]
- Citrate anticoagulants
- Tube thoracostomy supplies (Chapter 40)

Anticoagulation for an autotransfusion in the ED setting is generally recommended to help prevent the formation of clots in the autotransfusion system. While the administration of anticoagulants can be beneficial, it is not without risk and its use and dosage are at the discretion of the treating Physician. Heparin anticoagulation is one option. The local administration of heparin can lead to the formation of platelet microaggregates and potentially lead to systematic heparinization which can result in further hemorrhage in a patient who is already bleeding.[24]

Citrate anticoagulants are considered local anticoagulants and are preferred over heparin. Citrate works by binding with calcium ions to inhibit the conversion of fibrinogen into the insoluble protein fibrin, preventing the blood from clotting. Citrate only binds with calcium. It only anticoagulates the blood it is collected with. The liver rapidly metabolizes the citrate once the anticoagulated blood is reinfused.[22]

Citrate phosphate dextrose (CPD) is a commonly used anticoagulant. It is recommended to use 1 mL of CPD for every 7 mL of blood. It is difficult to estimate the amount of blood in a patient's chest cavity. One approach is to instill 60 mL of CPD into the autotransfusion bag, enough to anticoagulate 1 unit of blood. When 1 unit of blood has been collected (i.e., about 500 mL total volume of blood plus CPD), it may be reinfused or additional CPD may be added to continue the collection. Prepare a new collection bag if bloody drainage is ongoing and inject anticoagulant before disconnecting the filled collection bag for reinfusion. The CPD injection may be facilitated by using a volume-control IV chamber. Run the desired amount of CPD into the chamber and then infuse it via the IV tubing to the injection port.

Citrate phosphate dextrose adenine (CPDA) may be used as an anticoagulant at a ratio of 1 mL per 7 mL of blood. Some consider CPDA the anticoagulant of choice for an autotransfusion of a traumatic hemothorax. It provides a reduced pH mixture that is advantageous to the platelets. The CPDA solution maintains the viability of the platelets, reduces clot formation in autotransfusion equipment, plugs the blood filter with microemboli, and clots the IV lines.

PATIENT PREPARATION

Assess the patient for the signs and symptoms of hypovolemia, hypoperfusion, and/or a hemothorax. Describe the procedure, its risks, and benefits to the patient and/or their representative. Obtained written or oral consent, if possible.

This procedure requires strict aseptic technique. Clean any dirt and debris from the patient's chest wall. Don full sterile and personal protective equipment. This includes sterile gloves, a sterile gown, a face mask with an eye shield or goggles, and a cap. Apply povidone iodine or chlorhexidine solution to the skin over the chest wall and allow it to dry. Apply sterile drapes to delineate a sterile field.

Much preparation is required before starting an autotransfusion. The autotransfusion system requires prior assembly. Consider setting up the autotransfusion components for patients who are at high risk for a hemothorax before inserting the chest tube. Otherwise, blood that drains immediately upon chest tube insertion is lost to recovery. **Refer to all manufacturers' directions for use, warnings, and cautions regarding anticoagulant medications, microemboli filters, and IV blood administration sets prior to their use.** Prophylactic broad-spectrum antibiotics may be administered immediately before the chest incision is made for the tube thoracostomy or just prior to the reinfusion. Follow all hospital protocols for blood handling, waste disposal handling, and infection control. Insert a large-bore chest tube (Chapter 51).

TECHNIQUES

There are several manufacturers of chest drainage and autotransfusion systems. The Pleur-Evac System (Teleflex Medical, Research Triangle Park, NC) is one of the commonly used systems in many EDs.[25] The Atrium Chest Drain Autotransfusion System (Atrium Medical Corp., Hudson, NH) is the second of the two more commonly used systems (**Figure 228-1**).[26]

Three different techniques for blood collection are in-line, self-filling, and continuous. The in-line autotransfusion blood bag is a

FIGURE 228-1. The Oasis 3650 chest drain system with autotransfusion blood recovery. (Photo courtesy of Atrium Medical Corp., Hudson, NH.)

standard bag that attaches to the chest drain (**Figure 228-2**). The self-filling autotransfusion bag incorporates a low vacuum that is activated to improve the flow of fluid from the chest drain into the bag (**Figure 228-3**). The final option is the continuous autotransfusion. It is used to directly reinfuse shed autologous blood via a blood compatible infusion pump, a microemboli blood filter, and a nonvented blood compatible IV administration set. This is a true closed system and reduces the potential for contamination. It does not require the clamping and unclamping of tubes, exchanging autotransfusion bags, and the interruption of chest drainage to change autotransfusion bags. It results in less exposure to the patient's blood with a closed system. It saves time and money by not changing autotransfusion bags, filters, and IV tubing. Unfortunately, this closed system is rarely available in the ED and requires additional training for its use.

The technique chosen depends upon the chest tube drainage system used in your institution. Some chest tube drainage systems allow for more than one technique to collect blood for an autotransfusion. The specific port locations and connections will vary slightly among the different manufacturers but the general concepts are relatively universal. The general technique will be described below. The specific steps depend upon the actual equipment available.

Attach the autotransfusion bag to the chest drain. Inject anticoagulant into the collection unit as soon as possible during or before the blood collection if this is required. Collect the blood. Blood may be reinfused when 1 unit of blood has been collected (i.e., about 500 mL of blood plus the volume of anticoagulant). Remove the filled autotransfusion bag and replace it with a new one containing anticoagulant. Discontinue blood collection when it is determined that an autotransfusion is no longer required or there is no further bloody drainage from the chest tube.

FIGURE 228-2. An in-line autotransfusion bag. (Photo courtesy of Atrium Medical Corp., Hudson, NH.)

Reinfuse the blood. Prime the IV blood administration and microemboli blood filter with sterile saline. Invert the autotransfusion bag so that the spike port points upward. Remove the protective cap covering the spike. Insert blood tubing into the spike port by using a constant twisting motion. Attach a microfilter onto the blood tubing. **Remove the air from the autotransfusion bag.** Keep the unit inverted and squeeze all the air from the bag carefully through the filter and the drip-chamber assembly to prevent an air embolus. Close the infusion set clamp. Invert the autotransfusion bag and suspend it from an IV pole with the plastic strap. Open the infusion set and flush the administration line to remove the air. Attach the distal end of the infusion set to the IV line. Infuse the blood using gravity or pressure.

Keep in mind some general principles when performing an autotransfusion. Do not hang or hand carry the autotransfusion bag by its tubing. Use the hanger provided. Replace the spike port cap immediately following removal of the blood filters and IV tubing to prevent air entering the system. The air vent must remain closed when not in use. A new microemboli blood filter

FIGURE 228-3. A self-filling autotransfusion bag. (Photo courtesy of Atrium Medical Corp., Hudson, NH.)

and IV blood tubing must be used for each autotransfusion bag. When using vacuum suction with the collection apparatus it is important to use the lowest level possible to reduce the risk of red blood cell hemolysis during collection.[27] The recommended starting vacuum pressure is 20 mmHg.[21,28]

ASSESSMENT

Assess the patient's cardiopulmonary status and vital signs frequently during the autotransfusion and at least 1 hour after the autotransfusion is complete. Periodically monitor laboratory data to include arterial blood gas values, hematocrit, partial thromboplastin time, platelet count, prothrombin time, rapid thromboelastography (r-TEG), and serum lactate.[29] Monitor and mark the amount and type of drainage from the collection system hourly for the first 8 hours and then every 2 hours as volume loss can cause hypovolemic shock. Evaluate and maintain drainage tube patency every 2 to 4 hours. Mark the drainage level on the outside of the drainage-collection chamber in hourly or shift increments. Document the assessments and laboratory values in the medical record.

AFTERCARE

Monitor the patient's clinical status in response to the infusion during the stay in the ED once the infusion of the autologous blood has started. This will involve monitoring their vital signs and other hemodynamic parameters, including central venous pressure.[17] Monitor laboratory data to assess the success and possible complications of the autotransfusion process. This generally includes electrolytes, renal function, hemoglobin and hematocrit, platelet count, coagulation profile, DIC profile, and r-TEG to assess for a consumptive coagulopathy.[17]

The Emergency Physician, in conjunction with the nursing staff, must maintain the integrity of the autotransfusion system during the infusion of autologous blood. It is necessary to ensure the autotransfusion bag is replaced each time it is filled to prevent overflow of blood into the chest drainage apparatus. Ensure each collection bag has anticoagulant added to it prior to the collection of blood. A new microaggregate filter must be used for each collection bag prior to it being infused. Inspect the blood through the collection bag for evidence of clotting prior to infusing it. Discard the autotransfusion bag and do not reinfuse it if it contains clotted blood or particulate matter. Purge all the air from the collection bag, intravenous tubing, and microaggregate filter to prevent an air embolism. Observe the autotransfusion to ensure that the entire contents of the collection bag are not allowed to infuse through the microaggregate filter and intravenous tubing. The transfusion of shed blood collected under posttraumatic conditions should begin within 6 hours of initiating the collection at the longest to reduce the risk of bacterial overgrowth and minimize damage to the blood components during storage.[30]

COMPLICATIONS

The complications from an autotransfusion are clinically insignificant if the proper technique is followed and if less than 3000 mL of blood is reinfused. Overall, the complications are categorized as hematologic and nonhematologic. Refer to Chapter 51 regarding the complications associated with a tube thoracostomy.

HEMATOLOGIC

A coagulopathy may occur in trauma patients following an autotransfusion. The autotransfusion has not been overwhelmingly proven to be the etiology.[3] These patients are more likely to have other independent risk factors for a coagulopathy (e.g., acidosis and hypothermia).[31] This makes it difficult to determine the contribution of the autotransfusion to the development of a coagulopathy.[31] DIC is a rare complication that should be anticipated and treated accordingly. A dilutional coagulopathy may occur when greater than 3500 mL of autologous blood is transfused. Treatment includes administration of fresh frozen plasma and platelets to compensate for the proportional decrease in platelets and fibrinogen.[32] Hemolysis of red blood cells can be secondary to mechanical factors during the collection process, the use of hypotonic irrigation fluids, excessive suction pressure, aspiration of clotted blood, or the use of hemolytic solutions (e.g., povidone iodine, hydrogen peroxide, or alcohol) to collect blood.[33,34] Hemolysis can also occur secondary to the prolonged exposure of red blood cells to the serosal linings of

traumatized body cavities.[35] Thrombus formation may occur from increased plasma-free hemoglobin that promotes a procoagulant effect and increases the risk of free hemoglobin precipitation in the renal tubules.[36,37]

NONHEMATOLOGIC

Particulate embolism has been reported to occur during and after an autotransfusion. Most components of the equipment are designed for single use. Re-sterilization or re-use may compromise the structural integrity and lead to device failure. An air embolism may result from inadequate use of the IV set or the microemboli blood filter. An air embolism has also been associated with the reinfusion of the entire autotransfusion blood bag contents that contain residual air and a pressurized infusion of the autotransfusion bag with the air vents open. Citrate toxicity and myocardial depression may occur after the rapid infusion of citrate anticoagulated blood. The clinical manifestations of citrate toxicity include a perioral tingling sensation followed by abdominal cramping and cardiac dysrhythmias. Citrate toxicity can be prevented by ensuring that the ratio is not greater than 1:5 of the citrate anticoagulant to blood. Sepsis in patients with an isolated hemothorax is considered only a minimal risk. Sepsis has been associated with the difficulty that exists in maintaining a completely sterile environment in patients receiving an autotransfusion.[38-40]

SUMMARY

An autotransfusion is a basic technique that is possible in the ED. It is not the standard of care under all settings. The procedure requires familiarity with the equipment, continuing education, and quality control. The successful incorporation of this procedure likely requires the establishment of guidelines and protocols by a multidisciplinary group including representation from Emergency Medicine and Trauma Surgery. This may be the reason it is underutilized in many EDs. Anticipation of which patients would benefit from this technique might be problematic and time consuming, especially when assembling equipment that may not be used frequently. Proper use of the devices can be advantageous to avoid the multiple complications associated to hypovolemic shock in trauma patients. The low risk of complications related to this technique makes an autotransfusion a viable option to a homologous transfusion.

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229 Helmet Removal

Ashley N. Sanello and Atilla B. Uner

INTRODUCTION

The helmeted person who becomes injured presents unique challenges to athletic trainers, prehospital healthcare providers, and Emergency Department personnel in providing the initial stabilization and management. Greater numbers of people are wearing helmets due to the helmet laws and increasing public awareness for the prevention of head injuries associated with recreational and athletic activities. This practice will limit the most severe outcomes from head trauma. However, the helmeted patient is not immune from life-threatening head and neck injuries. **Secondary injury due to improper helmet removal can adversely affect patient outcome.**[1,2]

Helmets vary in size, type, and accessories based on the user's activity (**Figure 229-1**). They consist of a hard plastic, polycarbonate, and/or fiberglass shell over either a layer of foam covered by material, inflatable air bladders, or both. Bicyclists, kayakers, roller bladers, skateboarders, and skaters wear simple skull helmets. These helmets cover the top of the skull like a hat and have a strap that is snapped or clipped under the chin to maintain the helmet in position. Football, lacrosse, and hockey players use open-faced helmets. These may have clear visors and/or face cages whose bases are screwed into the helmets. Motorcyclists and racers often wear full-face helmets with or without retractable or removable visors.

Athletes playing football and hockey wear protective shoulder padding in addition to helmets. Because of the shoulder padding, their cervical spines are more adequately stabilized in comparison to those of helmeted motorcyclists without shoulder padding.[3,4] Helmet removal with and without shoulder pad removal has been shown to result in head and cervical spine movement.[5,6] This can increase the risk of spinal cord damage in the helmeted athlete with a cervical spine injury.[3,4]

Current recommendations for sports-related helmet removal, such as in ice hockey and football, are to leave the helmet in place until the patient arrives to an Emergency Department or Trauma Unit.[3-5,7-13] **The exception permitting the removal of an athlete's helmet in the field is when the helmet significantly delays lifesaving measures or if airway access is obstructed.**[3,4,7,14] This may occur in the unconscious and/or apneic patient. Prehospital healthcare providers should be able to maintain an adequate airway, stabilize a patient's cervical spine, and control associated hemorrhage with removal of only the face plate, face guard, and chin strap of the helmet.[7,8,12,13]

ANATOMY AND PATHOPHYSIOLOGY

The type and fit of protective equipment, the mechanism of injury, the patient's age, and physical development all influence cervical spine injury. There is a greater risk of injury when the helmet is too loose. It has been noted that most people wear helmets that are too large for their heads. Inertia is defined as a body's ability to resist change of position and motion. A 4 pound helmet can exert 200 pounds of force on the wearer's head and neck when an impact occurs at 50 mph. Injury is often more serious when inertial forces cause excessive extension and flexion of the cervical spine without adequate protection for the lower head and neck.[8,15]

The anatomic location of cervical spine injuries tends to differ between children and adults. The immature pediatric spine is more susceptible to flexion and extension injuries in the upper cervical (i.e., C2 to C3) segments due to the child's proportionally larger head size, shorter neck with immature development, weak neck muscles, and the neck's higher degree of flexion and extension.[8,15,16] The relative amount of cartilaginous tissue in comparison to bone makes the pediatric patient more prone to spinal cord injury when subjected to high inertial forces. These injuries may be more difficult to detect on plain radiographs, as cartilaginous injuries are radiolucent.[15,16]

Similar conditions of disproportionate head-to-neck size are artificially created in the helmeted patient. The motorcycle helmet will often cause hyperflexion of the neck. Flexion and extension injuries without adequate lower neck protection result in an increased incidence of upper cervical spine (i.e., atlantoaxial, C1, and C2) injuries in comparison to lower cervical spine segments.[16,17] There is a greater incidence of lower cervical spine (i.e., C5 and C6) injury among football and hockey players because the shoulder padding offers support to the lower head and upper neck. It decreases the amount of flexion and extension at the time of impact.[17] Head and neck restraint systems, such as the HANS (Head and Neck Support) device (HANS Performance Products, New Braunfels, TX) were developed for professional motorsport racers to prevent flexion-distraction injuries during high-velocity impacts. Since these devices are now required for use by many professional racing drivers and commercial drivers, they have significantly decreased fatal craniovertebral junction injuries.[18] The removal of these devices depends on the brand the patient is wearing, but all require the device to be unclipped from the helmet and cervical stabilization held while the device is removed.

Flexion and distraction while removing a helmet may cause spinal cord compression as demonstrated in unstable C1 to C2 injuries with helmet removal in cadaveric models.[17] Similar unfavorable results were demonstrated with neck flexion and extension during helmet and shoulder pad removal.[17] The properly fitting helmet is tight enough that a significant force must be applied to remove it in the field thus increasing the chance of cervical spine movement. **Apply proper in-line cervical immobilization before helmet removal or medical intervention while avoiding in-line traction. In-line traction increases the risk of subluxation or distraction at the site of injury.**

It is recommended that the helmet and padding in an athlete not be removed in the field. Overzealous manipulation of the patient or improper helmet and/or padding removal can complicate an underlying injury. The helmet can be secured to a long backboard if one is used. Since many prehospital agencies no longer use backboards, another way to secure the helmet is to place

FIGURE 229-1. Types of helmets. ***A.*** Simple skull helmet. ***B.*** Football helmet. ***C.*** Partial face covering helmet. ***D.*** Full face covering helmet without a face shield. ***E.*** Full face covering helmet with a face shield.

blankets or towels next to the head and tape them to the helmet. **If shoulder pads or helmets are removed in the field, the posterior aspect of the neck and shoulders must be adequately supported while in-line immobilization is maintained to avoid further spinal cord injury. Cervical immobilization, with and without helmet removal, is best accomplished with at least two people to assure that the patient's head and neck remain stable.**

INDICATIONS

Sports-related injuries can be assessed and stabilized in the field without removing the helmet or shoulder pads.[5,9-13] Once the patient is in the Emergency Department, it is best to remove the helmet to obtain an exam and possible imaging. The patient can sometimes be initially managed and radiographed and the cervical spine radiographically "cleared" prior to removal of the helmet in the Emergency Department.[1] Plain radiographs may be inadequate to visualize the bony structures if the helmet and pads remain on the patient.[19,20] If attempts to remove the helmet cause severe neck pain or focal neurologic symptoms, a computed tomography scan can be done while the helmet is on to evaluate the cervical spine as most helmets and pads are nonmetallic.[5,21]

Helmet removal is warranted emergently in patients who are apneic, in patients without an adequately sustainable airway, if the helmet interferes with airway management, if cardiopulmonary resuscitation (CPR) is required, and in patients who have uncontrollable hemorrhage.[14,22,23] If airway access is required, it is safer to only remove the face mask in the prehospital setting in a potential spine injury than to remove the entire helmet.[12,13] Helmets that are loose fitting or that do not adequately stabilize the head and neck should be removed. Other indications to remove a helmet include if it prevents proper patient immobilization or if the face mask cannot be removed. **When a helmet is removed, any pads and neck support devices attached to the pads must be removed to maintain the cervical spine in a neutral position and prevent secondary injury.[24]**

CONTRAINDICATIONS

Adhere to strict cervical spinal cord injury precautions throughout the prehospital period. Do not remove the helmet unless absolutely necessary.[25] It is not possible to completely exclude a spinal cord injury or vertebral fractures in the field.

Consider delaying the removal until radiographically cleared if the patient is unconscious, has an abnormal mental status, is under the influence of ethanol or drugs, is intoxicated or potentially intoxicated, or has distracting injuries; if there is evidence of a spinal cord injury; if a complete physical examination cannot be performed; or if the patient has neck pain. The only exception is if the patient's airway and breathing cannot be supported with the helmet in place.

EQUIPMENT

- Adjustable cervical collar
- Scissors or shears suitable to cut the face mask
- Cast saw with spare blades
- Screwdrivers, flat head and/or Phillips head
- Face mask removal tools
- Towels

PATIENT PREPARATION

A complete examination, including a neurologic examination, should be done prior to the removal of a helmet. Explain the removal procedure and its possible complications to the patient and/or their representative. Explain all the potential risks involved in helmet removal including the possibility of worsening an underlying spinal cord injury. The risk of permanent central nervous system damage should be stated as well as the fact that the precautions to avoid such injury are not foolproof but will be observed as carefully as possible. Answer any questions or concerns of the patient or their representative. **If the patient is awake and alert, instruct them to tell you immediately if they experience numbness, pain, paresthesias, or tingling during the removal procedure.**

Remove any removable parts if they were not detached from the helmet prior to arrival in the Emergency Department. The face mask, face shield, and/or visor can often be removed with a flat head screwdriver, a Phillips head screwdriver, or an electric screwdriver (i.e., battery operated or rechargeable).[26-30] If the screws cannot be removed because they spin in place or are rusted, cut the plastic clips holding the face mask onto the helmet. Some face masks can be removed easily if they are attached to the helmet with a quick release system.[31-33] A variety of tools are available to aid in face mask removal.[34] The hospital's maintenance department can provide an anvil pruner, bolt cutters, or PVC pipe cutter. Commercial devices have been designed to remove a face mask. These commercial devices are not often available in the Emergency Department.

Carefully look for any damage to the patient's helmet. External damage to the helmet may be the only initial indication of the severity of the impact. Inspect the integrity of the helmet shell for fit and structural breaks. The patient may be wearing eyeglasses or sunglasses under the helmet. Remove the glasses before helmet removal begins. Contact lenses may remain in place until the helmet is removed.

The helmet will cause flexion of the cervical spine when a patient is placed supine. This flexion is even more pronounced in a child due to their large occiput. Place rolled towels or sheets under the patient's shoulders to eliminate the excessive flexion of the cervical spine. Such flexion is minimal and requires no towels under the shoulders if the patient is wearing shoulder pads in addition to a helmet.

TECHNIQUES

MANUAL ONE-PERSON TECHNIQUE

Helmet removal can be performed by one or two people. **Reserve the one-person technique for extreme situations as the two-person technique is preferred (Figure 229-2).** With only one person present, it is necessary to pad voids behind the patient's neck and shoulders (**Figure 229-2*A***). **The goal is to keep the head and neck in the neutral position as much as possible while removing the helmet.**

Stand above the patient's head looking down toward their feet. Lift up the face mask, if present, and undo the helmet chin strap. Place the heels of both hands on the sides of the helmet (**Figure 229-2*B***). Insert the index, middle, ring, and little fingers into the space between the patient's head and the helmet. **Gently spread open the helmet with both hands to create clearance between the helmet padding and the patient's face and ears (Figure 229-2*B*).** Slowly and gently remove the helmet (**Figure 229-2*C***). **Stop removing the helmet immediately if the patient experiences pain or neurologic symptoms and use the two-person or cast saw technique.** If the helmet is of the full-face type, the lower part of the helmet may get hung up on the nose. If that occurs, gently tilt the helmet posteriorly and continue to pull it off until the nose is cleared. Return the helmet to a neutral position after the nose is cleared. **Avoid cervical spine hyperextension during this stage of helmet removal.**

Stop removing the helmet when the bottom of the helmet is just above the patient's occiput. Move the fingers of both hands onto

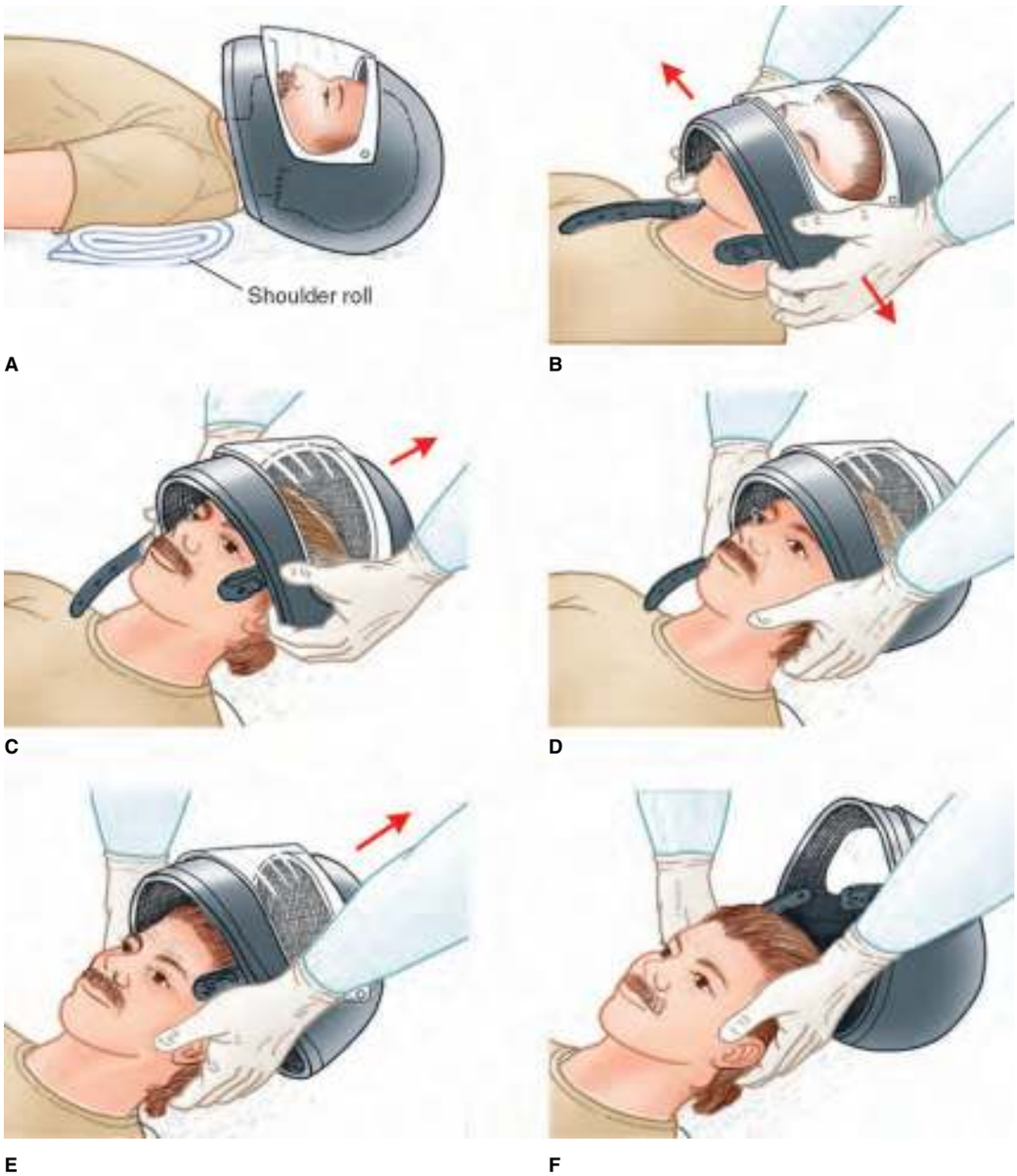

FIGURE 229-2. The one-person helmet removal technique. ***A.*** Patient positioning. ***B.*** The base of the helmet is spread open. ***C.*** The helmet is removed to expose the occiput. ***D.*** In-line immobilization is applied with the fingers. ***E.*** The helmet is pushed off with the heels of the hands. ***F.*** The helmet is removed while in-line immobilization is maintained.

the patient's occiput while keeping the heels of both hands on the helmet (**Figure 229-2*D***). The fingers will now maintain stabilization and immobilization of the head and neck. Push the helmet off the patient's head using the heels of both hands while maintaining immobilization of the patient's head and neck (**Figures 229-2*E* and 229-2*F***). Remove the shoulder pads if present. Maintain the head manually in line until immobilization can be accomplished with a cervical collar, backboard, sandbags, and/or tape.[6,22,35]

MANUAL TWO-PERSON TECHNIQUE

The two-person technique has the same goals as the one-person technique (**Figure 229-3**). The assistant provides in-line immobilization while the helmet is removed.[6,22,35] Instruct an assistant to stand at the patient's side at the level of the patient's neck and shoulders. Lift up the face mask, if present, and undo the chin strap. Place one of the assistant's hands under the patient's occiput and upper neck. Place the assistant's other hand under the patient's mandible so that the mental process (i.e., the patient's chin) rests in the assistant's first web space (**Figure 229-3*B***). Place the tip of the assistant's thumb on the angle of the patient's mandible. Place the index and long finger of the assistant's hand on the contralateral angle of the patient's mandible. Instruct the assistant to hold this position and maintain in-line immobilization of the patient's head and neck.[6,36]

The procedure to remove the helmet is similar to that described above for the one-person technique. Stand above the patient's head looking down toward their feet. Place the heels of both hands on the sides of the helmet (**Figure 229-3*B***). Insert the index, middle, ring, and little fingers into the space between the patient's head and the helmet. **Gently spread open the helmet with both hands to create clearance between the helmet padding and the patient's face and ears (Figure 229-3*B*).** Slowly and gently remove the helmet (**Figure 229-3*C***). **Stop removing the helmet immediately if the patient experiences pain or neurologic symptoms and**

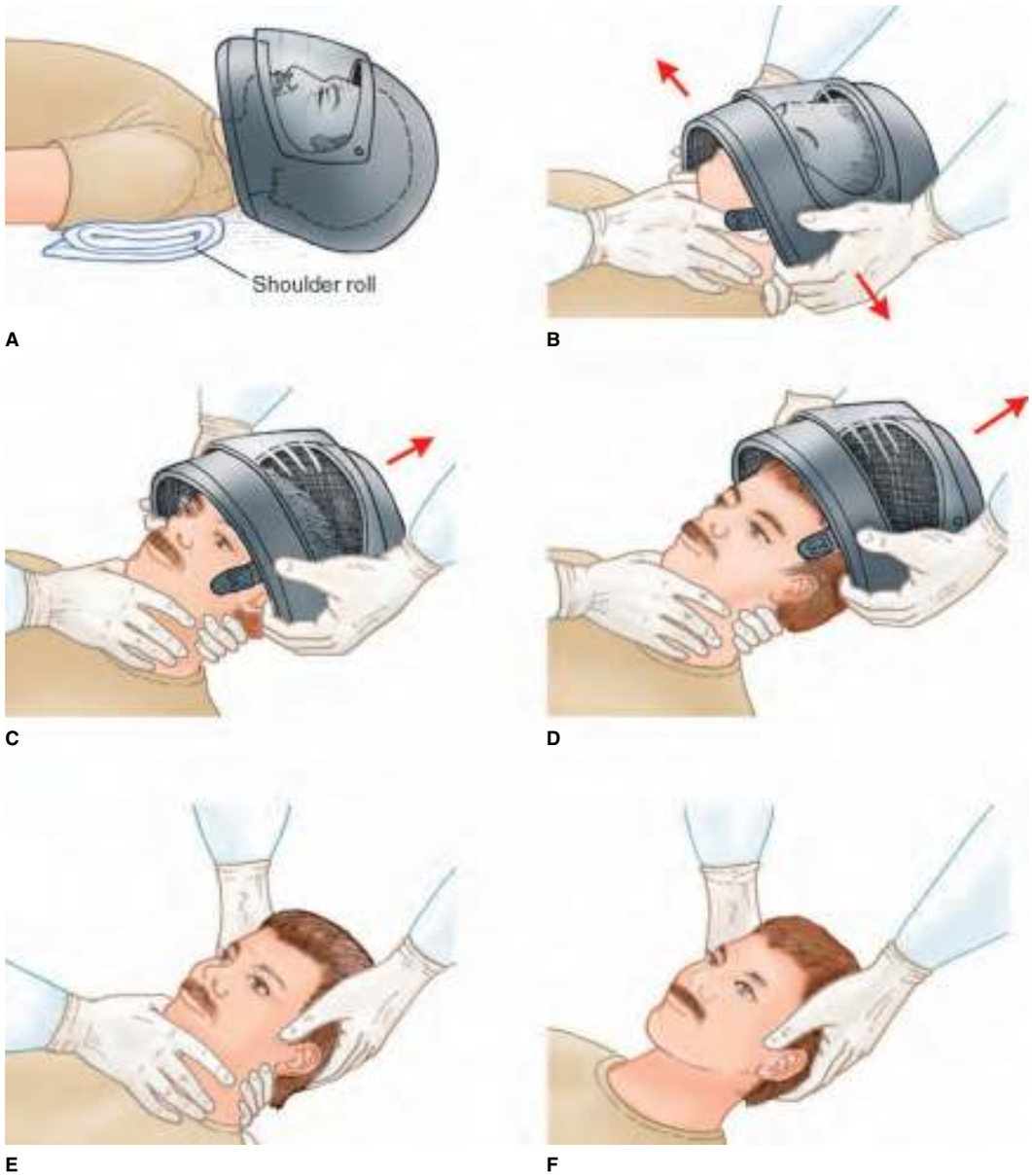

FIGURE 229-3. The two-person helmet removal technique. ***A.*** Patient positioning. ***B.*** An assistant provides in-line immobilization. The base of the helmet is spread open. ***C.*** Remove the helmet to clear the nose. ***D.*** The helmet is removed while the assistant continues in-line immobilization. ***E.*** The Emergency Physician provides in-line immobilization in addition to the assistant. ***F.*** The assistant has removed their hands and the Emergency Physician maintains the in-line immobilization.

use the cast saw technique. If the helmet is of the full-face type, the lower part of the helmet may get hung up on the nose. If that occurs, gently tilt the helmet posteriorly and continue to pull it off until the nose is cleared. Return the helmet to a neutral position after the nose is cleared. **Avoid cervical spine hyperextension during this stage of helmet removal.** Completely remove the helmet (**Figure 229-3*D***).

The Emergency Physician's hands should be applied to the patient's head and neck to maintain the in-line immobilization (**Figure 229-3*E***). Instruct the assistant to remove their hands (**Figure 229-3*F***). Remove the shoulder pads if present. Instruct the assistant to apply a cervical collar and secure the patient's head. The Emergency Physician can now release hold of the patient. Maintain the head manually in line until immobilization can be accomplished with a cervical collar, backboard, sandbags, and/or tape.[6,22,35]

CAST SAW BIVALVE TECHNIQUE

A cast saw (**Figure 229-4**) can effectively be used to cut through the shells of most helmets (**Figures 229-5 and 229-6**). This technique allows for removal of the helmet without cervical spine movement.[1,2] **Cut the helmet off any patient with a neurologic injury, abnormal cervical spine radiographs, if there is a concern for head and neck trauma, or if the helmet is extremely snug-fitting to prevent cervical spine movement.** This technique requires the presence of two providers.[1,2]

Protect the patient from secondary injury from the cast saw, the helmet-cutting process, and the sharp edges of the helmet. Instruct the responsive patient to keep their eyes closed throughout the cutting and removal process to prevent corneal foreign bodies, corneal abrasions, and globe penetration. Place a moist towel over

FIGURE 229-4. The cast saw.

the patient's face for protection. Explain that there may be sounds of material or Velcro being torn or pulled with the helmet and not to be alarmed. Warn the patient about the noise associated with cutting the helmet with the cast saw. Protect the patient's eyes from exposure to foreign body penetration or abrasions if they are unresponsive. This includes taping the eyelids closed and placing a moist towel over their face.

Instruct the assistant, as described above for the two-person technique, to stabilize and immobilize the patient's head and neck (**Figure 229-5*B***). Cut the helmet from ear to ear in a coronal plane with the cast saw (**Figure 229-5*C***). **Carefully cut the padding and straps with a heavy scissors. Use care to not cut the patient's ears as well not force the tips of the scissors into their ears or scalp.** Remove the anterior portion of the helmet (**Figure 229-5*D***). Gently slide out the posterior portion of the helmet while the assistant maintains in-line immobilization of the head and neck (**Figure 229-5*E***). The Emergency Physician should then support the patient's head and maintain in-line immobilization (**Figure 229-5*F***). Instruct the assistant to slowly and carefully remove their hands (**Figure 229-5*G***). Remove the shoulder pads if present. Instruct the assistant to apply a cervical collar and secure the patient's head. The Emergency Physician can now release hold of the patient.

CAST SAW-FACE MASK REMOVAL TECHNIQUE

The cast saw-face mask removal technique can be used to remove the front part of a full-face helmet to secure the airway and then continue to remove the helmet without cervical spine movement (**Figure 229-6**).[22,35,37] This procedure also requires two providers.[21,22,35,37]

This technique allows for removal of the helmet without cervical spine movement.[1,2] **Cut the helmet off any patient with a neurologic injury or abnormal cervical spine radiographs, if there is a concern for head and neck trauma, or if the helmet is extremely snug-fitting to prevent cervical spine movement.** This technique requires the presence of two providers.[1,2] Push the plastic eye shield up and unclip the chin strap.

Protect the patient from secondary injury from the cast saw, the helmet-cutting process, and the sharp edges of the helmet. Instruct the responsive patient to keep their eyes closed throughout the cutting and removal process to prevent corneal foreign bodies, corneal abrasions, and globe penetration. Place a moist towel over the patient's face for protection. Explain that there may be sounds of material or Velcro being torn or pulled with the helmet and not to be alarmed. Warn the patient about the noise associated with cutting the helmet with the cast saw. Protect the patient's eyes from exposure to foreign body penetration or abrasions if they are unresponsive. This includes taping the eyelids closed and placing a moist towel over their face (**Figure 229-6*B***).

Instruct the assistant, as described above, to stabilize and immobilize the patient's head and neck. Hold the cast saw with the dominant hand while the nondominant hand is placed between the inner helmet and the patient's face (**Figure 229-6*C***). This protects the patient's face and helps gauge the depth to cut. Cut the helmet starting at one side of the face mask (**Figure 229-6*C***). Repeat this process and cut the second side of the face mask (**Figure 229-6*D***). Remove the hard plastic front of the helmet, which includes the face mask, to expose the foam and fabric layer underneath. Use trauma sheers to cut through the fabric and foam of the face mask to expose the patient's face (**Figure 229-6*E***) After the airway is secure and/or spine imaging has taken place, if necessary, remove the rest of the helmet.

Gently spread open the cut ends of the helmet with both hands to create clearance between the helmet padding and the patient's face and ears (Figure 229-6*F*). Slowly and gently remove the helmet. **Stop removing the helmet immediately if the patient experiences pain or neurologic symptoms. Avoid cervical spine hyperextension during this stage of helmet removal.** Completely remove the helmet.

The Emergency Physician's hands should be applied to the patient's head and neck to maintain the in-line immobilization (**Figure 229-5*F***). Instruct the assistant to remove their hands (**Figure 229-5*G***). Remove the shoulder pads if present. Instruct the assistant to apply a cervical collar and secure the patient's head. The Emergency Physician can now release hold of the patient. Maintain the head manually in line until immobilization can be accomplished with a cervical collar, backboard, sandbags, and/or tape.[6,22,35]

ALTERNATIVE TECHNIQUES

There are some devices that are easy to install, low cost, and often recommended or required for use by many professional racing associations and organizations. An example is the Eject Helmet Removal System (Stilo USA, Mooresville, NC). The safety of using this device compared to manual helmet removal seems to be comparative, but few studies have been done.[38] The device consists of an accordion-folded airbag, tubing that exits the airbag and clips onto the edge of the helmet for easy access, and an insufflation bulb (**Figure 229-7**).

It is available in two forms. The first is the individual unit that can be purchased and installed in the helmet by the person wearing the helmet. This is usually only done in professional racing. The second is the EMS Kit for prehospital and hospital personnel to use if the device is not preinstalled by the helmet wearer. The EMS Kit contains several of the devices, insufflation bulbs, an insertion tool, and CO_2 cartridges that can be used instead of the insufflation bulb.

The device is simple to use. One person is required to provide in-line immobilization to the cervical spine. A second person is required to activate the device and remove the helmet. If the device is preinstalled in the helmet, locate the tubing insufflation port. If it is not already preinstalled, then use the insertion device to place the airbag under the helmet. Attach the insufflator bulb (**Figure 229-7*A***). Squeeze the insufflator bulb to inflate the airbag. Alternatively, attach the CO_2 cartridge to inflate the airbag. As the airbag inflates it will elevate and lift the helmet off the patient's head (**Figures 229-7*B* and 229-7*C***). While one person maintains cervical spine immobilization, the other person should remove the helmet followed by removal of the pads if present. Apply a cervical collar and secure the patient's head.

FIGURE 229-5. The cast saw bivalve removal technique. ***A.*** Patient positioning. ***B.*** An assistant provides in-line immobilization. ***C.*** The helmet has been cut with a cast saw. ***D.*** The anterior portion of the helmet is removed. ***E.*** The posterior portion of the helmet is removed. ***F.*** The Emergency Physician provides in-line immobilization in addition to the assistant. ***G.*** The assistant has removed their hands and the Emergency Physician maintains the in-line immobilization.

FIGURE 229-6. The cast saw-face mask removal technique. ***A.*** Patient positioning. ***B.*** A protective cloth has been placed over the patient's face and eyes. ***C.*** The cast saw is used to cut the first side of the helmet. ***D.*** The cast saw is used to cut the second side of the helmet. ***E.*** Trauma sheers are used to cut the fabric portions of the cut helmet. ***F.*** The cut sides of the face mask are extended out and the remainder of the helmet is slipped off.

FIGURE 229-7. The Eject Helmet Removal System. ***A.*** The system in place between the helmet and the patient's head. ***B.*** Inflation of the airbag releases the helmet and begins to elevate it off the head. ***C.*** The airbag is further inflated until the helmet is removed from the head.

ASSESSMENT

Reassess the patient after removing the helmet and securing their head and neck.[39] Examine the patient for secondary injury due to the helmet removal procedure such as abrasions, lacerations, and corneal foreign bodies. Perform and document a neurologic examination to ensure there is no change from the initial examination and that the helmet removal procedure did not result in an iatrogenic injury.

AFTERCARE

Clean and vacuum any helmet debris if the helmet was cut with the cast saw. This will prevent any of this material from getting into the patient's eyes or nose as well as preventing them from inhaling the debris.

COMPLICATIONS

The most concerning aspect of helmet removal is adequate immobilization of the patient's head and neck during the procedure. Gross movement of the head and neck can result in displacement of fractures, spinal cord injury, or exacerbation of a partial spinal cord injury. **Avoid any movement of the head and neck when there is any suspicion of a spinal cord injury or severe head trauma.** Some movement of the patient's head or neck is likely to occur during helmet removal. **Minimizing the amount of motion is paramount.**

Secondary injury to the ears, eyes, and nose can be avoided by using careful technique. Spreading open the sides of the helmet before removal in the one-person or two-person techniques will eliminate traction injuries to the scalp and ears.

A separate set of complications is applicable to the cast saw technique. Proper education of the patient is required to prevent them from moving because of the noise and vibration associated with this technique. Covering the patient's eyes and face with a moist towel will prevent corneal abrasions, corneal foreign bodies, oral and nasal foreign bodies, and inhalation of any debris. The cut edges of the helmet can be sharp. Careful removal will prevent cutting the patient's ears or scalp.

SUMMARY

The Emergency Physician who cares for trauma victims must be aware of the different types of helmets available and the means to safely remove them. Proper assessment of the helmeted patient will determine the need for emergent helmet removal. A thorough physical assessment and radiographic studies may take place prior to removing the helmet if the patient is stable upon the initial assessment. By using proper techniques, helmets may safely be removed without causing secondary injury.

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230 Pneumatic Antishock Garment (MAST Trousers)

Evan J. Weiner

INTRODUCTION

The pneumatic antishock garment (PASG) is a device aimed at treating hypovolemic shock in the prehospital setting. Its primary purpose has been in the transport of patients with hemorrhagic shock due to trauma. It has been used in cases of nontraumatic hemorrhage (i.e., ruptured ectopic pregnancy and ruptured aortic aneurysm). The PASG is more commonly known as the Military Antishock Trouser, Medical Antishock Trouser, or MAST (**Figure 230-1**). The PASG was first invented during the Vietnam War.[1,2] An earlier version called the "G-suit" dates to 1903 and served mainly to prevent postural hypotension and venous pooling in pilots.[3]

The evidence supporting PASG use is mixed. Initial reports described successes in increasing systemic blood pressure and controlling intra-abdominal hemorrhage in trauma patients with hypovolemic shock.[4,5] A large retrospective analysis of 1120 PASG-applied patients in 1983 revealed a 24-hour survival rate of 73%.[6] Approximately 84% of survivors had an increase in systolic blood pressure, a decrease in heart rate, or evidence of enhanced tissue perfusion. **Prospective studies have concluded that PASG provides no advantage to survival.[7,8] A Cochrane review concluded that there is no evidence to suggest that PASG reduces mortality or hospitalization.[9] The Advanced Trauma Life Support (ATLS) course has previously supported its use "to control bleeding from pelvis or lower extremity fractures" but states that its use "should not interfere with rapid reestablishment of intravascular volume by intravenous fluid infusion." It also cautions against prolonged application leading to compartment syndrome.[10] PASG is not part of the current ATLS protocol.[11]**

Another garment called the Non-Pneumatic Antishock Garment (NASG) has been recommended by the World Health Organization (**Figure 230-2**). It acts as a temporizing measure for postpartum obstetrical hemorrhage in poorly resourced areas of the world.[12]

ANATOMY AND PATHOPHYSIOLOGY

The antishock garment works by several proposed mechanisms. The garment provides circumferential pressure on the abdomen and lower extremities. It can enhance venous return from the lower extremities

FIGURE 230-1. An example of a PASG.

A

B

FIGURE 230-2. An example of an NASG. ***A.*** The garment opened. (Photo courtesy of LifeWrap NASG.) ***B.*** The garment on a patient. (Used with permission from reference 22.)

and acts as an autotransfusion. The antishock garment produces a small autotransfusion effect. It can increase the total peripheral vascular resistance (PVR). The increased PVR provides the most blood pressure elevation. Its actions on the lower extremity vasculature lead to increased preload, afterload, stroke volume, and cardiac output.[13,14] The antishock garment shunts the blood to the brain, heart, and vital organs. One study proposes that the antishock garment's blood pressure elevation effect may be due to sympathetic nervous system activation from the abdominal but not leg compression.[15] The antishock garment can tamponade hemorrhage. The antishock garment can be useful for applying direct external pressure on pelvic or lower extremity bleeding. It puts indirect pressure on deep structures. It may stabilize fractures (i.e., pelvic and femur) and tamponade bleeding.[16] It has been shown to decrease blood flow to areas distal to the renal vessels.[17] The antishock garment prevents bleeding into hematomas and the formation of hematomas.

INDICATIONS

There are no absolute indications for the use of PASG. Its main purpose is to function as an adjunct for uncompensated hypovolemic shock. PASG may be helpful in the situations of traumatic hypotension due to blunt abdominal trauma, major pelvic fractures, femur fractures, and lower extremity hemorrhage. It may be a reasonable adjunct in the prolonged prehospital transport of a hypotensive patient. The device assists in splinting pelvic and lower extremity fractures. Controversy exists regarding whether the PASG is useful in thoracic and penetrating abdominal trauma.

The antishock garment may be used for medical indications. It can be useful and effective for hypotension due to a ruptured aortic aneurysm.[18] Other uses include anaphylaxis, gynecologic hemorrhage, postpartum hemorrhage, ruptured ectopic pregnancy, septic shock, and spinal shock.[19-22]

CONTRAINDICATIONS

Absolute contraindications to use of PASG include congestive heart failure and pulmonary edema due to increased afterload. Suspected or actual lower thoracic spine and lumbar spine fractures can be made worse by the application and use of an antishock garment. A number of relative contraindications exist including cardiogenic shock, cardiac tamponade, myocardial infarction, penetrating thoracic trauma, diaphragmatic injury, lower extremity compartment syndrome, an impaled foreign body, abdominal evisceration, spinal instability, hemorrhage above the garment, and pregnancy (i.e., unless the PASG is not inflated or using a NASG).

EQUIPMENT

The most commonly available PASG is the MAST (**Figure 230-1**). The suit is made of three independently inflatable compartments (i.e., two leg compartments and one abdominal compartment) held together with Velcro. Newer versions are made of neoprene or urethane for prolonged storage between uses. The compartments are inflated with a foot pump and provide up to 104 mmHg in counterpressure regulated by individual pop-off valves. Each compartment has a stopcock to regulate the flow of air. The garment is available in three models and two sizes (i.e., adult and pediatric). One of the models is transparent and allows providers to monitor injury sites and gain access to insert femoral venous access (**Figure 230-3**). The antishock garment can be applied quickly and within 60 seconds.

The NASG is simple to apply (**Figure 230-2**). It is lightweight (1.5 kg), made of neoprene, and has Velcro closures. It is mostly used in low-income countries for postpartum hemorrhage.[21,22] The abdominal panel stretches and a foam ball compresses the abdomen to reduce blood flow (**Figure 230-2**). The design allows access to the perineum for procedures (e.g., bladder catheterization and episiotomy repair). There are no compartments to inflate, stopcocks, and hoses.

PATIENT PREPARATION

Explain the risks, benefits, and alternative procedures to the patient and/or their representative. Include an explanation of the application process. An informed consent is not required. Examine the

FIGURE 230-3. An example of a transparent PASG. (Used with permission from www.hiveminer.com.)

patient thoroughly. Determine if the patient has any sharp objects in their clothing or on the lower extremities. Remove anything sharp to prevent the objects from penetrating the antishock garment or the patient. Reduce any extremity deformities. Examination of the patient under the garment will be difficult once applied except when the transparent model is used.

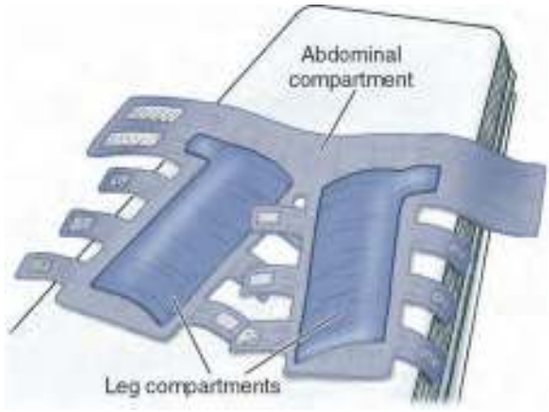

FIGURE 230-4. The three compartments of the PASG positioned on a gurney.

TECHNIQUE

PASG APPLICATION AND INFLATION

Open each compartment of the PASG and place the compartments on a flat surface (**Figure 230-4**). Place the PASG on the patient. Place the patient supine on the open PASG, log-roll the patient and put the PASG under them, slide the PASG under the supine patient, or apply the PASG as if it were a pair of pants. Close and secure the compartments around the patient's lower extremities and abdomen using the Velcro tabs (**Figure 230-5**). Connect the inflation hoses for each compartment to the foot pump. Place a blood pressure cuff on an upper extremity to monitor blood pressure.

The lower extremity compartments should always be inflated prior to the abdominal compartment. The stopcocks must all be closed before inflating any compartment. Open one stopcock at a time to inflate a compartment. Close the stopcock to the abdominal compartment and open the stopcocks to the lower extremities. Inflate the lower extremity compartments using the foot pump. Assess the systemic blood pressure using the blood pressure cuff. **The goal is to achieve a systolic blood pressure of 100 mmHg with the lowest inflation pressure.**[2] Proceed to inflate the abdominal

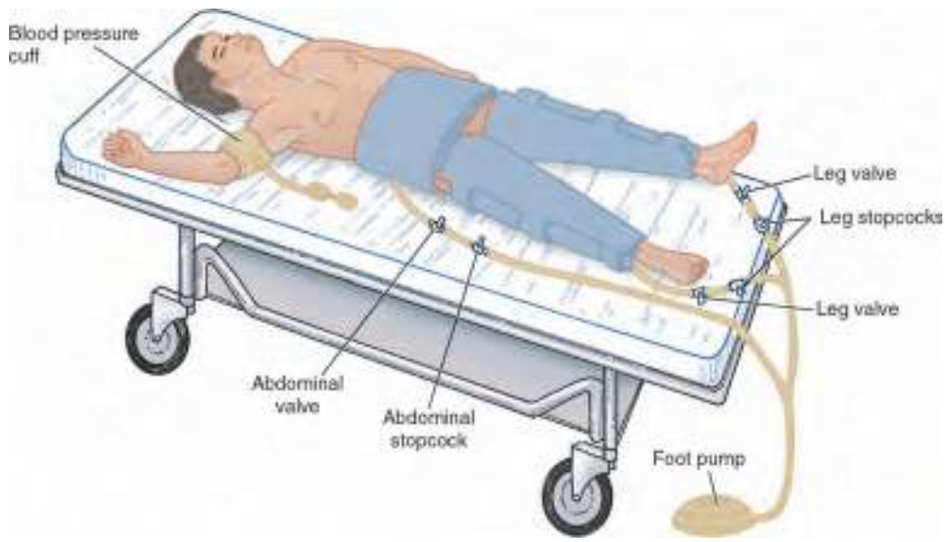

FIGURE 230-5. The PASG secured around the patient with Velcro straps.

compartment if the patient still has a blood pressure below 100 mmHg. This blood pressure goal is not important if the PASG is being applied to splint pelvic or lower extremity fractures. **Close off the stopcocks on inflated compartments to prevent deflation.**

PASG DEFLATION

The prehospital-applied PASG can be carefully deflated once under the direct supervision of a receiving physician and increased monitoring and support are instituted. Remove the PASG if a complication develops.

Deflate the compartments gradually. Sudden deflation is equivalent to a significant blood loss.[2] Blood pressure must be assessed after each compartment is deflated. Deflate the abdominal compartment prior to the lower extremity compartments. Use the stopcocks to achieve the slow and gradual deflation. Prepare and ready intravenous fluids to respond to any decrease in blood pressure.

NASG APPLICATION AND REMOVAL

The application and removal of the NASG is similar to the PASG without the inflation of compartments. Apply the NASG using the Velcro closures. Make sure the abdominal balloon is appropriately placed (**Figure 230-2**). Tighten the abdominal section as necessary to tamponade the bleeding. Removal is easy and does not involve the deflation of compartments. Just undo the Velcro.

COMPLICATIONS

Several complications may occur from the use of a PASG.[23] Its application may prolong scene time when used by poorly trained personnel. Hypotension may occur when garment removal is too rapid. Respiratory distress may be present in the setting of pulmonary edema or diaphragmatic rupture. A compartment syndrome may occur in injured and uninjured lower extremities during prolonged PASG application.[6,7,10,24] Other complications include renal hypoperfusion, congestive heart failure, vomiting, skin breakdown, lactic acidosis, fluid deficit, and hyperkalemia.[2,23,25]

The limitations of using a PASG include the limited ability to perform a physical examination, obtain vascular access, and perform any other procedures under the area of the garment. Another consideration is flight transport. PASG compartment pressures have been shown to increase as a function of increasing altitude.[26] Adjust compartment pressures during flight.

SUMMARY

The antishock garment may be a useful adjunct to treat traumatic hypovolemic shock and similar conditions. It may help increase venous return, increase systemic vascular resistance, tamponade hemorrhage, and splint fractures. The medical literature is conflicted concerning the device's efficacy. Its use is controversial.[27] Complications may occur, especially in the setting of prolonged use. The use of newer devices to treat fractures (i.e., pelvic and femur) and hemorrhage has resulted in less use of the antishock garments.

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231 Hazmat Patient Management

Atilla B. Üner

INTRODUCTION

Hazardous materials (hazmat) incidents occur most often during the transport of chemicals or at industrial site accidents. The work places, transportation, and hazardous substances are labeled with standard government labels or placards (**Figure 231-1**) Hazmat incidents may strike any community at any time.[1] Every Emergency Department must be prepared to respond to victims of a hazmat exposure.

Decontamination is the procedure of eliminating or reducing to a safe level any harmful substances on persons and equipment.[2] **Decontamination of victims in the field should be performed by Emergency Medical Service (EMS) providers before the patient enters the Emergency Department.** However, this does not always occur. Patients may be too ill for lengthy decontamination procedures prior to transport. Exposed patients may leave the scene and present to the Emergency Department on their own or be transported by EMS using specialized equipment (**Figure 231-2**).[3-8] Basic decontamination by Emergency Department personnel can be safely performed in a designated decontamination area.[6,9,10]

Exposure to a hazmat-contaminated patient who has been inadequately decontaminated or not decontaminated at all is a real possibility. This can result in exposure and secondary injury to healthcare personnel, other patients, and visitors.[5-7] The Emergency Department may be closed in part or in whole until the facility can be decontaminated. The panic and fear induced by rumors or odors can lead to unnecessary facility closure, delayed or inadequate patient treatment, and psychogenic illness in both healthcare personnel and bystanders.[3,4]

FIGURE 231-2. An example of a containment cell used by EMS. (Courtesy of Quantum EMS.)

The three primary goals for the Emergency Department are to isolate and contain the contamination; decontaminate and treat exposed patients while protecting staff, other patients, and visitors; and maintain normal services, or reestablish them, as soon as possible.[3,11]

Early recognition of potentially hazmat-contaminated patients will aid in preventing secondary contamination and Emergency Department closures. Numerous clues may identify a patient for hazmat contamination at triage.[3,4] These include accidents at agricultural or industrial sites, accidents involving chemical transports, suspected mass casualty incidents, a cholinergic toxidrome, mucous membrane irritation, chemical burns, soiling with unidentified liquids or powders, intentional overdose with chemicals, unexplained unconsciousness or cardiac arrest, and symptoms occurring in Paramedics or Emergency Medical Technicians (EMTs) after patient transport.

Information management is critical to the response.[4] Designate a person to obtain as much information as possible on the involved

FIGURE 231-1. An example of the labels and placards used to identify hazardous materials. (Courtesy of www.labelmakers.com.)

substance. Even preliminary and general information is useful and should be immediately conveyed to the treating Emergency Physician.[1] The maintenance of material safety data sheets (MSDS) is mandatory for each chemical used or stored at an industrial site or during transport. Request information on the MSDS from fire department personnel or EMS at the scene. Search for the material's MSDS online. Further information resources are listed after the "References" section of this chapter. Verify all information for accuracy by cross-referencing several sources.[4]

Separate decontamination procedures using diluted bleach for victims of biological weapons agents are no longer recommended for civilian mass casualty incidents.[6,12] Instead, follow the procedures outlined in this chapter. Most biological warfare agents are transmitted by inhalation; therefore, respiratory protection is highly recommended.

All Emergency Departments should conduct regular in-service training sessions and drills on hazmat decontamination procedures for ambulatory and nonambulatory patients. Personal protective equipment (PPE), including respiratory protection, require regular training for personnel safety and proficiency.[13-16] Information on decontamination best practices is available from the United States Department of Labor Occupational Safety & Health Administration, the US Department of Homeland Security, the US Department of Health and Human Services, and other resources.[6,10,17]

This chapter describes a general approach to the management of the hazmat-contaminated patient. The Emergency Department response and patient management will have to be tailored to the specific agent(s) and circumstances, such as the number of patients and facility resources.

ANATOMY AND PATHOPHYSIOLOGY

Hazardous materials can enter the body through inhalation, absorption, and ingestion.[18] The total dose is a function of the concentration and properties of the substance, the exposure time, and the exposure route. There is a dose-response relationship between the absorbed dose and toxic effects for most toxic chemicals, and decontamination is a time-critical medical countermeasure to prevent short- and long-term morbidity and mortality.[6,7,12,18] The effects can be local, systemic, and quite variable for different agents.

Inhalation is the most common route of exposure. It may lead to local irritation, airway obstruction, and/or pulmonary systemic absorption. Absorption through intact skin is facilitated by fat solubility, open wounds, burns, exposure of the eyes and mucous membranes, and dermal contamination underneath soiled clothing leading to an occlusive dressing effect.[2,4] The axillae, groin, and skin folds have a thin epidermal layer that facilitates the absorption of a contaminant. Children have a larger surface area to body weight ratio and more permeable skin making them more vulnerable to absorption of toxins.[19] Ingestions can be accidental or intentional.

Patients exposed to hazardous materials can show many signs.[4] Local effects include chemical burns and respiratory tract effects of inhaled toxins. Systemic effects include those from absorption of toxins, associated trauma, psychiatric reactions (e.g., agitation and anxiety), or contamination without apparent injury.[4]

The hazard to personnel, or risk of secondary contamination, is determined by the dose of the hazardous material that is carried by the victim and absorbed by the caregiver.[20] Toxic liquids and solids can pose a dermal contact hazard.[3] Solids or volatile liquids can pose an inhalational hazard.[3] Most gases or vapors dissipate quickly and are unlikely to pose a secondary contamination hazard.[6,7,10,20] However, the risk to emergency personnel is real.[4,5,21,22] Contamination with hazardous materials may indeed cause illness in personnel.[4,20-22] Toxins can rarely be emitted from vomitus (e.g., hydrogen cyanide after cyanide salt ingestion) or exhaled breath (e.g., after ethylene oxide exposure).[2]

INDICATIONS

The decision to initiate decontamination, whom to decontaminate first, and which level of PPE to use must be made according to circumstances and available information.[6,7] Any patient potentially contaminated with a toxic substance should be decontaminated prior to their entry into the Emergency Department.[6,21] Rapid and complete undressing is the most important initial step of decontamination. Decontamination is a time-sensitive intervention. The more toxic the substance, the more rapidly the decontamination should be performed. Pesticides and corrosives are common and worrisome contaminants.[20] Patients requiring immediate medical care, including antidotes, and those displaying signs and symptoms of toxicity should be prioritized. Children and patients with chronic medical conditions should follow those that require immediate care.[6] Ideally, enough healthcare personnel are present for those requiring immediate care and others that need decontamination before they need immediate care. Initiate any life support measures simultaneously with decontamination.[4] Patients who have been decontaminated or exposed to gases or vapors without signs of skin or eye irritation may not require further decontamination beyond removal of their clothing.[2,4,6,9]

Patients should never be made to wait in their contaminated clothing. They need to be given the means to undress in privacy immediately and cover themselves for modesty and protection from the elements.[6] The next step depends on the nature of the toxic exposure, the number of patients, and condition of the patients.[6] Self-decontamination occurs by the patient wiping, blotting, or taking a shower in a designated decontamination shower.[23] Patients may need instructions and towels or wipes to self-decontaminate. Gross decontamination is dealt with by deluge hosing down, showering with water, or using commercial decontamination products. Finally, there is a thorough technical decontamination by hospital personnel.[6] Rapid removal of clothing and immediate deluge gross decontamination with water will remove most contaminants and must be instituted without any delay.[12] All three decontamination tiers may be necessary. **Male and female patients should be in separate areas for all stages of decontamination.**

The absence of a system to isolate waste water is not a contraindication to water decontamination.[6,12] When in doubt, decontaminate the patient. Dilution will suffice to reduce the hazardous substance to a safe level.

CONTRAINDICATIONS

Water decontamination may be contraindicated if the risk of hypothermia outweighs the benefit of decontamination, especially in low-risk patients. Alternative approaches such as dry blotting or brushing, the use of chelating agents, the use of an absorbent (e.g., kitty litter), or the use of an adsorbent (e.g., activated charcoal) can be considered.[6]

Contraindications to water decontamination include contamination with calcium, cesium, lithium, metallic sodium, potassium, pure magnesium, rubidium, strontium, sulfur, titanium, uranium, yttrium, zinc, and zirconium.[1,24] These substances react violently with water and can cause secondary injury.[1,24] Remove the clothing and gently brush off all visible contaminants followed by covering the patient and/or substance with mineral oil, remove the substance with forceps, and store the substance in a container with mineral oil.[4,24] However, many of these substances may already have reacted with moisture on the patient's skin.[1,24,25] Rapid removal of the chemical agent is better than allowing continuing injury to occur.[9,25] Follow

this by rinsing the patient with copious amounts of water. Adding large amounts of water to the small amount of residual chemical on the patient's body poses little risk of creating a serious reaction hazard.[25,26]

Patients soaked in flammable liquids pose special problems. Do not cardiovert or defibrillate the patient until the material is completely removed from the skin by water decontamination to prevent an explosion or a fire.[9]

The use of chemical agents for neutralization is contraindicated. These substances may cause secondary injury, toxicity, or react with another substance in an adverse manner. Use large quantities of water for decontamination rather than a neutralization agent.[6,7,9]

EQUIPMENT

PERSONAL PROTECTIVE EQUIPMENT PER PERSON (FIGURES 231-3, 231-4, AND 231-5)

- Scrub suit and closed shoes
- Plastic shoe covers
- Protective goggles
- Water-resistant face mask
- Latex gloves
- Chemical-resistant (i.e., nitrile) gloves
- Chemical-resistant coverall suit with booties and hood
- Duct tape
- Rubber apron
- Butyl rubber gloves
- Chemical-resistant rubber boots or shoe covers
- Respiratory protection (i.e., powered air-purifying respirator [PAPR] with assigned protection factor 1,000)[10]
- Skin protectant for hands (optional)

PATIENT DECONTAMINATION

- Designated decontamination area
- Decontamination stretchers, if applicable
- Portable wading pools for ambulatory patients
- Tepid running water source with a hose and soft-stream nozzle (50 to 60 PSI water pressure)

FIGURE 231-3. An example of the PPE.

- Portable barriers to assure privacy and protect from wind
- Liquid hand or body soap
- Liquid shampoo
- Soft-bristled brushes or sponges
- Devices to decontaminate hands and nails (e.g., surgical scrub sets)
- Eye irrigation equipment

MISCELLANEOUS SUPPLIES

- Polyethylene plastic bags
- Name tags
- Yellow tape and signs to mark decontamination zones
- Shears to cut clothing
- Scrub suits, towels, and blankets for patients before and after decontamination

The Occupational Safety and Health Administration (OSHA) requires level B protection for responders to an unknown chemical hazard with a positive-pressure self-contained breathing apparatus and chemical-resistant clothing.[3,18,21] The high cost of such equipment and constant skills retraining required to use it may prohibit some hospitals from purchasing it and has made compliance difficult.[13-16,18] OSHA has published best practice guidelines for hospital-based chemical decontamination procedures including detailed recommendations for PPE.[10]

Hospital gowns, standard Tyvek suits, surgical masks, and latex gloves are not considered adequate protection.[4,6,20,24] PPE is available through specialized dealers and requires training to provide reliable protection.[4,18,21,24] The minimum protection recommended for hospital decontamination personnel includes a scrub suit, a chemical-resistant suit with booties and hood, plastic shoe covers, at least two layers of gloves (i.e., surgical and nitrile) taped at the sleeve, protective eyewear, and respiratory protection (**Figure 231-3**).[4,6,10,18] The exact PPE depends upon if the hazardous agent is known, what the hospital has available, and the knowledge of the personnel.

There is little consensus on how to provide respiratory protection. The required level of respiratory protection, if any, will depend upon the agent, the dose, prior training, and decontamination circumstances.[4,18,21] OSHA has published guidelines under which the use of PAPRs is sufficient.[10] The use of PAPRs requires regular training. Respiratory protection may not be necessary if decontamination is performed outdoors.[20] Information obtained about the contaminant will aid in determining the level of protection required.

PATIENT PREPARATION

Patients should be initially received in a secured area outside of the Emergency Department. Clearly mark the area with yellow tape or another similar method. This is considered a contaminated "hot zone." Triage, a primary survey, and any required immediate lifesaving interventions (e.g., basic life support and cardiopulmonary resuscitation) can be performed here by appropriately protected personnel.[4,18,27] Rapid removal of clothing is essential as absorption is time dependent and contaminated clothing can act as an occlusive dressing or continue to off-gas contaminant.[25] **Patients must not wait for decontamination in their contaminated clothing**. Patients who are able should remove their own clothing immediately and be given clean clothing or blankets for warmth and modesty. Children should not be separated from their caregivers if possible.[19,28]

Patients who present to the Emergency Department with real or imagined contamination are under high stress and are likely to act

FIGURE 231-4. Examples of air-purifying masks (APM) and powered air-purifying masks. ***A.*** The 3M half mask disposable APR. ***B.*** The 3M half mask APR. ***C.*** The North half mask APR. ***D.*** The 3M Breath Easy belt-mounted butyl rubber hood PAPR. ***E.*** The MSA CBRN PAPR. ***F.*** The 3M Versaflo Easy Clean PAPR.

A

B

C

D

E

FIGURE 231-5. Donning of PPE. See text for description.

F

I

J

G

K

H

L

FIGURE 231-5. (*Continued*)

M

N

O

P

Q

R

FIGURE 231-5. (*Continued*)

S

T

FIGURE 231-5. (*Continued*)

out if left unattended.[29] The early self-removal of clothing will help alleviate fears and provide a real early decontamination benefit.

The decontamination area is also considered a contaminated "hot zone."[4] **Perform basic life support and immediate lifesaving measures only in the decontamination area.**[11,24] However, the extent of medical treatment in the decontamination area will need to be decided on a case-by-case basis. Treatment of cardiac dysrhythmias and wounds or the administration of toxin-specific antidotes may have to be initiated prior to complete decontamination.[24,27] Advanced procedures such as endotracheal intubation (Chapter 18), video laryngoscopy (Chapter 20), or venous access (Chapter 61) will be difficult to accomplish while wearing PPE and may have to be supplanted by a supraglottic airway (Chapter 25) or intraosseous access (Chapter 70).[30-38] **Invasive procedures prior to decontamination may provide a percutaneous route of contamination with the hazardous substance.** Devices (e.g., monitors) used in the decontamination area will need to be decontaminated after use.[24] Use long leads, if available, that extend into the clean support area to prevent having to decontaminate monitoring devices.[24] **Patients are considered safe to move into the contamination reduction area ("warm zone") and then the Emergency Department (support area, "cold zone") only when decontamination is completed.** Perform a secondary survey, including a search for any toxidromes, and begin definitive medical treatment after decontamination.[4]

DONNING OF PPE

Putting on PPE is known as donning. Don PPE before approaching the contaminated patient.[4,10,39,40] This will prevent the healthcare worker from becoming contaminated, now making them a patient, and requiring the healthcare worker to be removed from duty. Suggested minimum PPE and its application is described here (**Figures 231-3 and 231-5**). Face mask and goggles provide splash protection only. An air-purifying respirator (APR) or a PAPR is required for respiratory protection (**Figures 231-3, 231-4, and 231-5**).[10] Information obtained about the contaminant will aid in determining the level of protection required.

Wear standard work attire (e.g., a scrub suit). The application of a skin protectant such as DermostyX (Vitamed Pharmaceutical Industries LTD., Israel) to the hands is optional.[41-44] The donning of PPE requires the help of an assistant. Put the PPE equipment in one place (**Figure 231-3**).

Assemble the mask power unit (**Figure 231-5*A***). Apply the cartridges to the mask power unit (**Figure 231-5*A***). Remove any shoes but leave the socks. Put on nitrile gloves (**Figure 231-5*B***). Step into the jumpsuit (**Figure 231-5*C***). Put on a pair of rubber boots (**Figure 231-5*D***). Insert the arms in the jumpsuit (**Figure 231-5*E***). Zip the jumpsuit mostly closed (**Figure 231-5*F***). Instruct the assistant to hold the mask and attached power unit (**Figure 231-5*G***). Place the mask power unit around the waist and cinch the belt

(**Figure 231-5*H***). Instruct the assistant to place the mask on and straighten the collar shields in the front and back (**Figures 231-5*I* and 231-5*J***). The collar shield is composed of an inner and an outer layer (**Figure 231-5*K***). Instruct the assistant to tuck in the circumference of the inner layer of the collar into the jumpsuit (**Figures 231-5*L* and 231-5*M***).

Close the zipper on the jumpsuit and secure it (**Figure 231-5*N***). Tighten the neck of the mask so it does not fall off or become disconnected from the jumpsuit (**Figure 231-5*O***). The PPE suit is now on and needs to be secured (**Figure 231-5*P***). Instruct the assistant to apply rubber gloves (**Figure 231-5*Q***). Tape the gloves to the jumpsuit to prevent any material from entering (**Figure 231-5*R***). Use tape specifically designed for this or duct tape. Taping the top of the boots to the jumpsuit is optional but recommended for added safety. It prevents material from getting into the boots and soaking the foot pieces of the jumpsuit. The PPE is now complete (**Figures 231-5*S* and 231-5*T***).

DECONTAMINATION TECHNIQUE FOR CHEMICAL AGENTS INCLUDING WARFARE AGENTS

Perform a primary survey and treat any life-threatening conditions with basic life support interventions.[4] Remove all the patient's clothing. This includes shoes and jewelry. **Cutting off clothing will aerosolize less contaminant than pulling off the clothing.[6]** Place the clothing in a plastic bag and seal it with duct tape. Place this bag into another bag and seal it to "double bag" the clothing. Double bag any jewelry, glasses, hearing aids, keys, and watches separately if time and circumstances permit. This allows for possible later decontamination and salvage. Label the bags with the patient's name or medical record number. In case of a criminal chemical release, these bags may become evidence for law enforcement. Patients may assist with their own decontamination. The patient should keep a form of identification on their person that can withstand the decontamination process (e.g., a laminated driver's license). Patients will object to surrendering their cell phone as it allows them to reach family and may contain telephone numbers that they do not know by heart. Depending on the toxic substance, jewelry, glasses, hearing aids, and cell phones can be later wiped and returned to the patient. For very toxic substances, however, everything on the patient's body must be considered contaminated and may not enter the support zone. **Do not irrigate the patient prior to complete clothing removal.** Chemicals on clothing will soak through wet clothing and onto the skin resulting in additional injury or absorption.[2,9]

Remove any visible chemicals on the patient's body by gently washing off liquids or brushing off solids. Avoid skin irritation or injury as both will lead to increased absorption of any remaining chemicals. Avoid aerosolization of the contaminant. Blot any heavy liquids with absorbent towels.[2,9]

Patients with open wounds require the area surrounding the wound to be gently washed with water. Irrigate the wound with clean water. Cover large wounds with plastic wrap to prevent runoff into the wound during the remaining decontamination procedure.[2] **Do not delay copious water irrigation if this wound care cannot be accomplished rapidly.**

Begin copious water irrigation with high-volume warm water using low water pressure (e.g., 50 to 60 PSI or normal shower pressure) for 3 minutes. Wash with copious water for 1 minute and then clean with soap and water, including the rinse, for 2 minutes. Washing for longer than 3 minutes may soften the skin and lead to increased absorption, a phenomenon called "wash-in effect."[6,7] Use liquid soap, shampoo, and soft brushes or sponges to gently cleanse the skin and any body hair.[5] **Do not delay decontamination with water if soap is not immediately available as most substances can be removed with water only.[6]** Thoroughly rinse the patient after soaping and shampooing. Repeat the procedure until all visible contamination is removed. **Do not forget to thoroughly clean any mucous membranes, the nails, the external auditory canals, the axillae, the groin, and any other skin folds.** Use cleaning devices such as surgical scrub sets for nail folds or syringes to flush the external auditory canals. Brushes, sponges, or scrub sets must be disposed as hazardous materials and cannot be used on the next patient.[6]

Irrigate the eyes, if contaminated, for 5 minutes (Chapter 187). Remove any contact lenses and discard them. A Morgan lens may be used if the globe is intact. Irrigation with running water is also an option but may be less thorough than **with a Morgan lens. A nasal oxygen cannula may be placed on the bridge** of the nose to irrigate the eyes if both eyes are affected.[2] Allow water to flow through the cannula and direct it from the medial canthus outward.

Do not let the caregivers carry children in a wet decontamination environment as they may fall and injure themselves and the child. Instead, have decontamination personnel carry the child. It may take two providers to safely carry a struggling wet child while wearing PPE.[19] An alternative is to use a gurney or, wheelchair, or a plastic laundry basket for infants.[6]

The nonambulatory contaminated trauma patient or a patient with altered mental status may need to be decontaminated on a specialized stretcher that is equipped to catch waste water. Autopsy tables from the morgue may be used after covering the surface with plastic. Enough decontamination personnel must be available to maintain cervical spine immobilization while log-rolling the patient as the decontamination process is performed.

ASSESSMENT

Move the patient after decontamination to the contamination reduction zone ("warm zone"). Assess the patient and initiate any required advanced life support measures. **There are no objective criteria to assess whether decontamination was sufficient.** There is, however, evidence that copious water irrigation and soap cleansing are highly effective in removing chemical contaminants.[4,6] **Successful decontamination can be implied if proper procedures for a given contaminant were followed.[4,6]** Measure the tear film pH if the eyes were exposed to corrosive agents. Continue eye irrigation until the tear film pH is neutral. This may take up to 15 minutes. Assess all wounds. Thoroughly irrigate any wounds. **Transfer the patient into the support zone (Emergency Department or "cold zone") when they are deemed decontaminated.**

HEALTHCARE PERSONNEL DECONTAMINATION TECHNIQUE

Health–care personnel must remove their personal protective equipment in an organized fashion to prevent self-contamination after decontamination of the patient.[18,45,46] Removing PPE is known as doffing. This is a suggested protocol and may need to be altered depending on institutional policy. **Figure 231-6** demonstrates using a plastic bag for doffing. Some institutions use tape on the floor to denote the clear area versus the contaminated area. The use of a plastic bag or a line taped on the floor is institution specific. The main advantage of using a plastic bag is that the contaminated equipment is already bagged without additional personnel touching the contaminated equipment. Doffing, like donning, requires the help of an assistant. The assistant does not need the level of PPE, depending on the substance, that the Emergency Physician requires (**Figure 231-6*F***).

Place the disposable plastic bag on the floor and in the area where the doffing is to take place. Step into the plastic bag (**Figure 231-6*A***

A

B

C

D

E

FIGURE 231-6. Doffing of PPE. See text for description.

F

G

H

I

J

FIGURE 231-6. (*Continued*)

K

L

M

N

O

FIGURE 231-6. (*Continued*)

P

Q

R

FIGURE 231-6. (*Continued*)

and 231-6*B*). The rest of the doffing procedure occurs in the confines of the plastic bag. Remove the tape attaching the rubber gloves to the jumpsuit and drop the tape into the bag (**Figure 231-6*C***). Remove the tape attaching the rubber boots to the jumpsuit and drop the tape into the bag. Place the hands between the knees and close the knees (**Figure 231-6*D***). Pull the hands upward and out of the rubber gloves (**Figure 231-6*E***). Open the knees and let the contaminated rubber gloves fall into the bag. Stand upright. Instruct the assistant to grasp and hold the mask power unit (**Figure 231-6*F***). Remove the mask power unit (**Figure 231-6*G***).

Prepare to remove the mask. Loosen the neck of the mask (**Figure 231-6*H***). Unzip the jumpsuit (**Figure 231-6*I***). Remove the arms of the jumpsuit (**Figures 231-6*J* and 231-6*K***). Push down the jumpsuit while only touching the inside. Step out of the jumpsuit and one boot (**Figure 231-6*L***). Move the free leg out of the bag (**Figure 231-6*M***). Remove the other leg from the jumpsuit and boot and place this outside the bag (**Figure 231-6*M***). Bend forward (**Figure 231-6*N***). Remove the mask (**Figure 231-6*O***). Place the mask in the bag (**Figure 231-6*P***). Instruct the assistant to place the power unit they are still holding into the bag (**Figure 231-6*Q***). Remove the nitrile gloves (**Figure 231-6*R***) and place them in the bag.

Instruct the assistant to seal the bag with duct tape.[47] Close off the dirty area until the level of contamination is established and the area is properly decontaminated. Move to a shower area. Shower thoroughly with soap and water. Dress in normal work attire.

AFTERCARE

Further patient management will depend on the type of exposure and other circumstances. Perform a thorough secondary survey to detect occult trauma or medical illness. Monitoring for cardiac dysrhythmias or for noncardiogenic pulmonary edema may be necessary after exposure to certain chemicals.[2,9] Some toxins necessitate treatment with specific antidotes which should be initiated as soon as possible.[48,49]

Dilute ingested chemicals with 4 to 8 ounces of water. Activated charcoal at 1 gm/kg may be given to prevent further absorption.[9] Ingestion of corrosive agents may necessitate early endoscopy, in which case charcoal should be withheld.[9] **Induction of emesis is rarely indicated.** Emesis may pose a hazard to the patient if they ingested corrosives or hydrocarbons and to healthcare personnel if the patient ingested cyanide.[2,9]

COMPLICATIONS

Hypothermia may be caused or worsened by the decontamination procedures.[2,9] This must be anticipated and treated accordingly. Children are especially vulnerable to hypothermia.[10,28] Residues of

highly toxic chemicals remaining in skin folds or under nails may pose a risk to the patient and caregivers.[9] This should be searched for and removed. Toxic vomitus may pose a risk to healthcare personnel, even after adequate decontamination.[2,9] Emesis must be contained and discarded appropriately.

Decontamination personnel are subject to dehydration, exhaustion, heat illness, and hyperthermia exhaustion while wearing PPE. Work cycles need to be adjusted to maintain personnel safety. The improper use of PPE can result in contamination of the healthcare personnel.[50] Frequent drills will prevent this from occurring.

SUMMARY

Emergency Departments must be prepared to decontaminate and treat victims of hazardous materials exposures at any time. Procedures must be developed, practiced, and incorporated into the hospital's mass casualty incident plan. Lack of preparation and practice will expose medical personnel to greater risks and lead to less than optimal patient care. This chapter presents a general approach to decontamination procedures that must be adapted to the individual circumstances and type of exposure.

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INFORMATION RESOURCES

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3. U.S. Department of Labor, Occupational Safety and Health Administration: OSHA best practices for hospital-based first receivers of victims from mass casualty incidents involving the release of hazardous substances. Accessed October 20, 2016, from www.osha.gov/dts/osta/bestpractices/html/hospital_firstreceivers.html.
4. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR), 4770 Buford Hwy NE, Atlanta, GA, 30341. The ATSDR website (www.atsdr.cdc.gov/) has online information for the management of specific hazardous materials exposures.
5. CHEMTREC: Information Service of the Chemical Manufacturers Association. CHEMTREC will provide chemical information and assist in obtaining medical information and site decontamination. 24-hour telephone number: (800) 424-9300. Website www.chemtrec.com/Chemtrec.
6. National Pesticide Information Center (NPIC), Oregon State University/Environmental Protection Agency. Will provide chemical information on pesticide products. The book "Recognition and Management of Pesticide Poisonings" can be downloaded under "Emergency" then "Related Topics" on the homepage. Phone (800) 858-7378, Monday through Friday, 8:00–12:00 Pacific Time, excluding holidays. Website www.npic.orst.edu.
7. Nuclear Regulatory Commission (NRC) Emergency Center. Will coordinate assistance for nuclear accidents or releases by one of four national NRC centers, including information, advice, and personnel if needed. 24-hour telephone number: (301) 816-5100 [Back-up tel. (301) 951-0550 or (301) 415-0550, fax (301) 816-5151].
8. National Response Center. Takes reports on any chemical or nuclear release or spill, or suspected terrorist attack, and forwards information to federal agencies, including but not limited to the FBI and the Chemical/Biological Defense Command of the U.S. Army. 24-hour telephone number: (800) 424-8802.
9. U.S. Army Military Research Institute on Infectious Diseases (USAMRIID). Information on diagnostics, medical management, and vaccines relating to biological weapons can be obtained by contacting the commander of the USAMRIID. Phone (301) 619-2833 or (888) 872-7443 during business hours Eastern Standard Time. Website www.usamriid.army.mil/index.htm.
10. Toxicology Data Network (ToxNet) at the National Library of Medicine (www.toxnet.nlm.nih.gov).
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232 Physical Restraints

Dean Sagun

INTRODUCTION

Psychiatric emergency services have become a major point of entry into the mental health system and a principal treatment site for many patients with chronic and severe mental illness.[1] Emergency Department (ED) patients with altered mental status, emotional disturbances, head trauma, psychiatric illness, psychological disturbances, or other medical conditions may be aggressive, physically injurious, or violent.[2,3] The aggression may be exhibited toward themselves or toward the healthcare personnel who are caring for them.[4] Emergency **Physicians must be prepared to cope effectively with agitated or violent patients to reduce the risk of serious injury to the patient and healthcare personnel. Physical restraints might be necessary to ensure the safety of the patient and the healthcare personnel given the volatile nature of some presenting conditions.** A prospective study found that 0.07% of ED patients were restrained during the 1-year study period.[5] Up to 8.5% of psychiatric patients seen in the ED may require the use of physical restraints.[6] Most patients were restrained for agitation, disruptive behavior, or violent behavior.[5,6]

Physical restraints are the direct application of physical force to a patient without the patient's consent to restrict their freedom of movement. The use of restraints for managing behavioral emergencies is allowed only when all other less restrictive measures have failed and severely aggressive or destructive behaviors place the patient or others in imminent danger. The Centers for Medicare and Medicaid Services (CMS) defines physical restraint as "any manual method, physical or mechanical device, material or equipment attached or adjacent to the patient's body that he or she cannot remove and that restricts freedom of movement or normal access to one's body."[7] The use of restraints in the ED may help to prevent patients from physically harming themselves or others. Imminent safety concerns are the only justification for application of physical restraints.[7] Physical restraints must be used for the shortest time possible and with the least restriction possible.[7,8] Physical restraints are humane and effective in the management and treatment of the patient while ensuring the safety of the patient and the health-care personnel if used properly and in the appropriate patient.

This chapter will present the rationale and technique for using locked-door seclusion and physical restraints in the ED. This chapter focuses on the aggressive, dangerous, and/or violent patient. It briefly reviews some of the techniques for physically restraining the young child during brief diagnostic or therapeutic procedures.

LEGAL CONSIDERATIONS

Informed consent is required before Emergency Physicians can lawfully treat competent adult patients, all of whom have the right to refuse medical treatment (Chapter 1). U.S. courts have consistently upheld the idea that a competent adult would consent to treatment to maintain health or life and that a patient could be restrained to protect others or self if the reasons for interventions are clearly documented.[9] **The competent adult retains their right to refuse treatment in an emergency setting. Coercive measures (e.g., restraints and the threat of restraints) cannot be used solely because a patient refuses treatment. It is never appropriate to restrain a competent patient against their will solely because they are refusing recommended treatment.**[10] The CMS, which governs

hospitals, have required specific training elements for personnel who will be applying physical restraints. They require hospitals to have clearly delineated policies as to who is certified as capable of applying restraints. The elements of training include the use of non-physical interventions and the demonstration of these skills.

INITIAL APPROACH TO THE PATIENT

Evaluation of the combative patient begins with a risk assessment and attention to safety measures. Assess for potentially dangerous organic causes of agitation when this can be done safely. Alcohol intoxication, drug intoxication, and substance withdrawal are common diagnoses in combative patients. Obtain a fingerstick glucose and complete set of vital signs. The typical patient first becomes angry, then resists authority, and finally becomes confrontational. **Signs of impending violence include aggressive acts, an angry demeanor, pacing, provocative behavior, speech that is aggressive and loud, and tense posturing.**

There are many risk factors for violence.[11] Young age and male sex are common characteristics in many violent patients. There is often current substance abuse, a history of substance abuse, or a history of violence. Patients with psychiatric disorders can be violent. Low socioeconomic status is associated with violence. The environment of the ED is associated with violence in some cases. This includes crowding, lack of privacy, and prolonged stays.

Use verbal de-escalation techniques prior to initiating physical restraints (Table 232-1).[12] This approach can assist the Emergency Physician in engaging the patient and calming the situation. Patients who are agitated but cooperative may be amenable to this approach. Carry out all techniques in a calm voice that is clear, slow, and low in volume. Use an attentive, nonconfrontational, and receptive manner without conveying vulnerability or weakness. Stand at least two arm's lengths away from the patient with direct access to the door or exit. Avoid approaching the patient from behind or moving suddenly. Gently question the patient as to why they are distressed. Allow the patient time to think about the question prior to offering an array of potential choices. Common reasons why ED patients become agitated include being frightened in a medical facility, being frustrated at the perceived lack of concern on the part of healthcare providers, being hungry, being made to undress and put on hospital attire, being in pain that they do not feel is being promptly addressed, and not understanding why things are happening. **Addressing the patient's specific concern can often de-escalate the agitated patient.**

Begin to orient the patient to their environment if they are unable to communicate their needs or are too agitated to tolerate questioning. Explain in clear language what needs to occur. For example, "Mr. Smith, you are in the ED because you are having a fever and we now need to get you dressed in a hospital gown. Can you do that for me? May I help you?" **Allow the patient some control to make decisions about the sequence of events as safety permits.** For example, if the patient first wants to use the restroom or drink water, allow the patient to do these things first. Patients who are confused or frightened often de-escalate with these techniques.

It is often possible to encourage the patient to take oral or intramuscular medication prior to the use of physical restraints. The ability to begin a patient on needed medical treatment (i.e., benzodiazepines for alcohol withdrawal or antipsychotic agents for paranoia) may allow the patient to de-escalate and eliminate the need to initiate physical restraint. Carefully monitor the patient including constant observation, pulse oximetry, telemetry if appropriate, and vital signs at regular intervals.

TABLE 232-1 Key Elements for Verbal De-Escalation[35]

1. Respect personal space (e.g., maintain two arm's lengths and provide space for an easy exit)
2. Do not be provocative (e.g., stay relaxed and do not stare at the patient)
3. Establish verbal contact (e.g., the first person to contact the patient should be the leader)
4. Use concise and simple language
5. Identify feelings and desires
6. Listen closely to what the patient is saying to improve the mutual understanding
7. Agree or agree to disagree
8. Lay down the law and set clear limits (e.g., violence and abuse will not be tolerated)
9. Offer choices and optimism
10. Debrief the patient and staff

INDICATIONS

Restraints are an emergent or urgent intervention when less restrictive interventions have failed.[12] Use restraints for patients who are currently demonstrating behaviors that would put them, the staff, or others in imminent danger.[12] This includes patients who are aggressive, agitated, combative, dangerous to self or staff, or violent and do not de-escalate with other interventions. **Do not delay using restraints if patients can harm themselves or others. The use of physical restraints is a last resort.**[13,14]

CONTRAINDICATIONS

Do not use physical restraints for the sake of convenience or if a competent adult refuses treatment. Do not use them for patients who are currently not demonstrating behaviors that would put them, the staff, or others in imminent danger. Patients with a history of violence but exhibiting no active imminent aggression cannot be restrained. The use of physical restraints is an urgent intervention when less restrictive interventions have failed.[12]

Physical restraints must be immediately released when the patient is no longer demonstrating dangerous behavior. There are potentially severe complications associated with the use of physical restraints. Hospitals must ensure appropriate staffing levels to meet this dynamic need. Every acute care hospital has developed policies and procedures for the monitoring and documentation associated with the use of physical restraints. A sample of the required documentation can be found in **Table 232-2**. The Emergency Physician must be cognizant of these requirements and be able to follow these policies and procedures. Asphyxiation and strangulation are the two most serious complications of physical restraint.[15] Personnel must be available to continuously monitor the patients' respiratory status throughout the application of physical restraints.

The use of leather restraints significantly increases the risk of neurovascular complications, rhabdomyolysis, and skin breakdown even when used correctly. Patients should demonstrate the ability to break free from soft restraints or require the additional security of leather restraints prior to application of leather restraints. **Leather restraints are to be considered a restraint of last resort.**

Do not use seclusion if the patient is unstable, if the patient must be medically monitored, or if they overdosed on medications and/or poisons. **Seclusion is contraindicated if the patient cannot be constantly monitored visually, either electronically with closed-circuit video or by direct observation.**

EQUIPMENT

- Gloves
- Additional personal protective equipment, if indicated
  - Gown
  - Face mask with an eye shield or goggles
  - Cap

TABLE 232-2 Typical Documentation Requirements for the Physical Restraint of a Patient

Physician Orders
- Date and time of order
- Alternatives to restraint employed (i.e., verbal de-escalation)
- Description of specific behavior that placed patient, staff, or others in imminent danger
- Patient response to alternatives
- Type of restraints and which limbs to restrain

Ongoing Assessment
- Description of behaviors that patient needs to demonstrate to release restraints
- Documentation of personnel involved in the application of restraint, including security and other trained staff
- Existence of an emergency
- Failure of less invasive techniques and list them
- Family notification of restraint as appropriate
- Injury from restraints and treatment
- Patient's preexisting medical or psychiatric condition that may increase the risk of harm
- Patient's reaction to restraints
- Reason for using restraints was/is explained to the patient
- Use of restraints was for patient benefit, patient safety, or staff safety

Conclusion of Restraint
- Debriefing of the patient regarding the episode of restraints including their reaction to the application of restraints
- Debriefing of the staff as appropriate
- Discontinuation of the restraint order
- Documentation of the expected behavior demonstrated by the patient that led to release of restraints and current behavior

- Standard ED bed
- Hospital-approved restraint devices
- Philadelphia collar, various sizes
- Additional personnel

The following equipment may be necessary for putting a patient in physical restraints. Utilize personal protective equipment (PPE) if contact with the patient's body fluids may occur. **Use only devices manufactured for the express purpose of restraining a patient.** Restraints are available in a variety of styles. They are made of many materials (e.g., leather, material, polypropylene, and polyurethane) with Velcro or buckles to secure them. Use a standard ED bed for two-point and four-point restraints. Use padding material to protect the patient's bony prominences. Soft restraint manufacturers usually include this already attached to the restraint. Leather restraints are physically stronger and less constricting than soft restraints. **Leather restraints are more effective in the violent patient than soft restraints.**

Makeshift restraints (i.e., gauze rolls) are difficult to apply and must be tied extremely tightly to secure the patient. **Avoid makeshift restraints in the ED because of the increased risk for injury to the skin, peripheral nerves, and vasculature.** Makeshift restraints can cause respiratory difficulties if placed around the chest.

PREPARATION

Appropriate use of restraints requires prior planning and education of physicians, nurses, and security staff. Physical restraints may pose a danger to the patient and the staff without sufficient training and safeguards.[2,16] The Emergency Physician must provide a timely diagnosis and an appropriate acute management plan to ensure the safety of a patient and the staff. **The "ABCs" of the emergency evaluation of aggressive and impulsive behavior include safety, diagnosis, and management. Safety is the first priority when confronted by a potentially violent patient.** Adequate evaluation, diagnosis, and management are impossible without proper safety mechanisms.

The application of physical restraints requires the understanding that physical restraints are mechanical devices that prevent patient interference with medical treatment, reduce the risk for falls and subsequent injury, and/or keep the patient from harming themselves or others. **Use physical restraints as a last resort for maintaining patient safety. Continually evaluate the alternatives to physical restraints.** It is important to be familiar with hospital procedures for ordering physical restraints, including documentation requirements (**Table 232-2**).

The Emergency Physician must understand the indications and purposes for use of the different types of restraints and their respective functions (e.g., wrist restraints to prevent arm movement). Observe and document the type of behavior that has led to the need for physical restraint (e.g., attempting to harm oneself or others, removing intravenous (IV) catheters, or removing other equipment) and the type of restraint prescribed (**Table 232-2**). An Emergency Physician order for physical restraint is required and must be documented within 1 hour of the application of the restraints (**Table 232-2**). It can be documented after the restraints are initiated. The treatment team (i.e., Emergency Physician and nursing) should each document the situation, the alternatives to physical restraint that were attempted, and that alternatives were attempted with lack of success. Review the manufacturer's directions for assembling and placing the physical restraint. Lower the bed as low to the floor as possible. Visually inspect the bed to ensure it is free of objects that may be trapped under a physically restrained patient.

The restraints, sufficient number of personnel, and a plan should be assembled and ready prior to initiating physical contact with the patient. This will reduce the risk of asphyxiation or other complications due to a prolonged physical contact time with the patient. All personnel must be approved for applying restraints to protect the patient and the staff. At least one of the staff members should be a female if the patient is female. It is best to have security standing ready to assist if needed. Their mere presence and visibility to the patient as a "show of force" is often enough to calm the patient. Emerging practices may include a scribe to document the initiation of restraint and to ensure the safety of the patient during the application of restraints by keeping time, monitoring the patient's status, and confirming respirations.

One or two people will usually be physically applying the restraints. It is common to need up to four additional personnel to hold the patient during the application of the restraints. Understanding the nature of the aggressive behavior and the physicality of the person who will need to be restrained may help determine how many additional personnel are required. **It is always better to have more people than needed than to not have enough. It is dangerous for the patient and the staff to have fewer people available to help than are needed. It is often better to have a "show of force" with multiple staff members and security present to be visible to the patient. The mere presence of multiple people conveys the message you are serious, often makes the patient more compliant, and gives the patient an excuse to cooperate with the application of physical restraints.** The application of physical restraints is time limited and is a personnel-intensive process. Be aware that other agitated patients may take this opportunity to elope from the facility.

Communicate with the patient and family. Evaluate whether the patient or family members require special considerations regarding communication (e.g., deafness, illiteracy, or language barriers). Arrange to meet these special needs if necessary. Briefly assess the patient and family for knowledge deficits and anxiety regarding the physical restraint procedure. Assign a staff member to provide information and emotional support as needed.

Family members should not be present for the physical restraint procedure. They may become agitated during the procedure and make it an unsafe environment for staff. They may serve as an agitating influence on the patient. All family should be politely asked and escorted to wait in the lobby or family room until the patient is secure. Inform the family that they may return once the patient is restrained and any procedures have been completed. Inform the family members that under no circumstances should they loosen or remove the physical restraints as this should be done solely by hospital staff.

All staff involved must have a knowledge of proper patient positioning.[12,13] **Attempt to not restrain a patient with one or both arms positioned above their shoulders. These "arm-elevated" positions can result in respiratory compromise. Restrain the patient in the supine position with their arms by their sides. The restraints must always be tied to the bed frame and not the side rails.**[17] The patient can experience significant pain and injuries if the bed rails are lowered while the restraints are tied to them.

TECHNIQUES

LOCKED-DOOR SECLUSION

One form of physical restraint is locked-door seclusion (LDS). **LDS provides a low-stimulus environment that is free of dangerous objects. It is a less restrictive form of physical restraint that is an appropriate and viable alternative if the patient is more likely to be an escape risk, dangerous to others, or dangerous to themselves.** Patients who are violent or self-destructive may be placed in LDS. Agitated patients can be placed in LDS by security that are authorized and trained to physically escort patients. A "show of force" often makes the patient more cooperative. ED staff can assist a patient who is not physically threatening but for whom the regular ED environment provides too much stimulation to allow effective treatment to occur. Have security present to prevent injury to the patient or the staff. Agitated patients may require that all treatment, including changing into hospital attire, commence in the LDS setting.

Hospitals who have LDS capability should have built the room according to The Joint Commission (TJC) parameters to provide safety, reduce stimulation, and lessen the risk of self-injury. These rooms have very limited equipment and supplies other than a bed so that the patient does not injure themselves. A patient can inflict significant self-injury with simple things (e.g., blood collection tubes, needles, and tongue depressors). **The LDS room must be continuously monitored via video or direct 1:1 monitoring.** State laws and institutional policies vary on what type of monitoring and documentation are required. **It is important to recognize that a patient is not absolutely safe in LDS.** Highly agitated patients can injure themselves.

The most likely cause of death in LDS is a cardiac event. It is especially important that a physical examination proceed as rapidly as possible after the patient is placed in LDS to address and correct any underlying causes of aggression and/or agitation (e.g., head trauma, hyperglycemia, hypoglycemia, and hypoxia).

PHYSICAL RESTRAINTS

Perform hand hygiene and apply personal protective equipment as needed. **Introduce yourself and your plan to the patient in a calm manner.** Assemble all the required personnel in the room. Provide privacy by closing the door to the room and/or pulling the curtain around the patient's bed if in a shared patient room.

Do not physically participate in holding the patient or the application of physical restraints. This allows the Emergency Physician to maintain the physician-patient relationship required to subsequently evaluate, manage, and treat the patient. Trained staff should physically assist the patient into a comfortable supine position that allows easy access to the area of the body to which the restraint will be applied. It may help to have a team member to hold and lock the patient's elbow or knee in extension. Apply the soft restraints to the patient's wrists and leave approximately 1 to 2 cm between their wrist and the restraint. It should fit snugly but not constrict (**Figure 232-1**).

Apply and tie the two long ends or strapping to the bed frame as a unit (**Figure 232-2**). Attach the ties to the portion of the frame that moves when the head of the bed is raised and lowered. **Do not attach the ties to the side rails.** Attaching the restraint ties to the side rails increases the patient's risk for injury when the side rails are moved down or up. Make a slip knot with the ties (**Figure 232-2**). The slip knot is easily released by pulling on the free ends of the ties (**Figure 232-3**). This allows for easy removal in case of an emergency. Secure the restraints to the bed frame in such a manner that both wrists remain at the patient's sides to reduce shoulder injury risk. **Use caution restraining the patient with one or both arms**

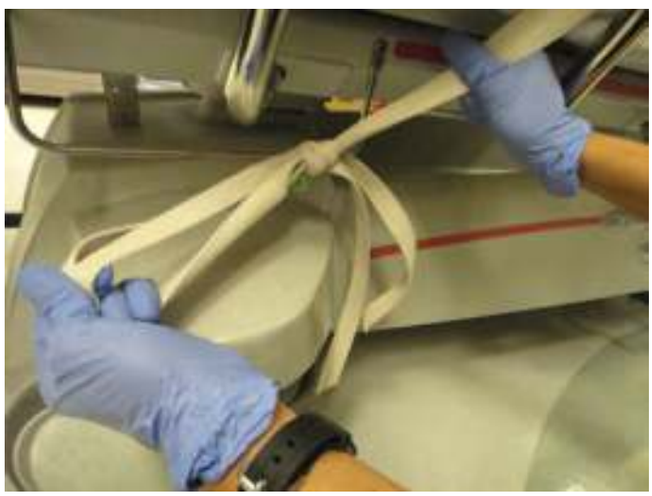
A

B

FIGURE 232-1. Soft wrist restraints. ***A.*** The restraint. (Photo courtesy of Posey Products LLC, Arcadia, CA.) ***B.*** It should have a gap of one to two finger breadths between the patient's wrist and the restraint when applied.

A

B



C

FIGURE 232-2. Soft restraints are attached to the bed frame using a slip knot. ***A.*** Starting the slip knot. ***B.*** Making the slip knot. ***C.*** Tightening the slip knot.

above their shoulders to ensure that the patient does not experience respiratory compromise (Figure 232-4). Avoid the prone or hobble restraint position (i.e., arms and legs restrained behind the patient) as this can result in respiratory complications.

FIGURE 232-3. Pulling the loose ends of the soft restraint will immediately release the knot.

The two-point soft restraint of the wrists is less restrictive than the four-point restraint of all limbs. Apply the four-point restraints to both wrists and ankles. Apply the soft restraints to the patient's ankles like that of the wrists. Leave approximately 1 to 2 cm between the ankle and the restraint (**Figure 232-5**). They are attached to the bed frame.

Soft restraints are less restrictive than leather restraints (Figure 232-6). The method for determining which type of restraint is required must be determined by clinical experience and the current situation involving the patient and their behavior. Patients who are physically frail and nonviolent may benefit from two-point soft restraints. Use of four-point restraints may be necessary for patient safety if they are able to move too freely and are agitated.[12] Severely agitated, aggressive, and dangerous patients may require leather restraints. **The goal is to utilize the least restrictive restraint method while ensuring the safety of the patient and the staff.**

FIGURE 232-4. A restrained patient using four-point restraints and one arm elevated.

FIGURE 232-5. Soft ankle restraints should have a gap of one to two finger breadths between the patient's ankle and the restraint when applied.

Other restraint devices have been used in conjunction with extremity restraints (**Figures 232-7 and 232-8**). Apply a Philadelphia cervical collar to minimize the potential for biting and head banging. An alternative that is more readily available in the ED is to apply a standard cervical collar that is used for the trauma victim. Exchange the standard cervical collar as soon as possible with the softer Philadelphia collar to prevent the standard collar from injuring the patient. **Never use a torso restraint or sheet around the torso.** Limiting chest movement and respirations can result in respiratory compromise and death. Use a device specifically designed to be wrapped around the patient's hips and that attaches to the bed frame (**Figure 232-7**). Consider the use of a net restraint in the very agitated patient who cannot be de-escalated (**Figure 232-8**). Chemical restraint can be safely administered once the patient is physically restrained (Chapter 233).

PEDIATRIC PATIENTS

Pediatric patients are rarely restrained for aggressive and violent behavior. The very young are commonly restrained for diagnostic and therapeutic procedures (**Figures 232-9 and 232-10**).[18-20] These are brief episodes of discrete restraint for specific indications. Use the same general indications, contraindications, and concerns with children as an adult. **Do not place young children in LDS by themselves.** This can result in them becoming more frightened and agitated. Allow the parent or guardian to remain in the LDS room if it is safe for them to do so. Some states have passed strict guidelines for using physical restraints on mentally and physically handicapped children.

A

B

C

FIGURE 232-6. Leather restraints. ***A.*** Buckle wrist restraint. ***B.*** Newer locking restraint. ***C.*** The restraints attached to a bed. (Photos courtesy of Posey Products LLC, Arcadia, CA.)

FIGURE 232-7. A hip restraint added to the four-point restrained patient. (Photo courtesy of Posey Products LLC, Arcadia, CA.)

A popular device is the papoose board for restraint. There are several companies that make restraint boards. These are temporary devices to be used during procedures and are commonly used (**Figure 232-10**). They come in neonatal through adult sizes. Restraining boards usually include canvas flaps that are strong with Velcro fasteners. They are easy and simple to clean after use. They can be configured for various procedures (**Figure 232-10C**). This includes arm extenders, straps for arms, and straps for the head.

The procedure is technically the same for pediatric patients who require physical restraint for aggressive and violent behavior.[19] The restraint time requirements are different. Limit the time an adult is physically restrained or in seclusion to 4 hours. Physical restraints and seclusion must be limited by decreased time intervals in pediatric patients: 2 hours for children and adolescents ages 9 to 17 and 1 hour for patients under 9 years old. Document an assessment and a new order for the continued use of physical restraints or seclusion after this time. Release the restraints every 1 to 2 hours, assist the patient to the bathroom, and offer food and fluids as appropriate in older children.[17] It is wise to document patient assessments at more frequent intervals than with an adult. Follow hospital policies regarding documentation and assessments.

FIGURE 232-8. The net restraint. (Photo courtesy of Posey Products LLC, Arcadia, CA.)

FIGURE 232-9. The Olympic Circumstraint Neonatal Immobilizer.

Frightened young children not able to respond rationally to their circumstances may be soothed when held by an adult which is termed therapeutic holding. This helps the pediatric patient regain control of their emotions, is an alternative to mechanical restraint, and is an alternative to seclusion.[2,21-25] Restraint is considered part of ongoing care if it is used for procedures. This requires specific training beyond the scope of this chapter. Adolescents may need to be separated from friends or family members who are causing their behaviors to escalate.

ASSESSMENT

Patients who are restrained require constant observation by specifically trained personnel. The complication rate for a patient in physical restraints has been estimated at 5.4% to 7%.[5,24] Evaluate the patient every 15 minutes for signs of injury or according to hospital protocol for the type of restraint. Documentation of monitoring includes confirming airway patency, breathing, and circulation. Monitor IV catheters, urinary catheters, drainage tubes, and other equipment at regular intervals to verify correct positioning and the absence of occlusion by the restraints. Assess the needs of the patient and/or family members. Offer additional information and emotional support as needed. **Continually assess whether restraint of the patient is necessary.** Evaluate the appropriateness of restraint use, including the level and characteristics of the patient's activity, at least once every 4 hours. Note whether the patient continues to exhibit the behaviors (e.g., agitation, combativeness) that have made restraint necessary. **The treating Emergency Physician must perform and document a face-to-face evaluation of the patient within 1 hour of the application of physical restraints.**[17] Provide written information as appropriate to reinforce patient and family teaching. Document the procedure in the patient's medical record. Include all teaching and the need for any follow-up education.

Patients will need to be continuously monitored if they are restrained secondary to violent and self-destructive behaviors. State

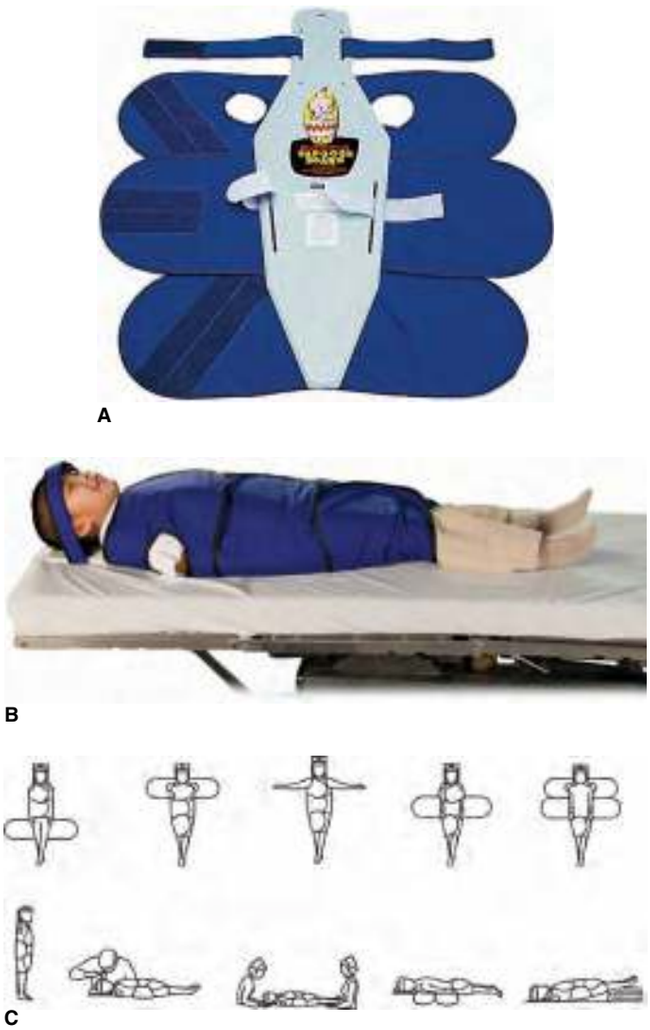


FIGURE 232-10. The Olympic Papoose Board. ***A.*** The device. ***B.*** A child restrained. ***C.*** Numerous configurations of the Papoose Board. (Photo courtesy of Natus Medical Inc., Pleasanton, CA.)

laws vary on monitoring if the patient is placed in restraints for nonviolent and non–self-destructive behavior. They may be monitored less frequently. Hospital policies determine these parameters so that they meet legal, TJC, and CMS requirements.

AFTERCARE

Take an organized approach to remove the restraints once the patient has calmed down. Talk with the patient about removing the restraints and review the patient's ability to stay calm and follow instructions. This process is known as debriefing.[17] Some Emergency Physicians release all restraints at once.

Releasing the restraints in a stepwise approach may prove safer. Release one of the patient's arms. Inform the patient that if they remain calm the other restraints will be removed. **Observe the patient to ensure that they do not release their other restraints once an arm is free.** Wait a short period of time before releasing the opposite arm if in two-point restraints or their opposite leg if they are in four-point restraints. Wait a short period of time if the patient was in four-point restraints before releasing the remaining two restraints at the same time. An alternative in the four-point restrained patient is to initially remove both leg restraints followed by one arm then the other.

Never leave the patient with only one arm restrained without a staff member at the bedside. This allows too much mobility and may cause harm to the patient and the staff members if the patient becomes combative or falls from the bed.[2] Never leave a patient restrained only by their legs as they can become injured if they fall off the bed.

Discuss with the patient the reason restraints were required.[26] Include in the discussion the alternatives that were first tried and that failed thus requiring physical restraint. Review the behaviors that should be avoided in the future that will prevent the application of physical restraints. Ensure that basic needs (e.g., diet, hydration, and toileting) are met for any patient restrained.

COMPLICATIONS

The use of physical restraints can result in asphyxia, strangulation, and sudden death if they are incorrectly applied or if the patient is not appropriately monitored.[27-31] Patients restrained in the prone position or who are under the influence of stimulants are at an increased risk of complications.[32-34] **Never restrain a patient in the prone position as this can compromise their respiratory function.** Tightly applied restraints or the patient fighting against the restraints can result in abrasions, contusions, extremity fractures, joint dislocations, neurovascular damage, rhabdomyolysis leading to acute renal failure, or skin breakdown.[2] Consider the use of chemical restraint (Chapter 233) to minimize these potential complications and to be able to release the physical restraints.

DOCUMENTATION

Thorough documentation of the incidents involving the use of restrictive physical intervention is essential (Table 232-2).[12] No single set of documentation guidelines has been drawn up to define what exactly to record after such incidents have occurred.[24] The documentation standards for restraint and seclusion episodes include circumstances leading to use, monitoring requirements, and staff debriefing.

SUMMARY

Patient violence occurs in many clinical settings and Emergency Physicians must be prepared to cope effectively with agitated patients to reduce the risk of serious injury to the patient and staff. On occasion, patients display behavioral disturbances that create an imminent danger for themselves and others. Alternatives to physical restraint including verbal de-escalation techniques must be attempted and documented before the initiation of physical restraints. It is necessary at times to physically restrain a person to facilitate their diagnosis, facilitate treatment, and prevent injury to the patient and medical staff. Every restrained patient must be carefully and frequently monitored. Remove physical restraints as soon as possible after reassessment.

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233 Chemical Restraint

David K. Duong and Dara Mendelsohn

INTRODUCTION

Acute agitation, psychosis, and violent behavior are common presentations in the Emergency Department (ED) and pose a threat to patients and the staff caring for them.[1-4] Management of the agitated patient presents a complex challenge of minimizing the risk of potential violence while enabling appropriate clinical evaluation of the agitated state. The underlying diagnosis is often unknown and treatment must often be rendered urgently with limited time for decision making. Such presentations can interfere with the ED evaluation and treatment as well as compromise both patient and staff safety. These behavioral emergencies may require pharmacologic intervention to reduce agitation, resume a more normal physician-patient relation, and facilitate safety (**Figure 233-1**).

The use of chemical restraint implies that medications are used to control behavior and confine a patient's bodily movement without an assessment and treatment plan.[5] This is rarely the case in the ED as medications to manage behavioral emergencies are administered as part of an evaluation and plan of care. **The Emergency Physician (EP) is addressing the medical emergency of agitation and violence in the patient and starting the treatment.** While the phrase "chemical sedation for acute agitation" may be more appropriate, for the purposes of this chapter, the phrase "chemical restraint" will refer to the emergent use of medications to control dangerous behavior in a patient. **Do not use the phrase "chemical restraint" on your charting.** This phrase sets off red flags among personnel from hospital administration, utilization management, and outside official review organizations.

GENERAL CONSIDERATIONS

Behavioral emergencies provide complicated medical and ethical considerations. The benefits of chemical restraint should be seriously considered against the potential side effects of the medication. The EP must be aware of alternatives to chemical restraint as well as the careful assessment, reevaluation, and treatment of the acutely agitated patient. Chemical restraint ideally provides a calming, rather than sedating, effect with a continued emphasis on doing no harm to the patient while simultaneously reducing the risk of violence.[6,7] An objectively good response to chemical restraint may still leave the patient feeling traumatized and angered. **Allow the patient to participate in treatment decisions to the extent possible.** This can be achieved through simple tasks such as asking the patient if they have a medication preference or offering a choice between potential medications.[7] **Attempt further verbal de-escalation or a "show of force" before applying any physical or chemical restraint (Figure 233-1).**[8-12]

EDs should have written policies regarding restraint use and monitoring as mandated by the Centers for Medicare and Medicaid Services (CMS) and The Joint Commission (TJC).[5,13,14] Both CMS and TJC have guidelines for the use of restraints. **The CMS defines chemical restraint as use of any medication to restrict a patient's behavior or freedom of movement but is not the standard treatment or dosage for the patient's underlying condition and strongly cautions against its use for discipline or medical staff convenience. The CMS and TJC recognize the legitimate use of restraint for the protection of the immediate physical safety of the patient and staff. Both organizations identify restrictions**

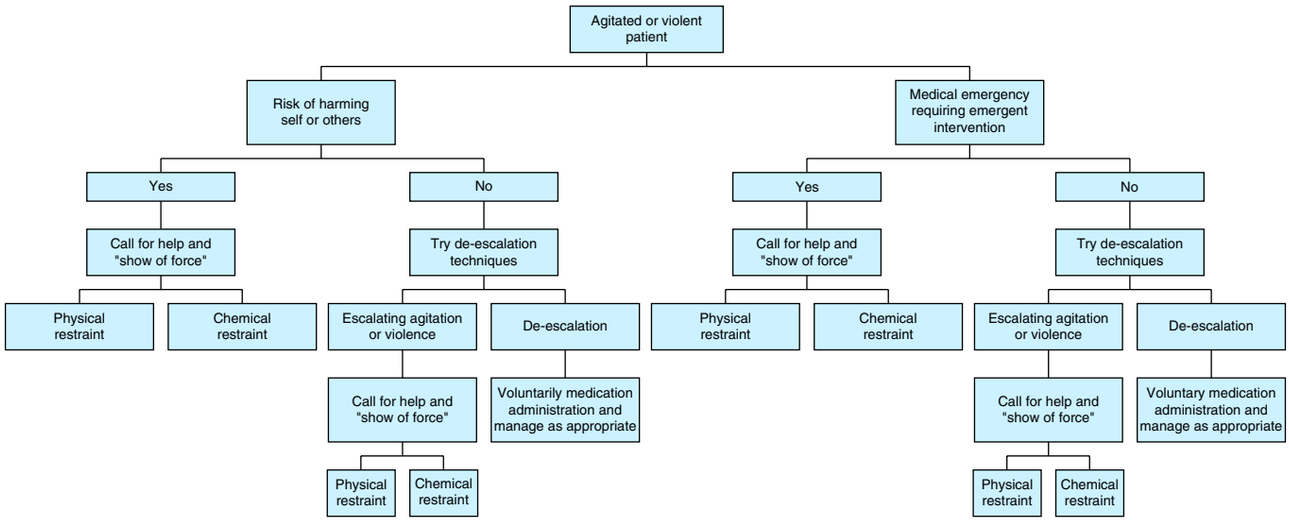

FIGURE 233-1. The management of the agitated or violent patient in the ED.

including that restraints must only be used when less invasive interventions have failed; must be ordered by a licensed clinician (i.e., can be a nonphysician); require the patient to be evaluated by a physician as soon as possible (but must be done within 1 hour); and cannot be written as a standing or as needed order.[5,9,15-17]

A brief consideration of the legal and ethical implications of chemical restraint deserves mention. Consent for evaluation and care is implied during an emergency. The courts assume a competent lucid adult would consent to treatment necessary to maintain health or life.[18] **The EP must consider whether the patient has the decisional capacity to refuse offered care.** The EP should feel comfortable in the treatment of life-threatening emergencies. **It is best to err in favor of treatment if there is doubt to the competency of the nonconsenting patient.**[18] Some leeway must be given to patient preference for treatment if the patient is competent. **The EP must document their thought process and justification for administering chemical restraint.** The covert administration of chemical restraint may be problematic and no universal ethical standard has been established in an ED setting.[19,20] It is advisable to review the ED and hospital restraint protocols that have been vetted through the hospital administration and legal review.

INDICATIONS

There is no precise definition of clinical agitation or violent behavior in the ED that necessitates chemical restraint.[21] Experts have agreed that explosive and/or unpredictable anger, intimidating behavior, restlessness, pacing, excessive movement, physical and/or verbal self-abusiveness, demeaning or hostile verbal behavior, and impulsive or impatient behavior are components of clinically significant agitation.[7,22] **A history of violence regardless of diagnosis, male sex, drug abuse, substance abuse, and alcohol abuse are associated with violent behavior (Table 233-1).**[18,21-24] **However, there is no reliable way to clinically predict which patients will be imminently violent.**[22] The history and physical examination will usually determine which patients are agitated, violent, or have imminently violent potential. Verbally engage the patient to the extent possible to obtain preliminary information and assess their potential for verbal de-escalation. The patient should ideally be asked about their intent to harm themselves or others, if they possess a weapon (e.g., on their person or at home), a history of recent violence, current alcohol or illicit drug use, medical conditions, and psychiatric conditions. **The possibility of violence should be taken seriously and acted upon quickly.**

TABLE 233-1 Causes of Agitation

Infectious	Medications and toxicology		Metabolic		Neurologic	Psychiatric
Brain abscess	Adrenergics	Gamma-hydroxybutyrate	Acute intermittent porphyria	Hypoglycemia	Alzheimer's disease	Agitated depression
Encephalitis	Alcohol (ethanol)	Hallucinogens	Cerebrotendinous xanthomatosis	Hyponatremia	Brain contusions	Anxiety
Meningitis	Amphetamines	Hydrocarbons	Creatine deficiency	Hypothermia	Epidural hematoma	Bipolar depression
Sepsis	Anticholinergics	Lithium	Diabetic ketoacidosis	Hypothyroidism	Huntington's disease	Personality disorders
	Antidepressants	LSD	Hepatic encephalopathy	Hypoxemia	Hydrocephalus	Posttraumatic stress disorder
	Antiemetics	Mushrooms	Homocystinuria	Niacin deficiency	Parkinson's disease	Schizophrenia
	Antihistamines	Opiates	Hypercalcemia	Renal failure	Seizures	
	Antipsychotics	Phencyclidine	Hypercarbia	Thiamine deficiency	Subarachnoid hemorrhage	
	Antispasmodics	Pseudoephedrine	Hyperglycemia	Thyrotoxicosis	Subdural hematoma	
	Aspirin	Sedative-hypnotics	Hypernatremia	Urea cycle disorders	Stroke	
	Atropine	Serotonin agonists	Hypertensive encephalopathy	Wilson's disease	Traumatic brain injury	
	Bath salts	Stimulants	Hyperthermia		Tumors	
	Carbon monoxide	Synthetic marijuana				
	Cocaine	Withdrawal syndromes				
	Ephedrine	Xanthenes				

Chemical restraint may be necessary if verbal interaction is not adequate to alleviate violent behavior.[9,11,12] **De-escalation through verbal communication should be attempted before initiating chemical restraint for most agitated patients. The EP must gauge the potential to manage agitation and violence through verbal and behavioral interaction given the clinical situation and available resources. Not every violent patient can be managed in a stepwise fashion. Do not delay appropriate intervention for violent behavior. The ultimate decision to use chemical restraint, either alone or after less restrictive alternatives, is a clinical decision that must be made at the time the behavior is occurring.[9]**

CONTRAINDICATIONS

Attempts should be made to determine if the patient has any known allergies through a review of the medical record, the history from the patient's family, and any known medical or psychiatric providers. A known allergy to a specific medicine is a contraindication to its use and to the use of other medications within the same class. Use dosing adjustment and caution when administering medications in patients who are children, elderly, debilitated, have comorbid medical conditions including pregnancy, or have a history of side effects with the use of such medications. Contraindications to the specific medication classes are listed in **Table 233-2**. **Chemical restraint is not indicated for a patient who refuses to cooperate or intensely stares. It is also not appropriate to use chemical restraint for punishment or for the convenience of staff.**

EQUIPMENT

- Personal protective equipment (gloves, mask, goggles or face shield, and gown)
- Alcohol pads
- Syringes, various sizes
- Needles, various gauges
- Pharmaceutical agents
- Resuscitation equipment
- Defibrillator
- Oxygen source
- Oxygen masks
- Nasal oxygen cannulas
- Oral airway
- Nasal airway
- Bag-valve mask device
- Continuous pulse oximetry
- Capnography, if available
- Continuous cardiac monitoring
- Intravenous (IV) access supplies
- Equipment for physical restraint

TABLE 233-2 Contraindication to Specific Medications Used for Chemical Restraint[32]

Agent	Contraindications
Lorazepam	1. Hypersensitivity to lorazepam or any component of formulation 2. Acute narrow angle glaucoma 3. Significant sleep apnea 4. Severe respiratory insufficiency
Midazolam	1. Hypersensitivity to midazolam or any component of formulation, including benzyl alcohol 2. Acute narrow-angle glaucoma 3. Concurrent use of CYP3A4 inhibitors (i.e., amprenavir, atazanavir, ritonavir)
Haloperidol	1. Hypersensitivity to haloperidol or any component of formulation 2. Significant Parkinson's disease 3. Severe central nervous system depression 4. Bone marrow suppression 5. Severe cardiac or hepatic disease
Droperidol	1. Hypersensitivity to droperidol or any component of formulation 2. Known or suspected QT prolongation
Risperidone	1. Hypersensitivity to risperidone or any component of formulation
Olanzapine	1. Hypersensitivity to olanzapine or any component of formulation
Ziprasidone	1. Hypersensitivity to ziprasidone or any component of formulation 2. History of (or current) prolonged QT interval 3. Concurrent use of other QTc-prolonging agents 4. Recent myocardial infarction or history of arrhythmia, uncompensated heart failure
Ketamine	1. Hypersensitivity to ketamine 2. Be cautious in patients with known coronary artery disease

PATIENT PREPARATION

Do not attempt to chemically restrain a patient until they are searched for dangerous objects and disarmed by hospital security or police. Identify a single team leader for the chemical restraint process. This is generally the EP who assigns roles and instructs other involved providers.[23,25] Less preparation is required if the patent is voluntarily willing to take oral medications compared to an agitated patient who is unwilling to take oral medications. Safely make attempts to decrease external stimuli (e.g., offering to place the patient in a quieter area).[9] **Consider how many staff members may be needed to intervene and formulate this team prior to any intervention.** It may be prudent to have more staff members readily available if needed. A patient may be so physically combative as to require five staff members in the intervention (i.e., one person to restrain each limb by immobilizing at the major joint and a fifth person to hold the head). Brief the team on what is expected and of the possible dangers. **Universal precautions should always be used to protect the healthcare provider.**

Always approach the patient face-to-face and not from behind or without warning. Staff must appear calm and in control. Speak in a nonprovocative, nonconfrontational, and noncondescending manner. The patient should not obstruct a staff member's ability to exit the room. Staff should initially not have the patient between them and a door until the situation is under reasonable control.

Always leave the door to the room open to facilitate escape if staff feel they are not safe. The open door also allows additional staff to enter and assist. It may be necessary to have security nearby and aware of the situation.

CHEMICAL RESTRAINT AGENTS

Improvements in pharmacologic treatment of the acutely agitated or psychotic patient have been recently made. This has resulted in more medication choices. The medication selected should be based on diagnostic or etiologic considerations, onset and duration of action, efficacy, route of administration available, patient's history of response to the medication, patient preference, ease of administration, and potential for adverse effects (**Table 233-3**).[6,7] The ideal agent for sedation of the acutely agitated patient in the ED is one that is rapidly effective without causing excess sedation, easily administered, available via multiple routes of administration, and safe.[26,27] Below is a summary of the pharmacologic options available for chemical restraint based upon the currently available evidence.

TABLE 233-3 General Choices for Chemical Restraint Based Upon Consensus Guidelines*[7,41,42]

Provisional diagnosis	First-line medication	Second-line medication	Comments
No data available; patient willing to take oral medications	Lorazepam PO (first choice) Risperidone PO	Olanzapine PO Haloperidol PO Quetiapine PO	
No data available; patient unwilling to take oral medications	Lorazepam IM (first choice) Ziprasidone IM Olanzapine IM Haloperidol IM		
Psychotic disorder; unwilling to take oral medications	Haloperidol IM + Benzodiazepine IM or Olanzapine IM	Ziprasidone IM Ziprasidone IM + Benzodiazepine IM Haloperidol IM Benzodiazepine IM	
Schizophrenia	Olanzapine PO Risperidone PO Risperidone PO + Benzodiazepine PO Haloperidol PO + Benzodiazepine PO	Quetiapine IM Ziprasidone IM	If IM medications are required, use Olanzapine, Haloperidol + Benzodiazepine, Ziprasidone, or Ziprasidone + Benzodiazepine
Mania	Olanzapine PO Risperidone PO	Quetiapine PO Ziprasidone PO Haloperidol PO	If IM meds required, use Olanzapine or Haloperidol + Benzodiazepine
EtOH withdrawal or stimulant toxicity Conduct disorder (pediatric)	Benzodiazepine PO or IM Nonpharmacologic means	Benzodiazepine PO or IM	

*The above table is adapted from information based on expert consensus recommendations and should not replace clinical judgment for the needs of a given patient and clinical scenario.

BENZODIAZEPINES

Benzodiazepines have achieved popularity because of their tolerability and safety. These medications bind to the alpha subunit of the $GABA_A$ receptor to enable inhibitory neurotransmission. Lorazepam and midazolam are the most often used benzodiazepines in the ED. They are an effective monotherapy for the initial treatment of the undifferentiated acutely agitated ED patient.[28] Multiple studies have shown that benzodiazepines reduce agitation and are as effective as haloperidol.[7,29] They should be used when no past medical history is available, when no specific treatment is indicated (i.e., personality disorder), or when benzodiazepines have specific benefits (e.g., ethanol intoxication). Benzodiazepines have fewer significant side effects than conventional antipsychotics. They have been associated with excessive sedation and paradoxical disinhibition. Combination therapy with a typical antipsychotic may be more effective than either benzodiazepine agent alone.

Clonazepam, diazepam, and flunitrazepam are benzodiazepines, which have been shown to be potentially effective in reducing agitation. This information is based upon extremely small studies in hospitalized patients with known psychiatric diagnoses. There have been inconsistent results and flunitrazepam is not available in the United States.[28] Diazepam has erratic absorption after intramuscular (IM) administration and its active metabolite makes this medication less useful in the ED. These three benzodiazepines will not be discussed.

Benzodiazepines are contraindicated if the patient has a known allergy to a benzodiazepine (**Table 233-2**). Administer benzodiazepines cautiously and adjust the dose in patients who are children, elderly, debilitated, have respiratory insufficiency or sleep apnea, hepatic disease, or renal disease. The most common side effects of benzodiazepines include excessive sedation and respiratory depression. These occur in over 10% of patients receiving these medications.

EPs should be careful not to overdose benzodiazepines due to with the possible adverse effects of excessive sedation and respiratory depression.[30,31] **Use flumazenil to reverse benzodiazepine-related sedation.** The initial dose is 0.2 mg IV over 30 seconds. This can be redosed at 0.3 mg IV over 30 seconds if the desired level of consciousness is not obtained.[32] The sole use of benzodiazepines was associated with repeated doses for sedation.[31] **Exercise extreme caution if flumazenil is administered. Its use may result in withdrawal seizures if the patient is chronically taking benzodiazepines.**

LORAZEPAM

Lorazepam has excellent absorption after IM administration with a rapid onset of action and a short duration of action (**Table 233-4**). Lorazepam has minimal risks when used as a single dose or for short-term administration.[27] The onset of action varies slightly depending upon the formulation administered. Sedative effects are

TABLE 233-4 Profiles of Benzodiazepines Used for Chemical Restraint[6,7,28,32]

Agent	Dose (mg)	Route	Onset of action (min)	Elimination half-life (hrs)	Metabolism	Excretion	Dosing limit	Side effects
Lorazepam	2 or 4	IV IM PO	5–10 IV 15–30 IM 20–30 PO	10–14	Hepatic	Urine	12 mg/d	Respiratory depression, excess sedation
Midazolam	5	IV IM PO	3–6 IV 10–20 IM 10–30 PO	1–4	Hepatic	Urine	30 mg/d	Respiratory depression, excess sedation

achieved within 15 to 30 minutes of IM administration. Peak plasma concentration is achieved within 60 to 90 minutes with a duration of action of approximately 8 hours.[32,33]

Lorazepam oral (PO) or IM is recommended as first-line therapy for undifferentiated agitation, alcohol-related agitation, and substance-induced agitation.[7] Expert consensus guidelines suggest optimal lorazepam dosing as 1 to 3 mg PO or 0.5 to 3 mg IM. Studies in the acutely agitated patient show that 2 or 4 mg of lorazepam IM is as effective in reducing agitation and violent behavior with significantly fewer side effects compared to 5 mg of haloperidol IM.[7,28,29]

MIDAZOLAM

Midazolam is a water-soluble benzodiazepine with good IM absorption, rapid time to onset, rapid time to recovery, and minimal side effects (**Table 233-4**). Traditionally, this medication has been used more for conscious sedation than rapid tranquilization. Sedation is achieved within approximately 15 minutes with the peak effect occurring at 30 to 60 minutes. The mean duration of action of IM midazolam is 2 hours. This medication is metabolized by the liver and excreted in the urine.[32]

Midazolam has a shorter time to onset of sedation and more rapid time to arousal compared to lorazepam and haloperidol.[34] One potential limitation includes the short half-life requiring frequent redosing to maintain tranquilization.[27,35] Midazolam can cause significant respiratory depression. Midazolam appears to be safest at a dose of 5 mg and without the concomitant use of narcotics.[6,35-37]

COMBINATION THERAPY

Medications can often be combined to achieve synergistic therapeutic effects while reducing the potential for adverse outcomes. Previous studies have focused on combining benzodiazepines with typical antipsychotic agents. Combination treatment allows for lower doses of antipsychotic medication to achieve adequate sedation. A lower incidence of extrapyramidal symptoms may be due to benzodiazepine prophylaxis or treatment of antipsychotic-induced side effects.[33]

The combination of lorazepam and haloperidol is the most commonly studied and utilized combination therapy in clinical practice. **A combination of 2 mg lorazepam with 5 mg haloperidol has been shown to more rapidly reduce agitation than either agent alone. The combination has fewer side effects when compared to higher dose haloperidol.** The combination can be mixed together in the same syringe and administered IM if used immediately.[29,38] It is important to note that studies supporting this data do not control for redosing. **Current American College of Emergency Physicians (ACEP) guidelines recommend that the combination of a benzodiazepine and haloperidol may produce more rapid sedation than monotherapy in the acutely agitated psychiatric patient in the ED.[28]**

There are increasing data to support the use of other benzodiazepine and antipsychotic medication combinations (e.g., midazolam-droperidol and midazolam-olanzapine). A dose of 5 mg of midazolam with 5 mg of droperidol or 5 mg of midazolam with 5 mg of olanzapine were found to be effective.[39,40] Combination treatment resulted in more patients being sedated than with use of just benzodiazepines.[31]

ANTIPSYCHOTICS

Typical antipsychotics (i.e., first-generation or conventional antipsychotics) and benzodiazepines have historically been the mainstay of treatment for acute agitation and psychosis. These medications have been available in parenteral formulations for years. Conventional antipsychotics and benzodiazepines can cause serious adverse effects. The utility of atypical antipsychotics (i.e., second-generation antipsychotics) is expanding as rapid-acting preparations are available. Atypical antipsychotics have differing mechanisms of action, a broader spectrum of response, and a lower side effect profile.[41]

Choosing the initial antipsychotic medication depends upon the patient's mental health and medical history, the need for concurrent sedation, and the side effect profile (**Table 233-5**). **A typical or atypical antipsychotic drug can be used as monotherapy in the acutely agitated patient with known psychiatric illness.** Use an oral preparation of either an antipsychotic or benzodiazepine in the cooperative but agitated patient. Options for the oral treatment of agitation related to schizophrenia or mania include olanzapine, risperidone, risperidone in combination with a benzodiazepine, or haloperidol in combination with a benzodiazepine. Use IM preparations for uncooperative schizophrenic patients. Options include olanzapine, ziprasidone, haloperidol in combination with a benzodiazepine, or ziprasidone in combination with a benzodiazepine. IM benzodiazepines are preferred over newer atypical antipsychotics in patients where no history is available or the underlying illness is undifferentiated.[7,41,42]

TYPICAL ANTIPSYCHOTICS

HALOPERIDOL AND DROPERIDOL

Haloperidol is a butyrophenone antipsychotic. It blocks dopaminergic D_1 and D_2 receptors in the brain. It is the best studied of all the typical antipsychotics. Most studies have been conducted in psychiatric patients with unclear applicability to the undifferentiated acutely agitated ED patient.[28] The peak serum concentration is achieved in approximately 20 minutes after IM administration and in 2 to 6 hours after PO administration. Adequate sedative effects can be expected within 30 to 60 minutes for both IM and IV administration. The duration of effect is approximately 24 hours.[8,32,33]

Dose-response studies have demonstrated that a single haloperidol dose of 7.5 to 10 mg produces the best outcome with the fewest side effects. Higher doses yield lesser degrees of improvement while increasing the risk of adverse effects.[6,7,43] **The utility of haloperidol as monotherapy is limited. The traditional combination therapy of haloperidol with a benzodiazepine was and remains a long-standing first-line option for schizophrenia, mania, and psychosis.** More recent expert consensus recommends second-generation antipsychotics over haloperidol alone or in combination for agitation due to known psychiatric disease.[44]

Droperidol is another butyrophenone neuroleptic that inhibits dopaminergic transmission. It is closely related to haloperidol but only available in a parenteral formulation. It has a rapid absorption, quick onset of action in 15 to 30 minutes, and a short half-life of 2.2 hours.[32,33] The peak effect is achieved within 30 minutes of IM administration and the duration of effect is 6 to 8 hours. A dose of 5 or 10 mg was effective in elderly patients with acute behavioral disturbances.[45] Droperidol has been shown to be more effective at acutely reducing agitation while requiring fewer repeat doses and causing fewer side effects when compared to haloperidol.[33] This medication has been compared to benzodiazepines. Droperidol produces better sedation, requires fewer repeat doses, and results in shorter ED length of stays when compared to lorazepam.[27,28,46-48]

The use of droperidol became controversial in the 2001 after the U.S. Food and Drug Administration (FDA) issued a black box warning regarding the drug's potential for QTc prolongation.[49] Many have criticized the evidence behind the FDA decision.[50] A large cohort study found that there was no increased risk of QT prolongation compared to the general population even if given in large doses of

10 mg or more.[51] The same study showed 70% of patients were effectively sedated after the first dose with a median time to sedation of 20 minutes.

Be aware of relevant side effects associated with typical antipsychotics. This includes extrapyramidal symptoms (EPS), cardiac arrhythmias, and neuroleptic malignant syndrome. Haloperidol has been associated with acute dystonias, parkinsonism, and akathisia.[52] The frequency of EPS associated with haloperidol use varies significantly across studies. It has been cited to affect between 0% and 50% of patients.[29,48] These side effects are of concern and may lead to increased patient agitation and medication refusal. Anticholinergic medications such as benztropine (e.g., 0.5 to 2 mg IM or IV) or diphenhydramine (e.g., 50 mg IM or IV) may be used to prevent or treat EPS. Droperidol has a lower incidence of EPS in the acute setting.[47,48,50] Caution should be used when administering typical antipsychotics in patients with Parkinson's disease, anticholinergic toxicity, phencyclidine (PCP) intoxication, or movement disorders. There was concern that antipsychotic agents lowered the seizure threshold in mice but this has not been found in humans.[18]

Typical antipsychotics cause QT interval prolongation due to their quinidine-like cardiac effects.[8,27] There have been reports of sudden death occurring with chemical restraint using typical antipsychotics. Haloperidol is considered to have a low risk of causing QTc prolongation. **It is important to understand the potential effects on the QTc interval when using antipsychotic medications. The likelihood of QTc interval change is increased when patients are already taking other medications that prolong the QTc or when they have certain comorbidities.** Caution should be used in patients with cardiac disease or those taking antipsychotics chronically. **Obtain an electrocardiogram prior to the administration of a typical antipsychotic if possible.** Withhold typical antipsychotic medication if the QTc is above 500 milliseconds or if the patient has risk factors associated with QT prolongation. **Do not give additional doses if the QTc increased by over 25% after administration.**[27]

There has been additional concern that typical antipsychotics may be affiliated with neuroleptic malignant syndrome (NMS). **NMS is characterized by mental status changes, muscular rigidity, fever, and autonomic instability.** The risk may increase in patients receiving large amounts of typical antipsychotics over a short time. The risk of NMS may be further exacerbated in those who are poorly hydrated and restrained in a poorly ventilated holding room. It has been estimated that NMS is a rare complication seen in approximately 0.2% to 1% of patients using typical antipsychotics.[6,8,33] These patients should be monitored for any physiologic changes.[6,8,33]

ATYPICAL ANTIPSYCHOTICS

Atypical antipsychotics are considered a breakthrough for the treatment of agitation and psychosis with the advent of newer IM formulations (**Table 233-5**). Olanzapine, ziprasidone, and risperidone are currently available in rapid-acting forms that can be used to treat acute agitation. These medications are generally well tolerated and do not result in excessive sedation compared to typical antipsychotics. This can lead to shorter ED stays and increased patient participation in their care. These medications are as effective as the typical antipsychotics with a better side effect profile.[41] Caution regarding QTc prolongation should still follow the general considerations for typical antipsychotics. There are fewer data on the use of these medications in the undifferentiated agitated patient. Most studies have been conducted in selected, consented, and less agitated individuals compared to the unselected and involuntary use in ED patients.[28]

ZIPRASIDONE

Ziprasidone is a benzylisothiazolylpiperazine antipsychotic with serotonin-dopamine antagonist effects. It was the first available atypical antipsychotic in the IM formulation. The IM preparation achieves peak concentration in 30 to 45 minutes and has a duration of effect of at least 4 hours.[27,32,35]

Multiple randomized controlled trials demonstrate that ziprasidone 20 mg IM is effective at rapid tranquillization of the agitated patient with a psychiatric history. There seems to be a dose-response effect as studies have shown increased efficacy when comparing 2 mg, 10 mg, and 20 mg doses. The 20 mg dose is well tolerated without evidence of increased EPS, dystonia, akathisia, respiratory depression, excessive sedation, tachycardia, or clinically significant QTc prolongation.[51,53] Ziprasidone appears to be comparable in efficacy to droperidol, midazolam, and haloperidol in combination with a benzodiazepine.[35,54] It may be more effective and better tolerated than IM haloperidol for treating acute psychosis.

Ziprasidone's high ratio of serotonin-2A to dopamine-2 neurotransmitter affinity results in a low incidence of EPS compared to conventional antipsychotics. Ziprasidone appears to cause greater

TABLE 233-5 Profiles of Antipsychotics Used for Chemical Restraint[7,32,33,41]

Class	Drug	Dose	Route	Onset of action (min)	Elimination half-life (hrs)	Metabolism	Elimination	Dosing limits	Side effects
Typical antipsychotic	Haloperidol	2.5–10 mg	PO IM IV	30–60	18	Hepatic	Urine + feces	30 mg	EPS, NMS
	Droperidol	5 mg	IV IM	10–30 IV 10–20 IM	1–4	Hepatic	Urine + feces	30 mg/d	Prolonged QTc
Atypical antipsychotic	Ziprasidone	10 mg q 2 hr or 20 mg q 4 hr	PO IM	15–20	7 PO 2–5 IM	Hepatic	Urine + feces	40 mg/d	Prolonged QTc
	Risperidone	2 mg q 2 hr	PO	< 90	20	Hepatic	Urine + feces	12 mg/d	Orthostatic hypotension, prolonged QTc at max dose
	Olanzapine	5–10 mg q 2–4 hr	PO IM	180–360 PO 15–45 IM	21–54	Glucuronidation and cytochrome P450–mediated oxidation	Urine + feces	20–40 mg/d PO 30 mg/d IM	Orthostatic hypotension, prolonged QTc at max dose

prolongation of the QTc interval compared to haloperidol, olanzapine, and risperidone.[55] This medication has not been associated with torsade de pointes or sudden cardiac death, and only rarely increases the QTc interval over 500 milliseconds.[27,55] In summary, ziprasidone seems to be a safe and well-tolerated medication for the treatment of acute agitation but should not be used in patients who are at risk for QTc prolongation.

RISPERIDONE

Risperidone is a benzisoxazole antipsychotic medication with serotonin-dopamine antagonist effects. Risperidone has a low risk of EPS due to its serotonin 5-HT_2 and dopamine D_2 antagonism. This medication comes as an orally disintegrating tablet that dissolves within seconds. It achieves peak plasma concentration within 1.5 hours.[32,33]

The combination of 2 mg PO risperidone plus 2 mg PO lorazepam has been shown to be equivalent to 5 mg IM haloperidol in combination with 2 mg lorazepam in reducing agitated psychosis in the emergent setting. Patients receiving the oral combination were also less somnolent and could be appropriately examined. **Such data suggest that oral risperidone is an acceptable agent for the agitated but cooperative patient.[28,56]**

OLANZAPINE

Olanzapine is another atypical antipsychotic with combined serotonin-dopamine antagonist effects. The medication is available in both an orally disintegrating tablet and an IM formulation. The IM preparation achieves peak plasma concentration in 15 to 45 minutes while it takes 5 hours for the PO form.[32,33] It has a duration of action of up to 24 hours.[32,33] The oral disintegrating tablet can be as effective as IM haloperidol.[57]

IM olanzapine effectively treats acute agitation in patients with schizophrenia, bipolar mania, and dementia.[58] Olanzapine appears effective at reducing agitation with a quicker onset of action, while having a lower incidence of dystonia and EPS when compared to haloperidol for the treatment of agitated schizophrenic inpatients.[59] IM olanzapine was shown to be more effective than IM lorazepam at acutely reducing agitation and required less redosing in agitated patients with bipolar mania.[60] IM olanzapine was as effective with quicker onset of action compared to IM lorazepam for the treatment of agitated patients with Alzheimer's disease and vascular dementia.[61] A Cochrane review of olanzapine use for agitation remarked that data for the oral formulation are insufficient and it essentially remains untested. Administration of IM olanzapine has some value in managing acute aggression.[58,62,63]

Pooled analysis of QTc data demonstrated a favorable QTc profile for olanzapine in the treatment of schizophrenia, dementia, and bipolar mania.[64] Studies have showed significantly higher rates of orthostatic hypotension in patients receiving IM olanzapine and ACEP guidelines recommend orthostatic vital signs to be taken if repeated dosing occurs.[27,28,65,66] **Olanzapine has been affiliated with fatalities.** Cardiopulmonary depression, bradycardia, and hypotension were noted in these cases.[7,28,33] These patients tended to have significant comorbidities and often received concomitant benzodiazepines or excessive olanzapine dosing. **A benzodiazepine and olanzapine should not be given concomitantly.[7,28,33]**

KETAMINE

Ketamine is a glutamate *N*-methyl-D-aspartate receptor antagonist and can function as a potent dissociative agent that provides analgesia, amnesia, no respiratory depression, and no hypotension. It has many established uses in the ED including pain control, procedural sedation, and induction for intubation. Studies have explored its utility in the management of refractory agitation in the prehospital and hospital settings. The results have been promising with ketamine demonstrating a rapid onset of sedation, wide therapeutic index, excellent safety profile, and a lack of respiratory depression.[67-69]

Ketamine is available in PO, IM, and IV formulations. It can be given IM if an IV preparation is not available or difficult to obtain.[70] The doses referenced in studies range from 1 to 2 mg/kg IV or 4 to 6 mg/kg IM. The onset of action is approximately 1 to 2 minutes with IV administration and 4 to 5 minutes with IM administration.[71] Ketamine IM has shown a much faster time to sedation compared to IM haloperidol.[71] Duration of action is frequently cited to be 10 to 15 minutes when administered IV but the approximate period of sedation is ill-defined for ketamine-induced sedation. One study required 60% of patients to be redosed or to receive a second agent to maintain sedation.[72] The side effect profile of ketamine includes emergence reactions, sympathomimetic effects (e.g., tachycardia and hypertension), laryngospasm, nausea, and vomiting. Ketamine has been shown to exacerbate schizophrenia, although there is paucity of specific data on exacerbation of other forms of psychosis (e.g., drug-induced). Its sympathomimetic effects may lead some EPs to take pause in patients with known coronary artery disease, although ketamine-induced rises in blood pressure and pulse appear to be insignificant.[73]

The exact setting and patient population that might benefit from ketamine over other agents still needs to be investigated. This is due to a lack of established studies on the efficacy and safety of ketamine to control acute agitation in the ED.[74] There are numerous prehospital studies on the use of ketamine to control agitation.[75,76] They showed that chemical restraint use was associated with endotracheal intubation and hospital admission. The rates of intubation and hospital admission increased with higher doses of ketamine.

MISCELLANEOUS AGENTS

Numerous agents have been tried to chemically restrain a patient. These are small studies with few patients. Two medications deserve mention. Dexmedetomidine has been used when other agents have failed.[77,78] It has been used for sedation of agitated patients related to cannabis and methamphetamine use in case reports. It must be administered by IV continuous infusion. Dexmedetomidine can cause bradycardia and hypotension.

Loxapine oral inhalation powder was FDA approved in 2015. It is supplied in 10 mg single-use inhalers. It is not approved for dementia-related psychosis in the elderly due to an increased rate of death. It can cause bronchospasm and is contraindicated in patients who have asthma, chronic obstructive pulmonary disease (COPD), and other respiratory disorders. It is useful in cooperative patients whose past medical history is known. Loxapine inhalation begins to work in 2 minutes with a half-life of 6 to 8 hours.

ASSESSMENT OF SUCCESS

Consensus guidelines endorse calming without sedation or with mild sedation to the point of drowsiness without sleep as the most appropriate goal.[7,23,79] This allows the resumption of a more functional patient-physician relationship, ensures the ability to obtain informed consent, and ensures the safety of both the patient and the staff. Such an approach may be associated with fewer injuries, shorter length of stay in the ED, and a quicker transition to the next appropriate level of care.[6] Sleep has been previously treated as an endpoint of chemical restraint. It eliminates the potential for patient participation in decision making and is not a guarantee of safety. **The desired therapeutic objective of reduction in agitation or aggression with minimal sedation is to allow for a timely**

assessment and treatment of any medical condition that requires acute intervention.

Further dosing or an alternative medication may be required if success is not achieved with the first dose of medication. The timing of repeated doses depends upon the pharmacokinetics of the medications. Most agents should exert their effects by 30 to 60 minutes. Administer another similar or higher dose of the initial medication if there is no improvement in agitation or violent behavior. **Consider administering lorazepam unless olanzapine was used.** A combination of an antipsychotic and benzodiazepine may be repeated. **Switching medications is only advised after three to four doses of the medication were given with insufficient effect or two doses of medication had no effect. Switching from one antipsychotic to another is generally not advised due to the different adverse effect profiles.**[7,80] An atypical antipsychotic may be used if further dosing is needed and a conventional antipsychotic was administered that resulted in EPS.[81]

AFTERCARE

The staff must continue to interact with the patient in a calm, nonjudgmental, and noncondescending manner after chemical restraint is employed. Patients still require a staff member to observe them throughout their ED stay. The issues of elimination and hydration should be anticipated and addressed. The degree and frequency of vital sign and cardiopulmonary monitoring are dictated upon medical concerns and the patient's clinical status. Sedated patients should have frequent cardiac monitoring, neurologic monitoring, pulmonary monitoring, vital sign monitoring, and respiratory assessments. Initiate aspiration precautions and the changing of positions to prevent pressure sores in the sedated patient. The chemically restrained patient must be evaluated for side effects and adverse reactions of the medication given (e.g., orthostasis, dystonic reactions, akathisias, EPS, and NMS).

Document the indication and description of the chemical restraint process, the outcome, and any complications. Document the less restrictive alternatives that were considered or performed prior to chemical restraint. If it is discovered that a specific person(s) is in danger, they should be warned and the local police notified. This is especially true if there is a situation where it is not possible to prevent a violent patient from leaving the ED (Tarasoff *v.* The Regents of the University of California, Supreme Court of California, 1976).

Psychiatric consultation should be a part of the evaluation of the violent patient. Obtain laboratory analyses and radiologic studies as indicated. The discussion of which patient needs laboratory analyses and radiology examinations depends on the medical situation and local requirements and is beyond the scope of this chapter. Standard medical clearance should be documented. This includes the resolution of abnormal vital signs, resolution of mental status and behavioral abnormalities, stability of ambulation and mobility, and ability to understand and follow discharge instructions.

ALTERNATIVES

The management of the patient exhibiting dangerous behavior usually requires several techniques that are used sequentially or in concert with other techniques. Although some of these techniques may be used as alternatives, they are generally used in conjunction with chemical restraint.

Verbal de-escalation is usually the first approach to the violent patient and may be the only intervention necessary. The overarching principle with verbal de-escalation is that staff convey their professional concern for the well-being of the patient.[18,21,23] Although a full description of verbal de-escalation techniques is not in the scope of this chapter, there are a few critical points to note. Use an approach during the interaction with a violent patient that is nonjudgmental, calm, and neutral. Give assurance that the patient will not be harmed and use empathic statements. Healthcare providers should be clear with limits on dangerous and violent behavior and the actions that will be taken to mitigate such behavior and actions. Avoid lying to or arguing with the patient. These actions can represent a challenge to the patient to justify violent behavior and actions.[21]

A "show of force" is intended to deter dangerous behavior. Have a daunting number of staff and security present in front of the violent patient. The personnel involved with the "show of force" should remain neutral and not provoke the patient. This often allows a single team leader to interact with the patient and de-escalate the behavior.[18,21]

Physical restraint is another alternative that may be employed in place of or temporarily in conjunction with chemical restraint. Both CMS and TJC have standards for use of physical restraints. The use of physical restraints is discussed in Chapter 232.

THE PREGNANT PATIENT

The safety, efficacy, and fetal outcome of using isolated doses of an antipsychotic medication and/or a benzodiazepine during pregnancy have not been directly studied. There is no strong evidence of any teratogenic effects from their use.[82,83] Benzodiazepines are considered safe to use during pregnancy. No definitive association between benzodiazepines and teratogenicity has been shown. The benefits of administering short-term doses of benzodiazepines generally outweigh the risks in emergent situations.[84,85] Most benzodiazepines cross into breast milk but this is not usually a concern in the agitated ED patient who receives short-term therapy.

A consensus panel of expert Emergency Psychiatrists recommends using haloperidol as a first-line agent in the agitated pregnant patient.[42] Successful use of haloperidol and risperidone, with and without lorazepam, has been described.[86] There is limited experience with the use of ziprasidone, olanzapine, and quetiapine in the management of the agitated pregnant patient.[86] Lurasidone is an atypical antipsychotic that, to date, has not been linked to congenital malformations.[86,87] It is currently classified as a class B pregnancy risk factor. There is no published evidence-based data for the use of lurasidone in acute agitation in the ED. Some Psychiatrists currently recommend its use in the agitated pregnant patient. It is only available in the PO form.

Monitor women in their second and third trimester for inferior vena cava obstruction if chemically restrained and placed in the supine position. Place them in the left lateral decubitus position. They may be at an increased risk of aspiration due to lower esophageal sphincter relaxation during pregnancy. The pregnant patient's respiratory status must be carefully monitored. The normal increase in respiratory rate and decrease in tidal volume can be affected if the patient is oversedated or somnolent.

THE PEDIATRIC PATIENT

In general, chemical restraint of the pediatric patient should follow that of the adult.[88,89] **Make every reasonable attempt possible to obtain parental consent for the medical management of a violent pediatric patient. Communication attempts and early discussion with the patient's parent(s) must be pursued and documented if an emergent situation precludes parental consent.** The child should be offered the option of taking the medication orally prior to the IM or IV routes of administration. This may help instill trust and help engage a likely frightened child as the idea of a needle may increase the agitation of a child and family

TABLE 233-6 Medications Used for the Chemical Restraint of Pediatric Patients[86,90]

Drug	Formulation	Dose
Lorazepam	PO/IM/IV	0.05–0.1 mg/kg/dose (max 2 mg/dose)
Diazepam	PO/IM/IV	0.04–0.2 mg/kg/dose (max 10 mg/dose)
Haloperidol	PO/IM/IV	0.025–0.075 mg/kg/dose (max 5 mg/dose)
Risperidone	PO	0.25 mg (school age) to 2 mg (late adolescent)
Olanzapine	PO/IM	2.5 mg (school age) to 10 mg (late adolescent)
Diphenhydramine	PO/IM/IV	1 mg/kg/dose (max 50 mg)
Ketamine	IM/IV	1–2 mg/kg/dose IV; 4–5 mg/kg/dose IM

members.[90-92] If minors are discharged, they should be in the care of a responsible adult who understands and agrees with the discharge plan.

The agents most commonly used for the chemical restraint of the pediatric patient are lorazepam, haloperidol, risperidone, and antihistamines.[7,93] Intranasal administration of midazolam has been studied for conscious sedation but not for chemical restraint. The use of intranasal midazolam cannot be recommended until there is further experience with this route of administration.[94,95] Benzodiazepines may cause paradoxical disinhibition in which there is increased agitation. This is more common in children with developmental delay and organic brain syndromes.[92] There are no data to support the use of stimulants to control behavior for children with established attention deficit disorder.[72]

Use weight-based and smaller doses in children (**Table 233-6**).[96] The use of haloperidol and droperidol in pediatric patients should follow the same guidelines as in adults. These medications are viewed as generally safe in children. Atypical antipsychotic use in pediatric patients is becoming more frequent. Olanzapine is approved for pediatric use and available in IM and orally disintegrating tablet forms (**Table 233-6**). Risperidone is an effective and safe alternative.[71] Experience with ziprasidone use in the ED for pediatric patients is extremely limited and there is no available pediatric dosing for this medication. Diphenhydramine can be an effective agent due to its sedative effect. Extrapyramidal side effects of antipsychotic medications may be treated with diphenhydramine (1.25 mg/kg/dose IV or IM) or benztropine (0.02 to 0.05 mg/kg/dose IV or IM).

THE ALZHEIMER'S PATIENT

Most Alzheimer's patients experience neuropsychiatric symptoms during their disease. Agitation is common and distressing for the patient and their family. The agitation can easily turn to violence. The use of de-escalating techniques can be difficult. The use of standard antipsychotics has been used on the violent and agitated patient with Alzheimer's disease. The side effects of medications in the elderly must be weighed against the benefits. The use of dextromethorphan bromide combined with quinidine sulfate appears to be a promising alternative to standard treatments.[97-99] This combination reduces agitation scores, reduces aggression scores, and improves behavior. Other tested medications include carbamazepine, citalopram, oxycarbazine, and prazosin.[98] It is too early to know whether these agents will be useful for acute intervention in the ED.

SUMMARY

Acute agitation and violent behavior may be due to a variety of medical and psychiatric etiologies. It is not an uncommon finding in ED patients. It may be appropriate at times to use pharmacologic intervention to calm the agitated patient and to facilitate the appropriate diagnostic and therapeutic workup while ensuring patient and staff safety. Typical antipsychotics and benzodiazepines have been the traditional options. The use of atypical antipsychotics is expanding with the development of new rapid-acting formulations with less adverse effects. It is advisable to review hospital and governmental policies regarding chemical restraint given the medical, ethical, and legal complexities involved in the treatment of acute agitation. Emergency Physicians should be aware of the indications, risks, and benefits of chemical restraint. Several recent guidelines are available to guide the Emergency Physician.[11,12]

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